

THE GENOME OF

Drosophila melanogaster

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ACADEMIC PRESS, INC.
Harcourt Brace Jovanovich, Publishers

San Diego New York Boston London Sydney Tokyo Toronto

Supported by National Institutes of Health Grants LM03689 and GM26810 and by National Science Foundation Grant BBES 86-16272 with supplemental support from The Department of Biology, University of California, San Diego; The Genetics Society of America; and the Drosophila Research Community as administered by the Genetics Society of America.

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ISBN 0-12-450990-8

Academic Press, Inc.
1250 Sixth Avenue, San Diego, California 92101-4311

United Kingdom Edition published by
Academic Press Limited
24-28 Oval Road, London NW1 7DX

PRINTED IN THE UNITED STATES OF AMERICA

92 93 94 95 96 97 EB 9 8 7 6 5 4 3 2 1

We dedicate this book to the memory of George Lefevre in recognition of his exhaustive cytogenetic analysis of the X chromosome and in gratitude for his many helpful comments on the manuscript version of this revision prior to his untimely death in January, 1990.

PREFACE

The last twenty years have witnessed a remarkable expansion in the definition of the *Drosophila* genome. The emergence of *Drosophila* as an organism of choice for molecular-genetic investigations of eukaryotic biology has attracted a large number of talented workers to the field, and the rapid advances in molecular technology have provided new and sophisticated tools and generated novel kinds of information.

This work is a revision of "The Genetic Variations of *Drosophila melanogaster*" by D. L. Lindsley and E. H. Grell, which appeared in 1968 and was essentially a complete catalogue of mutations and chromosome rearrangements of *Drosophila melanogaster* as of the end of 1966. The present volume purports to be such a catalogue current until the end of 1989. The illustrations are primarily the work of Edith M. Wallace, the artist employed by T. H. Morgan; they were mostly drawn between 1920 and 1940. The same illustrations were used in "The Genetic Variations of *Drosophila melanogaster*" and its predecessor "The Mutants of *Drosophila melanogaster*" by Calvin B. Bridges and Katherine S. Brehme. At the time of the 1968 publication, genes were identified exclusively through the existence of mutant alleles; the only wild-type alleles considered were electrophoretic variants of a few enzymes, and the only gene for which there was any molecular information was *bb*. Amino-acid and nucleotide sequencing and the polymerase chain reaction were concepts for the future; cloning of DNA sequences had not been imagined; transposable elements, hybrid dysgenesis, and transformation were unsuspected. These technologi-

cal advances have shifted the emphasis to normal gene structure and function rather than exclusive consideration of mutant alleles. This shift in emphasis is reflected in the title of the present volume, "The Genome of *Drosophila melanogaster*." The ability to identify a gene from either its protein product or the homologous product from another species, rather than the converse, has led to the discovery of many new genes for which no variant had been previously recognized. In addition, new genes with interesting expression patterns are being discovered in enhancer-detection lines.

Interim versions of the majority of the material contained herein have appeared in the form of volumes 62, 64, 65, and 68 of *Drosophila* Information Service. This volume contains information on upwards of 4000 genes and 9000 chromosome rearrangements. There are categories of effects, little if at all represented in the 1968 edition, that have assumed major proportions in the present version. These include developmental mutations, behavioral mutations, female-sterile mutations, meiotic and mitotic mutations, Y-autosome translocations, and transposable elements; in addition, many regions of the genome have been subjected to saturation mutagenesis so that large numbers of lethally mutable loci have been identified and deficiency mapped. A major consequence of the mapping efforts, utilizing both chromosome rearrangements and *in situ* hybridization, is that the polytene map has displaced the recombination map as the more useful standard. The 1968 volume was subdivided into seven sections: Mutations, Chromosome Aberrations,

PREFACE

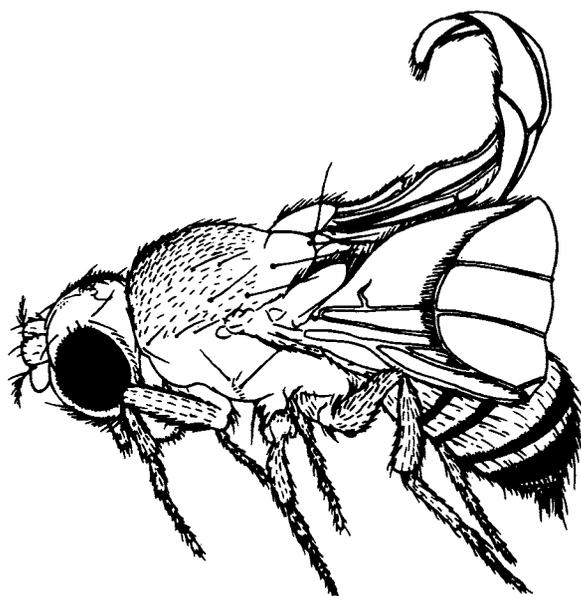
Special Chromosomes, Cytological Markers, Departures from Diploidy, Nonchromosomal Inheritance, and Wild-Type Stocks. In the present version, the section on Wild-Type Stocks has been eliminated, new sections on Transposable Elements and DNA Sequences have been added, and a molecular biology category has been added to the descriptions of genes and rearrangements if the information is available.

We are grateful to our colleagues throughout the world for their contributions and corrections to draft copies. Those who have submitted entries or sections of entries are acknowledged on the first line of the entry. Special thanks are due to a number of colleagues whose efforts on behalf of this volume have been more than substantial. In particular, Jeff Hall has provided almost all of the material on behavioral and neuronal genes; George Lefevre and especially his colleague, Catherine Coyle-Thompson, provided

massive amounts of information and corrections to the sections on X-linked lethals; Trudi Schüpbach provided descriptions of female-sterile and maternal-effect-lethal mutations. Jim Boyd and Scott Hawley provided the entries on mutagen-sensitive and meiotic mutants, respectively. Michael Ashburner has been especially helpful in keeping us supplied with his encyclopedic lists of mutations, chromosome rearrangements, and references; in addition, he has gone over the draft copies of the work and provided detailed additions and corrections. Finally, Loring Craymer and Abraham Schalet were most helpful in reviewing material and pointing out errors and omissions.

We apologize for the omissions, inconsistencies, and errors in this compilation. Every time we reread it we find new ones, but, mercifully, revision has to stop sometime.

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GENES

LOCI. When the last edition of this book was prepared, it was necessary to have two alleles of a gene in order to identify and map a locus genetically. The subsequent development of methods for identifying and mapping loci by gene-dosage manipulation and by *in situ* hybridization with cloned probes has led to the identification and cytological localization of many genes for which no allelic variability has yet been detected. Thus, genes are now recognized by virtue of a phenotypic response to the dosage of a specific circumscribed chromosome segment, by the *in situ* hybridization of a specific transcribed sequence to a polytene-chromosome band, or by the existence of allelic variation. Each locus so recognized is given a name which is descriptive of its mutant phenotype or its wild-type function. The name is concise and is preferably a simple noun or adjective; for example, cabbage, canoe, and kidney or Curly, outstretched, pink and rough. Loci recognized by virtue of the protein that they encode are named as the protein; e.g., Alcohol dehydrogenase, Actin, Calmodulin, etc.

The phenotype of flies obtained from natural populations is considered normal or wild type, and the alleles carried by such individuals, the normal or wild-type alleles. When the main characteristic of the nominate mutant allele is recognized when it is heterozygous with a wild-type allele, the mutant is considered dominant and its name begins with an upper-case letter; when the nominate allele is recessive, an initial lower-case letter is used. The names of genes specifying proteins, for many of which only the wild-type allele is known, begin with upper-case letters. Cases arise in which the same locus has received two or more names; other things being equal, the earlier-applied name is adopted.

For convenience, a symbol is assigned to each locus. This symbol is an abbreviation of the name that uniquely designates the locus in question; it combines brevity with

information. It usually begins with the same letter as the name, is always italicized, and does not contain subscripts, or spaces; e.g., *r* for rudimentary, *R* for Roughened, *ro* for rough, *rs* for rose, and *ry* for rosy. In designations of genotypes with several mutant genes, symbols of genes on the same chromosome are separated by spaces (e.g., *y w f B*); symbols of genes on homologous chromosomes are separated by a slash bar (e.g., *y w / f B*); symbols of genes on nonhomologous chromosomes are separated by semicolons and spaces (e.g., *bw; e; ey*). Names are not italicized in text.

ALLELES. The alternatives or alleles at a particular genetic locus are designated by the same name and symbol and are differentiated by distinguishing superscripts. The superscript notation designating alleles may take a number of different forms. A common device is an abbreviation that further characterizes the particular allele or that was used as the locus symbol before allelism was established. This practice is avoided because it has the disadvantage of preempting useful symbols and names from use as locus designations. Another unacceptable device is the use, as superscripts, of elements of the genotype in which the allele arose, since such a designation implies something more than a trivial connection between allele and element. Finally, lengthy acquisition numbers are avoided as allelic designations, since the information that they contain is of no use to the general user, and greatly exceeds what is necessary to differentiate one allele from another; in the present version, many such allelic designations have been abbreviated. In a large number of cases we have replaced complex, and to most users uninformative, superscripts with simple numerical designations; we have not, however, been consistent in this practice. Other acceptable superscripts for allelic designations are arbitrary numbers, capitalized initials of the finder or laboratory, or the date of discovery.

The numeral 1 is the implied superscript of nonsuperscripted symbols. Whereas genes in the same allelic series are designated by the same symbol but with different superscripts, mutants with similar phenotypes at different loci are not given the same symbol.

For a recessive allele of a preponderantly dominant series or a dominant allele of a predominantly recessive series, the superscripts *r* and *D*, respectively, may be used; e.g., Hn^r , Hn^{r2} , and bw^D . Finally, for the normal allele in a series, a superscript plus sign may be used; e.g., b^+ or B^+ . The plus symbol alone implies the normal (wild-type) allele or alleles in any context, such as $y/+$ or $y\ m\ fl/+$. Absence of a particular locus may be noted by use of a superscript minus sign with the symbol; e.g., bb^- . Revertants or partial revertants of mutant alleles are designated by the superscript *rv* followed by a distinguishing number; revertants of dominant mutations that are deficiencies are treated not as alleles but as deficiencies and are accordingly not superscripted but listed with the distinguishing number.

Loci encoding specific polypeptides or transcripts require special conventions. In many instances, such loci lack recognized allelic variants, but a single wild-type allele is known; in others, polymorphisms exist in such attributes as electrophoretic mobility, abundance, or stability, or mutants affecting activity, developmental-stage specificity, or tissue specificity may occur; these are designated as alleles in the standard manner; e.g., Adh^F and Adh^S . Alleles specifying the absence of a particular enzyme or other protein are designated by the superscript *n* (null) followed by a distinguishing number or letter, e.g., Adh^{n1} or where lack of function is inviable by *l* (lethal), e.g., Nrg^{l1} . All such loci identified by virtue of their wild-type gene product are treated as dominants and are thus named and symbolized with initial upper-case letters. Since all the genes described in this compilation are *Drosophila* genes, we have not used an initial "D" to designate the *Drosophila* homologues of genes originally characterized in other species. Abbreviations for the protein and the gene are frequently identical, and both are used in most discussions. The gene symbol may be differentiated from the protein symbol by having only its initial letter capitalized and by being italicized, whereas the protein symbol is in roman capitals; e.g., ADH.

In several instances where two members of the same allelic series were formerly given different locus names, both are here included under one name; e.g., $Pm = bw^{VI}$. In other cases, we assume allelism of mutants with similar phenotypes and genetic positions even though they have not been tested for phenotypic interaction. In such instances, the basis for the assumption is usually noted. Since the practice has not been consistent, some alleles may be described as different genes. Except in special cases, investigation of allelic interaction of sex-linked recessive lethals is not feasible; consequently, they are often given distinctive symbols where allelism may actually exist.

TRANSFORMANTS. Loci transposed to new chromosome locations by transposable elements are enclosed in brackets to indicate that they are not in their normal position, followed by a parenthetical indication of their new position; e.g., $[w^+](35BC)$ and $[ry^+](sd)$. As such constructs become more complex, a complete description cannot be incorporated into the symbol. Accordingly,

our policy is to sacrifice information in order to keep the symbol as simple as possible; thus, transformants of genes of interest selected by cotransformation with a selectable marker are designated according to the gene of interest rather than the selectable marker; e.g., $[Cp16](52D)$ designates an insertion of Chorion protein 16 into 52D, which was selected by cotransformation of ry^+ .

MIMICS. Mutants at different loci sometimes have similar phenotypic effects. Such loci may be handled in several ways. The simplest is to give each a distinctive name (e.g., vermilion, cinnabar, scarlet, karmoisin, cardinal); this method has the effect of scattering such mimics throughout the alphabetical listing. Or a common symbol followed by a distinguishing symbol may be used (e.g., $tu-1a$, $tu-1b$, $tu-2$ for genes controlling production of melanotic pseudotumors). Loci encoding proteins of similar function are differentiated by arbitrary numbers (e.g., $Sgs3$, $Sgs7$, $Sgs8$), by polytene chromosome position (e.g., $Act5C$, $Act42A$, $Act57A$, etc.), or by molecular weight (e.g., $Hsp68$, $Hsp70$, $Hsp83$, etc.). Distinctive suffixes are also useful (e.g., rough, roughoid, roughish, roughex; plexus, Plexate; dachs, dachsous; maroon, maroonlike). The latter schemes frequently have the virtue of placing like phenotypes or gene functions in sequence in an alphabetical listing. Some phenotypes result from mutation at many loci in all chromosomes; these are given a common symbol followed by a parenthetical designation of the chromosome and then by a distinguishing designation. Examples of this type of mutant are the female steriles, the lethals, the Minutes, and the male steriles [e.g., $fs(2)B$, $l(1)1Ac$, $M(1)18C$, $ms(2)73d$, respectively]. We endeavor in this work to replace arbitrarily chosen distinguishing designations with polytene locations where possible. This has become feasible as the result of remarkable strides in cytogenetic mapping made possible by the selection characterization and maintenance of many deficiencies and by *in situ* hybridization.

MODIFIERS. The primary effect of some mutants is to cause another mutant to exhibit a more-extreme departure from normal (enhancer) or a more nearly normal phenotype (suppressor). Such mutants are symbolized *e* or *E* and *su* or *Su*, followed in parentheses by the gene modified. Designation of the particular allele modified appears as a superscript within the parentheses and alleles of the modifier gene as superscripts outside the parentheses; e.g., $su(lz^{34})$ and $su(Hw)^r$. Terms such as dilutor, exaggerator, inhibitor, intensifier, and modifier were also formerly used, but we have usually attempted to classify such genes as enhancers or suppressors.

FORMAT. Mutants with their descriptions are listed alphabetically according to symbol and cross-indexed according to name. Current terminology is listed in bold face. All cases of synonymy are also listed in italics with cross-references to current usage. Mutants known to be lost are preceded by an asterisk; however, mutants not preceded by an asterisk are not therefore known to be extant. Each mutant is described according to the following format:

symbol: name (Author of entry)

location: The location is indicated by the chromosome number, separated by a hyphen from the genetic position

on the chromosome. Three levels of accuracy of the genetic location are indicated, those carried to tenths of a unit being the more accurately determined; e.g., 3.0 represents a more accurate location than 3. In regions saturated for mutants, map positions may be given in hundredths, either as the result of detailed recombinational mapping or by interpolation using deficiency mapping data. Map positions enclosed in braces are inferred from cytological map position. Accuracy of a map position determination is of course dependent on the accuracy of the positions assigned to the reference markers; i.e., on the accuracy of the map. We treat the map as a rough guide to the relative positions of loci but, considered on a refined level, it may be inaccurate with respect to both position and order of genes. Intense activity in determining cytological positions in recent years is resulting in the rapid replacement of the genetic map by the cytological map as the more useful indicator of gene position.

origin: For induced mutants, the agent is given; mutants recovered from untreated parents or a wild population are listed as spontaneous. Isoallelic variants found as major components of stocks or populations are listed as naturally occurring alleles. Mutagenic agents are frequently abbreviated, especially in tables of alleles; abbreviations used are indicated in the accompanying table:

abbreviation ^α	compound
CB 1246	triethylmelamine
CB 1414	nitrogen mustard
CB 1506	2-chloroethyl methanesulfonate
CB 1522	2-fluoroethyl methanesulfonate
CB 1528	ethyl methanesulfonate
CB 1540	methyl methanesulfonate
CB 1592	S-2-chloroethylcysteine
CB 1735	S-mustard
CB 2041	1:4-dimethanesulfonybutane
CB 2058	1:4-dimethanesulfonybut-2-yne
CB 2348	1:4-dimethanesulfony-1:4-dimethylbutane
CB 2511	D-1:6-dimethanesulfonyl mannitol
CB 2628	L-1:6-dimethanesulfonyl mannitol
CB 3007	DL-p-N,N-di-(2-chloroethyl)aminophenylalanine
CB 3025	L-p-N,N-di-(2-chloroethyl)aminophenylalanine
CB 3026	D-p-N,N-di-(2-chloroethyl)aminophenylalanine
CB 3034	p-N-N,di-(2-chloroethyl)aminophenylethylamine
CB 3086	strylquinoline
DCE	dichlorethane
DEB	diepoxy butane
EI	ethylenimine
EMS	ethyl methanesulfonate
ENU	ethyl nitrosourea
HCOH	formaldehyde
HD	hybrid dysgenesis
HMS	hycanthon methanesulfonate
ICR170	2-methoxy-6-dichloro-9-(3-ethyl-2-chloroethyl amino-propylamino)acridine dihydrochloride
MMS	methyl methanesulfonate
MR	male recombination factor = P element
NMS	nitrogen mustard
NMU	nitrosomethyl urea
NNG	N-methyl-N-nitro-N-nitrosoguanidine
P	P-element hybrid dysgenesis
SMS	sulfur mustard
spont	spontaneous
TEM	triethylmelamine

^α CB Chester Beatty; ICR Institute for Cancer Research.

The chromosome of origin of mutations is of interest when DNA sequence is being studied; such information is not generally available, however, and is usually not included.

discoverer: Name, date of discovery.

synonym: Alternative symbol or name or both, mostly obsolete terminology.

references: Sources of the major descriptive material are listed, but bibliographic material may also appear in some of the other categories. The second reference to a paper in an entry is generally abbreviated to just the author's name or to name and year. Such abbreviated references not preceded by a fuller reference in the same entry are generally to unpublished information. References to CP552 refer to Carnegie Publication 552, which is "The Mutants of *Drosophila melanogaster*" by C.B. Bridges and K.S. Brehme; CP627 refers to "Genetic Variations of *Drosophila melanogaster*" by D.L. Lindsay and E.H. Grell.

phenotype: The most important departures from normal, which are usually those suggested by the name, are described first. Other information about the phenotype follows, and finally there may be data on viability and fertility. This revision contains considerable information on the normal functions of the genes described, including observations on stage and tissue specificity of expression; the techniques of *in situ* hybridization and immunostaining of whole embryos and sectional material have added a new dimension to phenotypic description. The last item in the phenotypic description is the rank, abbreviated RK. In this revision, we have not attempted to assign rank to mutants; however, we have retained those assignments appearing in early editions. Mutants were classified by Bridges into three different ranks according to their utility in experiments in which counts are made: RK1 mutants are easily scored; RK2 mutants are usable but less convenient; RK3 mutants have limited usefulness. An RK3 mutant may be one with good expression and viability but simply not convenient to use in counting experiments; e.g., enzyme polymorphisms. The letter A follows the rank of mutants associated with chromosome aberrations.

alleles: Rather than describing alleles in separate entries, as was done in previous versions, we have attempted, wherever possible, to tabulate them. Different grouping of the types of information itemized above appear as columns in the tables of alleles. When a type of information, such as phenotype, is too extensive for tabulation, subentries follow the nominate entry. The types of information included in each table are decided on a case-by-case basis, but the order of columns approximates the order in which information is included in full entries. Deficiencies may be listed in tables of alleles for the purpose of cross-referencing.

cytology: This category is primarily to provide the cytological location of the gene, as determined by rearranged breakpoint-associated alleles, by deficiency mapping, or by *in situ* hybridization to polytene chromosomes. It may also indicate that the mutant was induced in a rearranged chromosome or occurred in association with a *de novo* rearrangement; in tables of alleles, pre-existing rearrangements are listed by name; *de novo* rearrangements likely to have caused the mutant receive the same designation as the mutant, and are listed by breakpoints in tables and by name in the section on chromosome rearrangements.

molecular biology: This is a new category of information that is expanding at an unprecedented rate. It includes references to the cloning, restriction mapping, sequenc-

ing, and conceptual amino-acid sequences with indicated homologies to other proteins and motifs. In allele tables, it includes sequence alterations associated with different alleles. Molecular mapping results are currently presented without regard to an established convention. Zero coordinates have frequently been chosen at sites not easily identified in a normal chromosome complement, such as a rearrangement breakpoint, the site of insertion of a transposable element, or the end of a random-shear fragment. We propose that the midpoint of an endonuclease restriction site, preferably one shown to be present in chromosomes of several independent origins, be chosen as the origin of a restriction map and that 0 marks not a nucleotide pair, but the plane of symmetry of the restriction site; ambiguity can result only from restriction site polymorphism. We also propose that when the chromosomal orientation of the map is determined, positive values extend to the right and negative values to the left so that all restriction maps have the same orientation; this

will be especially useful when adjacent restriction maps fuse. When two studies of the same region have used different sets of coordinates, we have perforce chosen the one that conforms more closely with the above conventions. No convention has been established for defining the origin of nucleotide maps of transcription units, and of course either orientation with respect to the restriction map may obtain, depending on which strand is transcribed.

other information: This category contains miscellaneous information that does not fit into one of the other categories.

Loci that share phenotypic and nomenclatural features (i.e., mimics) are frequently presented in a single entry in which the common information is presented once, and the information that distinguishes among loci is tabulated; the order of the columns of information roughly corresponds to the order in which the same categories of information appear in full entries.

a: arc

location: 2-99.2.

discoverer: Bridges, 12e24.

references: Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 202 (fig.).

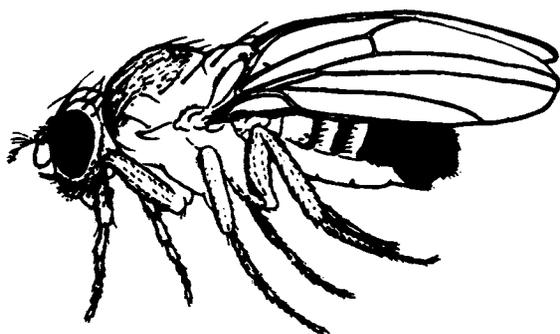
Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 212 (fig.).

Bridges, 1937, Cytologia (Tokyo), Fujii Jub., Vol. 2: 745-55.

phenotype: Wings broader; bent downward in slight, even arc; edges drawn down to diamond shape. Sometimes in stock, wings are bent upward instead of downward. Crossveins closer together. RK2.

alleles: a^1 , a^{ba} (see below), $*a^{ba1}$, $*a^{ba2}$, $*a^{ba3}$, $*a^{ba4}$, $*a^{bad6}$, $*a^{badp}$, $*a^{bar}$, $*a^{Ba}$, $*a^{BaC}$, $*a^{Bap1}$, $*a^{Bap2}$, $*a^{BaX}$, $*a^{Bay}$ (Goldschmidt, 1945, Univ. Calif. Berkeley Publ. Zool. 49: 351-56, 388-89, 519; CP627), and $*a^{M60}$ (Meyer, 1963, DIS 37: 50).

cytology: Placed between 57F11 and 58E4 on the basis of its inclusion within $Df(2R)M-1 = Df(2R)57F11-58A1;58F8-59A1$ but not $Dp(2;3)P = Dp(2;3)58E3-4;60D14-E2;96B5-C1$ (Bridges, 1937). Likely in band 58D6 or 7 based on $Df(2R)a-ba2 = Df(2R)58D5-6;58D7-8$.



a: arc

From Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 148.

 a^{ba} : arc-broad angular

origin: Spontaneous.

discoverer: Goldschmidt, 1934.

synonym: Referred to as *bran: broad angular* by Goldschmidt, but shown by him to be an allele of *arc*.

references: 1945, Univ. Calif. Berkeley Publ. Zool. 49: 351-56, 388-89, 519.

phenotype: Wings broader and shorter than wild type, blunt at the tip. Frequently shows upturned posterior scutellar bristles. In combination with svr^{poi} , produces soft blistered wing. Other interactions described by Goldschmidt, 1945, table 74. Wing grows in pupal stage to full length and then retracts, possibly with histolysis [Goldschmidt, 1934, Z. Indukt. Abstamm. Vererbungsl. 69: 38-131 (fig.)]. RK2.

cytology: Salivary chromosomes normal (Kodani).

other information: Claimed to recur repeatedly in certain lines (Goldschmidt, 1945).

α^1 : see *tyr1*

***A: Abnormal abdomen**

location: 1-4.5.

discoverer: Morgan, 11g.

synonym: *Abnormal*.

references: 1915, Am. Naturalist 49: 384-429 (fig.).

Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. No. 237: 27 (fig.).

phenotype: Tergites and sternites raggedly incomplete, exposing a thin crinkled cuticle; bristles and hairs on abdomen correspondingly eliminated. Highly variable, wild phenotype in old dry cultures. $A/+$ less extreme than A/A and A male; homozygous female fully viable and fertile. RK2 in well-fed cultures.

alleles: $*A^1$ (Morgan and Bridges, 1916), A^{53g} (see below), A^{70} [allelism conjectural (Gooskov, 1971, DIS 46: 41)].

other information: Lost by reversion to wild type.

A : see bw^A

A53g

location: 1- (between y and w ; may not be allelic to A).

origin: Spontaneous.

discoverer: Hillman, 53g.

references: 1953, DIS 27: 56.

1973, Genet. Res. 22: 37-53.

1977, Amer. Zool. 17: 521-33.

Hillman and Barbour, 1963, Proc. Intern. Congr. Genet., 11th, Vol. 1: 170.

phenotype: Epidermal foldings of abdomen abnormal. Tergite formation incomplete, ranging from loss of tergites 2-8 in extreme cases to loss of lateral part of tergite in one or more segments. Expression in $A53g/A53g$ females $> A53g/Y$ males $> A53g/+$ females. Expression maternally influenced (Shafer and Hillman, 1974, J. Insect Physiol. 20: 223-230). Highly variable; sensitive to modifiers on X , 2, and 3, including $E(A53g)$ on 2L. Sensitive to culture conditions; expression reduced in old cultures and under conditions of crowding, low temperature (TSP in late second and early third instar), and low humidity. Also reduced by agents that inhibit RNA or protein synthesis or oxidative phosphorylation (Hillman, Shafer, and Sang, 1973, Genet. Res. 21: 229-38). Supernatants from homogenates of $A53g$ -bearing adults stimulate amino acid incorporation and aminoacylation of tRNA more than those from wild type (Rose and Hillman, 1969, Biochem. Biophys. Res. Commun. 35: 197-204). Mutant late pupae and adults show increased concentrations of soluble protein. Expression of biochemical phenotype correlated with that of visible phenotype (Rose and Hillman, 1973, Genet. Res. 21: 239-245). RK2 in young cultures.

cytology: Deficiency analysis places $A53g$ in 3A5 (Hillman), which is at variance with the genetic position of A .

$a-3$: see $a(3)26$

***A-p: Abnormal abdomen-polygenic**

location: Polygenic.

discoverer: Sobels, 49i.

references: 1950, DIS 24: 62.

1951, DIS 25: 75-76.

1952, Genetica 26: 117-279 (fig.).

1952, Trans. Intern. Congr. Entomol., 9th, Vol. 1: 225-30.

synonym: AA ; Asy : *Asymmetric*.

phenotype: Incomplete mediodorsal fusion and on-sided reduction of tergites. When more than one tergite is abnormal, spiral segmentation types are most frequent. Expression strongly dependent on environment. Penetrance and expressivity correlated (Bezem and Sobels, 1953, Koninkl. Ned. Akad. Wetenschap., Proc. Ser. C. 56: 48-61). In strains selected for penetrance of *A-p*, mediodorsal fusion or asymmetrical reduction of head and thorax also occur. RK3.

***a(1)48: abnormal abdomen in chromosome 1**

location: 1- (not located).

origin: Spontaneous.

discoverer: Zimmerman, 1948.

references: 1952, DIS 26: 69.

1954, Z. Indukt. Abstamm. Vererbungs. 86: 327-72 (fig.).

phenotype: Used to describe three X chromosomes with little or no effect of their own but which increase the incidence of abdominal malformations in crosses with *a(2)* and *a(3)*. Viability and fertility good. RK3.

alleles: The three chromosomes designated **a(1)48*, **a(1)50*, and **a(1)51* (CP627). Genetic relations not worked out.

a(1)HM26

location: 1-(y-cv).

origin: Induced by ethyl methanesulfonate.

synonym: *l(1)HM26*.

references: Mayoh and Suzuki, 1973, Can. J. Genet. Cytol. 15: 237-54.

phenotype: Missing or reduced sternites; missing or angled tergites; black specks on ventral surface of abdomen in about one-third of males at 22° and more than half of males at 17°. Viability reduced at 17° relative to that at 22°.

a(1)HM27

location: 1-(near y).

origin: Induced by ethyl methanesulfonate.

synonym: *l(1)HM26*.

references: Mayoh and Suzuki, 1973, Can. J. Genet. Cytol. 15: 237-54.

phenotype: Same as *a(1)HM26*; more severe at 17° than at 22°. Viability slightly reduced at 17° relative to that at 22°.

***a(2)48**

location: 2- (not located).

origin: Spontaneous.

discoverer: Zimmerman, 1948.

references: 1952, DIS 26: 69.

1954, Z. Indukt. Abstamm. Vererbungs. 86: 327-72 (fig.).

phenotype: Abdominal irregularities most frequently involve anterior segments. Penetrance 7%. Also shows maternal effect. Viability and fertility good. RK3.

alleles: Second chromosomes with some or all of these effects are **a(2)50*, **a(2)51*, and **A(2)51*. Genetic relations not worked out.

a(3)26: see *abd*

***a(3)48**

location: 3- (not located).

origin: Spontaneous.

discoverer: Zimmerman, 1948.

references: 1952, DIS 26: 69.

1954, Z. Indukt. Abstamm. Vererbungs. 86: 327-72 (fig.).

phenotype: Only a maternal effect affecting 2.5% of progeny. Irregularities most frequently involve posterior segments of abdomen. Viability and fertility good. RK3.

A34: see *bw*^{V6}

aa: anarista

location: 3-0.

discoverer: Bridges, 23d10.

synonym: *al-b: aristaless-b*.

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 218.

phenotype: Aristae bare or tufted. Wings somewhat broader than wild type. Expression variable, overlaps wild type often in female and sometimes in male. RK3.

cytology: Placed between 61E2 and 62A6 on basis of its inclusion in *Df(3L)D = Df(3L)61E2-F1;62A4-6* from *T(Y;2;3)D*.

Aa: Altered abdomen

location: 1- (not located).

origin: X ray induced in the *In(1)dl-49, y w f* component of *C(1)DX*.

discoverer: Cicak, 56f.

references: Cicak and Oster, 1957, DIS 31: 80.

phenotype: Heavy deposition of melanin in tergites of females and males. *Aa* detachment-bearing males sterile. RK2A.

cytology: Possibly associated with a rearrangement in addition to *In(1)dl-49*.

AA: see *A-p*

ab: abrupt

location: 2-44.0.

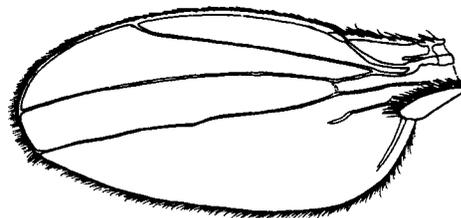
origin: Spontaneous.

discoverer: Bridges, 16j16.

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 218 (fig.).

phenotype: Vein L5 usually stops after posterior crossvein. Scutellar bristles usually fewer. Wing effect probably acts during contraction period (Waddington). Overlaps wild type. Expression more severe in females than in males and when pupal stage takes place at 20° than at 25°. TSP during the first 10% of pupal stage. (Thompson, Bruni, Carbonaro, and Russo, 1988, DIS 67: 86). RK2.

alleles: *ab*¹, *ab*² (see below), *ab*^{51g}, like *ab*² in *In(2L+2R)Cy*; **ab*^{l-60h}: *abrupt lethal* (CP627).



ab: abrupt

Edith M. Wallace, unpublished.

ab²

origin: Spontaneous.

discoverer: Bridges, 23g6.

synonym: *pt*: *parted*.

references: Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 232.

phenotype: Vein L5 does not reach margin. Scutellar bristles always fewer than wild type. Hairs parted down midline of thorax and abdomen. Supra-alar bristles sometimes absent. Coxae tend to be thickened. Males sterile and have rotated genitalia. *ab/ab²* resembles *ab/ab* but has a stronger bristle effect. RK2.

abb: abbreviated

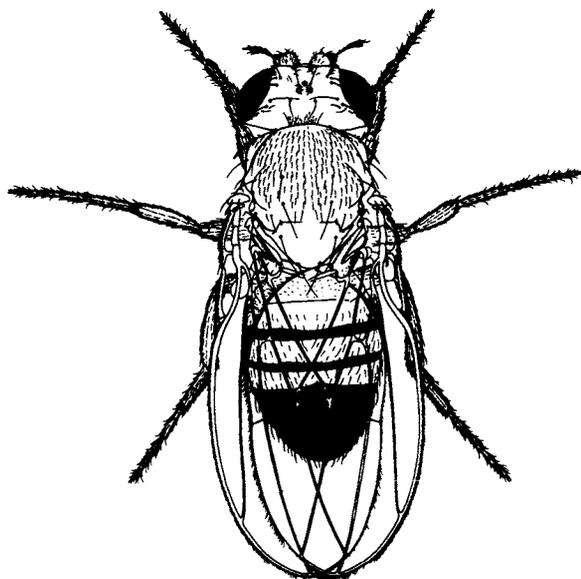
location: 2-105.5.

discoverer: Bridges, 28d6.

references: 1937, *Cytologia* (Tokyo), Fujii Jub., Vol. 2: 745-55.

phenotype: Bristles smaller, especially posterior scutellars. Developmental time slightly longer than normal. Viability only slightly reduced. Classification difficult, especially in early eclosions; improves with age of culture. Enhanced by *shr* (2-2.3), making classification easy. RK3; RK2 with *shr*.

cytology: Placed in region between 59E2 and 60B10 by Bridges (1937) on basis of its being to the right of *In(2R)bw^{VDel} = In(2R)41B2-C1;59E2-4* and to the left of *Df(2R)Px = Df(2R)60B8-10;60D1-2*.

**abb: abbreviated**

From Bridges and Brehme, 1944, *Carnegie Inst. Washington Publ. No.* 552: 11.

abd: abdominal

location: 3-27 (to the right of *se*).

origin: Spontaneous.

discoverer: H. A. and N. W. Timoféeff-Ressovsky.

synonym: *a-3*, *a(3)26*.

references: 1927, Wilhelm Roux's *Arch. Entwicklungsmech. Organ.* 109: 70-109.

Schäffer, 1935, *Z. Indukt. Abstamm. Vererbungsl.* 68: 336-60 (fig.).

phenotype: Irregular reduction of abdominal tergites, sternites, pigmentation, and bristles; more marked in females and increased by crowding and dry food (Braun, 1938, *Am. Naturalist* 72: 189-92). Schäffer's data (1935) suggest irregular dominance in heterozygote, overlapping of wild type in homozygote, and genetic modifiers. RK3.

alleles: *abd²*, spontaneous; recovered by Gottschewski, 1939. Partially complements *abd¹*. Allelism inferred from similarity in genetic location and phenotype and incomplete complementation.

cytology: Placed in 66D9-E1 based on its inclusion in *Df(3L)h-i22 = Df(3L)66D9-E1* (Ingham, Pinchin, Howard, and Ish-Horowitz, 1985, *Genetics* 111: 463-86).

abd-A: see *BXC*

Abd-B: see *BXC*

abdomen rotatum: see *ar*

abdominal: see *abd*

abe: see *mit15*

abero: see *abr*

Abl: Cellular abl oncogene sequence

location: 3-{44}.

origin: Isolated from genome library using *v-abl* probe.

synonym: *Dash*.

references: Shilo and Weinberg, 1981, *Proc. Nat. Acad. Sci. USA* 78: 6789-92.

Simon, Kornberg, and Bishop, 1983, *Nature* (London) 302: 837-39.

phenotype: Considered to be the *Drosophila* sequence homologous to mammalian *c-abl* based both on its origin and amino acid sequence as inferred from its nucleotide sequence.

ABL protein detected at the time of germ-band shortening in the axons of the central nervous system in a bilateral symmetrical series of points that correspond to the positions of neuromeres; later, protein appears in the axons growing across the midline, but not in the cell bodies of the CNS nor in the PNS. As development proceeds, staining of the longitudinal fascicles and to a lesser extent the commissural fascicles becomes intense; staining also seen in association with axonal outgrowth of neural cells in the eye imaginal disk (Bennett and Hoffmann).

Recessive alleles in combination with *Df(3L)st-j7 = Df(3L)73A1-2;73B1-2* either die as late pupae or pharate adults with complete cuticle and roughened eyes, *Abl¹¹*, or as short lived (5-6 days), rough-eyed adults, *Abl¹²* and *Abl¹³*. Surviving females lay few eggs, some of which develop into adults; surviving males have motile sperm, but do not mate and produce no progeny. The rough eye is a reflection of some loss of photoreceptor cells plus ommatidial fusion. In combination with a deficiency extending further to the left, e.g., *Df(3L)std11 = Df(3L)73A11-B1;73D1-2* to include the locus of *Dab*, *Abl¹/Abl⁻* genotypes die as late embryos or early first-instar larvae with disrupted axonal organization in the ventral nerve cord (Henkemeyer, Gertler, Goodman, and Hoffmann, *Cell* 51: 821-28). CNS of doubly deficient embryos, i.e., *Abl⁻ Dab⁻*, fails to form commissures and is defective in axonal outgrowth, although the PNS develops normally.

alleles: Three ethyl-methanesulfonate-induced recessive lethal or semilethal alleles recovered in combination with *Df(3L)st4* or *Df(3L)st-e5* by Belote, McKeown, and Hoffmann are designated *Abl*¹¹, *Abl*¹², and *Abl*¹³. Phenotypic descriptions given above.

cytology: Localized to 73B by *in situ* hybridization with genomic clone (Simon *et al.*).

molecular biology: Sequence isolated using *v-abl* probe from murine leukemia virus (Hoffman-Falk, Einat, Shilo, and Hoffmann, 1983, Cell 35: 393-401). cDNA and genomic sequencing (Telford, Burkhardt, Butler, and Pirrotta, 1985, EMBO J. 4: 2609-15; Henkemeyer, Bennett, Gertler, and Hoffmann, 1988, Mol. Cell Biol. 8: 843-53) reveal a gene of ten exons distributed over 26 kb of genomic DNA; the exons encode a protein of 1520 amino acids, whose sequence is more similar to mammalian *c-abl* sequence than to that of any other gene; between residues 187 and 656, which contains the tyrosine kinase essential domain, the *Drosophila* sequence is 75-85% similar to that of the human *abl* gene. 33 bp region beginning at tyrosine-416 84% homologous to mammalian nucleotide sequence and 62% homologous to *C-src* DNA from *Drosophila* (Hoffmann, Fresco, Hoffman-Falk and Shilo, 1983, Cell 35: 393-401). The polypeptide product as yet unidentified but presumed to be a protein kinase; *Drosophila* extracts do contain a tyrosine kinase activity (Simon *et al.*). The carboxy half of the ABL protein is not conserved between flies and mammals. Expression of the kinase essential domain in bacteria leads to excessive phosphorylation of proteins at tyrosine residues. Developmental Northern blots probed with 800 base-pair sequence from region of highest homology with *v-abl* reveal a 6.2 kb polyadenylated transcript in early but not late embryos, larvae or adults; most abundant in 0-4 hr embryos; absent after 8 hr (Lev, Liebovitz, Segev, and Shilo, 1984, Mol. Cell. Biol. 4: 982-84); returns in a burst of activity in early pupae.

Abnormal: see *A*

abnormal abdomen: see *a()*

Abnormal abdomen: see *A*

abnormal eye: see *mit15*

abnormal oocytes: see *abo*

abnormal tergites: see *abt*

abnormal wings: see *abw*

abo: abnormal oocyte

location: 2-44.0 (mapped with respect to *J*, 2-41).

origin: Naturally occurring allele recovered near Rome, Italy.

references: Sandler, Lindsley, Nicoletti, and Trippa, 1969, Genetics 64: 481-93.

Mange and Sandler, 1973, Genetics 73: 73-86.

Sandler, 1970, Genetics 64: 481-93.

1975, Israel J. Mol. Sci. 11: 1124-34.

1977, Genetics 86: 567-82.

phenotype: Probability of survival of embryos produced by *abo/abo* mothers reduced; male embryos more severely affected than female embryos. Both preblastoderm and postblastoderm embryonic death observed; partial rescue of postblastoderm mortality effected by pater-

nally inherited *abo*⁺ allele; partial rescue of preblastoderm mortality by heterochromatic *ABO* elements located in *Xh* between 3/4 and 7/8 of the distance from the centromere, in *YL* region h10-11, in *YS* region h19, in *2R* proximal, and perhaps in other heterochromatic regions (Pimpinelli, Sullivan, Prout, and Sandler, 1985, Genetics 109: 701-24). Gradual loss of phenotype in homozygous *abo* stocks accompanied by increase in quantity of ribosomal DNA (Kridler and Levine, 1975, Genetics 81: 501-13). New restriction fragments appear in Hind III/Hae III double digests of such homozygous lines probed with nontranscribed spacer sequences of ribosomal genes (Graziani, Vicari, Boncinelli, Malva, Manzi, and Mariani, 1981, Proc. Nat. Acad. Sci. USA 78: 7662-64). *abo* phenotype returns with subsequent maintenance in heterozygous condition. Homozygous *abo* females exhibit moderate decrease in recombination with concomitant increase in exceptional progeny (Carpenter and Sandler, 1974, Genetics 76: 453-75).

cytology: Located in 31F-32E based on its inclusion in *Df(2L)J39 = Df(2L)31A-B;32E* but not *Df(2L)J27 = Df(2L)31B-D;31F* or *Df(2L)Mdh = Df(2L)30D-F;31F*. *abo*-bearing chromosomes differ from others in having a *blood* insertion sequence in 32E (Lavorgna, Malva, Manzi, Gigliotti, and Graziani, 1989, Genetics 123: 485-94).

ABO

A series of heterochromatic elements capable of reducing the level of maternally influenced preblastoderm, but not postblastoderm, mortality among the progeny of *abo/abo* mothers; embryos that carry ABO elements survive better than those that do not (Pimpinelli, Sullivan, Prout, and Sandler, 1985, Genetics 109: 701-24). Elements identified to date and their heterochromatic locations, where known, are listed in the accompanying table. The rescuing capability of *ABO-X* approximates that of *ABO-YL + ABO-YS*. *ABO-X* apparently defective in *In(1)sc*⁴ (Malva, Labella, Manzi, Salzano, Lavorgna, De Ponti, & Graziani, 1985, Genetics 111: 487-94). Effectiveness of *ABO-X* and *ABO-2R* appears to be enhanced by maintenance in a homozygous *abo* stock (Sullivan and Pimpinelli, 1986, Genetics 114: 885-95).

element	cytology
ABO-2R	
ABO-X	h26-28 ^α
ABO-YL	h10-11
ABO-YS	h19

^α In region that includes proximal half of h26 and distal half of h28 (Pimpinelli, Sullivan, Prout, and Sander, 1985, Genetics 109: 701-24).

abr: abero

location: 2-83.

origin: Spontaneous.

discoverer: Bridges, 33b10.

phenotype: Abdominal banding etched and irregular.

Wing margins irregular. Eyes rough. Bristles and hairs sparse and disarranged. *abr*¹ + sometimes lacks anterior dorsocentrals. Viability usually poor. RK3.

other information: Not allelic to *fr* or *nw*.

abrupt: see *ab*

Abruptex: see *Ax*, listed under *N: Notch*

***abt: abnormal tergites**

location: 1-45.6.

origin: Induced by 2-chloroethyl methanesulfonate.

discoverer: Fahmy, 1955.

references: 1959, DIS 33: 83.

phenotype: Abdomen affected to various degrees, from extreme deformation of tergites to slight abnormalities in distribution of pigment and hairs. Eyes also deformed to various degrees from gross alterations in shape to slight derangement of ommatidia. Wings vary from alterations in size, outline, and venation to small incisions of the inner margin. Most-extreme effects not always positively correlated, and all flies show several atypical characters. Males viable; fertility severely reduced. RK3.

***abw: abnormal wings**

location: 1-60.

origin: X ray induced.

discoverer: Halfer, 1963.

phenotype: Wing size reduced; wings upturned; L5 and crossveins absent. Plexus of veins between L3 and L4. RK1.

abx: see **BXC**

ac: see **ASC**

Ac: see **Cu^A**

Ac-SD: see **Rsp**

acc: acclinal wing

location: 1-54.5.

origin: Induced by triethylenemelamine.

discoverer: Fahmy, 1952.

references: 1958, DIS 32: 67.

phenotype: Wings upheld but slope backward at 45° angle from abdomen. Unable to fly or jump; muscles normal in gross and ultrastructural morphology. Mosaic experiment suggests possible thoracic neural etiology (Deak, 1976, J. Insect Physiol. 22: 1159-65). Viability and fertility good in both sexes. RK1.

alleles: One allele each induced by D-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine and by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine.

Ace: Acetyl cholinesterase (J.C. Hall)

location: 3-52.2.

synonym: *l(3)26*.

references: Hall and Kankel, 1976, Genetics 83: 517-35.

Greenspan, Finn, and Hall, 1980, J. Comp. Neurol. 189: 741-74.

Hall, Alahiotis, Strumpf, and White, 1980, Genetics 96: 939-65.

phenotype: The structural gene for acetylcholinesterase [AChE, acetylcholine acetyl hydrolase (EC 3.1.1.7)], the enzyme that terminates synaptic transmission by rapidly hydrolyzing the neurotransmitter acetylcholine. Biochemical analysis (*e.g.*, Zingde, Rodrigues, Joshi, and Krishnan, 1983, J. Neurochem. 41: 1243-52; Gnagey, Forte, and Rosenberry, 1987, J. Biol. Chem. 262: 13290-98; Fournier, Bride, Karch, and Bergé, 1988, FEBS Lett. 238: 333-37; Haas, Marshall, and Rosenberry, 1988, Biochemistry 27: 6453-57; Toutant, Arpagaus, and Fournier, 1988, J. Neurochem. 50: 209-18; Fournier, Bergé, Almeida, and Bordier, 1988, J. Neurochem. 50: 1158-63), indicates that the mature enzyme

contains noncovalently associated subunits of 16 and 55 kd, which are processed from a primary translation product of *ca* 70 kd such that the 16-kd moiety is from the N terminus and the 55-kd moiety is from the C terminus; two such associations are linked via disulfide bonds connecting the 55-kd polypeptides anchored to membrane via a glycoinositol phospholipid anchor covalently linked to the C termini of the 55-kd subunits. Extracts contain amphiphilic dimers and monomers as well as hydrophilic dimers and monomers, which lack the glycoinositol phospholipid anchor. Developmental profile studied by Dewhurst, McCaman, and Kaplan (1970, Biochem. Genet. 4: 499-508; see also Arpagaus, Fournier, and Toutant, 1988, Insect Biochem. 18: 539-49); total AChE activity shows a transient peak during first larval instar and rises again to a maximum in the adult. In the developing eye disc, AChE first appears in retinula cells three to four days before they are functional and when it cannot have a synaptic function; levels are reduced in retinula cells midway through pupal development, and the enzyme accumulates rapidly in the neuropils of the optic lobes of the brain and the midbrain (Wolfgang and Forte, 1989, Dev. Biol. 131: 321-30). Putative nulls are lethal at end of embryonic stage; then ultrastructural observations of CNS in such mutants suggest neural-degenerative defects (Chase and Kankel, 1988, Dev. Biol. 125: 361-80). ACE-minus tissues survive in mosaics unless enzyme absent from posterior midbrain; surviving mosaics have defective visual physiology, optomotor behavior or courtship, depending on location of mutant clone. Such clones associated with defective morphology or neuropile of various ganglia in central nervous system (Greenspan *et al.*, 1980). In heat-sensitive combinations of *Ace* mutations (Greenspan *et al.*, 1980), both membrane-bound and soluble enzyme has reduced activity (Zador, 1989, Mol. Gen. Genet. 218: 487-90).

alleles: Unless noted otherwise in comments column alleles are null as is *Ace¹*.

allele	origin	discoverer	synonym	ref ^α	comments
Ace¹β	X ray	Schalet	<i>l(3)26</i>	4, 8	
Ace²	EMS	Deland	<i>l(3)m15</i>	4	
Ace³	EMS	Hilliker, Clark	<i>l(3)B2-5</i>	4	
Ace⁴	EMS	Hilliker, Clark	<i>l(3)B4-2</i>	4	
Ace⁵	EMS	Hilliker, Clark	<i>l(3)B8-2</i>	4	
Ace⁶	EMS	Hilliker, Clark	<i>l(3)B15-2</i>	4	hypomorphic
Ace⁷	EMS	Hilliker, Clark	<i>l(3)B22-1</i>	4	
Ace⁸	EMS	Hilliker, Clark	<i>l(3)B22-2</i>	4	hypomorphic
Ace⁹	EMS	Hilliker, Clark	<i>l(3)B27-1</i>	4	
Ace¹⁰	EMS	Hilliker, Clark	<i>l(3)B29-1</i>	4	
Ace¹¹	EMS	Hilliker, Clark	<i>l(3)B29-2</i>	4	
Ace¹²	EMS	Hilliker, Clark	<i>l(3)H36</i>	4	
Ace¹³	EMS	Hilliker, Clark	<i>l(3)H41</i>	4	
Ace¹⁴	EMS	Hilliker, Clark	<i>l(3)H89</i>	4	
Ace¹⁵	EMS	Hilliker, Clark	<i>l(3)B21-5</i>	4	
Ace¹⁶	EMS	Hilliker, Clark	<i>l(3)H15</i>	4	
Ace^{HD1}β	HD			6	
Ace¹¹⁹	EMS			1	
Ace¹²¹	EMS			1	
Ace¹²⁷	EMS			1	
Ace¹²⁹β	EMS			1, 2	cold sensitive
Ace¹³¹	EMS			1	
Ace¹³²	EMS			1	
Ace¹³³	EMS			1	
Ace¹³⁹	EMS			1	
Ace¹⁴⁰β	EMS			1, 2, 7	few survivors; suppressor of variegation
Ace¹⁴¹	EMS			1	

allele	origin discoverer	synonym	ref ^α	comments
<i>Ace</i> ^{J43}	EMS		1	
<i>Ace</i> ^{J44 β}	EMS		1	
<i>Ace</i> ^{J45}	EMS		1	
<i>Ace</i> ^{J50}	EMS		1	
<i>Ace</i> ^{J51}	EMS		1	
<i>Ace</i> ^{J53}	EMS		1	
<i>Ace</i> ^{J55}	EMS		1	
<i>Ace</i> ^{Im35 β}	EMS		3	hypomorphic
<i>Ace</i> ^{Im38}	EMS		3	
<i>Ace</i> ^{Im115}	EMS		3	
<i>Ace</i> ^{G11}	EMS Gelbart			
<i>Ace</i> ^{mr β}	spont		5	

^α 1 = Greenspan, Finn, and Hall, 1980, J. Comp. Neurol. 189: 741-74; 2 = Hall, Alahiotis, Strumpf, and White, 1980, Genetics 96: 939-65; 3 = Hall and Kankel, 1976, Genetics 83: 517-35; 4 = Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72; 5 = Mortan and Singh, 1982, Biochem. Genet. 20: 179-98; 6 = Nagoshi and Gelbart, 1987, Genetics 117: 487-502; 7 = Reuter, Gausz, Gyurkovics, Friede, Bang, Spierer, Hall, and Spierer, 1987, Mol. Gen. Genet. 210: 429-36; 8 = Schalet, Kernaghan, and Chovnick, 1964, Genetics 50: 1261-68.

^β More detailed description below.

cytology: Located in 87E3 based on its location between *Df(3R)ry1301 = Df(3R)87D2-4;87E1-2* and *Df(3R)GE41 = Df(3R)87E4* (Hall and Spierer 1986, EMBO J. 5: 2949-54).

molecular biology: Locus comprises ten exons and nine introns distributed within a 34-kb transcription unit (Fournier, Karch, Bride, Hall, Bergé, and Spierer, 1989, J. Mol. Biol. 210: 15-22) extending from approximately coordinates 18 to 52 kb on the molecular map of Bender, Spierer, and Hogness (1983, J. Mol. Biol. 168: 17-33) whose origin is 6.5 kb to the left of the left breakpoint of *In(3R)Cbx^{rv1}* with positive values extending to the right. Transcription takes place from right to left. The 5' untranslated region is transcribed from exons I and II; the signal sequence is encoded in exon II, the 16-kd polypeptide by exons II, III, and IV; the 55-kd polypeptide by exons IV, V, VI, VII, VIII, and IX; the hydrophobic peptide exchanged in mature protein with a glycolipid anchor as well as the 3' untranslated region by exon X. The mature transcripts are estimated at 4.2 and 4.5 kb (Nagoshi and Gelbart, 1987, Genetics 117: 487-502). cDNA sequencing (Hall and Spierer, 1986; Fournier *et al.*, 1989) indicates mature transcript of 4291 nucleotides encoding a 650-amino-acid protein product, which displays a high degree of homology with AChE from *Torpedo californica*; however, the *Drosophila* protein carries a 38-amino-acid signal sequence lacking in *Torpedo*, and a 41-amino-acid hydrophilic sequence extending from residues 140 to 180 that is not contained in the *Torpedo* sequence; the latter is encoded by exons III and IV, and contains the site of proteolytic cleavage of the *Drosophila* AChE primary translation product.

Ace^I

phenotype: Temperature insensitive lethal; lethal in homozygotes or in combination with deficiency for *Ace*⁺. Lethality at end of embryonic stage. Greatly reduced levels of acetylcholinesterase. AChE-minus tissues survive in mosaics unless enzyme absent from posterior midbrain; surviving mosaics have defective visual physiology, optomotor behavior, or courtship depending on location of mutant clone. Such clones associated with defective morphology of neuropile of various ganglia in

central nervous system.

molecular biology: DNA insert in fifth intron (Fournier *et al.*, 1989) observed at approximately coordinate +30; separable by recombination from the *Ace*^I mutation (Nagoshi and Gelbart, 1987, Genetics 117: 487-502).

Ace^{HD1}

phenotype: Retains some ACE activity (Nagoshi and Gelbart, 1987, Genetics 117: 487-502), but only as soluble enzyme outside CNS (Zudor *et al.*, 1986).

molecular biology: Deleted of promoter region and first (non-coding) exon (Fournier *et al.*, 1989).

Ace^{J29}

phenotype: The original allele of this complementation group. Cold sensitive lethal. Maximum survival of *Ace*^{J29}/*Df(3R)l26d* at 27°, no survival at 18°. Exposure to 18° does not reduce AChE activity. *Ace*^{J29} alters Km of enzyme, further implying structural gene locus.

Ace^{J40}

phenotype: Nearly completely lethal. Two percent survival in combination with *Df(3R)l26d* at 18°, none at 29°. Partial complementation of *Ace*^{J19} and *Ace*^{J50}; heat sensitive; extracts of *Ace*^{J40} lack the 110 kilodalton molecular species, whereas *Ace*^{J19} and *Ace*^{J50} lack the 64 and 75 kilodalton species (Zingde, Rodrigues, Joshi, and Krishnan, 1983, J. Neurochem. 41: 1243-52). Enzyme produced by heteroallelic combinations raised under permissive conditions is thermolabile. Exposure of *Ace*^{J40}/*Ace*^{J19} or *Ace*^{J40}/*Ace*^{J50} flies to restrictive temperature during late embryonic-early larvae stage lethal; little effect on mid and late larval stages; pupal exposure causes defects in adult phototaxis and motor activity. Heat treatment of adults causes no decline in ACE activity but decrements in phototaxis (29°), and cessation of movement (31°) observed. *Ace*^{J40} produces enzyme with altered Km.

molecular biology: Appears to map proximal to a DNA insert located between coordinates +43 and +48 (Nagoshi and Gelbart, 1987).

Ace^{J44}

molecular biology: Associated with a molecularly defined structural variation; probably loss of a *Bam* H1 site around coordinate +33 (Gausz, Hall, Spierer, and Spierer, 1986, Genetics 112: 65-78). Structural variant and mutation appear to be inseparable by recombination (Nagoshi and Gelbart, 1987).

Ace^{Im35}

phenotype: Hypomorphic allele. Exhibits reduced survival (< 30%) in combination with *Df(3R)l26d*. Enzyme activity in *Ace*^{Im35}/+ heterozygotes lower than in heterozygotes for more severe alleles.

Ace^{mr}: Acetylcholinesterase-malathion resistant

origin: Recovered from line selected for malathion resistance.

phenotype: Acetylcholinesterase from homozygotes has lower K_m, lower activity, and slightly increased electrophoretic mobility compared to wild type. Relation to malathion resistance unclear.

ACE1: Amplification Control Element on chromosome 1

A sequence required for amplification in ovarian follicle cells of the cluster of chorion-protein genes located at 7F1-2 (*Cp36* and *Cp38*); provisionally located between 654 and 266 base pairs upstream from *Cp38* (Wakimoto).

ACE3

A sequence required for amplification in ovarian follicle cells of the cluster of chorion-protein genes located at 66D11-15 (*Cp15*, *Cp16*, *Cp18* and *Cp19*); located between 615 and 187 base pairs upstream from *Cp18* (Kalfayan, Levine, Orr-Weaver, Parks, Wakimoto, deCicco, and Spradling, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 527-35).

Acetyl choline receptor: see *Acr*

achaete: see *ac* under *ASC*

Ach: see *emc*^D

Acp: Accessory gland protein

Genes inferred from bands on SDS polyacrylamide gels. Six polypeptides are highly polymorphic, exhibiting several electrophoretic variants; these all map to chromosome 2 and are tabulated below. Codominant expression indicates that variants are in structural genes and not attributable to differences in post-translational modification (Whalen and Wilson, 1986, Genetics 114: 77-92).

locus	genetic location	cytological location	molecular mass (kd)
<i>AcpA</i>	2-		165-70
<i>AcpB</i>	2-42.8	36D1-E4	130-140
<i>AcpC</i> ^α	2-53.0		125-128
<i>Acp-g1</i> ^α	2-13.5		145-163
<i>AcpJ</i>	2-		45
<i>AcpK</i> ^α	2-54.1		43

^α variants include a null allele.

Acp70A: Accessory gland peptide

location: 3-{40}.

references: Chen, Stumm-Zollinger, Aigaki, Balmer, Bienz, and Böhlen, 1986, Cell 54: 291-98).

phenotype: Encodes a 36-amino-acid peptide that is synthesized in the accessory gland and is transferred to the female where it represses female sexual receptivity and stimulates oviposition. The peptide contains a high concentration of basic amino acids, tryptophan and hydroxyproline as well as an unique residue of unknown nature that is encoded by a leucine codon.

cytology: Placed in 70A by *in situ* hybridization.

molecular biology: Gene cloned and sequenced; conceptual sequence indicates a hydrophobic amino-terminal signal sequence of 19 residues. mRNA for prepeptide accumulates exclusively in the male accessory gland.

AcpH-1: Acid phosphatase 1

location: 3-101.1 (between *ca* and *bv*).

discoverer: MacIntyre, 1964.

references: 1966, DIS 41: 61.
1966, Genetics 53: 461-74.

phenotype: Structural gene for acid phosphatase 1 [ACPH-1 (EC3.1.3.2)], the major phosphatase in adults; responsible for approximately 90% of the low-pH nucleotidase activity throughout development. Glycopro-

tein homodimer with subunit molecular weight of 50,000 daltons. Purification and biochemical characterization by Feigen, Mitrick, Johns, Postlethwait, and Sederoff (1980, J. Biol. Chem. 255: 10338-43). Serves as a reliable cytochemical marker in many tissues (Hall, 1979, Genetics 92: 437-57). Enzyme appears to be produced in nurse cells and follicular cells of ovary and transferred to oocyte through the ring canals and by pinocytosis, respectively (Sawicki and MacIntyre, 1977, Dev. Biol. 60: 1-13); maternally produced enzyme persists to third instar; paternal gene function detectable in gels after 9-10 hr of embryonic development (Yasbin, Sawicki, and MacIntyre, 1978, Dev. Biol. 63: 35-46); and after 5 hr histochemically (Sawicki and MacIntyre, 1978, Dev. Biol. 63: 47-58). Enzyme found in larvae, pupae, and adults; levels increase during adult life (Postlethwait and Gray, 1975, Dev. Biol. 47: 196-205).

alleles: In addition to the information tabulated below, pairwise combinations of *AcpH-1*ⁿ², *AcpH-1*ⁿ³, *AcpH-1*ⁿ⁶, and *AcpH-1*ⁿ⁹ exhibit 20-40% normal levels of cross reacting material (CRM).

allele	origin	derivative of	discoverer	ref ^α	comments
<i>AcpH-1</i> ^A	spont		MacIntyre	4	slow
<i>AcpH-1</i> ^B	spont		MacIntyre	4	intermediate
<i>AcpH-1</i> ^C	spont		MacIntyre	4	fast
<i>AcpH-1</i> ⁿ¹	EMS	<i>AcpH-1</i> ^B	MacIntyre	1	A-like mobility in heterodimer
<i>AcpH-1</i> ⁿ²	EMS	<i>AcpH-1</i> ^A	MacIntyre	1	
<i>AcpH-1</i> ⁿ³	EMS	<i>AcpH-1</i> ^B	MacIntyre	1	
<i>AcpH-1</i> ⁿ⁴	EMS	<i>AcpH-1</i> ^A	MacIntyre	1	0-5% normal CRM
<i>AcpH-1</i> ⁿ⁵	EMS	<i>AcpH-1</i> ^A	MacIntyre	1	0-5% normal CRM
<i>AcpH-1</i> ⁿ⁶	EMS	<i>AcpH-1</i> ^B	MacIntyre	1	A-like mobility in heterodimer
<i>AcpH-1</i> ⁿ⁷	EMS	<i>AcpH-1</i> ^B	MacIntyre	1	
<i>AcpH-1</i> ⁿ⁸	EMS	<i>AcpH-1</i> ^A	MacIntyre	1	0-5% normal CRM
<i>AcpH-1</i> ⁿ⁹	EMS	<i>AcpH-1</i> ^B	MacIntyre	1	
<i>AcpH-1</i> ⁿ¹⁰	EMS	<i>AcpH-1</i> ^A	MacIntyre	1	
<i>AcpH-1</i> ⁿ¹¹	EMS	<i>AcpH-1</i> ^B	MacIntyre	1	0-5% normal CRM
<i>AcpH-1</i> ⁿ¹²	EMS	<i>AcpH-1</i> ^B	MacIntyre	1	
<i>AcpH-1</i> ⁿ¹³	EMS	<i>AcpH-1</i> ^B	MacIntyre	1	0-5% normal CRM
<i>AcpH-1</i> ⁿ¹⁴	EMS	<i>AcpH-1</i> ^A	MacIntyre	1	B-like mobility in heterodimer
<i>AcpH-1</i> ⁿ¹⁵	EMS	<i>AcpH-1</i> ^A	MacIntyre	1	0-5% normal CRM
<i>AcpH-1</i> ^{nGB1}	spont	<i>AcpH-1</i> ^B		2,3	B-like mobility in heterodimer
<i>AcpH-1</i> ^{nGB2}	spont	<i>AcpH-1</i> ^B		2,3	
<i>AcpH-1</i> ^{nNC1}	spont	<i>AcpH-1</i> ^B		2,3	

^α 1 = Bell, MacIntyre, and Olivieri, 1972, Biochem. Genet. 6: 205-16; 2 = Burkhart, Montgomery, Langley, and Voelker, 1984, Genetics 107: 295-306; 3 = Langley, Voelker, Leigh Brown, Ohnishi, Dickson, and Montgomery, 1981, Genetics 99: 151-56; 4 = MacIntyre, 1968, DIS 3: 60.

cytology: Located between 99C5 and 7 based on its deletion by *Df(3R)ca-R14 = Df(3R)99A8-9;99D1-2* but not by *Df(3R)ca-165P = Df(3R)99B2-4;99C5-6* (Frisardi and MacIntyre, 1984, Mol. Gen. Genet. 197: 403-13).

Acr60C: Acetyl choline receptor in 60C

location: 2-{107}.

references: Shapiro, Wakimoto, Subers, and Nathanson, 1989, Proc. Nat. Acad. Sci. USA 86: 9039-43.

Onai, FitzGerald, Arakawa, Gocayne, Urquhart, Hall, Fraser, McCombie, and Venter, 1989, FEBS Lett. 255: 219-25.

phenotype: The structural gene encoding a Drosophila homologue of vertebrate muscarinic acetylcholine recep-

tor (mAChR). When expressed in Y1 adrenal cells it is physiologically active as measured by agonist dependent stimulation of phosphatidylinositol metabolism.

cytology: Placed in 60C7-8 by *in situ* hybridization.

molecular biology: Genomic clone isolated from library using a probes from vertebrate muscarinic acetylcholine receptor genes. Nucleotide sequences of cDNA clones reveal a long open reading frame that encodes a 788-amino-acid protein with calculated molecular weight of 84,807 (Onai *et al.*). The amino-acid sequence shows a number of features characteristic of the muscarinic/adrenergic receptor gene superfamily in vertebrates: three potential N-linked glycosylation sites (Asn 65, 84, and 87), seven putative membrane-spanning domains. It displays a high degree of amino-acid identity with vertebrate muscarinic acetylcholine receptors, ~60% overall and up to 88% in transmembrane regions; the segment between transmembrane domains 5 and 6 is considerably longer than that of vertebrate sequences; also the gene has three introns in the region.

Acr64B

location: 3-{8}.

synonym: *ard*.

references: Hermans-Borgmeyer, Zopf, Ryseck, Hovemann, Betz, and Gundelfinger, 1986, *EMBO J.* 5: 1503-08.

Wadsworth, Rosenthal, Kammermeyer, Potter, and Nelson, 1988, *Mol. Cell Biol.* 8: 778-85.

Sawruk, Hermans-Borgmeyer, Betz, and Gundelfinger, 1988, *FEBS Lett.* 235: 40-46.

phenotype: Structural gene encoding a *Drosophila* homologue of a subunit of vertebrate nicotinic acetylcholine receptors (nAChR). Antibody raised against *Acr64B* fusion proteins immunoprecipitate one of two high-affinity α -bungarotoxin-binding sites from detergent extracts of *Drosophila* head membranes (Schloss, Hermans-Borgmeyer, Betz, and Gundelfinger, 1988, *EMBO J.* 7: 2889-94). *In situ* hybridization localizes *Acr64B* expression to nervous tissue, especially in late embryos, pupae, and newly eclosed adults (Hermans-Borgmeyer, Hoffmeister, Sawruk, Betz, Schmitt, and Gundelfinger, 1989, *Neuron.* 2: 1147-56).

cytology: Placed in 64B by means of *in situ* hybridization.

molecular biology: Genomic clones identified using a *Torpedo californica* nAChR probe; these hybridize to a 3.2-kb mRNA present at high levels on developmental Northern blots in late embryos and during metamorphosis, periods of neuronal differentiation. Genomic probes used to isolate overlapping cDNA clones. The gene comprises six exons distributed over approximately seven kb of genomic sequence. The predicted mature protein after cleavage of a 24 amino-acid signal sequence, consists of 497 amino acids, has a calculated molecular weight of 57,340 and shows extensive homology to all known nAChR genes of other species along its entire amino acid sequence, conforming most closely to neuronal α subunits, although it lacks the cysteine doublet at residues 201 and 202 characteristic of all other α subunits. It contains four putative transmembrane domains, a potential amphipathic α helix, and a canonical N-glycosylation site Asn48; however, the N-linked glycosylation site found at residue 141 in all vertebrate nAChR's is absent in *Drosophila*.

Acr96A

location: 3-{83}.

synonym: *ALS: Alpha-Like Subunit*.

references: Bossy, Ballivet, and Spierer, 1988, *EMBO J.* 7: 611-18.

phenotype: Structural gene encoding a *Drosophila* homologue of a subunit of vertebrate nicotinic acetylcholine receptors (nAChR); inferred to be homologous to neuronal α subunits based on the cysteine doublet at amino-acid residues 201 and 202.

cytology: Placed in 96A by *in situ* hybridization.

molecular biology: Genomic clones identified using as a probe a fragment of the chick neuronal nAChR α 2 gene; these hybridize to a 10.5 kb mRNA present at high levels on developmental Northern blots from late embryo to pupation, decreasing in late pupae and adults; genomic probes used to isolate overlapping cDNA clones. The gene comprises ten exons distributed over 54 kb of genomic sequence; combined nucleotide sequence from the cDNA clones defines a single long open reading frame of 1701 nucleotides bracketed by 1282 5' and 514 3' nucleotides. The ORF encodes 567 amino acids, which show 40-44% sequence conservation with mammalian neuronal nAChR α subunits and with *Drosophila Acr64B* product. Structural domains homologous to those of vertebrate nAChR subunits include a cysteine doublet at residues 201 and 202 that characterizes all α subunits, four transmembrane domains, two potential glycosylation sites (Asn 24 and 212) characteristic of vertebrate neuronal α subunits, and an amphipathic α -helical region in the C-terminal quarter of the polypeptide. In addition, the positions of four *Drosophila* introns correspond exactly to those of four of seven vertebrate AChR introns.

Activator of SD: see *Rsp*

act: actidione sensitive

location: 3-90 (21 units to the right of *H*).

origin: Naturally occurring allele.

references: Marzluf, 1969, *Biochem. Genet.* 3: 229-38.

phenotype: *act/act* killed by 0.1 the concentration of actidione (cycloheximide) that *act*⁺-bearing strains survive.

alleles: Recessive allele fixed in Oregon-R and Canton-S strains. Urbana-S and Swedish-b carry *act*⁺.

Act5C: Actin in region 5C

location: 1-{14}.

references: Tobin, Zulauf, Sánchez, Craig, and McCarthy, 1980, *Cell* 19: 121-31.

Fyrberg, Kindle, Davidson, and Sodja, 1980, *Cell* 19: 365-78.

Fyrberg, Bond, Hershey, Mixer, and Davidson, 1981, *Cell* 24: 107-16.

phenotype: Codes for cytoplasmic actin; transcribed throughout development; one of two actin genes transcribed in Kc cells and several other cell lines (Fyrberg, Mahaffy, Bond, and Davidson, 1983, *Cell* 33: 115-23). One of three actin genes expressed in the wing disc during wing development, each with its characteristic profile. Peak expression at 44h of pupal age, little or no expression at 60h rising again at 80h. 44h peak corresponds to time of maximum change in cell shape (Peterson, Bond, Mitchell, and Davidson, 1985, *Dev.*

Genet. 5: 219-25). Transcripts present in the preblastoderm embryo suggesting maternal transcription; during blastoderm formation, *Act5C* transcripts accumulate at the periphery of the embryo; local concentrations of transcript observed in anterior and posterior midgut rudiments in stage 13 embryos; hybridization also observed in the developing ventral nervous system. Later in embryogenesis, transcript found in all tissues but with dramatic concentrations of transcripts in the anterior and posterior midgut and the proventriculus. Exon specific probes demonstrate that transcripts containing exon 1 tend to be concentrated in anterior portions of early embryos, including the anterior midgut primordium and the proventriculus, as well as in the posterior midgut primordium; during germ-band extension, small local concentrations of exon 2 transcripts are seen in posterior and ventral regions of the embryo (Burn, Vigoreaux, and Tobin, 1989, Dev. Biol. 131: 345-55).

cytology: Localized to 5C2-5 based on failure of *Act5* specific probe to hybridize to either *Df(1)N73 = Df(1)5C2;5C5-6* or *Df(1)C149 = Df(1)5A8-9;5C5-6* (Sodja, Rizki, Rizki and Zafar, 1982, Chromosoma 86: 293-98).

molecular biology: Genomic clone restriction mapped (Fyrberg *et al.*, 1980) and partially sequenced (Fyrberg *et al.*, 1981). Comparison with cDNA clones indicates the presence of three exons, two of which are included in any cDNA (Bond and Davidson, 1986, Mol. Cell Biol. 6: 2080-88). Partial sequence (Bond and Davidson; Vigoreaux and Tobin, 1987, Genes Dev. 1: 1161-71) indicate that all protein encoding sequences reside in exon 3, that either exon 1 or exon 2 is spliced to exon 3, and that there are three transcription start sites, one in exon 1 and two in exon 2, giving rise to different 5' untranslated regions; also there are indications of at least three polyadenylation sites generating messages with 3' untranslated regions of 375, 655, and 945 nucleotides. Using discriminating 5' and 3' probes, Burn, Vigoreaux, and Tobin (1989) have shown that transcripts differing in 5' untranslated regions display different tissue specificities; no 3' specificities are seen. Exons 1 and 2 are each preceded by a functional promoter (Bond-Matthews and Davidson, 1988, Gene 62: 289-300).

Act42A

location: 2-55.4 (inferred from polytene position).

references: Tobin, Zulauf, Sánchez, Craig, and McCarthy, 1980, Cell 19: 121-31.

Fyrberg, Bond, Hershey, Mixter, and Davidson, 1981, Cell 24: 107-16.

phenotype: Codes for cytoplasmic actin; transcribed throughout development; one of two actin genes transcribed in Kc cells and several other cell lines (Fyrberg, Mahaffy, Bond, and Davidson, 1983, Cell 33: 115-23). One of three actin genes expressed in the wing disc during wing development, each with its characteristic profile. Peak expression at 44h of pupal age, little or no expression at 60h rising again at 80h. 44h peak corresponds to time of maximum change in cell shape (Peterson, Bond, Mitchell, and Davidson, 1985, Dev. Genet. 5: 219-25).

cytology: Located in 42A by *in situ* hybridization.

molecular biology: Genomic clone = λ DmA3. Partial nucleotide sequence in Fyrberg *et al.* (1981).

Act57A

location: 2-{93}.

references: Tobin, Zulauf, Sánchez, Craig, and McCarthy, 1980, Cell 19: 121-31.

Fyrberg, Kindle, Davidson, and Sodja, 1980, Cell 19: 365-78.

Fyrberg, Bond, Hershey, Mixter, and Davidson, 1981, Cell 24: 107-16.

phenotype: According to Fyrberg and Davidson, *Act57A* encodes the actin I protein isoform, which is the major actin species of larval intersegmental muscle; also encodes adult cephalic and abdominal muscle (Fyrberg, Mahaffy, Bond and Davidson, 1983, Cell 33: 115-33).

cytology: Localized to 57A by *in situ* hybridization.

molecular biology: Genomic clone = λ DmA4; coding region restriction mapped and partially sequenced (Fyrberg *et al.*, 1981). Intervening sequence of approximately 630 base pairs inserted in the glycine codon at amino acid position 13 (Fyrberg *et al.*, 1981).

Act79B

location: 3-{47.5}.

references: Tobin, Zulauf, Sánchez, Craig, and McCarthy, 1980, Cell 19: 121-31.

Fyrberg, Kindle, Davidson, and Sodja, 1980, Cell 19: 365-78.

Fyrberg, Bond, Hershey, Mixter, and Davidson, 1981, Cell 24: 107-16.

Zulauf, Sánchez, Tobin, Rdest, and McCarthy, 1981, Nature 292: 556-58.

Sánchez, Tobin, Rdest, Zulauf, and McCarthy, 1983, J. Mol. Biol. 163: 533-51.

phenotype: According to Zulauf *et al.* (1981), *Act79B* appears to code for actin I, a larval muscle-specific actin. Initial translation product, which migrates as actin II, apparently acetylated to become actin I. Using probe from 3' transcribed-but-not-translated sequences, Sánchez *et al.* demonstrated two minor peaks of transcription during embryogenesis and major peaks during first and second instar and to a lesser degree in the pupa. Fyrberg, Mahaffy, Bond, and Davidson, (1983, Cell 33: 115-23) on the other hand, find *Act79B* to be expressed in adult legs and tubular muscles of thorax, including direct flight muscles, pleurosternal muscles, and muscles of various leg segments. In addition, *Act79B* is expressed in muscles that support the head and abdomen, in the scutellar pulsatile organ, and in two pairs of abdominal muscles that are present only in male flies (Courchesne-Smith, and Tobin, 1989, Dev. Biol. 133: 313-21). One of three actin genes expressed in wing development each with its characteristic developmental profile; peak activity at 80h of pupal age (Peterson, Bond, Mitchell, and Davidson, 1985, Dev. Genet. 5: 219-25).

cytology: Localized to 79B by *in situ* hybridization.

molecular biology: Genomic clone by Zulauf *et al.* (1981) and as λ DmA6 by Fyrberg *et al.* (1981). Coding region restriction mapped and partially sequenced by Fyrberg *et al.* (1981). Intervening sequence of 357 nucleotides within a glycine codon at position 307 (Fyrberg *et al.*, 1981). Coding sequences, intervening sequences and flanking sequences completely sequenced (Sánchez *et al.*). Encodes a 374-amino-acid 43-kd polypeptide which is 95% homologous with the product of *Act88F*

and 91% homologous with rabbit actin.

Act87E

location: 3-{52.3}.

references: Tobin, Zulauf, Sánchez, Craig, and McCarthy, 1980, Cell 19: 121-31.

Fyrberg, Kindle, Davidson, and Sodja, 1980, Cell 19: 365-78.

Fyrberg, Bond, Hershey, Mixter, and Davidson, 1981, Cell 24: 107-16.

phenotype: Encodes actin found in larval muscle and adult cephalic and abdominal muscle (Fyrberg, Mahaffey, Bond, and Davidson, 1983, Cell 33: 115-23).

alleles: No lethal alleles of *Act87E* recovered in a lethal-saturation study (Manseau, Ganetzky, and Craig, 1988, Genetics 119: 407-20).

cytology: Placed in 87E9-12 by *in situ* hybridization; included in *Df(3R)ry619 = Df(3R)87D7-9;87E12-F1*, but not in *Df(3R)ry1168 = Df(3R)87B15-C1;87E9-12* (Manseau *et al.*, 1988).

molecular biology: Genome clone restriction mapped and partially sequenced by Fyrberg *et al.* (1981). Comparison of genomic and cDNA sequence indicates a 556 nucleotide intron in the 5' untranslated region. Conceptual amino-acid sequence shows ~95% identity with other *Drosophila* actins (Manseau *et al.*, 1988).

Act88F

location: 3-57.1 (based on 41 *cu-sr* and 84 *red-e* recombinants).

references: Tobin, Zulauf, Sánchez, Craig, and McCarthy, 1980, Cell 19: 121-31.

Fyrberg, Kindle, Davidson, and Sodja, 1980, Cell 19: 365-78.

Fyrberg, Bond, Hershey, Mixter, and Davidson, 1981, Cell 24: 107-16.

Sánchez, Tobin, Rdest, Zulauf, and McCarthy, 1983, J. Mol. Biol. 163: 533-51.

phenotype: Structural gene encoding actin III; expressed only in the developing thorax, specifically in the indirect flight muscles (Fyrberg, Mahaffey, Bond, and Davidson, 1983, Cell 33: 115-23; Hiromi and Hotta, 1985, EMBO J. 4: 1681-87). The only actin expressed in indirect flight muscle (Fyrberg). Heterozygotes for null mutations or *Act88F* deficiencies are flightless; flightlessness is apparently caused by imbalance between myosin heavy chains and actin III; whereas hemizyosity for either *Mhc* or *Act88F* leads to complex myofibrillar defects and flightlessness, double hemizygotes have nearly normal fibrillar structure and are able to fly [Beall, Sepanski, and Fyrberg, 1989, Genes Dev. 3: 131-40 (fig.)]. Major peaks of transcription during pupal stage (Sánchez *et al.*, 1983). Heterozygotes and to a greater degree, homozygotes and heteroallelic heterozygotes for antimorphic alleles *Act88F⁴* and *Act88F⁵* show constitutive synthesis of heat-shock proteins, with HSP26 and HSP27 less actively synthesized than HSP22, HSP70, and HSP84; response to heat shock normal (Hiromi and Hotta).

alleles:

allele	origin	synonym	ref ^α	comments
<i>Act88F¹</i>	EMS	<i>Ifm(3)1, Ifm(3)2</i>	3, 4, 7	dominant antimorphic allele weak inducer of HSP arg 28 → cys
<i>Act88F²</i>	EMS	<i>Ifm(3)4</i>	3, 4, 7	dominant antimorphic allele

allele	origin	synonym	ref ^α	comments
<i>Act88F³</i>	EMS	<i>Ifm(3)6</i>	1, 7	weak inducer of HSP ile 76 → phe
<i>Act88F⁴</i>	EMS	<i>Ifm(3)7, Act88F^{KM75}</i>	3, 7, 8	dominant antimorphic allele strong inducer of HSP trp 356 → opal
<i>Act88F⁵</i>		<i>Act88F^{HH5}</i>	2, 8	dominant antimorphic allele strong inducer of HSP gly 366 → ser
<i>Act88F⁶</i>		<i>Act88F^{KM88}</i>	2, 8	dominant hypomorphous allele trp 75 → ambara
<i>Act88F⁷</i>		<i>Act88F^{KM129}</i>	2, 8	
<i>Act88F⁸</i>	spont	<i>Act88F^{rsd}</i>	5, 6	

^α 1 = Ball, Karlik, Beall, Saville, Sparrow, Bullard, and Fyrberg, 1987, Cell 51: 221-28; 2 = Hiromi and Hotta, 1985, EMBO J. 4: 1681-87; 3 = Karlik, Coutu, and Fyrberg, 1984, Cell 38: 711-19 (fig.); 4 = Karlik, Saville, and Fyrberg, 1987, Mol. Cell Biol. 7: 3084-91; 5 = Lang, Wyss, and Eppenberger, 1981, Nature 291: 506-08; 6 = Mahaffey, Coutu, Fyrberg, and Inwood, 1985, Cell 40: 101-10; 7 = Mogami and Hotta, 1981, Mol. Gen. Genet 183: 409-17; 8 = Okamoto, Hiromi, Ishikawa, Yamada, Isoda, Mackasa, and Hotta, 1986, EMBO J. 5: 589-96.

cytology: Placed in 88F by *in situ* hybridization.

molecular biology: Genomic clones isolated by Tobin *et al.* (1980) and by Fyrberg *et al.* (1981). Sequence analysis reveals a translated sequence accounting encoding a 374-amino-acid polypeptide of molecular weight 43 kd, which shows 95% homology with the *Act79B* gene product and 92% homology with rabbit actin. The genomic sequence shows an intron of 60 nucleotides within codon 307 (Sánchez *et al.*, 1983). Deletion analysis of upstream *cis*-acting regulatory sequences carried out by Geyer and Fyrberg (1986, Mol. Cell Biol. 6: 3388-96). Arthrin, a 55-kd protein found in indirect flight muscle shown to be an unquinated form of actin III (Ball, Karlik, Beall, Saville, Sparrow, Bullard, and Fyrberg, 1987, Cell 51: 221-28).

Act88F⁴

phenotype: Dominant flightless allele; actin III replaced by a truncated polypeptide of 42 kd that is stable and capable of incorporation into myofibrils; actin II reduced in homozygotes (Hiromi and Hotta, 1985). Myofibrils in indirect flight muscles of homozygotes severely deranged; sarcomere structure obliterated; indirect-flight-muscle nuclei enlarged. Skeins of morphologically normal but highly disorganized thick filaments present, but Z discs absent. Thin filaments scarce. Electron dense material of unknown origin seen in sections. Wild-type flies transformed with cloned *Act⁴* sequence produces both the 42-kd and the heat-shock proteins (Hiromi, Okamoto, Gehring, and Hotta, 1986, Cell 44: 293-301).

Act88F⁵

phenotype: Produces half normal amount of actin isoform III; shows increased synthesis of actin I, normally present in only trace amounts in indirect flight muscle. Indirect-flight-muscle nuclei enlarged and myofibrils disrupted. Heterozygotes flightless.

Act88F⁶

phenotype: Actin III entirely absent from indirect flight muscle in homozygotes; levels of actin II also reduced.

Act88F⁷

phenotype: Actin III replaced by a truncated polypeptide of 38 kd; its low concentrations on gels suggests high instability (Hiromi and Hotta, 1985).

Act88F⁸

phenotype: Studied only in combination with *rsd* at 95.4 on chromosome 3; not examined in *rsd*⁺ background. Wings of homozygotes held straight up, nearly meeting over thorax; heterozygotes have wings held normally, but are nearly flightless. Electron microscopy of homozygotes reveals grossly abnormal indirect-flight-muscle structure; lack thin filaments and Z discs (Deak, Bellamy, Bienz, Dubuis, Fenner, Gollin, Rahmi, Ramp, Reinhardt, and Cotton, 1982, J. Embryol. Exp. Morphol. 69: 61-81). Abnormal protein accumulation observed in thoraces. Actin III and its ubiquitinated derivative, arthrin, absent in *Act88F*⁸ homozygotes (Lang *et al.*); six other polypeptides, including an indirect-flight-muscle tropomyosin isoform and two indirect-flight-muscle tropomyosin-related isoforms, markedly reduced. Homozygotes transformed with *Act88F*⁺ show restoration to approximately normal levels of the six reduced polypeptides. Accumulation of actin III and arthrin still negative, however; the latter attributed to the failure of post-translational modification in the presence of homozygous *rsd*. Viability and fertility normal.

molecular biology: a null mutant; *Act88F* mRNA reduced 10-15 fold; alteration of normal sequence apparently outside the coding region; mRNA level, and to some degree, the phenotype rescuable by germ-line transformation using *Act88F* normal genomic sequence. Sequence of coding region of DNA including 60-nucleotide intron reveals differences from that of Canton-S that account for five amino-acids substitution (Mahaffey *et al.*).

other information: Conceivable that *Act88F*⁹ is a wild-type isoallele with normal phenotype in the absence of *rsd*.

actidione sensitive: see *act*

Actin: see *Act*

Actn: α Actinin

location: 1-1.0.

synonym: *l(1)2Cb*

phenotype: The structural gene for α Actinin (Fyrberg).

Both lethal and viable alleles recovered; allelism determined by Homyk and Emerson. Viable alleles unconditionally flightless; wing position normal, but unable to fly or beat wings; jump abnormally short distances. Gynandromorph studies of *Actn*¹ indicate a bilateral pair of submissive foci located mid ventrally close to the embryonic midline (Homyk and Emerson). ERG normal (Homyk and Pye, 1989, J. Neurogenet. 5: 37-48). *Actn*⁴ is a heat sensitive lethal, and when raised at low temperature, causes aberrant wing display of courting males; *Actn*¹/*Actn*⁴ jumps and flies abnormally when raised at 22° but normally when raised at 29°; temperature sensitive period for this effect in first half of pupal stage (Homyk *et al.*, 1980). Trans heterozygotes (i.e., *Actn*³, *Actn*⁴, *Actn*⁸, and *Actn*¹⁴) with *hdp-a*² also flightless. Lethal alleles die in late larval or early pupal stages; homozygous maternal germline clones produce normal ova. Polyphasic lethality of *Actn*⁸ attributed to position effect of the inversion on arm (Perrimon, Engstrom, and

Mahowald, 1985, Genetics 111: 23-41).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>Actn</i> ¹	EMS	Homyk	<i>ftiA</i> ¹	1, 2, 3	
<i>Actn</i> ²	EMS	Homyk	<i>ftiA</i> ²	1, 2, 3	
<i>Actn</i> ³	EMS	Homyk	<i>ftiA</i> ³	1, 2, 3	
<i>Actn</i> ⁴	EMS	Homyk	<i>ftiA</i> ⁴	1, 2, 4	heat-sensitive pupal lethal
<i>Actn</i> ⁵	X ray	Lefevre	<i>l(1)A115</i>	6, 8	larval lethal
<i>Actn</i> ⁶	X ray	Lefevre	<i>l(1)C212</i>	6	<i>T(1;3)1A7;2C3;80</i>
<i>Actn</i> ⁷	X ray	Lefevre	<i>l(1)GA17</i>	6, 8	embryonic lethal (double mutant)
<i>Actn</i> ⁸	X ray	Lefevre	<i>l(1)HC207</i>	1, 6, 8	<i>ln(1)2C3;7B1</i> ; polyphasic lethal larval-pupal lethal
<i>Actn</i> ⁹	X ray	Lefevre	<i>l(1)HC288</i>	6, 8	
<i>Actn</i> ¹⁰	X ray	Lefevre	<i>l(1)HF356</i>	6	
<i>Actn</i> ¹¹	X ray	Lefevre	<i>l(1)JC111</i>	6	<i>ln(1)2C3;9A2-3</i>
<i>Actn</i> ¹²	EMS	Lefevre	<i>l(1)EA43</i>	7, 8	larval lethal
<i>Actn</i> ¹³	EMS	Lefevre	<i>l(1)EA45</i>	7	
<i>Actn</i> ¹⁴	EMS	Lefevre	<i>l(1)EA82</i>	1, 7, 8	larval lethal
<i>Actn</i> ¹⁵	EMS	Lefevre	<i>l(1)EA111</i>	7	
<i>Actn</i> ¹⁶	EMS	Lefevre	<i>l(1)VE692</i>	7, 8	larval lethal
<i>Actn</i> ¹⁷	spont	Schalet	<i>l(1)4-3</i>		
<i>Actn</i> ¹⁸	spont	Schalet	<i>l(1)17-44-1</i>		
<i>Actn</i> ¹⁹	HMS		<i>l(1)HM29</i>	5	

^α

1 = Homyk and Emerson, 1988, Genetics 119: 105-21; 2 = Homyk and Grigliatti, 1983, Dev. Genet. 4: 77-97; 3 = Homyk and Shepard, 1977, Genetics 87: 95-104; 4 = Homyk, Szidonya, and Suzuki, 1980, Mol. Gen. Genet. 177: 553-65; 5 = Kramers, Schalet, Paradi, and Huizer-Hoogteyling, 1983, Mutat. Res. 107: 187-201; 6 = Lefevre, 1981, Genetics 99: 461-80; 7 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 8 = Perrimon, Engstrom, and Mahowald, 1985, Genetics 111: 23-41.

cytology: Placed in 2C3 based on breakpoint common to three rearrangement associated lethal alleles (Lefevre). Covered by *Dp(1;3)w^{vco} = Dp(1;3)2B17-C1;3C4-5;77D3-5;81* but not by *Dp(1;Y)w⁺³⁰³ = Dp(1;Y)2D1-2;3D3-4* (Perrimon, *et al.*).

ad: arcoid

location: 2-60.7.

origin: Spontaneous.

discoverer: Curry, 38a2.

references: 1939, DIS 12: 45.

phenotype: Wings arched, broad, and somewhat shortened; crossveins close; scutellar groove shallow. Legs may be slightly shorter than wild type. RK3.

add-B: see *dmd*²

Additional sex combs: see *Asx*

ade1: adenosine1

location: 1-57 (right of *f*).

origin: Induced by ethyl methanesulfonate.

synonym: *ade1-1^{sd}*.

references: Falk and Nash, 1974, Genetics 76: 755-766.

phenotype: Eclosion delayed 2 or 3 days; delay abolished by supplementation of minimal medium with adenosine or guanosine.

ade2 (S. Henikoff and D. Nash)

location: 2-17.7 [based on 73 *cl-spd* recombinants (Keizer, Nash, and Tiong, 1989, Biochem. Genet. 27: 349-53)].

references: Johnstone, Nash, and Naguib, 1985, Biochem. Genet. 23: 539-55.

Henikoff, Nash, Hards, Bleskan, Woolford, Naguib, and Patterson, 1986, Proc. Nat. Acad. Sci. USA 83: 3919-23.

Tiong, Keizer, Nash, Bleskan, and Patterson, 1989, *Biochem. Genet.* 27: 333-48.

phenotype: Purine nucleoside auxotroph supplementable with adenine, adenosine, and inosine. Eye color reddish-brown similar to rosy. Lacks detectable levels of the fourth purine *de novo* synthetic-pathway enzyme, formylglycineamide ribotide amidotransferase (FGARAT; EC 6.33.5.3). Homozygotes and heteroallelic combinations of many alleles have defective wings; the defects include reduced wing size, deranged posterior wing margins, and extra wing veins. Macrochaetae are somewhat thinner than normal and reduced in length. *ade2*⁵, *ade2*⁶, and *ade2*⁷ are sterile, perhaps owing to general debility, when homozygous or in heteroallelic combination with one another. Only *ade2*⁷ was not tested owing to a linked lethal. Most homozygotes and heteroallelic heterozygotes display reduced viability, with the appearance of pharate adults unable to eclose.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>ade2</i> ¹	EMS	Naguib		2, 3	weak allele
<i>ade2</i> ²	γ ray			5	
<i>ade2</i> ³	γ ray			5	
<i>ade2</i> ⁴	γ ray			5	
<i>ade2</i> ⁵	γ ray			5	
<i>ade2</i> ⁶	γ ray			5	
<i>ade2</i> ⁷	γ ray			5	
<i>ade2</i> ⁸	γ ray			5	<i>In(2LR)26B</i> ; 40-41;57B-C
<i>ade2</i> ⁹	γ ray			5	<i>T(2;3)26B1-2;97D</i>
<i>ade2</i> ¹⁰	spont	Bryson, 1939	<i>pym</i> ¹	1, 5	
<i>ade2</i> ¹¹	spont	Neel, 1941	<i>pym</i> ²	4	

^α 1 = Bryson, 1940, *DIS* 13: 49; 2 = Henikoff, Nash, Hards, Bleskan, Woolford, Naguib, and Patterson, 1986, *Proc. Nat. Acad. Sci. USA* 83: 3919-23; 3 = Johnstone, Nash, and Naguib, 1985, *Biochem. Genet.* 23: 539-55; 4 = Neel, 1942, *Am. Nat.* 76: 630-34; 5 = Tiong, Keizer, Nash, Bleskan, and Patterson, 1989, *Biochem. Genet.* 27: 333-48.

cytology: Placed in 26B, probably 26B1-2, based on breakpoints common to *In(2LR)ade*⁸ = *In(2LR)26B;40-41;57B-C* and *T(2;3)ade*⁹ = *T(2;3)26B1-2;97D*. Also included in *Df(2L)ade2-1* = *Df(2L)25F;26B5-6*, *Df(2L)ade2-2* = *Df(2L)25F2-3;26D-E*, and *Df(2L)ade2-3* = *Df(2L)26A;26B5-6*.

***ade3* (S. Henikoff and D. Nash)**

location: 2-20.

origin: Induced by ethyl methanesulfonate.

discoverer: Nash.

synonym: *Gart*.

references: Johnstone, Nash, and Naguib, 1985, *Biochem. Genet.* 23: 539-55.

Henikoff, Nash, Hards, Bleskan, Woolford, Naguib, and Patterson, 1986a, *Proc. Nat. Acad. Sci. USA* 83: 3919-23.

Henikoff, Keene, Sloan, Bleskan, Hards, and Patterson, 1986b, *Proc. Nat. Acad. Sci. USA* 83: 720-24.

phenotype: Purine nucleoside auxotroph supplementable with adenine, adenosine, and inosine. Recovery of *ade3* progeny from crosses between *ade3* and *ade3/SM5* is about 1% when raised on minimal medium. Less than 3% of the normal activity purine *de novo* synthetic pathway enzyme, glycineamide ribotide transformylase [EC 2.1.2.2 (GART)]. Eye color normal.

cytology: 27C by means of *in situ* hybridization of cloned

sequence.

molecular biology: Corresponds to the cloned sequence selected by Henikoff, Keene, Tatchell, Hall, and Nasmyth [1981, *Nature* (London) 289: 37] by its ability to complement *ade8* in yeast, which codes for GART. Seven exons specify a 4.7 kb mRNA encoding the second, third, and fifth *de novo* purine biosynthetic-pathway enzyme activities, glycineamide ribotide synthetase [EC 6.3.4.13 (GARS)], aminoimidazole ribotide synthetase [EC 6.3.3.1 (AIRS)], and GART, on a 1353 amino-acid polypeptide. The first four exons also specify a 1.7 kb mRNA encoding *Gars* alone on a 434 amino acid polypeptide (Henikoff *et al.*). This smaller polypeptide is identical to the NH₂-terminal portion of the larger, except for the last amino acid, as a consequence of alternative processing of the primary transcript. The *ade3* mutation is a single base transition changing a conserved glycine (found at that position in yeast *ade8*) to a serine at amino acid 1164 of the large polypeptide.

A functional pupal cuticle protein gene is found within the first intron (interrupting the GARS domain), is encoded on the other DNA strand, and is itself interrupted by a single intron between codons 4 and 5 of a 184 amino-acid open reading frame. This intronic gene (*Pcp*) is expressed primarily in abdominal epidermal cells that secrete the pupal cuticle (Henikoff, Keene, Fachtel, and Fristrom, 1986, *Cell* 44: 33-42).

Adenine phosphoribosyl transferase:

see *Aprt*

Adenylate kinase C: see *Adk-C*

***Adh*: Alcohol dehydrogenase (M. Ashburner)**

location: 2-50.1.

references: Johnson and Denniston, 1964, *Nature* (London) 204: 906-07.

Grell, Jacobson, and Murphy, 1965, *Science* 149: 80-82.

Ursprung and Leone, 1965, *J. Exp. Zool.* 160: 147-54.

phenotype: Structural gene for alcohol dehydrogenase [ADH (EC 1.1.1.1)]. Natural populations are polymorphic for three electrophoretic alleles (*Adh*^F, *Adh*^S, *Adh*^{F-ChD}) and for three rarer electrophoretic alleles (*Adh*^{US}, *Adh*^{F'}, *Adh*^{UF}). The frequency of the *Adh*^F allele increases, at the expense of *Adh*^S, with increasing latitude in both northern and southern hemispheres [Johnson and Schaffer, 1973, *Biochem. Genet.* 10: 149-63; Vigue and Johnson, 1973, *Biochem. Genet.* 9: 213-27; Wilks, Gibson, Oakeshott and Chambers, 1980, *Aust. J. Biol. Sci.* 33: 575-85; Anderson, 1981, *Genetic Studies of Drosophila Populations* (Gibson and Oakes, eds.). Australian National University Press, pp. 237-50; Anderson and Chambers, 1982, *Evolution* 36: 86-96].

Confers resistance to ethanol; flies lacking ADH rapidly become intoxicated and eventually die on exposure to ethanol (Grell, Jacobson and Murphy, 1968, *Ann. N.Y. Acad. Sci.* 151: 441-45; Vigue and Sofer, 1976, *Biochem. Genet.* 14: 127-135; David, Bocquet, Arens and Fouillet, 1976, *Biochem. Genet.* 14: 989-97). However, ethanol sensitivity is complex since even *Adh* nulls are more resistant to ethanol when young than when old (Vigue and Sofer, 1976; Tsubota). *Adh*⁺ flies are killed by low concentrations of unsaturated secondary alcohols (e.g. 1-penten-3-ol; 1-pentyn-3-ol) but not by unsaturated

turated primary alcohols (e.g. 1-penten-1-ol) (Sofer and Hatkoff, 1972, *Genetics* 72: 545-49), presumably due to the formation of toxic ketones. This allows the chemical selection of *Adh* nulls (Sofer and Hatkoff, 1972; O'Donnell, Gerace, Leister and Sofer, 1975, *Genetics* 79: 73-83). ADH may play a metabolic role independent of alcohol detoxication, i.e. in the metabolism of higher alcohols (see Winberg, Thatcher and McKinley-McKee, 1982, *Biochem. Biophys. Acta* 704: 7-16). ADH also catalyses the oxidation of acetaldehyde to acetate (Heinstra, Eisses, Schoonen, Aben, de Winter, van de Horst, van Marrewijk, Beenackers, Scharloo and Thörig, 1983, *Genetica* 60: 129-37; Moxon, Holmes, Parsons, Irving, and Doddrell, 1985, *Comp. Biochem. Physiol.* 80B: 525-35).

Specific activity of ADH changes with development, with peaks at the end of the third larval instar and about four days after eclosion (Ursprung, Sofer and Burroughs, 1970, *Wilhelm Roux's Arch. Entwicklungsmech. Org.* 164: 201-08; Dunn, Wilson and Jacobson, 1969, *J. Exp. Zool.* 171: 185-90; Leibenguth, Rammo and Dubiczky, 1979, *Wilhelm Roux's Arch. Dev. Biol.* 187: 81-88; Maroni and Stamey, 1983, *DIS* 59: 77-79; Anderson and McDonald, 1981, *Canad. J. Genet. Cytol.* 23: 305-13). Most of the activity is in the larval fat body and gut and the adult fat body (Ursprung, Sofer and Burroughs). Maternal inheritance of ADH by embryos and larvae (O'Donnell *et al.*; Leibenguth *et al.*). Half life of ADH-F *in vivo* estimated as 55.3 hours (Anderson and McDonald, 1981, *Biochem. Genet.* 19: 411-19). Not expressed in SL2 tissue culture cells, but transfected cloned gene is (Benyajati and Dray, 1984, *Proc. Nat. Acad. Sci.* 1701-05).

Ethanol tolerance usually correlated with ADH activity and polymorphic experimental populations exposed to ethanol usually show an increase in the frequency *Adh^F* (McDonald and Avise, 1976, *Biochem. Genet.* 14: 347-55; Cavener and Clegg, 1978, *Genetics* 90: 629-44; van Delden, Kamping and van Dijk, 1975, *Experientia* 31: 418-19; Oakeshott, Gibson, Anderson and Champ, 1980, *Aust. J. Biol. Sci.* 33: 105-14; McDonald, Chambers, David and Ayala, 1977, *Proc. Nat. Acad. Sci. USA* 74: 4562-66). Flies carrying *Adh^F* tend to be more resistant than those carrying only *Adh^S* to ethanol [Kamping and van Delden, 1978, *Biochem. Genet.* 16: 541-55; Ainsley and Kitto, 1975, *Isozymes* (C. Markert, ed.). Academic Press, Vol. II, pp. 733-43; Briscoe, Robertson and Malpica, 1975, *Nature* (London) 253: 148-49].

Electrophoresis of homozygous genotypes usually reveals three interconvertible isozymes [Ursprung and Leone; Johnson and Denniston; Grell *et al.*, 1965; Ursprung and Carlin, 1968, *Ann. N.Y. Acad. Sci.* 151: 456-75; Jacobson, Murphy and Hartmann, 1970, *J. Biol. Chem.* 245: 1075-83; Jacobson and Pfuderer, 1970, *J. Biol. Chem.* 245: 3938-44; Jacobson, Murphy and Ortiz, 1972, *Arch. Biochem. Biophys.* 149: 22-35; Knopp and Jacobson, 1972, *Arch. Biochem. Biophys.* 149: 36-41; Schwartz, Gerace, O'Donnell and Sofer, 1975, *Isozymes* (C. Markert, ed.). Academic Press, Vol. I, pp. 725-51]. These vary in activity and stability, the most cathodal being more active, but less stable, than the more anodal forms. They probably result from the binding of 0, 1 or 2 moles per mole of a NAD⁺ addition

complex with a carbonyl compound [Schwartz and Sofer, 1976, *Nature* (London) 263: 129-31; Schwartz, O'Donnell and Sofer, 1979, *Arch. Biochem. Biophys.* 194: 365-78; Winberg, Thatcher and McKinley-McKee, 1983, *Biochem. Genet.* 21: 63-80]. Feeding flies acetone, propan-2-ol, or 3-hydroxy-butanone, for example, converts isozymes to most anodal form and results in loss of enzyme activity *in vitro* and *in vivo* (Schwartz and Sofer, 1976; Papel, Henderson, van Herewege, David and Sofer, 1979, *Biochem. Genet.* 17: 533-63). ADH has been purified (Sofer and Ursprung, 1968, *J. Biol. Chem.* 243: 3118-25; Schwartz *et al.*, 1975; Thatcher, 1977, *Biochem. J.* 163: 317-23; Leigh Brown and Lee, 1979, *Biochem. J.* 179: 479-82; Juan and Gonzalez-Duarte, 1980, *Biochem. J.* 189: 105-10; Elliot and Knopp, 1975, *Methods Enzymol.* 41: 374-79; Chambers, 1984, *Biochem. Genet.* 22: 529-50). It is a homodimer with monomeric subunit molecular weight of 27500 daltons (Thatcher, 1980, *Biochem. J.* 187: 875-83); molecular extinction coefficient 4.8 X 10⁴ liter/mol/cm (Juan and Gonzalez-Duarte, for ADH-S). Complete amino acid sequence determined by Thatcher (1980; see also Schwartz and Jornvall, 1976, *Europ. J. Biochem.* 68: 159-68; Auffret, Williams and Thatcher, 1978, *FEBS Lett.* 90: 324-26; Benyajati, Place, Powers, and Sofer, 1981, *Proc. Nat. Acad. Sci. USA* 78: 2317-21; Chambers, Laver, Campbell and Gibson, 1981, *Proc. Nat. Acad. Sci. USA* 78: 3103-07) with secondary structure predictions (Thatcher and Sawyer, 1980, *Biochem. J.* 187: 884-86; Benyajati *et al.*, 1981). Limited homology in supposed catalytic region with ribitol dehydrogenase of *Klebsiella* (Jornvall, Persson and Jeffry, 1981, *Proc. Nat. Acad. Sci. USA* 78: 4226-30).

ADH shows a broad substrate specificity but is more active (by at least a factor of 5) with secondary than primary alcohols and shows highest activity to 3-6 carbon alcohols (Sofer and Ursprung; Thatcher and Camfield, 1977, Winberg *et al.*, 1982, Chambers *et al.*). Differences in substrate specificity, kinetic constants and stability of different electrophoretic variants often reported (Anderson and McDonald, 1983, *Proc. Nat. Acad. Sci. USA* 80: 4798-802). Considerable heterogeneity in the specific activity of ADH within and between different *Adh^F* and *Adh^S* strains, though *Adh^S* strains tend to be lower than *Adh^F* [Day, Hillier and Clarke, 1974, *Biochem. Genet.* 11: 141-53, 155-65; Day and Needham, 1974, *Biochem. Genet.* 11: 167-75; Gibson, 1970, *Nature* (London) 227: 959-61; Gibson, Chambers, Wilkes and Oakeshott, 1980, *Aust. J. Biol. Sci.* 33: 479-89; Gibson and Miklovitch, 1971, *Experientia* 27: 99-100; Kreitman, 1980, *Genetics* 95: 467-75; Oakeshott, 1976, *Aust. J. Biol. Sci.* 29: 365-73; Sampsell, 1977, *Biochem. Genet.* 15: 971-88; Sampsell and Sims, 1982, *Nature* (London) 296: 853-55; Thörig, Schoone and Scharloo, 1975, *Biochem. Genet.* 13: 721-31; Vigue and Johnson; Hewitt, Pipkin, Williams and Chakrabarty, 1974, *J. Hered.* 65: 141-44; Ward, 1974, *Biochem. Genet.* 12: 449-58; Ward, 1975, *Genet. Res.* 26: 81-93; Maroni, Laurie-Ahlberg, Adams and Wilton, 1982, *Genetics* 101: 431-66; Rasmuson, Nilson and Zeppezauer, 1966, *Hereditas* 56: 313-16; Clarke, Camfield, Garvin and Pitts, 1979, *Nature* (London) 180: 517-18; Laurie-Ahlberg, Maroni, Bewley, Lucchesi and Weil, 1980, *Proc. Nat. Acad. Sci. USA* 77: 1073-77; Barnes

and Birley, 1978, *Heredity* 40: 51-57; Barnes and Birley, 1978, *Biochem. Genet.* 16: 155-65; McDonald and Ayala, 1978, *Genetics* 89: 371-88; McDonald *et al.*, 1980; Lewis and Gibson, 1978, *Biochem. Genet.* 16: 159-70]. With the exception of the studies by Thatcher and Sheikh (1981, *Biochem. J.* 197: 111-17), Winberg *et al.* (1982), McDonald, Anderson and Santos (1980, *Genetics* 95: 1013-22); Eisses, Schoonen, Aben, Scharloo, and Thörig (1985, *Mol. Gen. Genet.* 199: 76-81) and Moxon *et al.* (1985), these were all done with crude extracts and not purified enzyme. Thatcher and Sheikh find the relative thermostabilities to be ADH-S > ADH-F > ADH-n5 > ADH-D. ADH-S shows slower dissociation of NADH from NADN-enzyme complex than ADH-F (Winberg, Hovik, and McKinley-McKee, 1985, *Biochem. Genet.* 23: 205-16).

ADH is not a metalloenzyme (Place, Powers and Sofer, 1980, *Fed. Proc.* 39: 1640); but, paradoxically, is inhibited by certain metal ion chelators, *e.g.* pyrazole (Place, Powers and Sofer; Winberg *et al.*, 1982; Moxon *et al.*, 1985).

Utilization of ethanol as an energy source (van Herrewege and David, 1974, *C. Rend. Acad. Sci. Paris* 279D: 335-38; van Herrewege, David and Grantham, 1980, *Experientia* 36: 846-47; Libion-Mannaert, Deltombe-Lietaert, Lenelle-Montfort and Elens, 1976, *Experientia* 32: 22-23) depends on ADH activity (David, Bocquet, van Herrewege, Fouillet and Arens, 1978, *Biochem. Genet.* 16: 203-11). *Adh^F* homozygotes usually show a better ability to survive on ethanol as a sole energy source than *Adh^S* homozygotes (Daly and Clarke, 1981, *Heredity* 46: 219-26; Anderson, McDonald and Santos, 1981, *Experientia* 37: 463-64).

Adh^F and *Adh^S* homozygotes also show behavioural differences in their response to ethanol (Parsons, 1977 *Oecologia* 30: 141-46; Cavener, 1979, *Behav. Genet.* 9: 359-65; Gelan and McDonald, 1980, *Behav. Genet.* 10: 237-49; Hougonto, Lietaert, Libion-Mannaert, Feytmans and Elens, 1982, *Genetica* 58: 121-28; Parsons, 1980, *Behav. Genet.* 10: 183-90; Parsons, 1980, *Experientia* 36: 1070-71).

D. simulans enzyme monomers form heterodimers with those of *D. melanogaster* (E.H. Grell); *D. simulans* enzyme purified (Juan and Gonzalez-Duarte, 1981, *Biochem. J.* 195: 61-69). Sequence of *D. simulans* ADH (from DNA) similar to that of *Adh^S* with following changes: ser1 → ala1; gln82 → lys82; ile184 → val184 (Bodmer and Ashburner, 1984, *Nature* 309: 425-30). *D. simulans* and *D. melanogaster* enzymes differentially regulated in hybrids (Dickenson, Rowan, and Brennan, 1984, *Heredity* 52: 215-25). The *Adh* genes from *D. orena* and *D. mauritiana* have also been sequenced (Bodmer and Ashburner), and those of *D. erecta*, *D. teissieri* and *D. yakuba* mapped with restriction enzymes (Langley, Montgomery and Quattlebaum, 1982, *Proc. Nat. Acad. Sci. USA* 79: 5631-35).

alleles: Large numbers of alleles have been selected and characterized. This information is summarized in the following tables: The first table describes the origins and phenotypes of the electrophoretic variants, the majority of which were isolated from natural populations; the second describes the origins and phenotypes of the null alleles; In addition 16 isolations of null alleles from four Australian locations have been described (Freeth and Gibson, 1985, *Heredity* 55: 369-74); not clear how many mutational events represented.

allele	origin	source	discoverer	ref α	migration β rate (pl)	thermo stability
Adh 71k γ	spont		Thörig	5, 6, 13, 22	(6.4)	> Adh ^F
Adh A1 δ	recomb.	<i>Adh</i> ⁿ¹ / <i>Adh</i> ⁿ⁵	Maroni	13, 15	(7)	< Adh ^S
Adh B7 δ	recomb.	<i>Adh</i> ⁿ¹ / <i>Adh</i> ⁿ⁵	Maroni	13, 15	(7)	< Adh ^S
Adh D	EMS	<i>Adh</i> ^F	Grell	9, 19	(6)	
Adh F	spont		Johnson and Denniston	1, 7, 12, 16, 20	6.4	
Adh F'	spont	(Congo)	David	4, 17, 20	6.5	
Adh F(o) $\gamma\epsilon$	spont		Eisses	6	(6.4)	> Adh ^F
Adh F-ChD ϵ	spont		Lewis	2, 3, 8, 14, 24	(6.4)	> Adh ^F ; > Adh ^S
Adh Fm ζ	spont		Sampsell	18	(6.4)	
Adh Fr ϵ	spont		Sampsell	13, 18	(6.4)	> Adh ^F
Adh Fs	spont		Sampsell	13, 18	(6.4)	< Adh ^F
Adh I ζ	spont		Ursprung and Leone	11, 23	(6.4)	
Adh II η	spont		Ursprung and Leone	11, 23	(7)	
Adh S	spont		Johnson and Denniston		7	
Adh Sm η	spont		Sampsell	18	(7)	
Adh Ss	spont		Sampsell	13, 18	(7)	< Adh ^F
Adh UF	spont			20, 21	6.0	
Adh US	spont	(Congo)	David	4, 10, 20	7.8	

α 1 = Benyajati, Place, Powers, and Sofer, 1981, Proc. Nat. Acad. Sci. USA 78: 2717-21; 2 = Chambers, Laver, Campbell, and Gibson, 1981, Proc. Nat. Acad. Sci. USA 78: 3103-07; 3 = Chambers, Wilks, and Gibson, 1981, Aust. J. Biol. Sci. 34: 625-37; 4 = David, 1978, Recherche 9: 482-83; 5 = Eisses, Schoonen, Scharloo, Thörig, 1985, Comp. Biochem. Physiol. 82: 863-68. 6 = Eisses, Thörig, and Scharloo, 1981, Genetics 97: s33; 7 = Fletcher, Ayala, Thatcher, and Chambers, 1978, Proc. Nat. Acad. Sci. USA 75: 5609-12; 8 = Gibson, Chambers, Wilkes, and Oakeshott, 1980, Aust. J. Biol. Sci. 33: 479-89; 9 = Grell, Jacobson, and Murphy, 1968, Ann. NY Acad. Sci. 151: 441-55; 10 = Grossman, Koreneva, and Ulitscaya, 1970, Genetika (Moscow) 6(2): 91-96; 11 = Hewitt, Pipkin, Williams, and Chakrabarty, 1974, J. Hered. 65: 141-48; 12 = Johnson and Denniston, 1964; Nature (London) 204: 906-07; 13 = Kreitman, 1980, Genetics 95: 467-75; 14 = Lewis and Gibson, 1978, Biochem. Genet. 16: 159-70; 15 = Maroni, 1978, Biochem. Genet. 16: 509-23; 16 = Retzios and Thatcher, 1979, Biochemie 61: 701-04; 17 = Retzios and Thatcher, 1980, Biochem. Soc. Trans. 9: 298-99; 18 = Sampsell, 1977, Biochem. Genet. 15: 971-88; 19 = Schwartz and Jormvall, 1976, Europ. J. Biochem. 68: 159-68; 20 = Thatcher, 1980, Biochem. J. 187: 875-83; 21 = Thatcher and Camfield, 1977, Biochem. Soc. Trans. 5: 271-72; 22 = Thörig, Schoone, and Scharloo, 1977, Biochem. Genet. 13: 721-731; 23 = Ursprung and Leone, 1965, J. Exp. Zool. 160: 147-54; 24 = Wilks, Gibson, Oakeshott, and Chambers, 1980, Aust. J. Sci. 33: 375-85.

β Numbers in parentheses inferred from phenotypic description; others represent actual measurements (Thatcher, 1980, Biochem J. 187: 875-83).

γ Unlike *Adh*^F, will oxidize dihydroorotic acid to orotic acid and sarcosine to glycine.

δ Probably identical.

ϵ Probably the same as *Adh*^{71k}.

ζ = *Adh*^F.

η = *Adh*^S.

allele	origin α	derivative of	discoverer	ref β	activity	CRM	forms active hybrid enzyme	notes
Adh fn4	formaldehyde	<i>Adh</i> ^D	Sofer	2-4, 9	-	-		
Adh fn6	formaldehyde	<i>Adh</i> ^D	Sofer	2-4, 9	-	-		
Adh fn23	formaldehyde	<i>Adh</i> ^D	Sofer	2-4, 9	-	+		γ
Adh fn24	formaldehyde	<i>Adh</i> ^D	Sofer	2-4, 9	-	-		
Adh n1	EMS	<i>Adh</i> ^S	E.H. Grell	6, 7, 10, 14	20%	+	+	δ
Adh n2	EMS	<i>Adh</i> ^S	E.H. Grell	6, 7, 10, 14	-	5%	-	
Adh n3	EMS	<i>Adh</i> ^S	E.H. Grell	6, 7, 10, 14	-	15%	-	
Adh n4	EMS	<i>Adh</i> ^D	E.H. Grell	6, 7, 10, 14	-	-		
Adh n5	EMS	<i>Adh</i> ^D	E.H. Grell	6, 12-14, 17, 18	leaky ts	-	+	ϵ
Adh n6	EMS	<i>Adh</i> ^F	Gerace	5, 9, 14	-	44%	-	
Adh n7	EMS	<i>Adh</i> ^F	Gerace	5, 9, 14	-	54%	-	
Adh n8	EMS	<i>Adh</i> ^F	Gerace	5, 9, 14	-	61%	-	
Adh n9	EMS	<i>Adh</i> ^F	Gerace	5, 9, 14	-	71%	-	
Adh n10	EMS	<i>Adh</i> ^F	Gerace	5, 9, 14	-	-	-	
Adh n11	EMS	<i>Adh</i> ^F	Sofer	9-12, 13, 14-17	0.02%	27%	+	ζ
Adh n12	EMS	<i>Adh</i> ^F	Sofer	9, 10, 14	-	73%	-	
Adh n13	EMS	<i>Adh</i> ^F	Sofer	9, 10, 14	-	5%	-	
Adh n14	EMS	<i>Adh</i> ^F	Sofer	9, 10, 14	-	-	-	
Adh n967	spont			18				η
Adh nA	EMS	<i>CyO</i> , <i>Adh</i> ^F	Sofer	5, 14	-	-		
Adh nB	EMS	<i>CyO</i> , <i>Adh</i> ^F	Sofer	5, 14	-	+		θ
Adh nC1	EMS	<i>Adh</i> ^{UF}	Ashburner					
Adh nC2	EMS	<i>Adh</i> ^{UF}	Ashburner		leaky ts			
Adh nLA2	X ray	<i>Adh</i> ^F	Aaron	1, 8	-	+		ι
Ash nLA73	X ray	<i>Adh</i> ^F	Aaron	1, 8	-	-		
Adh nLA74	X ray	<i>Adh</i> ^F	Aaron	1, 8	-	+		
Adh nLA80	X ray	<i>Adh</i> ^F	Aaron	1, 8	-	+		κ
Adh nLA248	X ray	<i>Adh</i> ^F	Aaron	1, 8	-	-		

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin α	derivative of	discoverer	ref β	activity	CRM	forms active hybrid enzyme	notes
Adh^{nLA249}	X ray	<i>Adh^F</i>	Aaron	1, 8	-	+		
Adh^{nLA252}	X ray	<i>Adh^F</i>	Aaron	1, 8	-	+		
Adh^{nLA319}	spont	<i>Adh^D</i>	Aaron	1, 8	-	+		
Adh^{nLA378}	X ray	<i>Adh^F</i>	Aaron	1, 8	-	+		
Adh^{nLA405}	X ray	<i>Adh^F</i>	Aaron	1, 8	-	+		1

α *Adh^{fn4}* - *Adh^{fn24}* and *Adhⁿ¹¹* - *Adhⁿ¹⁴* selected as larvae on pentenol; *Adhⁿ⁶* - *Adhⁿ¹⁰*, *Adh^{nA}*, *Adh^{nB}*, and the *Adh^{nLA}* series of alleles selected as adults on pentenol.

β 1 = Aaron, 1979, Mutat. Res. 63: 127-37; 2 = Benyajati, Place, Powers, and Sofer, 1981, Proc. Nat. Acad. Sci. USA 78: 2717-21; 3 = Benyajati, Place, and Sofer, 1983, Mutat. Res. 111: 1-7; 4 = Benyajati, Place, Wang, Pentz, and Sofer, 1982, Nucleic Acids Res. 10: 7261-72; 5 = Gerace and Sofer, 1972, DIS 49: 39; 6 = Grell, Jacobson, and Murphy, 1968, Ann. N.Y. Acad. Sci. 151: 441-55; 7 = Kamping and van Delden, 1980, DIS 55: 89; 8 = Kelley, Mims, Farnet, Dicharry, and Lee, 1985, Genetics 109: 365-77; 9 = O'Donnell, Gerace, Leister, and Sofer, 1975, Genetics 79: 73-83; 10 = Pelliccia and Sofer, 1982, Biochem. Genet. 20: 297-313; 11 = Reddy, Pelliccia, and Sofer, 1980, Biochem. Genet. 18: 339-51; 12 = Sampsel, 1977, Biochem. Genet. 15: 971-88; 13 = Schwartz and Jomvall, 1976, Europ. J. Biochem. 68: 159-68; 14 = Schwartz and Sofer, 1976, Genetics 83: 126-36; 15 = Thatcher, 1980, Biochem. J. 187: 875-83; 16 = Thatcher and Retzios, 1980, Protides of Biol. Fluids 28: 157-60; 17 = Thatcher and Sheikh, 1981, Biochem. J. 197: 111-17; 18 = Vigue and Sofer, 1974, Biochem. Genet. 11: 387-96.

γ Polypeptide smaller than wild type.

δ Polypeptide slightly larger than wild type on SDS gels and electrophoretic mobility altered on non-denaturing gels.

ϵ Purified enzyme thermostable; *Adhⁿ⁵* flies grown at 18° show loss of both ADH activity and CRM following a shift to 30° (Tsubota); recovery on return to 18° takes several days (Vigue and Sofer; Tsubota).

ζ Mutation in adenine ribose pocket of coenzyme binding domain; is not bound to 5' -AMP sepharose and cannot recognize NAD⁺ (Thatcher and Retzios), *Adh^F* / *Adhⁿ¹¹* forms an active dimer that migrates as *Adh^F* / *Adh^{UF}* (Schwartz and Jomvall; Schwartz and Sofer, Pelliccia and Sofer). Shows weak intracistronic complementation with *Adhⁿ⁶*, *Adhⁿ⁷*, *Adhⁿ¹²* and *Adh^{nA}* (Thatcher; Reddy *et al.*). *Adhⁿ⁶* / *Adhⁿ¹¹* heterozygotes display partial resistance to alcohol; hybrid enzyme activity heat labile; displays altered substrate binding properties (Pelliccia and Couper, 1984, DIS 60: 160-62).

η Isolated from natural population.

θ Polypeptide shorter than normal (24 kilodaltons).

ι *In vitro* translation product of mRNA smaller than that of wild type (Pelham and Bodmer).

κ Protein unstable by two-dimensional gel electrophoretic analysis.

cytology: Placed in 35B3 by *in situ* hybridization.

molecular biology: Structural gene cloned (Goldberg, 1980, Proc. Nat. Acad. Sci. USA 77: 5794-98) and sequenced [Goldberg; Benyajati *et al.*, 1981; Haymerle, 1983, Thesis, University of Cambridge; Kreitman, 1983, Nature (London) 304: 412-17; Benyajati, Place, Wang, Pentz, and Sofer, 1982, Nucleic Acids Res. 10: 7261-72]. Partial sequence (3' end) of cDNA clone by Benyajati, Wang, Reddy, Weinberg and Sofer (1980, Nucleic Acids Res. 8: 5649-67). Variation in restriction enzyme sites within and around *Adh* (Langley *et al.*). *Adh^F* alleles are polymorphic for insertion substitution changes within the 5' non-coding region intron (Kreitman, 1983). Sequence comparisons between 5' flanking regions and exons in *D. melanogaster* and *D. simulans* indicate excess polymorphism in the *D. melanogaster* 5' flanking region (Kreitman and Aguade, 1986, Genetics 114: 93-100; Aquadro, Desse, Blond, Langley, and Laurie-Ahlberg, 1986, Genetics 114: 1165-90).

Standard amino acid sequence taken to be that of ADH-S (Thatcher, 1980 with two corrections: glu25 (not gln) and an extra tryptophan at 251 (Benyajati *et al.*, 1981). Standard DNA sequence is that of *Adh^S* allele from clone pSAC1 of Goldberg (Benyajati *et al.*, 1981, 1982, 1983; Haymerle); numbered from -1/+1, +1 being the 'A' of the ATG initiating codon. All changes with respect to coding strand.

Two primary transcripts: major larval transcript initiated from -69, 24 bp from a TATA box (-100 to -94); major adult transcript initiated from -766, 24 bp from a TATA box (-808 to -800). The major adult transcript is processed by the removal of an intervening sequence between -689 and -36. There are two introns within the coding sequence, from +100 to +164 and from +571 to +639. The polyA addition site is from +1028 to +1034 and the 3' end of the mRNA at +1079. (Benyajati *et al.*, 1981, 1983, Henikoff, 1983, Nucleic Acids Res.

11: 4735-52).

An 11.8 kb *SacI* restriction fragment of the *Adh^F* allele shown by P-element-mediated germline transformation to contain all *cis*-acting DNA sequences necessary for correct expression (quantitative levels of mRNA and enzyme; tissue specificity; developmental switch in promoter usage)(Goldberg, Posakony, and Maniatis, 1983, Cell 34: 59-73. *In vitro* recombinants rule out the 5' flanking sequences as responsible for the two- to three-fold higher enzyme activity and increased amount of ADH protein in *Adh^F* compared to *Adh^S*; the only consistent differences are at three nucleotide positions, one at 1490 responsible for the electrophoretic difference and two silent third-codon substitutions at nucleotides 1443 and 1527 (Laurie-Ahlberg and Stamm, 1987, Genetics 115: 129-40); increase probably not attributable to increased levels of mRNA (Laurie and Stamm, 1988, Proc. Nat. Acad. Sci. USA 85: 5161-65). Transcript of *Drosophila* gene transfected into yeast spliced normally (Watts, Castle, and Beggs, 1983, EMBO J. 2: 2085-91). A fusion of *Adh* to the *Hsp70* promoter has been inserted into a *P* element and used in transformation experiments in *Adh* deficient flies; in such transformants *Adh⁺* function is under heat-shock control (Bonner, Parks, Parker-Thornberg, Mortin and Pelham, 1984, Cell 37: 979-91). Molecular information on alleles in following table.

allele	molecular biology	ref α
Adh^{71k}	lys 192 → thr 192; pro 214 → ser 214	7
Adh^D	lys 192 → thr 192; gly 232 → glu 232	10, 12
Adh^F	lys 192 → thr 192; A713 → C713	1, 10, 11, 14
Adh^{F'}	ala 51 → glu 51	10
Adh^{F-ChD}	lys 192 → thr 192; pro 214 → ser 214	4
Adh^{fn4}	No mature mRNA; 16 bp deletion in first intron (146-162); AG(163-164) splice acceptor changed to GG splicing defective.	2, 3
Adh^{fn6}	No mature mRNA; 6 bp deletion in	2, 3

allele	molecular biology	ref α
	first intron (106-111); 101-105 substituted by CGATC; splicing defective.	
Adh fn23	34 bp deletion in 3' coding region (724-758); read through of normal termination triplet.	2, 3
Adh fn24	50% wild-type mRNA level; 11 bp deletion in second exon (256-266); Premature chain termination	2, 3
Adh n4	C312 \rightarrow T312; gln 83 \rightarrow ter 83	2, 3
Adh n11	Destroys Pvu II site (Chia)	
Adh nB	gly 14 \rightarrow asn 14	10, 12, 13
	UGG \rightarrow UGA	8, 9
	in trp 234 codon; suppressible in vitro with yeast ochre suppressor tRNA	
Adh nLA248	250 bp insertion formed by unequal crossover between exon 3 (at +708) and exon 2 (at +465) with 7bp (GTGCAAC) inserted at the junction.	5, 6
Adh S	standard nucleotide sequence	1, 2, 3, 10, 13
Adh UF	asn ⁸ \rightarrow ala ⁸ ; ala ⁴⁵ \rightarrow asp ⁴⁵ ;	10, 13, 14
Adh US	lys 192 \rightarrow thr 192 lys 192 unchanged	11

α 1 = Benyajati, Place, Powers, and Sofer, 1981, Proc. Nat. Acad. Sci. USA 78: 2717-21; 2 = Benyajati, Place, and Sofer, 1983, Mutat. Res. 111: 1-7; 3 = Benyajati, Place, Wang, Pentz, and Sofer, 1982, Nucleic Acids Res. 10: 7261-72; 4 = Chambers, Laver, Campbell, and Gibson, 1981; Proc. Nat. Acad. Sci. USA 78: 3103-07; 5 = Chia, Karp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706; 6 = Chia, Savakis, Karp, Pelham, and Ashburner, 1985, J. Mol. Biol. 186: 679-88; 7 = de Boer, Andriess, and Weisbeek; 8 = Kubli, Schmidt, Martin, and Sofer, 1982, Nucleic Acids Res. 10: 7145-52; 9 = Martin, Place, Pentz, and Sofer, 1985, J. Mol. Biol. 184: 221-29; 10 = Retzios and Thatcher, 1979, Biochimie 61: 701-04; 11 = Retzios and Thatcher, 1980, Biochem. Soc. Trans. 9: 298-99; 12 = Schwartz and Jornvall, 1976, European J. Biochem. 68: 159-68; 13 = Thatcher, 1980, Biochem. J. 187: 875-83; 14 = Thatcher and Camfield, 1977, Biochem. Soc. Trans. 5: 271-72.

adl: see *l(1)adl*

adp⁶⁰: adipose

location: 2-83.4.

origin: Spontaneous.

discoverer: Doane, 1960.

references: 1961, DIS 35: 78.

1963, DIS 38: 32.

1963, Proc. 23rd Ann. Biol. Coll., Oregon State Univ. Press, Corvallis, pp. 65-88.

1969, J. Exp. Zool. 171: 321-42.

phenotype: Adult fat body hypertrophies as cells become distorted by enormous oil globules. Lipid accumulated at expense of glycogen in fat body; yolk deposition retarded (Doane, 1963, DIS 37: 73-74; Doane, 1980, Evolution 34: 868-74). Lipid content and fatty acid profiles compared for various developmental stages in *adp⁶⁰* and wild type (Teague, Clark, and Doane, 1986, J. Exp. Zool. 240: 95-104). Abnormal fat bodies visible through body wall of 6-day-old and older adults when submerged in 95% alcohol and then water. Corpus allatum of mated females hypertrophies. Females fertile but egg hatchability reduced to 45-90%, depending on residual genome; dorsal appendages of chorion convoluted or fused (King and Koch, 1963, Quant. J. Microscop. Sci. 104: 297-320); adult emergence lowered to 33-85%. (Doane, 1963, DIS. 37: 73-74). Males viable and fertile. Hetero-

zygotes show desiccation tolerance superior to that of wild type or *adp⁶⁰* homozygotes (Clark and Doane, 1983, Hereditas 99: 165-75). RK3.

cytology: Placed in 55A-C1 based on its inclusion in *Df(2R)PC4 = Df(2R)55A;55F* but not *Df(2R)P29 = Df(2R)55C1-2;56B1-2* (Doane and Dumapias, 1987, DIS 66: 49).

adp^{fs}: adipose-female sterile

origin: Spontaneous.

discoverer: Counce, 1956.

synonym: *fs(2)adp: female sterile(2) adipose*.

references: Doane, 1959, Genetics 44: 506.

1960a, J. Exp. Zool. 145: 1-22 (fig.).

1960b, J. Exp. Zool. 145: 23-42.

1961, J. Exp. Zool. 146: 275-98.

phenotype: Adult fat body phenotype like *adp⁶⁰*; lipid accumulated at expense of glycogen in fat body; yolk deposition in ovaries retarded; carbohydrate levels low in 8-day-old adults (Cummings and Ganetzky, 1972, DIS 30: 48. Corpus allatum hypertrophies in mated females to same degree as in *adp⁶⁰*. Females completely sterile; sterility autonomous. Eggs laid by homozygotes show meiotic or mitotic abnormalities, or both, never develop beyond early cleavage stages; chorion, chorionic filaments and vitelline membrane defective in some. Males 78% fertile. Heterozygotes fertile, but females become sterile with age. Viability generally good but longevity reduced; homozygotes with selective advantage under starvation; heterozygotes superior under desiccation. Average water content of well-fed adults reduced; percentage of lipids, as a function of dry body weight, almost double that of wild type. Iodine numbers show greater degree of saturation of mutant lipid extracts than of wild type. RK3.

***ae: aeroplane**

location: 2-55.8.

origin: Spontaneous.

discoverer: Mohr, 26k24.

references: Quelprud, 1931, Hereditas 15: 97-119 (fig.).

phenotype: Wings spread, balancers drooping. Overlaps wild type. RK3.

***Ae: Aechna**

location: 3- (rearrangement).

origin: X ray induced.

discoverer: Belgovsky, 45a14.

references: 1946, DIS 20: 63.

phenotype: Wings spread at right angles to body axis. Homozygous lethal. RK1A.

other information: Reduced crossing over in the *th-e* region suggests presence of pericentric inversion.

aea: see *dv²*

aeroplane: see **ae**

ag: agametic

location: 1-20.7.

origin: Spontaneous.

references: Engstrom, Caulton, Underwood, and Mahowald, 1982, Dev. Biol. 91: 163-70 (fig.).

phenotype: Maternal effect mutant; approximately 40% of gonads of progeny of homozygous females agametic. Although some eggs of homozygous females exhibit

abnormal polar granules, normal numbers of pole cells form; some pole cells abnormal with degenerating polar granules and nuclear bodies, but pole cells reach gonads at 14 hr of development and then in 40% of the gonads become necrotic and disappear; responses of right and left gonads correlated. Phenotype most pronounced at 25°, decreasing at higher and lower temperatures. Mutant not completely recessive; expression in progeny of heterozygous females half that in those of homozygotes.

cytology: Placed between 7B4 and 7C1 based on its position to the right of *ct* and its inclusion in *Df(1)ct268-42 = Df(1)7A5-6;7B8-C1*.

***agl: angle winglike**

location: 1- (not located).

origin: Recovered among descendants of flies treated with natural gas.

discoverer: Mickey, 49c7.

synonym: Originally called *angle wing* but this name preoccupied by *ang*.

references: 1950, DIS 24: 60.

phenotype: Wing bent upward in middle. Overlaps wild type. RK3.

agn: agnostic

location: 1-38.9.

references: Savvateeva, Korochkina, Peresleny, and Kamyshev, 1985, DIS 61: 144.

Savvateeva, Peresleny, Ivanushina, and Korochkin, 1985, Dev. Genet 5: 157-72.

phenotype: Identified as three temperature sensitive lethal mutations. Adenylate cyclase activity somewhat higher than normal at 22° and readily activated at 29°. Phosphodiesterase activity assayed in heat-pretreated homogenates higher than normal. Locomotor activity decreased and learning activity increased at 22°, like *dnc* at 29°.

alleles:

allele	origin	synonym
<i>agn</i> ¹	EMS	<i>l(1)ts398</i>
<i>agn</i> ²	EMS	<i>l(1)ts622</i>
<i>agn</i> ³	EMS	<i>l(1)ts980</i>

agq: atrophie gonadique

location: 2-3 polygenic.

origin: Recovered from natural population on French Mediterranean coast.

references: Periquet, 1970, DIS 45: 33.

1979, Biol. Cell. 33: 33-38.

phenotype: Gonads atrophic either unilaterally or bilaterally owing to pole cell degeneration. Degree of effect correlated with both temperature during the first hours of development and with the number of *agq*-derived autosomes. Pole cells, but not oocytes, thermosensitive. Responses of right and left gonads correlated. Penetrance higher in females than males.

al: aristaless

location: 2-0.4 [Golubovsky, Kulakov, and Korochkina, 1978, Genetika (Moscow) 14: 294-305].

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 213 (fig.).

Stern and Bridges, 1926, Genetics 11: 510 (fig.).

phenotype: Aristae strongly reduced. Postscutellars

widely separated and erect but strongly divergent. Scutellum shortened; sternopleurals irregular in size, position, and number; wings slightly bowed downward, narrowed, and pointed; first longitudinal vein raised and thickened. Tarsal claws transformed into bristle-like structures or absent; effect enhanced by presence of *th* or *ss*^{a40a} or both [Mglinetz and Ivanova, 1975, Genetika (Moscow) 11, 4: 88-96]. Enhances transformations of *Antp*^{NS} [Mikuta and Mglinetz, 1978, Genetika (Moscow) 14: 1578-85] and *ss*^{a40a}; *al ss*^{a40a} flies exhibit partial transformation of third antennal segment to basitarsus; anomalous outgrowths of distal basitarsus and foreshortening of second and third tarsal joints of thoracic legs; spineless phenotype also enhanced. *al* females exhibit reduced mating success (Burnet, Connolly, and Dennis, 1971, Anim. Behav. 19: 409-15). RK1.

alleles:

allele	origin	discoverer	ref ^α	comments
<i>al</i> ¹	spont	Bridges, 17k7	2, 6, 7	viable
<i>al</i> ²	spont	Stern, 26a	7	< <i>al</i> ¹ ; poorly viable
* <i>al</i> ³	spont	Bridges, 33g2		> <i>al</i> ¹ ; semilethal; female sterile
<i>al</i> ⁴	spont	Bridges, 33l27	1, 2	< <i>al</i> ¹ ; viable; <i>In(2LR)bw</i> ^{V1}
<i>al</i> ⁸	X ray		4	> <i>al</i> ¹ ; lethal; <i>In(2LR)21C1-2;41C</i>
<i>al</i> ³⁶	X ray	Glass, 36c	2, 3	= <i>al</i> ¹ ; viable
* <i>al</i> ^{M60}	X ray	Meyer, 60f	2, 5	lethal; <i>In(2LR)</i> ; variegated?
<i>al</i> ^v	X ray	E.B. Lewis, 1940	1	lethal; <i>In(2LR)21B-C1;41</i> ; variegated

^α 1 = Bridges, 1935, DIS 3: 5; 2 = CP627; 3 = Glass, 1939, DIS 12: 47; 4 = Korochkina and Golubovsky, 1978, DIS 53: 197-200; 5 = Meyer, 1963, DIS 37: 50; 6 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 213 (fig.); 7 = Stern and Bridges, 1926, Genetics 11: 510-511 (fig.).

^β Phenotype defined with respect to homozygous viability and strength of expression in comparison with *al*¹.

cytology: Placed in 21C1-2 doublet on the basis of its inclusion in *Df(2L)al = Df(2L)21B8-C1;21C8-D1* but not in *Df(2L)S5 = Df(2L)21C2-3;22A3-4* (Lewis, 1945, Genetics 30: 137-166).

***al*⁸**

phenotype: Homozygous lethal; 1% survival in combination with *Df(2L)al = Df(2L)21B8-C1;21C8-D1*; survivors have reduced aristae, broad thorax, arched wings with incomplete veins, enlarged eyes.

cytology: Associated with *In(2LR)al*⁸ = *In(2LR)21C1-2;41C*.

al-b: see *aa*

ala: see *dy*^{ala}

ala parvae: see *dy*^{ala}

^β *alanyl dopamine hydrolase*: see *t*

^β *alanyl dopamine synthetase*: see *e*

alarless: see *alr*

***alb*: alberich**

location: 2-(unmapped).

origin: Induced by ethyl methanesulfonate.

references: Tearle and Nüsslein-Volhard, 1987, DIS

66: 209-26.

phenotype: Maternal-effect lethal; occasional embryo lacks abdominal segments (Lehmann).

Alcohol dehydrogenase: see *Adh*

ald: *altered disjunction* (A. T. C. Carpenter)

location: 3-61.

origin: Induced by ethyl methanesulfonate.

references: O'Tousa, 1982, *Genetics* 102: 503-24.

phenotype: Homozygous females display elevated levels of nondisjunction of *X* and fourth chromosomes (9.5 and 6.0% respectively); double exceptions are predominantly *XX;0* and *0;44*, products expected from nonhomologous disjunction; behavior of large autosomes nearly normal. Exchange frequencies normal, and sex-chromosome exchange tetrads contribute to exceptional products.

Ald: Aldolase

location: 3-91.5.

origin: Naturally occurring polymorphism.

references: Voelker, Ohnishi, and Langley, 1979, *Biochem. Genet.* 17: 769-83.

phenotype: Structural gene for fructose-biphosphate aldolase (EC 4.1.2.13). Enzyme multimeric based on the formation of heteromultimeric bands on gels from heterozygotes for electrophoretic variants. Amino-acid sequence determined; protein is a 158-kd multimer comprising four 360-amino-acid polypeptides. Monomers show 71% identity with rabbit muscle aldolase (Malek, Suter, Frank, and Brenner-Holzach, 1985, *Biochem. Biophys. Res. Comm.* 126: 199-205). Mixed multimers of rabbit and *Drosophila* subunits able to function (Brenner-Holzach and Leuthard, 1972, *Eur. J. Biochem.* 31: 423-26). Sequence analysis suggests alternating domains of alpha helix and beta sheet; domain boundaries correspond to boundaries between exons as seen in rat-liver aldolase (Sawyer, Fothergill-Gilmore, and Freemont, 1988, *Biochem. J.* 249: 789-93).

cytology: Placed in 97A-B on the basis of its being between the autosomal breakpoints of $T(Y;3)R87 = T(Y;3)97A$ and $T(Y;3)B158 = T(Y;3)97B$.

alleles: Two electrophoretic variants described; *Ald*⁴ product migrates toward the anode more rapidly than that of *Ald*².

Aldehyde oxidase: see *Aldox*

Aldolase: see *Ald*

Aldox-1: Aldehyde oxidase-1

location: 3-57.2 (between *red* and *sbd*).

origin: Naturally occurring polymorphism.

synonym: *Ao*.

references: Dickinson, 1970, *Genetics* 66: 487-96.

Warner, Watts, and Finnerty, 1980, *Mol. Gen. Genet.* 180: 449-53.

Warner and Finnerty, 1981, *Mol. Gen. Genet.* 184: 92-96.

Bogart and Bernini, 1981, *Biochem. Genet.* 19: 929-46.

phenotype: Structural gene for aldehyde oxidase [AO-1 (EC 1.2.3.1)]. A molybdenum-containing homodimer with subunits of 140,000-dalton molecular weight. As with other molybdenum hydroxylases, activity is inhibited by tungsten and depends on the presence of a low-molecular-weight cofactor (Warner and Finnerty).

Enzyme activity increases nonuniformly during development with step increases at pupation and midway through pupal stage. First increase appears to be controlled by a closely linked *cis*-acting element (Dickinson, 1975, *Dev. Biol.* 42: 131-40) and the latter by *Aldox-2*⁺ (Bentley and Williamson, 1979, *Z. Naturforsch.* 34: 304-05). Control independent of that of *lpo* located 0.08 unit to the left (Dickinson and Weisbrod, 1976, *Biochem. Genet.* 14: 709-21). Enzyme activity absent in *cin* (Browder and Williamson, 1976, *Develop. Biol.* 53: 241-49) and *mal* and reduced in *lxd* (Courtright, 1967, *Genetics* 57: 25-39); cross-reacting material observed in all three genotypes (Browder, Wilkes, and Tucker, 1982, *Biochem. Genet.* 20: 111-24). These mutants presumably affect the availability of molybdenum cofactor (Warner and Finnerty). Autonomous in transplants. Enzyme composition in egg and early larva reflects maternal genotype, giving way to that of zygotic genotype during larval life. Tissue specificity varies with stage and strain (Dickinson, 1972, *Genetics* 71: s14; Cypher, Tedesco, Courtright and Kumaran, 1982, *Biochem. Genet.* 20: 315-32). Heptaldehyde serves as specific substrate (Cypher *et al.*). Differential staining for enzyme noted in different compartments of the wing imaginal disc (Kuhn and Cunningham, 1976, *Genetics* 83: s42).

alleles: Three electrophoretic variants superscripted 1, 2, and 3 in order of increasing mobility described by Dickinson (1970) presumably correspond to *Aldox-1*⁴, *Aldox-1*⁶, and *Aldox-1*⁸ (1978, *DIS* 53: 117); 6 electrophoretic variants numbered in order of increasing mobility from 1 through 6 described by Langley, Tobar, and Kojima (1974, *Genetics* 78: 921-36). Correspondence of two sets of alleles unknown. Two null alleles superscripted *n1* and *n2* are homozygous viable but produce no recognizable product either in the form of enzyme activity or by the formation of heterodimers with functional gene products; however, cross-reacting material found in larval hemolymph but not in extracts of adults (Browder *et al.*, 1982). Thirteen other null alleles isolated from natural populations in Great Britain (*Aldox*^{nGB1} to *Aldox*^{nGB4}) and North Carolina (*Aldox*^{nNC1} to *Aldox*^{nNC9}); all thirteen exhibit residual enzyme activity and except for *Aldox*^{nGB1} are *Aldox*⁴ derivatives and fail to participate in heterodimer formation with normal gene products. *Aldox*^{nGB1} is an *Aldox*⁸ derivative and forms heterodimers (Burkhart, Montgomery, Langley, and Voelker, 1984, *Genetics* 107: 295-306). *lao*: low aldehyde oxidase (Collins, Duck, and Glassman, 1971, *Biochem. Genet.* 5: 1-13) considered allelic based on its phenotype and genetic position (3-56); aldehyde oxidase levels of *Aldox*ⁿ/*Aldox*^{lao} higher than expected if *Aldox*ⁿ amorphic.

cytology: Placed in region between 88F9 and 89B4 on the basis of its inclusion in $Df(3)sbd105 = Df(3R)88F9-88A1,89B4-5$ but not $Df(3R)sbd45 = Df(3R)89B1-4;89B10-13$ (Spillmann and Nöthiger, 1978, *DIS* 53: 124). Narrowed to 89A1-2 by Langhout and van Breugel (1985, *DIS* 61: 181) on basis of reduced staining of that doublet in *Aldox*ⁿ¹.

Aldox-1^{Pa1}

origin: Polymorphic in laboratory and natural populations.

references: Dickinson, 1975, *Dev. Biol.* 42: 131-40.

phenotype: Exhibits substantial increase in enzyme

activity between late larval and early pupal stages followed by a second increase in late pupal stage. Results in relatively high pupal:adult activity ratio.

Aldox-1^{Pa2}

origin: Polymorphic in laboratory and natural populations.
references: Dickinson, 1975, Dev. Biol. 42: 131-40.
phenotype: Exhibits little change in enzyme activity between late larval and early pupal stages, but there is a substantial increase in late pupal and adult stages. Results in relatively low pupal:adult activity ratio. *Aldox-1^{Pa1}/Aldox-1^{Pa2}* has intermediate phenotype.
other information: 1.5% recombination with electrophoretic *Aldox-1* alleles recorded. The difference between *Pa1* and *Pa2* postulated to reside in a cis-acting site that exerts temporal control on gene activity.

Aldox-1^{Pb1}

origin: Polymorphic in natural population from Lima, Peru.
references: Dickinson, 1978, J. Exp. Zool. 206: 333-42.
phenotype: Cytochemically, enzyme activity in paragonia uniformly distributed and high at eclosion. By end of first week of adult life enzyme accumulated into intracellular bodies giving accessory gland a spotted appearance.
alleles: *Aldox-1^{Pb2}* characterized by lower activity that remains uniformly distributed in paragonia as detected histochemically.
other information: Not separated genetically from electrophoretic alleles of *Aldox-1*. *Pb1* and *Pb2* postulated to differ in a cis-acting control site with tissue-specific effects on gene activity.

aldox2: aldehyde oxidase 2

location: 2-82.9 (based on 112 *c-px* recombinants).
origin: Naturally occurring polymorphism.
references: Bentley and Williamson, 1979, Z. Naturforsch. 34: 304-05.
 1980, Genetics 94: s8.
 Meidinger and Bentley, 1986, Biochem. Genet. 24: 683-99.
 Bentley, Meidinger, and Braaten, 1989, Biochem. Genet. 27: 99-118.
phenotype: Not a structural gene for aldehyde oxidase. Homozygotes for *aldox2* fail to show increased levels of molybdoenzymes, aldehyde oxidase, pyridoxal oxidase, sulfite oxidase, and xanthine oxidase, that normally occur late in the pupal period; adult levels lower than normal. Enzyme activity less sensitive to tungsten and more responsive to molybdenum than in wild type; aldehyde oxidase activity, at least, more heat labile and pH optimum slightly more acidic than normal (Meidinger and Bentley, 1984, Genetics 107: s73). Duplication of *aldox2⁺* without effect on enzyme levels.
cytology: Placed in region 54 based on the failure of *Y^{P2D}* from *T(Y;2)H149 = T(Y;2)h21;54F* to cover it and its genetic map position to the right of *Amy*, which has been placed in 54A1-B1 by *in situ* hybridization.

ale: almond eye

location: 3-47.5 (located with respect to *D* and *Sb*).
origin: Spontaneous in natural population.
references: Golubovsky and Zakharov, 1972, DIS 49: 112.
 Golubovsky, Ivanov, and Zakharov, 1973, Genetika (Moscow) 9(8): 168-71.

phenotype: In homozygotes, eye almond shaped but with normal facet development. Phenotype normal in heterozygotes with normal third but mutant when heterozygous to *D. Dfd/ale* show normal eye size, and 20% of flies show tufted vibrissae characteristic of *Dfd/+*. *ale Mc* homozygotes completely eyeless.

other information: Both *Dfd* and *ale* map to 47.5. No wild-type recombinants recovered among 569 tested progeny of *Dfd/ale* females. *ale* acts as a transdominant suppressor of *Dfd*.

Aliⁿ: Aliesterase-negative

location: 3- (not located).
origin: Spontaneous.
synonym: *ali: aliesteraseless*.
references: Ogita, 1961, Botyu-Kagaku 26: 93-97.
 1962, DIS 36: 103.
phenotype: Probably the structural gene for aliesterase [ALI (EC 3.1.1.1)]. Homozygotes for *Aliⁿ* practically unable to hydrolyze methyl butyrate, whereas wild type shows high activity; *Aliⁿ/+* exhibits intermediate activity. Homozygotes shown by Beckman and Johnson to lack a normally present esterase that migrates slowly on starch gel (their band F). RK3.
alleles: *Aliⁿ* is a null allele; no other variants reported.

Alkaline phosphatase: see **Aph and Aph-2**

aliesteraseless: see *Aliⁿ*

almond: see *Dfd^r*

almondex: see **amx**

almondex-55: see *lz^K*

almond eye: see **ale**

***alo: alopecia**

location: 1-38.3.
origin: Induced by 2-chloroethyl methanesulfonate.
discoverer: Fahmy, 1956.
references: 1958, DIS 32: 67.
phenotype: Abdominal hairs much reduced in number; pigmentation frequently lighter and patchy. Effect very pronounced in females reared at 25° but overlaps wild type in both sexes when reared at a low temperature. Viability and fertility good in males but reduced in females. RK3.

Alp: Abnormal leg pattern

location: 2-10.
references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.
phenotype: Defined by two dominant gain-of-function alleles. Heterozygotes viable with fusion of metatarsal and second tarsal segments. *Alp¹/Alp²* is pupal lethal with more extreme tarsal fusions.
alleles: Two X-ray induced alleles. *Alp¹* is associated with *T(2;3)XT1* and *Alp²* is associated with *T(2;4)X2*.
cytology: Placed in 23F6-24A1 based on breakpoint common to translocations.

alpha: see *tyr-1*

alpha methylropa hypersensitive: see **amd**

alr: alarless*location:** 3- (not located).**origin:** Spontaneous.**discoverer:** Steinberg, 40b.**references:** 1940, DIS 13: 51.**phenotype:** Outer postalar bristle always missing; posterior supra-alar missing in about 80% of the flies. Anterior scutellars, humerals, and notopleurals frequently duplicated. Never overlaps. Viability and fertility excellent. RK3.**ALS:** see *Acr96A***Altered abdomen:** see *Aa***altered disjunction:** see *ald***Alu: Alula****location:** 2-54.9 (Muller places *Alu* to the left of *pr*).**origin:** Spontaneous.**discoverer:** Bridges, 38a12.**references:** Curry, 1939, DIS 12: 45.**phenotype:** Heterozygote has alula fused to main wing; wings often bent, broader. May overlap wild type but intensified by cold and by heterozygous *ds* with buckling effect increased. Homozygote at 19° shows extreme buckling owing to rotation of wing and alula. Homozygote viable and resembles heterozygote. RK2.**alleles:** **Alu*^{56c} (CP627).***alw: arclike wing****location:** 2- (near *b*).**discoverer:** Sturtevant, 1948.**references:** 1948, DIS 22: 55.**phenotype:** Wings evenly bent downward at tips. Overlaps wild type. RK2.**am:** see *Dfd*^r**Ama:** see *ANTC***Ama:** see *RpII*^{C4}**Ama-1: α-amanatin resistant 1****location:** 3-19.**origin:** Recovered from natural populations from India, Malaysia, and Taiwan.**references:** Phillips, Willms, and Pitt, 1982, Can. J. Genet. Cytol. 24: 151-62.**phenotype:** Flies homozygous or heterozygous for *Ama-1* and *Ama-2* have LD₅₀ to α-amanatin 10-30 times that of wild type. Sensitivity of RNA polymerase II activity to α-amanatin same as wild type. *Ama-1* alone sufficient to confer resistance, but in three independent isolations both *Ama-1* and *Ama-2* present.**Ama-2****location:** 3-100.**origin:** Recovered from natural populations in India, Malaysia, and Taiwan.**references:** Phillips, Willms, and Pitt, 1982, Can. J. Genet. Cytol. 24: 151-62.**phenotype:** Same as for *Ama-1*.**Amalgam:** see *Ama* under *ANTC***Amanatin resistant:** see also***Amr* and *RpII*****amb: amber****location:** 1-6.8.**origin:** Induced by triethylenemelamine.**discoverer:** Fahmy, 1950.**references:** 1958, DIS 32: 67.**phenotype:** *amb* has pale yellow body color; bristles very thin and short; hairs less affected. Eyes slightly brighter red. Males sterile. Viability 10-50% wild type. RK2. *amb*² less extreme, males viable and fertile, females sterile.**alleles:** **amb*¹, *amb*² (Fahmy, 1958 and CP627). Also one allele each induced by triethylenemelamine, DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine, 2-chloroethyl methanesulfonate, and nitrogen mustard and two alleles induced by *p*-N,N-di-(2-chloroethyl)amino-phenylethylamine.**cytology:** Placed in 4C7-8 on the basis of its inclusion in *Df(1)bi-D3 = Df(1)4C5-6;4C7-8* but not *Df(1)rb41 = Df(1)4B6-C1;4C7-8* (Banga, Bloomquist, Brodberg, Pye, Larrivee, Mason, Boyd, and Pak, 1985, Chromosoma 93: 341-46).**amd: alpha methyl dopa hypersensitive****location:** 2-53.9 [.002 units (2.1 kb) to the left of *Ddc*].**synonym:** *amd*; *l(2)amd*^H, *l(2)37Bk*.**references:** Sparrow and Wright, 1974, Mol. Gen. Genet. 130: 127-41.

Wright, 1977, Am. Zool. 17: 707-21.

Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26.

Gilbert, Hirsh, and Wright, 1984, Genetics 106: 679-94.

Marsh and Wright, 1986, Genetics 112: 249-65.

Black, Pentz, and Wright, 1987, Mol. Gen. Genet. 209: 306-12.

Wright, 1987, Adv. Genet. 24: 127-222.

phenotype: *amd*⁺ flies die when reared on levels of alpha methyl dopa that are not lethal to wild type; resistance proportional to the number of *amd*⁺ loci present. Adult *amd*⁺ females fed alpha methyl dopa become sterile and lay eggs that cannot complete embryogenesis. Dopa decarboxylase levels normal. *amd* homozygotes lethal; lethal phase at times of larval hatching, larval molts, and pupariation; larval anal organ extruded and necrotic; pupal cuticle thin and friable. Appears to play role in cuticle formation. *amd*¹/*amd*⁶ complementing adults deficient for one or more unidentified catecholamines involved in the colorless sclerotization of cuticle.**alleles:** The first seven alleles selected as alpha methyl dopa hypersensitive in the heterozygous condition; the remainder recovered as recessive lethal mutations. Interallelic complementation observed, suggesting dimeric product.

allele	origin	discoverer	synonym	ref ^α	comments
<i>amd</i> ¹	EMS	Sparrow	<i>l(2)amd</i> ^{H1}	1, 2	
<i>amd</i> ²	EMS	Sparrow	<i>l(2)amd</i> ^{H7}	2	
<i>amd</i> ³	EMS	Sparrow	<i>l(2)amd</i> ^{H14}	2	
<i>amd</i> ⁴	EMS	Sparrow	<i>l(2)amd</i> ^{H45}	2	
<i>amd</i> ⁵	EMS	Sparrow	<i>l(2)amd</i> ^{H82}	2	
<i>amd</i> ⁶	EMS	Sparrow	<i>l(2)amd</i> ^{H89}	1, 2	
<i>amd</i> ⁷	EMS	Sparrow	<i>l(2)amd</i> ^{H121}	1, 2	
<i>amd</i> ⁸	EMS	Wright	<i>l(2)amd</i> ^{H8}	3, 4	
<i>amd</i> ⁹	EMS	Wright	<i>l(2)amd</i> ^{H60}	3, 4	
<i>amd</i> ¹⁰	EMS	Wright	<i>l(2)amd</i> ^{H122}	3, 4	
<i>amd</i> ¹¹	EMS	Wright	<i>l(2)amd</i> ^{H149}	3, 4	
<i>amd</i> ¹²	X ray	Hodgetts	<i>l(2)amd</i> ^{HX1}	3, 4	

allele	origin	discoverer	synonym	ref ^α	comments
<i>amd</i> ²¹	EMS	Wright	<i>l(2)203</i>	4	
<i>amd</i> ²²	EMS	Wright	<i>l(2)245</i>	4	
<i>amd</i> ²³	EMS	Wright	<i>l(2)258</i>	4	
<i>amd</i> ²⁴	EMS	Wright	<i>l(2)283</i>	4	
<i>amd</i> ²⁵	EMS + HCOH	Wright	<i>l(2)305</i>	4	
<i>amd</i> ²⁶	EMS + HCOH	Wright	<i>l(2)329</i>	4	
<i>amd</i> ²⁷	EMS + HCOH	Wright	<i>l(2)337</i>	4	
<i>amd</i> ²⁸	EMS + HCOH	Wright	<i>l(2)341</i>	4	
<i>amd</i> ²⁹	EMS + HCOH	Wright	<i>l(2)346</i>	4	
<i>amd</i> ³⁰	EMS	Wright	<i>l(2)602</i>	4	
<i>amd</i> ³¹	EMS	Wright	<i>l(2)616</i>	4	
<i>amd</i> ³²	EMS	Wright	<i>l(2)638</i>	4	
<i>amd</i> ³³	EMS	Wright	<i>l(2)640</i>	4	
<i>amd</i> ³⁴	EMS	Wright	<i>l(2)674</i>	4	
<i>amd</i> ³⁵	EMS	Steward	<i>l(2)RS1</i>	4	
<i>amd</i> ³⁶	EMS + γ ray	Wright	<i>l(2)7301</i>	4	
<i>amd</i> ³⁷	EMS + γ ray	Wright	<i>l(2)7401</i>	1, 4	750-bp insert, destroys <i>EcoRI</i> site at -5.6-kb
<i>amd</i> ³⁸	EMS + γ ray	Wright	<i>l(2)7413</i>	4	
<i>amd</i> ³⁹	EMS + γ ray	Wright	<i>l(2)7433</i>	4	
<i>amd</i> ⁴⁰	EMS + γ ray	Wright	<i>l(2)7439</i>	1, 4	<i>BglII</i> site at -4.8-kb altered; at intron splice acceptor
<i>amd</i> ⁴¹	EMS + γ ray	Wright	<i>l(2)7445</i>	4	
<i>amd</i> ⁴²	EMS	Schüpbach	<i>l(2)WK26</i>		
<i>amd</i> ⁴³	EMS + γ ray	Cecil	<i>l(2)C7</i>		
<i>amd</i> ⁴⁴	EMS + γ ray	Cecil	<i>l(2)AA3</i>		
<i>amd</i> ⁴⁵	EMS + γ ray	Cecil	<i>l(2)BB2</i>		
<i>amd</i> ⁴⁶	EMS + γ ray	Cecil	<i>l(2)BB3</i>		
<i>amd</i> ⁴⁷	EMS + γ ray	Cecil	<i>l(2)B1</i>		

^α 1 = Black, Pentz, and Wright, 1987, *Mol. Gen. Genet.* 209: 306-12; 2 = Sparrow and Wright, 1974, *Mol. Gen. Genet.* 130: 127-41; 3 = Wright, Bewley, and Sherald, 1976, *Genetics* 84: 287-310; 4 = Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, *Mol. Gen. Genet.* 188: 18-26.

cytology: Placed in 37B9-C1 based on its inclusion in *Df(2L)NST* but not *Df(2L)VA17*.

molecular biology: Located between coordinates -4.7 and -2.42, where 0 is the axis of symmetry of the *HpaI* site near the terminus of the *Ddc* coding sequence and positive values extend to the right. Genomic sequence contains a 483-bp intron near the 5' end; the usual upstream regulatory sequences identified as well (Marsh, Erfle, and Leeds, 1986, *Genetics* 114: 453-67). A 2.0-kb *amd* transcript first detectable early in embryogenesis; reaching maximum level at 12-16 hours; low levels observed in adults; concentrated in the nurse cells of stage 8-9 oocytes; smaller transcripts with sequence homology to the 2.0-kb transcript observed in third-instar larvae. *amd* and *Ddc* transcribed from opposite strands; two regions of extensive homology between *amd* and *Ddc* detected; intron sequences and positions not conserved, although homology across intron junctions is high (Eveleth and Marsh, 1986, *Genetics* 114: 469-83). Sequence predicts a 50,481 dalton polypeptide with a slight negative charge; 38% amino acid homology with dopa decarboxylase.

***Amdr*: Alpha methyl dopa resistant**

location: 3- (between *h* and *tn*).

origin: Induced by ethyl methanesulfonate.

references: Bishop and Sherald, 1981, *DIS* 56: 21.

phenotype: Based on two of 16 chromosomes selected for conferring resistance to α methyl dopa when heterozygous. LD₅₀ to L-α-methyl dopa for the two chromosomes is 0.325 mM for *Amdr*^{1/+} and 0.35 mM for *Amdr*^{2/+}, compared to 0.10 mM for wild type. Both

chromosomes are homozygous lethal, and *Amdr*^{1/Amdr}² is nearly lethal, allowing the inference of a single locus.

***amethyst*:** see *amy*

amiel

location: Autosomal.

origin: Spontaneous.

synonym: *Amiel*.

references: Rushton and Metcalfe, 1971, *DIS* 46: 61.

phenotype: Homozygous males court abnormally; wing vibrations and copulation attempts more vigorous than in wild type, but mutant males take longer to achieve copulation and have higher incidence of unsuccessful courtships. Homozygous females behave normally.

Amiel: see *amiel*

Aminoimidazole ribotide synthetase: see *ade2*

***amn*: amnesiac (J.C. Hall)**

location: 1-63.

discoverer: Sziber.

origin: Induced by ethyl methanesulfonate.

references: Quinn, Sziber, and Booker, 1979, *Nature (London)* 277: 212-14.

phenotype: Homozygous or hemizygous mutant flies can be conditioned to avoid odors associated with electric shocks, but effects of conditioning decay with a half life of 15 min compared to 60 min for normal. Memory decay biphasic; rapid for first hour and slow thereafter (Tully). Substitution of reward (1.0 M sucrose) for punishment (electric shock) lengthens memory span from one hour to six hours (Tempel, Bonini, Dawson, and Quinn, 1983, *Proc. Nat. Acad. Sci. USA* 80: 1482-86). Groups of *amn* flies exhibit apparently abnormal acquisition of learning in tests using visual cues (Folkers, 1982, *J. Insect. Physiol.* 28: 535-39); it appears that short-term memory is defective in the mutant (in shock-odor tests), with long-term memory being normal (Tully and Quinn, 1985, *J. Comp. Physiol.* 157: 263-77); in experiments involving "operant" conditioning, with heat as the aversive unconditioned stimulus, *amn* exhibits a small decrement in learning *per se* and subsequently has no detectable memory (Mariath, 1985, *J. Insect Physiol.* 31: 779-81). In tests of "simple learning," amnesiac individuals habituate to or are sensitized by sugar stimuli subnormally; the sensitization defect maps to the same proximal locus as that affecting associative conditioning (Duerr and Quinn, 1982, *Proc. Nat. Acad. Sci. USA* 79: 3646-50). The effects on courtship behavior or pre-exposure to fertilized females decay more rapidly in amnesiac than in normal males (Siegel and Hall, 1979, *Proc. Nat. Acad. Sci. USA* 76: 3430-34; Ackerman and Siegel, 1986, *J. Neurogenet.* 3: 111-23), but amnesiac males are defective in expressing after-effects of exposure to immature wild-type males when tested immediately after such exposure (Gailey, Jackson, and Siegel, 1982, *Genetics* 102: 771-82). Females defective in ability to be primed by courtship song (Kyriacou and Hall, 1984, *Nature (London)* 308: 62-65).

cytology: Placed in 19A1 based on its inclusion in *Df(1)mal12 = Df(1)19A1;20F* but not *Df(1)mal11 = Df(1)19A2-3;19E1* or *Df(1)mal3 = Df(1)19A2-3;20E-F* (Tully and Gergen, 1986, *J. Neurogenet.* 3: 33-47).

Amplification Control Element: see **ACE**

Amr: Amanatin resistant

location: 3- (not mapped).

origin: Induced by ethyl methanesulfonate.

references: Nishiura, 1981, *Biochem. Genet.* 19: 31-46.

phenotype: Heterozygotes survive 5 µg/ml α amanatin. RNA polymerase II activity in *Amr*-bearing flies resistant to α amanatin.

alleles: Three lines possibly containing different alleles designated *Amr*⁰¹⁰, *Amr*⁰¹⁸, and *Amr*¹⁰⁶.

other information: Genetic analysis lacking. If it is demonstrated that this locus codes for an RNA polymerase II subunit, it will be renamed *RpII* plus a subunit designation.

amx: almondex

location: 1-27.7 [to the left of *lz* (Green and Green, 1956, *Z. Indukt. Abstamm. Vererbungsl.* 87: 708-21)].

origin: X ray induced.

discoverer: Ball, 32k20.

phenotype: Eyes slightly reduced, narrower below. Trident pattern stronger than in *lz*. Maternal effect lethal. Studies by Shannon [1972, *Genetica* (The Hague) 43: 244-56] show that *amx* progeny and many *amx*/+ progeny of *amx* mothers are embryonic lethals. Ovaries and egg production of *amx* females normal. General disorganization of early embryo with *amx*/+ progeny of *amx* mothers less extreme than *amx* progeny (Shannon, 1973, *J. Exp. Zool.* 183: 383-400); *amx*/+ daughters show 0.2% survival; *amx*/*Dp(I;I)lz-2* show considerably higher survival (Campos-Ortega); Lethal embryos exhibit hypertrophy of central nervous system at the expense of epidermal tissue (Lehmann, Dietrich, Jiménez, and Campos-Ortega, 1981, *Wilhelm Roux's Arch. Dev. Biol.* 190: 226-29; Lehmann, Jiménez, Dietrich, and Campos-Ortega, 1983, *Wilhelm Roux's Arch. Dev. Biol.* 192: 62-74). Similarly peripheral nervous elements, the sensilla, exhibit increased numbers and abnormal morphology; cells diverted from epidermal to neurological pathway (Hartenstein and Campos-Ortega, 1986, *Wilhelm Roux's Arch. Dev. Biol.* 195: 210-21). Embryonic phenotype locally rescuable by injections of ooplasm from wild-type or *pcx* ova during preblastoderm stages (Campos-Ortega, La Bonne and Mahowald, 1985, *Dev. Biol.* 110: 264-67). *lzl**amx* is wild type. Mosaics in *amx*/+ daughters of ±/± or *amx*/+ females show that ventral tissues are sensitive to reduced *amx*⁺ activity; no clones of *amx* tissue found in cuticle of *amx*/+ daughters of *amx* mothers (Germeraad and Disano, 1984, *Genetics* 107: s36). RK2.

cytology: Located in 8D (region 8D4 through 8E2) by Green and Green (1956).

*amx*⁵⁵: see *lz*^K

***amy: amethyst**

location: 2- (not located).

discoverer: Bridges.

references: Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 218.

phenotype: Transparent, light-purplish eye color. RK3.

Amy: Amylase

location: 2-77.9 (based on 5039 *c-wt* recombinants).

origin: Polymorphic locus.

discoverer: Kikkawa, 1957.

references: Kikkawa and Abe, 1960, *Annotationes Zool. Jpn.* 33: 14-23.

Kikkawa, 1960, *Jpn. J. Genet.* 35: 382-87.

Kikkawa and Ogita, 1962, *Jpn. J. Genet.* 37: 394-95.

Kikkawa, 1963, *DIS* 37: 94.

Bahn, 1967, *Hereditas* 58: 1-12.

1964, *Jpn. J. Genet.* 39: 401-11.

Doane, 1969, *J. Exp. Zool.* 171: 321-42.

1969, *Problems in Biology: RNA in development* (Hanly, ed.). U. of Utah Press, Salt Lake City, pp. 73-109 (fig.).

Hickey and Benkel, 1986, *CRC Crit. Rev. Biotech.* (fig.).

phenotype: The structural gene for α-amylase [AMY (EC 3.2.1.1)]. A monomeric protein based on failure to form hybrid enzyme molecules of intermediate mobility in heterozygotes for alleles coding for electrophoretic variants. Activity mainly in midgut and hemolymph with smaller amounts in other tissues; activity found in anterior or posterior, or both, but not middle, region of midgut; three spatial patterns of adult posterior midgut activity encountered on standard medium; controlled by the trans-regulatory effect of *map* (2-80) (Abraham and Doane, 1978, *Proc. Nat. Acad. Sci. USA* 75: 4446-50); adult anterior midgut activity under regulation of another separable regulatory locus (Doane, 1980, *DIS* 55: 36-39). Larval midgut activity affected by closely linked *cis*-acting regulatory elements (Klarenberg, Kisser, Willemse, and Scharloo, 1986, *Genetics* 114: 1131-45). Amylase activity is glucose repressible (Hickey and Benkel, 1982, *Biochem. Genet.* 20: 1117-29); the degree of repression can be greater than one hundred fold in larvae and occurs at a pretranslational, probably transcriptional, level of regulation (Benkel and Hickey, 1985, *Genetics* 110: S25; 1986, *Genetics* 114: 137-44, 943-54; 1987, *Proc. Nat. Acad. Sci. USA* 84: 1337-39).

alleles: Eight electrophoretic variants of α-amylase have been recorded; they are numbered, in order of decreasing rates of migration toward the anode, from -1 through +7 (Doane, Treat-Clemons, Gemmill, Levy, Hawley, Buchenberg, and Paigen, 1983, *Isozymes: Curr. Top. Biol. Med. Res.* 9: 63-70). Enzymes with mobilities 2 and 3 exist in forms with different heat sensitivities: *Amy*¹ the most frequent allele, may be expressed at three different activity levels in different strains, 1a, 1b, and 1c in which 1a has twice the activity of 1b and 1b has twice the activity of 1c; purified α-amylases from 1a and 1c strains have identical specific activities (Treat-Clemons and Doane, 1982, *Isozyme Bull.* 15: 90-91); enzyme levels here are apparently under the control of closely linked transacting regulatory elements (Hickey, 1981, *Biochem. Genet.* 19: 783-96). A chromosome may express none, one, or two of these forms. Bahn recovered one *Amy*^{1,3} and two *Amy*² recombinants from *Amy*¹/*Amy*^{2,3} heterozygotes and one *Amy*^{4,3} and two *Amy*^{2,6} recombinants from *Amy*^{4,6}/*Amy*^{2,3} heterozygotes. From these observations it was concluded that the *Amy* locus is duplicated and the two copies are separated by 0.008 cm; furthermore, flanking marker segregations indicated that determinants of forms 1,2 (thermostable), and 4 are to the left of those for 3 (thermostable) and 6. Conservation of res-

triction endocuclease sites in DNA from Bahn's *Amy*^{2,3} in comparison with *Amy*^{1,3} (from Canton-S) and *Amy*^{1,6} (from Suyama, Japan) indicates that the determinant of form 1 is to the left of those of forms 3 and 6 in the latter two chromosomes (Gemmill, Schwartz, and Doane, 1986, Nucl. Acids Res. 14: 5337-52). *Amy*¹ monomorphic allele in Oregon-R has been shown to be *Amy*^{1,1} (Hawley, 1989, Ph.D thesis, Arizona State University).

cytology: Placed in 54A based on *in situ* hybridization (Gemmill, Levy, and Doane, 1985, Genetics 110: 299-312).

Amy-d: Amylase distal

The distal member of the *Amy* repeat. Electrophoretic alleles include *Amy-d*³ (thermostable). *Amy-d*⁶ and likely *Amy-d*² (thermolabile); some chromosomes apparently lack *Amy-d* activity.

Amy-p: Amylase proximal

The proximal member of the *Amy* repeat. Electrophoretic alleles include *Amy-p*¹, *Amy-p*² (thermostable), *Amy-p*⁴, probably *Amy-p*³ (thermolabile), and *Amy-p*⁵; a null allele also exists. Allelic compositions of various strains are tabulated in the accompanying table.

strain	source	Amy-p	Amy-d	ref ^α
Amy 1a		<i>Amy-p</i> ^{1a}	inactive?	
Amy 1b		<i>Amy-p</i> ^{1b}	inactive?	
Amy 1c		<i>Amy-p</i> ^{1c}	inactive?	
Amy 1.2 β	<i>adp</i> ^{fs} , Kaduna	<i>Amy-p</i> ¹	<i>Amy-d</i> ²	2, 3, 4, 5
Amy 1.3	Canton-S	<i>Amy-p</i> ¹	<i>Amy-d</i> ³	3, 4, 5, 6
Amy 1.4		<i>Amy-p</i> ¹	<i>Amy-d</i> ⁴	
Amy 1.6	Suyama	<i>Amy-p</i> ¹	<i>Amy-d</i> ⁶	3, 4, 5
Amy 2		<i>Amy-p</i> ²	inactive?	
Amy 2.3	Copenhagen	<i>Amy-p</i> ²	<i>Amy-d</i> ³	1, 3, 5
Amy 2.6		<i>Amy-p</i> ²	<i>Amy-d</i> ⁶	
Amy 3.6 β	Kyoto	<i>Amy-p</i> ³	<i>Amy-d</i> ⁶	3, 4
Amy 4.5		<i>Amy-p</i> ⁴	<i>Amy-d</i> ⁵	
Amy 4.6	<i>adp</i> ⁶⁰ , Kaduna	<i>Amy-p</i> ⁴	<i>Amy-d</i> ⁶	1, 3, 4
Amy 5	Africa	<i>Adp-p</i> ⁵	inactive?	8
Amy 5.6	Africa	<i>Adp-p</i> ⁵	<i>Adp-d</i> ⁶	8
Amy n	Texas	inactive	inactive	7

^α 1 = Bahn, 1967, Hereditas 58: 1-12; 2 = Doane, 1967, J. Exp. Zool. 164: 363-78; 3 = Doane, 1969, Problems in Biology. RNA in Development (W. E. Hanley, ed.). Univ. Utah Press, Salt Lake City, pp. 73-109; 4 = Doane, 1969, J. Exp. Zool. 171: 321-42; 5 = Gemmill, Schwartz, and Doane, 1986, Nucl. Acids Res. 14: 5337-52; 6 = Levy, Gemmill, and Doane, 1985, Genetics 110: 313-24; 7 = Haj-Ahmed and Hickey, 1982, Nature 299: 350-52; 8 = Puijck and DeJong, 1972, DIS 49: 61.

^β Tentative assignments.

molecular biology: Clones homologous to mouse α-amylase gene isolated from Maniatis library (Doane, Treat-Clemons, Gemmill, Levy, Hawley, Buchberg, and Paigen, 1983, Curr. Top. Biol. Med. Res. 9: 63-90). λDm32 hybridizes to polytene region 53CD; no homologous mRNA detected; postulated to be pseudogene. λDm65 hybridizes to 54A1-B1 and is homologous to a 1450-1500 nucleotide transcript (Gemmill, Levy, and

Doane, 1985, Genetics 110: 299-312). 15kb insert in λDm65 contains reverse repeat by restriction mapping; subclones, each containing one of the repeated sequences, injected into *Xenopus* oocytes; one subclone capable of producing isozyme 1 of α-amylase; the other capable of producing isozyme 3; confirms duplicated nature of locus (Levy, Gemmill, and Doane, 1984, Isozyme Bull. 17; 1985, Genetics 110: 313-24). Directions of transcription of the two genes divergent. (Levy, 1985, Genetics 110: 137); separated by 4 kb. Seven strains exhibiting no, one, or two electrophoretic forms of α-amylase all carry the duplication as ascertained from restriction analysis. *Amy*^{1c} contains an approximately 10 kilobase insert some 10 kb proximal to *Amy-p*. 12% of chromosomes isolated from diverse natural populations contain large inserts in the vicinity of the *Amy* loci (Langley, Shrimpton, Yamazaki, Miyashita, Matsuo, and Aguadro, 1988, Genetics 119: 619-29). Two molecular inversions that could have arisen through interlocus exchange recorded; one had normal levels of amylase activity (Langley *et al.*) and the other was a null allele that produced reduced levels of mRNA and was insensitive to glucose repression (Hickey, Benkel, Abukashawa, and Haus, 1988, Biochem. Genet. 26: 757-68; Schwartz and Doane, 1989, Biochem. Genet. 27: 31-46). An amylase cDNA has been cloned and sequenced (Boer, and Hickey, 1986, Nucleic Acid Res. 14: 8399-8411). This sequence shows 57% identity to mouse amylase; the predicted amino-acid sequence indicates a 54.5-kd polypeptide of 493 residues, the 18 N-terminal ones of which are signal sequence; there is 55.4% amino-acid identity with mouse amylase. Upstream sequence contains a repeated motif also found in a negatively regulated mammalian gene (Hickey, Genest, and Benkel, 1987, Nucleic Acid Res. 15: 7184). Northern blots probed with this cDNA show that the glucose repression effect is at the level of amylase mRNA abundance.

*Amy*⁴: see *Amy*^{1,4}

Amy⁺: see *Amy*¹

Amy^{ad}: see *Amy*^{4,6}

Amy^s: see *Amy*^{2,6}

Amy^{wh}: see *Amy*^{1,4}

Amyloid protein precursor-like: see *App1*

an: ancon

location: 2-44 (34-54).

discoverer: Bridges, 30e3.

phenotype: Wings and legs somewhat short in *an*¹; *an*² (CP627) more extreme with gnarled legs, scraggly abdominal bristles, etched sclerites; eyes small and roughish. *an*¹/*an*² like *an*².

alleles: *an*¹ and *an*².

anarista: see *aa*

ancon: see *an*

And: Andante (J.C. Hall)

location: 1-36.2

origin: Induced by ethyl methanesulfonate.

discoverer: Konopka, R. Smith and Orr, 1976.

references: Jackson, Gailey, and Siegel, 1983, J. Comp.

Physiol. 151: 545-52.

phenotype: The normal free-running 24 hr periods of the circadian rhythms of eclosion and adult locomotor activity (in constant conditions) are lengthened by 1.5-2 hr/cycle; *And*/*+* heterozygotes have a period phenotype intermediate between wild-type and mutant homozygotes (Konopka, R. Smith and Orr). The phase-response curves (PRCs) for eclosion and activity rhythms, indicating light-induced phase shifts, show a similar degree of lengthening as seen in free-running periodicities. *And* rhythms are highly temperature-compensated, as are those of wild-type (Konopka *et al.*). *And* males are defective in after effects on courtship behavior that are usually induced by prior exposure to mated females or very young males (Jackson *et al.*, 1983).

cytology: Placed in 10E1-2;10F1. The *And* homozygote-like activity rhythm phenotype is uncovered by *Df(1)KA6 = Df(1)10E1;11A7-8* and *Df(1)KA7 = Df(1)10A9;10F6-7*, but heterozygotes involving *And* and *Df(1)N105 = Df(1)10F7;11D1*, *Df(1)RA47 = Df(1)10F1;19F9-10*, or *Df(1)m²⁵⁹⁻⁴ = Df(1)10C2-3;10E1-2* are like *And*/*+* (Konopka *et al.*). The two *And*-uncovering *Df*'s just noted over wild type give normal periods. An anomaly then, is that *Df(1)HA85 = Df(1)10C1-2;11A1-2*, which uncovers *And* as it should (see above), leads to significantly longer-than-normal periods when over wild type (Konopka *et al.*).

other information: *And* lengthens in an additive manner, the periodicities associated with certain other rhythm mutants, *i.e.*, those which by themselves cause shorter- or longer-than-normal locomotor activity periods (*viz.*, *per^S*, *per^{L1}*, *per^{L2}*, *Clk*). *And*, on its isolation, was associated with a *dy* wing phenotype, and the rhythm abnormality maps to the *dy-m* locus (see "cytology"); but *dy* and *m^D* have normal activity rhythm periods, and *And* over either of these two visibles gives the same periods as seen in *And*/*+* (Konopka *et al.*); however, of four gamma-ray induced *dy*'s - *dyⁿ¹*, *dyⁿ²*, *dyⁿ³*, and *dyⁿ⁴*, all but *dyⁿ²* are *And*-like in their locomotor activity rhythms (Jackson, Newby, and DiBartolomeis, 1989, *Neurosci. Abstr.* 15: 461).

ang: angle wing

location: 2-10.5.

origin: Spontaneous.

discoverer: Mittler and Goldberg, 48i16.

references: Mittler, 1950, DIS 24: 61.

phenotype: Wings held up from dorsal surfaces and extended outward 15-90° from the mid-dorsal line. Longitudinal dorsal median muscles 5 and 6 fused (Goldberg, 1954, Ph.D. Thesis, Ill. Inst. Technol.). No increase in expressivity with temperature. Does not overlap wild type. RK2.

ang: see *ano*

angle wing: see *ang*

angle wing: see *agl*

angle winglike: see *agl*

***ano: anomogenitals**

location: 1-35.7.

origin: Induced by triethylenemelamine.

discoverer: Fahmy, 1952.

synonym: Originally symbolized *ang*, but this symbol preoccupied.

references: 1958, DIS 32: 67.

phenotype: Many bristles on head and thorax either reduced in size or absent. Thoracic and abdominal hairs appreciably fewer. External male genitalia invariably abnormal, sometimes completely absent. Melanized exudate frequently present in furrow between mesonotum and scutellum near anterior scutellar bristles. Males sterile; viability less than 10% wild type. RK3.

ant: antennaless

location: 2- (not located).

origin: Spontaneous.

discoverer: Gordon, 1936.

references: 1941, DIS 14: 39.

1941, *Proc. Intern. Congr. Genet.*, 7th. p. 131.

Gordon and Sang, 1941, *Proc. Roy. Soc. (London)*, Ser. B 130: 151-84 (fig.).

Vogt, 1947, *Biol. Zentr.* 66: 388-95 (fig.).

phenotype: Antennae missing on one or both sides. Expression affected by residual genotype, nutritional environment, and temperature. Time of action about 70 hours after hatching [Begg and Sang, 1945, *J. Exp. Biol.* 21: 1-4 (fig.)]. Used in experiments to locate chemoreceptors [Begg and Hogben, 1946, *Proc. Roy. Soc. (London)*, Ser. B 133: 1-19] and in studies of mating behavior (Begg and Packman, 1951, *Nature* 168: 953). RK3.

ANTC: The Antennapedia Complex (T.C. Kaufman)

location: 3-47.5.

references: Kaufman, Lewis, and Wakimoto, 1980, *Genetics* 94: 115-33.

Lewis, Wakimoto, Denell, and Kaufman, 1980, *Genetics* 95: 393-97.

Denell, Hummels, Wakimoto, and Kaufman, 1981, *Dev. Biol.* 81: 43-50.

Kaufman, 1983, *Time, Space, and Pattern in Embryonic Development*, Alan R. Liss, New York, pp. 365-83.

Kaufman and Abbott, 1984, *Molecular Aspects of Early Development*, Plenum, New York, pp. 189-218.

Wakimoto, Turner, and Kaufman, 1984, *Dev. Biol.* 102: 147-72.

Regulski, Harding, Kostriken, Karch, Levin, and McGinnis, 1985, *Cell* 43: 71-80.

Gehring and Hiromi, 1986, *Ann. Rev. Genet.* 20: 147-73.

Akam, 1987, *Development* 101: 1-22.

Fechtel, Natzle, Brown, and Fristrom, 1988, *Genes* 120: 465-74.

Mahaffey and Kaufman, 1988, *Developmental Genetics of Higher Organisms: A Primer in Developmental Biology*, Macmillan, New York, pp. 329-59.

Kaufman, Seeger, and Olsen, 1990, *Genetic Regulatory Hierarchies in Development*, Academic Press, New York, pp. 309-62.

phenotype: The existence of the homeotic *ANTC* was originally proposed based on the tight linkage of the proboscipedia (*pb*). Sex combs reduced (*Scr*) and Antennapedia (*Antp*) loci. All were found to reside in a set of three doublet bands at the proximal end of section 84 (84A1,2-3,4 and 84B1,2) in the right arm of polytene chromosome 3. Subsequent genetic analyses have shown that two other homeotic loci labial (*lab*) and Deformed

(*Dfd*) are also members of the complex. The homeotic loci of the *ANTC* are involved in the specification of segmental identity in the posterior head (gnathocephalic) and anterior thoracic regions of the embryo and adult. Moreover the linear order of the homeotic loci in the complex *lab*, *pb*, *Dfd*, *Scr*, and *Antp* corresponds to the anterior posterior order of altered segments (intercalary, mandibular, maxillary, labial, and thoracic) found in animals bearing mutations in each of the resident loci. Specifically *Antp* transforms posterior T1, all of T2, and the anterior of T3, *Scr* transforms T1 and labial, *Dfd* affects the maxillary and mandibular lobes, *pb* affects the derivatives of the maxillary and labial segments and finally *lab* functions in the intercalary segment. Taken together the results of mutational analyses indicate that members of the complex are necessary to repress head development in the thorax (*Antp*) and elicit normal segmental identity in the anterior thorax (*Scr*) and posterior head (*Scr*, *Dfd*, *pb*, and *lab*). The *ANTC* is distinguished from the bithorax complex not only by virtue of the domain of action of its homeotic loci (anterior vs. posterior) but also by the residence of loci which are not homeotic in nature. Two of these fushi tarazu (*ftz*) and zerknüllt (*zen*) have been shown to affect segment enumeration (*ftz*) and the formation of dorsal structures (*zen*) in the early embryo. A third nonhomeotic gene is bicoid (*bcd*). Mutations in this locus result in female sterility and maternal effect lethality. Eggs laid by *bcd* females fail to develop normal anterior ends and instead produce mirror image duplications of structures normally produced at the posterior terminus of the embryo. In addition to these genetically defined loci there are several other "genes" which have been found in the *ANTC* by molecular mapping. The first of these is a cluster of cuticle-protein-related genes which map between the *lab* and *pb* loci. Eight small (*ca.* 1 kb) transcription units make up the cluster and all have sequence similarities to known cuticle protein genes. These "genes" are also apparently regulated by ecdysone in imaginal discs. Deletion of the entire cluster has no apparent effect on the development or cuticle morphology of embryos, larvae, or adults. The second molecularly identified "gene" is the Amalgam (*Ama*) transcription unit. The encoded protein places the gene in the immunoglobulin superfamily and like the cuticle cluster the locus can be deleted from the genome with no discernible effect on the organism.

cytology: Placed in the 84A1-B2 interval by the inclusion of the complex in *Df(3R)Scr* and the location of breakpoint associated inactivations of the *lab*, *pb*, *Dfd*, *Scr*, *ftz*, and *Antp* loci.

molecular biology: The entire complex has been cloned and has been shown to cover 335 kb of genomic DNA. The most distal transcription unit is *Antp* which covers the distal-most 100 kb of the complex and is made up of eight exons. Proximally the next 75 kb contain the *Scr* and *ftz* loci. The distal 50 kb of this interval house sequences necessary for *Scr* expression as well as the two exons of the *ftz* locus and its associated regulatory elements. The proximal 25-kb contain the three identified exons of the *Scr* transcription unit. The five exons of the *Dfd* gene are found in the central portion of the next-most-proximal 55-kb interval. The *Dfd* transcription unit covers only 11 kb of this region and it is likely that one or both of the 20-kb intervals flanking the gene are the

location of *cis*-acting regulatory elements for the locus. The next 25-kb interval contains four of the nonhomeotic transcription units which help distinguish the *ANTC* and *BXC*. The distal most is *Ama*, next *bcd*, and finally *zen* and *z2*. The *z2*, *zen*, and *Ama* transcription units are all relatively small (1-2 kb) and comprise two exons each. The *bcd* gene is somewhat larger (3.6 kb) and is made up of four exons. Immediately proximal to the *z2* transcription unit (*ca.* 1 kb from its 3' end) is the 5' end of *pb*; the latter gene extends over the next 35 kb of genomic DNA and contains nine exons. The next 25 kb of the complex contain the cuticle cluster and its eight identified transcription units. The final 25 kb are the residence of the *lab* gene which is made up of three exons.

Despite the nonhomeotic nature of three of the smaller transcription units (*zen*, *bcd*, and *ftz*) resident in the complex, these loci are tied to the larger homeotic genes of the region by the nature of their protein products. All five of the large homeotics (*Antp*, *Scr*, *Dfd*, *pb*, and *lab*) and the three small genes have a homeobox motif and their protein products are found in the nuclei of the cells in which they are expressed. Thus eight of the genes in the *ANTC* encode regulatory proteins which act as transcription factors. The *z2* gene also contains a homeobox; however, the biological significance of the gene is not known as deletions of this transcription unit have no discernible effect. The cuticle-like genes and *Ama* do not contain a homeobox.

The reasons for the clustering of these developmentally significant loci of similar function is not known. The existence of common or overlapping regulatory elements, the need to insulate regulatory sequences from position effect and the possibility of higher order chromatin structures for proper expression have all been proposed. Whatever the reason, the homeotic complex structure has a long evolutionary standing. Similar clusters are found in vertebrates, an observation consistent with a very early origin of these genes, likely predating the separation of protostomes and deuterostomes.

Ama: Amalgam

location: 1-{47.5}.

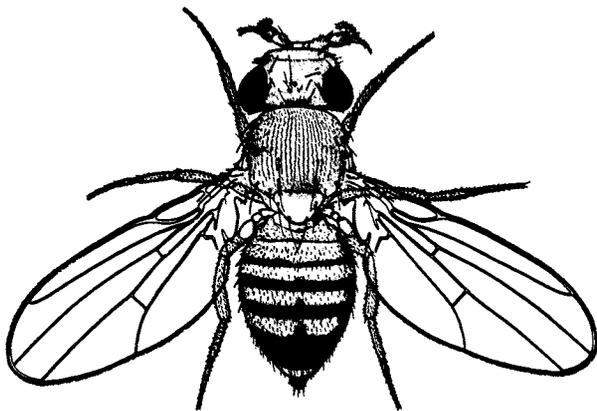
origin: Isolated as an unidentified third transcription unit in a 50 kb region known to harbor *bcd* and *zen*.

references: Seeger, Haffley, and Kaufman, 1988, Cell 55: 589-600.

phenotype: Antibody staining first detects Amalgam in the mesoderm during gastrulation; as neuroblasts delaminate from the ectoderm staining appears in a row of mesectodermal cells along the ventral midline of the extended germ band. Amalgam appears in the first neurons generated from the ganglion-mother cells, but not in the neuroblast precursors of these cells. Neuronal accumulation of *Ama* gene product increases during CNS development, but appears to be confined to the CNS and initially does not extend to axons exiting the CNS in segmental, intersegmental, or peripheral nerves; with time three rows of PNS-associated cells accumulate *Ama* protein; staining heavy around spiracle sensory organ and several cephalic sensory structures. Simultaneously there is a complicated temporal and spatial sequence of staining of mesodermal derivatives. Embryonic phenotype of deletion of *Ama* attributable to simultaneous deletion of *zen*; no effect of *Ama*⁻ detectable.

cytology: Placed in 84A1 based on its juxtaposition with *bcd*.

molecular biology: Sequence of a putatively full-length cDNA clone compared with that of the corresponding genomic region reveals a gene with a 316 bp intron in the 5' untranslated region. Transcription from left to right. Conceptual amino-acid sequence indicates a protein product of 333 amino acids, the first 23 of which have the characteristics of a signal sequence. The sequence contains three internal repeats of approximately 100 amino acids each that exhibit homology to the immunoglobulin or Ig domain of vertebrates; each contains two widely spaced cysteine residues and the show 22-36% identity to one another with greatest identity found around the cysteines. There are two potential N-linked glycosylation sites in the first domain and one in the third. In addition there is a potential C-terminal membrane-attachment domain of amino acids. Comparison with sequences in the data base indicate that the Amalgam sequence is closest to members of the Ig class of proteins that act as cell-adhesion molecules.



Antp^{LC}: Antennapedia of Le Calvez

From Le Calvez, 1948, Bull. Biol. France Belg. 82: 97-113.

Antp: Antennapedia

location: 3-47.5.

references: Denell, 1973, Genetics 75: 279-97.

Struhl, 1981, Nature 292: 635-38.

Garber, Kuroiwa, and Gehring, 1983, EMBO J. 2: 2027-34.

Hafen, Levine, Garber, and Gehring, 1983, EMBO J. 2: 617-23.

Hazelrigg and Kaufman, 1983, Genetics 105: 581-600.

Levine, Hafen, Garber, and Gehring, 1983, EMBO J. 2: 2037-46.

Scott, Weiner, Polisky, Hazelrigg, Pirrotta, Scalenghe, and Kaufman, 1983, Cell 35: 763-76.

Hafen, Levine, and Gehring, 1984, Nature 307: 287-89.

Abbott and Kaufman, 1986, Genetics 114: 919-42.

Carroll, Laymon, McCutcheon, Riley, and Scott, 1986, Cell 47: 113-22.

Frischer, Hagen, and Garber, 1986, Cell 47: 1017-23.

Laughon, Boulet, Bermingham, Laymon, and Scott, 1986, Mol. Cell Biol. 6: 4676-89.

Martinez-Arias, 1986, EMBO J. 5: 135-41.

Schneuwly, Kuroiwa, Baumgartner, and Gehring, 1986, EMBO J. 5: 733-39.

Wirz, Fessler, and Gehring, 1986, EMBO J. 5: 3327-34.

Jorgensen and Garber, 1987, Genes Dev. 1: 544-55.

Schneuwly, Klemenz, and Gehring, 1987, Nature 325: 816-18.

Schneuwly, Klemenz, and Gehring, 1987, EMBO J. 6: 201-06.

Bermingham and Scott, 1988, EMBO J. 7: 3211-22.

Boulet and Scott, 1988, Genes Dev. 2: 1600-14.

Gibson and Gehring, 1988, Development 102: 657-75.

Muller, Affolter, Leupin, Otting, Wuthrich, and Gehring, 1988, EMBO J. 7: 4299-304.

Otting, Gottfried, Qian, Muller, Affolter, Gehring, and Wuthrich, 1988, EMBO J. 7: 4305-09.

Perkins, Daly, and Tjian, 1988, Genes Dev. 2: 1615-26.

Stroeher, Gaiser, and Garber, 1988, Mol. Cell Biol. 8: 4667-75.

Bermingham, Martinez-Arias, Pettitt, and Scott, 1990, Development 109: 553-66.

phenotype: Null loss-of-function alleles result in embryonic lethality. Animals succumb at the end of embryogenesis and show homeotic transformations in the larval cuticle of the first, second, and third thoracic segments. Specifically the cuticle derived from parasegments 4 and 5 are transformed to a more anterior identity such that the posterior of the first thorax produces fragments of mouth hook material on its dorsal surface presumably owing to a new posterior labial identity, whereas the anterior of the second thorax resembles the first thorax. The anterior of the third thoracic segment is weakly transformed toward a T1-like identity. The posterior of T2 is presumably T1 like as there are no gnathal structures seen in this compartment. There are also partial loss-of-function mutations which allow survival into the larval, pupal, and adult stages. Those that allow adult survival produce animals in which the anterior of the dorsal mesothorax shows a transformation to prothorax. There are no other apparent defects associated with these lesions. Those "leaky" mutants which die in the pupal and larval stages show similar parasegmental transformations as the null alleles, except that only the parasegment 4 to 3 homeosis is generally apparent. Animals which survive to the pupal stage fail to evert their anterior spiracles resulting in a blunt appearance of the anterior pupa. This same phenotype is seen in genotypes which survive to the adult stage. These partial mutants in many cases are associated with chromosome rearrangements notably deletions which approach the locus from its distal end. Moreover these mutations have been shown to complement fully other seemingly null mutations. Subsequent molecular analyses have shown that these results are accounted for by the presence of two promoters, one, P1, distal to the other, P2. The partial mutants affect the ability of the P1 promoter to initiate transcription, while the complementing lesions inactivate P2. Null mutants affect the transcription unit and protein encoding portion of the gene which is common to both promoters (see below).

X-ray induced somatic clones of *Antp*⁻ cells demonstrate that the locus is required in the adult for the proper development of the dorsal pro and mesothorax, and legs. The former is reduced in size presumably reflecting an anteriorward transformation while the latter are

transformed to antennae. Thus *Antp*⁺ function is required in the embryo and adult in parasegments 4 and 5 to prevent more anterior segmental identities, specifically those normally found in the anterior thorax and head.

The *Antp* locus was initially recognized by virtue of several striking dominant gain-of-function alleles. Thirteen of these transform the antenna of the adult into a mesothoracic leg (*Antp*⁴⁹, *Antp*^B, *Antp*^{Yu}, *Antp*^{Pw}, *Antp*^{LC}, *Antp*^R, *Antp*^{Wu}, *Antp*⁵⁰, *Antp*^{RM}, *Antp*^{73b}, *Antp*^{CB}, *Antp*^{72j}, and *Antp*^{Ns}). Three of these also have effects on the orbit of the eye and the vibrissal region of the ventral head (*Antp*^{RM}, *Antp*^{72j}, and *Antp*^{Ns}). There are also two dominant alleles (*Antp*^{Ctx} and *Antp*^W) which transform portions of the head capsule (dorsal and posterior) and the eye to a dorsal mesothoracic identity. In some cases this includes the production of wing tissue in the eye. Finally, a unique dominant *Antp*^{Hu} produces bristles on the normally bald propleurae just ventral to the mesothoracic spiracle. This latter phenotype has been interpreted as the production of sternopleural bristles on the propleurae, and thus a T1 to T2 transformation. With the exception of *Antp*^{Ns} and *Antp*^{72j} all these dominant lesions are associated with recessive lethality and gross chromosome rearrangements. All the breakpoints fall in the interval between the distal and proximal promoters. The dominant gain-of-function phenotype results from the misregulation of the P2 promoter by position affect or by the production of novel transcripts initiated in the newly juxtaposed sequences and spliced to the downstream *Antp* coding sequences. Both events result in the ectopic accumulation of the *Antp* protein product in the eye-antennal disc where the normal head repressive function of the gene causes the observed alteration. The recessive lethality associated with these lesions falls into the partially deficient category mentioned above. That is, these lesions show complementation with the P2 specific (*Antp*¹ and *Antp*²³) mutations and in general show only strong parasegment 4 → parasegment 3 transformations. However, there is a gradient of this affect among the breakpoints. Those closest to P1 and furthest from P2 are the weakest, whereas those close to P2 show the strongest phenotype and earlier lethal phase. This same result is obtained with breakpoint mutations in the P2-to-P1 interval which are not associated with a dominant phenotype. Therefore this interval likely contains sequences necessary for the proper regulation of the P2 promoter.

Three of the dominant gain-of-function lesions (*Antp*^{Hu}, *Antp*^{73b}, and *Antp*^{Ns}) have been reverted. The revertants are either complete nulls, thus obviating the potential for ectopic expression, or are partial mutants; the latter mutants likely remove the potential for ectopic expression by altering the juxtaposed sequences required for abnormal P2 activity.

Both *in situ* hybridization and immunostaining have been used to determine the spatio-temporal pattern of *Antp* expression. Both the protein and RNA are strongly accumulated in the ventral nerve cord and more weakly in the epidermis and mesoderm of the embryo. Protein and RNA are first detected during cellular blastoderm in a band of cells in the parasegment 4-6 anlagen. This initial spatial pattern is further elaborated at full germ-band extension. In the ectoderm *Antp* products are found starting in the region of the first thoracic segment (parasegments 3 and 4) and extending posteriorly to the level of

the seventh abdominal segment. In the mesoderm, they are found in parasegments 4-6. During germ band shortening the gene products are accumulated in the CNS from parasegment 4 (posterior T1) through to the posterior end of the ventral nerve cord. In the integument transcripts and protein are mainly restricted to the parasegments 4-5 interval although some weak expression can be seen in parasegments 3. As embryogenesis proceeds, the posterior CNS expression diminishes but is still detectable at the end of embryogenesis. The major accumulation in the CNS at this time is in the neuromeres of parasegments 4 and 5. The mesodermal expression is found in the anterior midgut; quenching of *Antp* expression is found in the posterior portion of the anterior midgut and has been shown to be dependent on the expression of *Ubx*. In later stages *Antp* protein can be detected in the leg, dorsal prothoracic, and wing discs.

alleles:

allele	origin	discoverer	synonym	type	cytology
<i>Antp</i> ¹	X ray	Abbott	<i>Antp</i> ^{a58}	hypomorphic	normal
<i>Antp</i> ²	X ray	Abbott	<i>Antp</i> ^{a60}	hypomorphic	<i>Df(3R)84B2;84D3</i>
<i>Antp</i> ³	X ray	Abbott	<i>Antp</i> ^{a62}	null	normal
<i>Antp</i> ⁴	X ray	Abbott	<i>Antp</i> ^{a71}	null	normal
<i>Antp</i> ⁵	X ray	Abbott	<i>Antp</i> ^{a74}	null	<i>In(3R)84B2;87C</i>
<i>Antp</i> ⁶	X ray	Abbott	<i>Antp</i> ^{a75}	hypomorphic	<i>Df(3R)84B2-C6</i>
<i>Antp</i> ⁷	EMS	Denell	<i>Antp</i> ^{d7}	null	normal
<i>Antp</i> ⁸	DEB	Stephenson	<i>Antp</i> ^{e8}	null	normal
<i>Antp</i> ⁹	EMS	Fornili	<i>Antp</i> ^{f9}	hypomorphic	normal
<i>Antp</i> ¹⁰	EMS	Fornili	<i>Antp</i> ^{f22}	null	normal
<i>Antp</i> ¹¹	EMS	Fornili	<i>Antp</i> ^{f36}	null	normal
<i>Antp</i> ¹²	EMS	Fornili	<i>Antp</i> ^{f40}	null	normal
<i>Antp</i> ¹³	EMS	Fornili	<i>Antp</i> ^{f69}	null	normal
<i>Antp</i> ¹⁴	X ray	Kaufman	<i>Antp</i> ^{k4}	null	<i>T(2;3)36C-D;</i> <i>84B1-2</i> <i>+ In(3LR)62B;</i> <i>98F</i> normal
<i>Antp</i> ¹⁵	EMS	Kaufman	<i>Antp</i> ^{k5}	null	normal
<i>Antp</i> ¹⁶	EMS	Matthews	<i>Antp</i> ^{kml}	null	?
<i>Antp</i> ¹⁷	X ray	Lopez	<i>Antp</i> ^{l1}	hypomorphic	<i>T(2;3)25F;84B1-2</i>
<i>Antp</i> ¹⁸	X ray	Lopez	<i>Antp</i> ^{l2}	null	normal
<i>Antp</i> ¹⁹	X ray	Pultz	<i>Antp</i> ^{l4}	null	normal
<i>Antp</i> ²⁰	EMS	R. Lewis	<i>Antp</i> ^{r4}	hypomorphic	normal
<i>Antp</i> ²¹	EMS	R. Lewis	<i>Antp</i> ^{r10}	null	normal
<i>Antp</i> ²²	EMS	R. Lewis	<i>Antp</i> ^{r17}	null	normal
<i>Antp</i> ²³	X ray	Scott	<i>Antp</i> ^{s1}	hypomorphic	normal
<i>Antp</i> ²⁴	X ray	Scott	<i>Antp</i> ^{s2}	hypomorphic	<i>In(3R)80;84B1-2</i>
<i>Antp</i> ²⁵	EMS	Wakimoto	<i>Antp</i> ^{w10}	null	normal
<i>Antp</i> ²⁶	EMS	Wakimoto	<i>Antp</i> ^{w24}	null	normal
<i>Antp</i> ⁴⁹	X ray	Piternick	<i>Antp</i> ⁴⁷⁰³	weak	lesion in 84B1-2
<i>Antp</i> ⁵⁰	X ray	Piternick	<i>Antp</i> ⁴⁷¹⁵	strong	extra band distal to 84B1-2 = <i>Antp</i> ⁴⁹ ? = <i>Antp</i> ⁵⁰ ?
<i>Antp</i> ⁵⁹	X ray	Piternick		weak	
<i>Antp</i> ⁶⁰	X ray	Piternick		weak	
<i>Antp</i> ^{72j}	spont	Baker		viable	normal
<i>Antp</i> ^{73b}	spont	Green		strong	<i>In(3R)84B1-2</i>
<i>Antp</i> ^{73b-rv1}	spont	Green	<i>Antp</i> ^{73b}	revertant	
<i>Antp</i> ^{73b-rv2}	spont	Green	<i>Antp</i> ^{73b}	revertant	
<i>Antp</i> ^{73b-rv5}	X ray	Hazelrigg	<i>Antp</i> ^{73b}	revertant	<i>T(2;3)57B6-8;</i> <i>84B1-2;97B3</i>
<i>Antp</i> ^{73b-rv7}	X ray	Hazelrigg	<i>Antp</i> ^{73b}	revertant	<i>T(2;3)40;84B1-2</i>
<i>Antp</i> ^{73b-rv8}	X ray	Hazelrigg	<i>Antp</i> ^{73b}	revertant	<i>Dp(3;3)84D5-8;</i> <i>85F5-8</i>
<i>Antp</i> ^{73b-rv9}	X ray	Hazelrigg	<i>Antp</i> ^{73b}	revertant	<i>In(3R)84B1-2;</i> <i>84C5-6</i>
<i>Antp</i> ^B	X ray	Bacon		moderate dominant	<i>In(3R)84B1-2;85E</i>
<i>Antp</i> ^{CB}	X ray	Black		moderate dominant	<i>In(3R)84B1-2;</i> <i>99F-100A</i>
<i>Antp</i> ^{Ctx}	X ray	Lewis	<i>Ctx</i>	strong dominant	<i>T(2;3)35B;84B1-2</i>
<i>Antp</i> ^{Hu}	X ray	Ruch	<i>Hu</i>	moderate dominant	<i>In(3R)84B1-2;</i> <i>84F4;86C7-8</i>
<i>Antp</i> ^{Hu-rv1}	X ray	Hazelrigg	<i>Antp</i> ^{Hu}	revertant	<i>Df(3R)84B1-2;</i> <i>84D6-F4</i>

allele	origin	discoverer	synonym	type	cytology
<i>Antp</i> JK	spont	Kennison		recessive	
<i>Antp</i> LC	Neutron	LeCaivez	<i>Ar</i> , <i>ss</i> ^{Ar}	moderate dominant	<i>In(3R)84A5-6;92A5-6</i>
<i>Antp</i> Ns	spont	Gehring	<i>Ns</i>	viable dominant	normal
<i>Antp</i> Ns-rv1	X ray	Denell		<i>Antp</i> ^{Ns} revertant	<i>In(3R)81;84B1-2</i>
<i>Antp</i> Ns-rv2	γ ray	Duncan		<i>Antp</i> ^{Ns} revertant	<i>In(3R)81F;90BC</i>
<i>Antp</i> Ns-rv3	γ ray	Duncan		<i>Antp</i> ^{Ns} revertant	<i>T(Y;3)Y;84A4-B2</i>
<i>Antp</i> Ns-rv6	γ ray	Duncan		<i>Antp</i> ^{Ns} revertant	<i>In(3LR)79D1-2;84A4-B2</i>
<i>Antp</i> Ns-rv8	γ ray	Duncan		<i>Antp</i> ^{Ns} revertant	normal
<i>Antp</i> Ns-rv11	X ray	Denell		<i>Antp</i> ^{Ns} revertant	normal
<i>Antp</i> Ns-rv13	γ ray	Duncan		<i>Antp</i> ^{Ns} revertant	<i>T(2;3)84A4-B2;40-41</i>
<i>Antp</i> Ns-rv16	γ ray	Duncan		<i>Antp</i> ^{Ns} revertant	Complex
<i>Antp</i> Ns-rv18	γ ray	Duncan		<i>Antp</i> ^{Ns} revertant	<i>T(Y;3)Y;84A4-B2</i>
<i>Antp</i> Ns-rv19	γ ray	Duncan		<i>Antp</i> ^{Ns} revertant	<i>T(Y;3)Y;84B1-3</i>
<i>Antp</i> Ns-rv25	X ray	Denell		<i>Antp</i> ^{Ns} revertant	<i>In(3R)81;84B1-2;85A</i>
<i>Antp</i> Ns-rv70	X ray	Denell		<i>Antp</i> ^{Ns} revertant	normal
<i>Antp</i> Ns-rv72	X ray	Denell		<i>Antp</i> ^{Ns} revertant	<i>Df(3R)84B3;84D</i>
<i>Antp</i> Ns-rv85	X ray	Denell		<i>Antp</i> ^{Ns} revertant	<i>In(3R)81;84B1-2</i>
<i>Antp</i> Ns-rv96	X ray	Denell		<i>Antp</i> ^{Ns} revertant	<i>T(Y;3)Y;84B1-2;94C</i>
<i>Antp</i> Ns-rvC1	EMS	Struhl		<i>Antp</i> ^{Ns} revertant	normal
<i>Antp</i> Ns-rvC2	EMS	Struhl		<i>Antp</i> ^{Ns} revertant	normal
<i>Antp</i> Ns-rvC3	EMS	Struhl		<i>Antp</i> ^{Ns} revertant	normal
<i>Antp</i> Ns-rvC4	EMS	Struhl		<i>Antp</i> ^{Ns} revertant	<i>In(3LR)75B;84B1-2</i>
<i>Antp</i> Ns-rvC5	EMS	Struhl		<i>Antp</i> ^{Ns} revertant	normal
<i>Antp</i> Ns-rvC6	EMS	Struhl		<i>Antp</i> ^{Ns} revertant	normal
<i>Antp</i> Ns-rvC8	EMS	Struhl		<i>Antp</i> ^{Ns} revertant	<i>T(2;3)41;84B1-2</i>
<i>Antp</i> Ns-rvC9	EMS	Struhl		<i>Antp</i> ^{Ns} revertant	normal
<i>Antp</i> Ns-rvC10	EMS	Struhl		<i>Antp</i> ^{Ns} revertant	<i>T(Y;3)Y;84B1-2</i>
<i>Antp</i> Ns-rvC11	EMS	Struhl		<i>Antp</i> ^{Ns} revertant	normal
<i>Antp</i> Ns-rvC12	EMS	Struhl		<i>Antp</i> ^{Ns} revertant	normal
<i>Antp</i> PW	MDN ^α	Pinchin		strong dominant	<i>In(3LR)71F;84B1-2</i>
<i>Antp</i> R	X ray	Rappaport	<i>ss</i> ^a	moderate dominant	<i>In(3R)84B1-2;86C</i>
<i>Antp</i> RM	X ray	R. Meyer		moderate dominant	<i>In(3R)82E1;84B1-2</i>
<i>Antp</i> Scx	spont	Hannah	<i>Scx</i>	weak dominant	normal
<i>Antp</i> W	X ray	Wohlwill		moderate dominant	<i>T(3;4)84B1-2;102F + T(2;3)33E;66C</i>
<i>Antp</i> Wu	γ ray	Wu		strong dominant	<i>In(3LR)75C;84B1-2</i>
<i>Antp</i> Yu	X ray	Yu		strong dominant	<i>T(2;3)22B;83E-F + T(2;3)38E;98A</i>

^α MDN = methoxy diethylnitrosamine.

cytology: Placed in 84B1-2 based on *Antp*'s inclusion in the overlap region between *Df(3R)Scr* and *Df(3R)A41* as well as the commonly held breakpoint of four forward, eleven gain-of-function and eighteen revertant of gain-of-function mutations (see table of alleles).

molecular biology: The *Antp* transcription unit lies at the distal end of the *ANTC* and is transcribed in a distal to proximal (*i.e.*, left to right) direction with respect to the right arm of the third chromosome. The locus has been identified in the DNA through the localization of breakpoints associated with both loss- and gain-of-function mutations. Additionally regulatory portions of the gene have been used to drive the expression of β galactosidase reporter constructs *in vivo* and these constructs produce spatial patterns of expression similar to those seen for the normal gene. The identified transcription unit is 100 kb long and is made up of eight exons. Exons 1 and 2 are the most distal and are found at the 5' end of RNAs initiated from the P1 promoter mentioned previously. Exon 3 is approximately 60 kb downstream of the P1 5' end and represents the leader sequences unique to transcripts initiated at the P2 promoter. The remaining five exons (E4-E8) are common to transcripts initiated at both P1 and P2. Exon 4 also encodes a leader sequence and the identified open reading frame begins in exon 5, 36 nucleotides downstream of the splice acceptor. The open

reading frame continues through exons 6, 7, and 8 ending 240 nucleotides downstream of the splice acceptor of E8. Two polyadenylation sites are used at the downstream end of E8. The first (A1) is *ca.* 875 nucleotides downstream of the 5' end of the exon; the other (A2) is *ca.* 2300 nucleotides more proximal. The two promoters coupled with the two adenylation sites result in the production of four size classes of transcript (P1/A1 = 3.2 kb, P1/A2 = 4.6 kb, P2/A1 = 3.4 kb, P2/A2 = 4.8 kb). All of these have been seen on Northern blots. There is no apparent preferential association of promoter with respect to 3' end formation. However, the two promoters do have different spatial patterns of expression. Notably the P1 promoter is seen to be strongly expressed in the anlagen of the dorsal prothoracic disc, a tissue dramatically affected by its deletion. The P2 promoter is more evenly expressed in *Antp*'s spatial domain (see below), consistent with the defects associated with its inactivation. The 3' end of the transcription unit is *ca.* 30 kb distal to the 3' end of *ftz* and 50 kb distal to the identified 5' end of *Scr*. The distance to the next most distal transcription unit from the P1 5' end is nearly 50 kb. The site of the *Antp*^{Hu} breakpoint is in this 50 kb interval.

In addition to the transcript heterogeneity mentioned above, *Antp* also undergoes alternate splicing among the ORF-containing introns. Specifically exon 6 which encodes thirteen amino acids is found predominantly in embryonic transcripts and less frequently in imaginal disc derived RNAs. Additionally there is an alternate splice at the 3' end of exon 7, resulting in the deletion of four amino acids just upstream of the homeobox motif if the short splice is made. It appears that the long form splice is used preferentially but that all four potential protein forms are made in imaginal discs. The exon-6-less transcripts are rare in embryonic RNA. There is no apparent preferential association of alternate splicing patterns with either of the two promoters. The longest potential protein (E6 + 7L) is 378 amino acids in length, and has a predicted molecular weight of 43 kd. The homeobox motif is encoded in E8 and the opa like repeats in E5.

bcd: bicoid

location: 3-{47.5} (between *zen* and *Ama*).

synonym: *mum: multimorph*.

references: Frohnhöfer and Nüsslein-Vollhard, 1986, *Nature* (London) 324: 120-25 (fig.).

Frohnhöfer and Nüsslein-Vollhard, 1987, *Genes Dev.* 1: 603-14.

phenotype: Maternal-effect lethal mutations showing defective head and thorax development. Females homozygous for strong alleles produce embryos in which head and thorax are replaced by duplicated telson, including anal plates, tuft, spiracles, and flizkörper; however, no pole cells formed at the anterior end. Deletions and fusions of anterior abdominal segments and occasionally anterior abdominal segments in reversed polarity are also observed. Strong alleles amorphic based on phenotypic similarities of embryos produced by homozygous and hemizygous females. Weak alleles result in pattern defects in heads of embryos; lack only labral derivatives (median tooth, dorsal bridge); intermediate weak genotypes produce reduced head but retain normal thoracic development; intermediate strong produce further reduction of head, deletion of second and third and reduction

of first thoracic dentical belts; thoracic segments fused. Partial rescue of embryonic phenotype effected by injection of cytoplasm (5% of volume) from the anterior ends of unfertilized wild-type eggs into the anterior pole of newly fertilized eggs of *bcd* mothers; injection into ectopic sites stimulates differentiation of anterior structures at site of injection; efficiency proportional to number of *bcd*⁺ alleles carried by cytoplasm donor. Phenocopies result from leakage of 5% of egg volume from anterior perforation of normal embryos. The distance of the head fold at gastrulation is proportional to the number of *bcd*⁺ alleles in the maternal genotype. *bcd* mRNA appears as a flattened disc plastered to the anterior extremity of early embryos; by the time of pole cell migration it has become localized to the clear cytoplasm at the periphery, forming a cap over the anterior end of the egg and is distributed in a steeply decreasing gradient such that 90% of the RNA is in the anterior 18% of egg length; by nuclear cycle 14 the RNA begins to disappear and becomes undetectable by midway through cellularization. *bcd* protein on the other hand forms a shallower gradient in which 57% of protein is in the anterior 18% of egg length, and the gradient doesn't reach baseline until the posterior 30% of egg length; the gradient forms from two to four hours after oviposition in both fertilized and unfertilized eggs, and except during mitosis is concentrated in nuclei; diffusion postulated to account for the establishment of the protein gradient following translation from anteriorly anchored RNA. Protein levels decrease during cellularization, although some nuclear staining persists until the end of germ-band elongation. *bcd* transcript first detectable in the ovaries of *bcd* females; forms a ring around the anterior margin of the developing oocyte in stages 5 and 6; in stages 9 and 10 nurse-cell accumulation observed to be localized toward the periphery of the cyst; by stage 12 the nurse cells have emptied their contents into the oocyte and the *bcd* transcript appears as an anterior cap (St. Johnston, Driever, Berleth, Richstein, and Nüsslein-Volhard, 1989, Development Supplement: 13-19). No evidence of translation of *bcd* protein during oogenesis. Formation of the *bcd* gradient is regulated by three maternally active genes *exu*, *sww*, and *stau*; *exu* appears necessary for nurse cell accumulation; *sww* is required for anterior localization of *bcd* mRNA in the oocyte; and *stau* appears to be involved in RNA localization in the embryo. A defect in any of these functions results in little or no gradient of *bcd* activity. *bcd* in turn appears to control the activity of anterior gene activity; specifically the anterior pattern of *hb* expression is not observed and is replaced by a mirror-image posterior *hb* stripe in *bcd*⁻ embryos (Tautz, 1988, Nature 332: 281-84; Schröder, Tautz, Seifertz, and Jäckle, 1988, EMBO J. 7: 2881-87).

alleles:

allele	origin	synonym	ref ^α	comments
<i>bcd</i> ¹	EMS	<i>bcd</i> ⁰⁸⁵	2, 5	intermediate allele; 2564 C → T; 184 gln → amber
<i>bcd</i> ²	EMS	<i>bcd</i> ²⁻¹³	2, 5	weak allele; 3885 T → A; 453 leu → his
<i>bcd</i> ³	EMS	<i>bcd</i> ²³⁻¹⁶	2	strong allele
<i>bcd</i> ⁴	EMS	<i>bcd</i> ³³⁻⁵	2	strong allele
<i>bcd</i> ⁵	EMS	<i>bcd</i> ¹¹¹	2, 5	weak allele; 2798 C → T; 262 gln → amber
<i>bcd</i> ⁶	EMS	<i>bcd</i> ^{E1}	1, 2, 5	strong allele; 2482-2650 deleted

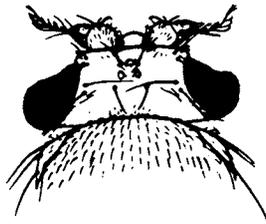
allele	origin	synonym	ref ^α	comments
<i>bcd</i> ⁷	EMS	<i>bcd</i> ^{E2}	1, 2	+ TA inserted; frameshift → 55 out-of-frame amino acids replacing amino acids 156-494, including homeodomain
<i>bcd</i> ⁸	EMS	<i>bcd</i> ^{E3}	2, 5	Strong allele; 260 base-pair deletion overlapping homeodomain
<i>bcd</i> ⁹	EMS	<i>bcd</i> ^{E4}	2, 5	intermediate allele; strongly temperature sensitive; 2406 C → T; 131 ser → leu
<i>bcd</i> ¹⁰	EMS	<i>bcd</i> ^{E5}	2, 5	intermediate allele; 2393 C → T; 127 leu → phe
<i>bcd</i> ¹¹	EMS	<i>bcd</i> ^{E6}	5	weak allele; 2804 C → T; 264 gln → amber
<i>bcd</i> ¹²	EMS	<i>bcd</i> ^{GB}	2, 5	2388-2420 deleted; amino acids 125-135 deleted
<i>bcd</i> ¹³			3	strong allele; 2486 C → T; 158 gln → amber
<i>bcd</i> ¹⁴			3	hypomorphic allele
<i>bcd</i> ¹⁵			4	hypomorphic allele
<i>bcd</i> ¹⁶			4	strong hypomorphic allele

^α 1 = Berleth, Burri, Thoma, Bopp, Richstein, Frigerio, Noll, and Nüsslein-Volhard, 1988, EMBO J. 7: 1749-56; 2 = Fronhöfer and Nüsslein-Volhard, 1986, Nature 324: 120-25; 3 = Lambert, 1985, PhD Thesis, Indiana University; 4 = Seeger, 1989, PhD Thesis, Indiana University; 5 = Struhl, Struhl, and MacDonald, 1989, Cell 12: 59-73.

cytology: Placed in region 84A1 on the basis of failure to be complemented by *Df(3R)9A99 = Df(3R)83F2-84A1;84B1-2*; *Df(3R)LIN*, and *Df(3R)Scr = Df(3R)84A1-2;84B1-2*, and complementation by *Df(3R)4SCB = Df(3R)84A6-B1;84B2-3*, and *Df(3R)Antp17 = Df(3R)84A6;84D13-14*.

molecular biology: Gene identified in an 8.7-kb genomic fragment from coordinates -42 to -33 kb of the chromosome walk of Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman (1983, Cell 35: 763-76) by germ-line transformants that completely rescue the mutant phenotype (Berleth, Burri, Thoma, Bopp, Richstein, Frigerio, Noll, and Nüsslein-Volhard, 1988, EMBO J. 7: 1749-56; see also Frigerio, Burri, Bopp, Baumgardner, and Noll, 1986, Cell 47: 735-46; Kilchherr, Baumgardner, Bopp, Frei, and Noll, 1986, Nature 321: 493-97). The transcription unit comprises four exons and produces a major mRNA of 2.6 kb, which contains all four exons, and a minor 1.6-kb mRNA from which exons 2 and 3 are spliced. Splice-acceptor-site variation in the third exon leads to translation products of 489 and 494 amino acids (53.9 kd). The first exon contains a PRD repeat, consisting essentially of alternating histidines and prolines, found within a number of genes, including *prd*, expressed early in development; the 5' end of exon 3 encodes a novel homeodomain with no more than 40% amino-acid homology with other homeobox sequences; the 3' end contains a series of repeated glutamines, opa repeats. Also contains a RNA-recognition motif, mostly in exon 4 (Rebagliatti, 1989, Cell 58: 231-32). A highly acidic C-terminal domain is thought to provide transcriptional activation; the latter can be replaced with heterologous activating sequences and still display *bcd*⁺ activity (Driever, Ma, Nüsslein-Volhard, and Ptashne, 1989, Nature 342: 149-54). The sequence responsible for the anterior localization of *bcd* RNA at the anterior embryonic pole localized to 625 nucleotides in the 3' untranslated region, which include regions capable of forming extensive secondary structure

(Macdonald and Struhl, 1988, Nature 336: 595-600). The ten residues from 138 to 147 comprise the DNA recognition helix of the *bcd* homeodomain; replacing the lysine in the ninth position of this ten-amino-acid sequence with either alanine or glutamine is sufficient to destroy recognition of *hb* sequences; in addition, the latter substitution confers a new specificity for *Anp* and *Ubx* upstream target sequences (Hanes and Brent, 1989, Cell 57: 1275-83). Bicoid protein binds to five high-affinity binding sites (consensus sequence TCTAATCCC) upstream from the *hb* transcription start site (Driever and Nüsslein-Volhard, 1989, Nature 337: 138-43). The posterior boundary of the anterior *hb* domain responds to changes in the number or affinity of these sites as well as to the dose of *bcd*⁺ such that increases cause a more posterior and decreases a more anterior boundary (Driever, Thoma, and Nüsslein-Volhard, 1989, Nature 340: 363-67; Struhl, Struhl, and Macdonald, 1989, Cell 57: 1259-73).



Dfd: Deformed

From Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 94.

Dfd: Deformed

location: 3-47.5.

references: Chadwick and McGinnis, 1987, EMBO J. 3: 779-89.

Hazelrigg and Kaufman, 1983, Genetics 105: 581-600.

Jack, Regulski, and McGinnis, 1988, Genes Dev. 2: 635-51.

Kuziora and McGinnis, 1988, Cell 55: 477-85.

Martinez-Arias, Ingham, Scott, and Akam, 1987, Dev. 100: 673-83.

Merrill, Turner, and Kaufman, 1987, Dev. Biol. 122: 379-95.

Regulski, McGinnis, Chadwick, and McGinnis, 1987, EMBO J. 3: 767-77.

Chadwick, Jones, Jack, and McGinnis, 1990, Dev. Biol. 141: 130-40.

phenotype: Null mutations act as recessive lethals. Homozygous or hemizygous animals die at the end of embryogenesis and show a spectrum of defects in the head. There are no discernible defects in the trunk. The head defects are associated with missing structures normally derived from the mandibular and maxillary segments, the dorsal lateral papillae of the maxillary sense organ, the mouth hooks, and the maxillary cirri. The remaining gnathal structures are present albeit disarranged likely due to abnormalities in the movements associated with head involution. A weak homeotic

transformation (30-50% penetrance) has also been noted in animals hemizygous for a breakpoint-associated revertant of the single dominant gain-of-function allele (*Dfd*^{rv1}). The phenotype is an apparent transformation of the H piece and lateral-graten which appear to be replaced by cephalopharyngeal plates. This phenotype has not been observed in any other mutant genotype and the reason for its low-penetrance production by this particular allele is not known. X-ray-induced somatic clones of *Dfd*⁻ cells have shown that the locus is also required for adult head development. These cells develop normally in the thorax and abdomen but do not form structures in the ventral anterior aspect of the head; specifically the vibrissae and maxillary palps. Clones in the dorsal posterior part of the head form ectopic bristles which have been interpreted as a head to thoracic transformation. A temperature-conditional allele has been used to define two temperature-critical periods for *Dfd*⁺ activity. The first is during embryogenesis during segmentation and head involution, while the second occurs in the late third instar larval through mid pupal stages. These times correlate nicely with the observed cuticular defects in mutant animals and the times of peak gene product accumulation. There is a single dominant gain-of-function allele which causes defects in the ventral aspects of the adult head similar to those seen in the *Dfd*⁻ head clones mentioned above. There are no defects seen in the posterior of the head nor does this allele cause any embryonic or larval defects as a heterozygote, homozygote, or hemizygote. This allele is associated with a group of *B104* (*roo*) insertion elements (ca. 50 kb of inserted DNA) as well as a duplication of the 3' exons of the *Dfd* transcription unit (see below). The mutant causes an extended spatial domain of expression of the locus into the eye portion of the eye-antennal disc as compared to the pattern seen in normal animals. The precise cause-effect relationship between the observed molecular defect and the mutant phenotype is not known except that partial deletion of the *B104* elements but not the 3' end duplication causes a reversion of the dominant phenotype and has no apparent effect on the wild type function of the resident *Dfd* gene. This dominant allele has been reverted and these revertants act as a simple recessive loss-of-function alleles with the one exception noted above. The *Dfd* transcript is initially detected at the blastoderm stage in a band of cells at the position of the future cephalic furrow. This RNA shows maximal accumulation from 6-12 hours of embryogenesis when it is found in the mandibular and maxillary lobes, as well as in the subesophageal region of the CNS. The amount of *Dfd* RNA diminishes through the first and second larval instars and peaks again during the third instar. At this point, it is found in the peripodial membrane cells of the eye-antennal discs. The cells which accumulate the RNA are those which have been fate mapped to give rise to the adult-head-capsule structures which are defective in *Dfd*⁻ clones. Antibodies raised to *Dfd* protein have shown a similar pattern of accumulation to that seen for the RNA. The protein is first detected in cellular blastoderm stage in a stripe of six cells which circumscribes the embryo. As germ-band elongation proceeds and segmentation becomes evident *Dfd* protein is detected in the mandibular and maxillary lobes and a portion of the dorsal ridge. During germ-band shortening protein is no longer detect-

able in the mandibular lobe or in the anterior lateral aspect of the maxillary lobe. The process of head involution carries the *Dfd*-expressing cells interiorly where they are found in portions of the pharynx at the end of embryogenesis. *Dfd*-positive cells are also found in the subesophageal region of the CNS in the maxillary ganglion. This expression pattern has been shown to be dependent on the prior expression of the gap and pair-rule segmentation genes for its inception and on an autonomous regulatory element upstream of the *Dfd* transcription initiation site for the maintenance of *Dfd* expression into the later stages of embryogenesis. Immunostaining of imaginal discs shows *Dfd*-positive cells in the peripodial membrane of the eye-antennal discs with no detectable accumulation in the disc proper. There are also a few cells in the stalk of the labial discs which appear to accumulate *Dfd* protein. The *Dfd* cDNA driven by a heat shock promoter has been returned to flies and used to ectopically express *Dfd* protein. Animals carrying this construct subjected to heat shock produce ectopic mouth hooks and maxillary cirri in the ventral aspect of their thoracic segments, two structures missing in *Dfd*⁻ animals. There is no phenotypic affect on abdominal pattern; however, head development is severely disrupted in heat-pulsed animals.

alleles:

allele	origin	discoverer	synonym	type	cytology
<i>Dfd</i> ¹	spont	Cattell, 13g		dominant allele	normal
<i>Dfd</i> ²	EMS	Cain	<i>Dfd</i> ^{rC9}	hypomorphic allele	normal
<i>Dfd</i> ³	EMS	Cain	<i>Dfd</i> ^{rC11}	temperature sensitive	normal
<i>Dfd</i> ⁴	EMS	Fornili	<i>Dfd</i> ^{rf1}	hypomorphic allele	normal
<i>Dfd</i> ⁵	EMS	Fornili	<i>Dfd</i> ^{rf78}	hypomorphic allele	normal
<i>Dfd</i> ⁶	X ray	Kaufman	<i>Dfd</i> ^{rK2}	null allele	?
<i>Dfd</i> ⁷	X ray	Kaufman	<i>Dfd</i> ^{rK26}	hypomorphic allele	?
<i>Dfd</i> ⁸	EMS	Matthews	<i>Dfd</i> ^{rKM2}	hypomorphic allele	normal
<i>Dfd</i> ⁹	EMS	Matthews	<i>Dfd</i> ^{rKM24}	hypomorphic allele	normal
<i>Dfd</i> ¹⁰	EMS	R. Lewis	<i>Dfd</i> ^{rR1}	hypomorphic allele	normal
<i>Dfd</i> ¹¹	EMS	R. Lewis	<i>Dfd</i> ^{rR3}	null allele	normal
<i>Dfd</i> ¹²	EMS	R. Lewis	<i>Dfd</i> ^{rR11}	hypomorphic allele	normal
<i>Dfd</i> ¹³	EMS	Merrill	<i>Dfd</i> ^{rV8}	hypomorphic allele	normal
<i>Dfd</i> ¹⁴	EMS	Merrill	<i>Dfd</i> ^{rV13}	hypomorphic allele	normal
<i>Dfd</i> ¹⁵	EMS	Wakimoto	<i>Dfd</i> ^{rW6}	hypomorphic allele	normal
<i>Dfd</i> ¹⁶	EMS	Wakimoto	<i>Dfd</i> ^{rW21}	null allele	normal
<i>Dfd</i> ^{rv1}	X ray	Hazelrigg	<i>Dfd</i> ^{+RX1}	<i>Dfd</i> ¹ revertant	<i>Tp(3;3)83D4-5;</i> <i>84A4-5;98F1-2</i>
<i>Dfd</i> ^{rv2}	X ray	Hazelrigg	<i>Dfd</i> ^{+RX13}	<i>Dfd</i> ¹ revertant	<i>Df(3R)83E3;</i> <i>84A4-5</i>
<i>Dfd</i> ^{rv3}	X ray	Hazelrigg	<i>Dfd</i> ^{+RX16}	<i>Dfd</i> ¹ revertant	<i>Tp(3;3)86F11;</i> <i>87D14;84A4-5</i>
<i>Dfd</i> ^{rv4}	X ray	Hazelrigg	<i>Dfd</i> ^{+RX17}	<i>Dfd</i> ¹ revertant	normal

cytology: Placed in 84A4-5 by its inclusion in *Df(3R)Scr*, *Df(3R)Antp17*, and *Df(3R)Dfd13* and the location of two revertant-associated breakpoints *Dfd*^{rv1} and *Dfd*^{rv2}.

molecular biology: The *Dfd* transcription unit has been identified in the *ANTC* by its association with two *Dfd* revertant breakpoints which interrupt it and result in the recessive lethal mutant phenotype. The identified transcription unit covers 11 kb of genomic DNA and is made up of five exons. The 5'-most three exons are separated by two relatively small introns and these are separated from the 3'-most two exons by a large 7-kb intron. Transcription proceeds from proximal to distal (with respect to the chromosome centromere to telomere). This orientation is opposite to that of all the other homeotic loci in the *ANTC*. The next most proximal gene in the complex is *Ama*, the 3' end of which is just over 20 kb from the 5'

end of *Dfd*. Distally the 3' end of *Dfd* is 20 kb from the 3' end of *Scr*. The five exons sum to 2.75 kb, a figure in good agreement with the 2.8 kb transcript size seen in Northern blots. Sequence analysis of a full length cDNA shows a long open reading frame of 1758 nucleotides encoding a protein of 586 amino acids, yielding a molecular weight of 63.5 kd. The homeobox is encoded by exon four and the opa repeats are downstream in exon five.

ftz: fushi tarazu

location: 3-47.5.

references: Hafen, Kuroiwa, and Gehring, 1984, Cell 37: 833-41.

Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

Kuroiwa, Hafen, and Gehring, 1984, Cell 37: 825-31.

Laughon and Scott, 1984, Nature 310: 23-31.

Wakimoto, Turner, and Kaufman, 1984, Dev. Biol. 102: 147-72.

Weiner, Scott, and Kaufman, 1984, Cell 37: 843-51.

Carroll and Scott, 1985, Cell 43: 47-57.

Hiromi, Kuroiwa, and Gehring, 1985, Cell 43: 603-13.

Duncan, 1986, Cell 47: 297-309.

Hiromi and Gehring, 1987, Cell 50: 963-74.

Doe, Hiromi, Gehring, and Goodman, 1988, Science 239: 170-75.

phenotype: Null loss-of-function mutations result in embryonic lethality. Animals survive to the end of embryogenesis and exhibit a pair-rule mutant phenotype in the cuticle. This same phenotype is observable in animals at the beginning of segmentation of the germ band. Prior to deposition of cuticle, *ftz*⁻ animals have two rather than three mouth (gnathocephalic) segments and five as compared to ten trunk metameres. The material deleted is derived from the even-numbered parasegments, ps2 through ps12. Similar metamer deletions/fusions are seen in the neuromeres of the ventral nerve cord of the CNS. The name of the locus derives from the phenotype and is Japanese for "segment" (*fushi*) "deficient" (*tarazu*) (*N.B.* — there is only one letter t in tarazu; it is at the start of the word *i.e.*, there is **no** second t preceding the z). Temperature-sensitive alleles of the gene have shown that the temperature-critical period for viability and phenotype falls between 1 and 4 hours of embryogenesis with the mid point of 2.5 hours at the blastoderm stage. The recovery of clones of *ftz*⁻ cells created by X-ray-induced somatic exchange after cellular blastoderm have demonstrated that *ftz*⁺ activity is not necessary for normal cuticular morphogenesis subsequent to this point in development. In addition to these recessive null and hypomorphic alleles there are two classes of dominant gain-of-function lesions at the *ftz* locus. The first, *ftz-Regulator of postbithorax-like*, causes a variable transformation of the posterior haltere into posterior wing. The second, *ftz-Ultra-abdominal-like*, is associated with a patchy transformation of the adult first abdominal segment toward third abdominal identity. The former (*ftz*^{Rpl}) lesion also shows a recessive loss-of-function phenotype while the latter class (*ftz*^{Ual}) has no discernable embryonic phenotype and is homozygous viable. The fact that these dominant alleles produce mutant phenotypes that mimic lesions in the *BXC* has been inter-

preted as demonstrating a regulatory link between the segment enumeration genes and the homeotics. alleles:

allele	origin	discoverer	synonym	type	cytology
<i>ftz</i> ¹	DEB	M. Bender	<i>ftz</i> ^{b54ts}	hypomorphic allele	normal
<i>ftz</i> ²	DEB	M. Bender	<i>ftz</i> ^{b5ts}	hypomorphic allele	normal
<i>ftz</i> ³	EMS	Cain	<i>ftz</i> ^{c15}	null allele	normal
<i>ftz</i> ⁴	EMS	Cain	<i>ftz</i> ^{clts}	hypomorphic allele	normal
<i>ftz</i> ⁵	EMS	Fornili	<i>ftz</i> ^{f47ts}	hypomorphic allele	normal
<i>ftz</i> ⁶	EMS	Kaufman	<i>ftz</i> ^{k5}	null allele	normal
<i>ftz</i> ⁷	EMS	Matthews	<i>ftz</i> ^{km13}	null allele	normal
<i>ftz</i> ⁸	EMS	K. Matthews	<i>ftz</i> ^{kmQ}	null allele	normal
<i>ftz</i> ⁹	EMS	R. Lewis	<i>ftz</i> ^{R13}	null allele	normal
<i>ftz</i> ¹⁰	EMS	R. Lewis	<i>ftz</i> ^{R14}	null allele	normal
<i>ftz</i> ¹¹	EMS	Wakimoto	<i>ftz</i> ^{w20}	null allele	normal
<i>ftz</i> ¹² α	EMS	Jürgens	<i>ftz</i> ^{7B}		
<i>ftz</i> ¹³	EMS	Jürgens	<i>ftz</i> ^{9H34}		
<i>ftz</i> ¹⁴	EMS	Jürgens	<i>ftz</i> ⁹⁰⁹³		
<i>ftz</i> ¹⁵	EMS	Jürgens	<i>ftz</i> ^{E66}		
<i>ftz</i> ¹⁶	EMS	Jürgens	<i>ftz</i> ^{E193}		
<i>ftz</i> ^{Rpl}	X ray	Duncan	<i>ftz</i> ^{Rpl}	dominant allele	<i>T(2;3)84A6-B1;41</i>
<i>ftz</i> ^{Ual1}	EMS	E.B. Lewis	<i>ftz</i> ^{Ual1}	dominant allele	normal
<i>ftz</i> ^{Ual2}	ENU	Chiang	<i>ftz</i> ^{Ual2}	dominant allele	normal
<i>ftz</i> ^{Ual3}	EMS	Duncan	<i>ftz</i> ^{Ual3}	dominant allele	normal
<i>ftz</i> ^{Ual2rv1}	EMS	Duncan	<i>ftz</i> ⁻	revertant of <i>ftz</i> ^{Ual2}	normal
<i>ftz</i> ^{Ual2rv2}	X ray	Duncan	<i>ftz</i> ⁻	revertant of <i>ftz</i> ^{Ual2}	normal
<i>ftz</i> ^{Ual2rv3} β	spont	Duncan	<i>ftz</i> ⁻	revertant of <i>ftz</i> ^{Ual2}	?

α Associated with a 5-kb insertion element in the transcribed region of *ftz*.
 β Behaves genetically as a deletion of *ftz*, *Scr*, and *Antp*.

cytology: Placed in 84B1-2 based on its inclusion in *Df(3R)Scr* and the 3R breakpoint of *T(2;3)ftz^{Rpl}*, which is known to interrupt the coding region of the *ftz* transcription unit.

molecular biology: The localization and identification of the *ftz* transcription unit within the *ANTC* has been accomplished through the mapping of *ftz*-associated aberrations in the DNA [*ftz*¹¹ and *T(2;3)ftz^{Rpl}*] and the rescue of *ftz*⁻ genotypes using *P*-element mediated transformation. The transcription unit is just over 2 kb in length and is made up of two exons of 800 and 980 base pairs and a single 150-base-pair intron. The open reading frame is 1,239 nucleotides long and initiates in the (800 bp) 5' exon. Conceptual translation of the open reading frame predicts a protein of 398 amino acids with a molecular weight of 43 kd. The most prominent motifs in the protein are the homeodomain (encoded in the second exon) and a PEST domain which may be important in the dynamic pattern of *ftz* expression. Northern blots have shown that the *ftz* transcript is accumulated in early embryos starting at about 2 hours (syncytial blastoderm), peaking shortly afterwards and declining at about 4 hours. These times are coincident with the temperature-sensitive-period data noted above. The spatial pattern of RNA accumulation is first seen as a broad band at syncytial blastoderm extending from the position of the future cephalic furrow posteriorly to about 15% egg length. At cellular blastoderm this broad single band resolves into seven transverse stripes which circumscribe the embryo. These stripes disappear as gastrulation proceeds and are gone by mid gastrulation. Protein accumulation lags behind the RNA and is first detected at cellular blastoderm in the seven-stripe pattern. The position and width of the stripes indicates that *ftz* expression occurs within the even-numbered parasegmental anlagen, which are missing in *ftz*⁻ animals. Subsequent to the ectodermal expression in the germ band, the *ftz* protein product is

again detected in the later stages of germ-band shortening, in a subset of cells in each of the segmental ganglia of the ventral nerve cord. This expression continues to the end of embryogenesis and has been shown to be important in the proper morphogenesis of a specific set of neurons repeated in each ganglion. Transformation studies have resulted in the identification of at least three *cis*-acting upstream regulatory elements necessary for normal *ftz* expression. An 1-kb fragment just upstream of the start of transcription is necessary for the establishment of the striped pattern at cellular blastoderm. Another fragment just distal to this element is needed for expression in the CNS, while about 6 kb upstream is an element necessary for the maintenance of stripes. It has also been shown that this *cis*-acting maintenance element requires the presence of *ftz* protein and therefore that *ftz* is apparently autogenously regulated in the later stages of its expression.

lab: labial

location: 3-47.5.

references: Diederich, Merrill, Pultz, and Kaufman, 1989, *Genes Dev.* 3: 399-414.

Merrill, Diederich, Turner, and Kaufman, 1989, *Dev. Biol.* 135: 376-91.

Mlodzik, Fjose, and Gehring, 1988, *EMBO J.* 7: 2569-78.

phenotype: Null mutations act as recessive embryonic lethals. Animals survive to the end of embryogenesis and have normal thoracic, abdominal, and caudal segments. However, the head is abnormal, and shows defects in derivatives of all of the gnathocephalic segments. There is no obvious homeotic transformation in these animals. Analysis of earlier stages shows abnormalities in the process of head involution. X-ray-induced clones of *lab*⁻ cells demonstrate that *lab* function is unnecessary for the development of the adult thorax and abdomen. However, clones in the head fail to develop normally and show deletions in the maxilla and eye. Dorsally the posterior head capsule is transformed toward an apparent thoracic identity. A temperature conditional allele has been used to show a temperature critical period between 6 and 14 hours of embryogenesis. This period coincides with an interval in which head involution, a process disrupted by *lab*⁻, takes place. Antisera raised to *lab* protein have shown it to initially accumulated just anterior to the gnathocephalic region of the germ band at the early stages of segmentation. This protein also is found in a row of cells extending above the gnathal region in the procephalic lobe and more dorsally into the dorsal ridge. As segmentation, germ-band shortening and head involution proceed, the cells expressing the protein are involved in the process complexities of head involution. Finally at the end of morphogenesis, *lab* positive cells are found in the lateral aspects of the pharynx, the tritocerebral ganglia of the CNS, and the frontal sac. In addition to this expression in the head, *lab* protein is also found in endodermal cells at the posterior of the anterior midgut and the anterior cells of the posterior midgut. The position and movements of the cephalic cells accumulating *lab* is consistent with the interpretation that this locus is expressed in the intercalary or most anterior of the gnathal segments.

alleles:

allele	origin	discoverer	synonym	comments
lab 1	EMS	R. Lewis	<i>lab</i> ^{r9}	hypomorphic allele
lab 2	X ray	Kaufman	<i>lab</i> ^{k3}	temperature-sensitive allele
lab 3	EMS	Fornili	<i>lab</i> ^{f7}	hypomorphic allele
lab 4	EMS	Fornili	<i>lab</i> ^{f8}	null allele
lab 5	EMS	Fornili	<i>lab</i> ^{f10}	hypomorphic allele
lab 6	EMS	Fornili	<i>lab</i> ^{f33}	hypomorphic allele
lab 7	EMS	Fornili	<i>lab</i> ^{f40}	null allele
lab 8	EMS	Fornili	<i>lab</i> ^{f56}	hypomorphic allele
lab 9	X ray	Abbott	<i>lab</i> ^{a76}	null allele; <i>In(3R)84A1-2;84E</i>
lab 10	EMS	Merrill	<i>lab</i> ^{v14}	hypomorphic allele
lab 11	DEB	Seeger	<i>lab</i> ^{l5}	null allele
lab 12	DEB	Seeger	<i>lab</i> ^{l10}	hypomorphic allele
lab 13	DEB	Seeger	<i>lab</i> ^{lB1}	hypomorphic allele
lab 14	X ray	Diederich	<i>lab</i> ^{vd1}	null allele; (insertion)
lab 15	X ray	Diederich	<i>lab</i> ^{vd2}	hypomorphic allele; (deletion)
lab 16	X ray	Merrill	<i>lab</i> ^{vd21}	null allele; <i>T(3;4)84A1-2;101</i>
lab 17	X ray	Merrill	<i>lab</i> ^{vd22}	hypomorphic allele
lab 18	X ray	Merrill	<i>lab</i> ^{vd35}	hypomorphic allele

cytology: Placed in 84A1-2 based on its inclusion in *Df(3R)Scr* and the location of the proximal 3R breakpoints of two rearranged alleles *In(3R)lab*⁹ and *T(3;4)lab*¹⁶. These latter two breakpoints have been located in the DNA and are known to interrupt the *lab* transcription unit.

molecular biology: The *lab* transcription unit is the most proximal in the *ANTC* and has been localized and identified by mapping the position of four *lab*⁻ associated rearrangements in the DNA (*lab*⁹, *lab*¹⁴, *lab*¹⁵, and *lab*¹⁶) and the rescue of *lab*⁻ animals by a minigene constructed from the transcription unit implicated by the breakpoints. The *lab* transcription unit is 17 kb in length, is made up of three exons and is transcribed from distal to proximal on the chromosome. Exons two and three are separated by a 245-bp intron and these from the 5' exon by a 13.8-kb intron. The open reading frame begins at nucleotide +239 in the first exon and extends through the third. Conceptual translation of the open reading frame predicts a protein of 629 amino acids and a molecular weight of 67.5 kd. Northern blot analysis detects a single Poly(A)⁺ RNA of 3.0 kb, a size in good agreement with the identified exons of 1455, 416, and 935 bp. This RNA is first detected at 2-4 hours of embryogenesis and remains present through the larval and pupal stages. There is no detectable accumulation in adults. The encoded protein contains opa sequences as well as a homeodomain. The latter is encoded by sequences in exons two and three, has its closest similarity to the *pb* homeodomain, and shares with that homeobox the position of its intronic interruption.

pb: proboscipedia

location: 3-47.5.

references: Bridges and Dobzhansky, 1933, Wilhelm Roux's Arch. Dev. Biol. 127: 575-90.

Kaufman, 1978, Genetics 90: 579-96.

Lewis, Wakimoto, Denell, and Kaufman, 1980, Genetics 95: 383-97.

Pultz, Diederich, Cribbs, and Kaufman, 1988, Genes Dev. 2: 901-20.

phenotype: Null alleles transform the labial palps of the adult into portions of the prothoracic leg. The distal tarsal segments are present, including claws and pulvilli. The distal portion of the first tarsal segment including the sex comb in males is fused directly to the proximal portion of the femur. Thus proximal first tarsus, tibia, and distal femur are absent. Leg segments proximal to femur are not present. Hypomorphic alleles produce a labial-palp-to-antenna transformation. Generally only more distal (arista) antennal structures are seen. Extremely weak hypomorphic alleles exist which produce no ostensible phenotype as homozygotes but do reveal a weak antennal transformation in combination with a deletion or null allele. Both null and hypomorphic alleles also show an alteration in maxillary palp morphology which has been interpreted as a transformation toward an antennal identity.

alleles:

allele	origin	discoverer	synonym	phenotype	cytology
pb 1	spont	Bridges and Dobzhansky		18° → antenna 29° → leg	+ +
pb 2	γ ray	Duncan and Kaufman		leg	+
pb 3	γ ray	Duncan and Kaufman		leg	+
pb 4	γ ray	Duncan and Kaufman		antenna	+
pb 5	γ ray	Duncan and Kaufman		leg	+
pb 6	spont	Baker, W.K.	<i>pb</i> ^{72j}	weak antenna	+
pb 7	X ray	Abbott	<i>pb</i> ^{a19}	leg	+
pb 8	X ray	Abbott	<i>pb</i> ^{a21}	leg	+
pb 9	X ray	Abbott	<i>pb</i> ^{a70}	leg	+
pb 10	X ray	Diederich	<i>pb</i> ^{bd4}	leg	+
pb 11	EMS	Cain	<i>pb</i> ^{c13}	antenna	+
pb 12	X ray	Kaufman	<i>pb</i> ^{draw1}	leg	+
pb 13	EMS	Fornili	<i>pb</i> ^{f4}	weak antenna	+
pb 14	EMS	Fornili	<i>pb</i> ^{f73}	18° → antenna 29° → leg	+
pb 15	EMS	Hazelrigg	<i>pb</i> ^{h62}	antenna	+
pb 16	X ray	Kaufman	<i>pb</i> ^{lose1}	leg	<i>In(3LR)66B;84A4-5</i>
pb 17	EMS	Matthews	<i>pb</i> ^{m1}	leg	+
pb 18	EMS	Matthews	<i>pb</i> ^{m2}	leg	+
pb 19	EMS	Matthews	<i>pb</i> ^{m3}	leg	+
pb 20	X ray	Pultz	<i>pb</i> ^{map2}	leg	<i>Df(3R)84A4-5</i>
pb 21	X ray	Pultz	<i>pb</i> ^{map3}	leg	<i>T(2;3)84A4-5;26D-F</i>
pb 22	X ray	Pultz	<i>pb</i> ^{map6}	leg	+
pb 23	X ray	Pultz	<i>pb</i> ^{map8}	leg	+
pb 24	X ray	Pultz	<i>pb</i> ^{map9}	leg	(deletion)
pb 25	X ray	Pultz	<i>pb</i> ^{map10}	leg	<i>In(3R)84A4-5;84D</i>
pb 26	X ray	Pultz	<i>pb</i> ^{map12}	leg	<i>In(3R)84A4-5;het</i>
pb 27	X ray	Pultz	<i>pb</i> ^{map13}	leg	<i>In(3R)84A4-5;85D</i>
pb 28	X ray	Pultz	<i>pb</i> ^{map17}	leg	(deletion)
pb 29	X ray	Pultz	<i>pb</i> ^{map17}	variegating leg	<i>In(3R)84A4-5;het</i>
pb 30	EMS	Merrill	<i>pb</i> ^{v10}	leg	+
pb 31	EMS	Merrill	<i>pb</i> ^{v12}	leg	+
pb 32	EMS	Wakimoto	<i>pb</i> ^{w4}	18° → leg 29° → antenna	+
pb 33	EMS	Wakimoto	<i>pb</i> ^{w19}	antenna	+
pb 34	X ray	Kaufman	<i>pb</i> ^{win1}	leg	<i>Df(3R)84A4-5;84B1-2</i>
pb 35	X ray	Kaufman	<i>pb</i> ^{win3}	leg	<i>Df(3R)84A4-5;84C1-2</i>
pb 36	X ray	Kaufman	<i>pb</i> ^{win5}	leg	+
pb 37	X ray	Kaufman	<i>pb</i> ^{win12}	leg	<i>In(3R)84A4-5;87A5</i>
pb 38	X ray	Kaufman	<i>pb</i> ^{x1}	leg	+
pb 39	X ray	Kaufman	<i>pb</i> ^{x2}	leg	<i>Df(3R)84A4-5;84B1-2</i>
pb 40	X ray	Matthews	<i>pb</i> ^{x3}	leg	<i>T(2;3)44F;84D</i>
			<i>pb</i> ^{x4}	leg	+

cytology: Placed in 84A4-5 based on its inclusion in *Df(3R)Scr* and the location of eleven *pb*-associated breakpoints in this doublet (see table of alleles).

molecular biology: The *pb* transcription unit extends over

35 kb of DNA in the proximal portion of the *ANTC*. It is bounded distally by the z2 transcription unit and proximally by a cluster of at least eight cuticle-like genes. Neither the proximal nor distal transcripts have any demonstrable function in the fly. The transcription unit produces a single 4.3-kb mRNA which is derived from nine exons distributed over the interval. The open reading frame begins in exon two and ends in exon nine. The homeobox motif is encoded in exons four and five and is split by intron four in the same position as the homeobox is split in the labial gene. The opa sequences are in exon eight and are therefore downstream of the homeobox. Exon three is roughly equidistant between its two flanking exons and the two largest introns of the gene. This exon is 15 nucleotides long and is alternately spliced. The RNA and protein products of the gene are accumulated in the maxillary and mandibular lobes of the embryo and the labial discs of the larvae.

Scr: Sex combs reduced

location: 3-47.5.

- references:** Kuroiwa, Kloter, Baumgartner, and Gehring, 1985, *EMBO J.* 4: 3757-64.
 Sato, Hayes, and Denell, 1985, *Dev. Biol.* 111: 171-92.
 Mahaffey and Kaufman, 1987, *Genetics* 117: 51-60.
 Martinez-Arias, Ingham, Scott, and Akam, 1987, *Development* 100: 673-83.
 Riley, Carroll, and Scott, 1987, *Genes Dev.* 1: 716-30.
 Carroll, DiNardo, O'Farrell, White and Scott, 1988, *Genes Dev* 2: 350-60.
 Glicksman and Brower, 1988, *Dev. Biol.* 127: 113-18.
 LeMotte, Kuroiwa, Fessler, and Gehring, 1989, *EMBO J.* 8: 219-27.
 Mahaffey, Diederich, and Kaufman, 1989, *Development* 105: 167-74.

phenotype: Null mutations at the locus result in embryonic lethality. Animals die at the end of embryogenesis and show evidence of homeotic transformation in the cuticle derived from the labial and first thoracic segments. The first thorax is transformed to a second thoracic identity and the labial segment toward maxillary. This latter phenotype is seen as a duplication of the maxillary sense organs and the cirri. Deletions of the locus as well as null alleles also produce a dominant phenotype most clearly seen in males as a reduction in the number of sex-comb teeth. This reduction is indicative of a partial transformation of first leg to second, a conclusion borne out by the recovery of hypomorphic alleles of the locus which as hemizygotes allow survival to the adult stage and have no obvious effect in the embryo. These survivors show a complete transformation of ventral prothorax to mesothorax including the presence of steno-pleural bristles on the propleurae; they also show an apparent transformation of the dorsal prothorax toward a mesothoracic identity. In addition to these thoracic transformations, the labial palps are transformed toward a maxillary palp morphology. All of these adult transformations can also be seen in X-ray-induced somatic clones of *Scr*⁻ cells. Thus *Scr* activity is needed for proper segmental identity in both the embryo and adult in the anterior-most segment of the thorax and the posterior-most metamere of the head. In the absence of *Scr* product these two segments are transformed divergently to the identity of the next most posterior and ante-

rior metamere respectively. The only other homeotic mutation to produce such a divergent homeosis is *pb*, which appears to act similarly in the adjacent maxillary and labial segments of the adult head. In addition to these loss-of-function mutations there are several gain-of-function dominant alleles. All result in a similar phenotype in adults, most clearly seen in males as the production of sex combs on the second and third thoracic legs. Additionally, strong alleles of this type (*Scr*^{*ScxW*}, *Scr*^{*ScxP*}, and *Scr*^{*ScxS*}) show the loss of sternopleural bristles indicative of a more complete transformation of mesothorax to prothorax. All of these dominants are associated with genomic rearrangements and with the exception of *Scr*^{*ScxS*} act as recessive lethals (*Scr*^{*Msc*}, *Scr*^{*ScxT1*}, *Scr*^{*ScxT2*}, and *Scr*^{*ScxP*}) or semilethals (*Scr*^{*ScxW*} and *Scr*^{*ScxT3*}) at the locus. Examination of animals carrying these lesions at the end of embryogenesis as heterozygotes with a normal chromosome or hemizygotes reveals no evidence of the gain-of-function transformation of T2 and T3 → T1, only the loss-of-function phenotypes described above. These phenotypic observations have been extended by showing that *Scr* protein is accumulated ectopically in the second and third leg imaginal discs in dominant gain-of-function genotypes but not in the second and third thoracic segments at any point in embryogenesis. Thus it appears that the spatial pattern of *Scr* expression is differentially regulated at these two times. Genetic analyses have shown that at least one difference lies in *Scr* imaginal expression being subject to a transvection-like effect. The gain-of-function lesions cause or allow the ectopic expression of the structural gene on the *trans*- rather than the *cis*-coupled transcription unit. This is most clearly seen in the case of *Scr*^{*ScxT1*}, which is broken within the transcribed portion of *Scr* and is therefore incapable of making a functional gene product. *Scr* mRNA is first detected in embryos in early gastrulae in a band of cells just posterior to the cephalic furrow. Protein is not detected at this time but later during germ-band elongation; it is found in the region of the labial lobe. Subsequently, during germ-band retraction, RNA and protein are detected in the first thoracic segment with the highest concentration at the anterior border of this segment. RNA and protein are also detected in the subesophageal region of the CNS in the labial ganglion and in mesodermal cells associated with the anterior midgut. As head involution proceeds, the *Scr*-expressing cells of the labial segment are carried inside where they are found associated with the pharynx and the mouthparts at the end of embryogenesis. In the third larval instar, protein is found in the prothoracic leg discs, the dorsal prothoracic discs, the labial discs, and a small group of cells in the stalk of the antennal portion of the eye-antennal disc where it attaches to the mouthparts. In addition to this disc expression, *Scr* protein is accumulated in the subesophageal region of the CNS. This spatial pattern of expression in the epidermis is consistent with the spectrum of defects seen in *Scr*⁻ animals and clones.

alleles:

allele	origin	discoverer	synonym	type	cytology
Scr¹	EMS	Denell	<i>Scr</i> ^{<i>d8</i>}	null allele	normal
Scr²	X ray	Kaufman	<i>Scr</i> ^{<i>k6</i>}	null allele	normal
Scr³	EMS	R. Lewis	<i>Scr</i> ^{<i>r18</i>}	hypomorphic allele	normal

allele	origin	discoverer	synonym	type	cytology
<i>Scr</i> ⁴	EMS	Wakimoto	<i>Scr</i> ^{w17}	null allele	normal
<i>Scr</i> ⁵	EMS	Wakimoto	<i>Scr</i> ^{w22}	hypomorphic allele	normal
<i>Scr</i> ⁶	EMS	Fornili	<i>Scr</i> ^{f2cs}	cold-sensitive	normal
				hypomorphic allele	
<i>Scr</i> ⁷	EMS	Fornili	<i>Scr</i> ^{f71}	hypomorphic allele	normal
<i>Scr</i> ⁸	EMS	Fornili	<i>Scr</i> ^{f76cs}	cold-sensitive	normal
				hypomorphic allele	
<i>Scr</i> ⁹	X ray	Abbott	<i>Scr</i> ^{a68}	null allele	<i>In(3LR)77D</i> ; <i>84B1-2</i>
<i>Scr</i> ¹⁰	X ray	Abbott	<i>Scr</i> ^{a72}	null allele	<i>In(3LR)75B</i> ; <i>84B1-2</i>
<i>Scr</i> ¹¹	EMS	Lambert	<i>Scr</i> ^{c12}	null allele	normal
<i>Scr</i> ¹²	EMS	Stephenson	<i>Scr</i> ^{e40}	null allele	normal
<i>Scr</i> ¹³	EMS	Matthews	<i>Scr</i> ^{km0}	null allele	normal
<i>Scr</i> ¹⁴	EMS	Matthews	<i>Scr</i> ^{km7}	hypomorphic allele	normal
<i>Scr</i> ¹⁵	EMS	Matthews	<i>Scr</i> ^{km12}	hypomorphic allele	normal
<i>Scr</i> ¹⁶	EMS	Matthews	<i>Scr</i> ^{km15}	null allele	normal
<i>Scr</i> ¹⁷	X ray	Pultz	<i>Scr</i> ^{p18}	null allele	normal
<i>Scr</i> ¹⁸	X ray	Merrill	<i>Scr</i> ^{VD30}	null allele	<i>In(3R)84B1-2</i> ; <i>95F</i>
<i>Scr</i> ¹⁹	X ray	Jürgens	<i>Scr</i> ^{XF5}	null allele	<i>T(2;3)?</i>
<i>Scr</i> ²⁰	X ray	Jürgens	<i>Scr</i> ^{XT145}	null allele	<i>T(2;3)?</i>
<i>Scr</i> ^{Msc}	spont	Tokunaga	<i>Msc</i>	Dominant allele	<i>In(3R)84B1-2</i> ; <i>84F1-2</i>
<i>Scr</i> ^W	EMS	Wakimoto	<i>Scr</i> ^{w15}	Dominant allele	50 kb inversion in 84B1-2
<i>Scr</i> ^{Wrv1}	X ray	Hazelrigg		<i>Scr</i> ^W revertant	<i>T(2;3)58F1-2</i> ; <i>84B1-2</i>
<i>Scr</i> ^{Wrv3}	X ray	Hazelrigg		<i>Scr</i> ^W revertant	normal
<i>Scr</i> ^{Wrv5}	X ray	Hazelrigg		<i>Scr</i> ^W revertant	<i>In(3R)81;84B1-2</i>
<i>Scr</i> ^{Wrv6}	X ray	Hazelrigg		<i>Scr</i> ^W revertant	<i>T(2;3)22D</i> ; <i>63A1-2+</i> <i>T(2;3)54A1</i> ; <i>80-81</i>
<i>Scr</i> ^{T1}	X ray	Tiong	<i>Scr</i> ^{T1}	Dominant allele	<i>Tp(3;3)80-81</i> ; <i>84B1-2;84D5-6</i>
<i>Scr</i> ^{T2}	X ray	Tiong	<i>Scr</i> ^{T2}	Dominant allele	<i>T(2;3)40-41</i> ; <i>84B1-2</i>
<i>Scr</i> ^{T3}	X ray	Tiong	<i>Scr</i> ^{T3}	Dominant allele	<i>T(2;3)25D;40</i> ; <i>84B1-2+</i> <i>T(2;3)29B</i> ; <i>91E</i>
<i>Scr</i> ^P	X ray	Pultz	<i>Scr</i> ^{P1}	Dominant allele	<i>T(3;4)80-81</i> ; <i>84B1-2;102F</i>
<i>Scr</i> ^S	DEB	Seeger	<i>Scr</i> ^{msl}	Dominant allele	

cytology: Placed in 84B1-2 based on its inclusion in *Df(3R)Scr*, and the common 84B1-2 breakpoints of eleven *Scr* mutations.

molecular biology: The *Scr* transcription unit has been identified by the localization of twelve *Scr*-associated breakpoints and the overlap junction of six deletions. Three of these approach *Scr* from its distal limit [*Df(3R)Antp7*, *Df(3R)A41*, and *Df(3R)Hu*]; the remainder delete the proximal end of the gene [*Df(3R)Dfd13*]. The identified transcription unit spans 25 kb of genomic DNA and is made up of three exons. Proceeding from 5' to 3' they are 0.5, 1.0, and 2.5 kb in length. The two introns are 6.0 and 15 kb respectively. The 3' end of *Scr* is 20 kb distal to the 3' end of *Dfd*, and the 5' end of *Scr* is 18 kb proximal to the 5' end of *ftz* and 50 kb proximal to the 3' end of *Antp*. Breakpoint associated mutations in this latter 50-kb interval all affect *Scr* function indicating that this region is important for the normal expression of the transcription unit. The sum of the three identified exons is in close agreement with the 3.9-kb mRNA detected on Northern blots. There is a single large open reading frame, which initiates in exon 2 just downstream of the splice acceptor, and terminates in exon 3 about 300 nucleotides downstream of the splice acceptor. Thus the 3' tail is just over 2 kb in length. The total open reading

frame is 1,245 nucleotides in length and encodes a protein of 413 amino acids with a predicted molecular weight of 45 kd. The homeobox motif is encoded in exon 3 and opa-like repeats are found in exon 2.

z2: zen-2

location: 3-47.5 (inferred from close proximity to *zen*).

synonym: *zpr: zen pattern related*.

references: Rushlow, Doyle, Hoey, and Levine, 1987, *Genes Dev.* 1: 1268-79.

Pultz, Diederich, Cribbs, and Kaufman, 1988, *Genes Dev.* 2: 901-20.

phenotype: Simultaneous deletion of both *z2* and *pb* produces only a *pb* mutant phenotype. Thus absence of *z2* function has no discernible effect on development or morphology.

cytology: Placed in 84A4-5 based on its inclusion in *Df(3R)Scr* and exclusion from *Df(3R)LIN*.

molecular biology: The *z2* transcription unit maps 10 kb proximal to *zen* and about 1 kb distal to the transcription initiation site of *pb*. An open reading frame starts 67 bp downstream of the transcription start site in exon 1 and extends to 114 bp upstream of a consensus poly(A) addition site. The transcript produced is 1.0 kb in length and shows the same spatio-temporal expression pattern as the neighboring *zen* gene. Like *zen* conceptual translation of the *z2* open reading frame reveals the presence of a homeobox domain (encoded in exon 2). This sequence shows good similarity to the *zen* homeobox (75%), but there is little other sequence similarity found in the remainder of the proteins. The function of this locus is not known, but in light of the fact that its deletion causes no detectable effect, and *zen* mutants can be rescued by a genomic fragment which does not contain *z2*, it is likely the locus represents a pseudogene. Consistent with this conclusion is the finding that a *z2* homologue is not found in the *ANTC* of *D. pseudoobscura*.

zen: zerknüllt

Location: 3-47.5 (between *pb* and *bcd* in the *ANTC*).

references: Wakimoto, Turner, and Kaufman, 1984, *Dev. Biol.* 102: 147-72.

Rushlow, Doyle, Hoey, and Levine, 1987, *Genes and Dev.* 1: 1268-79.

Rushlow, Frasch, Doyle, and Levine, 1987, *Nature* 330: 583-86.

Doyle, Harding, Hoey, and Levine, 1986, *Nature* 323: 76-9.

phenotype: Null mutations result in embryonic lethality and the loss of several dorsally derived embryonic structures, including the amnioserosa, optic lobe, and dorsal ridge. These animals also fail to fully extend their germ bands and go through the process of head involution. The name for the locus derives from the characteristic "wrinkled" appearance of the germ band seen in the SEM at the time of normal germ-band retraction. Hypomorphic mutations result in the absence of dorsal structures but do undergo normal gastrulation movements. A temperature-sensitive allele has been used to define the time of *zen*⁺ action between 2 and 4 hours of embryogenesis, just prior to and overlapping the earliest observable morphogenic defects. X-ray induced somatic clones have further shown that *zen*⁺ function is unnecessary for postembryonic development. The RNA product of *zen* is first detected at about 2 hours of development during the

eleventh to twelfth cell cycle of the syncytial blastoderm. At this early stage the RNA is found on the dorsal surface of the embryo extending around the anterior and posterior poles. As cellularization proceeds and the early events of gastrulation begin, the RNA becomes restricted to a mid-dorsal stripe of cells. These cells have been fate mapped and give rise to the amnioserosa and the lobes in the dorsal posterior of the embryonic head, *i.e.*, the structures absent in *zen*⁻ animals. The time of appearance of *zen* RNA also correlates nicely with the temperature-sensitive-period data obtained using the conditional allele. Antisera to the *zen* protein product has been used to follow its accumulation pattern, and this analysis agrees with and expands the *in situ* results. The protein is located in the nuclei of cells expressing the gene and at cellular blastoderm is found in a mid-dorsal stripe seven cells wide and seventy cells in length. During gastrulation these cells eventually give rise to the amnioserosa, the optic lobe, and dorsal ridge; these structures continue to show *zen* protein accumulation until the end of germ-band extension at about 4 to 6 hours of development. This end point also correlates well with the end of the temperature-sensitive period of the conditional allele. The spatial pattern of *zen* expression has been shown to be dependent on the products of several of the maternally expressed genes which specify the anterior-posterior and dorsal-ventral polarity of the embryo, and *zen* would appear to lie near the end of the axis-determining pathway.

alleles: Seven ethyl-methanesulfonate-induced alleles, all of which have normal cytology.

allele	discoverer	synonym	comments
zen ¹	Forniii	<i>zen</i> ^{f16}	temperature-sensitive hypomorphic allele
zen ²	Forniii	<i>zen</i> ^{f27}	null allele
zen ³	Forniii	<i>zen</i> ^{f55}	null allele
zen ⁴	Forniii	<i>zen</i> ^{f62}	null allele
zen ⁵	Forniii	<i>zen</i> ^{f75}	hypomorphic allele
zen ⁶	Merrill	<i>zen</i> ^{v1}	null allele
zen ⁷	Wakimoto	<i>zen</i> ^{w36}	null allele

cytology: Placed in 84B1-2 based on its inclusion in *Df(3R)Scr* and *Df(3R)SCB-XL2*.

molecular biology: One of a pair of regions between *pb* and *bcd* in the *ANTC* has been shown to be *zen* by *P*-element mediated transformation and rescue of a *zen*⁻ genotype. The rescuing fragment is 4.5 kb in length and carries a single 1.3 kb transcription unit. It is composed of two exons separated by a 64-base pair intron. The start of translation is 52 base pairs downstream of the transcription start site in the first exon. The open reading frame ends 169 base pairs upstream of a poly(A) addition site in exon 2. The predicted size of mature message from genomic and cDNA sequence analysis is 1.3 kb which is in agreement with the transcript size observed on Northern blots. Conceptual translation of the open reading frame shows the presence of a homeodomain and PEST sequences, which are enriched for the amino acids serine, threonine, proline, and glutamic acid. The presence of both of these motifs correlates well with the DNA binding activity of *zen* protein and the dynamic pattern of protein accumulation seen for *zen* protein *in vivo*.

antenna: see *ems*

Antennapedex: see *Apx*

Antennapedia: see *Antp* under *ANTC*

anterior open: see *aop*

anterobithorax: see *abx* under *BXC*

Antp: see *ANTC*

ants: antennas

location: 3-49.4.

origin: Spontaneous.

references: Ribó, 1968, DIS 43: 59.

phenotype: Antennae modified—lengthened or reduced—especially in males. Viability good.

aop: anterior open

location: 2-12 (approximate).

references: Nüsslein-Volhard, Wieschaus, and Kluding, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82. (fig.).

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Embryonic lethal. Homozygous embryos have anterior dorsal hole in epidermis. Brain and sometimes gut extrude through hole. Head involution normal. Visible during dorsal closure.

alleles: Six ethyl-methanesulfonate induced alleles; *aop*¹ and *aop*² (recovered as *IP* and *IIS*) retained.

aor: abdominal one reduced

location: 3-{85}.

references: González, Molina, Casal, and Ripoll, 1989, Genetics 123: 371-77.

phenotype: Hemizygotes lack the first abdominal segment. Histoblasts of third-instar larvae normally present in A1.

cytology: Placed in 96A1-7 based on its association with *In(3R)Ubx*^{7L} = *In(3R)89E;96A1-7* and its inclusion in *Df(3R)L16* = *Df(3R)96A1-10;96E*.

***ap: apterous** (T.G. Wilson)

location: 2-55.2.

references: Metz, 1914, Am. Naturalist 48: 675-92.

Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 236 (fig.).

Stevens and Bryant, 1985, Genetics 110: 281-97.

1986, Genetics 112: 217-28.

phenotype: Wings and halteres reduced to traces. Bristles eliminated from area around wing base (including posterior notopleurals, anterior and posterior supra-alars, and anterior postalars); posterior scutellars erect when present but missing in first counts; dorsocentrals smaller and fewer; hairs on thorax sparse and irregular. Sutural furrow reduced; thorax disproportionately small. Flies small, pale, weak, and very short lived. Viability about 70% that of wild type but erratic. Both sexes sterile. RK2.

alleles: Apterous alleles generally fall into three groups based on phenotypic differences. Most of the characterized apterous alleles belong to the first group and have basically the *ap* or *ap*⁴ phenotype. Some alleles (*ap*^{blt2} and *ap*^{T60}) have a less severe wing phenotype, being straplike. Alleles also vary in their expressivity of the precocious adult death and nonvitellogenic ovary phenotypic characters; some alleles result in a low number of escapers, similar to *ap*⁴, while others have an escaper percentage of as much as 50%. There is little correlation between expressivity of the wing deficiency phenotype

and either precocious adult death or nonvitellogenic ovary development, but a good correlation exists between expressivity of the latter phenotypic characters (Wilson, 1980). Generally, heterozygous combinations of these alleles do not show complementation for any phenotypic characters. Another group, represented by *ap^{blt}*, exhibits a less severe, somewhat different phenotype; attributable to localized lysosomal cell death in the presumptive wing blade. (Sedlock, Mango, and Stevend, 1984, Dev. Biol. 104: 489-96). A third group includes two dominant alleles. The apterous locus appears to be a complex locus, containing several partially complementing groups for the wing deficiency and adult-death/female-sterility phenotypic characteristics. However, by studying the effects of a number of different temperature regimens on phenotypic expression of three different temperature-sensitive alleles, Stevens and Bryant (1986) conclude that all phenotypes are explicable in terms of changes in quantity rather than quality of gene product.

allele	origin	discoverer	synonym	ref ^α	phenotype ^β
<i>*ap¹</i>	spont	E.M. Wallace, 13h		4, 8, 14	
<i>*ap²</i>	spont	Bridges, 16j20	<i>ap-c</i>	3, 4, 8	<i>ap⁴</i>
<i>ap^{2a}</i>	EMS	Wilson, 1978		20	
<i>*ap³</i>	spont	Morgan, 23a	<i>ap-c</i> , no wings	8, 17	<i>ap⁴</i>
<i>ap^{3a}</i>	EMS	Wilson, 1978		20	<i>ap^{56f}</i>
<i>ap⁴</i>		Medvedev, 32a15	<i>ap-d</i>	8, 13, 20	γ
<i>ap^{4a}</i>	EMS	Wilson, 1978		20	
<i>ap⁵</i>	U.V.	Byers, 49f		8, 16	<i>ap⁴</i>
<i>ap⁶</i>	spont	Faulhaber		6, 8, 10	<i>ap⁴</i>
<i>ap¹³</i>	EMS	Wilson		20	
<i>ap¹⁸</i>	EMS	Wilson, 1978		20	
<i>ap²⁵</i>	EMS	Wilson, 1978		20	
<i>ap³²</i>	EMS	Wilson, 1978		20	
<i>ap⁴⁰</i>	EMS	Wilson, 1978		20	<i>ap^{56f}</i>
<i>ap⁴⁶</i>	EMS	Wilson, 1978		20	
<i>ap⁴⁹</i>	EMS	Wilson, 1978		20, 21	
<i>ap^{49j}</i>	spont	Ritterhoff, 49j		8, 11, 20	<i>ap⁴</i>
<i>ap⁵⁴</i>	EMS	Wilson, 1978		8, 20, 21	<i>ap^{78j}</i>
<i>ap^{56f}</i>	spont	Thompson, 56f		5, 8, 20	γ
<i>ap⁵⁷</i>	EMS	Wilson, 1978		20	
<i>ap⁵⁸</i>	EMS	Wilson, 1978		20	
<i>ap^{67e}</i>	spont	Lee		12	
<i>ap^{69c1}</i>	EMS	Au		7	
<i>ap^{69c2}</i>	EMS	Gottschalk		7	
<i>ap^{69c3}</i>	EMS	Nadler		7	
<i>ap⁷³ⁿ</i>	spont	Altorfer, 73n		1	<i>ap⁴</i>
<i>ap^{77f}</i>	EMS	Wilson, 77f		20, 23	γ
<i>ap^{78e}</i>	EMS	Wilson, 78e			
<i>ap^{78j}</i>	EMS	Wilson, 78j		20, 21, 24	γ
<i>ap^{blt}</i>	spont	Groscurth, 31b1	<i>blt</i>	8	γ
<i>ap^{blt*}</i>				20	
<i>ap^{blt2}</i>	spont	Whittinghill, 44h		8, 22	< <i>ap⁴</i>
<i>ap^{blt3}</i>	spont	Semenza, 49k	<i>blt^{S49k}</i>	2, 8	<i>ap⁴</i>
<i>ap^c</i>				20	
<i>ap^e</i>	spont			18	γ
<i>ap^{ID}</i>			<i>ap^{id}</i>	20	<i>ap^{Xa?}</i>
<i>ap^{T60}</i>	X ray	Thomas, 60g		8, 15	< <i>ap⁴</i>
<i>ap^{trw}</i>	spont		<i>trw</i>	9	γ
<i>ap^{Xa}</i>	X ray	Serebrovsky, 28a	<i>Xa</i>	8, 19, 20	γ

^α 1 = Altorfer, 1977, DIS 52: 2; 2 = Barigozzi, 1950, DIS 24: 54; 3 = Bridges, 1919, J. Exp. Zool. 28: 370. 4 = Bridges and Morgan,

1919, Carnegie Inst. Washington Publ. 278: 236 (fig.); 5 = Burdick, 1956, DIS 30: 69; 6 = Butterworth and King, 1965, Genetics 52: 1153-74; 7 = Butterworth, Nolph, Au, Gottschalk, Nadler, and Tuma, 1970, DIS 45: 36; 8 = CP627; 9 = Crist, Fontaine, and Merrell, 1980, DIS 55: 204; 10 = Faulhaber, 1963, DIS 37: 48; 11 = Glass, 1951, DIS 25: 76-77; 12 = Lee, 1972, DIS 48: 18; 13 = Medvedev and Bridges, 1935, Tr. Inst. Genet. Akad. Nauk. SSR 10: 119-209; 14 = Metz, 1914, Am. Nat. 48: 675-92 (fig.); 15 = Meyer, 1963, DIS 37: 50; 16 = Meyer, Edmondson, Byers, and Erickson, 1950, DIS 24: 59; 17 = Morgan, 1929, Carnegie Inst. Washington Publ. 399: 183; 18 = Roberts and Bownes, 1982, DIS 58: 209; 19 = Serebrovsky and Dubinin, 1930, J. Hered. 21: 259-65; 20 = Stevens and Bryant, 1985, Genetics 110: 281-97; 21 = Stevens and Bryant, 1986, Genetics 110: 217-28; 22 = Whittinghill, 1947, DIS 21: 71; 23 = Wilson, 1980, Dev. Genet. 1: 195-204; 24 = Wilson, 1981, Dev. Biol. 85: 425-33.

^β Designation of allele with similar phenotype.

γ Phenotypes described below in separate entries.

cytology: Placed in salivary region 41B-C (Schultz).

ap⁴

phenotype: Wings less than 10% normal length, lacking all wing blade structures. Halteres reduced to structure-less remnants less than 25% normal size. Scutellar and dorsocentral bristles sometimes missing (Butterworth and King, 1965, Genetics 52: 1153-74). Wing phenotype disc autonomous in *ap⁴/ap⁺* mosaic flies, although small patches of *ap⁴* wing structures are found in *ap⁴/ap⁺* mosaic wings. Haltere phenotype disc autonomous (Wilson, 1981, Dev. Biol. 85: 434-45). Adults become paralyzed about 30 hr following eclosion and die soon thereafter. Around 1% of adults are long-lived "escapers" of this phenotype (Wilson, 1980, Dev. Genet. 1: 195-204). Precocious adult-death phenotype fate-maps to proximity of Malpighian tubules, and tubule malfunctioning postulated to result in this phenotype (Wilson, 1981). Foregut of females swollen owing to accumulation of peritrophic membrane (King and Sang, 1958, DIS 32: 133). Female sterile with underdeveloped ovaries; nurse cell nuclei become pycnotic after stage 7, and stage-8 oocytes are the most advanced (King and Burnett, 1957, Growth 21: 263-80; Wilson, 1980). *ap⁴* ovaries develop nonautonomously when transplanted to a wild-type host (King and Bodenstern, 1965, Z. Naturforsch. 20B: 292-97). Application of juvenile hormone mimic, ZR-515, to newly eclosed *ap⁴* females results in vitellogenic oocytes [Postlethwait and Weiser, 1973, Nature (London) New Biol. 244: 284-85]. Membranes of vitellogenic oocytes lack microvilli and pinocytotoxic vesicles normally present; development of these structures stimulated by administration of ZR-515 (Tedesco, Courtwright, and Kumaran, 1981, J. Insect. Physiol. 27: 895-902). Corpora allata from adult *ap⁴* are juvenile-hormone deficient when bioassayed [Postlethwait, Handler, and Gray, 1975, The Juvenile Hormones (L.I. Gilbert, ed.), pp. 449-69]. Nonvitellogenic oocyte phenotype fate-maps to same or similar location as precocious adult death phenotype (Wilson, 1981). Escaper females develop stage-14 oocytes (King and Sang, 1958) and are fertile (Wilson, 1980). Males show immature sexual behavior and are sterile, but testes appear normal with motile sperm (King and Sang, 1958). Larval fat body histolysis delayed; this phenotype is nonautonomous as determined by transplantation experiments (Butterworth, 1972, Dev. Biol. 28: 311-25). Application of ZR-515 accelerates larval fat body histolysis in *ap⁴* adults (Postlethwait and Jones, 1978, J. Expt.

Zool. 203: 207-14). Ovarian acid phosphatase level low in ap^4 females and is restored after application of ZR-515 (Postlethwait *et al.*, 1975). ap^4 ovaries cultured *in vitro* are capable of yolk protein synthesis (Redfern and Bownes, 1982, Mol. Gen. Genet. 195: 181-83). $ap^4/Df(2L)M41A-54$ hemizygote has nearly normal complement of bristles but otherwise resembles ap^4 homozygote (Butterworth and King, 1965).

ap^{56f}

phenotype: Wing and haltere phenotype like ap^4 . Scutellar and dorsocentral bristles missing (Butterworth and King, 1965, Genetics 52: 1153-74). Rear and middle legs occasionally twisted, more frequently in female than in male. Both sexes fertile and long lived when homozygous and in combination with other ap alleles. $ap^{56f}/M(2)S2^4$ have normal complement of dorsocentral and scutellar bristles (Butterworth and King, 1965).

ap^{77i}

phenotype: Weakest non-temperature-sensitive allele known. Wing has reasonably good wing blade development, with missing triple-row elements and posterior wing margin. Haltere less well developed but more so than ap^4 . Adults long lived and fertile. Less dominant in heteroallelic combination with ap^4 -like alleles than is ap^{56f} . $ap^{77i}/Df(2R)M41A4$ has more severe phenotype than ap^{77i} homozygotes.

ap^{78j}

phenotype: A temperature-sensitive allele of apterous. When raised at 22°, wing and haltere phenotype approaches wild type except for missing patches of triple-row bristles and posterior wing margin. When raised at higher temperatures, phenotype becomes more severe and resembles ap^4 at 29°. Two nonoverlapping temperature-sensitive periods in development, one in late-second to middle-third instar for wing and haltere deficiency phenotype and the other during the first day of pupal development for precocious adult death and nonvitellogenesis phenotype. Wing discs of heat-pulsed larvae failed to exhibit cell death by trypan blue exclusion.

ap^{blt} : apterous-blot

phenotype: Wings blistered, sometimes inflated and dark due to trapped hemolymph. Mirror-image duplication of posterior wing blade structures occurs [Waddington, 1939, Proc. Nat. Acad. Sci. USA 25: 299-307; Whittle, 1979, J. Embryol. Exp. Morphol. 53: 292-303 (fig.)]. Wing venation may be disrupted. Portions of posterior wing compartment may be transformed into anterior compartment structures, an effect like that of engrailed (*en*; 2-62.0). Despite relatively mild adult phenotype, extensive cell death observed, localized to wing pouch of imaginal discs; associated with acid phosphatase and lysosomal activity (Sedlak, Manzo, and Stevens, 1984, Dev. Biol. 104: 489-96). Clonal analysis revealed nonautonomous expression of phenotype. Heterozygotes with ap^4 or ap^{56f} and hemizygotes show blistering phenotype only (Whittle). ap^{blt}/ap^{73n} shows transformation phenotype, and aldehyde oxidase histochemical staining of these wing discs is consistent with transformation (Whittle and Sprey, 1982, Wilhelm Roux's Arch. Dev. Biol. 191: 285-88). Much overlapping with wild type, and expressivity variable. Adults long lived and fertile.

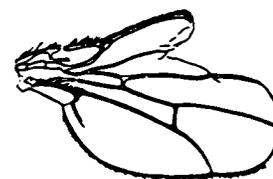
ap^e

phenotype: Homozygotes display extreme wing reduction, particularly of the posterior wing compartment. Approximately 50% of the flies have duplications of the anterior wing margin, distal costa, and triple row bristles. In wings with large amounts of wing blade, very little venation is present; however, these may often have triplications or even four copies of the anterior wing margin, some located in the posterior part of the wing. Dried hemolymph sometimes trapped between the dorsal and ventral wing surfaces giving the wing a puffy blackened appearance. This mutant therefore has duplications and deficiencies characteristic of cell death followed by regulation in the wing, but also has transformations of the posterior wing compartment to the anterior wing compartment. 8% of the flies have defective third legs, more frequently in females than in males. Halteres and scutellar bristles appear to be normal. Homozygotes viable and fertile.

ap^{trw} : apterous-torn wing

phenotype: Distal part of wing in homozygotes shows sawtooth pattern as if tip torn away. Expression uniform in males and females. Viability and fertility good.

other information: Genetic location and phenotype suggests allelism with apterous, but not tested with viable ap alleles.



ap^{Xa} : apterous-Xasta

From Bridges and Brehme, 1944, Carnegie Inst. Washington Publ. No. 552: 228.

ap^{Xa} : apterous-Xasta

phenotype: Wings reduced in length to about 70% normal; irregular in outline with a V-shaped incision with apex at L2, uniformly present giving wing a mitten-like shape with the thumb between marginal vein and L2. Excellent dominant with no overlap. Fertile and fully viable in heterozygote. Usually lethal in homozygous conditions, but occasionally ecloses very late as pale dwarf with wings and balancers like *vg*. Deep notch visible in tip of wing fold in prepupa (Waddington, 1939, Proc. Nat. Acad. Sci. USA 25: 299-307). In homozygotes and in combination with ap^4 , ap^6 , or $Df(2R)M41A4$, wings are straplike and 30-70% normal length, and haltere length is 25-50% normal; longevity and fertility like ap^4/ap^4 except for an occasional long-lived $ap^{Xa}/Df(2R)M41A4$ female that may be fertile [Butterworth and King, 1965, Genetics 52: 1153-74 (fig.)]. In heterozygous combination with ap^{ID} , duplications of the notum occur frequently. Wing disc cell death found in both $ap^{Xa}/+$ (Fristrom, 1969, Mol. Gen. Genet. 103: 363-79) and ap^{Xa}/ap^{ID} [Postlethwait, 1978, Genetics and Biology of *Drosophila* (Ashburner and Wright, eds.), Academic Press, London, New York, San Francisco, Vol. 2C,

pp. 418-19 (fig.).

cytology: Shown by Sturtevant (1934, DIS 2: 19) to be associated with $T(2;3)ap^{Aa} = T(2;3)41F;89E8-F1$ which is superimposed on $In(2R)Cy$ and $In(3R)P$ (Morgan, Bridges, and Schultz, 1936, Year Book - Carnegie Inst. Washington 35: 294; Lewis, 1951, DIS 25: 109).

ap-c: see *ap*²

ap-c: see *ap*³

ap-d: see *ap*⁴

apang: see **apg**

Apart: see **Apt**

***apb**: **apterblister**

location: 2-44.7.

origin: Ultraviolet induced.

discoverer: Edmondson, 49k.

references: Meyer, Edmondson, Byers, and Erickson, 1950, DIS 24: 59-60.

phenotype: Wings always notched, nearly always spread, and usually blistered but expression somewhat variable. Homozygous imagos live less than 24 hr, owing to intestinal obstructions. Abdomens characteristically turn dark grey before death because of accumulation of digested food products. Although not at same locus as *ap*, *apb* $+/+$ *ap*⁴, flies show slight notching of wings and many die within a day; those that survive are fertile. *ap*⁵ gives a similar heterozygous effect. RK2.

Ape: **Apurinic endonuclease**

location: 3-{47}.

synonym: AP3.

references: Kelly, Venugopal, Harless, and Deutsch, 1989, Mol. Cell Biol. 9: 965-73.

phenotype: Encodes an apurinic-apyrimidinic DNA endonuclease, AP3. Biochemical studies by Spiering and Deutsch (1986, J. Biol. Chem. 261: 3222-28).

cytology: Placed in 79C-D by *in situ* hybridization.

molecular biology: Isolated from a cDNA expression library using antiserum directed against human enzyme known to cross react with the Drosophila enzyme (Spiering and Deutsch). The conceptual translation product predicts a 317-amino-acid polypeptide of molecular weight 34.2 kd. Region between nucleotides 30 and 173 shows 66% homology with *recA* of *E. coli* and 42% amino-acid identity. Two helix-turn-helix domains detected in the carboxy-terminal end of the polypeptide. Northern blots identify a 1.3-kb transcript at all stages of development, but somewhat reduced in pupae and adult males; there is also transiently present, a 3.5-kb transcript in four-to-eight-hour embryos that disappears after second larval instar.

apexless: see **apx**

aperA: **abnormal proboscis extension reflex A** (J.C. Hall)

location: 1-22.1.

origin: Induced by ethyl methanesulfonate.

discoverer: Kimura.

references: Kimura, Shimozawa and Tanimura, 1986, J. Exp. Zool. 239: 393-99.

phenotype: Variable phenotypic defects in the sugar-induced proboscis extension reflex (PER): some *aperA*

flies cannot extend their probosces at all, whereas they are able to open the labellar lobes; some individuals extend their probosces only to the right or the left side of the body; each mutant individual seems to have a fixed phenotype, e.g., a fly which shows one-sided PER always extends its proboscis to the same side; the array of aberrant phenotypes is different under the influence of the two mutant alleles: for *aperA*¹, 47.0% were unable to extend their probosces, 16% only to the left side, and the remainder extended their probosces normally; for *aperA*²: 32% could not extend their probosces, 23% could extend them only to the right side, and 22% only to the left side (the remainder behaved normally).

alleles: Two alleles; *aperA*¹ (=TT1), *aperA*² (=TT360), with the overall penetrance for the former ca. 79%; 77% for the latter.

aperB (J.C. Hall)

location: 1-0.6.

origin: Induced by ethyl methanesulfonate.

discoverer: Kimura.

references: Kimura, Shimozawa and Tanimura, 1986, J. Exp. Zool. 239: 393-99.

phenotype: Given sugar stimuli *aperB* flies extended their probosces, not straight forward (as does wild-type), but backward; when these mutants show a partial extension of their probosces, the direction of the extensions is normal; the expression of the *aperB* gene is sensitive to culture temperature: when the *aperB*¹ mutants were reared at low temperature (18° or 20°C), over 90% of the flies were normal, whereas the high culture temperature (over 25°C) caused an abnormal PER; the temperature at which the proboscis extension reflex was tested did not affect the phenotype.

cytology: Maps to 2D3-F3; based on its inclusion in $Df(1)Pgd = Df(1)2D3;2F5$ but not $Df(1)JC19 = Df(1)2F3;3C5$; $w^+Y = Dp(1;Y)2D2;3D2-3$ covers *aperB*¹.

alleles: Two alleles: *aperB*¹ (=TT665) and *aperB*² (=TF48), which lead to indistinguishable phenotypic defects.

other information: *aperB* mutations are completely recessive, and complement the closely linked *aperC* mutation.

aperC (J.C. Hall)

location: 1-0.4.

origin: Induced by ethyl methanesulfonate.

discoverer: Kimura.

references: Kimura, Shimozawa and Tanimura, 1986, J. Exp. Zool. 239: 393-99.

1986, Devel. Biol. 117: 194-203.

1987, J. Neurogenet. 4: 21-28.

phenotype: Sugar-induced proboscis extension nearly absent (*i.e.* no extension at all of rostrum and haustellum), but not until adults are three to six days old; this defect, which is completely recessive, wanes such that at least half of the adults behave normally again by approximately day 10-11; correlated with these behavioral changes is time-dependent degeneration and regeneration of a pair of muscles, the rostral protractors; behavioral and histological phenotypes are temperature-sensitive: 18° causes defects later in adult life, and yet there is no recovery; 29° causes lower than usual (*i.e.* 25°) proportion of adults developing the defects, and high temperature is compatible with recovery; temperature-sensitive

period is from two to four days post-eclosion.

cytology: Maps to 1F5-2A, based on its inclusion in $Df(1)A94 = Df(1)1F5;2B15$ and $Df(1)S39 = Df(1)1E4;2B11-20$ plus the fact that the X-chromosome duplication from the distal tip to 2A, from $T(1;Y)G20$, covers *aperC*.

other information: *aperC* completely recessive and complements the closely linked *aperB* mutations.

apg: apang

location: 2-7.7.

origin: Induced by ethyl methanesulfonate.

references: Shakaron and Sharma, 1983, DIS 59: 110 (fig.).

phenotype: Homozygotes when raised at 19° show occasional absence of one or both claws; veins L4 and L5 interrupted; fertile at 19° but become sterile when shifted to 28°; produce embryos with range of germ band abnormalities. Homozygous pupal lethal when raised at 28°; pharate adults show defective tarsal development of all six legs; condensed, poorly developed and curved metatarsus and tarsi; duplications in tibial and tarsal segments; claws absent. Temperature sensitive period first instar to early pupa.

Aph-1: Alkaline phosphatase-1

location: 3-47.3 (between *W* and *p*) (Wallis and Fox).

references: Beckman and Johnson, 1964, Nature 201: 321 (fig.).

1964, Genetics 49: 829-35 (fig.).

Wallis and Fox, 1969, Biochem. Genet. 2: 141-58.

phenotype: Locus responsible for one of several different alkaline phosphatase species [APH1 (EC 3.1.3.1)] formed during the life cycle. Specifies the enzyme that becomes active in the larval cuticle and muscle during the third instar. Electrophoretic mobility of a pupal form of the enzyme, which differs from that found in the larva, also appears to be controlled by this locus (Wallis and Fox). Dimeric nature of enzyme inferred from the presence of enzymes of hybrid mobility in larvae heterozygous for electrophoretic variants. Biochemical characterization of larval enzyme by Harper and Armstrong (1972, Biochem. Genet. 6: 75-82; 1973, Biochem. Genet. 10: 29-38; 1974, Biochem. Genet. 11: 177-80).

alleles: Naturally occurring alleles superscripted *F* and *S* reported by Beckman. Wallis and Fox describe *Aph^A* which specifies larval enzyme migrating faster than *Aph^F*, but a pupal enzyme with same characteristics as that produced by *Aph^F*. *Aph⁰* reported by Johnson (1966, Science 152: 361-62) produces no detectable enzyme activity but causes the appearance in extracts of *Aph^S/Aph⁰* larvae of a band migrating slightly faster than the hybrid band produced by *Aph^F/Aph^S* larvae. Naturally occurring alleles superscripted 2, 4, 6, and 10 characterized by Harper and Armstrong (1972, 1973, 1974); 4 is synonymous with *F* as is 6 with *S*; 2 migrates more slowly than *S* and 10 more rapidly than *F*; not clear that 10 and *A* are different. That the larval and pupal enzymes are differently modified products of the same locus is indicated by genetic inseparability and by concordance in the orders of mobilities of electrophoretic alleles (Wallis and Fox).

Aph-2

location: 2- not mapped.

references: Schneiderman, Young, and Childs, 1966, Science 151: 461-63.

phenotype: The alkaline phosphatase found in adult hindgut.

alleles: Two different alleles recorded superscripted *A* and *B*. Enzyme produced by *Aph-2^A* homozygotes migrates more rapidly than that produced by *Aph-2^B* homozygotes; enzyme produced by *Aph-2^A/Aph-2^B* has same mobility as that produced by *Aph-2^A* homozygotes.

apo: altered pattern orientation (J.C. Hall)

location: 1- (not localized).

origin: Induced by ethyl methanesulfonate.

synonym: *apo^{SI29}*.

references: Heisenberg, 1979, Handbook of Sensory Physiology (H. Autrum, ed.). Springer-Verlag, Berlin, Vol. VII/6A, pp. 665-79.

Bülthoff, 1982, DIS 58: 31.

1982, Biol. Cybernet. 45: 63-70.

phenotype: Poor orientation to objects, including spots in Y-maze test; electroretinogram normal.

app: approximated

location: 3-37.5.

discoverer: Curry, 34a25.

references: 1935, DIS 3: 6.

phenotype: Crossveins close together; veins diverge at greater angle than wild type; effect visible in prepupal wing [Waddington, 1940, J. Genet. 41: 75-139 (fig.)]. Legs short with four-jointed tarsi; the penultimate joint characteristically swollen [Waddington, 1939, Growth Suppl. 37-44 (fig.)]. Joint between second and third tarsal segments often incomplete; invaginations or internalization of cuticle seen in tarsi 1, 3, and 4 (Held, Duarte, and Derakhshanian, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 145-57). Thickset body. Posterior scutellars farther apart than normal. Eyes smaller and flatter than normal, also bumpy. Spread wings; thickened veins. RK1.

alleles: *app^{61e}* (CP627).

cytology: Placed in 69A2-4 on the basis of its inclusion in $Df(3L)vin6 = Df(3L)68C8-11;69A4-5$ but not $Df(3L)vin5 = Df(3L)68A3;69A1-2$ (Akam, Roberts, Richards, and Ashburner, 1978, Cell 13: 215-26).

Appl: β -Amyloid protein precursor like (K. White; J.C. Hall)

Location: 1-{0}.

origin: Isolated as cDNA clones derived from cloned genomic DNA in the 1B division.

references: Rosen, Martin-Morris, Luo, and White, 1989, Proc. Nat. Acad. Sci. USA 86: 2478-82.

Martin-Morris, and White, 1990, Dev. (In press).

molecular biology: A 6.5 kb transcript corresponding to the cDNA clones encodes a polypeptide that is conceptually an 886 amino acid transmembrane protein; this predicted amino-acid sequence shows strong homology in certain of its regions to the β -amyloid protein precursor protein of humans (Rosen *et al.*, 1989). Two forms of the actual protein, which is N-glycosylated, are detectable (in studies involving extracts, primary cultures, and transfected cells); an 145 kd membrane-associated precursor and a 130 kd secreted form lacking the

cytoplasmic domain inferred from sequencing (Luo, Martin-Morris, and White, 1990, J. Neurosci., in press). The source of the *Appl* transcript spans ca. 38 kb of genomic DNA; this RNA localizes to post-mitotic neurons (and apparently not to non-neuronal tissues) in all developmental stages and in adults (Martin-Morris and White, 1990). Consistent with these *in situ* hybridization data are those showing APPL protein immunoreactivity in developing neurons, concomitant with axonogenesis; this staining remains associated with differentiated neuronal cell bodies and axonal tracts (including neuropil regions) in embryos, APPL immunoreactivity is observed exclusively in post-mitotic CNS and PNS neurons (Luo *et al.*, 1990).

other information: The APPL-encoding gene initially suggested (Rosen *et al.*, 1989) to correspond to *vnd* (which was defined originally by embryonic neural-lethal mutations). This has been disproved, in that a terminal deletion *Df(1)78* which retains *vnd*⁺ function removes most of the *Appl* coding sequences (Martin-Morris and White).

apr: see *w*^a

Appt: Adenine phosphoribosyltransferase

location: 3-1.49 (0.13 cM to the right of *R*; estimated by Johnson and Friedman to be 3.03 units from the tip of *3L*).

synonym: *aprt*.

references: Johnson and Friedman, 1981, Science 212: 1035-36.

1983, Proc. Nat. Acad. Sci. USA 80: 2990-94.

phenotype: Is the structural gene for adenine phosphoribosyltransferase [APRT, AMP: pyrophosphate phosphoribosyltransferase (EC 2.4.2.7)], a homodimer with 23,000 dalton subunits. It is a purine salvage enzyme which catalyzes the synthesis of AMP from 5-phosphoribosyl-1-pyrophosphate. Flies homozygous for a null allele *Appt*¹ survive on 15 times the concentration of purine that wild type tolerates and show about 2% wild-type enzyme activity; *Appt*¹/₊ exhibit about half wild-type activity. *Appt*² has 9% normal enzyme activity. The dosage response suggests that the mutant affects the structural gene for APRT.

alleles: Electrophoretic variants *Appt*^A (more acidic) and *Appt*^B (more basic) in wild-type stocks Oregon R and Canton S, respectively. *Appt*¹ (Duck), *Appt*² and *Appt*³ (Gelbart and Chovnick) induced by ethyl methanesulfonate; *Appt*⁴ and *Appt*⁵ selected on purine food by Johnson and Friedman (1983).

cytology: Placed in 62B8-12 based on its inclusion in the region of overlap of *Df(3L)R-G7 = Df(3L)62B8-9;62F2-5* and *Df(3L)R-G2 = Df(3L)62B2-4;62B11-12* (Sliter, Henrich, Tucker, and Gilbert, 1989, Genetics 123: 327-36). *in situ* hybridization identifies 62B9 as the site of *Appt* (Johnson *et al.*).

molecular biology: Genomic clone isolated by chromosomally walking from sequences isolated and cloned by microdissection of region 62B from polytene chromosomes. Gene recognized by hybrid selection of an 1-kb mRNA that translated an APRT product. cDNA's have a common 5' initiation site but two different 3' polyadenylation sites. The primary transcript contains two introns, the first of which has alternative 5' sites, which are spliced to the same 3' site; one product encodes the

functional enzyme and the other a prematurely terminated and presumably nonfunctional polypeptide (Johnson and Henikoff, 1989, Mol. Cell Biol. 9: 2220-23). Conceptual amino-acid sequence predicts a polypeptide of 194 amino acids and about 20 kd in molecular weight. *Drosophila* APRT amino-acid sequence displays approximately 40% identity and nearly 80% homology with all known APRT proteins (Johnson, Edström, Burnett, and Friedman, 1987, Gene 59: 77-86).

***Apt: Apart**

location: 3- (between *h* and *p*).

origin: X ray induced.

discoverer: Belgovsky, 34e23.

references: 1935, DIS 3: 27.

phenotype: Wings spread widely. Viability, fertility, and separability good. Homozygous lethal. RK2A.

cytology: Associated with *In(3L)Apt* — no salivary analysis.

other information: *Apt/D* survive; therefore not an allele of *D*.

apterblister: see *apb*

apterous: see *ap*

Apurinic endonuclease: see *Ape*

***apx: apexless**

location: 1-11.3.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)aminophenylalanine.

discoverer: Fahmy, 1954.

references: 1959, DIS 33: 83.

phenotype: Slightly larger fly with large eyes containing various numbers of deranged ommatidia. Wings broad and blunt; in many flies, margin removed to various degrees, from a small incision of inner margin to removal of entire inner margin, costal vein, and parts of the membrane as far as L3. Region from L3 to costal cell unaffected. Rarely L4 and 5 are interrupted. Males viable and fertile; female fertility reduced. RK3.

Apx: Antennapedex (R.E. Denell)

location: 1-70 (said to map 12 units to the right of *B*).

origin: Neutron induced.

references: Ginter, 1969, DIS 44: 50.

phenotype: Males and heterozygous females show variable expression from small additional segment on the third antennal segment to a nearly complete leg including femur, tibia, and tarsus. Arista usually present. Homozygous females lethal but *X0* males survive. Crosses involving either *Apx* males or females produce many inviable embryos.

cytology: Polytene X appears normal, but genetic results suggest a *T(1;3)* with breakpoints in the proximal part of *Xh* and at *Antp*.

Apx-2: see *Antp*

ar: abdomen rotatum

location: 4- (proximal to *bt*; Fung and Stern, 1951, Proc. Nat. Acad. Sci. USA 37: 403-4.

origin: Spontaneous.

discoverer: Beliajeff, 1926.

references: 1931, Biol. Zentralbl. 51: 701-8 (fig.). Bridges, 1935, Biol. Zh. 4: 401-20.

Marengo and Howland, 1942, *Genetics* 27: 604-11 (fig.).

phenotype: Abdomen twisted clockwise through 45° to 60°. No overlapping with wild type. Male external genitalia often missing. Males usually sterile; females partially fertile. Puparia not so smooth as normal; larval segmentation remains. Puparia have deep constriction near posterior end just anterior to spiracles. Existing chromosomes marked *ar* also carry *l(4)* and, in combination with *Df(4)M*, show counterclockwise rotation of male abdomen when viewed from behind (Hochman). RK2.

alleles: *ar*², **ar*^{57d}, and **ar*^{57g} X ray induced (CP627). Ethyl methanesulfonate induced alleles superscripted *65f*, *65h*, *68i*, and *69g* described by Hochman (1971, *Genetics* 67: 235-52; 1972, *DIS* 48: 17). *ar*⁶⁸ⁱ shows best viability and fertility.

cytology: Placed in salivary chromosome region 101E through 102B16 on basis of its inclusion in *Df(4)M* = *Df(4)101E-F;102B6-17*.

Ar: see *Antp*^{LC}

arc: see *a*

arch: *arch*

location: 2-60.5.

origin: Spontaneous.

discoverer: Curry, 36g3.

references: 1937, *DIS* 7: 5.

phenotype: Wings curved evenly downward, both longitudinally and transversely; sometimes shorter and blunter; rarely divergent. RK2.

arclike wing: see *alw*

arcoid: see *ad*

arctops: see *at*

arctus oculus: see *at*

ard: see *Acr64B*

aret: *arrest* (*T. Schüpbach and E. Wieschaus*)

location: 2-48.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus.

phenotype: Female sterile; homozygous females often have underdeveloped ovaries which seem to lack germ cells altogether. In some females a small number of developing egg chambers is found. These never develop beyond the first few stages of oogenesis.

alleles: Eight, *aret*^{WQ} = *aret*^l, *aret*^{WH}, *aret*^{PA}, *aret*^{PC}, *aret*^{PD}, *aret*^{PE}, *aret*^{QB}, *aret*^{RM}. Females homozygous for *aret*^{PC} and *aret*^{QB} usually have a number of egg chambers in their ovaries in which the nurse cells and oocyte never seem to develop beyond stage 2 or 3 of oogenesis, but the follicle cells nevertheless synthesize a tiny round chorion around the cysts. Females homozygous for *aret*^{PA}, *aret*^{PD} and *aret*^{RM} usually have almost normal numbers of early stages of egg chambers in their ovaries which degenerated before yolk uptake occurs.

cytology: Placed in 33B3-F2, since uncovered by *Df(2L)Prl* = *Df(2L)32F1-2;33F1-2* and *Df(2L)prd1.7* = *Df(2L)33B2-3;34A1-2*.

Argentine Curly: see *Cu*^A

Argk: *Arginine kinase*

location: 3-25.2.

references: Fu and Collier, 1981, *Genetics* 97: 537-38.

1983, *Bull. Inst. Zoo. Acad. Sinica* 22: 25.

James and Collier, 1988, *J. Exp. Zool.* 248: 185-91.

phenotype: Structural gene for arginine kinase [ARGK (EC 2.7.3.3)] based on gene dosage studies. Cellular and mitochondrial forms behave as if both are products of the gene. (Munneke and Collier, 1985, *Genetics* 110: s85). In 108-hour third-instar larvae, activity is high in body-wall and digestive-tract musculature; lower in brain, imaginal discs, and salivary glands; present in head, thorax, and abdomen of adults, being highest in indirect flight muscle of thorax. Activity levels increase during development reaching a peak at the pupal stage; then an abrupt decrease during the pupal stage is followed by a second increase beginning around the time of eclosion and climbing to high adult levels (James and Collier).

cytology: Located to 66B by segmental aneuploidy.

Arista: see *Ata*

aristaless: see *al*

aristaless-b: see *aa*

Aristapedia: see *Antp*^{LC}

Aristapedioid: see *Arp*

arm: *armadillo*

location: 1-1.2.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Voelhard, Wieschaus, and Jürgens, 1982, *Verh. Dtsch. Zool. Ges.* 1982: 91-104.

Gergen and Wieschaus, 1986, *Wilhelm Roux's Arch. Dev. Biol.* 195: 49-62.

Klingsmith, Noll, and Perrimon, 1989, *Dev. Biol.* 134: 130-45.

phenotype: Homozygous lethal; embryonic segmentation defective by time of germ-band shortening; naked cuticle ordinarily comprising the posterior two thirds of each segment replaced by mirror-image duplication of the anteriorly situated denticle belt; strong alleles delete first denticle row in abdominal segments. May have dorsal hole in cuticle. Embryonic CNS development quasi normal (Patel, Schafer, Goodman, and Holmgren, 1989, *Genes Dev.* 3: 890-904). Autonomous at the level of single cells as shown by denticulate clones of homozygous cells in the naked cuticle of abdominal segments in *arm*⁺ embryos (Wieschaus and Riggelman, 1987, *Cell* 49: 177-84). Clones of homozygous female germ cells arrested at stage 10 of oogenesis (Wieschaus and Noell, 1986, *Wilhelm Roux's Arch. Dev. Biol.* 195: 63-73). An exception is *arm*⁸ for which progeny from homozygous germ-line clones have been recovered (Klingsmith *et al.*). Cell lethal in imaginal discs; although clones of homozygous cells not observed in adults, their formation seems to engender mirror-image duplications, which are not seen in response to homozygosing other cuticular cell lethals (Wieschaus). Transcript found with minor fluctuations in amount, in all cell types at all stages in development (Riggelman, Wieschaus, and Schedl, 1989, *Genes Dev.* 3: 96-113).

alleles:

allele	origin	synonym	ref ^α	comments
arm 1	EMS	<i>arm XK22</i>	3	strong allele
arm 2	EMS	<i>arm XM19</i>	3	hypomorphic allele
arm 3	EMS	<i>arm XP33</i>	3	strong allele
arm 4	EMS	<i>arm YD35</i>	3	strong allele
arm 5	P	<i>arm TD5</i>	2	1.2-kb P insert
arm 6	P	<i>arm 18.3</i>	2	1.3-kb P insert
arm 7	P	<i>arm TD5</i>	2	complex molecular rearrangement
arm 8	EMS	<i>arm H8.6</i>	1	temperature-sensitive allele

^α 1 = Klingsmith, Noll, and Perrimon, 1989, Dev. Biol. 134: 130-45; 2 = Riggleman, Wieschaus, and Schedl, 1989, Genes Dev. 3: 96-113; 3 = Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307.

cytology: Located in region 2B15 by *in situ* hybridization.

Complementation with other lethals in 2B apparently not tested.

molecular biology: Gene isolated by transposon tagging and identified by germ-line transformation (Klingsmith, Noll, and Perrimon, 1989, Dev. Biol. 134: 130-45). Genomic sequences identify a single 3.2-kb transcript; two classes of cDNA's isolated which have different first exons spliced to six common exons; the first exons are 500 base pairs apart, so that exon 1 of one transcript is within the first intron of the other; they also show slightly different 3' polyadenylation sites. The mature transcript contains an open reading frame of 2529 nucleotides, which encodes a predicted 843-amino-acid, 91.1-kd acidic protein with an isoelectric point of 5.86. The conceptual amino-acid sequence contains no indication of either a signal or a transmembrane sequence; it does contain a 23-amino-acid glycine-rich sequence, which lacks charged amino acids, near the C terminus. In addition, internally there are 12.5 tandem repeats of a 42-amino-acid sequence with a consensus sequence to which the various repeats show 28 to 80% identity.

arp-1: see *ss^{asp}*

Arp: Aristapedloid (P. Adler)

location: 2-67 (inseparable from *vg*)

origin: Hybrid dysgenesis.

references: Adler, 1984, Genetics 107: s1.

Adler, Charlton, and Brunk, 1989, Dev. Genet. 10: 249-60.

Brunk and Adler, 1990, Genetics 124: 145-56.

phenotype: Homozygous lethal. Two dramatic dominant phenotypes: One is a partial transformation of arista to tarsus, the other is the loss or reduction in size of medially located macrochaetae on the dorsal thorax. Both phenotypes show high penetrance but variable expressivity.

alleles: *Arp¹* and *Arp²*.

cytology: Both alleles associated with *In(2R)49A12-B3;49E5-F1* with P-derived sequences at either end. Deficiency analysis shows that lethality common to the two alleles associated with the 49A12-B3 breakpoint and thus localizes *Arp*. Both alleles revert by reinversion in dysgenic (1/200) and control (1/2000) crosses.

other information: Concluded to be homeotic gain-of-function alleles of *Su(z)2* (Brunk and Adler).

arr: arrow

location: 2-66.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82 (fig.).

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Embryonic lethal. Additional denticle bands anterior and posterior to normal denticle bands, especially in ventral midline. Strongest at 18°.

alleles: Nine ethyl-methanesulfonate-induced alleles; *arr¹* and *arr²* (recovered as *IB* and *IIV*) retained.

Arr1: Arrestin 1

location: 2-{53}.

references: Smith, Shieh, and Zuker, 1990, Proc. Nat. Acad. Sci. USA 87: 1103-07.

Hyde, Mecklenburg, Pollock, Vihtelic, and Benzer, 1990, Proc. Nat. Acad. Sci. USA 87: 1108-12.

LeVine, Smith, Whitney, Malicki, Dolph, Smith, Burkhart, and Zuker, 1990.

phenotype: Encodes a Drosophila homologue of mammalian arrestin, a protein that interacts stoichiometrically with activated rhodopsin, inhibiting its ability to interact with the G protein, transducin, thus terminating the visual response. ARR1 presumed to be the 41-kd protein that is phosphorylated by exposure to light in the presence but not the absence (i.e., *ninaE* flies) of rhodopsin. Expression first detected in late pupae, when other genes involved in phototransduction are also first expressed. Transcript localized to photoreceptor cells of the compound eye, the ocelli, and the larval light sensitive organ.

cytology: Placed in 36D1-2 by *in situ* hybridization.

molecular biology: Sequence isolated from an eye specific genomic library produced by subtractive hybridization with cDNA from body and *eya* flies. The genomic clone identifies an 1.4-1.5-kb transcript in late pupae. Sequencing of genomic and cDNA clones indicates a primary transcript with four exons separated by introns of 421, 233, and 60 nucleotides; the conceptual amino-acid sequence indicates a protein of 364 residues that displays 42-45% amino-acid identity with bovine and human arrestins. Drosophila arrestin, however, lacks the 30 C-terminal amino acids, which share homology with α -transducin in human and bovine arrestins.

Arr2

location: 3-{26}.

references: Yamada, Takeuchi, Komori, Kobayashi, Sakai, Hotta, and Matsumoto, 1990, Science 248: 483-86.

Levine, Smith, Whitney, Malicki, Dolph, Smith, Burkhart, and Zuker, 1990.

phenotype: Encodes a second Drosophila homologue of mammalian arrestin, a 49-kd (or 46-kd) protein that is phosphorylated by exposure to light in wild type but not in *ninaE* flies which lack rhodopsin in photoreceptor cells 1-6, nor *norpA* flies which are deficient in phospholipase C activity. Phosphorylation dependent on the presence of Ca^{++} , whose intracellular levels are regulated by phospholipase C. Expression first detected in late pupae, when other genes involved in phototransduction are also first expressed. *Arr2* expression is approximately seven times that of *Arr1*. In the adult, transcript localized to photoreceptor cells of the compound eye and the ocelli.

cytology: Placed in 66D10-11 by *in situ* hybridization

(Levine *et al.*).

molecular biology: Sequence isolated by screening an expression library with a monoclonal antibody raised to the *Drosophila* 49-kd protein (Yamada *et al.*) or with a synthetic oligonucleotide based on partial amino-acid sequence (LeVine *et al.*). Also apparently isolated as autonomous head-specific clones by Levy, Ganguly, Ganguly, and Manning (1982, *Dev. Biol.* 94: 451-64). The sequence identifies a single transcript of 1.8 kb (or 1.65 kb) on Northern blots; the conceptual amino-acid sequence indicates a protein of 401 amino acids, a calculated molecular mass of 44,972 daltons, and an isoelectric point of 8.9. ARR2 shares 206 residues with ARR1, and 181 with bovine arrestin; 146 residues are conserved in all three polypeptides. ARR2 shares with bovine arrestin virtually identical hydropathy plots and potential glycosylation sites but differs in being basic (pI = 8.9) rather than an acidic (pI = 6.0). The carboxy terminus of ARR2 displays only partial homology to the sequence of the proposed rhodopsin binding site of α -transducin; also, the arrestin sequences that resemble the adenosine diphosphate- ribosylation sites of transducin are not found in ARR2.

Ars: Arylsulfatase

references: MacIntyre, 1974, *Isozyme Bul.* 7: 23-24.

phenotype: Inferred as structural gene for arylsulfatase [ARS (EC 3.1.6.1)] on basis of response of enzyme level to gene dosage.

cytology: Placed in 74A-79D by segmental aneuploidy.

art: aristatarsia

location: 3- (not mapped; modifying factors on chromosome 2).

origin: Spontaneous.

discoverer: Ouweneel, 69e.

references: 1970, *DIS* 45: 35.

phenotype: In homozygotes, arista replaced by tarsus-like structure. Penetrance more than 70%. Complements *ss*^a but enhances *Antp*^B in heterozygous condition; penetrance of *Antp*^B/+ less than 1%, of *Antp*^B/*art* more than 60%; *Antp*⁴⁹ also enhanced.

arth: arthritic

location: 1-0.0 [between *ewg* and *y* (Fleming)].

references: Schalet and Roberts, 1973, *DIS* 50: 23.

phenotype: Legs weak with pigmented joints; tarsal segments frequently askew with claws fused; movements somewhat uncoordinated. Brownish-black pigment present at joints of over 90% of males, most frequently in meso- and metathoracic legs between femur and tibia but sometimes between coxa and trochanter or proximal to coxa.

cytology: Placed in 1A5-8 based on arthritic phenotype of males carrying *Df(1)y15 = Df(1)1A4-5;1A8-B1* in combination with *l(1)1Ar*^Y. Included in *Df(1)sc*¹⁹, whereas *ewg* is not (Schalet).

as: ascute

location: 3-46.

origin: Spontaneous.

discoverer: Bridges, 16j21.

references: Bridges and Morgan, 1923, *Carnegie Inst. Washington Publ.* No. 327: 170.

phenotype: In *as*¹, front of scutellum elevated, with partial obliteration of transverse furrow; deep chested. Bub-

ble in scutellum or midline of thorax; dried black exudate, often at each side of scutellum, may appear at any of the sutures of head and thorax; black deformed lump behind cheek. Wings droop at sides. Overlaps wild type. RK3.

alleles: **as*¹, **as*² (CP627), *as*^{hg}.

cytology: Placed in region 72D3-73A5 by Velissariou and Ashburner, 1981, *Chromosoma* 84: 173-85.

as^{hg}: ascute-hängende

origin: Spontaneous.

references: Franke, 1934, *DIS* 2: 9.

Gottschewski, 1935, *DIS* 4: 15.

phenotype: Wings held laterally downward, ends occasionally resting on legs; eyes small and knobby. RK2.

ASC: achaete-scute complex

location: 1-0.0.

references: García-Bellido, 1979, *Genetics* 91: 491-520.

Carramolino, Ruiz-Gómez, Guerrero, Campuzano, and Modolell, 1982, *EMBO J.* 1: 1185-91.

Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, *Cell* 40: 327-38.

Dambly-Chaudière and Ghysen, 1987, *Genes Dev.* 1: 297-306.

Ghysen and Dambly-Chaudière, 1988, *Genes Dev.* 2: 495-501.

phenotype: The achaete-scute complex (ASC) produces a number of transcripts whose translation gene products function in the specification of diverse sensilla and their centrally projecting neurons. Insertions of transposable elements and rearrangement breakpoints in ASC result in the loss of certain subsets of bristles (both macrochaetae and microchaetae) and other sensilla, including their associated accessory cells, such as those forming sockets and peripheral neurons; these lesions, with few exceptions, occur outside of transcribed regions. The complex was subdivided into four components based on the sites of lesions causing mutant phenotypes by García-Bellido (1979); they are, from left to right, *ac*, *sc α* , *l(1)sc*, and *sc β* ; a fifth component, *sc γ* , has been identified by Dambly-Chaudière and Ghysen (see also Ghysen and Dambly-Chaudière). Four genes that function in neurogenesis, and thus comprise ASC, are *ac*, *sc* (in the *sc α* region), *l(1)sc*, and *ase* (in the *sc γ* region); some lesions in *sc* affect sex determination and are currently denoted *sis-b*. *ac*, and *ase* function in the genesis of both larval and adult peripheral nervous-system elements; *l(1)sc* functions in central-nervous-system development. *Hw* alleles are dominant gain-of-function mutations in ASC; unlike loss of function mutations, at least two *Hw* mutations have foreign sequences inserted into structural genes of ASC (Campuzano, Balcells, Villares, Carramolino, García-Alonzo, and Modolell, 1986, *Cell* 44: 303-12). Deficiencies for most regions of the complex are hemizygous and homozygous viable; however, deficiency for *l(1)sc* is lethal. Interestingly, *sc* rearrangements with breakpoints in the *sc α* region have their second breakpoints in pericentric heterochromatin, whereas those broken in the *sc β* region have euchromatic second breakpoints (García-Bellido, 1979). Different segments of ASC tend to affect different subsets of bristles in the adult, as will be described in entries to follow; detailed descriptions of these bristle patterns are given by Dubinin (1933, *J. Genet.* 27: 446) and García-Bellido (1979). ASC

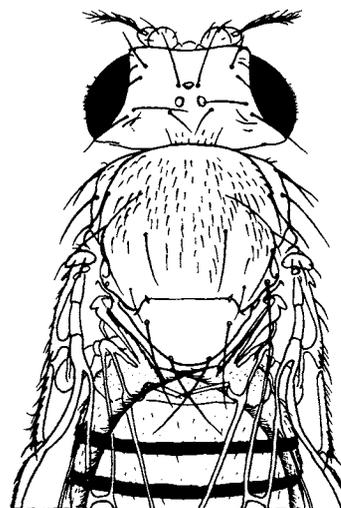
deficiencies also result in absences of campaniform and trichoid sensilla (García-Bellido and Santamaria, 1978, *Genetics* 88: 469-86). In larvae, *ASC* specifies neurons innervating peripheral sensory organs plus another class of neurons of uncertain (perhaps proprioceptive) function, but not chordotonal-organ-innervating neurons; here too, different components of *ASC* specify different and sometimes overlapping subsets of pattern elements (Dambly-Chaudière and Ghysen). Double hemizygotes for *ASC* and *da* [*Df(1)260-1/+; Df(2L)J27/+*] or *ASC* and *Df(4)M101-62f* show loss of macrochaetae, which none of the single hemizygotes does (Dambly-Chaudière, Ghysen, Jan, and Jan, 1988, *Roux's Arch. Dev. Biol.* 197: 419-23). Homozygous *ASC* deficiencies tend to counteract the embryonic effects of deficiencies for the neurogenic loci; *i.e.*, they cause a partial return to epidermigenesis (Brand and Campos-Ortega, 1988, *Roux's Arch. Dev. Biol.* 197: 447-56). In the following treatment of *ASC*, *ac*, *ase*, *Hw*, *l(1)sc*, *sc*, and *sis-b* are discussed in turn; a general discussion of phenotype of each element is followed by a table of alleles, after which specific aspects of various alleles are described.

cytology: Placed in 1B1-7 based on its being positioned to the right of the left breakpoint of $ln(1)y^{3P} = ln(1)1B1-2;20F$ and to the left of the left breakpoint of $Tp(1;2)sc^9 = Tp(1;2)1B1-2;1B4-7;25-26$. The region has been subdivided by rearrangement breakpoints in the following order: $ln(1)y^{3P}$, $ln(1)y^4$, *ac*, $ln(1)sc^8$, *sc α* , $ln(1)sc^{L8}$, $ln(1)sc^{S1}$, $ln(1)sc^4$, *l(1)sc*, $ln(1)sc^9$, *sc β* , *Df(1)sc19*, *sc γ* , *Df(1)260.1*.

molecular biology: Most of the complex has been cloned and restriction mapped in a chromosome walk of over 100 kb (Campuzano *et al.*); insertions and rearrangement breakpoints associated with different *sc* mutations located on restriction map. The coordinate system designates 0 as an arbitrarily chosen *EcoRI* site in the *sc β* region with positive values extending to the left (Carramolino, Ruiz-Gómez, Guererro, Campuzano, and Modolell, 1982, *EMBO J.* 1: 1185-92). Sequence variation in *ASC* region of chromosomes isolated from natural populations investigated by Aguadé, Miyashita, and Langley (1989, *Genetics* 22: 607-15). Nine different transcription units, originally designated T1 to T9, identified in the *ASC* region. Four of these, T5, T4, T3, and T8 (formerly T1a), appear to be responsible for the neurogenic functions of the complex and correspond to *ac*, *sc*, *l(1)sc*, and *ase*, respectively; they are transcribed from left to right and encode members of the helix-loop-helix class of proteins, which are able to bind, as dimers, to DNA. In addition, the *ac*, *sc*, and *l(1)sc* transcripts share a 15-nucleotide acidic C-terminal sequence (Villares and Cabrera, 1987, *Cell* 50: 415-24; Alonso and Cabrera, 1988, *EMBO J.* 7: 2585-91; González, Romani, Cubas, Modolell, and Campuzano, 1989, *EMBO J.* 8: 3553-62). Transcription unit T2 is found in the *sc β* region; its protein product and spatial pattern of expression rule out a neurogenic function and therefore membership in *ASC*.

ac: achaete

phenotype: *ac* specifies the formation of the anterior and posterior dorsocentral, the posterior supra-alar (as does *sc*), the anterior vertical bristle, and in addition the acrostichal rows of microchaetae on the notum. Absence of bristles accompanied by absence of associated socket and



ac: achaete

From Bridges and Brehme, 1944, Carnegie Inst. Washington Publ. No. 552: 12.

underlying centrally projecting neuron (Stern, 1938, *Genetics* 23: 172-73). In addition mutant alleles of *ac* tend to remove the interocellar hairs and the hairs on the surface of the eye and a restricted subset of the campaniform sensilla on the wing blade (Leyns, Dambly-Chaudière and Ghysen, 1989, *Roux's Arch. Dev. Biol.* 198: 227-32). Trichomes are not affected. *ac* deficiencies, *e.g.*, $ln(1)y^{3PL}sc^{8R}$, survive as fully mobile and fertile adults (García-Bellido, 1979, *Genetics* 90: 491-529). A series of terminal deficiencies approaching the *ac* coding sequence from the left a few hundred base pairs at a time, when tested in heterozygotes with $ln(1)y^{3PL}sc^{8R}$ or *Df(1)sc19*, cause, with few exceptions, progressive loss of chaetae as the amount of deleted material increases; response of anterior verticals erratic. First effects of deficiencies noted with chromosomes broken 10 kb upstream of the transcription start site. Despite loss of most of the DNA upstream from the transcribed region, the phenotypes associated with these deletions still suppressed by *emc* and *h* (Ruiz-Gómez and Modolell, 1987, *Genes Dev.* 1: 1238-46). Deficiencies for *ac* act as suppressors of *h* (Sturtevant, 1970, *Dev. Biol.* 21: 48-63), whereas extra doses of ac^+ enhance expression of *h* (Moscoso del Prado and García-Bellido, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 242-51). Longitudinal stripes of expression on either side of the midline during gastrulation become internalized and segmented into four longitudinal rows of clusters of expressing cells at half-segment intervals. *ac* RNA undetectable in germ band at time of germ-band shortening. Several regions of high expression seen in cephalic region. Also expressed in posterior midgut rudiment (Romani, Campuzano, and Modolell, 1987, *EMBO J.* 6: 2085-92; Cabrera, Martínez-Arias, and Bate, 1987, *Cell* 50: 425-33). In third-instar larvae, expression in wing imaginal disks restricted to regions where precursors of cuticular organs specified by *ac* are known to reside (Romani, Campuzano, Macagno, and Modolell, 1989, *Genes Dev.* 3: 997-1007).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
ac¹	spont	Weinstein, 1918	<i>sc¹¹</i>	8	deletion for kb 84 to 64; includes <i>y³</i>
*ac²	X ray	Dubinin, 1929		2, 6	induced with <i>sc³</i>
ac³	X ray	Dubinin, 1929	<i>sc¹⁰, sc¹¹</i>	3, 4, 5	<i>In(1)1B2-3;1B14-C1</i> ; kb 59.6 to 58.8
ac^{3B}				1	<i>gypsy</i> at kb +63.7
*ac⁴	X ray	Dubinin, 1929	<i>ac³</i>	3, 4, 5	with <i>y^{54e}</i> in <i>y⁺ y</i>
ac^{54e}	X ray	W. K. Baker			
ac²⁶⁰⁻²⁸	X ray	Sutton, 1916		7	

^α 1 = Campuzano, Carramolino, Cabrera, Ruíz-Gómez, Villares, Boronat, and Modolell, 1985, Cell 40: 327-38; 2 = Dubinin, 1929, Biol. Zentr. 49: 328-39; 3 = Dubinin, 1930, Zh. Eksperim. Biol. 6: 300-46; 4 = Dubinin, 1932, J. Genet. 25: 163-81; 5 = Dubinin, 1933, J. Genet. 27: 443-64; 6 = Serebrovsky and Dubinin, 1930, J. Hered. 21: 259-65 7 = Sutton, 1943, Genetics 28: 210-17; 8 = Weinstein, 1918, Genetics 3: 133-72.

cytology: Placed in 1B1-2 based on its position between left breakpoints of *In(1)y⁴ = In(1)1A8-B1;18A3-4* and *In(1)sc⁸ = In(1)1B2-3;20F*.

molecular biology: The *ac* transcription unit, designated T5 by Campuzano *et al.* is located from kb 59 to 58, and transcription is from left to right. cDNA and genomic sequences of the putative *ac* transcribed region determined by Villares and Cabrera (1987, Cell 50: 415-24); the transcription unit of 911 nucleotides is without introns and specifies a 201-amino-acid polypeptide of 22.7 kd.

ac¹

phenotype: Hypomorphic; phenotype of homozygous females weaker than in hemizygous females (García-Bellido, 1979, Genetics 91: 491-520). Posterior dorso-central bristles missing; also posterior supra-alar and anterior vertical bristles frequently missing. Anterior dorso-centrals displaced anteriorly (Claxton, 1969, Genetics 63: 883-96). García-Bellido (1979) finds anterior dorso-central bristles more strongly decreased than posterior dorso-centrals. Hairs usually fewer near position of posterior dorso-centrals; interocellar hairs invariably fewer, typically absent. Eyes partly devoid of hairs. Trichomes unaffected. Limited nonautonomy near the borders of somatic spots with respect both to numbers and positions of bristles and hairs (Stern, 1954, Am. Sci. 42: 212-47; Roberts, 1961, Genetics 46: 1241-43; Claxton, 1976, Genet. Res. 27: 11-22). *ac* partially suppresses *h*; *Hw/ac* = *Hw*/+ (Sturtevant, 1969).

molecular biology: A deficiency extending from a point between kb 64.9 and 63.5, which is approximately 5 kb upstream from the presumptive *ac* transcription unit, to the left to approximately kb 82. (Campuzano, Carramolino, Cabrera, Ruíz-Gómez, Villares, Boronat, and Modolell, 1985, Cell 40: 327-38).

***ac²**

phenotype: Since *ac²* and *sc³* were for practical purposes inseparable by crossing over, the effect of *ac²* alone could not be assessed. The double mutant removed all bristles except scutellars and postdorso-centrals. *ac²/ac²* and *ac²/+* suppress *h* (Sturtevant). Viability of males low; females nearly inviable. RK2.

cytology: Salivary chromosomes normal (Schultz).

ac³

synonym: Called *ac²* by Dubinin, the earlier *ac²* with *sc³* having been omitted from the series, *sc¹⁰, sc¹¹* (Sturtevant and Schultz, 1931, Proc. Nat. Acad. Sci. USA 17: 265-70).

phenotype: Posterior and usually anterior dorso-centrals lacking; other bristles wild type. Hairs removed from areas across rear and front edges of thorax, through mid-dorsal area, and between ocelli. *ac³* even in *ac³/+* heterozygotes exerts strong suppression on *h* (Sturtevant, 1970, Dev. Biol. 21: 48-61). RK2A.

cytology: Inseparable from *In(1)ac³ = In(1)1B2-3;1B14-C1* (Muller, Prokofyeva, and Raffel, 1935, Nature (London) 135: 253-55).

molecular biology: Left breakpoint of *In(1)ac³* at DNA coordinate +59 near the transcription start site of the *ac* transcript; transcript levels substantially reduced in both embryos and pupae; the *l(1)sc* transcript but not the *sc* transcript also reduced (Campuzano, Carramolino, Cabrera, Ruíz-Gómez, Villares, Boronat, and Modolell, 1985, Cell 40: 327-38).

ac^{3B}

phenotype: Low level of absence of dorso-central bristles as well as microchaetae. Bristles normally removed by *sc* mutations not missing.

molecular biology: A *gypsy* insert in the *ac* region of *ASC* at +63.7; in addition there is a 0.5 kb insert in the *sc^β* region at approximately -5.0 to -6.5, which shows sequence homology with the *gypsy* LTR.

other information: *ac^{3B}* is a derivative of *sc^{3B}*, which originally displayed a *sc¹*-like phenotype. The original phenotype is no longer present and a weak achaete phenotype appears instead. Apparently a *gypsy* element originally inserted between -5.0 and -6.5 has excised, leaving a 0.5 kb remnant and resulting in reversion of the *sc* phenotype, and a new *gypsy* has inserted at 63.7, about 4 kb upstream from the *ac* transcription unit, to produce the present *ac* phenotype. Level of *ac* transcript reduced. *ac^{3B}* complements *ac¹* (García-Bellido, 1979, Genetics 91: 491-520).

ase: asense

location: 1-0.0.

references: Ghysen and Dambly-Chaudière, 1988, Genes Dev. 2: 495-501.

González, Romani, Cubas, Modolell, and Campuzano, 1989, EMBO J. 8: 3553-62.

phenotype: *ase¹* (formerly *sc²*) shown to be a molecular deletion including the *ase* transcription unit. *ase* embryos lack a subset of peripheral neurons (Dambly-Chaudière and Ghysen, 1987, Genes Dev. 1: 297-306); third-instar larvae show disrupted optic-lobe development; in adults almost all abdominal chaetae are removed as are the extra chaetae induced by *Tft*. Abdomen tends to be swollen; wings poorly expanded; viability of homozygous and hemizygous females low (García-Bellido, 1979, Genetics 91: 491-520). Gene expression first detectable in the neural primordium, presumably in the neuroblasts. In later embryos, RNA is detected in most cells of the CNS primordium as well as in the labrum, optic lobe rudiment, procephalic neurogenic region, and the posterior midgut rudiment. Expression lasts into germ-band retraction. Also expressed sparsely in third-instar larvae; scarce in imaginal disks except for

one large cell cluster in each leg disk; strong in the CNS, especially in a cap of cells over each optic lobe, which are destined to generate ganglion mother cells of the lamina and medulla; expression also seen in inner anlagen of optic lobes, which give rise to cells of the medulla and lobula complex. Otherwise expression in brain and ventral ganglion occurs in isolated clusters of cells and single cells. Expression thought to identify actively proliferating cells (González *et al.*).

cytology: Placed in 1B4-7, the 22-kb region defined as *scy* by Dambly-Chaudière and Ghysen (1987), based on its being included in the apparently terminal deficiency *Df(1)260.1 = Df(1)1B4-6* but in neither *Df(1)sc19 = Df(1)1B1-2;1B4-5* nor in the terminal duplication afforded by the $4^P X^D$ element of *T(1;4)sc^H*.

molecular biology: Both cDNA and genomic clones isolated and sequenced (González *et al.*); 1596 ORF encodes a protein of 486 amino acids; in addition to the helix-loop-helix motif, it contains a centrally located acidic region and a proline-rich region near the N terminus; it lacks the N-terminal acidic domain characteristic of the other three *ASC* transcripts. The protein also contains PEST and *opa* sequences. The genomic sequence is without introns. *ase¹* mutation is a small deletion from coordinates -11.5 to -30 kb that includes the *ase* transcription unit, which is located at approximately coordinate -25 kb.

Hw: Hairy wing

references: Campuzano, Balcells, Villares, Carramolino, García-Alonzo, and Modolell, 1986, Cell 44: 303-312. García-Alonzo and García-Bellido, 1986, Wilhelm Roux's Arch. Dev. Biol. 193: 259-64. Balcells, Modolell, and Ruiz-Gómez, 1988, EMBO J. 7: 3899-3906.

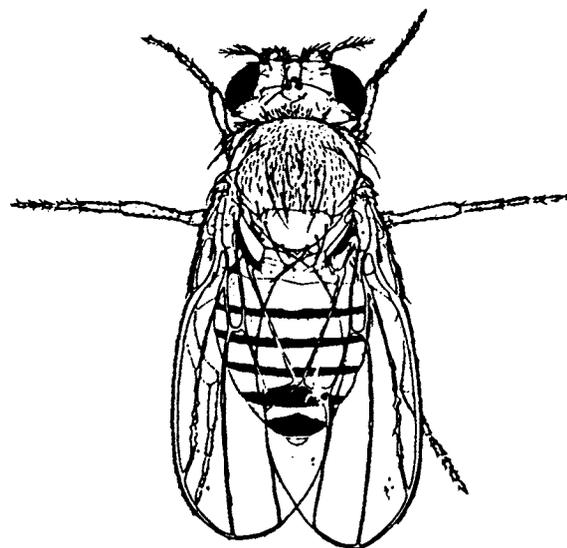
phenotype: Gain of function alleles of *ASC*, which lead to the development of supernumerary bristles and hairs in all segments of the fly: in the prefrons, postfrons, postgena, and occipital regions of the head; in the preepisternum, episternum, anepisternum, scutum, scutellum, postnotum, wingblade, legs, humerus, and halteres of the thorax; and in the tergites, pleura, and sternites of the abdomen. Phenotype suppressed by three doses of *h⁺* (Botas, Moscoso del Prado, and García-Bellido, 1982, EMBO J. 1: 307-10) and enhanced by *h*, *emc*, and *pyd* (Neel, 1941, Genetics 26: 52-58; Moscoso del Prado and García-Bellido, 1984, Wilhelm Roux's Arch Dev. Biol. 193: 242-45). Numbers of super numerary bristles reduced in *da⁺* hemizygotes (Dambly-Chaudière, Ghysen, Jan and Jan, 1988, Roux's Arch Dev. Biol. 97: 419-23).

alleles:

allele	origin	discoverer	ref ^α	comments
<i>Hw¹</i>	spont	Bridges, 23c12	1, 3, 6	in y
<i>Hw²</i>	spont	Nichols-Skoog, 35a9	2	derivative of <i>Hw¹</i>
<i>Hw^{49c}</i>	32 P	King, 49c21	4, 8	with <i>sc^{49c}</i>
<i>Hw^{59g}</i>	X ray	Green	5, 7	in <i>sc¹</i> ; <i>In(1)1B;2B3-4</i>
<i>Hw^{bap}</i>		Cline	9	<i>T(1;2)1B;21B</i>
<i>Hw^{BS}</i>	spont	García-Bellido	1	derivative of <i>Hw¹</i>
<i>Hw^{Ua}</i>	spont	García-Bellido	1, 3	in y ² <i>sc¹</i>

^α 1 = Campuzano, Balcells, Villares, Carramolino, García-Alonzo, and Modolell, 1986, Cell 44: 303-312; 2 = CP627; 3 = García-Alonzo and García-Bellido, 1986, Wilhelm Roux's Arch. Dev. Biol. 193: 259-64; 4 = Gottlieb, 1964, Genetics 49: 739-60; 5 = Green,

1961, Genetics 46: 671-82; 6 = Neel, 1941, Genetics 26: 52-58; 7 = Lee, 1972, DIS 48: 18-19; 8 = Poulson and King, 1949, DIS 23: 62-63; 9 = Roseland and Schneiderman, 1979, Wilhelm Roux's Arch. Dev. Biol. 186: 235-65.



Hw: Hairy wing

Edith M. Wallace, unpublished.

Hw¹

phenotype: Males and heterozygous females have extra bristles on the head (especially occipitals), the notum and the mesopleurae; also extra bristles, including sensory ones and campaniform sensilla (Palka, Schubiger, and Hart, 1981, Nature 294: 447-49), along longitudinal veins and in membrane of wing. Classifiable in a single dose in triploids (Schultz, 1934, DIS 1: 55). Homozygous females more extreme; 110 extra chaetae on wing vs. 49.5 for *Hw¹/+*; 11 on scutellum and postnotum vs. 0.7 in *Hw/+* (García-Bellido and Merriam, 1971, Proc. Nat. Acad. Sci. USA 68: 2222-26). Number of extra bristles inversely correlated with temperature (Ohn and Sheldon, 1970, Genetics 66: 517-40). *Hw¹/Hw¹* females exhibit 40-80% wild-type viability and are agametic steriles; clones of homozygous germinal cells in *Hw/+* females capable of producing progeny (García-Bellido and Robbins, 1983, Genetics 103: 235-47); however, García-Alonzo and García-Bellido claim that their strain is no longer homozygous female sterile. Viability and fertility of *Hw¹/Y* males and *Hw¹/+* females good. *Hw¹/Hw¹* and \pm autonomous in somatic clones until 8 hr before puparium formation; altering cellular genotype after that time is without effect owing to perdurance (García-Bellido and Merriam). X-ray-induced full and partial revertants are frequently mutant for *ac* (García-Alonzo and García-Bellido). RK1 as male or heterozygous female.

cytology: Claimed by Demerec and Hoover (1939, Genetics 24: 68) to be associated with duplication for the 1B1-2 doublet; however restriction mapping of *ASC* fails to confirm this claim (Campuzano *et al.*). Many rearrangements with breakpoints in distal portion of *sc* com-

plex have a weak *Hw* effect, especially as indicated by microchaetae on the mesopleurae; duplications for rearranged scute loci have enhanced effects; duplications of *Hw*⁺ without phenotypic effects except that they suppress *h* (Botas *et al.*).

molecular biology: Carries a *gypsy* insert near the midpoint of the *ac* structural gene (T5) as well as a 2.6 kb insert (*Sancho 2*) located 8.5 kb to the left of the *gypsy*. Transcript 5 shortened from 1.1 kb seen in wild type to 0.9 kb; initiation normal; termination takes place in insert. Developmental Northern blots show peak accumulations in early embryos and early pupae; this transcript, which contains only the 5' half of the normal T5, is still functional in that no *ac* bristles are removed; it is present in many fold excess at all developmental stages examined (Campuzano *et al.*). The distribution of expressing cells in the wing disk is much less localized than in wild type (Balcells *et al.*).

*Hw*²

phenotype: Females homozygous for *Hw*² show only occasional extra hairs along wings. Overlaps wild type. RK3A.

cytology: Salivary chromosome analysis by Schultz (Morgan, Schultz, and Curry, 1941, Year Book - Carnegie Inst. Washington 40: 284) shows small inversion of the region from 1A3 through 1B1 *i.e.*, associated with $In(1)Hw^2 = In(1)1A2-3;1B1-2$.

Hw^{49c}

phenotype: More extreme than *Hw*¹. Homozygous female has doubling and tripling of many bristles, three or four extra dorsocentral bristles per side, extra wing veins, gap in posterior crossvein, and extra hairs on vein L2 and in wing cells. Width of mesonotum in region between dorsocentral bristles increased leading to increased numbers of acrostichal rows as well as extraneous extra microchaetae (Gottlieb, 1964, Genetics 49: 739-60); many lack one or more ocellar or postvertical macrochaetae (Stoddard, 1972, DIS 48: 137-38). Heterozygous female has normal bristles, extra hairs on L2 and L3 and in wing cells, and often an extra free vein from posterior crossvein; also extra acrostichal rows. *Hw*^{49c} male much like homozygous female but bristle duplication less extreme. Low degree of non autonomy reported at junction between *Hw*^{49c}/*Hw*^{49c} and +/- twin spots (Gottlieb). Male and heterozygous female fertile; homozygous female sterile. Revertants of *Hw*^{49c} lose their dominant phenotypes; however they remain *sc* and may exhibit a weak *ac* phenotype or be lethal in combination with *Df(1)sc*¹⁹ (García-Alonzo and García-Bellido). Not suppressed by *su(Hw)*. *Hw*^{49c} and *Oce* act synergistically in removing head bristles but cancel each others effects on thorax in *Hw/Oce* females (Stoddard). *ac*, *sc*, and *l(1)sc* transcripts considerably more abundant than in wild type; also more generally expressed in wing disks than normal (Balcells *et al.*). RK1.

cytology: Associated with $In(1)1B;2B3-4$, with one breakpoint between 14.8 kb and 13.5 kb in the walk of Campuzano *et al.* and the other between 111 kb and 122 kb in the walk of Chao and Guild (Balcells *et al.*).

other information: Three X-ray-induced revertants of *Hw*^{49c}, all of which lack ectopic chaetae, characterized by Balcells *et al.*; *Hw*^{49c-rv1} is associated with $T(1;3)1B;95A$; its breakpoint in region 1B is in the same

restriction fragment as that of $In(1)Hw^{49c}$. *Hw*^{49c-rv4} is associated with $T(1;2)1B;21A$; its 1B breakpoint is between *sc* and *l(1)sc* in 26.8 kb to 25.0 kb. *Hw*^{49c-rv5} is associated with an eight-base-pair deletion in the *sc* coding region; it produces a polypeptide containing the first 114 amino acids of the *sc* translation product followed by 53 nonsense amino acids; the truncated polypeptide retains the basic region but not the helices of the helix-loop-helix motif. A null allele of *sc*.

Hw^{59g}

phenotype: Extra vertical, dorsocentral, and scutellar bristles. Suppression of *sc* with $su(Hw)^2$ does not suppress *Hw*^{59g}.

*Hw*⁶⁸⁵

references: Ruiz-Gómez and Modolell, 1987, Genes Dev. 1: 1238-46.

Balcells, Modolell, and Ruiz-Gómez, 1988, EMBO J. 7: 3899-3906.

phenotype: $Df(1)Hw^{685}/Df(1)sc^{19}$ generates lateral clusters of microchaetae on the scutellum and promotes differentiation of extra sensilla campaniformia on the dorsal radius of the wing, of microchaetae or other sensilla on wing vein 3, and occasional microchaetae on wing vein 2. Slight increases in numbers of microchaetae on notum as well. Displays a very weak achaete effect despite presumed homozygous deficiency for *ac*.

cytology: Claimed to be associated with terminal deficiency, $Df(1)yT1b-685$, which was broken at 45.4 kb to 43.9 kb. That this determination may be in error is suggested by the observation: (1) that no other terminal $Df(1)yT1b$, including two others broken in the same restriction fragment, shows a hairy-wing effect; (2) terminal deficiencies induced in *mu2* females are unstable losing about 75 base pairs per generation (Biessman and Mason, 1988, EMBO J. 7: 1081-86); yet *Hw*⁶⁸⁵ appears to be stable, and (3) *Hw*⁶⁸⁵ does not appear to be completely deficient for *ac*.

Hw^{bap}: Hairy wing-bristly abdominal pleura

phenotype: Nearly all abdominal segments have on the pleurae two rows of bristles, which are the same size as those on the sternites. Mutant-bearing flies have a row of bristles arising immediately posterior to each pigment band that are shorter than other tergite bristles.

cytology: Associated with the 2^{PX^D} element of $T(1;2)Hw^{bap} = T(1;2)1B;21B$.

Hw^{BS}

phenotype: Spontaneous derivation of *Hw*¹ with slightly weaker phenotype.

molecular biology: Contains an 8 kb insert near the distal LTR in the *gypsy* element that is inserted in T5, the presumptive *ac* transcript, in *Hw*¹. The identity of this sequence is unknown, but its internal 1.7 Pst1 fragment is repeated fifteen times in the genome of *Hw*^{BS} and Oregon R. The developmental profile of the *Hw*^{BS} T5 transcript is similar to that of *Hw*¹ (Campuzano *et al.*).

Hw^{Ua}

phenotype: Weak *Hw* phenotype in heterozygous females. Phenotype only slightly enhanced by suppressing the pre-existing *sc*¹ allele with $su(Hw)^2$ (García-Alonzo and García-Bellido).

molecular biology: Complete *copia* element inserted into

T4, the presumed *sc* structural gene, about one third of the distance from termination of the transcription unit. The *Hw^{Ua}* transcript, which carries the 5' two-thirds of the presumptive *sc* and terminates in *copia*, is present in excess in early larvae but not in crawling larvae or early pupae (Campuzano *et al.*).

l(1)sc: lethal at scute

synonym: *l'sc*.

references: Muller, 1935, *Genetica* 17: 237-52.

García-Bellido, 1979, *Genetics* 91: 491-520.

Jiménez and Campos-Ortega, 1987, *J. Neurogenet.* 4: 179-200.

phenotype: Deficiency from which existence of *l(1)sc* inferred, *i.e.*, *In(1)sc^{4Lsc9R}*, embryonic lethal. Volume of embryonic ventral nerve cord slightly reduced; posterior commissures thinner than in wild type; longitudinal connectives virtually lacking. Concomitant deletions for *scα* or *scα* and *ac* cause more severe CNS disruptions, although by themselves these deletions have no observable CNS effects; simultaneous deletion of the *scγ* region also enhances the CNS disruptions (Jiménez and Campos-Ortega). Transiently expressed at periphery of syncytial blastoderm; late blastoderm shows paired dorsolateral and ventrolateral longitudinal stripes of expression, the latter being coincident with the presumptive neurogenic ectoderm. During germ-band expression, *l(1)sc* expression seen in many cell clusters over most of the ectoderm; segmental distribution becomes apparent both internally and externally. *l(1)sc* expression seen in many foci in the head region and in the posterior midgut rudiment (Romani, Campuzano, and Modolell, 1987, *EMBO J.* 6: 2085-92; Cabrera, Martínez-Arias, and Bate, 1987, *Cell* 50: 425-33). Little if any expression in later stages, except in the central nervous system (Romani, Campuzano, Macagno, and Modolell, 1989, *Genes Dev.* 3: 997-1007).

molecular biology: The gene encodes a 258-amino-acid, 29-kd polypeptide with a helix-loop-helix motif in the N-terminal end and a 15-residue C-terminal acidic domain (Alonso and Cabrera, 1988, *EMBO J.* 7: 2585-91). Transcription unit between coordinated 19.9 and 17.8 kb.

other information: Inferred from the inviability of *In(1)sc^{4Lsc9R} = In(1)1B3-4;19F-20CI^L1B2-3;18B8-9^R* [left break of *In(1)sc⁹* in doubt], except in the presence of *Dp(1;2)sc¹⁹*. No mutant recovered (García-Bellido).

sc: scute

phenotype: Specifies the differentiation of numerous macrochaetae on the head and thorax as well as microchaetae on the tergites: anterior, medial, and posterior orbital, posterior vertical, ocellar, and postvertical bristles on the head plus humeral, presutural, anterior and posterior notopleural, anterior supra-alar, sternopleural, anterior and posterior postalar, and anterior and posterior scutellar bristles on the mesothorax; also participates with *ac* in specifying the anterior vertical, posterior supra-alar, and anterior dorsocentral bristles; specifies the majority of the campaniform sensilla on the wing blade (Leyns, Dambly-Chaudière and Ghysen, 1989, Wilhelm Roux's *Arch. Dev. Biol.* 198: 227-32). Males deficient for *sc*, *i.e.*, *In(1)sc^{8Lsc4R}*, are poorly viable and catatonic; homozygous deficient females lethal; males deficient for both *ac* and *sc*, *i.e.*, *In(1)y^{3PLsc4R}*, are lethal (García-

Bellido, 1979, *Genetics* 91: 491-520). *sc* deficiencies suppress the phenotype of *emc*; extra doses of *ASC* enhance the *emc* phenotype (Moscoco del Prado and García-Bellido, 1984, Wilhelm Roux's *Arch. Dev. Biol.* 193: 242-51).

alleles: Numerous alleles described, all but one of which are associated either with chromosome rearrangements with one breakpoint in region 1B or with inserts of foreign DNA. All but two chromosomal lesions with *sc* effects map proximal to the presumptive *sc* transcription unit; two inversions with breakpoints just distal to the transcription unit have slight effects; proximal lesions map to either side of *l(1)sc*, and strength of expression is inversely correlated with the molecular distance between the lesion and the scute transcription unit. Proceeding from right to left, lesions remove bristles from the mesonotum in a roughly hierarchical fashion in the following order: scutellars, (postverticals, ocellars, sternopleurals, anterior and medial orbitals, postalars), anterior notopleurals, (posterior orbitals, postverticals, anterior supra-alar) (presuturals, orbitals), with those enclosed in parentheses tending to be removed together (see Ghysen and Dambly-Chaudière, 1988, *Genes Dev.* 2: 495-501). Transcription first observed in early gastrula in regions with neurogenic potential, but before any overt evidence of neurogenesis apparent. As development proceeds, a complex temporal and spatial program of expression, mostly in neurogenic precursor cells ensues; expression ceases during the period of germ band shortening (Cabrera, Martínez-Arias, and Bate, 1987, *Cell* 50: 425-33; Romani, Campuzano, and Modolell, 1987, *EMBO J.* 6: 2085-92). In third-instar larvae, expression in wing disks is confined to restricted subsets of cells known to correspond to regions giving rise to precursors of cuticular sense organs that are under control of *sc* (Romani, Campuzano, Macagno, and Modolell, 1989, *Genes Dev.* 3: 997-1007).

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>sc¹</i>	spont	Bridges, 16a22	ase¹	12, 20	<i>gypsy</i> at 1.9 to 2.4
<i>sc²</i>	X ray	Dubinín, 1928		7, 12	17-19 kb deletion between -11.5 and -30
*sc³	X ray	Dubinín, 1928		7, 35	
sc³⁻¹	X ray	Sturtevant		12, 39	partial reversion of <i>sc³</i>
sc^{3B}	spont	Bridges, 26d26		12	
sc⁴		Agol, 1928		1, 2, 34, 35	<i>In(1)1B3-4;20F</i> ; in <i>y</i> ; kb 25.8 to 24.0
sc⁵	X ray	Gaissinovitsch, 1923		12, 13, 35	in <i>w^a</i> ; 1.2 kb deletion around -17
sc⁶	X ray	Serebrovsky, 29a21		12, 34, 35	17.4 kb deletion in 7.4 to -10.8 kb
sc⁷	X ray	Dubinín, 1929		8, 10, 35, 41	<i>In(1)1B3-4;6D-E</i> ; in <i>y</i> ; kb -1.8 to -5.9; -2.4 to -4.9 kb fragment deleted
sc⁸	X ray	Sidorov, 1929		29, 37, 38	<i>In(1)1B2-3;20F</i> ; kb 47.9 to 46.8
sc⁹	X ray	Levit, 1929		18	<i>In(1)1B3-4;18C1</i> ; kb 5.6 to 4.7
sc¹⁰			ac³		
sc¹⁰⁻¹	X ray	Sturtevant, 1930		39, 40	derivative of <i>In(1)ac3</i> ; nonsense mutation in <i>sc</i> transcript
sc¹¹			ac³		
*sc¹²	X ray	Shapiro, 1929		36	
*sc¹³		Dubinín, 1929		8, 9, 10	with <i>ac⁴</i> ; derivative of <i>sc¹</i>
*sc¹⁵	X ray	Muller	<i>scutex</i>	30	Df?
sc¹⁹	X ray	League		24	<i>Tp(1;2)1B1-2;1B4-5;25A5</i> ; kb -12.2 to

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>sc</i> 28					-13.2
<i>sc</i> 29		Agol, 1930		17	<i>In(1)1B;13A2-5;</i> kb -6.1 to -6.4 with <i>Hw</i> ^{49c} with <i>su(s)</i> ^{52c}
<i>sc</i> 49c	32 p	Lewis, 1965			
<i>sc</i> 52c	spont	Green, 52c		15	
<i>sc</i> 60d19				33	
<i>sc</i> 67b5				17	
* <i>sc</i> 90	X ray	Goldat		14	derivative of <i>sc</i> ⁶ ; <i>In(1)1B4-7;1D2-E1</i> derivative of <i>sc</i> ⁶ ; <i>T(1;2)1A6-B1;25F</i>
<i>sc</i> 115	X ray	Goldat		14	
<i>sc</i> 260-14	X ray	Sutton, 39b		43	<i>In(1)1B2-3;11D3-8;</i> kb 17.6 to 16.4
<i>sc</i> 260-15	X ray	Demerec, 38l		43	<i>T(1;3)1B4-5;71C-D;</i> kb 25.8 to 24.0
* <i>sc</i> 260-16	X ray	Sutton, 1938		43	
* <i>sc</i> 260-17	X ray	Sutton, 39d		43	<i>T(1;2)1B2-3;31C</i>
* <i>sc</i> 260-18	X ray	Sutton, 39d		43	<i>T(1;2)1A6-B1;41D-E</i>
* <i>sc</i> 260-20	X ray	Sutton, 39e		43	<i>T(1;3)1A8-B1;61A1-2</i>
<i>sc</i> 260-22	X ray	Sutton, 39f		43	<i>In(1)1B2-3;1E2-3;</i> kb -10.9 to -11.5
* <i>sc</i> 260-23	X ray	Sutton, 1939		43	<i>T(1;?)</i>
<i>sc</i> 260-25	X ray	Sutton, 39k		42	<i>In(1LR)1B2-3</i>
* <i>sc</i> 260-26	X ray	Sutton, 39l		43	<i>T(1)2)1B4-5;41F2-3;58B2-3</i> ^β + <i>In(2LR)27D2-3;41A</i>
* <i>sc</i> 260-27	X ray	Sutton, 39l		43	<i>T(1;2)1A8-B1;15E;19F;</i> <i>33-34;57B-C</i>
* <i>sc</i> 260-29	X ray	Sutton, 40a		43	<i>T(1;2;3)1A6-B1;22A-B;</i> <i>34A-B;75C-E</i> insert in -6.1 to -7.2
<i>sc</i> 1744		Schalet, '85			
* <i>sc</i> A		Agol		3	<i>In(1)?</i>
<i>sc</i> A	spont	Capdevila			insert in -13.2 to -16.8
<i>sc</i> B	X ray	Brande, 37g			<i>In(1)y</i> ⁴
<i>sc</i> B57				4	
<i>sc</i> C				17	
<i>sc</i> D1	X ray	Dobzhansky, 1930		6, 19	<i>gypsy</i> at 2.4 to 1.9; with a y allele
<i>sc</i> D2	spont	Dobzhansky, 1931		6, 12	<i>gypsy</i> at 2.4 to 1.9; in y
<i>sc</i> dr				17	
<i>sc</i> Fah	CB3007	Fahmy, 1954		11	<i>Df(1)1A8-B1;1B2-3</i>
<i>sc</i> H	γ ray	Hackett, 46a		28	<i>T(1;4)1B4;101D-E;</i> kb 6.5 to 6.0
<i>sc</i> J1	X ray	Jacobs-Muller		21, 25	<i>In(1)1A4-5;1B4-5,</i> with <i>l(1)1Ac</i> ¹
<i>sc</i> J4	X ray	Jacobs-Muller		22, 25	<i>T(1;3)1B;3A3-C2;61A;</i> kb 26.1 to 20.9
* <i>sc</i> K		Krivshenko		3	<i>T(1;3)?</i>
* <i>sc</i> K3	X ray	Krivshenko, 53j29		16	<i>T(1;3)1B2-3;61A1-2</i>
<i>sc</i> KAB					<i>T(1;3)1B4-5;98;</i> kb 16.3 to 14.9
<i>sc</i> L3		Levy, 1932		12	<i>gypsy</i> at 2.4 to 1.9
<i>sc</i> L6				17	
<i>sc</i> L8		Levy, 1932		26, 27, 31	<i>In(1)1B3-4;20F;</i> kb 30.9 to 28.8
* <i>sc</i> P1		Panshin, 1934			<i>T(1;2;3)sc</i> ^{P1}
<i>sc</i> R				17	
<i>sc</i> S1		Sinitskaya, 34c		23, 26, 31, 5	<i>In(1)1B3-4;20F;</i> kb 28.2 to 26.9
<i>sc</i> S2		Sinitskaya, 1934			<i>T(1;2)1B4-7;60C-E;</i> kb 1.2 to -3.2; 0.3 to -2.7 fragment deleted
<i>sc</i> SJ	X Ray			32	<i>In(1)1B3-4;2E3</i>
* <i>sc</i> So		Sytko		3	
<i>sc</i> V1	γ ray	J. I. Valencia, 46h23		28	<i>In(1LR)1A8-C3</i>
<i>sc</i> V2	γ ray	J. I. Valencia, 46h23		28	<i>In(1)1B2-3;20F</i>

^α 1 = Agol, 1929, Zh. Eksperim. Biol. 5: 86-101 (fig.); 2 = Agol, 1931, Genetics 16: 254-66; 3 = Agol, 1936, DIS 5: 7; 4 = Bier, Ackerman, Barbel, Jan and Jan, 1988, Science 240: 913-16; 5 = Crew and Lamy, 1940, J. Genet. 30: 273-83; 6 = Dobzhansky, 1935, DIS 3: 16; 7 = Dubinin, 1929, Biol. Zentr. 49: 328-39; 8 = Dubinin, 1930, Zh. Eksperim. Biol. 6: 300-24; 9 = Dubinin, 1932, J. Genet. 26: 37-58 10 = Dubinin, 1933, J. Genet. 27: 443-64; 11 = Fahmy, 1958, DIS 32: 74; 12 = Garcia-Bellido, 1979, Genetics

91: 491-520; 13 = Gassinovitsch, 1930, Eksperim. Biol. 6: 15-14; 14 = Goldat, 1936, Biol. Zh. 5: 803-12; 15 = Green, 1952, DIS 26: 63; 16 = Krivshenko, 1959, DIS 33: 95-96; 17 = Lee, 1973, Aust. J. Biol. Sci. 26: 903-909; 18 = Levit, 1930, Wilhelm Roux Arch. Entwicklungsmech. Organ. 122: 770-83; 19 = Morgan, Bridges, and Schultz, 1935, Carnegie Inst. Wash. Year Book 34: 290; 20 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 211, 235 (fig.); 21 = Muller, 1932, Proc. Intern. Congr. Genet., 6th., Vol. 1: 225; 22 = Muller, 1934, DIS 2: 60; 23 = Muller, 1935, DIS 3: 50; 24 = Muller, 1935, Genetica 17: 237-52; 25 = Muller, Prokofyeva, and Raffel, 1935, Nature, 135: 253-55; 26 = Muller and Raffel, 1938, Genetics 23: 160; 27 = Muller, Raffel, Gershenson, and Prokofyeva-Belgovskaya, 1937, Genetics 22: 87-93; 28 = Muller and Valencia, 1947, DIS 21: 69-70; 29 = Noujdin, 1935, Zool. Zh. 14: 317-52; 30 = Patterson and Muller, 1930, Genetics 15: 495-577 (fig.); 31 = Raffel, and Muller, 1940, Genetics 25: 541-83; 32 = Rowan, 1968, DIS 43: 61; 33 = Scowcroft, 1973, Heredity 30: 289-301; 34 = Serebrovsky, 1930, Wilhelm Roux Arch. Entwicklungsmech. Organ. 122: 88-104; 35 = Serebrovsky and Dubinin, 1930, J. Hered. 21: 259-65 (fig.); 36 = Shapiro, 1930, Zh. Eksperim. Biol. 6: 347-64; 37 = Sidorov, 1931, Zh. Eksperim. Biol. 7: 28-40; 38 = Sidorov, 1936, Biol. Zh. 5: 3-26; 39 = Sturtevant, 1935, DIS 3: 15; 40 = Sturtevant, 1936, Genetics 21: 444-66; 41 = Sturtevant, 1969, Dev. Biol. 21: 48-61; 42 = Sutton, 1940, Genetics 25: 628-35; 43 = Sutton, 1943, Genetics 28: 210-17;
^β DNA coordinates from Carramolino, Ruiz-Gómez, Guerrero, Campuzano, and Modolell (1982, EMBO J. 1: 1185-91) and Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell (1985, Cell 40: 3227-38). 0 defined as the more distal EcoRI site within the *sc*⁵² molecular deficiency; positive values extend to the left. Where known, restriction fragment interrupted by the 1B breakpoint of *sc* rearrangements indicated.

cytology: Placed in 1B3 based on its location between the left breakpoints of *In(1)sc*⁸ = *In(1)1B2-3;20F* and *In(1)sc*⁴ = *In(1)1B3-4;20F*.

molecular biology: *sc* assigned to transcript T4 of Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell (1985, Cell 40: 3227-38); the *sc* transcription unit is transcribed from left to right and occupies DNA coordinates approximately 34 to 32 kb. A series of terminal deletions approaching the *sc* transcription unit from the left were placed in heterozygous combination with *In(1)sc*^{8L}*sc*^{4R}, among other *sc* deficiencies, and the phenotypes assessed (Ruiz-Gómez and Modolell, 1987, Genes Dev. 1: 1238-46). The majority of deficiencies between coordinates 55 and 38 kb showed significant but weak *sc* phenotypes; those ending within 4-5 kb of the transcription start caused stronger phenotypic effects, removing first posterior supra-alar, and then posterior notopleural bristles (see also Ghysen and Dambly-Chaudière, 1988, Genes Dev. 2: 495-501). cDNA and genomic sequences of the *sc* transcribed region determined by Villares and Cabrera (1987, Cell 50: 415-24); the transcription unit of 1430 nucleotides is without introns; the longest open reading frame encodes a 345-amino-acid polypeptide of 38.1 kd. Contains both the helix-loop-helix motif and the C-terminal acidic region characteristic of *ac* and *l(1)sc*.

sc¹

phenotype: Causes loss or marked reduction in number of scutellar, coxal, ocellar, first and second orbital, anterior notopleural, postvertical, tergal, and sternal bristles. Bristle sockets missing; bristle cells absent 19 hr after pupation, when normally present [Lees and Waddington, 1942, DIS 16: 70-70a; 1943, Proc. Roy. Soc. (London), Ser. B 131: 87-110]. Suppressed by *su(Hw)* and *su(Hw)*². RK1.

other information: Revertable by X rays (Green, 1961, Genetics 46: 671-82).

*sc*²: see *ase*

****sc*³**

phenotype: Most bristles affected, principally ventrals, orbitals, verticals, postverticals, ocellars, humerals, presuturals, notopleurals, supra-alars, postalars, sternopleurals, abdominals, and anterior dorsocentrals; scutellars and postdorsocentrals usually present. Viability of male low; female virtually lethal. RK2.

cytology: Salivary chromosomes appear normal (Morgan, Bridges, and Schultz, 1935, Year Book – Carnegie Inst. Washington 34: 290).

***sc*³⁻¹**

phenotype: Partial reversion from *sc*³. Homozygous females show reduced viability; hemizygous females lethal (García-Bellido, 1979, Genetics 91: 491-520). RK2.

****sc*^{3B}: scute-3 of Bridges**

phenotype: Like *sc* but does not affect anterior notopleurals. Suppressed by *su(Hw)*² (Lee, 1973, Aust. J. Biol. Sci. 26: 903-09). RK1.

other information: Some stocks currently labelled *sc*^{3B} are reverted for *sc* but are now *ac*^{3B}.

***sc*⁴**

phenotype: Extreme scute. Bristles of head, except anterior verticals, absent. Only posterior notopleurals and alars remain on sides of mesothorax; abdominals, ventrals, coxals, and scutellars also missing. Slight variegation for *Hw*. RK1A.

***sc*⁵**

phenotype: Sternital and scutellar bristles reduced in number; others rarely affected. *sc*⁵/*sc*⁶ is practically wild type. RK1.

***sc*⁶**

phenotype: Slight *sc*; removes coxals, ocellars, first and second orbitals, postverticals, and anterior notopleurals. Scutellars and sternitals not affected. RK1.

cytology: No inversion.

***sc*⁷**

phenotype: Like *sc* but anterior notopleurals not affected. *sc*⁷ tends to suppress expression of *h* (Steinberg, 1942, DIS 16: 68). RK1A.

other information: *w*^a separable from *sc*⁷ by exchange in triploid female.

***sc*⁸**

phenotype: Slight *sc*; supra-alars, sternopleurals, or other bristles sometimes affected. Extra bristles may be present. Shows *Hw* effect and may be recognized in heterozygote, homozygote, or male by presence of one or more hairs on anterior mesopleural region. The *Hw* effect interacts strongly with *h* to produce extremely hairy wings (Steinberg, 1942, DIS 16: 68). *sc*⁸/*0* male nearly lethal; survivors show variegation for *y* and *ac*; lethality suppressed by a Y chromosome, partially suppressed by parts of the Y (Hess, 1962, DIS 36: 74-75; 1963, Verhandl. Deut. Zool. Ges., Zool. Anz. Suppl. 26: 87-92). RK2A.

*sc*⁸*c.o.* X: see *Df(1)sc*⁸

*sc*⁸*ENc.o.* X: see *Df(1)sc*⁸

***sc*⁹**

phenotype: Like *sc* but scutellars always absent. Abdomen swollen and wings poorly expanded, like *sc*². RK2A.

*sc*¹⁰: see *ac*³

***sc*¹⁰⁻¹**

phenotype: Like *sc*³ but more extreme; most extreme viable *sc* allele. Viability low. RK2A.

cytology: Originally thought to be associated with a minute deficiency (Schultz); not confirmed by molecular analysis.

molecular biology: Sequence data reveal a silent C→T transition at nucleotide 669, a C→G transversion at nucleotide 1143 causing ser¹⁶¹→arg¹⁶¹, and a C→T transition in nucleotide 1147 resulting in a stop codon; (first nucleotide of initial ATG codon at nucleotide 660) (Villares and Cabrera, 1987, Cell 50: 415-29).

*sc*¹¹: see *ac*³

****sc*¹²**

phenotype: First and second orbitals reduced or absent; other head bristles, posterior scutellars, and coxals also affected. Also shows achaete effect. Viability of homozygous female low. RK2.

****sc*¹³**

phenotype: Like *sc* but scutellars invariably absent and ocellars, postverticals, and first and second orbitals less frequent. Anterior and posterior dorsocentrals also absent, as are thoracic hairs, because of *ac*⁴. Viability low. RK2.

****sc*¹⁵**

phenotype: Originally allelic to *sc* and semilethal in male. Subsequently, allelic to *y*, *ac*, and *sc*, and male lethal. Lethal form exaggerates other *ac* and *sc* alleles in heterozygote. RK2A.

cytology: Presumably associated with *Df(1)sc*¹⁵; break-points unknown.

other information: Apparently, *y*⁺ and *ac*⁺ were inserted elsewhere in the genome (as in *sc*¹⁹ or *sc*^{V1}), became separated from the left end of X, and were lost.

***sc*¹⁹**

phenotype: Scutellar bristles absent and sternitals reduced. RK1A.

***sc*²⁸**

phenotype: Strong scute; not suppressed by *su(Hw)*².

***sc*²⁹**

phenotype: Similar to *sc*⁷. Viable and fertile. RK2A.

***sc*^{49c}**

other information: Overlooked at the time *Hw*^{49c} was described. Possibly of subsequent spontaneous origin.

***sc*^{52c}**

other information: Association with *In(1)sc*^{52c} (break-points unknown) inferred, since *sc*^{52c} has been inseparable from *ras v*.

sc^{67b5}

phenotype: Strong scute; not suppressed by *su(Hw)*².

sc²⁶⁰⁻¹⁴

phenotype: Both sexes viable and fertile. RK2A.

sc²⁶⁰⁻¹⁵

phenotype: Male sterile. Viability reduced. RK2A.

***sc²⁶⁰⁻¹⁶**

phenotype: *sc²⁶⁰⁻¹⁶/sc* overlaps wild type. Lethal homozygous and hemizygous. RK2.

cytology: Salivary chromosomes normal.

***sc²⁶⁰⁻¹⁷**

phenotype: Male and homozygous female viable and fertile. RK2A.

***sc²⁶⁰⁻¹⁸**

phenotype: Male sterile. RK2A.

***sc²⁶⁰⁻²⁰**

phenotype: Male and homozygous female viable and fertile. RK2A.

sc²⁶⁰⁻²²

phenotype: Both sexes viable and fertile. RK2A.

***sc²⁶⁰⁻²³**

phenotype: Both sexes viable. RK3A.

sc²⁶⁰⁻²⁵

phenotype: Variegates for *y*, *ac*, and probably *l(1)Ia*c but not for *svr*; more extreme than *sc^{V1}*. Homozygous lethal. RK2A.

***sc²⁶⁰⁻²⁶**

phenotype: Viability reduced in male. Male fertile. RK2A.

***sc²⁶⁰⁻²⁷**

phenotype: Male viable but sterile. RK2A.

***sc²⁶⁰⁻²⁹**

phenotype: Male viable but sterile. RK2A.

***sc^A: scute of Agol**

phenotype: Similar to *sc*. Viability low. RK2A.

***sc^{B1}: scute of Brande**

phenotype: Similar to *sc*. Viability good. RK2A.

sc^{B57}

phenotype: Embryo displays reduced numbers of CNS and PNS neurons.

sc^{D1}: scute of Dobzhansky

phenotype: Weak *sc*. RK2.

cytology: Salivary chromosomes apparently normal (Schultz).

sc^{D2}

phenotype: Slight *sc*. RK2.

sc^{Fah}: scute of Fahmy

phenotype: Bristles (principally orbitals, verticals, post-verticals, and ocellars) missing. Scutellars and postdorsocentrals left nearly intact. Male viable and fertile; female homozygous lethal. *sc^{Fah}/sc* has only occasional absence of postvertical or ocellar bristles. RK2A.

sc^H: scute of Hackett

phenotype: Similar to *sc* but more extreme. RK2A.

sc^{J4}

phenotype: Scute and achaete effects. RK3A.

***sc^K: scute of Krivshenko**

phenotype: Similar to *sc* but semilethal in male and lethal in homozygous female. RK2A.

sc^{L3}: scute of Levy

phenotype: In addition to scute, it has a spoon-like wing caused by a mutation to the right of *sc*. Viable. Suppressed by *su(Hw)*² (Lee, 1973, Aust. J. Biol. Sci. 26: 903-09). RK2.

sc^{L6}

phenotype: Moderate scute; suppressed by *su(Hw)*² (Lee, 1973, Aust. J. Biol. Sci. 26: 903-09)

sc^{L8}

phenotype: Similar to *sc⁴* but more extreme. Homozygous female sterile. RK2A.

sc^{S1}: scute of Sinitskaya

phenotype: Rather extreme *sc* allele; slight *Hw* effect; hairs often removed from abdomen and wings. Homozygous female sterile and low in viability. Male fertile and fairly viable. RK2A.

sc^{S2}

phenotype: Similar to *sc⁷*. RK1A.

***sc^{So}: scute of Sytko**

phenotype: Like *sc*; viability of homozygous female low. RK2.

sc^{V1}: scute of Valencia

phenotype: Extreme scute and achaete. Viability low. RK2A.

sc^{V2}

phenotype: Both achaete and scute variably affected. Some tendency for extra or twin bristles. Abdominal bristles markedly fewer both dorsally and ventrally. Male and homozygous female viable and fertile. RK2A.

sis-b: sisterless b (T. W. Cline)

location: 1-0.0.

synonym: *sc³⁻¹*.

references: Cline, 1988, Genetics 119: 829-62.

Torres and Sánchez, 1989, EMBO J. 8: 3079-86.

Cline, 1989, Cell 59: 231-34.

phenotype: Original hypomorphic allele recovered as a reversion of *sc³* to nearly wild-type *ac* and *sc* phenotype in hemizygote and homozygote; originally designated *sc³⁻¹* by Sturtevant, renamed *sis-b* by Cline. Locus also characterized by dominant effects of deficiencies and duplications of the *ASC* region, and later by mutant alleles *sc¹⁰⁻¹* and *Hw^{49c}* that affect *ASC* functions more than *sis-b* functions. *sis-b* reduces viability of homozygous females and hemizygous females are lethal; yet hemizygous males fully viable. Dominant synergistic female-specific lethal interactions with loss-of-function alleles of *Sxl*, *sis-a*, and/or maternal *da*; magnitude of viability effects depends on genetic background and inversely correlates with background effects on male-lethal effects; interactions temperature dependent, generally more extreme at higher temperatures. Female via-

bility effects suppressed by gain-of-function *Sxl^{M1}* allele, and by duplications of *Sxl⁺* or *sis-a⁺*. Duplication of *sis-b⁺* male-lethal in combination with duplication of *Sxl⁺* and/or *sis-a⁺*, more so at lower temperatures. Male lethality of duplication combinations suppressed by *Sxl⁻*. Phenotype of 2X;3A intersexes strongly dependent on dose of *sis-b⁺*. The dose-dependent interactions of this gene identify it as a positive regulator of *Sxl⁺* and part of the numerator of what is referred to as the X/A balance, the primary sex-determination signal. This is a character it shares with *sis-a*.

cytology: Located in 1B3, based on its location between the left breakpoints of *In(1)sc⁸ = In(1)1B2-3;20F* and *In(1)sc⁴ = In(1)1B3-4;20F*.

molecular biology: Activity mostly (but not entirely) confined to a region of approximately 24 kb as determined by Cline (1988), which includes the scute-alpha member of the ASC, and is known to encode one (T4) or possibly two (also T7) transcripts. Location refined further by Torres and Sánchez (1989) to the 8.3 kb fragment between the breakpoint of *In(1)sc^{L8}* distally and the breakpoint of *Df(1)yT2-650* proximally, a region containing only transcription unit T4; however, there are uncertainties connected with this assignment: *Df(1)yT* rearrangements are reported to be highly unstable, losing 75 base pairs per generation (Biessmann and Mason, 1988, EMBO J. 7: 1081-86), the assay used to assess *sis-b* function was not always sufficient to establish whether wild-type levels of *sis-b* activity were present. Molecular characterization of *sc¹⁰⁻¹* and *Hw^{49c}* indicates that *sis-b* function requires protein encoded by the *sc* transcription unit; nevertheless, it seems worthwhile to retain both gene names for the time being, since different regulatory information, and perhaps even somewhat different protein activities, are involved in sex determination versus neurogenesis.

ascutex: see *asx*

asense: see *ase* under **ASC**

ash1: absent, small, or homeotic discs

location: 3-46.

references: Shearn, 1974, Genetics 77: 115-25.

Shearn, Hersperger, and Hersperger, 1987, Roux's Arch. Dev. Biol. 196: 231-42 (fig.).

phenotype: Most alleles homozygous lethal; stage of lethality variable. Surviving and pharate adults display transformations including haltere to wing, first and third legs to second leg, genitalia to leg or antenna; *ash1⁸* and *ash1¹⁰* show transformation of posterior to anterior wing margin. Homeotic transformations also seen in clones of homozygous cells in otherwise heterozygous flies. Larvae homozygous for *ash1¹²*, a severe allele, displays a high incidence of failure to form imaginal disks, especially antennal, genital, haltere, and wing disks.

alleles:

allele	origin	synonym	comments
<i>ash1¹</i>	ICR170	<i>III-10</i>	prepupal-pupal lethal
<i>ash1²</i>	ICR170	<i>XVI-18</i>	pupal lethal
<i>ash1³</i>	EMS	<i>RD317</i>	pharate-adult lethal
<i>ash1⁴</i>	EMS	<i>RE418</i>	L2 lethal
<i>ash1⁵</i>	EMS	<i>RF327</i>	L2 lethal
<i>ash1⁶</i>	EMS	<i>RF605</i>	L1 lethal
<i>ash1⁷</i>	EMS	<i>RL031</i>	adults eclose

allele	origin	synonym	comments
<i>ash1⁸</i>	EMS	<i>RZ426</i>	adults eclose
<i>ash1⁹</i>	EMS	<i>RZ606</i>	L1 lethal
<i>ash1¹⁰</i>	EMS	<i>SP1017</i>	pharate-adult lethal
<i>ash1¹¹</i>	γ ray	<i>TK117</i>	L1 lethal
<i>ash1¹²</i>	γ ray	<i>TN402</i>	L2-to-prepupal lethal

cytology: Placed in 76B5-E based on its position between the 3L breakpoints of *T(Y;3)L14 = T(Y;3)h3;76B5-10* and *T(Y;3)A112 = T(Y;3)h11;76E*.

ash2

location: 3-{85} (distal to *aor*).

references: Shearn, 1974, Genetics 77: 115-25.

Shearn, Hersperger, and Hersperger, 1987, Roux's Arch. Dev. Biol. 196: 231-42 (fig.).

phenotype: Most alleles homozygous lethal; stage of lethality variable. Surviving and pharate adults display transformations similar to those indicated for *ash1¹*.

alleles:

allele	origin	synonym	comments
<i>ash2¹</i>	NNG	<i>703</i>	L2-L3 lethal
<i>ash2²</i>	NNG	<i>1803R</i>	prepupal lethal
<i>ash2³</i>	EMS	<i>R0631</i>	pupal-adult lethal
<i>ash2⁴</i>	EMS	<i>SE420</i>	
<i>ash2⁵</i>	EMS	<i>SH536</i>	prepupal-pupal lethal

cytology: Placed in 96A1-25 based on its inclusion in *Df(3R)96A1-7;96A20-25* associated with *In(3R)Ubx^{7L}ats^R* (González, Molina, Casal, and Ripoll, 1989, Genetics 123: 371-77).

asp: abnormal spindle

location: 3-85.2 (based on 64 *hh¹-tx* recombinants).

origin: Induced by ethyl methanesulfonate.

references: Ripoll, Pimpinelli, Valdivia and Avila, 1985, Cell 41: 907-12.

González, Molina, Casal, and Ripoll, 1989, Genetics 123: 371-77.

phenotype: Cold-sensitive recessive semilethal; 12% survive to adulthood at 25°; most larvae with small or absent imaginal discs; delayed development; survivors with nicked wings and rough eyes. Males sterile at 18° and fertile at 25°; produce abundant MI nondisjunction; females sterile. Larval neuroblasts frequently polyploid, especially when held at 18°. Phenotype enhanced by duplication for 97B, i.e., the *Y^{P3D}* element of *T(Y;3)B158 = T(Y;3)B^{SXh};97B*, but not that of *T(Y;3)R71 = T(Y;3)h1-2;97B*

cytology: Localized to 96A21-B10 between third chromosome breakpoints of *T(Y;3)A117 = T(Y;3)h24;96A21-25* and *T(Y;3)B197 = T(Y;3)h3;96B1-10*.

L-aspartate: 2-oxyglutarate aminotransferase: see **Got**

ast: asteroid

location: 2-1.3 (0.02 unit to right of S).

origin: Spontaneous.

discoverer: E. B. Lewis, 38b.

synonym: *S^r*: *Star-recessive*.

references: 1938, DIS 10: 55.

1942, Genetics 27: 153-54.

1945, Genetics 30: 137-66.

1951, Cold Spring Harbor Symp. Quant. Biol. 16: 159-

74 (fig.).

phenotype: Eyes small and rough. Veins L2, L3, L4, and L5 do not always extend to margin. Overlaps wild type rarely. *S +/+ ast* has very small eyes with fused facets; veins L2 to L5 incomplete at tip. *S ast/+ ast* has slightly larger eye than *S +/+ ast*. *S ast/+ +* resembles *S +/+ +*. *S +/+ ast* and *ast/ast* partially suppress *px* and *net*. Eyes of *ast/E(S)* rough. RK2.

alleles: **ast²* and *ast³* show eye but not wing effect of *ast* (CP627). *ast⁴*, **ast⁵*, and *ast^X* homozygotes look normal but enhance expression of *S* in heterozygotes (Lewis, 1951). Three reversions of *ast* superscripted **rv1*, **rv2*, and **rv3* (Lewis, 1951). *ast^v* is variegating allele associated with *T(2;4)21E2-3;101* (Lewis, 1951).

cytology: Placed in the 21E1-2 doublet on the basis of its being included in the synthetic deficiency derived by combining the *Y*-centric portion of *T(Y;2)21E = T(Y;2)21D4-E1* and the 2-centric portion of *T(2;4)ast^v = T(2;4)21E2-3;101* (Lewis, 1945). See also Roberts, Brock, Rudden, and Evans-Roberts, 1985, Genetics 109: 145-56.

***asx: ascutex**

location: 1-26.

origin: Spontaneous.

discoverer: Bridges, 24b14.

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 218.

phenotype: Furrow between scutellum and thorax much shallower; scutellum inflated. Body color pale. Legs have blackened leaky joints. Character less extreme in old dry cultures. Viability 60% wild type. RK3.

Asx: Additional sex combs

location: 2-72.

origin: Induced by ethyl methanesulfonate.

discoverer: Jürgens.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82. Jürgens, 1985, Nature 316: 153-55.

Breen and Duncan, 1986, Dev. Biol. 118: 442-56 (fig.)

phenotype: *Asx/+* males have extra sex-comb teeth on meso- and metathoracic legs (e.g., *Asx⁶* → 11.8 sex-comb teeth per prothoracic, 0.5 per mesothoracic, and 0.2 per metathoracic leg). Homozygote embryonic lethal. Abdominal denticles in head and thoracic segments; abdominal segments 1-7 transformed into more posterior segments. With respect to the degree of transformation to more posterior structures, *Df(2R)trix/Df(2R)trix* > *Df(2R)trix/Asx⁵* > *Asx⁵/Asx⁵*, indicating that *Asx⁵* is hypomorphic mutation. Similar genotypes derived from *Df(2R)trix/Asx⁵* oocytes achieved by means of pole-cell transplatanation display more severe transformation than their counterparts above derived from mothers carrying one dose of *Asx⁺* (Breen and Duncan). In double mutant combinations with *Pcl*, *Psc*, or *Scm* shows strong posterior transformation of all segments and failure of head involution. The presence of *BXC⁺* required for expression of phenotype.

allele	origin	discoverer	synonym	ref ^α	comments
Asx¹	EMS		<i>Asx^{IIF}</i>	3	
Asx²	EMS		<i>Asx^{ET21}</i>	3	
Asx³	X ray		<i>Asx^{XT129}</i>	3	
Asx⁴	HD		<i>Asx^{P2}</i>	2, 3	recovered as dominant

allele	origin	discoverer	synonym	ref ^α	comments
Asx⁵	EMS	Duncan	<i>Asx^{D1}</i>	1	enhancer of <i>Pc</i> recovered as suppressor of <i>Pc T(2,3)</i>
Asx⁶	γ rays	Tiong, 1982	<i>Asx^{T1}</i>		
^α					1 = Breen and Duncan, 1986, Dev. Biol. 118: 442-56 (fig.); 2 = Dura, Brock, and Santamaria, 1985, Mol. Gen. Genet. 198: 213-20; 3 = Jürgens, 1985, Nature 316: 153-55.

cytology: Placed in 51A1-B4 based on its being deleted by both *Df(2R)trix = Df(2R)51A2-4;51B3-6* and *Df(2R)L+48 = Df(2R)51A1;51B4*.

Asy: see *A-p*

Asymmetric: see *A-p*

***at: arctus oculus**

location: 2-60.1.

origin: Spontaneous.

discoverer: Fernandez Gianotti, 42g28.

synonym: *bar eye*; *arctops*.

references: 1943, DIS 17: 48.

1944, DIS 18: 45.

1945, Rev. Inst. Genet. Fac. Agron. Vet. Univ. Buenos Aires 2(14): 171-77.

1948, DIS 22: 53.

phenotype: Eyes similar to *B* but with more facets. Classification, fertility, and viability excellent. RK1.

At: Attenuated

location: 1- (in the *B* region).

origin: Induced with soft X rays in *In(1)sc^{SIL}sc^{8R}+dl-49, sc^{S1}sc⁸B*; associated with loss of *B* phenotype.

discoverer: Valencia and Valencia, 1949.

references: 1949, DIS 23: 64.

phenotype: In *At/+* females, wings incised medially and laterally, usually have one large central blister. *At/At* females have badly crumpled, blistered, and sometimes poorly developed wings. Wings of *At* males tend to be more like those of *At/+* females, although many fall somewhere between *At/+* and *At/At* in phenotype. Thus there is evidence for only a slight dosage compensation for *At*. This mutant is similar to some Beadex alleles, but allelism not tested. Both males and homozygous females viable and fertile. RK1A.

cytology: Associated with *In(1)At = In(1)16A4-5;18C4-6;20A2-3*.

Ata: Arista

location: Not located.

origin: X ray induced.

discoverer: Krivshenko, 1949.

synonym: *At* (symbol preoccupied).

references: 1954, DIS 28: 74-75.

1955, DIS 29: 73.

phenotype: Lateral branches of arista reduced, especially branches extending upward from central axis and situated at base of arista. Axis of arista often abnormal. Wings have small transparent spots distally. Homozygous lethal. Heterozygous viability and fertility comparatively high. RK2A.

cytology: Associated with *T(2;3)Ata = T(2;3)40;66F-67A + T(2;3)47;81*.

athrin: see *Act88F*

atn: see *ems*

Atpα: (Na⁺ + K⁺) ATPase α subunit**location:** 3-{70}.**references:** Lebovitz, Takeyasu, and Fambrough, 1989, EMBO J. 8: 193-202.**phenotype:** Structural gene for the α or catalytic subunit of Na⁺ K⁺ ATPase (ouabain sensitive). Immunofluorescence microscopy with a monoclonal antibody demonstrates high concentrations of αATPase in adult muscle, nervous tissue, and Malpighian tubules; strong immunofluorescence observed in brain, optic lobes, photoreceptor cells of retina, and ventral thoracic neuromere. Flies heterozygous for *Df(3R)r1-G6* which lacks *Atpα*, are sluggish and less tolerant of physical stress than normal.**cytology:** Placed in 93B by *in situ* hybridization. Included in *Df(3R)r1-G6 = Df(3R)93A2-B1;93E-F*.**molecular biology:** cDNA clones isolated by probing a late-pupal and six-hour embryonic cDNA libraries with probes from the homologous gene from chicken. All tissues and all stages tested produce three homologous transcripts of 3.6, 3.8, and 4.8 kb, with the 4.8-kb species being the most abundant, except in the adult thorax where all are equally abundant. The sequence reveals an open reading frame encoding a polypeptide of 1038 amino acids; the conceptual sequence is 75-80% homologous to other α subunits. Genomic clones contain more than 10 kb of intron sequence.**atrophy gonadique:** see *agg****ats: abnormal tarsi*****location:** 3- (not localized).**origin:** Spontaneous in a third chromosome carrying three non-overlapping paracentric inversions.**references:** Sierra and Comendador, 1984, DIS 60: 244-45(fig.)**phenotype:** Homozygotes exhibit different degrees of tarsal aplasia; most frequently second, third, and fourth tarsal joints missing; less frequently the fifth tarsal joint arises from tibia; often a rudimentary tarsal appendage arises from tibia or the fifth tarsal joint. Homozygous males and females sterile. Adult and preadult survival reduced; more than 50% of imagoes die during the first day.**Attenuated:** see *At****aub: aubergine (T. Schüpbach)*****location:** 2-39.**origin:** Induced by ethyl methanesulfonate.**references:** Schüpbach and Wieschaus.**phenotype:** Female sterile; homozygous females lay eggs which are of variable shapes; in the most extreme cases, the eggs are longer than the normal, more pointed at the posterior end, and lack dorsal appendages, resembling eggs produced by the dominant female-sterile mutation *Fs(2)G*.**alleles:** *aub*^{OC} = *aub*¹, *aub*^{AHN}, *aub*^{HN}.**augenwulst:** see *awu****aur: aurora*****location:** 3-53.**synonym:** *early-A; frühe2*.**references:** Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.**phenotype:** Maternal-effect lethal. Embryos abnormal

from nuclear cycle 9 onward. Multiple spindles assemble in long arrays with shared centrosomes. Polyploid nuclei develop with multiple centrosomes. Larval mitosis not investigated (Glover and Nüsslein-Volhard).

alleles: Three ethyl-methanesulfonate-induced alleles, *aur*¹, *aur*², and *aur*³ recovered as *074, 175, and 287*.**cytology:** Placed in 87A7-B2 based on inclusion in the region of overlap of *Df(3R)E229 = Df(3R)86F6-7;87B1-2* and *Df(3R)kar-D1 = Df(3R)87A7-8;87D1-2*.****aw: awry*****location:** 1-32 (not allelic to *wy*).**origin:** Induced by ingested radiophosphorus.**discoverer:** Bateman, 1949.**references:** 1950, DIS 24: 54.
1951, DIS 25: 77.**phenotype:** Wings upcurled, slightly wavy, convex, opaque, or vestigial-like. Variable; overlaps wild type. Viability about 50% wild type. Not enhanced in presence of *y*, as is *dvr* (1-28.1). RK3.****aw-b: awry-b*****location:** 1-38 to 39.**origin:** Induced by ingested radiophosphorus.**discoverer:** Bateman, 1950.**synonym:** *aw*².**references:** 1950, DIS 24: 54.
1951, DIS 25: 77.**phenotype:** Like *aw*. Good expression at 25°. Viability 10% that of wild type. Most males fail to eclose. RK3.***awd: abnormal wing disc (A. Shearn)*****location:** 3-102.9 (based on mapping of *awd*^K).**origin:** *P* mutagenesis.**discoverer:** Dearolf, 1983.**references:** Dearolf, Hersperger, and Shearn, 1988, Dev. Biol. 129: 159-68.

Dearolf, Tripoulas, Biggs, and Shearn, 1988, Dev. Biol. 129: 169-78.

Biggs, Tripoulas, Hersperger, Dearolf, and Shearn, 1988, Genes Dev. 2: 1333-43.

phenotype: Homozygotes die in late third larval instar; wing discs variably hypoplastic; other discs appear grossly normal. Trypan blue staining reveals cell death in wing disc, either in area of presumptive wing blade, or scattered throughout disc; other discs reveal lesser amounts of cell death later in development than in wing discs. Extensive lipid vacuolization in larval ventral ganglion, brain lobes, and proventriculus. Mutant discs exhibit slow growth when transplanted into wild-type hosts; normal discs in mutant hosts grow normally. Metamorphosis of eye-antennal, wing, and leg discs when transplanted into normal larvae severely restricted; few or no adult structures develop; mutant ovaries do not survive transplantation to wild-type hosts; however, transplanted *awd* pole cells capable of producing both *awd*⁺ and *awd*^{awd} progeny. Epidermal clones nearly lethal and with thin, short, bent bristles and hairs when induced early; survive in reduced numbers and size when induced late.**alleles:** Five *P*-induced alleles and twelve revertants of *awd*^K reported but only a subset of them identified in

publications; some revertants listed under *Df(3R)awd*.

allele	origin	discoverer	synonym	ref ^α	comments
<i>awd^{b3}</i>	P	Dearolf, 1983		2, 3	amorphic allele P element in exon 1
<i>awd^K</i>		Sturtevant, 54a	<i>Kpn</i>	1, 4, 5, 6, 7	Killer of prune; description follows
<i>awd^{Krv14}</i>	γ ray	Biggs	<i>awd^{KR14}</i>	1	hypomorph; lesion in 5' untranslated region; 20% normal protein
<i>awd^{Krv17}</i>	γ ray	Biggs	<i>awd^{KR17}</i>	1	0.8-kb deletion; mutates <i>awd</i> and adjacent downstream gene

^α 1 = Biggs, Tripoulas, Hersperger, Dearolf, and Shearn, 1988, *Genes Dev.* 2: 1333-43; 2 = Dearolf, Hersperger, and Shearn, 1988, *Dev. Biol.* 129: 159-68; 3 = Dearolf, Tripoulas, Biggs, and Shearn, 1988, *Dev. Biol.* 129: 169-78; 4 = Orevi and Falk, 1974, *Arch. Genet.* 47: 172-83; 5 = Orevi and Falk, 1974, *Mutat. Res.* 33: 193-200; 6 = Sturtevant, 1955, *DIS* 29: 75; 7 = Sturtevant, 1956, *Genetics* 41: 118-23.

cytology: Placed in 100C-D by *in situ* hybridization.

molecular biology: Locus cloned by transposon tagging and restriction mapped. 850 base pair transcript detected by northern analysis. Rescue of mutant phenotype achieved by germ-line transformation. Transcript detected by northern analysis, abundantly in larval and less abundantly in adult tissues as well as in cultured cells. Sequencing shows exon 1 = ~130 nucleotides, intron 1 = ~250 nucleotides, and exon 3 = ~300 nucleotides. Sequence determined by Biggs *et al.* indicates a protein of 153 amino acid residues and 16-17 kd in molecular weight. Gel-filtration experiments of embryonic extracts indicate that the AWD protein is present in cells in a 100 kd species. The conceptual amino acid sequence shows 78% identity and 95% similarity to that of the human metastasis inhibiting factor, NM23 (Rosengard, Krutzsch, Shearn, Biggs, Barker, Inger, Margulies, King, Liotta, and Steeg, 1989, *Nature* 342: 177-80). Both sequence and activity (Shearn) indicate that the protein is a nucleoside diphosphate kinase.

awd^K

phenotype: An antimorphic allele; no phenotypic effects, either when homozygous or heterozygous; however, it is a dominant conditional lethal, which is lethal in combination with *pn*, i.e., *pn/Y; awd^K/+* males or *pn/pn; awd^K/+* females. *awd^K* does not interact with any other eye-color mutant. Interacts lethally with all standard alleles of *pn* tested; however, *pn^{ts-ek}* insensitive to *awd^K* at permissive temperatures, whereas nine other temperature-sensitive alleles of *pn* are insensitive under all conditions. Lethal phase of *pn; awd^K* genotypes begins in early second larval instar and lasts until after the time that normal larvae pupate. Using *pn^{ts-ek}*, the temperature-sensitive period of the *pn* component of the lethal interaction determined to begin in second instar and last until eclosion. *pn; awd^K* sons of *pn* mothers appear to die earlier than those of *pn/+* mothers, suggesting a maternal effect of *pn⁺*, which is revealed in the presence of *awd^K* (Hackstein, 1971, *Mol. Gen. Genet.* 111: 371-76). Fate mapping in *pn/pn/+; awd^K/+* gynandromorphs places

the focus of the lethal interaction in the midventral part of the blastoderm, not a focus of pyridine biosynthesis. *awd^K* eye disks transplanted into *pn* hosts develop autonomously as do the reciprocal transplants (Grell, 1958, *DIS* 32: 123-24). The levels of *awd* transcript are normal in (1) *pn*, (2) *awd^K/+*, and (3) *pn; awd^K/+* flies; however, only the first two genotypes accumulate normal levels of the AWD polypeptide, the last having little or none, despite the presence of one copy of *awd⁺*. *pn; awd^K/+* larvae die in early third instar, whereas those that are *pn; awd^K/+/+* die at the end of L3.

other information: Allelism of *awd^K* demonstrated by two observations. First induced revertants of *awd^K*, as recovered on the basis of their failure to interact lethally with *pn*, are lethal in all pairwise combinations (Lifschytz and Falk, 1969, *Genetics* 62: 353-58) and in combination with *awd^{b3}*. Second transformants carrying an *awd* homologous sequence isolated from homozygous *awd^K* DNA are able to rescue *awd^{b3}* lethality and are able to kill *pn* in the presence of homozygous *awd⁺* (Biggs *et al.*).

awry: see *aw*

awu: augenwulst

location: 2-53.7 (based on mapping of *awu²* by Carfagna and Melen).

origin: Spontaneous.

discoverer: Rosin, 1951.

references: Volkart, 1959, *DIS* 33: 100.

phenotype: Eyes deformed; in most extreme expression, deeply indented at middle of anterior margin where invaginating integument forms a pad-like swelling with bristles. Expression variable, often asymmetrical. Overlaps wild type. Heterozygote occasionally has minor effects. Good viability. RK3.

alleles: **awu¹*; *awu²*, spontaneous (1971, Carfagna and Melen, *DIS* 47: 38). Shows more dominance than *awu*.

Ax: see under *N*

Axs: Aberrant X segregation (A.E. Zitron and S. Hawley)

location: 1-56 (not separated from *f* in 80 recombinants between *v* and *car*).

origin: Induced by ethyl methanesulfonate.

references: Zitron, and Hawley, 1989, *Genetics* 122: 801-21.

phenotype: Dominant meiotic mutant that affects distributive pairing; disrupts meiotic segregation of non-exchange chromosomes in females. Has no effect on the frequency of exchange. Loss of function mutation at a dosage sensitive locus; *Axs⁺* duplication can rescue mutation in *trans*. Specific to female meiosis. In *Axs/Axs/Y* females, X-chromosomes nondisjunction is accompanied by random disjunction of the *Y* rather than directed disjunction from the *X*'s as observed in *+/+/Y* females. *Axs* homozygotes increase X nondisjunction many fold; parallel but lesser effects observed on autosomal disjunction.

cytology: Associated with a small chromosome aberration at 15D1.

b: black (M. Ashburner)

location: 2-48.5.

phenotype: Black pigment on body and tarsi and along wing veins. Reflectance of cuticle 40% that of wild type (Pedersen, 1982, Carlsberg Res. Comm. 47: 391-400). Heterozygote somewhat darker than wild type, especially on trident, but never confused with homozygote. Body color darker at low temperature (Sherald, 1981, Mol. Gen. Genet. 183: 102-06). Puparium lighter than wild type. Not easily classified in newly emerged flies (Waddington, 1941, Proc. Zool. Soc. London Ser. A. 111: 173-80). Tyrosinase formed in adult flies (Horowitz). Fails to synthesize beta-alanine (Hodgetts, 1972, J. Insect Physiol. 18: 937-47), and feeding or injection of beta-alanine to *b* produces normal phenotype [Jacobs, 1974, J. Insect Physiol. 20: 859-66; Hodgetts, Hodgetts and Choi, 1974, Nature (London) 252: 710-11]. Also corrected by feeding 6-azauracil (Pedersen, 1982, Hereditas 97: 329); feeding 6-azathymine produces weak *b* phenocopy which is suppressed by *Su(b)* (Pedersen). ERG normal [Hotta and Benzer, 1969, Nature (London) 22: 354-56]. Puparial case structurally abnormal with wide, diffuse, exocuticular lamellae and indistinct endocuticular fibrils (Jacobs, 1978, Insect Biochem. 8: 37-41). Pupae UV sensitive (Jacobs 1978). Suppressed by *su(b)* (Sherald, 1981, Mol. Genet. 181: 102-106) and *Su(b)* (Pedersen); enhanced by *sp*² (Gubb) and *su(r)* (Pedersen). *su(r) su(b) b* is enhanced black; *su(r) b* not enhanced by feeding 6-azauracil (Pedersen). Possibly structural gene for beta-ureidopropionase (EC 3.5.1.6; Sherald). RK1 in aged flies.

alleles:

allele	origin	discoverer	ref ^α	cytology
<i>b</i> ¹	spont	Morgan, 1910	4, 5, 7, 8	
<i>*b</i> 14b28	spont	Bridges	7	
<i>*b</i> 18c27	spont	Wallace	7	
<i>*b</i> 23b2	spont	L.V. Morgan	7	
<i>*b</i> 24b15	spont	L.V. Morgan	7	
<i>*b</i> 29	spont	Promptov	7	
<i>*b</i> 34g	spont	Gottschewski	7	
<i>b</i> 36f		Nichols-Skoog,	11	
		36f1		
<i>b</i> 39	spont	Glass, 39f22	7, 9	
<i>*b</i> 39j	spont	Bryson	7	
<i>b</i> 40c	spont	Buzzati- Traverso,	6, 7	
		40c15		
<i>b</i> 50d β	UV	Meyer, 50d	8, 12	
<i>b</i> 51f β	UV	Meyer, 51f	8, 12	
<i>b</i> 661	γ ray	Alexandrov	1	
<i>b</i> 66h				
<i>b</i> 71k1	γ ray	Alexandrov	1	<i>Tp(2;2)34D2-4;</i> <i>34D8-E1;</i> <i>43C2-4;</i> lethal
<i>b</i> 71k2	γ ray	Alexandrov	1	
<i>b</i> 74b2	caffeine + γ ray	Alexandrov	1	
<i>b</i> 74b4	caffeine + γ ray	Alexandrov	1	
<i>b</i> 74b5	caffeine + γ ray	Alexandrov	1	
<i>b</i> 74c2	caffeine + γ ray	Alexandrov	1	
<i>b</i> 74c4	caffeine + γ ray	Alexandrov	1	
<i>b</i> 74c5	caffeine + γ ray	Alexandrov	1	
<i>b</i> 74d2	caffeine + γ ray	Alexandrov	1	
<i>b</i> 74d4	caffeine + γ ray	Alexandrov	1	
<i>b</i> 74d6	caffeine + γ ray	Alexandrov	1	
<i>b</i> 75a	γ ray	Alexandrov	1	
<i>b</i> 75f	EMS		2	
<i>b</i> 76b1	caffeine + γ ray	Alexandrov	1	
<i>b</i> 76b2	caffeine + γ ray	Alexandrov	1	

allele	origin	discoverer	ref ^α	cytology
<i>b</i> 76e1	γ ray	Alexandrov	1	
<i>b</i> 76e2	γ ray	Alexandrov	1	
<i>b</i> 76f3 β	γ ray	Alexandrov	1	
<i>b</i> 76j1	actin.-D + γ ray	Alexandrov	1	
<i>b</i> 76j2	actin.-D + γ ray	Alexandrov	1	
<i>b</i> 76j3	actin.-D + γ ray	Alexandrov	1	
<i>b</i> 76k1	actin.-D + γ ray	Alexandrov	1	
<i>b</i> 76k2	actin.-D + γ ray	Alexandrov	1	
<i>b</i> 77a1	actin.-D + γ ray	Alexandrov	1	
<i>b</i> 77a2	actin.-D + γ ray	Alexandrov	1	
<i>b</i> 77a3	actin.-D + γ ray	Alexandrov	1	
<i>b</i> 77a4	actin.-D + γ ray	Alexandrov	1	
<i>b</i> 77a5	actin.-D + γ ray	Alexandrov	1	
<i>b</i> 77j	γ ray	Alexandrov	1	
<i>b</i> 77.1X	X ray		3	<i>In(2L)Cy^L_t^R, Roi</i>
<i>b</i> 77.2X	X ray		3	<i>In(2L)Cy^L_t^R, Roi</i>
<i>b</i> 77.3e	EMS		2	
<i>b</i> 77.4e	EMS		2	
<i>b</i> 77.5X	X ray			
<i>b</i> 78.1	DEO γ	Detwiler	3	
<i>b</i> 78a	γ ray	Alexandrov	1	
<i>b</i> 78f1	NaF + γ ray	Alexandrov	1	
<i>b</i> 78f2	NaF + γ ray	Alexandrov	1	
<i>b</i> 78g	NaF + γ ray	Alexandrov	1	
<i>b</i> 78k1	NaF + γ ray	Alexandrov	1	
<i>b</i> 78k2	NaF + γ ray	Alexandrov	1	
<i>b</i> 78k3	NaF + γ ray	Alexandrov	1	
<i>b</i> 78k4	NaF + γ ray	Alexandrov	1	
<i>b</i> 78k5	NaF + γ ray	Alexandrov	1	
<i>b</i> 79a1	0.85 MeV n	Alexandrov	1	
<i>b</i> 79a2	0.85 MeV n	Alexandrov	1	
<i>b</i> 79b1	γ ray	Alexandrov	1	
<i>b</i> 79b7	0.85 MeV n	Alexandrov	1	
<i>b</i> 79d2	γ ray	Alexandrov	1	
<i>b</i> 79d4	0.85 MeV n	Alexandrov	1	
<i>b</i> 79d5	0.85 MeV n	Alexandrov	1	<i>In(2L)34D4;</i> <i>35B10;</i> lethal
<i>b</i> 79d6	0.85 MeV n + γ ray	Alexandrov	1	<i>T(2;3)34A2-3;</i> <i>34D8-E1;79B;</i> <i>80C;</i> lethal
<i>b</i> 79d8 β	0.85 MeV n + γ ray	Alexandrov	1	
<i>b</i> 79dt0	0.85 MeV n + γ ray	Alexandrov	1	
<i>b</i> 79dt1	0.35 MeV n	Alexandrov	1	
<i>b</i> 79dt3	γ ray	Alexandrov	1	
<i>b</i> 79df2	γ ray	Alexandrov	1	
<i>b</i> 79g2	γ ray	Alexandrov	1	
<i>b</i> 79h1	γ ray	Alexandrov	1	<i>Tp(2;2)34D2-4;</i> <i>34D8-E1;</i> <i>41;</i> lethal
<i>b</i> 79h2	γ ray	Alexandrov	1	
<i>b</i> 79h3	γ ray	Alexandrov	1	
<i>b</i> 80c1	γ ray	Harrington		
<i>b</i> 80c2	γ ray	Angel	3	<i>In(2L)b^{80c2}</i>
<i>b</i> 80i2	γ ray	Harrington		
<i>b</i> 80i3	γ ray	Harrington		
<i>b</i> 80j1	γ ray	Harrington		<i>T(1;3)CA17</i>
<i>b</i> 80j2	γ ray	Harrington		
<i>b</i> 80k1	γ ray	Harrington		
<i>b</i> 80k2	γ ray	Harrington		
<i>b</i> 80l1	γ ray	Angel		<i>Tp(2;2)Sco</i>
<i>b</i> 80l2	γ ray	Angel		<i>Tp(2;2)Sco</i>
<i>b</i> 81a	γ ray	Alexandrov	1	<i>Tp(2;2)34D2-4;</i> <i>34D8-E1;</i> <i>41D-E1;</i> lethal
<i>b</i> 81a2	γ ray	Alexandrov	1	
<i>b</i> 81c	0.7 MeV n	Alexandrov	1	
<i>b</i> 81c2	γ ray	Alexandrov	1	
<i>b</i> 81c17	γ ray	Alexandrov	1	
<i>b</i> 81dt				
<i>b</i> 81ff δ	γ ray	Gubb		<i>CyO +</i> <i>Dp(2;2)34D3;</i> <i>35B2</i>
<i>b</i> 81f2	γ ray	Gubb		<i>CyO</i>
<i>b</i> 81f3 β	0.1 MeV n	Alexandrov	1	<i>In(2L)34D2-4;</i> <i>35B10</i>
<i>b</i> 81h2	γ ray	Gubb		<i>CyO</i>

allele	origin	discoverer	ref ^α	cytology
<i>b</i> 81 <i>k</i> β	γ ray	Alexandrov	1	
<i>b</i> 8112	γ ray	Durrant		<i>In(2L)Gla</i>
<i>b</i> 81117	γ ray	Alexandrov	1	<i>In(2L)34D2-4</i> ; 40 <i>F</i> ; lethal
<i>b</i> 81140	γ ray	Alexandrov	1	
<i>b</i> 81142	γ ray	Alexandrov	1	lethal
<i>b</i> 82a1	γ ray	Harrington		
<i>b</i> 82c1	spont	Harrington		<i>In(2LR)Gla</i>
<i>b</i> 82c3	252 Cf fission n	Alexandrov	1	
<i>b</i> 82c7	252 Cf fission n	Alexandrov	1	
<i>b</i> 82c16	252 Cf fission n	Alexandrov	1	
<i>b</i> 82c44	252 Cf fission n	Alexandrov	1	<i>In(2L)34D4</i> ; 40 <i>F</i> ; homozygous sterile
<i>b</i> 82c54	252 Cf fission n	Alexandrov	1	homozygous sterile
<i>b</i> 83b1	γ ray	Alexandrov	1	homozygous sterile
<i>b</i> 83b2	γ ray	Alexandrov	1	<i>In(2L)34D4</i> ; 35 <i>B10</i> ; lethal
<i>b</i> 83b40	γ ray	Alexandrov	1	
<i>b</i> 83c20	γ ray	Alexandrov	1	
<i>b</i> 83c25	γ ray	Alexandrov	1	
<i>b</i> 83c26	γ ray	Alexandrov	1	
<i>b</i> 83c35a	γ ray	Alexandrov	1	homozygous sterile
<i>b</i> 83c35b	γ ray	Alexandrov	1	
<i>b</i> 83c36	γ ray	Alexandrov	1	
<i>b</i> 83c47	γ ray	Alexandrov	1	homozygous sterile
<i>b</i> 83d29b	252 Cf fission n	Alexandrov	1	
<i>b</i> 83d35	252 Cf fission n	Alexandrov	1	
<i>b</i> 83d36	252 Cf fission n	Alexandrov	1	
<i>b</i> 83f17	γ ray	Alexandrov	1	
<i>b</i> 83f18	γ ray	Alexandrov	1	
<i>b</i> 83f51	γ ray	Alexandrov	1	
<i>b</i> 83f52	γ ray	Alexandrov	1	
<i>b</i> 83f <i>XD</i>	X ray	Alexandrov	1	
<i>b</i> 83l	0.7 MeV n	Alexandrov	1	
<i>b</i> 84b1	γ ray	Harrington		
<i>b</i> 84g	X ray	Alexandrov	1	
<i>b</i> 84h34	X ray	Alexandrov	1	
<i>b</i> 84h40	X ray	Alexandrov	1	
<i>b</i> 84h70	γ ray	Alexandrov	1	
<i>b</i> 85b3	γ ray	Alexandrov	1	
<i>b</i> 85b4	γ ray	Alexandrov	1	
<i>b</i> 85c2	0.85 MeV n	Alexandrov	1	<i>T(2;3)34C7-D1</i> ; 34 <i>E1-2</i> ; 95 <i>C4-D1</i> ; homozygous sterile
<i>b</i> 85c3	0.85 MeV n	Alexandrov	1	
<i>b</i> A51	EMS	Sherald		
<i>b</i> A53	EMS	Sherald		
<i>b</i> A54	EMS	Sherald		
<i>b</i> A55	EMS	Sherald		
<i>b</i> D ^e	spont	Goldschmidt,	10	
		1945		

α 1 = Alexandrov and Alexandrova, 1986, DIS 63: 159-61; 2 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95; 3 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91; 4 = Ashburner, Aaron, and Tsubota, 1982, Genetics 102: 421-35; 5 = Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 144 (fig.); 6 = Buzzati-Traverso, 1940, DIS 13: 49; 7 = CP552; 8 = CP627; 9 = Glass, 1939, DIS 12: 47; 10 = Goldschmidt, 1945, Univ. Calif. Berkeley Publ. Zool. 49: 504-20; 11 = Nichols-Skoog, 1937, DIS 7: 60; 12 = Meyer and Edmonson, 1951, DIS 25: 71.

β Lighter than *b*¹.

γ Diepoxyoctane.

δ Induced simultaneously with *rK* 81*f*2.

ε Synonym = *b*^B; homozygous lethal, said to be associated with *Df(2L)35C;35D*, but this was almost certainly a misinterpretation of ectopic pairing (Ashburner).

cytology: Placed in region 34D4-6 (Ashburner).

β: see *Tyr2*

B: Bar

location: 1-57.0.

origin: Spontaneous in a female.

discoverer: Tice, 13b.

references: 1914, Biol. Bull. 26: 221-30 (fig.).

Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. No. 237: 66 (fig.).

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 29-33.

phenotype: Eye restricted to narrow vertical bar of about 90 facets in the male and 70 facets in the female, as contrasted with normal numbers of about 740 for males and 780 for females [Sturtevant, 1925, Genetics 10: 117-47 (fig.)]. Homozygous female fully viable. *B/+* female has about 360 facets and shows indentation terminating in horizontal fissure on anterior margin of eye, producing a kidney-shaped eye. *B/B* and *B/+* completely separable from wild type; in some genetic backgrounds, *B/B* overlaps *B/+* slightly. Classifiable in single dose in triploids by slight anterior nick in eye (Schultz, 1934, DIS 1: 55); is useful in the recognition of triploids. Variegated position effect derivatives of *B*^S(*B*^{SVY}) exhibit nonmutant phenotypes in *XY* males but narrow eyes in *XYX* males; other enhancers of variegation, *M(2)S10* and *E(var)7*, also shift phenotype toward normal (Brosseau, 1960, Genetics 45: 979). Eyes of female heterozygous for a deficiency for *B* and a normal *X* are normal (Sutton, 1943, Genetics 28: 97-107). Log of facet number inversely proportional to temperature of development (Hersh, 1930, J. Exp. Zool. 57: 283-306).

Nonautonomous over short distances (Sturtevant, 1932, Proc. Intern. Congr. Genet., 6th, Vol. 1: 304-7). Facet development enhanced in organ culture by addition of wild type cephalic complexes [Kuroda and Yamaguchi, 1956, Jpn. J. Genet. 31: 97-102 (fig.)]. Disc size reduced; morphogenetic furrow absent; deep cleft at anterior margin of preommatidial cell clusters. Only four to five rows of clusters. Those at cleft edge look mature (Renfranz and Benzer, 1989, Dev. Biol. 136: 411-29). Cell death observed in anterior presumptive ommatidium-forming region of eye disc [D. Fristrom, 1969, Mol. Gen. Genet. 103: 363-79; 1972, Mol. Gen. Genet. 115: 10-18; Michinomae and Kaji, 1973, Jpn. J. Genet. 49: 353-71 (fig.); Michinomae, 1974, Jpn. J. Genet. 49: 353-71; 1976, Jpn. J. Genet. 51: 315-26 (fig.)]; high acid phosphatase levels characteristic of liposomal activity seen at same time (Michinomae, 1974, 1976). Reduction in facet number, increase in acid phosphatase activity, and cell death inhibited in larvae grown on medium supplemented with acetamide or lactamide (Kaji, 1954, Annot. Zool. Jpn. 27: 194-200; Fristrom, 1972; Michinomae and Kaji, 1973; Michinomae, 1974, 1976). Double amides more effective than single (DeMarinis and Sheibley, 1968, DIS 43: 138). Facet development responds strongly to environmental factors around 60 hr after oviposition (Luce, Quastler, and Chase, 1951, Genetics 36: 488-99). Pigmented but non-faceted part of eye shows retinulae and dioptic apparatus lacking, but rudimentary ommatidia present, consisting of hypertrophied accessory cells (Wolsky and Huxley, 1936, Proc. Zool. Soc. London 485-89). Introduction of *B* into a strain of flies characterized by jumping in response to a sudden decrease in light intensity nearly eliminates response; response rescued by increasing facet number

allele	origin		ref α	phenotype		cytology δ
	chromosome	treatment		eye β	viability γ	
<i>B</i> ³	<i>B</i>	spont	6, 7	< <i>B</i>	+	
* <i>B</i> ⁴	<i>B</i>	spont	6, 7	< <i>B</i>	+	
<i>B</i> ^{36b}	<i>BB</i>	spont	6, 7	< <i>B</i>	+	
<i>B</i> ^{36d}	<i>B</i>	spont	6, 7	> <i>B</i>	+	
* <i>B</i> ^{36j}	+	spont	6, 7	< <i>B</i>	+	
* <i>B</i> ^{48g}	+	X ray	7	< <i>B</i>	+	<i>T(1;2)15F-16A1;33B +</i> <i>ln(1)sc</i> ⁴
<i>B</i> ^{58l}	+	X ray	7	> <i>B</i>	+	<i>T(1;3)16A;88F</i>
<i>B</i> ^{68f}	+	X ray	3	< <i>B</i>	+	<i>T(1;4)15F;101</i>
* <i>B</i> ²⁶³⁻⁵	<i>B</i>	X ray		+	-	<i>Df(16A1-7;17A3-4</i>
* <i>B</i> ²⁶³⁻¹⁴		X ray	6			
<i>B</i> ²⁶³⁻²⁰	<i>B</i>	X ray	6	+	-	<i>Df(1)15F9-16A1;16A7-B1</i>
* <i>B</i> ²⁶³⁻²⁴	<i>BⁱBⁱ</i>	X ray	6	+	-	<i>Tp(1;1)10C1-2;12E1-2;16A5-6</i>
* <i>B</i> ²⁶³⁻²⁸	<i>BⁱBⁱ</i>	X ray	6, 7	< <i>B</i>	+	Deletes one 16A7-16A1 Junction no change
* <i>B</i> ²⁶³⁻³⁴	<i>BⁱBⁱ</i>	X ray	6, 7	+	-	no change
* <i>B</i> ²⁶³⁻³⁸	<i>BⁱBⁱ</i>	X ray	6, 7	+	-	no change
* <i>B</i> ²⁶³⁻⁴³	<i>BⁱBⁱ</i>	X ray	6	+	+	+
* <i>B</i> ²⁶³⁻⁴⁶	<i>B</i>	X ray	6	+	+	+
* <i>B</i> ²⁶³⁻⁴⁷	+	X ray	6, 7	<i>B</i>	+	<i>ln(1)16A2-4;20A2-3</i>
* <i>B</i> ²⁶³⁻⁴⁸	+	X ray	6, 7	< <i>B</i>	+	<i>Tp(1;1)3E2-3;15F9-16A1;20A2-3</i>
* <i>B</i> ²⁶³⁻⁴⁹	<i>BB</i>	X ray	6, 7	<i>BB to +</i>	+	no change
* <i>B</i> ²⁶³⁻⁵¹	<i>BB</i>	X ray	6, 7	+	+	no change
<i>B</i> ^{bd}	+	X ray	6, 7	< <i>B</i>	+	<i>T(1;2)16A1-2;48C2-3 +</i> <i>ln(2R)41A;47A</i>
* <i>B</i> ^{DG}	+	X ray	6, 7		-	<i>T(1;2)4;15F-16A;20;40;41</i>
<i>B</i> ⁱ	<i>B</i>	spont	6, 7	< <i>B</i>	+	no change
* <i>B</i> ^{i40b}	<i>BB</i>	spont	6, 7	< <i>B</i>	+	no change
* <i>B</i> ^{i67b}	<i>FM6</i>	spont	8	< <i>B</i>	+	no change
* <i>B</i> ^{i67f}	<i>FM6</i>	spont	8	< <i>B</i>	+	no change
<i>B</i> ^{M1}	+	X ray	6, 7	< <i>B</i>	+	<i>ln(1)16A2-5;20A3-B</i>
<i>B</i> ^{M2}	+	X ray	6, 7	< <i>B</i>	+	<i>ln(1)16A2-5;20E</i>
* <i>B</i> ^{par}	<i>B</i>	X ray	6, 7	< <i>B</i>	+	
<i>B</i> ^r	+	X ray	2	+	+	
* <i>B</i> ^R	<i>B</i>	X ray	7	> <i>B</i>	+	
* <i>B</i> ^{rev-1}	<i>B</i>	X ray	6, 7	+	+	no change
* <i>B</i> ^{rev-2}	<i>B</i>	X ray	6	+	+	<i>ln(1)3F8-4A1;16A2-4</i>
* <i>B</i> ^{rev-3}	<i>B</i>	X ray	6	+	+	<i>ln(1)16A6-A1;20A4-5</i>
<i>B</i> ^S	<i>B</i>	X ray	6, 7	> <i>B</i>	+	<i>T(1;4)16A7-A1;102F</i>
<i>B</i> ^{S3i}	<i>B</i>	X ray	7	> <i>B</i>	+	<i>Tp(1;3)16A7-B1;19-20;66B13-C1</i>
<i>B</i> ^{SR}	<i>T(1;4)B^S</i>	spont	5, 9	+	+	no change
<i>B</i> ^{SV}	<i>B^SY</i>	X ray	1	< <i>B</i>		

α 1 = Brosseau, 1960, Genetics 45: 979 and the report of the Seattle-La Jolla Drosophila Laboratories, 1971, which appeared as an unbound supplement to DIS 47; 2 = Brosseau, 1967, DIS 42: 38; 3 = Brosseau, 1969, DIS 44: 45; 5 = Childress, 1973, Mol. Gen. Genet. 121: 133-38; 6 = CP552; 7 = CP627; 8 = Kaplan, 1969, DIS 44: 45; 9 = Novitski, 1970, DIS 45: 87-88.

β The phenotype of females carrying the allele in heterozygous combination with +. <*B* = less extreme than *B*/+; >*B* = more extreme than *B*/+, etc.

γ Viability of males hemizygous and, where testable, of females homozygous for the allele. Where tested the lethal alleles are cell lethal in cuticular spots.

δ + = loss of tandem duplication or triplication to produce normal sequence.

through lactamide feeding during larval development (Nakashima-Tanaka and Matsubara, 1980, Jpn. J. Genet. 55: 275-82). RK1A.

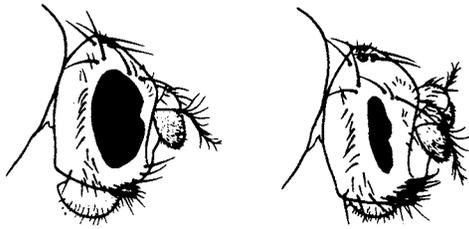
alleles: Derivatives of *B* with altered phenotypes, including reversions to normal phenotype have been given allelic designations. These derivatives are summarized in the accompanying table. Unless otherwise indicated, fuller descriptions are found in CP627. See table for allelic information.

cytology: Located in 16A1-2. Associated with *Dp(1;1)B = Dp(1;1)15F9-16A1;16A7-B1*.

molecular biology: Region cloned by transposon tagging from hybrid-dysgenesis-induced *B* reversion (Tsubota, Rosenberg, Szostak, Rubin, and Schedl, 1989, Genetics 122: 881-90). Analysis of genomic DNA reveals presence of *roo* element at the junction between 16A7 and 16A1, indicating that original duplication may have arisen by unequal recombination between *roo* elements inserted at 16A1 and 16A7. Three hybrid-dysgenesis-induced partial revertants have *P*-element inserts close to

the junction as does *B*³. Breakpoints of *B*, *B*^{M1}, *B*^{M2}, and *B*^{bd} span a region of approximately 37 kb.

other information: Since *B* is a tandem duplication, *B* homozygotes may give rise to a nonduplicated chromosome (reversal to normal phenotype) and a triplicated chromosome (*i.e.*, double Bar = *BB*) as reciprocal products of unequal crossing over (Sturtevant and Morgan, 1923, Science 57: 746-47; Gabay and Laughnan, 1973, Genetics 75: 485-95). From successive unequal crossovers in attached X's, Rapoport (1940, Zh. Obshch. Biol. 1: 235-70; 1941, DIS 15: 36-37) was able to accumulate as many as 7 or 9 Bar regions in a single chromosome. Bar is the first recorded instance of position effect. Presumably results from the new band association 16A7-16A1 and can be reversed by rearrangements that separate these bands. Also the first case of *cis-trans* position effect, two 16A7-16A1 associations in the same chromosome producing greater facet reduction than one association in each of two homologous chromosomes.



B: Bar

Left: heterozygous female. Right: hemizygous male. From Sturtevant and Beadle, 1939. An Introduction to Genetics, Saunders, p. 24.

B^Z : see B^R

$b-l^{33g18}$: see *tri*

$B2t$: see *TubB85D*

ba: balloon

location: 2-107.4.

origin: Spontaneous.

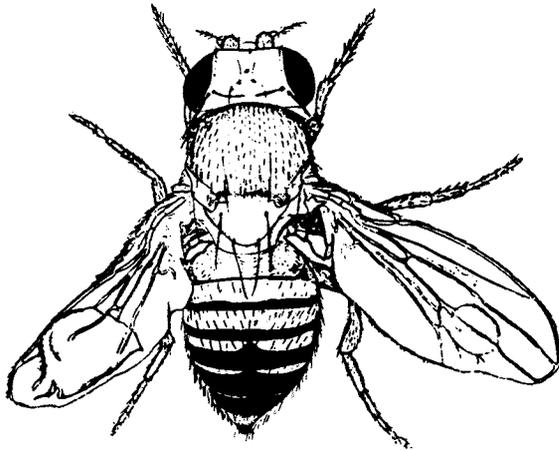
discoverer: Morgan, 10k.

references: Marshall and Muller, 1917, J. Exp. Zool. 22: 457-70 (fig.).

Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 148 (fig.).

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 212 (fig.), 218.

Bridges, 1937, Cytologia (Tokyo), Fujii Jub., Vol. 2: 745-55.



ba: balloon

From Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 148.

phenotype: Wings at first inflated with hemolymph to produce blisters and vesicles; venation weak, plexus like; wings smaller, warped, discolored, and divergent. Effect caused by inadequate contraction of epithelium after inflated state of pupal wing [Waddington, 1940, J. Genet. 41: 75-139 (fig.)]. Sensitive to temperature. RK3 above 25°; RK2 at 19° or below.

alleles: $*ba^2$ (CP627).

cytology: Located in 60C5-D2 based on inclusion within $Df(2R)Px = Df(2R)60B8-10;60D1-2$ and within $Df(2R)Px^2 = Df(2R)60C5-6;60D9-10$ (Bridges, 1937).

other information: May be part of a pseudoallelic complex with *bs* and *Px*.

ba^2 : see *blo*

ba^{33f26} : see *blo*

Ba: Brista

location: 2-107.8 (0.8 unit to right of *sp*).

synonym: *Dll*: Distal-less.

references: Sato, 1984, DIS 60: 180-82 (fig.)

Sunkel and Whittle, 1987, Wilhelm Roux's Arch. Dev. Biol. 196: 124-32.

Cohen and Jürgens, 1989, EMBO J. 8: 2045-55.

phenotype: Null alleles are recessive embryonic lethals, with dominant developmental defects of distal appendages. Lethal embryos lack certain sensilla, including Keilin's organs, and antennal, maxillary, labial, and labral sense organs, all of which are thought to correspond to vestiges of the distal sensilla of rudimentary larval appendages. No other embryonic sensilla are affected, nor are the neurons innervating the rudimentary appendages detectably abnormal. In homozygous-viable or pharate-adult-lethal hypomorphic alleles defects are found in all appendages represented by the above larval structures. Heterozygotes for lethal alleles are characterized by transformations of distal antennal segments (*i.e.*, AII, AIII, and arista) to mesothoracic leg and by variable loss of distal leg segments, depending on severity of allele. Transformation in the case of Ba^1 sensitive to low-temperature pulses throughout larval development, whereas the TSP for leg truncation is at end of the third larval instar. Clones of homozygous Ba^- cells incapable of contributing to any but the coxal segment of the legs, and for the most part, the first antennal segment; in the relatively infrequent cases, in which Ba^- clones involve distal antennal segments, Ba^- portions, which always include at least the arista, are absent, and Ba^+ portions develop normally indicating cellular autonomy.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments ^β
Ba^1	γ ray	Whittle		2, 3, 6	cold sensitive allele
$*Ba^2$	EMS	Sunkel		6	
Ba^3	EMS	Sunkel		6	recessive pharate-adult lethal
Ba^5	EMS	Tiong, 1983		3, 6	
Ba^6				2	
Ba^7				2	
Ba^8				2	
Ba^9	spont	Sato	Ba^M	2, 5, 6	recessive; homozygous viable
Ba^{10}	HD	Adler	$E(Arp)$	1, 2, 3	8-kb insert in 13-kb intron at ~ 120 + <i>P</i> insert distal to transcription unit at 30 kb; recovered as enhancer of <i>Arp</i>
Ba^{11}	X ray	Botas	Art^1, Ba^B	2, 3, 6	$In(2LR)48E;60E5-6$; break at ~ 115 kb ^γ
Ba^{12}	EMS	Kennison	Ba^{E1}	4	
Ba^{13}		Gelbart	Ba^G	2, 3	$Tp(2;3)52E;60E;81$; break at ~ 100 to 110 kb
Ba^{14}	spont	Bussey	$anto, Ba^{1B}$	2	
Ba^{15}	γ ray	Jürgens	Ba^J	2, 3, 6	$T(2;3)60E5-6;64B12-C12$; break at ~ 105 kb; 3' to putative coding region ^δ
Ba^{16}	HD	Posakony	Ba^P	2	insert of ? at ~ 125 kb
Ba^{17}			Ba^{SA1}	2, 3	~ 6 kb deletion around ~ 120 kb; removes middle exon
Ba^{18}	spont	Williams	Art^1, Ba^W	2, 3, 6	$T(1;2)3C3-7;60E5-6$; break at ~ 120 kb

allele	origin	discoverer	synonym	ref ^α	comments ^β
α	1 =	Brunk and Adler, 1990, Genetics 124: 145-56;			
	2 =	Cohen, Brönner, Küttner, Jürgens, and Jäckle, 1989, Nature 338: 432-34;			
	3 =	Cohen and Jürgens, 1989, EMBO J. 8: 2045-55;			
	4 =	Kennison and Tamkun, 1988, Proc. Nat. Acad. Sci. USA 85: 8136-40;			
	5 =	Sato, 1984, DIS 60: 180-82;			
	6 =	Sunkel and Whittle, 1987, Wilhelm Roux's Arch. Dev. Biol. 196: 124-32.			
β					Coordinates estimated from figure of Cohen <i>et al.</i> ; origin undefined; positive values extend to left.
γ					Differs from other tested alleles in showing some dominance in combination with two + alleles.
δ					Differs from other tested alleles in being embryonic rather than pupal lethal in combination with <i>Ba</i> ³ and <i>Ba</i> ⁹ .

cytology: Placed in 60E5-6 based on its inclusion in *Df(2R)Ba = Df(2R)60E3;60E6* and on common breakpoints of *Ba* rearrangements.

molecular biology: Region isolated in a chromosome walk of 180 kb (Cohen, Jürgens, and Jäckle, 1989, Nature 338: 432-34). *Ba* lesions extend over a region of 38 kb. A single transcription unit detected that is homologous to transcripts of ~2.5 and ~3.4 kb which hybridize to restriction fragments extending over 25 kb of genomic sequence; three exons identified; sequence of genomic fragments hybridizing transcript indicate the presence of a putative homeodomain that is interrupted by an intron between residues 44 and 45 as is the homeodomain of *lab*.

*Ba*¹

phenotype: Homozygous lethal; dies in first instar and has normal morphology. Heterozygote shows antenna-to-leg transformation; entire arista and part of third antennal segment transformed to distal metathoracic leg structures. Expression temperature sensitive; transformation complete at 19°; phenotype normal at 29°. *Ba/Df(2R)Ba4* shows abnormal segmentation both dorsally and ventrally and loss of head structures; antennal, maxillary, and labial segments affected. "H" piece malformed or absent as are Keilin's organs in all three thoracic segments. Recessive to two doses of 60D-F.

*Ba*³

phenotype: *Ba*³/+ wild type; *Ba*³/*Ba*¹ fail to eclose; pharate adults lack third antennal segment; second segment transformed to leg; distal arista normal. Legs lack all structures distal to tibiae; tibiae enlarge and bear ectopic bristles; no remaining leg bristles are bracted.

*Ba*⁵

phenotype: *Ba*⁵/+ like *Ba*¹/+. *Ba*⁵/*Ba*⁵ and *Ba*⁵/*Ba*¹ are late embryonic lethals; show loss of antennae, maxillary, and labial sense organs, as well as the "H" piece and Keilin's organs from all three thoracic segments. Occasional abnormalities in setal bands of segments posterior to prothorax; setal bands or spinules may be deleted or adjacent bands fused. Posterior end of larva normal.

*Ba*⁹

phenotype: Recessive allele. Homozygotes show partial transformation of antennae to legs as well as deletions of some leg structures. Tarsal tissue, sometimes including claws, develops in place of arista and part of third antennal segment. Third segment usually contains patchwork of incompletely differentiated leg tissue; first and second segments normal. Leg effects distal to mid tibia; legs proximal to mid tibia normal. In extreme cases number

of tarsal segments reduced to two or three; basitarsus and distal tibia may be missing; in most extreme cases claws may also be missing. In the case of mild expression abnormal bristle patterns, including polarity reversal, occur around basitarsus-tibia joint, where there may also develop cuticular hyperplasia. Penetrance greater in females than males, in antennae than legs, and in metathoracic than other legs. *Ba*¹/*Ba*^M die as pharate adults with extreme malformations of antennae and legs.

*Ba*¹²

phenotype: *Ba*¹²/+ wild type; recessive lethal. Heterozygote exhibits nearly complete suppression of extra-sex-combs phenotype of *Pc*⁴/+.

bab: see *y*^{bab}

Bag: see *Bg*

bag of marbles: see *bam*

Baksa: see *Fs(3)Sz3*

**bal*: bandy legged

location: 2- (not located).

origin: Spontaneous.

discoverer: Ströher, 1958.

references: Mainx, 1958, DIS 32: 82.

phenotype: Legs extremely shortened and crippled. All parts of legs from femur to tarsi shortened, broadened, and irregularly curved. Deformities most extreme in metathoracic legs. Movement unsteady and tottering. Manifestation increased by selection. Viability poor, especially in males; fertility good. RK2.

bald: see *ra*²

balding: see *bd*

bald: see *bld*

baldhead: see *bh*

ballet: see *bit*

balloon: see *ba*

Balloon: see *Bb*

balloon wing: see *bs*³

bam: bag of marbles

location: 3-{85}.

origin: Hybrid dysgenesis.

references: González, Molina, Casal, and Ripoll, 1989, Genetics 123: 371-77.

genetics: Homozygous males and females sterile.

cytology: Placed in 96A-E based on its inclusion in *Df(3R)L16 = Df(3R)96A1;96E*.

band: see *bn*

bandy legged: see *bal*

bang senseless: see *bss*

bang sensitive: see *bas*

Bar: see *B*

Bar + Bar: see *BB*

Bar double: see *BB*

bar eye: see *at*

bar-3: see *hh*

Bar-infra double: see $B^i B^i$

Barlike eye: see **Be**

baroid: see B^{bd}

barrel: see h^{brr}

Barsa: see $Fs(2)Sz1$

bas: bang sensitive (J.C. Hall)

location: 1-50.7 (based on 31 *m-f* recombinants).

origin: Induced by ethyl methanesulfonate.

references: Grigliatti, L. Hall, Rosenbluth, and Suzuki, 1973, *Mol. Gen. Genet.* 120: 107-14.

Ganetzky and Wu, 1982, *Genetics* 100: 597-614.

synonym: *bas-A*.

phenotype: Striking culture vial sharply on hard surface immobilizes *bas* flies for 30-40 seconds. Rapid recovery followed by refractory period of an hour. Suppressed by *nap^{ts}* at permissive temperatures; at temperatures above 37.5° *bas* flies quickly paralyzed; no detectable physiological abnormalities at neuromuscular junction in *bas* larvae (Ganetzky and Wu). In experiments on photoreceptor function, recovery of prolonged depolarization afterpotentials (induced by strong blue light) abnormally slow following exposure to orange light (Homyk and Pye, 1989, *J. Neurogenet.* 5: 37-48).

cytology: Included in neither $Dp(1;4)r^+ = Dp(1;4)13F;16A1-2$ nor $Df(1)sd72b = Df(1)13F1-14B1$; therefore *bas* to the left of 13F1. Placed in 12F by Homyk.

other information: Jan and Jan (1978, *Proc. Nat. Acad. Sci. USA* 75: 515-19) erroneously termed what is now *bss¹* a *bas* allele.

bas-B: see *bss*

Bashed: see $Mhc-m^5$

basket: see **bsk**

bat: bat

location: 2-71.0.

discoverer: Bridges, 22j26.

synonym: *ext-b: extended-b*.

phenotype: Wings extended and bent backward. RK2.

***baton: baton**

location: 2-52.

phenotype: Abdomen elongated with defective plates; eye resembles L^4 . Extremely inviable; most homozygotes die in larval and pupal stages, appearing as elongated corpses. Heterozygote shows some eye effect. RK3.

baz: bazooka

location: 1-56.7.

origin: Induced by ethyl methanesulfonate.

references: Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 296-307.

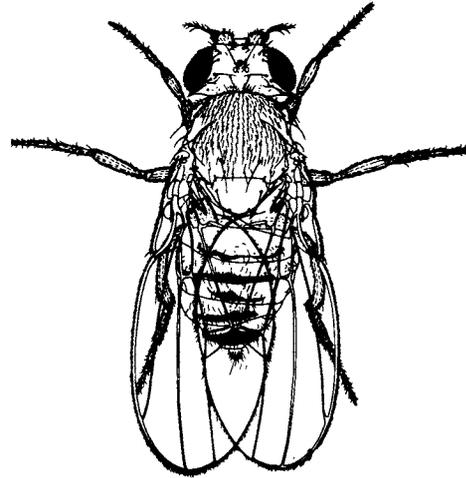
Wieschaus and Noell, 1986, *Wilhelm Roux's Arch. Dev. Biol.* 195: 63-73.

phenotype: Homozygous lethal, large dorsal and ventral hole in embryo. Homozygous *baz* germ-line clones undergo oogenesis, but the heterozygous progeny produced display major reductions in viability (Wieschaus

and Noell).

alleles: Three alleles, $baz^1 = D9$, $baz^2 = K7$, and $baz^3 = i^1$.

cytology: Placed in 14A1-B1, or 14E1-F6, or 15A6-16A2 based on its being covered by $Dp(1;4)exd^+82b = Dp(1;4)14A1-2;14B5-18 + 14E1-4;16A1-2$ but not included in $Df(1)r-D17 = Df(1)14F6;15A6$.



an extreme bobbed

Edith M. Wallace, unpublished.

bb: bobbed (K.D. Tartof and R.S. Hawley)

location: 1-66.0 (Bridges) and on *YS* (Stern, 1927; Cooper, 1959, *Chromosoma* 10: 535-88). Proximal to *ks-1* and *ks-2* (Kennison, 1981, *Genetics* 98: 529-48).

origin: Many alleles. In laboratory stocks, *bb* mutants may arise spontaneously or inadvertently while selecting for mutagen-induced mutations at other loci. *bb* mutations may be induced at high frequency by carcinogenic hydrocarbons such as 7,12-dimethyl benz[α]anthracene (DMBA) (Fahmy and Fahmy, 1969, *Nature* 224: 1328-29; 1970, *Mutat. Res.* 9: 239-43) or multifunctional alkylating agents such as triethylenemelamine (Tartof). Mutations of the X chromosome *bb* locus may be specifically induced genetically in four different ways. First, alterations of the X chromosome *bb* locus may occur *bb* in males or females carrying the aberrant chromosome Ybb^- . These are high frequency (10^{-2} - 10^{-1}) events and may be observed as either stable reversions from *bb* to bb^+ (magnification: Ritossa, 1968, *Proc. Nat. Acad. Sci. USA* 60: 509-16; Komma and Endow, 1986, *Genetics* 114: 859-74) or as mutations (10^{-3} - 10^{-2}) from bb^+ to *bb* or from *bb* to bb^1 (reduction: Tartof, 1973, *Cold Spring Harbor Symp. Quant. Biol.* 38: 491-500; Tartof, 1974, *Proc. Nat. Acad. Sci. USA* 71: 1272-76; Locker and Prud'homme, 1973, *Mol. Gen. Genet.* 124: 11-19). It has been suggested that both magnification and reduction may be the result of unequal sister-chromatid exchanges occurring at the X chromosome *bb* locus in the germ line of X/Ybb^- males (Tartof, 1974); evidence for this is from studies of ring-X *bb* chromosomes (Endow, Komma, and Atwood, 1984, *Genetics* 108: 969-83); alternatively, a model of excision and reintegration of circular molecules rDNA has been proposed by Ritossa [1972, *Nature (London)*, *New Biol.*

240: 109-11]. *C(1)RM* stocks carrying *Ybb*⁻ accumulate modifiers that suppress the *bb* phenotype (Marcus, Zitron, Wright, and Hawley, 1986, *Genetics* 113: 305-19). Second, in males carrying *mei-41*, *bb*⁺ mutates to *bb* at a frequency of 10⁻³-10⁻² (Hawley and Tartof, 1983, *Genetics*, 104: 63-80). *mei-41* does not induce reversions of *bb* to *bb*⁺ as does *Ybb*⁻ nor does it induce *bb* mutations on the *Y*. Third, when males carrying certain XY chromosomes are mated to females heterozygous for *Rex*, free *Y* chromosomes carrying *bb* mutants are generated at high frequency (10⁻²; Robbins, 1981, *Genetics* 99: 443-59). Fourth, hybrid dysgenic crosses may also generate *bb* (Thompson and Woodruff, 1980, *Proc. Nat. Acad. Sci. USA* 77: 1059-62). Despite such high frequency genetic mutations, homozygous stocks of *bb*², *bb*⁴, *bb*⁶, and *bb*⁸ have been maintained for 10 years with no evidence of reversions (Tartof). Unless otherwise stated, the origin of most *bb* mutations is unclear.

discoverer: Sturtevant, 20b.

references: Stern, 1927, *Z. Indukt. Abstamm. Vererbungsl.* 44: 187-231.

Ritossa, Atwood, and Spiegelman, 1966, *Genetics* 54: 819-34.

Ritossa, 1976, *Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 801-47.

phenotype: Three phenotypes are associated with bobbed mutants: thinning and shortening of bristles, etching of the abdomen, and, in extreme cases, lethality. Of these phenotypes, bristle size and lethality are the most reliable. These phenotypes are somewhat variable from fly to fly and the bristle abnormality may be obscured by such mutations as *f*, *sn*, and *ty* to name a few. *bb/0* males, and *bb/In(1)sc^{4L}sc^{8R}* females have phenotypes similar to, but more extreme than, that of homozygous females. *bb/Y* males are wild type, owing to presence of a normal allele of *bb* in *YS*; *bb/bb/Y* females are similarly normal in phenotype. Viability is variable. Ritossa, Atwood, and Spiegelman (1966) showed that *bb* contains about half as many ribosomal RNA genes (rDNA) as *bb*⁺. They conclude that the *bb* locus is the site of ribosomal RNA synthesis and interpreted *bb* mutations as partial deletions of the locus. They postulated that in *bb* flies the rate of protein synthesis is limited by the amount of ribosomal RNA, and the *bb* phenotype results in part because normal bristle production requires maximum protein synthesis on the part of the trichogen cells during a particular interval in development. The rate of rRNA synthesis is reduced in *bb* (Mohan and Ritossa, 1970, *Dev. Biol.* 22: 495-512; Mohan, 1975, *Genetics* 81: 723-38; Shermoen and Kiefer, 1975, *Cell* 4: 275-80)

alleles: Many independent occurrences of *bb* have been recovered and designated without a superscript; consequently, alleles designated as *bb* are often unrelated. We redesignate Sturtevant's original *bb*⁵ as *bb*¹, but, given the propensity for *bb* alleles to change spontaneously and the ambiguities of labeling, it is unlikely that any particular *bb* stock contains the original allele. The same arguments apply to *Ybb* originally described by Bridges. The accompanying tables summarize the specifically desig-

nated *bb* alleles on both the *X* and the *Y* chromosomes.

allele	origin	X-linked <i>bb</i> alleles discoverer	ref ^α	comments
<i>bb</i> ¹		Sturtevant 20b	2	
<i>bb</i> ²		Schultz	13	severe; ~120 genes
<i>bb</i> ^{2r}	reduction of <i>bb</i> ²	Tartof	14	lethal; ~20 genes
<i>bb</i> ^{2m}	magnification of <i>bb</i> ²	Tartof	14	normal; ~220 genes
<i>bb</i> ⁴		Schultz	13	standard <i>bb</i> ; ~160 genes
<i>bb</i> ⁵	not <i>bb</i> ⁵ of Sturtevant	Fahmy and Fahmy	3	
<i>bb</i> ⁶		Schultz	13	severe; ~150 genes
<i>bb</i> ⁷		Schultz	13	severe; ~140 genes
<i>bb</i> ⁸		Schultz	21	severe; ~100 genes
<i>bb</i> ¹¹	in <i>C(1)RM</i>	Gabritschevsky	2	
<i>bb</i> ²⁰	spont	Bridges 30b24	2	lethal
<i>bb</i> ^{28l}	spont		2	
<i>bb</i> ⁴¹	<i>mei-41</i> induced	Hawley	6	severe
<i>bb</i> ^{68F}		Lee	7	
<i>bb</i> ^{69d}	7-bromomethyl 12 methyl benz[α] anthracene	Fahmy and Fahmy	4	
<i>bb</i> ⁸³	in Binsc			
<i>bb</i> ⁸⁴	in Binsc			
<i>bb</i> ^{amg}				abnormal male genitalia
<i>bb</i> ^D	X ray	Lefevre 48g28	2	dominant allele
<i>bb</i> ^{ds}	spont	Goldschmidt	9	no longer deficiency sensitive; ~100 genes
<i>bb</i> ^{G1}	spont	Goldschmidt	2	
<i>bb</i> ^{G2}	spont	Goldschmidt	2	
<i>bb</i> ^{G3}	spont	Goldschmidt	2	
<i>bb</i> ^{G4}	spont	Goldschmidt	2	
<i>bb</i> ^{G5}	spont	Goldschmidt	2	
<i>bb</i> ^K	EMS	Merrakechi	5	
<i>bb</i> ^l	spont	Bridges, 1926	2	lethal; ~20 genes
<i>bb</i> ^{l2}	spont	Stem, 28k	2	
<i>bb</i> ^N		1		
<i>bb</i> ⁿ⁵				
<i>bb</i> ^{Of}	X ray		2	
<i>bb</i> ^{Of-2}	X ray		2	
<i>bb</i> ^{P2}			8	
<i>bb</i> ^{P7}			8	
<i>bb</i> ^{Rex}	Rex ^β	Robbins	11, 12	
<i>bb</i> ^{S1}		Schultz	13	~130 genes
<i>bb</i> ^{T6}	EMS		10	

^α 1 = CP552, 2 = CP627; 3 = Fahmy and Fahmy, 1970, *DIS* 45: 74; 4 = Fahmy and Fahmy, 1971, *Mut. Res.* 13: 19; 5 = Gans, Audit, and Masson, 1975, *Genetics* 81: 683-704; 6 = Hawley and Tartof, 1983, *Genetics* 104: 63-80; 7 = Lee, 1972, *DIS* 48: 18; 8 = Lenevea and Prud'homme, 1974, *European Drosophila Conference Abstracts* 4; 9 = Lindsley, Edington, and Von Halle, 1960, *Genetics* 45: 1645-90; 10 = Makai and Marrakechi, 1978, *Biol. Cell.* 33: 39a; 11 = Robbins, 1981, *Genetics* 99: 443-59; 12 = Swanson, 1984, Ph.D. dissertation, Michigan State University; 13 = Tartof, 1973, *Genetics* 73: 57-71; and 14 = Tartof, 1974, *Proc. Nat. Acad. Sci. USA* 71: 1272-76.

^β A group of *bb* alleles regularly produced by *Rex*-induced mitotic exchange in *YSX•YL* or in *X* chromosomes having separated blocks of rDNA (e.g. *In(1)w^{m51bl}w^{m4R}*; *In(1)sc^{51L}sc^{4R}*). Phenotype ranges from bobbed-lethal to wild-type.

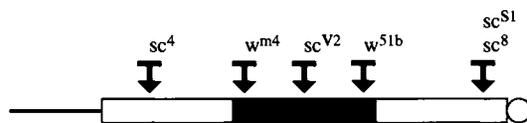
allele	origin	Y-linked <i>bb</i> alleles discoverer	ref ^γ	comments
<i>Ybb</i>	spont	Bridges	1	designated several unrelated <i>Y</i> -linked <i>bb</i> mutations
<i>Ybb</i> ⁻	spont	Schultz	1, 7	~40 genes; lacks <i>cr</i> ⁺
<i>Ybb</i> ⁻	spont	Schultz	2	~10 genes, mostly with type II

Allele	Origin	Number	Characteristics
<i>Ybb^{P2}</i>	Tartof		inserts, does not participate in nucleolus formation
<i>Ybb^{N2}</i>	spont? Ritossa	4	weak; viable over <i>bb¹</i> ; ~100 genes
<i>Ybb^{N3}</i>	spont Ritossa	4	weak; viable over <i>bb¹</i> ; ~100 genes
<i>Ybb^{Rex}</i>	Rex δ	5, 6	strong; viable over <i>bb¹</i> ; ~50 genes
<i>Ybb^{suVar}</i>		4	strong; viable over <i>bb¹</i> ; ~50 genes
<i>Ybb^{ts1}</i>	EMS Williamson	3	~117 genes; extreme <i>bb</i> at 18°; normal at 29°
<i>Ybb^{ts2}</i>	EMS Williamson	3	~127 genes; extreme <i>bb</i> at 18°; normal at 29°

γ 1 = CP627; 2 = Endow, 1982, *Genetics* 102: 91-99; 3 = Procnier and Williamson, 1974, *Dev. Biol.* 39: 198-109; 4 = Ritossa, 1968, *Proc. Nat. Acad. Sci. USA* 59: 1124-31; 5 = Robbins, 1981, *Genetics* 99: 443-59; 6 = Swanson, 1987, *Genetics* 115: 271-76; 7 = Tartof, 1973, *Genetics* 73: 57-71.

δ A regular product of *Rex*-induced mitotic exchange in progeny of crosses of *Rex*⁺ or *Rex*/*Rex* females to *YSX*•*YL*/0 males; *X/Ybb^{Rex}* males are fertile.

cytology: Shown to be to the right of 20E-F by Schalet and Lefevre [1976, *Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, vol. 1b, pp. 848-902)]. The *bb* locus lies approximately in the center of the proximal heterochromatin and accounts for about one-third of its length, or about 3.2×10^6 bp. The nucleolus organizer, located between heterochromatic regions *hB* and *hC* (Cooper, 1959), = *h29* of Gatti and Pimpinelli, and the rDNA are probably the same (Ritossa, Atwood, and Spiegelman, 1966). Presence of *bb*⁺ on *Y* chromosome postulated by Burlingame and demonstrated by Stern (1927). This *bb*⁺ is located on *YS* proximal to *ks-1* and *ks-2* (Kennison, 1981); it is located in the Hoechst-33258 dully fluorescing segment of *YS* designated h20 by Gatti and Pimpinelli (1983, *Chromosoma* 88: 349-73). The rDNAs of the *X* and *Y* chromosomes combine to form a single nucleolus in which the rDNA components of each chromosome can be discerned by electron microscopy (Schultz, 1965, *Brookhaven Symp. Biol.* 18: 116-47).



Positions of *X*-chromosome inversion breakpoints with respect to the rDNA. Thin line represents euchromatin and shaded box represents ribosomal DNA.

molecular biology: The *X* and *Y* chromosome *bb* loci each contain approximately 225 rRNA genes organized as a large tandem array of transcription units separated by nontranscribed spacers. (Ritossa *et al.*, 1966, Tartof, 1973, *Genetics* 73: 57-71). Copy number of *Y*-linked sequences varies over a six-fold range in *Y* chromosomes sampled from a natural population (Lyckegeard and Clark, 1989, *Proc. Nat. Acad. Sci. USA* 86: 1944-48). Sequences of nontranscribed spacers and external tran-

scribed spacers determined (Simeone, deFalco, Macino, and Boncinelli, 1982, *Nucleic Acid Res.* 10: 63-72; Simeone, LaVolpe, and Boncinelli, 1985, *Nucleic Acid Res.* 13: 1089-1101). There are three kinds of repeating units in the array as defined by digestion with the restriction enzyme, *EcoRI*. The first class is the functional 11-kb gene containing an external transcribed spacer followed by the sequence encoding 18S rRNA and then an internal transcribed spacer within which is the sequence encoding 5.8S rDNA; this is followed by the sequence encoding 28S rRNA, which is interrupted by a short transcribed spacer and finally there is a long non-transcribed spacer (White and Hogness, 1977, *Cell* 10: 177-92; Wellauer and David, 1977, *Cell* 10: 193-212; Pellegrini, Manning, and Davidson, 1977, *Cell* 10: 213-24). A second class is composed of repeats that contain a 0.5- to 5-kb insert known as Type I (White and Hogness; Wellauer and Dawid). The third class of repeats possesses a different insertion, Type II (Dawid, Wellauer, and Long, 1978, *J. Mol. Biol.* 126: 749-68). Type I and Type II insertions are located at different but nearby sites within the 28S coding region (Rioha, Miller, Woods, and Glover, *Nature (London)* 1981, 290: 749-53; see also Rae, 1981, *Nucleic Acids Res.* 9: 4997-5010). Type I and Type II containing genes appear to be transcriptionally inactive under normal conditions (Long and Dawid, 1979, *Cell* 18: 1185-96; Long, Rebert, and Dawid, 1980, *Cold Spring Harbor Symp. Quant. Biol.* 45: 667-72) and are intermingled at random with the noninsert-bearing genes throughout the array (Tartof and Dawid, 1976, *Nature* 263: 27-36; Hawley and Tartof, 1983, *J. Mol. Biol.* 163: 499-503). During magnification, however, all types of sequences appear to be transcribed (deCicco and Glover, 1983, *Cell* 32: 1217-25; Labella, Vicari, Manzi, and Graziani, 1983, *Mol. Gen. Genet.* 190: 487-93). The ribosomal genes of only one homolog are amplified in polytene nuclei of both males and females (Endow, 1980, *Cell* 22: 149-55).

A single rDNA gene is inserted ectopically into euchromatic locations by *P*-element transformation capable of high-level transcription in polytene chromosomes and micronucleolus formation; also able to partially alleviate effects of partial deletions of *bb* (Karpen, Schaefer, and Laird, 1988, *Genes Dev.* 2: 1745-63).



Map of basic repeating unit of ribosome DNA. Thick segments represent rDNA sequences; segments of intermediate width represent internal (ITS) and external (ETS) transcribed spacer sequences. Thin line represents non-transcribed (NTS) spacer sequences. Inverted triangle denotes positions of type I and type II inserts.

X and *Y* chromosome rDNA differ from each other in three important respects. (1) 65-70% of the *X* rDNA contains Type I-bearing genes (Wellauer, Dawid, and Tartof, 1978, *Cell* 14: 269-78). The *Y* contains type-I repeats detectable by *in situ* hybridization to metaphase chromosomes (Hatsumi and Endow, 1990); however, the major

type-I class is greatly reduced in amount in *Y* chromosomes (Tartof and Dawid, 1979, *Nature* 263: 27-30; Komma, Glass and Endow, 1990). (2) Yagura, Yagura, and Muramatsu (1979, *J. Mol. Biol.* 133: 533-47) showed that the 18S RNA transcribed from *X* rDNA differs from that of the *Y* by at least one base substitution. (3) Coen, Thoday, and Dover (1982, *Nature* 295: 564-68) found that the 5' end of the nontranscribed spacer is different in *X* and *Y* rDNA. These data indicate that there is little, if any, interchange of rDNA between *X* and *Y* chromosomes. However, rDNA is apparently responsible for regular pairing and segregation of *X* and *Y* chromosomes in males (McKee and Karpen, 1990, *Cell* 61: 61-72).

other information: Exchange between *Xh* and the *Y* chromosome at the bobbed locus results in *YSXh* and *X·YL* products that arise at a frequency of about 2×10^{-4} (for review, see Lindsley, 1955, *Genetics* 40: 24-44; Hawley and Tartof, 1983, *Genetics* 104: 63-80). Orientation of the *X* rDNA cluster has no effect on the frequency of exchange (Maddern, 1981, *Genet. Res.* 38: 1-7). Similar events occur in females [Williamson and Parker, 1976, *Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Vol. 1b, pp. 701-20]. Recombination events involving rDNA sequences of *X* chromosomes and *X* and *Y* chromosomes investigated using restriction-fragment-length polymorphisms (Williams, Kennison, Robbins, and Strobeck, 1989, *Genetics* 122: 617-24).

bb^{*a px sp*}: see *bb*^{*G1*}

bb^{*a px sp hi*}: see *bb*^{*G2*}

bb^{*poi*}: see *bb*^{*G3*}

bb^{*poi47*}: see *bb*^{*G4*}

b^{*poi hi*}: see *bb*^{*G5*}

bb^{*x*}: see *bb*^{*28l*}

Bb: Bubble

location: 1- (not located) or 3-48.

origin: X ray induced.

discoverer: R. L. King, 32d.

synonym: *Balloon*.

phenotype: Wings of heterozygous female smaller, trimmed, and inflated. Bubble in first posterior cell. In extreme cases and usually in males, the wing is a small inflated sac. Sexual difference in expression may indicate that *Bb* is on the *X*. Female fertile; male entirely sterile; therefore, homozygous females not obtainable. RK3A.

cytology: Associated with *T(1;3)Bb = T(1;3)13E;84F* (Morgan, Bridges, and Schultz, 1937, *Year Book - Carnegie Inst. Washington* 36: 301).

BB: Bar + Bar

origin: Spontaneous through unequal crossing over in *B/B* (see description of *B*).

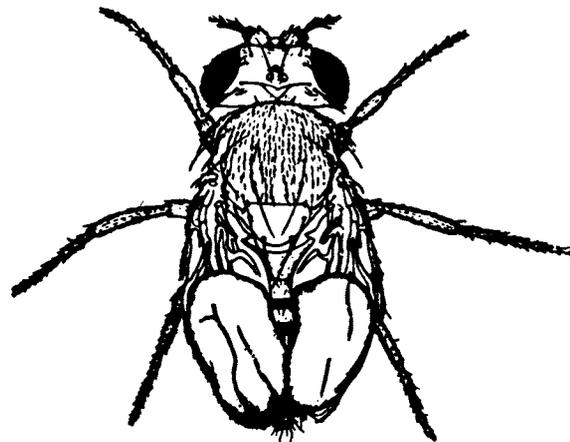
discoverer: Zeleny.

synonym: *Bar double*; *Ultra-bar*; *double Bar*.

references: 1920, *J. Exp. Zool.* 30: 292-324 (fig.).

Sturtevant, 1925, *Genetics* 10: 117-47 (fig.).

phenotype: Eye more reduced than in *B*. Facet numbers are 25, 29, and 45 in *BB/BB* female, *BB* male, and *BB/+* female, respectively. Median ocellus lacking or strongly



Bb: Bubble

From Bridges and Brehme, 1944, *Carnegie Inst. Washington Publ. No. 552: 23*.

reduced (Lefevre, 1941, *DIS* 14: 40). Optic disc reduced (Power, 1942, *Genetics* 27: 161); deep cleft anteriorly; cell clusters at cleft look mature (Renfranz and Benzer, 1989, *Dev. Biol.* 136: 411-29). RK1A.

alleles: Different alleles of *B* that are not involved in gross chromosome aberrations can be combined in all pair-wise combinations by means of unequal crossing over in heterozygous females. Described combinations are **BBⁱ*, **BⁱB*, and *BⁱBⁱ* (Sturtevant, 1925, *Genetics* 19: 117-47); **B^{i40b}B^{i40b}* (Steinberg, 1942, *DIS* 16: 53); **BB³*, *BB^{36b}*, and *BB^L* (Muller).

cytology: Associated with a tandem triplication of the region duplicated in *Dp(1;1)B = Dp(1;1)15F9-16A1;16A7-B1* [Bridges, 1936, *Science* 83: 210-11 (fig.)].

Bc: Black cells

location: 2-80.6.

origin: Induced by ethyl methanesulfonate.

references: Grell, 1969, *DIS* 44: 46.

Rizki, Rizki, and Grell, 1980, *Wilhelm Roux's Arch. Dev. Biol.* 188: 91-99 (fig.).

phenotype: Quadrate crystalline bodies in crystal cells of hemolymph replaced by amorphous melanotic mass. Black cells appear in 11-hour embryos in *Bc/Bc* and in late first-instar larvae in *Bc/+*. Crystal cells replaced by black cells during first instar in *Bc/+*. Numerous black cells visible through the integument of larvae, pupae, and of head, thorax, and abdomen in adults. *Bc/Bc* larvae have no phenol oxidase activity and larval hemolymph fails to darken upon exposure to air; *Bc/+* intermediate between *+/+* and *Bc/Bc* in these respects. *Bc/Bc* adults viable and fertile when separated from a closely linked simultaneously recovered lethal. RK1 in larvae; RK2 in pupae and adults.

cytology: Placed in 55A based on its inclusion in *Df(2R)PC4 = Df(2R)55A;55F* but not *Df(2R)Pd-w5 = Df(2R)55A-B;55C* (Deng and Rizki, 1988, *Genome* 30, Suppl. 1: 192).

other information: Suppressed by some *lz* alleles (Rizki and Rizki, 1981, *Genetics* 97: s90). *Dox-A1* and *Phox* reside in the same cytological interval.

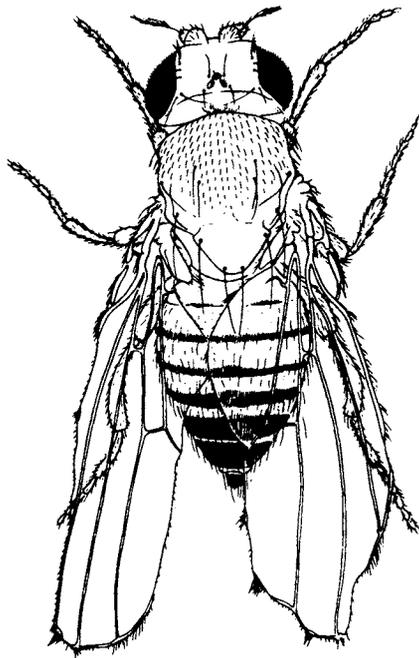
bch: branch

location: 3-46 (approximately).
origin: Induced by ethyl methanesulfonate.
references: Jürgens, Wieschus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.
phenotype: Homozygous lethal; incomplete fusion of denticle bands.

bd; see *ra*²

***bd: balding**

location: 2-56.1 (between *Bl* and *L*).
origin: Spontaneous.
discoverer: Ives, 71j25.
synonym: *bald* (preoccupied).
references: 1972, DIS 49: 38.
phenotype: Semicircular, medial bald spot on thorax, about one-third width of thorax. Very little variation at 25°. RK1.



Bd: Beaded

From Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 152.

Bd: Beaded

location: 3-91.9 [based on 102 *ro-ca* and 182 *Pr-ca* recombinants in crosses involving *Bd*^S (Curry, 1939, DIS 12: 46)].
references: Dexter, 1914, Am. Nat. 48: 712-58.
 Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 37, 152 (fig.).
 Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 45.
 Fleming, Scottgale, Diederich, Artavanis-Tsakonas, 1990, (manuscript).
phenotype: Originally recovered alleles were recessive lethal with a dominant incised-wing phenotype; *Bd*¹ was very weak and highly variable when first recovered, but

gained expressivity with selection; subsequently isolated alleles were stronger. Wings reduced by marginal excision both anteriorly and posteriorly. Phenotype of *Bd*¹/*Bd*¹/+ extreme (Peter Lewis). Expression and interaction studied by Goldschmidt and Gardner [1942, Univ. Calif. (Berkeley) Publ. Zool. 49: 103-24]. Expression of *Bd*¹, *Bd*³, and *Bd*^S suppressed by *H* (DIS 9) and *Ax* alleles (Bang). In combination with several different Minutes, causes incomplete development of anal and genital imaginal discs in males and less frequently in females (Goldschmidt, 1948, Proc. Nat. Acad. Sci. USA 34: 245-52; Sturtevant, 1949, Proc. Nat. Acad. Sci. USA 35: 311-13). *Bd*^S (originally designated *Ser: Serrate*) homozygous viable; initially thought to be homozygous lethal, but lethality removable by recombination (Belt, 1971, DIS 46: 116). The closely linked recessive lethal persists in many *Bd*^S-bearing chromosomes. Recessive lethal alleles, which lack the dominant wing phenotype, recovered as revertants of *Bd*^S (symbolized *Bd*^{Srv}) or selected on the basis of their failure to complement the lethality of *Bd*³ (symbolized *Bd*^r). Allelism of *Bd*^{r1} (originally designated *std: serratoid*) inferred from enhanced wing incising in heterozygotes with *Bd*^S and genetic map position similar to that of *Bd*^S; homozygous viability unknown. Cuticle preparations of embryos homozygous for *Bd*^S revertants reveal lack of germband retraction, improper deposition of cuticle, lack of head and thoracic structures, lack of Filzkörper, and in severe cases, only a remaining patch of cuticle (either ventral or dorsal). Central-nervous-system defects revealed by anti-horseradish peroxidase preparations include breaks in the longitudinal and/or commissure nerve tracts, twisted or unretracted nerve tracts, only a single nerve tract, and occasionally only the presence of groups of staining cells scattered throughout the embryo (Fleming, *et al.*). Each *Bd*^{Srv} allele displays the whole range of embryonic phenotypes but the proportions of individuals with a particular phenotype varies between alleles.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>Bd</i> ¹	spont	Morgan, 10e		1, 2 10	dominant wing phenotype; homozygous lethal; fails to complement <i>Bd</i> ³
* <i>Bd</i> ²	spont	Wallace, 15i10	<i>Bd</i> ^W	2	more extreme; ends of L3 and L4 split or disturbed
<i>Bd</i> ³	heat	Goldschmidt, 1934	<i>Bd</i> ^G	2, 3, 4	dominant wing phenotype; homozygous lethal; molecular polymorphisms at 0 to 1.0 and 14 to 17 kb
* <i>Bd</i> ⁴	spont	Goldschmidt	<i>Bd</i> ^{G45}	2, 6	more extreme scalloping effect
* <i>Bd</i> ⁵	X ray	Piternick, 1949	<i>Bd</i> ^P	2, 5	more highly penetrant; 100% when heterozygotes like extreme <i>Bd</i> ; variable; overlaps wild type
* <i>Bd</i> ⁶	X ray	Ohnishi, 49i16	<i>Bd</i> ⁴⁹	2, 11	
<i>Bd</i> ^{r1}	spont	Lee	<i>std</i>	7, 8	
<i>Bd</i> ^{r2}	EMS	Hecht	<i>Bd</i> ^{43.5}	3	homozygous lethal; no dominant phenotype
<i>Bd</i> ^{r3}	EMS	Hecht	<i>Bd</i> ^{862.5}	3	homozygous lethal; fails to complement <i>Bd</i> ³ ; no dominant phenotype
<i>Bd</i> ^{r4}	EMS	Hecht			homozygous lethal; fails to complement <i>Bd</i> ³ associated with <i>pll</i> ¹¹ ; possible small inversion in 97F region; molecular lesion at 17 to 19 kb
<i>Bd</i> ^{r5}	X ray			3, 9	fails to complement <i>Bd</i> ³ ; no dominant phenotype; <i>T(Y;3)R128 = T(Y;3)YS;97F;</i>

allele	origin discoverer	synonym	ref ^α	comments
<i>Bd^S</i>	spont Spencer, 3517	<i>Ser</i>	2, 3	breakpoint at 25 to 28 kb dominant wing phenotype; homozygous viable; 5.5-kb insert at 0 to 3.0 kb ^β
<i>Bd^{Srv1}</i>	X ray Fleming	<i>Ser^{rev2-3}</i>	3	homozygous lethal; complements <i>Bd^S</i>
<i>Bd^{Srv2}</i>	X ray Fleming	<i>Ser^{rev2-11}</i>	3	homozygous lethal; fails to complement <i>Bd^S</i> ; <i>In(3R)97F;98C</i>
<i>Bd^{Srv3}</i>	X ray Fleming	<i>Ser^{rev3}</i>	3	homozygous lethal; fails to complement <i>Bd^S</i> <i>T(2;3)56E;97F</i>
<i>Bd^{Srv4}</i>	X ray Fleming	<i>Ser^{rev5-5}</i>	3	homozygous lethal; fails to complement <i>Bd^S</i>
<i>Bd^{Srv5}</i>	X ray Fleming	<i>Ser^{rev6-1}</i>	3	homozygous lethal; fails to complement <i>Bd^S</i>

^α 1 = Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 37, 152 (fig.). 2 = CP627; 3 = Fleming, Scottgale, Diederich, Artavanis-Tsakonas, 1990, (manuscript); 4 = Gottschewski, 1935, DIS 4: 14, 16; 5 = Goldschmidt, 1953, J. Exp. Zool. 123: 79-114; 6 = Goldschmidt, 1942, Univ. Calif. (Berkeley) Publ. Zool. 49: 520; 7 = Lee, 1972, DIS 48: 18-19; 8 = Lee, 1973, Aust. J. Biol. Sci. 26: 189-99; 9 = Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, and Gould-Somero, 1972, Genetics 71: 157-84; 10 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 45; 11 = Ohnishi, 1950, DIS 24: 61.

^β More detailed description follows.

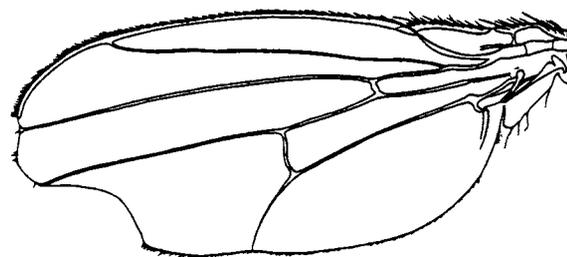
cytology: Placed in 97F by *T(2;3)Bd^{Srv3}* = *T(2;3)56E;97F*, *In(3R)Bd^{Srv2}* = *In(3R)97F;98C*, *Df(3R)Ser* = *Df(3R)97D;97F-98A1*, and *T(Y;3)128* = *T(Y;3)97F;YS*. Also *in situ* chromosomal studies displayed hybridization at 97F.

molecular biology: An 85 kb walk and Southern analysis showed that *Bd^G* and *Bd^S* contain molecular lesions in the 97F region (*Bd^S* contains a 5.5 kb repeated DNA sequence, and *Bd^G* has a complex rearrangement). Two major transcripts of 5.5 and 5.6 kb appear to differ at their 5' ends. Sequencing of two overlapping cDNAs produced a sequence of 5561 base pairs that contained a large open reading frame of 4329 nucleotides. Conceptual translation indicates a protein of 1404 amino acids that contains a signal peptide, a region of sequence homology to the neurogenic locus *Delta*, a partial EGF-like repeat, 14 EGF-like repeats with interruptions in the 4th, 6th, and 10th repeats, a transmembrane domain, and an intracellular domain of ~160 amino acids. Analyzing whole mount *in situs* using probes that recognize both transcripts, revealed a complex and dynamic pattern of embryonic mRNA expression in the head segments; the dorsal, ventral, and lateral epidermis in the thorax and abdomen; the proventriculus, and hindgut; the trachea; and a reiterated array of cells in the CNS. Expression appears to be limited to tissues of ectodermal origin (Fleming *et al.*, 1990).

other information: *Bd/In(3R)C*, *l(3)a* was the first described case of a balanced lethal [Muller, 1918, Genetics 3: 422-99 (fig.)].

Bd^S: Beaded-Serrate

phenotype: Wings of *Bd^{S/+}* and *Bd^{S/Df(3R)Ser}* notched at tip; deepest notch at second posterior cell. In triploids, one dose of *Bd^S* overlaps wild type. *Bd^S* is homozygous viable; initially thought to be homozygous lethal, but lethality removable by recombination (Belt, 1971, DIS 46: 116); the closely linked recessive lethal persists in



Bd^S: Beaded-Serrate

Edith M. Wallace, unpublished.

many *Bd^S*-bearing chromosomes. Homozygous *Bd^S* produces extreme incision of wing margins especially in second posterior cell (Belt, 1971). As with other *Bd* alleles expression suppressed by *H* and *Ax* (Bang).

bdw: see *os^{bdw}*

**be-3*: benign tumor in chromosome 3

location: 3-25.

origin: Spontaneous.

discoverer: Stark, 16k.

references: 1919, Proc. Nat. Acad. Sci. USA, 5: 573-80 (fig.).

Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 179 (fig.).

Stark and Bridges, 1926, Genetics 11: 249-66.

Stark, 1935, DIS 4: 62.

phenotype: Melanotic tumors appear in larvae and persist in adults. Subject to modification by genetic factors. Nonlethal. RK3.

Beaded: see *Bd*

Beadex: see *Bx*

Beadexoid: see *Bxd*

Bearded: see *Brd*

bef: beta lobes fused

location: 1- (not located).

origin: Induced by ethyl methanesulfonate.

synonym: *bef^{BG17b}*.

references: Heisenberg, 1980, Development and Neurobiology of *Drosophila* (Siddiqi, Babu, Hall, and Hall, eds.), pp. 373-90.

phenotype: Beta lobes of mushroom bodies (in anterior supraesophageal ganglion) are fused across midline. Mutant isolated on basis of this morphological defect in strain with aberrant visual responses and optic lobes, but genetic etiology of aberrant mushroom bodies is apparently separable from X-chromosome factor(s) causing the visual defects.

bel: belle (M.T. Fuller)

location: Just proximal to *p*.

synonym: *l(3)L3; ms(3)neo30*.

references: Bender, Turner, and Kaufman, 1987, Dev. Biol. 119: 418-32.

Jones and Rawls, 1988, Genetics 120: 733-42.

Deuring, Wolf, Schuske, Cooley, Spradling, and Fuller, (unpublished).

phenotype: Recessive larval lethal. Homozygous null individuals hatch, but remain as first instar larvae until their death several days later.

allele	synonym	origin	ref ^α	phenotype
bel¹	EMS	<i>belle</i> <i>b3</i>	1	larval lethal
bel²	EMS	<i>belle</i> <i>b10</i>	1	larval lethal
bel³	EMS	<i>belle</i> <i>b58</i>	1	larval lethal
bel⁴	EMS	<i>belle</i> <i>b68</i>	1	larval lethal
bel⁵	EMS	<i>belle</i> <i>eke24</i>	1	larval lethal
bel⁶	EMS	<i>belle</i> <i>pr134</i>	1	larval lethal
bel⁷	P	<i>ms(3)neo30</i>	2	male sterile ^β

^α 1 = Bender, Turner, and Kaufman, 1987, Dev. Biol. 119: 418-32; 2 = Deuring, Wolf, Schuske, Cooley, Spradling, and Fuller, (unpublished).

^β Male sterile with defects in meiosis and spermatid differentiation. Hemizygotes are more extreme than homozygotes, and poorly viable if raised at 18°. Female fertile, but hemizygous females sterile and produce eggs with weak chorion.

cytology: Placed in region 85A4-6 on the basis of the position of the P-element insert in *bel⁷*, and on the basis of being uncovered by *Df(3R)p7* but not *Df(3R)p5*.

molecular biology: The 4 kb *bel* transcript is present in unfertilized eggs, embryos, larvae, pupae, and adult males and females; it encodes a protein of 781 amino acids with a central core region of 414 residues that is highly homologous to *vasa* (Deuring *et al.*, unpublished). Based on this core homology, the *bel* gene product is a member of the DEAD box family and is likely to be a nucleic-acid-dependent ATPase.

bellyache: see *bly*

ben: *bendless* (J. Hall; R. Wyman)

location: 1-45.0 (1.2 cM to the left of *na*).

origin: Induced by ethyl methanesulfonate.

synonym: *nj-262*; *nonjumper-262*.

references: Thomas, 1980, Neurosci. Abstr. 6: 742.

Thomas and Wyman, 1982, Nature (London) 298: 650-51.

Thomas and Wyman, 1983, Cold Spring Harbor Symp. Quant. Biol. 48: 641-53.

1984, J. Neurosci. 4: 530-38.

phenotype: Adults have an aberrant startle response; they do not jump when presented with a lights-off stimulus. The cervical giant fiber, a brain neuron, has abnormal morphology. The normal giant fiber terminates in the thorax at a synapse onto the motoneuron innervating the tergo-trochanteral (TT) muscle (= jump muscle). In *ben*, this synapse is abnormal or absent. The normal lateral bend of the giant fiber toward the TT motoneuron in the mesothoracic neuromere is absent; the axon usually terminates at the midline with fine branches extending from its tip. Latency of response of the TT muscle to stimulation of the giant fiber is abnormally long, and muscles cannot follow stimulation at rates above 5 Hz. Rhabdomeres of the photoreceptor cells in the ommatidia heart shaped in cross section with indentation centrally oriented rather than round as is normal. Furthermore, the axons from photoreceptor cells R7 and R8 fail to make the right-angle turn into the optic medulla after traversing the lamina (Benzer). *ben* flies choose visible over ultraviolet wave lengths whereas wild-type flies make the opposite choice (Benzer).

alleles: Six ethyl-methanesulfonate-induced alleles; all are jumpless; all but *ben¹* have normal giant-fiber physiology (Tanouye).

allele	discoverer	synonym
ben¹	Wyman, and Thomas	
ben²	Tanouye	<i>ben</i> ¹³⁷²
ben³	Tanouye	<i>ben</i> ¹³⁹⁴
ben⁴	Tanouye	<i>ben</i> ¹⁴⁹⁹
ben⁵	Tanouye	<i>ben</i> ¹⁵⁶¹
ben⁶	Tanouye	<i>ben</i> ¹⁶⁸²

other information: *ben/Df(1)HA92* said to be indistinguishable from *ben/ben*, indicating that the mutation is amorphic (Thomas and Wyman, 1983).

cytology: Located within salivary gland chromosome region 12A6-B based on its inclusion in *Df(1)HA92 = Df(1)12A6-7;12D3* (Thomas and Wyman, 1984), but not the segmental deficiency *Y^PX^DB166/X^PY^DB89 = Df(1)12A-B;12B9-C1* (Tanouye).

ben(2)gcn: see *bgn*

bending wings: see *os^{bdw}*

bendless: see *ben*

benign gonial cell neoplasm: see *bgn*

benign tumor in chromosome 3: see *be-3*

bent: see *bt*

bent scutellars: see *bsc*

***ber:** *berrytail*

location: 1-52.4.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 67.

phenotype: Abdomen narrow, ending in a berry-like protrusion carrying defective genitalia. Wings opaque, with areas of deranged hairs (some with cut inner margins and interrupted or abnormally positioned longitudinal veins). Anterior scutellars often acutely bent; eyes occasionally misshapen. Males sterile and viability about 40% wild type. RK3.

Bercel: see *Fs(3)Sz4*

beta: see *Tyr2*

beta lobes fused: see *bef*

bf: *brief*

location: 3-95.

origin: Spontaneous.

discoverer: Curry, 38i3.

references: 1939, DIS 12: 45.

phenotype: Fly small; bristles Minute like. Classification perfect; viability fair. Male completely sterile; female with low fertility. RK3.

Bg: *Bag*

location: 1-51.6 (to the right of *sd*).

origin: Spontaneous.

discoverer: Bridges, 33d22.

phenotype: Heterozygous female with wings shorter and blunter, shortened L5, extra veins or gaps near anterior

crossvein, and inflated bag centering in first basal cell. Frequently overlaps wild type. Lethal in male. RK2 as a lethal; RK3 as a dominant.

alleles: Lost alleles described in CP627: **Bg*¹, **Bg*², **Bg*^{49h}, and **Bg*^{52c}.

cytology: Probably in 13C, based on *Bg*-like variegation of *Tp(1;3)ras*^v = *Tp(1;3)9E;13C;81F*.

other information: Possibly an allele of *Bb*, although published information suggests that *sd* lies between *Bb* and *Bg*.

***bgn*: benign gonial cell neoplasm**

location: 2- (between *dp* and *b*).

origin: Induced by ethyl methanesulfonate.

synonym: *ben(2)gcn*.

references: Gateff, 1981, DIS 56: 191.

phenotype: Gametocyte differentiation defective; gonial proliferation unchecked; gonads of both males and females become engorged with gonial cells. Ovarioles appear sausage like; testes smaller than normal. Autonomous in transplants of young adult ovaries and testes into body cavity of wild-type females.

***bh*: baldhead**

location: 3-81.

origin: Spontaneous.

references: Robertson, 1973, DIS 50: 24 (fig.).

phenotype: Ocelli and associated bristles absent; wings shortened. Both sexes sterile.

***bhe*: broad head (C. Nüsslein-Volhard)**

location: 2-0.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

phenotype: Embryonic lethal; embryos have incompletely involuted heads. In double mutants with *Pc*-like mutants, abdominal transformations occur. At least one *bhe* allele fails to complement *l(2)gl*, which is a larval lethal (Kennison).

alleles: *bhe*¹ (formerly *bhe*^{1J}) and *bhe*² (formerly *bhe*^{1M}).

cytology: Placed in 21A on basis of being covered by terminal 2L duplications that cover *l(1)gl*.

***bi*: bifid**

location: 1-6.9.

discoverer: Morgan, 11k.

references: Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. No. 237: 28 (fig.).

phenotype: Longitudinal veins fused at base of wing into bifid stalk. L3 delta-like at tip; L4 often incomplete at tip. Wing margins often excised at tip of L4. Wings spread in proportion to their shortness. High temperature enhances and low temperature produces overlapping of wild type. Stronger in male than in female. Hypomorphic allele; females heterozygous for *bi* and a deficiency for *bi* have outspread, crumpled, and very flimsy wings with an extreme bifid fusion of the basal regions of the longitudinal wing veins (Craymer and Roy, 1980, DIS 55: 204). Enhances *Bx* alleles as well as *sd*, *cp*, and *vg*^{np} (Waletzky). RK1.

alleles: **bi*³⁵ (CP627).

cytology: Placed in 4C5-6 based on its inclusion in *Df(1)rb13* = *Df(1)4C5-6;4D3-E1* but not in *Df(1)GA56* = *Df(1)4C5-6;4D1*, as well as the *bi* phenotype of

T(1;2)bi^{D2} = *T(1;2)4C5-6;46B5-7* and *T(1;3)bi*^{D1} = *T(1;3)4C5-6;65C3-5*. (Banga, Bloomquist, Brodberg, Pye, Larrivee, Mason, Boyd, and Pak, 1986, Chromosoma 93: 341-46).

***bib*: big brain**

location: 2-34.7.

references: Lehmann, Dietrich, Jiménez, and Campos-Ortega, 1981, Wilhelm Roux's Arch. Dev. Biol. 190: 226-29.

Lehmann, Jiménez, Dietrich, and Campos-Ortega, 1983, Wilhelm Roux's Arch. Dev. Biol. 192: 62-74.

phenotype: Recessive embryonic lethal, a neurogenic mutant. Homozygotes fail to form ventral, lateral, and most of the cephalic epidermis. Central nervous system hypertrophied by recruitment of presumptive epidermal cells, but exhibiting considerable architectural normality. Most epidermal sense organs can be recognized in electron microscope preparations; chordotonal organs prevalent but with abnormal structures, perhaps owing to disrupted attachment. Supernumerary peripheral elements formed by recruitment of presumptive epidermal cells [Hartenstein and Campos-Ortega, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 210-21 (fig.)]. Cuticular clones of cells homozygous for *bib*¹ or *bib*⁶ exhibit normal development (Dietrich and Campos-Ortega, 1984, J. Neurogenet. 1: 315-32). Effects on central nervous system intermediate with respect to mutations at other neurogenic loci. Insensitive to changes in dosage of other neurogenic genes (de la Concha, Dietrich, Weigel, and Campos-Ortega, 1988, Genetics 118: 499-508).

alleles: Eight alleles with similar phenotypes.

allele	origin	discoverer	synonym	ref ^α
<i>bib</i> ¹	EMS	Nüsslein-Volhard and Wieschaus	<i>bib</i> ^{ID05}	1, 2, 3
<i>bib</i> ²	EMS	Nüsslein-Volhard and Wieschaus	<i>bib</i> ^{IJ66}	3
<i>bib</i> ³	EMS	Nüsslein-Volhard and Wieschaus	<i>bib</i> ^{IIP39}	3
<i>bib</i> ⁴	EMS	Nüsslein-Volhard and Wieschaus	<i>bib</i> ^{IIV46}	3
<i>bib</i> ⁵	EMS	Nüsslein-Volhard and Wieschaus	<i>bib</i> ^{IIID118}	3
<i>bib</i> ⁶	EMS	Nüsslein-Volhard and Wieschaus	<i>bib</i> ^{6N37}	3
<i>bib</i> ⁷	X ray	Campos-Ortega	<i>bib</i> ^{FX1}	3
<i>bib</i> ^{8β}	X ray	Lehmann	<i>bib</i> ^{C7A}	3

^α 1 = Dietrich and Campos-Ortega, 1984, J. Neurogenet. 1: 315-32; 2 = Hartenstein and Campos-Ortega, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 210-21 (fig.); 3 = Lehmann, Jiménez, Dietrich and Campos-Ortega, 1983, Wilhelm Roux's Arch. Dev. Biol. 190: 226-29.

^β Associated with *In(2L)30A9;30F*.

cytology: Placed in region 27D-31E based on the lethal phenotype of the deficient segregant from *T(Y;2)B231* in combination with *bib*. *In(2L)bib*⁸ = *In(2L)30A9;30F* further restricts position to 30A9-F (Campos-Ortega).

molecular biology: Gene cloned and sequenced; conceptual amino-acid sequence indicates a gene product with 700 amino acids. The predicted *bib* product shows significant sequence similarity to a family of transmembrane proteins, some of which form channels permeable to small molecules (Rao, Jan, and Jan, 1990, Nature 345: 163-67).

bic: bicaudal

location: 2-67.0 (inferred from cytological position; maps to 68 with respect to *Bl* and *L*, but penetrance problems introduce uncertainty).

origin: Spontaneous.

references: Bull, 1966, J. Exp. Zool. 161: 221-42 (fig.).

Nüsslein-Volhard, 1977, Wilhelm Roux's Arch. Dev. Biol. 183: 249-68 (fig.).

1979, Symp. Soc. Dev. Biol. 37: 185-211 (fig.).

phenotype: Homozygous *bic* females produce variable numbers of eggs that fail to hatch (e.g., 25-50%). *bic/Df(2R)vg^B* females produce higher embryonic mortality. Males of either genotype are normal. A wide array of developmental anomalies observed among arrested embryos. Many fail to show outward signs of initiation of development, although in at least some zygotes nuclear divisions can be demonstrated. Others show replacement of anterior embryonic segments by a mirror-image set of posterior segments. The majority of these bicaudal embryos are symmetrical with opposing sets of the two to five posterior-most segments; mirror-imaged structures include denticle belts, posterior spiracles, Malpighian tubules, genital disc, and posterior midgut invagination. Polar cells form at the original but not the duplicated posterior end. Asymmetrical bicaudal embryos such as 3/6 or 3/7 have fewer duplicated than original posterior segments. Other embryos have the head replaced by posterior abdominal structures or show abnormal development of the cephalopharyngeal apparatus. Finally, some embryos are foreshortened and seem to lack internal segments. Maximum production of bicaudal embryos occurs in the first eggs produced by females developing at 28°-29°. The incidence of bicaudal embryos falls rapidly during the first 60 hr after eclosion. Incidence of bicaudal embryos and nondeveloping eggs depends on genotype: *bic/Df(2R)vg^B* > *bic/Df(2R)vg^D* > *Df(2R)vg^B/+* > *bic/bic* > *bic/+* = 0.

alleles: *bic⁴⁴* (Basel).

cytology: Placed between 49D3 and 49E6 based on the absence of normal allele in *Df(2R)vg^B* = *Df(2R)49D3-4;50A* and *Df(2R)vg^D* = *Df(2R)49C1-2;49E2-6*.

BicC: Bicaudal C

location: 2-52.0.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Jürgens, 1982, Verh. Dtsch. Zool. Ges. 91-104.

Mohler and Wieschaus, 1986, Genetics 112: 803-22.

phenotype: A maternal-effect semilethal; the majority of embryos produced by *BicC/+* mothers given rise to normal larvae a minority fail to hatch and vary in phenotype. Double-abdomen embryos have a normal posterior end and the anterior end replaced by mirror image series of posterior segments, e.g. A8-A4|A4-A8 to A8-A6|A6-A8; they need not have the same number of segments on either side of the reversal of polarity, the duplicated portion having fewer segments; generally more segments seen ventrally than dorsally. Less severely affected embryos may be headless, or with reduced mouth parts, or even normal appearing but failing to hatch. The incidence and severity of abnormal embryos vary with genetic background and temperature, the incidence being highest at 25° and decreasing at lower and higher temperatures. The majority of the embryos produced by

BicC/BicD trans heterozygotes are abnormal. Homozygotes are abnormal. Homozygous *BicC* females sterile, germ-cell differentiation blocked at the beginning of vitellogenesis. Follicle cells do not invade between oocyte and nurse cells; they do synthesize a chorion which remains open ended like a chalice or a cup; such eggs never fertilized.

alleles: Five ethyl-methanesulfonate-induced alleles; *BicC³* and *BicC⁵* more severe than the others.

allele	discoverer	synonym	ref ^α
<i>BicC¹</i>		<i>BicC^{IIF34}</i>	1, 2
<i>BicC²</i>	Nüsslein-Volhard	<i>BicC^{C96}</i>	1
<i>BicC³</i>	Schüpbach	<i>BicC^{RU35}</i>	1
<i>BicC⁴</i>	Schüpbach	<i>BicC^{WC45}</i>	1
<i>BicC⁵</i>		<i>BicC^{YC33}</i>	1, 2

^α 1 = Mohler and Wieschaus, 1986, Genetics 112: 803-22; 2 = Nüsslein-Volhard, Wieschaus, and Jürgens, 1982, Verh. Dtsch. Zool. Ges. 91-104.

cytology: Placed in 35D4-E6 based on the sterility of females carrying *BicC* and heterozygous for *Df(2L)osp29* = *Df(2L)35B3;35E6* but not *Df(2L)75c* = *Df(2L)35A1-2;35D4-7* (Mohler and Wieschaus).

BicD

location: 2-52.91 (to the right of *dl*; based on an estimated four recombinants between *dl* and *BicD* out of 4,000 tests).

references: Nüsslein-Volhard, Wieschaus, and Jürgens, 1982, Verh. Dtsch. Zool. Ges. 91-104.

Mohler and Wieschaus, 1986, Genetics 112: 803-22.

Steward and Nüsslein-Volhard, 1986, Genetics 113: 665-78.

Suter, Romberg, and Steward, 1989, Genes Dev. 3: 1957-68.

Wharton and Struhl, 1989, Cell 59: 881-92.

phenotype: A maternal-effect semilethal; substantial numbers of embryos produced by *BicD/+* mothers give rise to normal larvae; the remainder fail to hatch and vary in phenotype. The array of phenotypes encountered is the same as that produced by *BicC/+* females. The incidence and severity of abnormal embryos vary with genetic background and temperature, the incidence being highest at 18°, and decreasing at higher temperatures. *BicD* homozygotes, either homoallelic or heteroallelic, and *BicC/BicD* heterozygotes are female fertile, although producing eggs with fused or reduced chorionic appendages; the majority of their embryos are abnormal and the influence of temperature, if any, obscure. *BicD/+* duplication-bearing females produce few if any abnormal embryos, whereas *BicD/Df(2L)TW119* females produce more abnormal embryos than *BicD/+*, i.e., with respect to the severity of phenotype, *BicD/0* > *BicD/+* > *BicD/+* > *BicD/+*; however, simply a deficiency of *BicD* product does not account for the abnormal phenotype, since embryos produced by females carrying one dose of *BicD⁺* over a deficiency develop normally. Embryos produced by homozygous *BicD* females display symmetrical patterns of *cad⁺* polypeptide distribution during early development (Mlodzik and Gehring, 1987, Cell 48: 465-78).

Abnormal embryo production by *BicD/+* females enhanced by the heterozygosity for the mutant allele of or deficiency for *l(2)49*; Mohler and Wieschaus speculate that *l(2)49* is an allele of *bic*. In addition, eg, *stau*, *tor*,

and *trk*, maternal-effect mutants that affect early anterior but not posterior development, act as dominant enhancer of *BicD*; other maternal-effect mutants ineffective.

Bicaudal embryos exhibit nanos protein at both anterior and posterior poles and the absence of hunchback protein in both ends of the embryo; embryos produced by *BicD*¹/*BicD*²;*osk/osk* females do not express *nos*; display a burst of anterior, *bcd*-dependent *hb* expression not seen in bicaudal embryos, which is correlated with a dramatic expansion of *kni* expression (abdominal) and failure to express *Kr* (thorax and anterior abdomen). *BicD* expressed early in oogenesis; in wild type, protein first appears in the cytoplasm of all cells in 16-cell cysts in the middle of the germarium; upon entering the vitellarium, protein begins to accumulate in the oocyte and continues to do so for as long as observations are possible. The early embryos produced by such females show uniform distribution of *BicD* protein, which becomes localized to the cortical cytoplasm at blastoderm formation; anterior-to-posterior distribution remains uniform. Protein disappears at gastrulation. In *BicD*¹/*BicD*² females protein accumulation in the oocyte is precocious and greater than normal and the adjacent nurse cells may become visibly depleted; in the embryo, the protein appears to be concentrated as a cap over the anterior third and uniformly less concentrated in the remainder of the embryo. *BicD*^{rv1} females display extreme concentration of product in the presumptive oocyte and virtually none in the nurse cells; however, the presumptive oocyte never develops as an oocyte but remains nurse-cell like until the cyst degenerates.

alleles:

allele	origin	synonym	ref ^α	comments
<i>BicD</i> ¹	EMS	<i>BicD</i> ^{71.34}	3	TTC → ATC = lys ²²⁴ → glu
<i>BicD</i> ²	EMS	<i>BicD</i> ^{III E48}	3	GAG → AAG = ile ⁶⁸⁴ → phe
<i>BicD</i> ^r		<i>BicD</i> ^{PA66}	2	recessive female sterile allele
<i>BicD</i> ^{rv1}	X ray	<i>BicD</i> ^{7134R26}	1, 3	recessive female sterile; revertant of <i>BicD</i> ^r ; deletion of residues 376-379 superimposed on <i>BicD</i> ¹ lesion

^α 1 = Mohler and Wieschaus, 1986, Genetics 112: 803-22; 2 = Suter, Romberg, and Steward, 1989, Genes Dev. 3: 1957-68. 3 = Wharton and Struhl, 1989, Cell 59: 881-92.

cytology: Placed in 36C2-D1 based on its inclusion in *Df(2L)TW137 = Df(2L)36C2-4;37B9-C1*, but not *Df(2L)VA18 = Df(2L)36C4-D1;37C2-D1* (Steward and Nüsslein-Volhard).

molecular biology: The gene has been cloned and sequenced (Suter, Romberg, and Steward, 1989, Genes Dev. 3: 1957-68; Wharton and Struhl, 1989, Cell 59: 881-92); identified by transformation rescue of *BicD*^{rv1} female sterility. Two transcripts found in early embryos of 3.6 and 4.0 kb (Wharton and Struhl; 3.8 and 4.4 kb according to Suter *et al.*), which differ only in their 3' untranslated regions; transcription occurs from right to left. Gene contains at least eight exons; encodes a polypeptide of 782 amino acids and ~89 kd molecular mass; pI = 4.93. Sequence similar to that of the C-terminal half of myosin-heavy-chain polypeptides, and similar coiled-coil molecules, which contain long α helices that form double- or triple-chain coils. These species contain an underlying heptad amino-acid repeat in which residues one and four are generally hydrophobic; about

half of the *BicD* protein comprises such heptad repeats.

other information: Mohler and Wieschaus describe a polygenic strain, designated YC67 that parallels the behavior of *BicD* in all respects except that it is unmappable.

BicF

location: 3- (unmapped).

references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Dominant maternal effect giving bicaudal embryos in some genetic backgrounds (*e.g.*, with *ln(2LR)O*, *Cy l(2)DTS513*). High temperatures and short egg shape may enhance this effect. Homozygous phenotype is probably collapsed eggs.

bie: bientot (T. Schüpbach)

location: 2-97.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

phenotype: Maternal-effect female-sterile mutant; homozygous females lay eggs which show no visible signs of development when observed under transmitted light in a stereo microscope. These eggs are defective in fertilization or very early embryonic development. *bie*^{PV} sometimes gives rise to embryos which form irregular blastoderms and develop abnormally, producing fragmented pieces of cuticle.

alleles: Three, *bie*¹ - *bie*³ isolated as *RD*, *PV*, and *SD*, respectively.

***bifid*:** see *bi*

***big brain*:** see *bib*

***Billa*:** see *Fs(2)Sz2*

bip: bipolar oocyte

location: 3-10.

origin: Induced by ethyl methanesulfonate.

references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Female sterile; no eggs laid. Oocytes often have nurse-cell clusters at both ends (15 nurse cells altogether). Mature oocytes can have micropiles at both ends, no dorsal appendages and no polarity.

bis: bistre

location: 1-21+ (T. K. Johnson).

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)aminophenylalanine (CB. 3007).

discoverer: Fahmy, 1954.

references: 1958, DIS 32: 67.

phenotype: Very dark brown eye color; ocelli also dark.

Wings frequently unexpanded. Males sterile, but homozygous females fertile (T. Johnson). Viability varies from less than 10% to 70% wild type. RK2A.

cytology: Placed between 7B5 and 9 by Lefevre.

**Bit: Bitten*

location: 3- (not located; crossing over between *ru* and *th* almost completely suppressed).

origin: X ray induced.

discoverer: Lefevre, 48g5.

references: 1949, DIS 23: 58.

phenotype: Inner margin of wing indented. Wings, nor-

mally folded, appear to have had a bite taken out of the back. Marginal hairs present unlike *N* and *ct*. Flight is impeded, although little wing area lost. Homozygous lethal. RK1A.

cytology: Associated with *In(3L)Bit*; breakpoints not determined.

bithorax: see *bx* under *BXC*

Bithoraxlike: see *Ubx* under *BXC*

bithoraxoid: see *bx* under *BXC*

Bitten: see *Bit*

***bk: buckled**

location: 1-59.8.

origin: Induced by *p*-N,N-di-(2-chloroethyl)aminophenylethylamine (CB. 3034).

discoverer: Fahmy, 1955.

references: 1959, DIS 33: 83.

phenotype: Wings slightly altered in shape and frequently divergent; membranes warped between longitudinal veins. Veins slightly thickened at wing margins. Eye shape slightly altered. Scutellar bristles frequently abnormal, either inserted in base atypically, bent, or duplicated. Males viable and fertile. RK3.

alleles: **bk*² (CP627).

Bkd: Blackoid

location: 2-65 (Braun).

origin: Spontaneous.

discoverer: Goldschmidt, 1938.

phenotype: Body color black in homozygote, distinctly darker than wild type in heterozygote. RK2.

alleles: **Bkd*¹ (spontaneous, Goldschmidt, 1938); *Bkd*^M (induced by ethyl methanesulfonate + formaldehyde, Marsh and Mack, 1985, DIS 61: 214); allelism of *Bkd*^M inferred from phenotype and position between *cn* and *c*.

***bkl: buckledlike**

location: 1-59.9.

origin: Induced by *D*-*p*-N,N-di-(2-chloroethyl)aminophenylalanine (CB. 3026).

discoverer: Fahmy, 1955.

references: 1959, DIS 33: 83.

phenotype: Wings slightly divergent with membranes warped between longitudinal veins, which themselves are often slightly thickened. Abnormally-shaped eyes, frequently compressed dorsoventrally. Both sexes viable and fertile. RK3.

other information: Probably a complementing allele of *bk*. One X-ray-induced allele.

Bl: Bristle

location: 2-54.8 (crossing over may be reduced).

origin: Spontaneous.

discoverer: R. L. King, 25d11.

references: 1927, Biol. Bull. 53: 465-68.

phenotype: Bristles one-half to two-thirds normal length, blunt, thicker, and beaded in outline. Posterior scutellars often cross and adhere to body. Eyes somewhat larger and rougher. Overlaps wild type when reared at 20° (Ashburner). Probably affects nature of bristle secretion, particularly outer layer [Lees and Waddington, 1942, DIS 16: 70; Lees and Picken, 1945, Proc. Roy. Soc. (London), Ser. B 132: 396-423 (fig.)]. Viability of

heterozygote is good but erratic; homozygotes usually lethal; survivors female sterile with roughish eye character. RK1 as dominant.

alleles: **Bl*³⁰ and **Bl*³¹ (Plough and Ives, 1935, Genetics 20: 42-69).

cytology: Between 38A6 and 38E9 based on inclusion within *Df(2L)TW2 = Df(2L)37D2-E1;38E6-9* but not *Df(2L)TW9 = Df(2L)37E2-F4;38A6-C1* or *Df(2L)TW158 = Df(2L)37B2-8;37E2-F4* (Wright, Hodgetts, and Sberald, 1976, Genetics 84: 267-85).

***bla: bladderwing**

location: 1-43.2.

origin: Induced by *L*-*p*-N,N-di-(2-chloroethyl)aminophenylalanine (CB. 3025).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 67-68.

phenotype: Wings grossly deformed, small, and normally full of fluid. Eyes slightly abnormal in shape. Males fertile; females sterile. Viability about 50% wild type. RK3.

Bla: see *nw*^B

black: see *b*

Black cells: see *Bc*

black leg: see *bleg*

Blackoid: see *Bkd*

bladderwing: see *bla*

Blastoderm-specific gene: see *Bsg*

bhc: blocked (T. Schüpbach)

location: 2-43.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus.

phenotype: Female-sterile; homozygous females often have underdeveloped ovaries which seem to lack germ cells altogether. In some females a small number of developing egg chambers is found; may contain abnormal numbers of nurse cells, and never develop beyond the first few stages of oogenesis.

alleles: *bhc*^{WE} = *bhc*¹, *bhc*^{HM}.

bld: bald

location: 3-48.1 (in *3R* to the left of *Ki*).

origin: Induced by ethyl methanesulfonate; detected by the presence of X-ray-induced somatic spots in the progeny of treated males.

references: García-Bellido and Dapena, 1974, DIS 50: 179.

1974, Molec. Gen. Genet. 128: 117-30.

phenotype: Homozygous lethal (lethal may be independently induced); not cell lethal. In homozygous cuticular spots, chetae, trichomes and cuticle appear depigmented and transparent; trichomes long, thin, and wooly. RK1 as a marker for cuticular clones.

Bld: Blond

location: 1- or 2- (associated with rearrangement).

origin: Spontaneous in chromosome containing *In(2R)Cy*.

discoverer: Burkart, 1930.

references: 1931, Rev. Fac. Agron. Vet. Univ. Buenos Aires 7: 393-491.

Burkart and Stern, 1933, Z. Indukt. Abstamm.

Vererbungs. 64: 310-25 (fig.).

phenotype: Bristles of heterozygote are gleaming yellow at tips and for varying lengths of more basal regions. Hairs not much paler and bristles of abdomen only slightly affected. Larval mouth parts wild type. No overlap. ERG normal [Hotta and Benzer, 1969, Nature (London) 222: 354-56]. Viability and fertility excellent. Most available *T(1;2)Bld* chromosomes carry a lethal in *2R*; lethal easily separated from translocation by recombination in *T(1;2)Bld/In(2R)Cy* females.

cytology: Associated with *T(1;2)Bld* = *T(1;2)1B13-C1;60B12-13* (Lefevre).

other information: *Bld* phenotype associated with the $2R^{DX^P}$ element of the translocation.

Ble: Barlike eye

location: 3-94.

origin: X ray induced.

discoverer: Crowell, 57i.

references: Meyer, 1958, DIS 33: 97.

phenotype: Eye shape indistinguishable from Bar. Expression of *Ble/+* varies, best at 26°. Excellent expression in homozygote at all temperatures. *Ble/Ble* in combination with *B* results in an extremely narrow eye. RK1.

other information: If *Ble* represents a transposition of the Bar locus to chromosome 3, the flanking loci of f^+ and od^+ have not been transposed. Also against transposition is absence of sexual dimorphism that dosage compensation of *B* should produce in such a case.

***bleg: black leg**

location: 3- (near *p*).

discoverer: Bridges, 16b23.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 158.

phenotype: Legs black; body color pallid; wings flimsy. RK3.

blistered: see bs

Blisterlike: see Bsl

blistery: see by

blo: bloated

location: 2-58.5.

origin: Recovered among descendants of heat-treated flies.

discoverer: Ives, 33f26.

synonym: Originally referred to as ba^2 : *balloon* and ba^{33f26} .

references: Plough and Ives, 1934, DIS 1: 33.

1934, DIS 2: 10.

1935, DIS 3: 6.

Bridges, Skoog, and Li, 1936, Genetics 21: 788-95.

phenotype: Wings spread, crumpled, and vesiculated; wing shows irregular plexus of extra veins. In extreme cases, wings unexpanded. Occasional hooked or wavy bristles. Developmental studies by Waddington [1939, Proc. Nat. Acad. Sci. USA 25: 299-307 and 1940, J. Genet. 41: 75-139 (fig.)] show intervein material spongy and veins swollen with inadequate contraction after inflated stage of pupal wing. Droplets of hemolymph often become clothed with cells liberated from epithelium and remain along basal processes. Does not overlap wild type but has poor viability and hatches later. RK2.

cytology: Not included within and does not recombine with (0/1098) *Df(2R)Np* = *Df(2R)44F1-2;45E1-2* (Bridges, Skoog, and Li, 1936).

blocked: see bic

Blond: see Bld

blot: see ap^{blt}

***blt: ballet**

location: 1- (not located).

origin: X ray induced.

discoverer: Iyengar.

references: 1962, DIS 36: 38.

phenotype: Wings one-third the normal length, stretched outward and slightly upward; wing tip broadened; venation markedly altered as in fused. Male viability impaired; females almost completely lethal. RK2.

blt: see ap^{blt}

***blu: blunt**

location: 3- (near *ru*).

origin: Spontaneous.

discoverer: Walbrunn, 46j23.

references: 1947, DIS 21: 71.

phenotype: Wings slightly shorter and broader than normal, giving a squared appearance. Sometimes difficult to classify. RK3.

Blunt short bristle: see Bsb

bly: bellyache

location: 1-28.3.

origin: Induced by ethyl methanesulfonate.

references: Eberl and Hilliker, 1988, Genetics 118: 109-20.

phenotype: Recessive lethal. Dispersed or poorly formed Malpighian tissue and often a yolk plug; cuticle appears normal.

alleles: Two putative alleles, bly^1 and bly^2 ; isolated as $l(1)EH290$ and $l(1)EH740a$.

***bn: band**

location: 3-72.

origin: Spontaneous.

discoverer: Morgan, 12g.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 79 (fig.).

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 215 (fig.), 218.

phenotype: Trident pattern and scutellum darker with dark transverse band across anterior portion of mesonotum. Thorax vacuolated; hairs on thorax sparse and directed medially in bowed lines. RK2.

bo: bordeaux

location: 1-12.5.

discoverer: Nazarenko.

phenotype: Eye color dark wine; not completely separable from wild type. Transplantation indicates *bo* may be nonautonomous (Ephrussi and Beadle, 1937, Genetics 22: 65-75). Larval Malpighian tubules bright yellow (Beadle, 1937, Genetics 22: 587-611). RK3.

other information: CalTech stock, *bo v*, contains *w* alleles. Schalet thinks *bo* is a dark allele of *w*; *bo/w^a* looks like dark allele of *w* series.

bobbed: see *bb*

bobbed on the Y chromosome: see *bb^Y*

bod: bowed

location: 3-48.3.

origin: Spontaneous.

discoverer: Nichols-Skoog, 35b20.

references: 1937, DIS 7: 6.

phenotype: Wings bowed downward over abdomen with curvature along both axes; curvature occasionally reversed. Wings somewhat smaller than wild type. Whole fly smaller and humpy; eyes slightly bulged. Overlaps wild type slightly. Viability 75% wild type. RK3.

Bojla: see *Fs(3)Sz5*

***bord: bordered**

location: 1-70.

origin: Spontaneous.

discoverer: Bridges, 1916.

references: Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 220.

phenotype: Wings smaller and slightly extended; venation ragged; veins bordered by darker bands. Viability poor; classification unreliable. RK3.

bordeaux: see *bo*

bordered: see *bord*

***bos: bordosteril**

location: 3-0.0.

origin: Spontaneous.

discoverer: Fabian, 1941.

references: 1948, *Arch. Julius Klaus-Stift. Vererbungsforsch. Sozialanthropol. Rassenhyg.* 23: 512-17.

phenotype: Eye color dark brownish red, darkens with age. Malpighian tubules and testis sheaths colorless. Male fertile; female sterile. RK2.

other information: Possibly an allele of *she*.

boss: bride of sevenless

location: 3.90.5 (just proximal to *Pr*).

references: Reinke and Zipursky, 1988, *Cell* 55: 321-30.

phenotype: Homozygotes lack photoreceptor cell R7; phenotype indistinguishable from that of *sev*. Mosaic studies demonstrate that *boss⁺* activity required in R8 and in no other photoreceptor cell for the normal development of the R7 cell in the same ommatidium. *boss⁺* R8 cell cannot rescue R7 development in adjacent ommatidia.

alleles: Sixteen alleles induced by X rays, ethyl methanesulfonate, and hybrid dysgenesis.

cytology: Placed in 96F5-14 by deficiency mapping.

bot: botch (T. Schüpbach)

location: 2-20.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1989, *Genetics* 121: 101-17.

phenotype: Maternal-effect female-sterile mutant; embryos from homozygous mothers do not hatch and show irregular segmentation, variable segment fusions, and holes in cuticle.

alleles: *bot^{Q1}* = *botⁱ*.

Botond: see *Fs(3)Sz6*

***bow: bow wings**

location: 1- (not located).

discoverer: Bridges, 12h15.

references: Morgan and Bridges, 1916, *Carnegie Inst. Washington Publ. No. 237*: 46 (fig.).

phenotype: Wings curved downward over abdomen and also sideways, like bowl of a spoon. Overlaps wild type. RK3.

Bow: Bowed (M. Ashburner)

location: 2-55.1.

origin: Induced with ethyl methanesulfonate.

discoverer: Detwiler.

phenotype: Wings of heterozygotes at 45° to body axis; macrochaetae, especially scutellars, with hooked tips.

cytology: Cytologically normal (Ashburner).

bow-legged: see *bwl*

bowed: see *bod*

Bowed: see *Bow*

bp: see *bul^{bp}*

br: see *BRC*

***Br: Bridged**

location: 1- (right half; crossing over suppressed to the right of *v*).

origin: X ray induced.

discoverer: Muller, 2713.

references: 1935, DIS 3: 29.

phenotype: Plexus-like wings with extra crossveins bridging longitudinals. L4 bent. Wings arched. Male lethal. RK3A.

cytology: Associated with *In(1)Br*.

Br: see *Sp*

brachymacrochaetae: see *brc*

brahma: see *brm*

braille: see *brl*

bran: see *a^{ba}*

Bran: see *a^{Ba}*

branch: see *bch*

Branchlet: see *Bt*

***brb: broad abdomen**

location: 1-52.9.

origin: Induced by styrylquinoline (CB. 3086).

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 83.

phenotype: Fly with broad abdomen and slightly shortened thorax and wings. Wings frequently slightly divergent. Eyes small and dull red with reflection spots. Bristles slightly shortened and lying flatter on thorax. Males and females viable and fertile. RK2.

alleles: One allele induced by *L-p-N,N-di-(2-chloroethyl)amino-phenylalanine*.

brc: brachymacrochaetae

location: 1-0.0 [no recombinants with *sc* among 6746 sons; placed between *su(s)* and *tw* by duplication analysis (Maddern, 1972, DIS 49: 40); placed just proximal to *tw* between *l(1)1Da* and *l(1)1Dc*, by Voelker, using terminal deficiencies].

origin: Induced by triethylenemelamine (CB. 1246).

discoverer: Fahmy, 1952.

references: 1958, DIS 32: 68.

phenotype: One or more thoracic bristle much reduced in size; scutellars and dorsocentrals most frequently affected. Occasional bristles duplicated. Extra-bristle phenotype enhanced by duplications for the tip of X with breakpoint between *su(s)* and *tw* (Maddern, 1972, DIS 49: 40). Good viability and fertility in both sexes. *brc*⁶, a lethal allele, causes pupal death with substantial pupal histolysis, especially at anterior end (Eberl, Hilliker, and Voelker, 1988, DIS 67: 36).

alleles: Majority of alleles are lethal.

allele	origin ^α	discoverer	synonym	ref ^β	comments
<i>brc</i> ¹	TEM	Fahmy, 1952		1	viable
<i>brc</i> ²	CB. 3025	Fahmy, 1952		1	viable
<i>brc</i> ³	X ray	Fahmy, 1952		1	viable
<i>brc</i> ⁴	X ray	Lefevre	<i>l(1)C93</i>	2	
<i>brc</i> ⁵	EMS	Lefevre	<i>l(1)VA23</i>	3, 4	polyphenic; no maternal effect
<i>brc</i> ⁶	DCE	Kramers	<i>l(1)DCE12</i>		
<i>brc</i> ⁷	ENU	Voelker	<i>l(1)B1</i>		
<i>brc</i> ⁸	ENU	Voelker	<i>l(1)B5</i>		semilethal; <i>brc</i> phenotype
<i>brc</i> ⁹	ENU	Voelker	<i>l(1)B20</i>		semilethal; <i>brc</i> phenotype
<i>brc</i> ¹⁰	ENU	Voelker	<i>l(1)B29</i>		
<i>brc</i> ¹¹	ENU	Voelker	<i>l(1)B43</i>		
<i>brc</i> ¹²	ENU	Voelker	<i>l(1)B45</i>		

^α 1 = Fahmy, 1958, DIS 32: 68; 2 = Lefevre, 1971, Genetics 67: 497-513; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 4 = Per-rimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14.

BRC: Broad Complex

location: 1-0.28 (left of *dor*); mapped to 1-0.43 based on 15 crossovers between *npr*⁴ and *y*, 36 between *npr*⁴ and *w*, and 159 between *npr*⁴ and *ec* (Belyaeva *et al.*).

synonym: *o.c.c.*: overlapping complementation complex; *ecs*: ecdysone sensitivity (Zhimulev, Belyaeva, Fomina, Protopopov, and Bolshakov, 1987, Chromosoma 94: 492-504).

references: Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelova, and Zhimulev, 1980, Chromosoma 81: 281-306.

Belyaeva, Protopopov, Baritcheva, Semeshin, and Izquierdo, 1987, Chromosoma 95: 295-310.

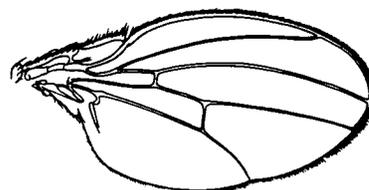
Kiss, Beaton, Tardiff, Fristrom, and Fristrom, 1988, Genetics 118: 249-57.

genetics: The Broad Complex resides in the early ecdysone-induced puff at the left end of the X chromosome; it comprises a number of mutations with complicated phenotypic and complementation characteristics. Russian investigators originally defined four mutually complementing, lethally mutable loci, which function in ecdysone-dependent induction of metamorphosis: *br* = broad, *rbp* = reduced bristles on palpus, *l(1)2Bc*, and *l(1)2Bd*. Based on amorphic mutations, defined on the basis of having equivalent phenotypes in homozygous and hemizygous females, Kiss *et al.* define two complementation groups: *br* [comprising *br*, *rbp*, and *l(1)2Bd*

(Belyaeva *et al.*)] and *l(1)2Bc;npr* alleles are non-complementing. In the following treatment we retain the subdivisions as defined by Belyaeva *et al.*; however, as the molecular structure of the complex becomes understood, simpler terminology will be indicated.

cytology: Placed in 2B5 based on its deletion by *Df(1)S39* = *Df(1)1E3-4;2B5* and by the deficiency within *Dp(1;Y)S280* = *Df(1)2B4-5;2B6-7*; *Df(1)S39/Dp(1;Y)S280* males die as puffless third-instar larvae (Belyaeva, Vlassova, Biyasheva, Kakpakov, Richards, and Zhimulev, 1981, Chromosoma 84: 207-19).

molecular biology: Region entered by transposon tagging (Chao and Guild, 1986, EMBO J. 5: 143-50) and by microdissection (Galcerán, Jiménez, Edström, and Izquierdo, 1986, Insect Biochem. 16: 249-54). Chromosomal walk of 230 kb includes polytene bands 2B1-6; coordinate 0 defined as the most distal point of the walk, with positive values extending to the right. Mutational lesions are spread over 50 kb and are confined to two disjunct regions, the distal one is between 100 and 115 kb and the proximal is between 150 and 175 kb (Sampedro, Galcerán, and Izquierdo, 1989, Mol. Cell Biol. 9: 3588-91); both *br* and *l(1)2Bc* lesions are found in the proximal group. The region is transcriptionally complex; transcripts from the distal region seem unrelated to *BRC* function, whereas, the proximal region produces an array of transcripts that display alternative initiation and termination sites as well as alternatively spliced exons which show stage specificity, with constitutive and early transcripts originating at coordinate 143 kb and late transcripts at 165 kb (Galcerán, Llanos, Sampedro, Pongs, and Izquierdo, 1990, Nucleic Acids Res. 18: 539-45).



br: broad

Edith M. Wallace, unpublished.

br: broad

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 145, 220 (fig.).

phenotype: The *br* complementation group contains both amorphic and hypomorphic mutant alleles; amorphic alleles cause early prepupal developmental arrest; hypomorphic alleles cause late pupal or pharate adult developmental arrest or are viable. Null alleles display normal larval development but prevent elongation and eversion of discs giving rise to appendages in the pupal stage. Wings of the viable allele, *br*¹, somewhat broader than normal; about 80% of normal length, with round full tip; crossveins closer together. Shape difference visible in middle prepupal stage immediately after eversion [Waddington, 1939, Proc. Nat. Acad. Sci. USA 25: 299-307; 1940, J. Genet. 41: 75-139 (fig.)]. A haplo-insufficient locus in that heterozygosity for a deficiency including the

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br locus leads to a slight *br* phenotype (Craymer and Roy, 1980, DIS 55: 200-04); furthermore, the deficiency in combination with *br*¹ or *br*³ leads to drastic reduction in viability, especially at 18°, and an extreme phenotype among survivors, including reduced palpi characteristic of *rbp* alleles, short rounded wings with interrupted veins, and malformed third legs, *i.e.*, shortened and thickened femora and tibiae as well as misshapen basitarsi. The malformed-leg syndrome is enhanced by heterozygosity for *Sb* or *sbd* alleles (Beaton, Kiss, Fristrom, and Fristrom, 1988, Genetics 120: 453-64). *br*¹⁶/+ and *Df(1)S39*/+ display slight dominance of *br* effects in the presence of *Rpl1215*^{Ubl} (Mortin and Lefevre, 1981, Chromosoma 82: 237-47).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>br</i> ¹	spont	Bridges, 15i26		4, 5, 7, 13	viable; 412 insert at 164.4 kb
<i>br</i> ³	spont	Bridges, 31e1		4, 5, 13	viable; 412 insert at 164.4 kb + B104 insert at 159.4 kb
<i>br</i> ⁴	DNA	Fahmy	<i>br</i> ^{59j}	4, 5	viable; inserts at 109 and 115 kb + small deletion between 102 and 104 kb
<i>br</i> ⁵	EMS		<i>l(1)t35</i>	1, 2, 7, 8	amorph, prepupal lethal
<i>br</i> ⁶	EMS		<i>l(1)t103</i>	1, 2	<i>ln(1)2B3-4;3C1</i> , hypomorph, pupal lethal; breakpoint at 146 to 151 kb
<i>br</i> ⁷	EMS		<i>l(1)t336</i>	1, 2, 7	
<i>br</i> ⁸	EMS		<i>l(1)t366</i>	1, 2, 8	amorph, prepupal lethal
<i>br</i> ⁹	γ ray	Pak	<i>l(1)t476</i>	1, 2	viable
<i>br</i> ¹⁰	X ray	Lefevre	<i>l(1)A18</i>	9	<i>T(1;2)2A4;60E8-9</i>
<i>br</i> ¹¹	X ray	Lefevre	<i>l(1)A64</i>	9	<i>T(1;2)2B7;42</i>
<i>br</i> ¹²	X ray	Lefevre	<i>l(1)C123</i>	9	<i>T(1;2)2B6;54A</i>
<i>br</i> ¹³	X ray	Lefevre	<i>l(1)GA106</i>	9	<i>ln(1)2B6;20F</i>
<i>br</i> ¹⁴	X ray	Lefevre	<i>l(1)GE222</i>	9	<i>T(1;3)2B6;83E</i>
<i>br</i> ¹⁵	X ray	Lefevre	<i>l(1)GE246</i>	9	<i>ln(1)2B6;3D5-6</i> (complex)
<i>br</i> ¹⁶	X ray	Lefevre	<i>l(1)GF325</i>	4, 11	<i>Tp(1;2)2B7;7D;36C</i>
<i>br</i> ¹⁷	X ray	Lefevre	<i>l(1)N34</i>		<i>ln(1)2B6;4D6</i>
<i>br</i> ¹⁸	EMS	Lefevre	<i>l(1)VE662</i>	10	
<i>br</i> ²⁰	EMS	Lefevre	<i>l(1)VE736</i>	10	
<i>br</i> ²¹	EMS	Lefevre	<i>l(1)VE891</i>	10	<i>ln(1)2B6-7;20A</i>
<i>br</i> ²²	EMS	Lefevre	<i>l(1)VE901</i>	10	<i>T(1;2)2B;25E-F</i>
<i>br</i> ²³	spont	Schalet	<i>l(1)14-153</i>		
<i>br</i> ²⁴	spont	Schalet	<i>l(1)20-68</i>		
<i>*br</i> ^{24h}	spont	L.V. Morgan		4	viable
<i>br</i> ²⁵	X ray			7	<i>Df(1)1E1-2;2B4-5 = Df(1)pn7b</i> breakpoint at 144.5 to 147 kb
<i>br</i> ²⁶	DEB		<i>br</i> ^{JTD1}	7	<i>Df(1)1D-E;2B5</i> ; hypomorph
<i>br</i> ²⁷	DEB		<i>br</i> ^{JTD2}	7	viable, hypomorph
<i>br</i> ²⁸	HD		<i>br</i> ^{32110B}	13	0.2 kb deletion at 102 to 104 kb
<i>br</i> ²⁹	X ray		<i>br</i> ^{X195}	13	1.5 kb insert at 174 kb
<i>*br</i> ^{33a}	spont	L.V. Morgan		4	viable
<i>*br</i> ³⁷¹	spont	Bridges		4	viable
<i>br</i> ^D	spont	Muller, 19h		12	<i>D = Dominant</i>
<i>*br</i> ^{1-a}	spont	Muller, 19h		12	
<i>*br</i> ^{1-b}	spont	Muller, 19h		12	
<i>*br</i> ^{sh}	spont	Bridges, 14g20		3	viable; <i>sh</i> = short
<i>*br</i> ^{uq}	spont	Fahmy, 1955		6	viable; <i>uq</i> = unequal wings
<i>*br</i> ^w	spont	Duncan		4	viable; <i>w</i> = wide

^α 1 = Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90; 2 = Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelova, and Zhimulev, 1980, Chromosoma 81: 281-306; 3 = Bridges, 1916, Genetics 1: 151; 4 = CP552; 5 = CP627; 6 = Fahmy, 1958, DIS 32: 77; 7 = Kiss, Beaton, Tardiff, Fristrom, and Fristrom, 1988, Genetics 118: 247-59; 8 = Kiss, Szabad, Belyaeva, Zhimulev, and Major, 1980, Development and Neurobiology of *Drosophila* (Siddiqi, Babu, Hall, and Hall, eds.) Plenum Press, New York and London, pp. 163-81; 9 = Lefevre, 1981, Genetics 99: 460-80; 10 = Lefevre; 11 = Morton and Lefevre, 1981, Chromosoma 82: 237-47; 12 = Muller and Altenburg, 1921, Anat. Rec. 20: 213. 13 = Sampedro, Galcerán, and Izquierdo, 1989, Mol. Cell Biol. 9: 3588-91.

other information: Some *br* and *br/rbp* genotypes display reduced palpi, leading Kiss *et al.* to place *br* and *rbp* in the same complementation group.

l(1)2Bab

phenotype: So named because of its failure to complement lethality of *br* and *rbp* mutations. Homozygotes and hemizygotes die in pupal stage; puparium formation delayed three [*l(1)2Bab*³] to six [*l(1)2Bab*¹] hr. *l(1)2Bab*¹ pupae have normal imaginal organs, and escapers have faded wings and reduced bristles on palpi. Prepupal lethal in combination with *npr1*¹ or *Df(1)S39*.

other information: Surviving *l(1)2ab*¹/*rbp*¹ and *l(1)2ab*¹/*l(1)2Bc*¹ exhibit malformed-leg syndrome.

allele	origin	synonym	ref ^α
<i>l(1)2Bab</i> ¹	EMS	<i>l(1)t4</i>	1, 2, 3, 4
<i>l(1)2Bab</i> ²	EMS	<i>l(1)t126</i>	1, 2, 3
<i>l(1)2Bab</i> ³	EMS	<i>l(1)t143</i>	1, 2, 3, 4
<i>l(1)2Bab</i> ⁴		<i>l(1)d.norm.24</i>	3, 5

^α 1 = Aizenzon and Belyaeva, 1982, DIS 58: 3-7; 2 = Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90; 3 = Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelova, and Zhimulev, 1980, Chromosoma 81: 281-306; 4 = Kiss, Beaton, Tardiff, Fristrom and Fristrom; 5 = Kiss, Bencze, Fekete, Fodor, Gausz, Maróy, Szabad, and Szidonya, 1976, Theoret. Appl. Genet. 48: 217-26.

l(1)2Bc

phenotype: Die in prepupal or early pupal stage after formation of a gas bubble. Imaginal discs fail to fuse, especially dorsally, to produce a continuous integument. Puparium formation variably delayed: 6 hr in *l(1)2Bc*¹, 12 hr in *l(1)2Bc*², and 9 hr in *l(1)2Bc*³ and *l(1)2Bc*⁴. Many late ecdysone puffs both in larvae and prepupae either absent or underdeveloped [*l(1)2Bc*¹] [Zhimulev, Belyaeva, and Aizenzon, 1980, Genetika (Moscow) 16: 1613-31]. *l(1)2Bc*¹ an amorphic allele fully complements the amorphic *br*⁵ (Kiss *et al.*).

alleles:

allele	origin	synonym	ref ^α	comments
<i>l(1)2Bc</i> ¹	EMS	<i>l(1)t10</i>	1, 2, 3, 4, 5	amorph
<i>l(1)2Bc</i> ²	EMS	<i>l(1)t76</i>	1, 2, 3, 4	amorph
<i>l(1)2Bc</i> ³	EMS	<i>l(1)t149</i>	1, 2, 3	
<i>l(1)2Bc</i> ⁴	EMS	<i>l(1)t197</i>	1, 2, 3	
<i>l(1)2Bc</i> ⁵	HD	<i>l(1)2Bc</i> ^{lp14}	6	defective <i>P</i> at 172 kb
<i>l(1)2Bc</i> ⁶	HD	<i>l(1)2Bc</i> ^{lp69}	6	defective <i>P</i> at 172 kb

^α 1 = Aizenzon and Belyaeva, 1982, DIS 58: 3-7; 2 = Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90; 3 = Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelova, and Zhimulev, 1980, Chromosoma 81: 281-306; 4 = Kiss, Beaton, Tardiff, Fristrom and Fristrom; 5 = Kiss, Szabad, Belyaeva, Zhimulev, and Major, 1980, Development and Neurobiology of *Drosophila* (Siddiqi, Babu, Hall and Hall, eds.) Plenum Press, New York and London, pp. 163-81. 6 = Sampedro, Galcerán, and Izquierdo, 1989, Mol. Cell Biol. 9: 3588-91.

l(1)2Bd

phenotype: Males and homozygous females display normal phenotype and viability; however, in heterozygous combination with deficiencies or *npr* mutations, *l(1)2Bd* acts as a temperature-sensitive lethal; completely lethal at 29°; at 25° or 18° most individuals die in late pupal stage, and survivors have faded wings, swollen abdomen, and reduced bristle number on palpi.

alleles:

allele	origin	synonym	ref ^α	comments
<i>l(1)2Bd¹</i>	EMS	<i>l(1)t252</i>	1, 2, 3, 4	hypomorph

^α 1 = Aizenzon and Belyaeva, 1982, DIS 58: 3-7; 2 = Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90; 3 = Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelova, and Zhimulev, 1980, Chromosoma 81: 281-306; 4 = Kiss, Beaton, Tardiff, Fristrom and Fristrom.

npr1: nonpupariating

phenotype: Hemizygous male larvae fail to pupariate, although they survive 10-15 days after their normal sibs have pupariated. Four-day-old larvae appear normal as do their imaginal discs; normal ecdysteroid levels achieved. Discs become abnormal beginning on the sixth day; peripodial membrane becomes enormously distended and highly distorted; partially evaginated structure becomes visible in the disc lumen; do not undergo detailed morphological changes characteristic or metamorphosis, either *in situ* or in transplants into normal larvae [Fristrom, Fekete, and Fristrom, Wilhelm Roux's Arch. Dev. Biol. 190: 11-21 (fig.)]. Both salivary glands and fat bodies fail to undergo histolysis *in situ* or *in vitro*. Mutant flies able to produce ecdysone, but tissues unable to respond normally. In gynandromorphs, the female tissue forms a puparium, whereas *npr1* male tissue remains larval; no adults survive (Kiss, Szabad, and Major, 1978, Mol. Gen. Genet. 164: 77-83; Kiss, Bencze, Fodor, Szabad, and Fristrom, 1975, Nature 262: 136-38). Implantation of wild-type ring glands into *npr1* larvae does not rescue pupariation; however implanted wild-type or *npr1* ring glands are able to rescue *npr3* larvae [Kiss, Szabad, Belyaeva, Zhimulev, and Major, 1980, Development and Neurobiology of Drosophila (Siddiqi, Babu, Hall, and Hall, eds.) Plenum Press, New York and London, pp. 163-81]. No maternal effect of either *npr1*³ or *npr1*⁴ (Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14). *npr1*⁶ homozygous and hemizygous larvae die without exhibiting any sign of ecdysone-inducible puff formation; culture of salivary glands in 20-OH ecdysone produces partial development of some early, but none of late ecdysone-inducible puffs, and extraneous puff appears at 75CD (Belyaeva, Vlassova, Biyasheva, Kakpakov, Richards, and Zhimulev, 1981, Chromosoma 84: 207-19). *npr1*⁺ gene product also required for regression of the intermolt 68C glue puff [Belyaeva, *et al.* (*npr*⁶); Crowley, Mathers and Meyerowitz, 1984, Cell 39: 149-56 (*npr*³)] and for the transcription of the three 68C glue protein genes (Crowley *et al.*).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>npr</i> ¹	EMS	Stewart	<i>l(1)d.norm.-1^a</i>	2, 4, 6, 7, 8, 9, 12	
<i>npr</i> ²	EMS	Stewart	<i>l(1)d.norm.-1^b</i>	8, 10, 12	
<i>npr</i> ³	EMS	Kiss	<i>l(1)npr-1</i>	2, 4, 6, 7, 9	
			<i>l(1)d.norm.-12</i>	5	
<i>npr</i> ⁴	EMS	Kiss	<i>l(1)npr-2</i>	2, 6, 9	
<i>npr</i> ⁵	EMS		<i>l(1)t324</i>	1, 2, 5	
<i>npr</i> ⁶	EMS		<i>l(1)t435</i>	1, 2, 3	
<i>npr</i> ⁷	DEB		<i>npr</i> ^{JTD3}	6	<i>T(1;3)2B5;61F3-4</i>
<i>npr</i> ^{fs}	P		<i>fs(1)del12</i>	11	

^α 1 = Aizenzon and Belyaeva, 1982, DIS 58: 3-7; 2 = Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelova, and Zhimulev, 1980, Chromosoma 81: 281-306; 3 = Belyaeva, Vlassova, Biyasheva, Kakpakov, Richards, and Zhimulev, 1981, Chromosoma 84: 207-19; 4 = Fristrom, Fekete, and Fristrom, 1981, Wilhelm Roux's Arch. Dev. Biol. 190: 11-21; 5 = Kiss, Bencze, Fekete, Fodor, Gausz, Maróy, Szabad, and Szidonya, 1976, Theoret. Appl. Genet. 48: 217-26; 6 = Kiss, Beaton, Tardiff, Fristrom, and Fristrom; 7 = Kiss, Bencze, Fodor, Szabad, and Fristrom, 1976, Nature 262: 136-38; 8 = Kiss, Szabad, Belyaeva, Zhimulev, and Major, 1980, Development and Neurobiology of Drosophila (Siddiqi, Babu, Hall, and Hall, eds.) Plenum Press, New York and London, pp. 163-81; 9 = Kiss, Szabad and Major, 1978, Mol. Gen. Genet. 164: 77-83; 10 = Murphy, 1974, Dev. Biol. 39: 23-36; 11 = Orr, Galanopoulos, Romano, and Kafatos, 1989, Genetics 122: 847-58 (fig.). 12 = Stewart, Murphy, and Fristrom, 1972, Dev. Biol. 27: 71-83.

rbp: reduced bristles on palpus

phenotype: Late pupal lethal; most animals reach the pharate adult stage. *rbp*² homozygotes and males survive with fewer than normal bristles on the palpus; when raised at 29°, females exhibit faded wings and swollen abdomens; when raised at 18° males have faded wings. *rbp*²/*Df(1)RA19* females virtually lethal; females carrying *rbp*² and any of the other *rbp* alleles are completely viable when reared at 18°, but at higher temperatures most die and escapers have reduced bristles on the palpus, shortened bristles on the scutellum, shrivelled or swollen abdomen shrivelled wings and eyes with crumpled surface.

alleles:

allele	origin	synonym	ref ^α	comments
<i>rbp</i> ¹	EMS	<i>rbp</i> ^{t99}	1, 2, 3, 4	
<i>rbp</i> ²	EMS	<i>rbp</i> ^{t132}	1, 2	hypomorph
<i>rbp</i> ³	EMS	<i>rbp</i> ^{t144}	1, 2	
<i>rbp</i> ⁴	EMS	<i>rbp</i> ^{t358}	1, 2	
<i>rbp</i> ⁵	EMS	<i>rbp</i> ^{t376}	1, 2	

^α 1 = Aizenzon and Belyaeva, 1982, DIS 58: 3-7; 2 = Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelova, and Zhimulev, 1980, Chromosoma 81: 281-306; 3 = Kiss, Beaton, Tardiff, Fristrom, and Fristrom; 4 = Kiss, Szabad, Belyaeva, Zhimulev, and Major, 1980, Development and Neurobiology of Drosophila (Siddiqi, Babu, Hall, and Hall, eds.) Plenum Press, New York and London, pp. 163-81;

***brd: broadened**

location: 1-33.

origin: X ray induced.

discoverer: Muller, 26127.

references: 1935, DIS 3: 29.

phenotype: Wings expanded. Viability 20% wild type. RK3.

Brd: Bearded (M. Leviten)

location: 3-42 (between *G1* and *th*).

phenotype: Causes production of supernumerary chaetae and sensilla at or near normal positions. *Brd*¹ homozygotes survive and exhibit more severe phenotypes than heterozygotes. *Brd*¹/*+* = *Brd*¹/*Df(3L)Brd*. *Brd* alleles affect all classes of adult sensory organs. *Brd*¹ strongly affects macrochaetae and other imaginal-disc sensilla (*i.e.*, trichoid, campaniform, basiconic), but only mildly increases microchaete density. *Brd*³ and other alleles produce more severe microchaete phenotypes, in both disc and histoblast derived tissues, as well as exhibiting the *Brd*¹ phenotypes. In addition to sensillum multiplication, *Brd*¹ homozygotes also exhibit bristle loss, with

anterior orbitals absent (>90%) and a less frequent loss of ocellar macrochaetae (<5%). This bristle loss occurs in other *Brd* genotypes and is most severe for *Brd*³ homozygotes. The combination of sensilla multiplication and loss phenotypes is made more severe by loss-of-function mutations at Notch and neuralized, and are decreased in the presence of three wild-type copies of these genes. The ethyl-methanesulfonate-induced point revertants of *Brd*¹ tested (*Brd*^{rv1-4}) are homozygous viable, viable in *trans* to *Brd* deficiencies, and display no mutant phenotypes in either situation.

alleles: Alleles other than *Brd*¹ are primary or secondary derivatives of *Brd*¹. *Brd*^{rv5} and *Brd*^{rv6} are revertants of *Brd*³, and the *Brd*^{Prv} alleles are revertants of *Brd*^{P1}.

allele	origin	discoverer	phenotype ^α	comments
<i>Brd</i> ¹	spont	Groger		
<i>Brd</i> ²	γ ray	Posakony	> <i>Brd</i> ¹ ; severe eye defects; homozygous early pupal lethal	<i>In(3L)71A:71F</i>
<i>Brd</i> ³	P	Leviten	homozygotes → <i>fz</i> -like rough-eye and bristle polarity defects	<i>In(3L)70D-E;71A</i>
<i>Brd</i> ⁴	γ ray	Posakony	< <i>Brd</i> ¹ ; homozygous viable	<i>In(3)71A1-2;80-81</i>
<i>Brd</i> ⁵	γ ray	Posakony	<< <i>Brd</i> ¹ ; homozygous viable	<i>In(3)71A1-2;80-81</i>
<i>Brd</i> ⁶	γ ray	Posakony	< <i>Brd</i> ¹ ; homozygous lethal	<i>In(3)71A1-2;80-81</i>
<i>Brd</i> ^{rv1}	EMS	Shim	wild type; homozygous viable	
<i>Brd</i> ^{rv2}	EMS	Shim	wild type; homozygous viable	
<i>Brd</i> ^{rv3}	EMS	Leviten	wild type; homozygous viable	
<i>Brd</i> ^{rv4}	EMS	Leviten	wild type; homozygous viable	
<i>Brd</i> ^{rv5}	EMS	Leviten	wild type; homozygous lethal	
<i>Brd</i> ^{rv6}	EMS	Leviten	wild type; homozygous lethal	
<i>Brd</i> ^{P1}	P	Leviten	< <i>Brd</i> ¹ ; homozygous viable	
<i>Brd</i> ^{P11}	Δ2-3	Leviten	> <i>Brd</i> ¹ ; homozygous lethal	
<i>Brd</i> ^{P12}	Δ2-3	Leviten	> <i>Brd</i> ¹ ; homozygous viable	
<i>Brd</i> ^{P13}	Δ2-3	Leviten	> <i>Brd</i> ¹ ; homozygous viable	
<i>Brd</i> ^{Prv1}	Δ2-3 ^α	Leviten	wild type; homozygous viable	
<i>Brd</i> ^{Prv2}	Δ2-3	Leviten	wild type; homozygous viable	
<i>Brd</i> ^{Prv3}	Δ2-3	Leviten	wild type; homozygous viable	

^α P-element excisions from *Brd*^{P1} induced by Δ2-3.

other information: Possibly *Hi* was a *Brd* allele.

***bre: bright eye**

location: 1-24.6.

origin: Induced by L-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 68.

phenotype: Eye color brighter red. Wings shorter, often crumpled or waned. Abdomen disproportionately large. Male viability and fertility good; females have reduced fertility. Not easily classified. RK3.

alleles: One allele induced by methyl methanesulfonate.

brevis: see *bv*

brh: brown head

location: 2-61.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82 (fig.).

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Embryonic lethal. Defect in head involution; denticle bands abnormal.

alleles: Two retained, *brh*¹ and *brh*² isolated as *IB* and *IID*; plus six discarded alleles.

bri: bright

location: 2-54.3.

origin: Spontaneous.

discoverer: Nichols-Skoog, 34b23.

references: Beadle and Ephrussi, 1937, Am. Naturalist 71: 91-95.

phenotype: Eye color bright red, like *cn*² or *v*²; difficult to separate from wild type. Malpighian tubules pale yellow (Beadle, 1937, Genetics 22: 587-611). RK3.

bride of sevenless: see *boss*

Bridged: see *Br*

brief: see *bf*

bright: see *bri*

bright eye: see *bre*

Brista: see *Ba*

Bristle: see *BI*

Bristled: see *Sp*

brl: braille (M.P. Scott)

origin: Enhancer trap P-element mutagenesis.

discoverer: L.E. Rost and M.P. Scott, 1989.

phenotype: Recessive mutation with complete penetrance and variable expressivity. Strongest phenotypes include partial to full transformation of one or both eyes into antennae. Less severe adult phenotypes include excess bristles under the eyes, duplication of the more anterior set of scutellar bristles (macrochaetae), and a spoon-like curvature of the wings. Homozygotes are viable and fertile.

cytology: P-element insertion in 60B-C; mobilization of the P-element restored wild-type morphology.

molecular biology: Gene cloned by plasmid rescue from bacterial sequences in the P-element.

other information: Presumed additional alleles obtained by mobilization of P-element. All are recessive lethals that die during early larval stages.

brm: brahma (J.A. Kennison)

location: 3-43.0.

origin: Induced by ethyl methanesulfonate.

discoverer: Kennison, 1983.

references: Kennison and Tamkun, 1988, Proc. Nat. Acad. Sci. USA 85: 8136-40.

phenotype: Dominant suppressor of *Pc* and *Pcl* alleles. Recessive embryonic lethal with strong maternal contribution. Maternal effect lethality is non-rescuable even by two wild-type zygotic alleles. There is a single 5.5 kb mRNA present throughout development with the greatest amounts in the unfertilized egg and early embryo. *brm*¹ isolated as a dominant suppressor of the antennal to leg transformation associated with a *Pc*² *Anp*^{Ns} double heterozygote.

alleles: Fifteen alleles induced by ethyl methanesulfonate, three alleles induced by gamma irradiation, and four alleles induced by hybrid dysgenesis.

cytology: Placed in 72A3-5 based on *in situ* hybridization to salivary gland chromosomes.

brn: see *ca*^G

broad: see *br* in *BRC*

broad abdomen: see *brb*

broad head: see *bhe*

broad head: see *l(2)gl*

broadened: see *brd*

broader wing: see *brw*

bronze: see *sf*²

bronzy: see *mal*^{bz}

brown: see *bw*

brown head: see *brh*

brown-like: see *red*

brown spots: see *bsp*

brr: see *h*^{brr}

brunette: see *Hn*^{r2}

***brw: broader wing**

location: 1-39.8.

origin: X ray induced.

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 83.

phenotype: Wings broad and rounded at the tips. Males show reduced viability and are sterile. RK3.

bs: blistered

location: 2-107.3.

origin: Spontaneous.

discoverer: Bridges, 11k16.

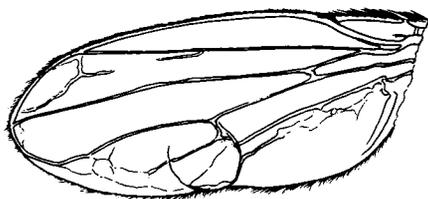
references: Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 155 (fig.).
Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 219 (fig.).

phenotype: Wings blistered, small, pointed; venation thick and plexus-like with branches from and parallel to L5 beyond second crossvein, where there is a semidominant free vein effect. Temperature sensitive. RK2 at 19°; RK3 at 25°.

alleles: *bs*², *bs*³, **bs*⁴, *bs*⁸, **bs*^{52d}, **bs*^{54j}, **bs*^{bl}, **bs*³⁸ⁱ, *bs*^{cy}, **bs*^P, and *bs*^{PP} (CP627).

cytology: Located between 60C5 and 60D2, based on its inclusion within *Df(2R)Px = Df(2R)60B8-10;60D1-2* and within *Df(2R)Px² = Df(2R)60C5-6;60D9-10* (Bridges, 1937, Cytologia (Tokyo), Fujii Jub., Vol. 2: 745-55).

other information: May be part of a pseudoallelic complex with *ba* and *Px*.



bs²: blistered-2

Edith M. Wallace, unpublished.

Bsb: Blunt short bristle

location: 3-100.6 (based on distribution of *Bsb* among 387 crossovers between *ro* and *ca* from *Bsb/ca ro* females).

origin: Induced by ethyl methanesulfonate.

references: B.S. Baker, 1980, DIS 55: 197.

phenotype: Bristles markedly shortened; sharply tapered at tip resembling sharpened pencil under compound microscope. Excellent cell marker. Bristles reduced to very short stubs in *Pr Bsb* heterozygotes. Homozygous lethal; viability and fertility of heterozygote excellent.

cytology: Salivary-gland chromosomes normal.

***bsc: bent scutellars**

location: 1-1.1.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).

discoverer: Fahmy, 1954.

references: 1958, DIS 32: 68.

phenotype: One or more scutellars bent on themselves in form of inverted V. Other bristles irregularly bent. Eyes slightly smaller. Wings slightly abnormal in shape. Male viability about 50% wild type; fertility much reduced. RK3.

alleles: One allele each induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine and D-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine.

Bsg: Blastoderm-specific gene

Three loci identified in a differential screen of a genomic library with labeled blastoderm cDNA plus competing amounts of unlabeled preblastoderm cDNA; localized by *in situ* hybridization to polytene chromosomes.

locus	genetic location	cytological location	synonym	ref ^α
Bsg25D	2-{16}	25D3		2, 3
<i>Bsg75C</i>	3-{45}	75C1-2	term	1, 3
Bsg99D	3-{101}	99D4-8		3

^α 1 = Balderelli, Mahoney, Salas, Gustavson, Boyer, Chang, Roark, and Lengyel, 1988, Dev. Biol. 125: 85-95; 2 = Boyer, Mahoney, and Lengyel, 1987, Nucleic Acids Res. 15: 2309-25; 3 = Roark, Mahoney, Grahm, and Lengyel, 1985, Dev. Biol. 109: 476-88.

Bsg25D

molecular biology: Genomic subclone hybridizes to overlapping 2.7-, 3.0-, and 4.5-kb mRNA's on Northern blots. The two larger transcripts present in 0-8 hr embryos; whereas the 2.7-kb transcript is found only in the blastoderm stage. The 2.7- and 4.5-kb messages encode the same polypeptide; they come from a transcript with three exons, the first two of which are shared, and the third of which differs between the two in the length of its 3' untranslated region. The conceptual amino-acid sequence contains 741 amino acids and contains a 96-amino-acid domain with 22% identity to c-fos and a 21-amino-acid domain that resembles repeated actin-binding segments of tropomyosin.

Bsh: see *Mhc-m*⁵

bsk: basket

location: 2-33.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82 (fig.).

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Homozygous lethal; embryos have large dorsal anterior hole.

alleles: Three, *bsk*¹, *bsk*², and *bsk*³, isolated as *IIIJ*, *IIP*, and **IL*.

cytology: Placed in 31B-32A based on its inclusion in *Df(2L)J27 = Df(2L)31B-E;32A*.

bsp: brown spots

location: 2-40.6.

origin: Spontaneous.

references: Di Pasquale, 1959, DIS 33: 128.

Di Pasquale and Zambruni, 1963, DIS 37: 73 (fig.).

1965, DIS 40: 80.

1966, DIS 41: 119.

1967, DIS 42: 74.

phenotype: Spots of brown pigment appear in integument of *bsp/bsp* females only after they have mated. Di Pasquale and Zambruni (1963) showed that copulation with any male, sterile or fertile, triggers formation of brown spots in cuticle and brown masses in tissues. Courtship without copulation ineffective; virgin females never show brown spots. Simulated copulation with a glass needle, with or without accessory-gland fluid, leads to phenotype. Mating of etherized females less effective in inducing brown spot formation. No phenotype in males. Penetrance of 60-80%; viability excellent. RK3.

bss: bang senseless (J.C. Hall)

location: 1-54.6.

origin: Induced by ethyl methanesulfonate.

synonym: *bas*^{MW1}: *bang sensitive*.

references: Jan and Jan, 1978, Proc. Nat. Acad. Sci. USA 75: 515-19.

Ganetzky and Wu, 1982, Genetics 100: 597-614.

phenotype: Mechanical shock or vortexing induces paralysis lasting for 2-3 minutes; heterozygous female are paralyzed for 40-50 seconds. Homozygotes and hemizygotes have abnormally prolonged release of neurotransmitter at larval neuromuscular junctions, which is associated with multiple firing of action potentials in the nerves; behavioral and electrophysiological phenotypes suppressed by *nap*^{ts} at its permissive (low) temperature.

alleles: *bss*¹ (formerly *bas*^{MW1}) and *bss*² phenotypically alike.

cytology: Placed between 14B5 and 14B13 based on its being deleted by *Df(1)81112h = Df(1)14B5-18;15A6-11* (Steller) but not carried by *Dp(1;2)r⁺75c = Dp(1;3)14B13;15A9;35D-E* (Ganetzky and Wu).

other information: Separable by recombination from *eas*, which causes a similar phenotype and to which *bss* is closely linked.

bt: bent

location: 4-1.4 [mapped in diplo-4 triploids by Sturtevant (1951, Proc. Nat. Acad. Sci. USA 37: 405-7)].

origin: Spontaneous.

references: Muller, 1914, J. Exp. Zool. 17: 325-36.

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 216 (fig.), 219.

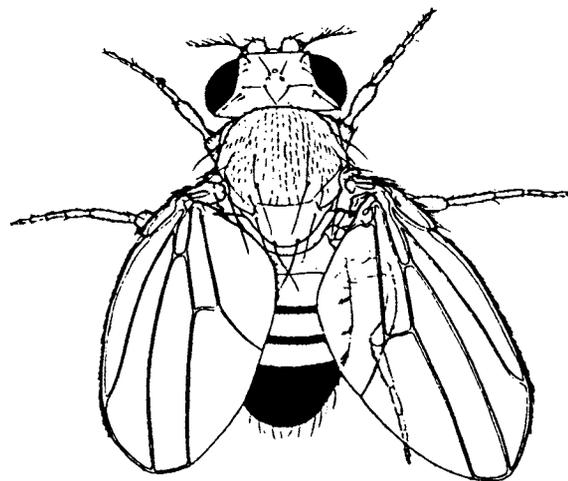
Bridges, 1935, Biol. Zh. 4: 401-20.

phenotype: Wings held out at base and bent sharply backward. Rear legs often lumpy at first tarsal joint. May have one to four "preleg" or "first ventral" bristles on ventral surface of thorax anterior to first pair of legs, in

space otherwise devoid of bristles or hairs. Overlaps wild type at 25°, very much at 19°, and little if any at 29° (Metz, 1923, Proc. Soc. Exp. Biol. Med. 20: 305-10). RK2 at 28°.

cytology: Tentatively placed in 102B10-E9 between *Df(4)M4 = Df(4)101E-F;102B9-10* and *Df(4)G = Df(4)102E2-10;tip* (Hochman, 1971, Genetics 67: 235-52).

other information: First mutant found on chromosome 4.



bt: bent

From Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 216.

bt^D: bent-Dominant

origin: X ray induced.

discoverer: Schultz, 33a11.

references: Bridges, 1935, Biol. Zh. 4: 401-20.

phenotype: When found, *bt^D/+* showed regularly divergent wings with some angular bend near base. Legs lumpy at low temperature. Preleg bristles present as in *bt*. Homozygous lethal. Fails to complement *l(4)2* and *l(4)23* [Hochman, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 903-28]. RK3 as lethal.

other information: Balanced stocks in existence today show only preleg bristle character and recessive lethality (Lewis).

***Bt: Branchlet**

location: 1- (rearrangement).

origin: Induced by ³²P.

discoverer: Bateman, 1950.

references: 1950, DIS 24: 54.

1951, DIS 25: 77.

phenotype: Heterozygous female has posteriorly directed branchlet on posterior crossvein as well as other extra venation. Abdominal segments often poorly chitinized. Male lethal. RK3A.

cytology: Associated with *Dp(1;1)Bt = Dp(1;1)3B2-C1;6F6-7*.

other information: Phenotype may be *Co* effect in 3C7 or more likely *dx* in region 6.

btd: buttonhead**location:** 1-31.**origin:** Induced by ethyl methanesulfonate.**references:** Wieschaus, Nüsslein-Volhard and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307 (fig.)**phenotype:** Homozygous lethal; head involution incomplete.**cytology:** Localized to 8A5-9A1 by segmental aneuploidy.**btdl: buttonheadlike****location:** 1-65.**origin:** Induced by ethyl methanesulfonate.**references:** Eberl and Hilliker, 1988, Genetics 118: 109-20.**phenotype:** Mouthparts sclerotized but not completely internalized; the head is often open. A fraction of *btdl*¹/+ females have bristled growths on the head; those in eye region resemble eye-to-antenna transformations.**alleles:** *btdl*¹ and *btdl*² isolated as *l(1)EH564* and *l(1)EH793*.**cytology:** Placed in 18B4-19A1 based on its being covered by *X^PY^DB50* from *T(1;Y)18B4-11;YL* but not by *Ymal*⁺, which carries 18F3-19A1 to 20F from the X.***bu: bulging****location:** 1-58.**origin:** X ray induced.**discoverer:** Muller, 2618.**references:** 1935, DIS 3: 29.**phenotype:** Eyes rough and bulging. Semilethal. RK3.*bu*: see *Hn*^{r2}*bu-w*^{61j}: see *vs*^{61j}**Bubble:** see **Bb***bubble wing*: see *vs*^{61j}**buckled:** see **bk****buckledlike:** see **bkl****bul: bulge****location:** 3-43.6.**origin:** Spontaneous.**discoverer:** Spencer, 36d28.**references:** 1937, DIS 7: 6.

Curry, 1939, DIS 12: 45.

phenotype: Eyes very large and bulging; facets rounded, in irregular rows, and some quite large. Wing margin heavy; end of wing somewhat squared off to L3. RK3.**cytology:** In 72E4-5 (Velissariou and Ashburner, 1981, Chromosoma 84: 173-85).**bul^{bp}: bulge-bumpy****origin:** Spontaneous.**synonym:** *bp*.**references:** E. H. Grell, 1955, DIS 29: 72.**phenotype:** About one-half the eye surface erupted into irregular yellowish blisters. Facets larger than normal in nonblistered areas. Homozygotes occur with 1% of expected frequency. Surviving homozygotes vigorous and male fertility high; females lay eggs abundantly, but only rarely does an egg hatch. RK3.**bul^D: bulge-Dominant (E.H. Grell)****origin:** Induced by ethyl methanesulfonate.**phenotype:** Eyes rough with large facets in both heterozygote and homozygote. Good viability.**bulging:** see **bu*****buo: burnt orange****location:** 2-57.1.**origin:** Spontaneous.**discoverer:** T. Hinton and Kleiner, 1941.**references:** Hinton, 1942, DIS 16: 48.**phenotype:** Eye color bright orange-brown. Malpighian tubules colorless in larva (Brehme and Demerec, 1942, Growth 6: 351-56). RK2.**other information:** Not an allele of *cn*. Allelism with *ltd* (2-56) apparently never tested.**bur: burgundy****location:** 2-55.7.**origin:** Ultraviolet induced.**references:** Meyer and Edmondson, 1949, DIS 23: 60.**phenotype:** Eye color dull, darkish brown (like *pr*), brilliant orange in combination with *cn*. *bur*^{gua} supplementable by guanosine but not other ribonucleosides or nucleic acid bases; inosine-5'-monophosphate dehydrogenase activity reduced compared to normal (Johnston, Nash, and Naguib, 1985, Biochem. Genet. 23: 539-55). Classification and viability excellent. Fertility of females good; of males, variable. RK1.**alleles:** *bur*² (CP627) and *bur*^{gua}.**cytology:** In 42B1-3 (Johnstone).**other information:** *bur* not allelic to *lt*, *ltd*, or *pr*. Allelism of *bur*^{gua} inferred from map position of 2-54 and phenotype.**bur^{gua}****origin:** Induced by ethyl methanesulfonate.**synonym:** *gua2-1*.**phenotype:** Recessive auxotroph for guanosine; supplementable by 3.2 mM of guanosine but not by other ribonucleosides or nucleic acid bases. Homozygotes have dark red eyes; this character segregates with the nutritional requirement. Inosine dehydrogenase activity of *bur*^{gua} larval extracts an order of magnitude lower than that of wild type (Johnston, Nash, and Naguib, 1985, Biochem. Genet. 23: 539-55).**burnt orange:** see **buo****buttonhead:** see **btd****buttonheadlike:** see **btdl****bv: brevis****location:** 3-102.7 (recalculated from Sturtevant, 1956, Genetics 41: 118-23).**discoverer:** Bridges, 33e25.**phenotype:** Bristles uniformly short and stubby. Body chunky. Hatches late but viability excellent. RK1.**cytology:** Tentatively placed in distal to 100B7-8 by Frisardi and MacIntyre (1984, Mol. Gen. Genet. 197: 403-13) based on the retention of *bv*⁺ by *Df(3R)ca48 = Df(3R)98F14;100B7-8*.

bw: brown

location: 2-104.5.

discoverer: Waaler, 19j15.

references: 1921, Hereditas 2: 391-94.

Dreesen, Johnson, and Henikoff, 1988, Mol. Cell Biol. 8: 5206-15.

Sullivan and Sullivan, 1975, Biochem. Genet. 13: 603-13.

Mount, 1987, Nature (London) 325: 487.

phenotype: Eye color light brownish wine on emergence, darkening to garnet. Red pigments lacking. Xanthommatin mostly replaced by dihydroxanthommatin (Phillips, Forrest, and Kulkarni, 1973, Genetics 73: 45-56). Pigment granules present but somewhat smaller than in wild type. Adult testes and vasa colorless. Larval Malpighian tubules pale yellow (Beadle, 1937, Genetics 22: 587-611). Produces white eyes in combination with *v*, *cn*, or *st*. Eye color autonomous when transplanted into wild-type host (Beadle and Ephrussi, 1936, Genetics 21: 230). RK1.

alleles: Mutant alleles of *bw* are listed in the accompanying table and their phenotypes compared to those of alleles described below. More complete descriptions in CP627 unless otherwise indicated.

allele	phenotype ^α
<i>bw</i> ¹	<i>bw</i>
* <i>bw</i> ²	<i>bw</i> ⁵
<i>bw</i> ^{2b}	<i>bw</i> ⁵
* <i>bw</i> ^{2c}	<i>bw</i> ⁴
<i>bw</i> ⁴	<i>bw</i> ⁴
<i>bw</i> ⁵	<i>bw</i> ⁵
<i>bw</i> ⁶	<i>bw</i> ⁶
* <i>bw</i> ⁺²¹	<i>bw</i> ⁴
* <i>bw</i> ²⁴	<i>bw</i> ⁴
* <i>bw</i> ³⁰	<i>bw</i>
* <i>bw</i> ^{33f}	<i>bw</i>
* <i>bw</i> ^{33g}	<i>bw</i>
* <i>bw</i> ^{37g}	<i>bw</i> ⁵
<i>bw</i> ^{38jβ}	<i>bw</i> ⁵
<i>bw</i> ^{45a}	<i>bw</i> ⁴
* <i>bw</i> ^{47j}	<i>bw</i> ⁵
<i>bw</i> ⁵⁹	<i>bw</i> ⁴
<i>bw</i> ⁷²	<i>bw</i>
<i>bw</i> ⁷⁵	<i>bw</i> ⁵
<i>bw</i> ⁸¹	<i>bw</i> ⁵
* <i>bw</i> ^{53l}	<i>bw</i> ⁵
* <i>bw</i> ⁶⁰⁹	<i>bw</i>
<i>bw</i> ^{979γ}	<i>bw</i>
<i>bw</i> ^a	<i>bw</i> ^a
* <i>bw</i> ^{AD}	<i>bw</i>
* <i>bw</i> ^{au}	<i>bw</i>
* <i>bw</i> ^{CB}	<i>bw</i>
* <i>bw</i> ^{le}	<i>bw</i>
* <i>bw</i> ^{M58}	<i>bw</i> ⁵
* <i>bw</i> ^{M590}	<i>bw</i>
* <i>bw</i> ^{ptm}	<i>bw</i> ^{ptm}
<i>bw</i> ^{Raδ}	<i>bw</i>

^α The phenotype column lists the allele described below that the allele listed resembles; *bw*⁵ is homozygous lethal, but other *bw*⁵-like alleles are not.

^β Ives, 1968, DIS 43: 64.

^γ Valadé del Rio, 1983, Genet. Iberica, 35: 47.

^δ Trippa, Loverre, and Cichetti, 1980, Genetics 95: 399-412.

cytology: Placed between 59E1-2 or the 59E2-3 interband by examination of *In(2LR)bw*^{85f4} = *In(2LR)30A;59E2-3* (Nash and Tiong) and by *in situ* hybridization of a brown (Dreesen, *et al.*, 1988).

molecular biology: The genomic locus as well as cDNAs have been cloned and sequenced. The genomic region contains six small introns. The *bw* mutation is an insertion of about 8 kb of unknown origin into the coding portion of the locus. In the heads of newly eclosed wild-type flies there are two major transcripts of 2.8 and 3.0 kb that differ as a consequence of alternative poly(A) addition and encode the same predicted protein of 675 amino acids. The 8 kb *bw* insertion results in termination of transcription to give two truncated poly(A⁺) transcripts (Dreesen, *et al.*, 1988).

other information: Uptake studies indicate that *bw*, like *w*, blocks the transport of guanine and xanthine, likely pteridine precursors (Sullivan and Sullivan, 1975). Sequence comparisons between the predicted *bw*- and *w*-encoded proteins show that they are similar to one another and to subunits of active transport family members (Mount, 1987; Dreesen, *et al.*, 1988).

bw⁴

origin: Spontaneous.

discoverer: Mohr, 31k28.

phenotype: Homozygotes have normal eye color; in heterozygotes with other alleles, red pigment is reduced. *bw*⁴/*bw*⁵ is purpleoidlike. *bw*⁴/*bw* like *bw* but darker. RK3.

bw⁵

origin: Spontaneous.

discoverer: Mohr, 31k28.

phenotype: *bw*⁵/*bw*⁴ is purpleoidlike (see *bw*⁴); *bw*⁵/*bw*⁵ is light yellowish brown; *bw*⁵/+ is wild type; *bw*⁵/*bw*⁵ is lethal. RK2A.

cytology: Shown to be a deficiency based on failure to hybridize *in situ* with clones derived from this region (Dreesen, Johnson, and Henikoff).

bw⁶

origin: Spontaneous.

discoverer: Farmer, 1974.

references: 1977, Heredity 39: 297-303.

Farmer and Fairbanks, 1986, DIS 63: 50-51.

synonym: *Su(w^{co2})⁺*; *Su(w^{coJ})⁺*.

phenotype: A subliminal recessive allele of *bw*; homozygotes are wild type in all combinations except in flies that are homozygous or hemizygous for *w^{co2}*, which are like *bw* or *w^{co}*. *bw*⁶/*bw*⁴ suppresses *w^{co2}*, but all other heterozygotes between *bw*⁶ and *bw* alleles which were tested did not suppress. RK1 in combination with *w^{co2}*.

bw^{6lj}: see *vs*^{6lj}

bw^a: brown-amber

origin: Spontaneous.

discoverer: R. C. King, 48f15.

references: Poulson and King, 1948, DIS 22: 54.

phenotype: Eye color light brownish yellow. Adult testes and vasa colorless. Larval Malpighian tubules slightly paler yellow than wild type. *bw*^a/*bw* gives eye color slightly lighter than *bw*. RK1.

***bw^A: brown-Auburn**

origin: X ray induced.

discoverer: Dubinin.

synonym: *A*; *Pm*^{DI}.

references: Dubinin and Heptner, 1935, J. Genet.

30: 423-46 (fig.).

Dubinín, 1936, Biol. Zh. (Moscow) 5: 851-74.

phenotype: Nearly uniform brown but with extra *Y* chromosome shows strong variegation. Homozygote usually lethal. RK1A.

cytology: Associated with $In(2R)bw^A = In(2R)A1;59D$.

***bw^D*: brown-Dominant**

origin: Spontaneous.

discoverer: T. Hinton, 1940.

references: 1940, DIS 13: 49.

1942, DIS 16: 48.

Slatís, 1955, Genetics 40: 246-51.

phenotype: Eye color varies with age from purple to brown. Shows slight variegation in combination with *st* (Slatís, 1955). Wings pebbled. $bw^D/+$ shows nearly a 100-fold reduction in pteridine levels; no accumulation of *bw* RNA detectable. $bw^D/bw^{VI} > bw^D/+ > bw^D/bw^D$ in severity of effect (Henikoff, and Dreesen, 1989, Proc. Nat. Acad. Sci. USA 86: 6704-08). Variegation suppressed by extra *Y* chromosomes (Brosseau, 1959, DIS 33: 123). Homozygote viable and fertile. Larval Malpighian tubules bright yellow (Brehme and Demerec, 1942, Growth 6: 351-56). RK1A.

alleles: bw^{D114} , X ray induced (Wright, Hodgetts, and Sherald, 1976, Genetics 84: 267-285).

cytology: Schultz reports an extra band in 59E that tends to pair with a band in the homolog, suggesting a duplication of one band from 59E. Slatís (1955) reports insertion of three or four bands, probably of heterochromatic origin. Reverts to wild type when extra bands separated from *bw* locus (Hinton and GoodSmith, 1950, J. Exp. Zool. 114: 103-14). Examination of stained metaphase preparations reveals a block of heterochromatin in the approximate position of bw^D (Dimitri and Pimpinelli).

other information: Since 1950 some and perhaps all lines of bw^D have undergone a secondary event that removed a portion of the coding region of the *brown* gene, generating a null allele (Dreesen and Henikoff).

****bw^{ptm}*: brown:pteridine modifier**

origin: Naturally occurring allele.

synonym: *ptm*.

references: Clancy, 1967, DIS 42: 57.

phenotype: Undetectable in wild-type background. In w^{sar} or w^{cf} flies that are at the same time *v*, *cn*, or *st*, the amount of red eye pigment in $+/+ > +/bw^{ptm} > bw^{ptm}/bw^{ptm}$.

***bw^R*: brown-Rearranged**

origin: X-ray-induced derivatives of *bw* or bw^+ .

discoverer: Slatís, 48k16.

references: 1955, Genetics 40: 5-23.

phenotype: These rearranged derivatives exhibit variegated phenotype in combination with *+*. Mostly homozygous lethal; survivors have brown eyes.

alleles: These derivatives and those associated rearrangements are listed in the accompanying table. More complete descriptions in CP627.

cytology: All associated with chromosome rearrangements with one breakpoint in 59DE and one in proximal heterochromatin.

allele	rearrangement ^α
bw^A	$In(2R)A1;59D$

allele	rearrangement ^α
bw^D	heterochromatic insertion in 59E
$*bw^{R3}$	$In(2LR)40F;51F;55E;57E;58D8-9$
$*bw^{R4}$	$T(2;3)59E2-3;80-81$
$*bw^{R12}$	$T(2;3)59D;80C$
$*bw^{R14}$	$T(2;3)59E2-3;80$
$*bw^{R15}$	$T(2;3)59D;80C$
$*bw^{R18}$	$In(2)40-41;59E4-F1$
$*bw^{R20}$	$In(2LR)40D;59D5-6$
$*bw^{R25}$	$T(2;4)59D;101E$
$*bw^{R27}$	$T(Y;2)59D11-E1$
$*bw^{R32}$	$In(2R)41A;59D$
$*bw^{R33}$	$In(2R)41;59DE$
$*bw^{R35}$	$In(2)40F-41A;59D11-E1$
$*bw^{R40}$	$Df(2R)59C5-6;$
$*bw^{R45}$	$In(2)40F-41A;59E3-4$
$*bw^{R47}$	$In(2)40-41;59D-E1$
$*bw^{R50}$	breakpoint at 59D2-3
$*bw^{R55}$	$In(2LR)24E1-D;42E + In(2R)40F-41A;59D4-5$
$*bw^{R56}$	$In(2)40F-41A;59DE$
$*bw^{R57}$	$T(Y;2)59D5-6$
$*bw^{R58}$	$T(2;3;4)59D;65;101C$
$*bw^{R67}$	$In(2)40F-41A;59E4-F1$
$*bw^{R68}$	breakpoint near 58F
$*bw^{R73}$	$In(2)40F-41A;59E4-F1$
$*bw^{R79}$	$In(2)40F-41A;59F2-3$
bw^{V1}	$In(2LR)21C8-D1;60D1-2 + In(2LR)40F;59D4-E1$
$*bw^{V2}$	$In(2R)$
$*bw^{V3}$	$T(2;3)$
bw^{V4}	$T(2;3)$
bw^{V5}	$T(2;3)$
$*bw^{V6}$	$T(2;3)$
$*bw^{V7}$	$In(2R)$
$*bw^{V8}$	$T(2;3)$
$*bw^{V29I}$	$In(2LR)$
$*bw^{V30a}$	
$*bw^{V30k1}$	$In(2LR)$
$*bw^{V30k10}$	$In(2R)$
$*bw^{V30k12}$	$T(2;3)$
$*bw^{V30k13}$	$T(2;3)$
bw^{V30k18}	$T(2;3;4)$
bw^{V32g}	$In(2LR)40F;59E$
bw^{V34k}	$In(2R)41;59E + In(2R)Cy$
bw^{V40b}	$In(2R)41A-B;59D-E$
$*bw^{V54a}$	$In(2R)41A-B;59D4-9$
$*bw^{V54b}$	$In(2R)41A;60D9-11$
$*bw^{V54c}$	$In(2R)41;59E1$
bw^{V57e}	<i>SM1</i> derivative
bw^{VA}	$T(2;3)$
bw^{VD}	$T(2;3)$
bw^{VDe1}	$In(2R)41B2-C1;59E2-4$
bw^{VDe2}	$In(2R)41A-B;59D6-E1$
bw^{VDe3}	$T(2;3)59D;81F$
bw^{VDe4}	$T(2;3)59D2-4;80$
$*bw^{VI}$	$In(2R)41A;59D$

^α Polytene analysis unavailable where breakpoints not indicated.

***bw^{V1}*: brown-Variegated**

origin: X ray induced.

discoverer: Muller, 1929.

synonym: *Pm*: *Plum*.

references: 1930, J. Genet. 22: 299-334 (fig.).

Glass, 1934, J. Genet. 28: 69-112 (fig.).

1934, Am. Naturalist 68: 107-14.

Bridges, 1937, Cytologia (Tokyo), Fujii Jub., Vol. 2: 745-55.

phenotype: Eye color like *bw* or *pr*, mottled with darker spots that deepen in red color with age. With *st* or *v*, has pale orange ground with dark orange spots. bw^{V1}/bw shows sharply reduced levels of transcript from both the bw^+ allele in *cis* with the rearrangement and the *bw* allele on the homologous second chromosome (Henikoff, and Dreesen, 1989, Proc. Nat. Acad. Sci. USA

86: 6704-08). Extra *Y* chromosome, as with other variegated browns, suppresses brown color, giving red eye sparsely speckled or spotted with darker spots. Amount of wild-type eye color also responds to fourth-chromosome constitution; $4/C(4) > C(4) = 4/4 > 4/0$, with the first approaching wild type and the latter nearly *bw* (Lindsley and Rokop). Larval Malpighian tubules normal (Glass, Brehme). Generally lethal homozygous and in combination with other brown-Varietateds. Heterozygotes fully viable and fertile. RK1A.

alleles: Other variegated alleles of *bw*, all of which are associated with X-ray-induced chromosome aberrations, are listed in the accompanying table. More complete descriptions in CP627.

cytology: Associated with $In(2LR)bw^{VI} = In(2LR)21C8-D1;60D1-2 + In(2LR)40F;59D4-E1$ (Schultz and Bridges).

bw-b: see *ca*

bwn^G: see *ca^G*

bx*:** see ***BXC

bx^D*:** see ***Ubx in ***BXC***

***Bx*: Beadex**

location: 1-59.4.

phenotype: Male and homozygous female with Beaded-like wings; long, narrow, and excised along both margins; fully viable. Heterozygous female less extreme and overlaps wild type. Some venation abnormality. Development studied by Goldschmidt [1935, Biol. Zentr. 55: 535-54 (fig.)]. According to Waddington (1940), embryological effect is same as that of *vg*. RK2 (RK3 as *Bx/+*).

alleles:

allele	origin	discoverer	ref ^α	mol. biol. ^β
<i>Bx¹</i>	spont	Bridges, 23a3	2, 3, 9, 13, 17	8.5 kb (roo)
<i>Bx^{2γ}</i>	spont	Mohr, 24129	2, 3, 10, 14, 16	gypsy
<i>Bx^{3γ}</i>	spont	Gershenson, 1927	2, 3, 6	10.3 kb (roo)
<i>Bx⁹</i>	P	Engels	13	-1.2 kb
<i>Bx¹⁵</i>	P	Engels	13	-2.0 kb
<i>Bx^{32e}</i>	X ray	Moore, 32e	2	
<i>Bx⁴⁶</i>			13	4.2kb (copia)
<i>*Bx^{59h}</i>	spont	T.J. Lee, 59h	3, 12	
<i>*Bx^C</i>	spont	Catcheside 39c3	1, 2	
<i>*Bx^{In}</i>	spont	Mohr, 24d4	2, 15	
<i>Bx^{Jγ}</i>	heat?	Jollos, 1930	2, 3, 4, 5, 13	6.2 kb (3S18)
<i>*Bx^L</i>	spont	Lancefield	2, 13	
<i>Bx^M</i>	spont	Muller (in Winscy)		12-16 kb
<i>Bx⁰</i>	spont	Oster, 1964 (in <i>C(1)Dx</i>)		
<i>Bx^{rγ}</i>	spont	Ives, 35k	2, 3, 8, 9, 11	
<i>Bx^{r49kγ}</i>	spont	Mossige, 49k22	2, 3, 10, 17	

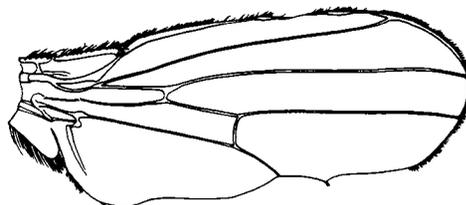
^α 1 = Catcheside, 1939, DIS 12: 49; 2 = CP552; 3 = CP627; 4 = Jollos, 1933, Naturwissenschaften 21: 831-34; 5 = Jollos and Waltzky, 1937 DIS 8: 9; 6 = Gassinovitsch and Gershenson, 1928, Biol. Zentrabl. 48: 385-87 (fig.); 7 = Gottschewski, 1935, DIS 4: 7, 14, 16; 8 = Green, 1952, Proc. Nat. Acad. Sci. USA 38: 949-53; 9 = Green, 1953, Genetics 38: 91-105 (fig.); 10 = Green, 1953, Z. Indukt. Abstamm. Vererbungsl. 85: 435-39 (fig.); 11 = Ives, 1937, DIS 7: 6; 12 = Lee, 1964, DIS 39: 60; 13 = Mattox and Davidson, 1984, Mol. Cell. Biol. 4: 1343-53 (fig.); 14 = Modolell, Bender, and Meselson, 1983, Proc. Nat. Acad. Sci. USA 80: 1678-82; 15 = Mohr, 1927, Hereditas 9: 178; 16 = Mohr, 1927, Nyt. Mag. Natur. 65: 265-74; 17 = Morgan, Bridges, and Sturtevant, 1925, Bibliogr. Genet. 2: 219; 18 = Mossige, 1950, DIS 24: 61.

^β Size of DNA insert in *Bx* region.
^γ Phenotypes described below.

cytology: Placed in 17C2-3 based on the position of break-points of hybrid-dysgenesis-induced *hdp* mutants (Engels and Preston, 1981, Cell 26: 421-28).

molecular biology: 49kb, including the *Bx* locus, cloned and restriction mapped by Mattox; 0 coordinate defined as *Sma*I site within a 1.8 kb *Bam* HI fragment within or very close to the *Bx* coding sequence; positive values to the right. Six mutants differ from wild type in having different segments of DNA inserted into the same 0.4kb restriction fragment (-2.0 to -1.6). *Bx²* has two gypsy elements inserted in opposite orientation at coordinates -0.5 and -1.5. Hybrid-dysgenesis-induced mutations, *Bx⁹* and *Bx¹⁵*, are imprecise excisions of the P factor, which is inserted at coordinate -0.8 in the Π2 X chromosome and is without phenotypic effect. *Bx⁹* deletion includes 1.2kb of flanking DNA including -0.8 to -2.0, and *Bx¹⁵* deletion of 2.0kb includes -1.3 to +0.4 (Mattox and Davidson, 1984, Mol. Cell. Biol. 4: 1343-53).

other information: Interpreted by Lifschytz and Green (1979, Mol. Gen. Genet. 171: 153-59) as mutation in *cis*-acting control element causing overproduction of *hdp-a* gene product. Tandem duplication of *Bx⁺* and *hdp-a⁺* has recessive *Bx* effect (e.g., *Bx^r*); tandem tripllication and quadruplication have dominant *Bx* effects in combination with a normal X, but not in combination with *hdp-a* or a *Bx* deficiency (Lifschytz and Green, 1979). Amorphic or extreme hypomorphic *Bx* mutations and deficiencies for *Bx* act as trans suppressors of *Bx*. Suppression used by Lifschytz and Green to select null alleles or deficiencies from treated + or *Bx³* chromosomes; seven derivatives of *Bx³* and those of *Bx⁺* described; all have *hdp-a* effects as well.



***Bx²*: Beadex-2**

Edith M. Wallace, unpublished.

Bx²

phenotype: Wings of males and homozygous females narrowed by marginal excision. Wings often bubbly and ragged. Homozygous female fully viable. *Bx^{2/+}* less extreme; overlaps wild type. Classifiable in a single dose in triploids (Schultz, 1934, DIS 1: 55). RK1 (RK3 as *Bx^{2/+}*).

Bx³

phenotype: Extreme allele usually without the bubbles in the wing. Shortened L5 a constant character (few *Bx²* show this). Wings more pointed than *Bx²* and hairs at tip of wing clumped. Third-instar larvae show 20-40 degenerating cells in area of wing disk corresponding to future wing margin (D. Fristrom, 1969, Mol. Gen. Genet. 103: 363-79). Scalloping visible in prepupal

wing bud [Waddington, 1940, *J. Genet.* 41: 75-139 (fig.)]. Bx^3 in heterozygous combination with a Bx deficiency or $hdp-a$ has normal wing phenotype (Lifschytz and Green, 1979, *Mol. Gen. Genet.* 171: 153-59). $Bx^3/+$ fully separable. RK1.

Bx^J : *Beadex of Jollos*

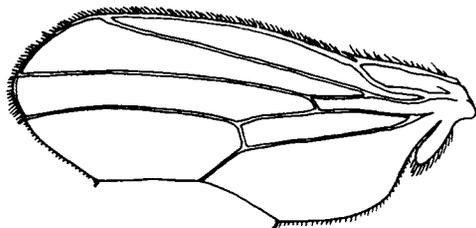
phenotype: Wings reduced to slender strip; only posterior cell present at tip. $Bx^J/+$ have half and Bx^J/Bx^J one third the normal number of cells in membrane of wing. Femur shortened or legs otherwise abnormal, especially third pair. Halteres abnormal. Homozygous female viable. Interacts with bi to give more nearly normal wings. The only dominant Bx allele not suppressed by a Bx deficiency [e.g., $In(1)Cl^{Ly}4R$] or hdp (Lifschytz and Green, 1979, *Mol. Gen. Genet.* 171: 153-59). Embryology like Bx [Goldschmidt, 1935, *Biol. Zentr.* 55: 535-54; Waddington, 1940, *J. Genet.* 41: 75-139 (fig.)]. Clonal analysis of wing disk development indicates massive cell loss during third larval instar. Clones of Bx^+ cells in $Bx^J/+$ wings that reach the margin but are confined to the dorsal or ventral surface often cause reconstitution of both surfaces and appearance of marginal elements derived from both surfaces (Santamaria and García-Bellido). RK1. 1975, *Wilhelm Roux' Arch. Entwickl.-lungsmech. Organ.* 178: 233-45.

Bx^r : *Beadex-recessive*

phenotype: $Bx^r/+$ is normal. Male and homozygous female show less extreme narrowing of wings than Bx . Anterior crossvein short and thickened and that region blistered. May overlap wild type in old crowded cultures at 25°, more extreme at 19°. RK3A.

cytology: Associated with $Dp(1;1)Bx^r = Dp(1;1)17A;17E-F$ (Green, 1953, determined by E.B. Lewis). Recessive Bx effects are due not to an altered gene but to increased dosage of the normal allele.

other information: $Bx/Dp(1;1)Bx^r$ produces recombinants of genotype Bx^+Bx and $BxBx^+$, which are more extreme than Bx . Same holds for $Bx^2/Dp(1;1)Bx^r$.



Bx^{r49k} : *Beadex-recessive 49k*

From Green, 1953, *Z. Indukt. Abstamm. Vererbungsl.* 85: 435-49.

Bx^{r49k}

phenotype: Slight scalloping of posterior wing margin only; overlaps wild type. RK3A.

cytology: Associated with $Dp(1;1)Bx^{r49k} = Dp(1;1)17A;17C$ (E.B. Lewis).

other information: This duplication undergoes unequal crossing over readily and forms triplications and quadruplications. Duplication is recessive; triplication is dominant. Phenotypic interaction with Bx same as for Bx^r .

BXC: Bithorax Complex (I. Duncan)

The bithorax complex (BXC) is a gene cluster that functions to assign unique identities to body segments in the abdomen and posterior thorax (Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, *Science* 221: 23-29). Most, perhaps all, BXC functions are expressed within parasegments, metameric units composed of the posterior compartment of one segment and the anterior compartment of another. Complementation studies indicate that the BXC is organized into three large functionally integrated regions, known as the *Ultra-bithorax* (*Ubx*), *abdominal-A* (*abd-A*), and *Abdominal-B* (*Abd-B*) domains. Apparent point mutations have been recovered that totally inactivate each of these domains. The *Ubx* domain functions primarily to assign identities to parasegments 5 and 6 (PS5 and PS6), the *abd-A* domain functions in PS7-PS13, and the *Abd-B* domain functions in PS10-PS14. Each BXC domain contains subregions that are responsible for the assignment of identities to specific compartments or parasegments. The *Ubx* domain contains two major subregions. These are the *anterobithorax-bithorax* (*abx-bx*) region, which specifies PS5 identities, and the *bithorax-postbithorax* (*bxd-pbx*) region, which specifies PS6 identities. Molecular studies indicate that most or all *Ubx* domain functions are executed by a family of homeodomain-containing *Ubx* proteins. The *abx-bx* and *bxd-pbx* subregions appear to *cis*-regulate the expression of these proteins in PS5 and PS6, respectively. *abd-A* and *Abd-B* functions are also executed by homeodomain-containing proteins. The *abd-A* and *Abd-B* domains each contains parasegment- or segment-specific subregions which have been named *infra-abdominal* (*iab*) regions. Each *iab* region is named according to the anterior compartment of the segment or parasegment whose identity is specifically abolished by mutations in that region. For example, *iab2* mutations cause a transformation of the anterior second abdominal segment (A2) toward A1, and *iab3* mutations cause transformations of anterior A3 (and more posterior segments) toward A2. The *iab2*, *iab3*, and *iab4* subregions are contained within the *abd-A* domain whereas the *iab5* through *iab9* regions are located within the *Abd-B* domain. Some evidence suggests that the *bxd-pbx* region and some of the *iab* regions may be bifunctional and affect the expression of both adjacent BXC domains. Remarkably, the order of subregions within the complex is the same as the order of the parasegments or segments that each affects. Although it is not known whether this correlation has functional significance, two mutations (*Uab¹* and *Cbx¹*) that change the relative order of BXC regions alter the spatial expression of BXC products.

abd-A: abdominal-A (I. Duncan)

location: 3-58.5 (to the right of *iab2*; to the left of *iab3*).

references: Lewis, 1978, *Nature* 276: 565-70.

Morata, Botas, Kerridge, and Struhl, 1983, *J. Embryol. Exp. Morphol.* 78: 319-41.

Sánchez-Herrero, Vernós, Marco, and Morata, 1985, *Nature* 313: 108-13.

Sánchez-Herrero, Casanova, Kerridge, and Morata, 1985, *Cold Spring Harbor Symp. Quant. Biol.* 50: 165-172.

Tiong, Bone, and Whittle, 1985, *Mol. Gen. Genet.* 200: 335-42.

Karch, Wiefenbach, Peifer, Bender, Duncan, Celniker,

Crosby, and Lewis, 1985, Cell 43: 81-96.
 Duncan, 1987, Annual Review of Genetics 21: 285-319.
 Cumberledge, Zaratzian, and Sakonju, 1990, Proc. Nat. Acad. Sci. USA 87: 3259-63.

phenotype: Null alleles are recessive lethal. Homozygous larvae show transformations of the ventral and dorsal setal belts of A2 through A8 toward A1. These transformations are complete in A2 through A4, but are incomplete more posteriorly. Partial Keilin's organs composed of monohairs occur variably on all segments from A1 through A7. In the adult cuticle, homozygous *abd-A* mitotic recombination clones are completely transformed to A1 in segments A2 through A4 and show characteristics of A1 to A4 in segments A5 to A7.

alleles:

allele	origin	discoverer	synonym	cytology	molecular biology ^α
<i>abd-A</i> ³⁹	ENU	R.H. Baker	<i>Hab</i> ^{rev28390.39}		
<i>abd-A</i> ⁴¹	ENU	R.H. Baker	<i>Hab</i> ^{rev28390.41}		
<i>abd-A</i> ⁵¹⁷	X ray	R.H. Baker	<i>iab2</i> ^{31616.517}	<i>T(2;3)34-35;41</i> ; 40-45 kb <i>78;83-84;89E</i>	
<i>abd-A</i> ^{C26}	X ray	Crosby	<i>iab2</i> ^{C26}	normal	34.5-36 kb deleted
<i>abd-A</i> ^{C51}	X ray	Crosby	<i>Hab</i> ^{revC51} <i>iab2</i> ^{C51}	<i>In(3LR)80;89E</i>	41.5-43 kb
<i>abd-A</i> ^{C53}	X ray	Crosby	<i>Hab</i> ^{revC53} <i>iab2</i> ^{C53}	normal	undetected
<i>abd-A</i> ^{D24}	EMS	Duncan	<i>iab2</i> ^{D24}	normal	undetected
<i>abd-A</i> ^{M1}	EMS	Morata		not reported	not studied
<i>abd-A</i> ^{M3}					
<i>abd-A</i> ^{MX1}	X ray	Morata		<i>T(2;3)40;89E</i>	54-56 kb
<i>abd-A</i> ^{MX2}	X ray	Morata		normal	55-58.5 kb deleted
<i>abd-A</i> ^{P10}	X ray	Shaw	<i>iab2</i> ^{P10} <i>T(2;3)P10</i>	<i>Tp(3;2)29A-C</i> ; <i>89C1-2;89E1-2</i>	35-36 kb
<i>abd-A</i> ^{S2}	X ray	Tiong			50-55 kb

^α Coordinates of rearrangement breakpoints unless otherwise indicated: Karch, Wiefenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.

Abd-B: Abdominal-B (I. Duncan and S. Celniker)

location: 3-58.8 (to the right of *iab7*; to the left of *iab8*,9).

references: Karch, Wiefenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.
 Sánchez-Herrero, Casanova, Kerridge, and Morata, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 165-172.
 Sánchez-Herrero, Vernós, Marco, and Morata, 1985, Nature 313: 108-13.
 Tiong, Bone, and Whittle, 1985, Mol. Gen. Genet. 200: 335-42.
 Casanova, Sánchez-Herrero and Morata, 1986, Cell 47: 627-36.
 Duncan, 1987, Annual Review of Genetics 21: 285-319.
 Sánchez-Herrero, and Crosby, 1988, EMBO J. 7: 2163-73.
 Celniker, Keelan, and Lewis, 1989, Genes Dev. 3: 1425-37.
 Zavortnik and Sakonju, 1989, Genes Dev. 3: 1969-81.
 DeLorenzi and Bienz, 1990, Development 108: 323-29.

phenotype: Heterozygotes for null alleles show weak anteriorly-directed transformations of A5, A6, and A7. In the male, this results in the presence of a tiny extra tergite in A7 and a loss of pigmentation on the A5 tergite. Heterozygotes are partially to completely sterile in both sexes, but are fertile if a duplication for the BXC [such as *Dp(3;5)P5* or *Dp(3;1)P68*] is present. Hemizygotes and homozygotes are lethal; embryos lack posterior spiracles

and filzkörper, have the ventral setal bands of A6, A7 and A8 transformed toward A5 or A4, and develop rudimentary chitinized plates in posterior A8.

alleles:

allele	origin	discoverer	synonym	molecular biology
<i>Abd-B</i> ^{D3}	DEB	Duncan	<i>iab7</i> ^{D3}	undetected
<i>Abd-B</i> ^{D16}	EMS	Duncan	<i>iab7</i> ^{D16}	undetected
<i>Abd-B</i> ²⁹⁷	ENU	Lewis	<i>iab7</i> ²⁹⁷	undetected
<i>Abd-B</i> ^{M1}	EMS	Morata		not examined

abx: anterobithorax (E.B. Lewis)

location: 3-58.8 (to the right of *Cbx3*; to the left of *bx*).

references: Lewis, 1978, Nature 276: 565-70.

Lewis, 1980, DIS 55: 207-08.

Lewis, 1981, Developmental Biology Using Purified Genes (Brown and Fox, eds.). Academic Press, New York, pp. 189-208.

Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, Science 221: 23-29.

Casanova, Sánchez-Herrero, and Morata, 1985, Cell 42: 663-69.

Peifer and Bender, 1986, EMBO J. 5: 2293-2303.

phenotype: Homozygotes show variable transformations of the anterior-most portion of the third thoracic segment (T3) toward the corresponding part of T2. Homozygotes also show variable transformations of posterior T2 to posterior T1. The latter effect is enhanced by low temperature. Partially complements and shows transvection with *bx*¹, *bx*³, and *bx*^{34e}. *abx/pbx* has the posterior portion of the distal segment of the haltere very slightly transformed into wing tissue; *abx/pbx*² is similar if heterozygous for a rearrangement that suppresses transvection.

alleles:

allele	origin	discoverer	synonym	molecular biology ^α
<i>abx</i> ¹	X ray	Lewis, 59j	<i>bx</i> ⁷	-79 to -73 kb deleted
<i>abx</i> ²	X ray	Kerridge	<i>bx</i> ^{SK} , <i>abx</i> ^{sk}	-79.0 to -77.5 kb deleted
<i>abx</i> ^{CAC4}	dysgenesis	Adler, 1984		-80 to -66 kb deleted, with Hobo element inserted at point of deletion.

^α Peifer and Bender, 1986, EMBO J. 5: 2293-2303.

bx: bithorax (E.B. Lewis)

location: 3-58.8 (to the right of *abx*; to the left of *Cbx*¹.)

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. 327: 137.

Morgan, Bridges, and Sturtevant, 1925, Bibliogr. Genet. 2: 79.

Lewis, 1951, Cold Spring Harbor Symp. Quant. Biol. 16: 159-74.

Lewis, 1963, Am. Zool. 3: 33-56.

Finnegan, Rubin, Young, and Hogness, 1978, Cold Spring Harbor Symp. Quant. Biol. 42: 1053-63.

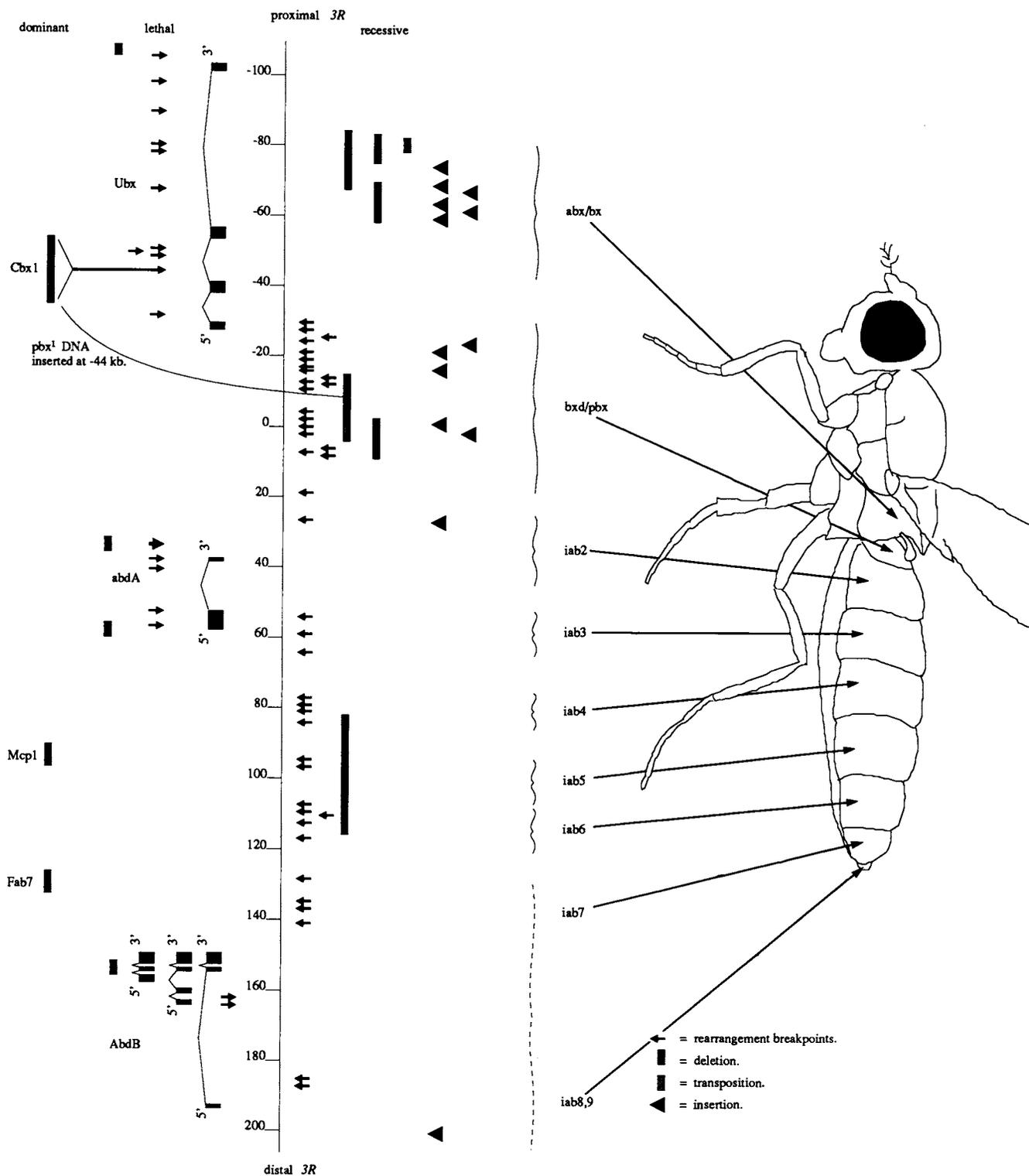
Modolell, Bender, and Meselson, 1983, Proc. Nat. Acad. Sci. USA 80: 1678-82.

Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, Science 221: 23-29.

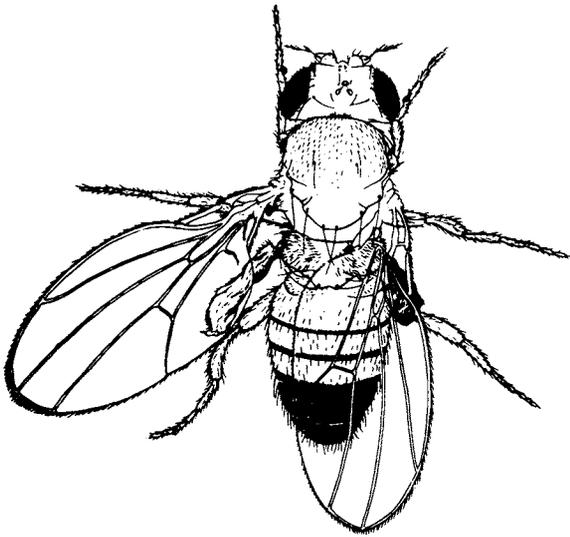
Casanova, Sánchez-Herrero, and Morata, 1985, Cell 42: 663-669.

Peifer and Bender, 1986, EMBO J. 5: 2293-2303.

phenotype: Homozygote has anterior portion of third



BXC: The Bithorax Complex Map
 From data supplied by M. O Connor. Program by D. Conner.



bx: bithorax

From Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 152.

thoracic segment (T3) transformed toward corresponding region of second (T2). The extent of this transformation is allele dependent and is most extreme in *bx*³ and weakest in *bx*⁴. Although the transformations caused by most *bx* alleles are uniform, those caused by *bx*¹ and *bx*^{34eprv} are highly variable. At 17°C several *bx* alleles show weak and variable transformations of posterior T2 to posterior T1. *bx*³, *bx*⁸, and *bx*^G over *pbx* show a very slight *pbx* effect (as described for *abx*) if heterozygous for a rearrangement that suppresses transvection.

alleles:

allele	origin	discoverer	molecular biology ^α
<i>bx</i> ¹	spont	Bridges, 1915	412 at -60 kb
<i>bx</i> ³	spont	Stern, 1925	gypsy at -57 kb; doc at -53 kb
<i>bx</i> ⁴	spont	Lewis	undetected
<i>bx</i> ⁸	EMS	Lewis, 1965	harvey at -59.5 kb
<i>bx</i> ⁹	spont	Kuhn, 1981	gypsy at -64 kb
<i>bx</i> ^{34e}	spont	Schultz, 1934	gypsy at -63.5 kb
<i>bx</i> ^{34eprv}	X ray	Lewis	-66.5 to -57 kb deleted
<i>bx</i> ^{AF}	spont	Lewis	identical to <i>bx</i> ^{34e}
<i>bx</i> ^{AF2}	spont	Lewis	identical to <i>bx</i> ^{34e}
<i>bx</i> ^{AV}	spont ^β	Rosenfeld	gypsy at -66 kb
<i>bx</i> ^{F31}	spont	Adler, 1982	I factor at -74 kb
<i>bx</i> ^G	spont	Gans	gypsy at -66 kb
<i>bx</i> ^{IS}	spont	Ising	identical to <i>bx</i> ¹
<i>bx</i> ^{Ka}	spont	Kuhn, 1983	gypsy at -61.5 kb
<i>bx</i> ^{Kb}	spont	Kuhn, 1983	gypsy at -55 kb
<i>bx</i> ^X	spont	Lewis	identical to <i>bx</i> ^{34e}

^α Peifer and Bender, 1986, EMBO J. 5: 2293-2303.
^β Probably spontaneous.

***bx*: bithoraxoid (E.B. Lewis)**

location: 3-58.8 (to the right of *Ubx*¹; to the left of *pbx*¹).
references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. 327: 137.
 Morgan, Bridges, and Sturtevant, 1925, Bibliogr. Genet. 2: 79.
 Lewis, 1951, Cold Spring Harbor Symp. Quant. Biol. 16: 159-74.
 Lewis, 1955, Am. Nat. 89: 73-89.
 Lewis, 1963, Am. Zool. 3: 33-56.
 Kerridge and Sang, 1981, J. Embryol. Exp. Morphol.

61: 69-86.

Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, Science 221: 23-29.
 Peattie and Hogness, 1984, Genetics 107: s81.
 Bender, Weiffenbach, Karch, and Peifer, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 173-80.
 Lipshitz, Peattie, and Hogness, 1987, Genes Dev. 1: 307-22.

phenotype: Homozygotes show transformation of the anterior first abdominal segment (A1) to the corresponding region of the third thoracic segment (T3). In addition, *bx**d* homozygotes have posterior T3 and posterior A1 transformed toward posterior T2. Hemizygotes for the stronger *bx**d* alleles show (with variable expression) formation of one or a pair of well-developed thoracic legs and, rarely, an extra haltere on A1; the frequency of these abdominal halteres is greatly enhanced in hemizygotes for *bx**d*⁹ *iab2*^K. The A1 legs in *bx**d* hemizygotes contain underdeveloped posterior compartments, indicating that posterior A1 is partially transformed toward thorax.

alleles: See table on the following page for allele information.



***bx*: bithoraxoid**

From Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 225.

***Cbx*: Contrabithorax (E.B. Lewis)**

location: 3-58.8.

references: Lewis, Proc. Int. Congr. Genet. 9th, 1954, 1: 100-05.
 Lewis, 1963, Am. Zool. 3: 33-56.
 Lewis, Proc. Int. Congr. Genet. 12th, 1968, 2: 96-97.
 Morata, 1975, J. Embryol. Exp. Morphol. 34: 19-31.
 Lewis, 1978, Nature 276: 565-70.
 Capdevila and García-Bellido, 1978, Wilhelm Roux's Arch. Dev. Biol. 185: 105-26.
 Lewis, 1981, Developmental Biology Using Purified Genes (Brown and Fox, eds.). Academic Press, New York, pp. 189-208.
 Lewis, 1982, Embryonic Development: Genes and Cells (Burger, ed.). Alan Liss, Inc., New York, pp. 269-88.
 Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis,

allele	origin	discoverer	synonym	cytology	molecular biology ^α	type ^β
<i>bx^d1</i>	spont	Bridges		normal	gypsy at -21 kb	I
<i>bx^d9</i>	spont	Kuhn and Lewis		normal	gypsy at -19 kb	I
<i>bx^d55i</i>	spont	Green		normal	gypsy at -23 kb	I
<i>bx^d51j</i>	spont	Lewis		normal	gypsy at -17.5 kb	I
<i>bx^d68</i>	X ray	Baker	<i>bx^d1068</i>	<i>T(2;3)A1;89E</i>		III
<i>bx^d100</i>	X ray	Lewis		<i>Tp(3;3)66;89B5-6;89E</i>		I
<i>bx^d101</i>	X ray	Lewis		<i>T(3;4)89E;101F</i>		I
<i>bx^d106</i>	X ray	Lewis		<i>In(3LR)72D11-E2;89E</i>		I
<i>bx^d110</i>	X ray	Lewis	<i>bx^d107</i>	<i>Tp(3;3)89E;91D1-2;92A2-3</i>		I
<i>bx^d111</i>	X ray	Lewis	<i>bx^d23240.1</i>	<i>Tp(3;1)4D;89E;90B2</i>		II
<i>bx^d113</i>	X ray	Lewis		<i>In(3LR)69C3-4;89E</i>		I
<i>bx^d114</i>	X ray	Lewis	<i>bx^d3978.114</i>	<i>In(3R)89E;94</i>		I
<i>bx^d121</i>	X ray	Lewis		normal	-15 kb to +60-65 kb deleted	?
<i>bx^d123</i>	X ray	Crosby		<i>T(2;3)A1;89E</i>		?
<i>bx^d125</i>	ENU	Chiang	<i>bx^d27830.C5A</i>	<i>In(3R)89B;89E</i>		I
<i>bx^d127</i>	X ray	Lewis	<i>bx^dC5A</i> <i>bx^d29315.4G</i> <i>bx^d54G</i>	<i>T(2;3)59C;89E + In(3R)88C-D;92</i>		II
<i>bx^d183</i>	EMS	Lewis	<i>bx^d17756.83</i>	<i>In(3R)89C;89E</i>		I
<i>bx^d194</i>	X ray	Lewis	<i>bx^d19409.2X</i> <i>bx^d92</i> <i>bx^d24032.266</i>	<i>In(3LR)80F;89E</i>		II
<i>bx^d266</i>	X ray	Tung	<i>bx^d16765.7</i>	<i>T(2;3)A0;89E</i>		III
<i>bx^d657</i>	EMS	Lewis		<i>In(3R)81;89E</i>		I
<i>bx^dDB1</i>	X ray	D. Baker		<i>T(2;3)A1;89E</i>		I
<i>bx^dDB3</i>	X ray	D. Baker		<i>T(2;3)A8;89E</i>		II
<i>bx^dDB4</i>	X ray	D. Baker		<i>T(2;3)32;89E</i>		?
<i>bx^dDB5</i>	X ray	D. Baker	<i>bx^dB231</i>	<i>T(2;3)A1;89E</i>		?
<i>bx^dDB6</i>	X ray	D. Baker		<i>T(2;3)22A;43A-C;60D;84;89E;92F</i>		II
<i>bx^dDB7</i>	X ray	D. Baker		<i>T(Y;3)89E</i>		?
<i>bx^dDB9</i>	X ray	D. Baker		<i>Tp(3;3)80;89E</i>		?
<i>bx^dK</i>	spont (?)	Kuhn		normal (?)	gypsy at -2.5 kb; P-element at -6.5 kb	II
<i>bx^dSR</i>	spont	Rosenberg				?
<i>bx^dUab1</i>	EMS	Lewis	<i>bx^dG</i>	normal		I
<i>bx^dX</i>	X ray	Lewis	<i>bx^d22290.11X</i>	<i>T(2;3)A2B-C;89E1-2</i>		?

^α Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, Science 221: 23-29; Bender, Weiffenbach, Karch, and Peifer, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 173-80.

^β Type is as described by Bender *et al.*, 1985.

- I Strong *bx^d* (Hemizygotes): Transformation of posterior portion of haltere towards wing, loss of A1 tergite, and appearance of T3 legs on A1. Ventral setal belt of A1 is transformed toward that of T3, and ventral pits appear on abdominal segments A1 to A7, inclusive.
- II Intermediate *bx^d* (Hemizygotes): Transformation of posterior portion of haltere toward wing, replacement of A1 tergite by post-notal tissue as in (I), no extra legs. A1 setal belt is intermediate between A1 and T3 type belts. Ventral pits on all abdominal segments.
- III Weak *bx^d* (Hemizygotes): Only two *bx^d* effects remain: A1 tergite is slightly reduced in hemizygote but not in homozygote, and ventral pits are formed on A1 to A7, inclusive, in the hemizygote and on A1 in the homozygote. These weak alleles when homozygous survive to adults and appear wild type.

and Hogness, 1983, Science 221: 23-29.

Casanova, Sánchez-Herrero, and Morata, 1985, J. Embryol. Exp. Morphol. 90: 179-196.

White and Akam, 1985, Nature 318: 567-69.

phenotype: *Cbx¹/+* has a strong transformation of the posterior region of the second thoracic segment (T2) toward the corresponding region of the third (T3), and a weak and variable transformation of anterior T2 toward anterior T3. The *Cbx¹* homozygote differs in having a stronger, but still variable, transformation of anterior T2 toward T3. *Cbx¹/Ubx* has a slight enhancement of the *Ubx* phenotype (see also *su-Cbx*). *Cbx²* has both anterior and posterior regions of T2 moderately transformed toward T3. *Cbx^{Hm}* affects only the wing, which is strongly transformed to haltere. Flies carrying two doses of *Cbx^{Hm}* plus a normal allele have a virtually complete transformation of wing to haltere (as figured in Lewis,

1982). *Cbx²* and *Cbx^{Hm}* have inseparable recessive *bx^d* effects. *Cbx³/+* transforms anterior portions of T2 variably toward anterior T3. It has no effect in posterior T2. For an overview of the effects of *Cbx* mutants on specific structures see Table 1: Lewis, 1982.

allele	origin	discoverer	synonym	cytology	molecular biology ^α
<i>Cbx¹</i>	X ray	Bacon		normal	insert of -3 kb to +14 kb in inverted orientation at -44 kb
<i>Cbx²</i>	?	Kreber		<i>In(3R)89E;91C-E</i>	+8 to +10 kb
<i>Cbx³</i>	X ray	Akam		<i>In(3R)89A;89E</i>	-110 to -103 kb
<i>Cbx^{Twt}</i>	X ray	Abbot		<i>In(3R)87EF;89E</i>	-110 to -103 kb
<i>Cbx^{Hm}</i>	X ray	Slatis	<i>Hm</i>	<i>T(2;3)breaks?</i>	-27.5 kb to -29.5 kb

^α Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, Science 221: 23-29.

Hab: Hyperabdominal (E.B. Lewis)

location: 3-58.8 (between *iab2* and *iab3*: to the right of *abd-A*^{C53}).

synonym: *Cbx*d (Contrabithoraxoid).

references: Lewis, Proc. Int. Congr. Genet. 12th, 1968, 2: 96-97.

Lewis, 1978, Nature 276: 565-70.

Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.

Bender, Weiffenbach, Karch, and Peifer, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 173-80.

phenotype: *Hab*/+ has the third thoracic segment (T3) and first abdominal segment (A1) variably transformed toward the second abdominal segment (A2), occasionally resulting in the loss of one or both metathoracic legs and one or both halteres; an A2 type tergite and sternite appear on T3; but A1 is only weakly transformed toward A2. Strongly enhanced when mother is from stock of *Df(3R)red-P93, l(3)tr Sb/In(3L)P + In(3R)P18, Me Ubx*.

alleles:

allele	origin	discoverer
<i>Hab</i> ¹	EMS	Bacher, 66k
<i>Hab</i> ²	EMS	Duncan

iab2: infra-abdominal 2 (I. Duncan)

location: 3-58.8 (to the right of *pbx*; to the left of *abd-A*).

references: Lewis, 1978, Nature 276: 565-70.

Kuhn, Woods, and Cook, 1981, Mol. Gen. Genet. 181: 82-86.

Sánchez-Herrero, Vernós, Marco, and Morata, 1985, Nature 313: 108-13.

Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.

Duncan, 1987, Annual Review of Genetics 21: 285-391.

phenotype: Adult homozygotes or hemizygotes show transformations of A2 toward A1. The known alleles cause only partial transformations. Wheeler's organs (A2 structures) are reduced or absent and A2 tergite bristles are reduced in size. These alleles do not affect the larval cuticle pattern.

alleles:

allele	origin	discoverer	synonym	cytology	molecular biology
<i>iab2</i> ⁶⁷¹	X ray	Lewis		<i>T(Y;2;3)21E;26;86;89E</i> on <i>In(3R)81F;90C-D</i>	
<i>iab2</i> ^K	Spont	Woods		normal	gypsy insert at 27.5 kb
<i>iab2</i> ^{S3}	X ray	Tiong	<i>abd-A</i> ^{S3}	<i>In(3R)89E;93F</i>	24-28 kb

iab3: infra-abdominal 3 (E.B. Lewis)

location: 3-58.8 (to the right of *abd-A*; to the left of *iab4*).

references: Lewis, 1978, Nature 276: 565-70.

Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.

Lewis, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 155-64.

Sanchez-Herrero and Akam, 1989, Development 107: 321-89.

phenotype: Hemizygote and homozygote have third, fourth, fifth and sixth abdominal segments (A3, A4, A5, and A6) transformed toward the second abdominal segment (A2). The Wheelers Organ (normally only on A2)

is now partially to fully developed on A3 to A6, inclusive. Hemizygotes are viable, and show a loss of gonads in both sexes. In homozygotes A1 is weakly transformed toward A2.

alleles:

allele	origin	discoverer	synonym	cytology	molecular biology ^α
<i>iab3</i> ³³	X ray	R.H. Baker	<i>iab3</i> ^{35250.33}	<i>In(3LR)74A;89E</i>	
<i>iab3</i> ⁸⁶	X ray	Lewis		<i>In(3R)62C;89E</i> on <i>In(3R)81F;90C-D</i>	
<i>iab3</i> ²⁷⁷	X ray	D. Baker	<i>Mcp</i> ^{revB277} , <i>iab3</i> ^{B277} , <i>iab3</i> ^{DB}	<i>Tr(3;3)89E;</i> <i>94A;96F;</i> <i>89E</i> on <i>In(3R)81;</i> <i>92D-E</i>	63-64.5 kb
<i>iab3</i> ⁹⁹⁹	X ray	Lewis		<i>In(3R)68F;89E</i> on <i>In(3R)81E;90C-D</i>	
<i>iab3</i> ²⁵⁷⁶	X ray	Lewis		<i>T(2;3)51D-E;89E</i> on <i>In(3R)81E;90C-D</i>	
<i>iab3</i> ^{Uab4}	EMS	Lewis	<i>iab3</i> ^{Uab} , <i>Uab</i> ⁴	<i>In(3R)80C;</i> <i>85A;89E</i>	58.5-61.5 kb

^α Coordinates of rearrangement breakpoint (Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96).

iab4: infra-abdominal 4 (E.B. Lewis)

location: 3-58.5 (to the right of *iab3*; to the left of *Mcp*).

references: Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.

Lewis, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 155-64.

Cumberledge, Zaratzian, and Sakonju, 1990, Proc. Nat. Acad. Sci. USA 87: 3259-63.

phenotype: Both the hemizygote and homozygote are viable and have a transformation of A4 toward A3 as well as a weak transformation of A5 toward A4 or A3. Gonads are absent in both sexes (or partially developed in some alleles). In some of the alleles, A2 transforms weakly to A3, especially in the homozygote.

alleles: Eight alleles induced by X rays.

allele	discoverer	synonym	cytology	molecular biology ^α
<i>iab4</i> ⁴⁵	R.H. Baker	<i>iab4</i> ^{31616.1045} , <i>iab4</i> ¹¹⁰⁴⁵	<i>T(2;3)32;41;89E</i>	78.5-82 kb
<i>iab4</i> ¹²⁵	R.H. Baker	<i>Mcp</i> ^{rev 31125.27} <i>iab4</i> ^{31125.27}	<i>T(3;4)89E;101</i>	81-83 kb
<i>iab4</i> ¹⁶⁶	Von Der Ahe	<i>Mcp</i> ^{rev 31166.1} <i>iab4</i> ^{31166.1}	<i>In(3LR)79D-E;89E</i>	76-83 kb
<i>iab4</i> ¹⁸⁶	Lewis		<i>In(3LR)73D-F;89E</i> on <i>In(3R)81F;90C-D</i>	
<i>iab4</i> ³⁰²	R.H. Baker	<i>iab4</i> ^{31616.302}	<i>T(2;3;4)60D;81;</i> <i>89E;100F;101</i>	83-86.5 kb
<i>iab4</i> ³¹¹⁰	Lewis		<i>In(3R)81F;89E</i> on <i>In(3R)81F;90C-D</i>	
<i>iab4,5</i> ⁸⁴⁹	Lewis		<i>In(3LR)76F-G;89E</i> on <i>In(3R)81F;90C-D</i>	
<i>iab4,5</i> ^{DB}	D. Baker	<i>iab5</i> ^{DB}		85-113 kb deleted

^α Coordinates of rearrangement breakpoint unless otherwise indicated (Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96).

iab5: infra-abdominal 5 (I. Duncan)

location: 3-58.8 (to the right of *iab4*; to the left of *iab6*).

references: Lewis, 1978, Nature 276: 565-70.

Lewis, 1981, Developmental Biology Using Purified Genes (Brown and Fox, eds.). Academic Press, New York, pp. 189-208.

Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker,

Crosby, and Lewis, 1985, Cell 43: 81-96.

Lewis, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 155-64.

Duncan, 1987, Annual Review of Genetics 21: 285-319.

phenotype: Hemizygotes show strong transformation of A5 toward A4, resulting in a loss of black pigment in the A5 tergite of the male. In addition, A6 may be weakly transformed toward A4. When homozygous, *iab5*³⁰¹ causes a weak transformation of A3 toward A4 as well as a transformation of A5 to A4.

alleles: Five alleles induced by X rays.

allele	discoverer	synonym	cytology	molecular biology ^α
<i>iab5</i> ¹⁹⁸	Lewis		<i>In(3R)89A;89E</i> on <i>In(3R)81F;90C-D</i>	
<i>iab5</i> ³⁰¹	R.H. Baker	<i>iab5</i> ^{31616.301}	<i>Tp(2;3)41;86E;</i> <i>89E</i>	95-104 kb deleted
<i>iab5</i> ⁷⁷¹	Lewis		<i>In(3LR)69E-F;89E</i> on <i>In(3R)81F;90C-D</i>	
<i>iab5</i> ⁸⁴³	R.H. Baker	<i>iab5</i> ^{31616.843}	<i>T(2;3)24E-F;</i> <i>53F;81;89B;</i> <i>89E</i>	93-99.5 kb
<i>iab5,6</i> ¹⁸⁸¹	Lewis		<i>T(2;3)30D-E;89E</i> on <i>In(3R)81F;90C-D</i>	

^α Coordinates of rearrangement breakpoint unless otherwise indicated (Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96).

iab6: infra-abdominal 6 (I. Duncan)

location: 3-58.8 (to the right of *iab5*; to the left of *iab7*).

references: Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.

Duncan, 1987, Annual Review of Genetics 21: 285-319.

phenotype: Hemizygotes show strong transformations of both A5 and A6 toward A4. Males show a loss of pigment on the A5 and A6 tergites and show the development of bristles on the A6 sternite. Some alleles cause weak transformations of A4 toward A5.

alleles: *iab6*^{C7} induced by ethyl methanesulfonate. The other alleles induced by X rays.

allele	discoverer	synonym	cytology	molecular biology ^α
<i>iab6</i> ¹¹	Lewis		<i>In(3LR)72;89E</i> on <i>In(3R)81F;90C-D</i>	113.2-115 kb
<i>iab6</i> ⁷⁵	R.H. Baker	<i>iab5</i> ⁷⁵ <i>Mcp</i> ^{rev31075.26}	<i>T(2;3)30A-B;89F;</i> <i>31D-E;59F;89E</i>	103-108 kb
<i>iab6</i> ¹⁰⁵	Lewis	<i>iab5</i> ¹⁰⁵	<i>T(2;3)60C;89E</i>	108-111 kb
<i>iab6</i> ²⁰⁰	Lewis		<i>T(2;3)29E-F;89E</i> on <i>In(3R)81F;90C-D</i>	120-121.2 kb
<i>iab6</i> ¹⁸²¹	Hong		<i>T(2;3)30F-31A;89E</i> on <i>In(3R)81F;90C-D</i>	116-118 kb
<i>iab6</i> ^{C1}	Crosby	<i>Mcp</i> ^{revC1}	<i>T(2;3)60B;81;</i> <i>89E</i>	108-111 kb
<i>iab6</i> ^{C7}	Crosby	<i>iab5</i> ^{C7} , <i>Mcp</i> ^{rvC7}		insertion of DNA from 90E at 124 kb
<i>iab6</i> ^{Vno}	E.H. Grell	<i>Vno</i>	<i>Tp(3;3)89E;93F;</i> <i>97A</i>	108-111 kb

^α Coordinates of rearrangement breakpoint unless otherwise indicated (Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96).

iab7: infra-abdominal 7 (I. Duncan)

location: 3-58.8 (to the right of *iab6*; to the left of *Abd-B*).

references: Sánchez-Herrero, Vernós, and Morata, 1985, Nature 313: 106-13.

Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.

Sánchez-Herrero, Casanova, Kerridge, and Morata, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 165-72.

Duncan, 1987, Annual Review of Genetics 21: 285-319.

phenotype: Hemizygotes show strong transformations of A5, A6, and A7 toward A4. In males, the A5 and A6 tergites show a loss of pigmentation and an unpigmented A4-type tergite develops in A7. Both A6 and A7 show the development of sternites with bristles. Heterozygotes show a small A7 tergite in the male. Two gain-of-function alleles recorded. *iab7*^{Spth} (split thorax) heterozygotes display a longitudinal furrow in the mesothorax; *iab7*^{SGA} heterozygotes causes abdominal structures to develop in the back of the head (Awad, Gausz, Gyurkovics, and Párducz, 1981, Acta Biol. Acad. Sci. Hung. 32: 219-28; Kuhn and Packert, 1988, Dev. Biol. 125: 8-18).

alleles: Seven alleles induced by X rays.

allele	discoverer	synonym	cytology	molecular biology ^α
<i>iab7</i> ¹⁶⁴	Lewis		<i>In(3R)65;89E</i> on <i>In(3R)81F;90C-D</i>	126-128 kb
<i>iab7</i> ⁷⁷⁰	Hong		<i>In(3LR)68;89;94</i> on <i>In(3R)81F;90C-D</i>	
<i>iab7</i> ³⁰⁸¹	Charles		<i>In(3R)66;79;89</i> on <i>In(3R)81F;90C-D</i>	
<i>iab7</i> ^{MX1}	Casanova	<i>iab6</i> ^{MX1} <i>Abd-B</i> ^{MX1}	<i>T(Y;3)63A;</i> <i>66B;68A;72E;</i> <i>89B;92A-D;97D-F;</i> <i>98F;Y</i>	126-129 kb
<i>iab7</i> ^{MX2}	Casanova	<i>iab6</i> ^{MX2} <i>Abd-B</i> ^{MX2}	<i>In(3LR)64A;</i> <i>89A;89E</i>	139.5-142 kb
<i>iab7</i> ^{SGA}	Gyurkovics	<i>SGA</i> ⁶²	<i>In(3R)88C;89E</i>	133-139.5 kb
<i>iab7</i> ^{Spth}	Kempthues	<i>iab6</i> ^{Spth}	<i>In(3R)89A;89E</i>	133-139.5 kb

^α Coordinates of rearrangement breakpoint unless otherwise indicated (Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96).

iab8: infra-abdominal 8 (I. Duncan and S. Celniker)

location: 3-54.8 (to the right of *Abd-B*; to the left of *iab-9*).

references: Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.

Casanova, Sánchez-Herrero, and Morata, 1986, Cell 47: 627-36.

Celniker and Lewis, 1987, Genes. Dev. 1: 111-23.

Duncan, 1987, Annual Review of Genetics 21: 285-319.

phenotype: Hemizygous adult males show strong transformation of A5, A6, A7 toward A4. In addition, an A8 tergite develops which is half the size of a normal tergite. In these males, A5 and A6 tergites show a loss of pigmentation and an unpigmented A4-type tergite develops in A7. A6, A7, and A8 all development sternites, the first two with bristles.

alleles:

allele	origin	discoverer	synonym	molecular biology
<i>iab8</i> ³⁸⁰	ENU	Lewis	<i>iab7</i> ³⁰⁸	
<i>iab8</i> ^{D6}	EMS	Duncan	<i>iab7</i> ^{D6}	
<i>iab8</i> ^{D14}	EMS	Duncan	<i>iab7</i> ^{D14}	157-157.4 kb detected
<i>iab8</i> ^{D15}	EMS	Duncan	<i>iab7</i> ^{D15}	

***iab9*: infra-abdominal 9**

(I. Duncan and S. Celniker)

location: 3-58.8 (to the right of *Abd-B*; this is the distal most region of the BXC).

references: Gardner and Woolf, 1949, *Genetics* 34: 573-85.

Lewis, 1978, *Nature* 276: 565-70.

Kuhn, Woods, and Cook, 1981, *Mol. Gen. Genet.* 181: 82-86.

Lewis, 1981, *Developmental Biology Using Purified Genes* (Brown and Fox, eds.). Academic Press, New York, pp. 189-208.

Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, *Cell* 43: 81-96.

Casanova, Sánchez-Herrero, and Morata, 1986, *Cell* 47: 627-36.

Celniker and Lewis, 1987, *Genes. Dev.* 1: 111-23.

Duncan, 1987, *Annual Review of Genetics* 21: 285-319.

phenotype: Adult homozygotes or hemizygotes show absent or defective genitalia and analia in both sexes. Adults homozygous or heterozygous for *iab9*⁶⁵ show in addition a partial transformation of A6 toward A7. Embryos hemizygous for *iab9* mutations show the development of a zone of naked cuticle and a rudimentary ninth abdominal setal belt posterior to the eighth abdominal setal belt. Posterior spiracles are absent in *iab9*⁶⁵ and *iab9*⁴⁸ hemizygotes and are defective in *iab9*^{Uab1}, *iab9*^{tuh-3}, and *iab9*^{Tab} hemizygotes. *iab9*⁶⁵ and *iab9*⁴⁸, but not the other *iab9* mutations, cause transformations of the A8 setal belt (located in anterior A8) toward A7.

alleles:

allele	origin	discoverer	synonym	cytology	molecular biology
<i>iab9</i> ⁴⁸	X ray	R.H. Baker		<i>In(3R)89E</i> ; <i>100C on</i>	+163-166.5 kb
<i>iab9</i> ⁶⁵	X ray	R.H. Baker	<i>iab7</i> ⁶⁵	<i>In(3R)81;92E</i>	+163-166.5 kb
<i>iab9</i> ¹³⁹²	X ray	Hong		<i>T(2;3)41;89E</i> <i>In(3R)63A;89E</i> <i>on In(3R)81F;90C-D</i>	182-187 kb
<i>iab9</i> ¹⁶⁴⁵	X ray	Hong		<i>T(2;3)26A;89E</i> <i>on In(3R)81F;90C-D</i>	182-187 kb
<i>iab9</i> ^{Tab}	X ray	Lewis	<i>Tab</i>	<i>In(3R)89E;90D</i>	+187-188 kb
<i>iab9</i> ^{tuh-3}	spont	Woolf	<i>tuh-3</i>		insert of Delta 88 at +200 kb
<i>iab9</i> ^{Uab1}	EMS	Lewis	<i>Uab1</i>		inversion of -14 to +185 kb
<i>iab9</i> ^{x23-1}	X ray	Casanova	<i>x23-1</i>		

***Mcp*: Miscaldastral pigmentation (E.B. Lewis)**

location: 3-58.5 (to the right of *iab4*; to the left of *iab6*).

origin: Spontaneous.

discoverer: Crosby.

synonym: *Male chauvinist pigmentation*.

references: Lewis, 1978, *Nature* 276: 565-70.

Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, *Cell* 43: 81-96.

Duncan, 1986, *Cell* 47: 297-309.

phenotype: *Mcp* homozygotes have the fourth (A4) and fifth (A5) abdominal segments transformed to a state intermediate between A5 and A6. Similar, but weaker, transformations occur in *Mcp*¹/+ heterozygotes. *Mcp*¹ can be scored in males by dark pigmentation of the A4 tergite and in females by an effect on the orientation of the lateral bristles of the A4 tergite.

alleles:

allele	origin	discoverer	cytology	molecular biology ^α
<i>Mcp</i> ¹	spont	Crosby	normal	94-97.6 kb deleted
<i>Mcp</i> ²	spont	Crosby		

^α Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, *Cell* 43: 81-96.

***pbx*: postbithorax**

location: 3-58.8 (to the right of *bx4*; to the left of *iab2*).

references: Lewis, *Proc. Int. Congr. Genet.* 9th, 1954, 1: 100-05.

Lewis, 1955, *Am. Nat.* 89: 73-89.

Lewis, 1981, *Developmental Biology Using Purified Genes* (Brown and Fox, eds.). Academic Press, New York, pp. 189-208.

Lewis, 1982, *Embryonic Development: Genes and Cells* (Burger, ed.). Alan Liss, Inc., New York, pp. 269-88.

Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, *Science* 221: 23-29.

phenotype: Adult homozygotes and hemizygotes have posterior region of third thoracic segment (T3) transformed toward corresponding region of the second (T2).

alleles:

allele	origin	discoverer	cytology	molecular biology ^α
<i>pbx</i> ¹	X ray	Lewis, 1954	normal	-3 to +14 kb deleted
<i>pbx</i> ²	X ray	Lewis, 1980?	normal	14 to +1 kb deleted

^α Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, *Science* 221: 23-29.

***Sab*: Superabdominal (E.B. Lewis)**

location: 3-58.8 (just to the right of *Mcp*).

synonym: *Uab-5*^{*Sab*}.

references: Sakonju, Lewis, and Hogness, 1984, *Genetics* 107: s93.

Duncan, 1986, *Cell* 47: 297-309.

phenotype: *Sab*⁺ adults show patchy transformations of A3 and A4 to A5. Homozygote viable and more extreme than heterozygote. *Mcp*¹ *Sab*¹ homozygotes show transformations of A3, A4, and A5 to a state intermediate between A5 and A6.

alleles:

allele	origin	discoverer	synonym	cytology
<i>Sab</i> ¹	ENU	Sakonju	<i>Uab4</i> , <i>Uab4</i> ^{SS}	normal
<i>Sab</i> ²	ENU	Lewis	<i>Uab4</i> -like, <i>Uab4</i> ^b <i>Uab4-427995</i> , <i>Sabb</i>	normal

***Tab*: Transabdominal (E.B. Lewis)**

location: 3-58.8.

origin: X ray induced.

discoverer: E.B. Lewis.

synonym: *Net*: Neo-ectopic tergite; *Eat*: Ectopic abdominal tergite; *Comma*-like.

references: Celniker and Lewis, 1984, *Genetics* 107: s17.

Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, *Cell* 43: 81-96.

Celniker and Lewis, 1987, *Genes Dev.* 1: 111-23.

phenotype: Heterozygous adults have a pair of laterally disposed longitudinal stripes of tissue on the second thoracic segment (T2) probably corresponding to tissue from the dorsal sixth (A6) and/or seventh (A7) abdominal segments. These ectopic stripes of tissue are entirely

black in males but only partially pigmented in females. *Tab/+* flies are virtually sterile, and have a thin seventh tergite in males. Fertility is partially restored in the presence of a duplication for *BXC*, such as *Dp(3;1)P68*. When hemizygous, *Tab* embryos have a reduction of the posterior spiracles and filzkörper, and a tiny ninth abdominal ventral setal belt that appears as a small row of denticles posterior to the A8 setal belt.

cytology: Associated with *In(3R)Tab = In(3R)89E;90D*.

molecular biology: The inversion puts the 90D region into the *BXC* at +188 kb., in the presumed *iab9* region (see Karch *et al.*, 1985).

Uab: Ultraabdominal (E.B. Lewis)

location: 3-58.8.

references: Kiger, 1976, *Dev. Biol.* 50: 187-200.

Davis and Kiger, 1977, *Dev. Biol.* 58: 114-23.

Lewis, 1978, *Nature* 276: 565-70.

Lewis, 1982, *Embryonic Development: Genes and Cells* (Burger, ed.). Alan Liss, Inc., New York, pp. 269-88.

Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, *Cell* 43: 81-96.

phenotype: *Uab*¹, *Uab*², and *Uab*⁴ cause A1 to transform toward A2. *Uab*⁵ causes A1 and A2 to transform toward A3. *Uab*¹ is an intra-complex inversion with a breakpoint in the *bx*d region, *Uab*² is associated with a *Uab* mutation, the *Uab*⁴ breakpoint is an *iab-3* mutation, and the *Uab*⁵ breakpoint is a weak *bx*d mutation. Reversion studies show that *Uab*¹, *Uab*⁴, and *Uab*⁵ cause abnormal expression of *abd-A* domain functions in A1.

alleles:

allele	origin	discoverer	synonym	cytology	molecular biology
<i>Uab</i> ¹	EMS	Lewis			-14 kb; +185 kb inverted
<i>Uab</i> ²	X ray	Lewis			
<i>Uab</i> ⁴	EMS	Lewis	<i>iab3</i> <i>Uab4</i>	<i>In(3LR)80C;85A;89E</i>	58.5-61.5 kb
<i>Uab</i> ⁵	EMS	N. Shaw		<i>T(1;3)1F;89E3-4</i>	

Ubx: Ultrabithorax (E.B. Lewis)

location: 3-58.8 (to the right of *Cbx*, to the left of *bx*d).

synonym: *bx*^D, *bx*d^D, *bx*l.

references: Bridges and Brehme, 1944, Carnegie Inst.

Washington Publ. 552: 35.

Lewis, 1951, Cold Spring Harbor Symp. Quant. Biol. 16: 159-74.

Lewis, 1955, *Am. Nat.* 89: 73-89.

Lewis, 1963, *Am. Zool.* 3: 33-56.

Akam, 1983, *EMBO J.* 2: 2075-84.

Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, *Science* 221: 23-29.

Akam, Moore, and Cox, 1984, *Nature* 309: 635-37.

Hayes, Sato, Denell, 1984, *Proc. Nat. Acad. Sci. USA* 81: 545-49.

Struhl, 1984, *Nature* 308: 454-57.

White and Wilcox, 1984, *Cell* 39: 163-71.

Akam and Martinez-Arias, 1985, *EMBO J.* 4: 1689-1700.

Beachy, Helfand, and Hogness, 1985, *Nature* 313: 545-51.

Hogness, Lipshitz, Beachy, Peattie, Saint, Goldschmidt-Clermont, Harte, Gavis, and Helfand, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 181-94.

Struhl and White, 1985, *Cell* 43: 507-19.

Beachy, Krasnow, Gavis, and Hogness, 1988, *Cell* 55: 1069-81.

O'Connor, Binari, Perkins, and Bender, 1988, *EMBO J.* 7: 435-45.

Kornfeld, Saint, Beachy, Harte, Peattie, and Hogness, 1989, *Genes Dev.* 3: 243-58.

Mann and Hogness, 1990, *Cell* 60: 597-610.

phenotype: Larvae homozygous and hemizygous for strong *Ubx* mutants, such as the *Ubx*¹-type, have the ventral setal bands or "hooklets" of the first abdominal segment (A1) and T3 transformed toward those of T2; dorsally, the hair patterns of posterior T2 and posterior T3 are transformed toward posterior T1; Keilin organs with 2 hairs appear on A1; ventral pits appear on segments A1 thru A7; two extra sets of anterior spiracles appear, one on T3 and one on A1. Homozygotes die, usually as tiny third-instar larvae, but occasionally grow to a normal-sized third-instar larvae and may pupate. Homozygotes of some weaker alleles, such as *Ubx*^{61d}, survive to adult stage and show weak *bx*, *bx*d, and *pbx* effects, especially in the haltere, and A1 is generally reduced.

THE GENOME OF *DROSOPHILA MELANOGASTER*

alleles:

allele	origin	discoverer	synonym	cytology	type
Ubx¹	spont	W.F. Hollander, 34	<i>bx^D</i> , <i>bx^d 1D</i> , <i>Bxl</i>	normal	
Ubx^{1A}	X ray		<i>Ubx^{19649.1}</i>	<i>T(2;3)41;89E</i>	
Ubx^{1K}	X ray	Lewis, 64c	<i>Ubx^{15152.1K}</i>		
Ubx^{1U}			<i>Ubx^{18264.1U} ***</i>	<i>T(2;3)31;89E</i>	
Ubx^{1X}	EMS	Smit, 71k	<i>Ubx^{1007.14}</i> <i>Ubx^{19061X}</i> <i>Ubx^{23240.2E}</i>		unusual <i>Ubx</i>
Ubx^{2E}			<i>Ubx^{18498.2J}</i>		like <i>Ubx¹¹</i>
Ubx^{2J}	EMS	Lewis, 69g	<i>Ubx^{19649.2P}</i>	<i>T(Y;2;3)39;89E1-2;91F</i>	
Ubx^{2P}	X ray		<i>Ubx^{16612.2R}</i>		like <i>Ubx¹¹</i>
Ubx^{2R}	EMS	Bacher, 66h	<i>Ubx^{16800.2V}</i>		weak <i>Ubx</i>
Ubx^{2V}	EMS	Bacher, 66h	<i>Ubx^{18264.2V} ***</i>		like <i>Ubx¹¹</i>
Ubx^{2W}			<i>Ubx^{26074.4}</i>		
Ubx⁴	EMS	Tung, 81b	<i>Ubx^{19530.4A}</i>		
Ubx^{4A}	EMS	Lewis, 73k	<i>Ubx^{3944.4}</i>		
Ubx^{4B}			<i>Ubx^{15152.6C}</i>		
Ubx^{6C}	X ray	Lewis, 64c	<i>Ubx^{21560.6Q}</i>	<i>In(3R)89E3-4;96F;97A</i>	<i>Cbx</i> -like rev. " <i>Ubx</i> "
Ubx^{6Q}			<i>Ubx^{19286.7L}</i>	<i>In(3R)89E1-2;96A</i>	
Ubx^{7L}	X ray	Smit, 72i	<i>Ubx^{15152.7M}</i>	<i>In(3R)</i>	
Ubx^{7M}	X ray	Lewis, 64c	<i>Ubx^{12110.8}</i>		
Ubx⁸			<i>Ubx^{21560.8A}</i>	<i>T(1;3)5B;89E1-2</i>	<i>Cbx</i> -like rev. " <i>Ubx</i> "
Ubx^{8A}			<i>Ubx^{26074.8}</i>		
Ubx^{8B}	EMS	Tung, 81b	<i>Ubx^{19286.8M}</i>		
Ubx^{8M}	X ray	Smit, 72i	<i>Ubx^{15152.8N}</i>	<i>In(3R)</i>	
Ubx^{8N}	X ray	Lewis, 64c	<i>Ubx^{26074.10}</i>		
Ubx¹⁰	EMS	Tung, 81c	<i>Ubx²⁸⁵¹⁰</i>		A1 missing
Ubx^{10A}	ENU	Chiang, 82f	<i>Ubx^{26074.12}</i>		
Ubx¹²	EMS	Tung, 81c	<i>Ubx^{19191.12C}</i>		weak <i>Ubx</i>
Ubx^{12C}	EMS	Shaw, 72a	<i>Ubx^{26930.13}</i>		very weak <i>Ubx</i>
Ubx¹³	ENU	Lewis, 81i	<i>Ubx^{19286.16N}</i>	<i>In(3R)89E;99</i>	
Ubx^{16N}	X ray	Smit, 72i	<i>Ubx^{19709.16V}</i>	normal	
Ubx^{16V}	X ray		<i>Ubx^{19709.18W}</i>	normal	like <i>Ubx¹¹</i>
Ubx¹⁸	X ray		<i>Ubx^{27510.C9A}</i>	normal	larvae die over <i>bx^d</i> at 2-3instar
Ubx¹⁹	ENU	Chiang, 82b			
Ubx^{21J}	EMS	Lewis, 73k	<i>Ubx^{19530.21J}</i>		
Ubx^{21R}	X ray		<i>Ubx^{19649.21R}</i>	<i>T(Y;3)89E</i>	
Ubx²⁴			<i>Ubx¹⁸⁶²⁴ ***</i>		
Ubx^{24K}	EMS	Lewis, 73k	<i>Ubx^{19530.24K}</i>		
Ubx²⁷	EMS	Lewis, 73k	<i>Ubx^{19530.27L}</i>		
Ubx³⁰	X ray		<i>Ubx^{19709.30G}</i>		moderate
Ubx^{30A}			<i>Ubx^{3966.30}</i>	<i>In(3R)89E1-2;91B</i>	
Ubx³¹			<i>Ubx^{16184.31E}</i>	<i>86E-F;87A</i>	
Ubx³⁴	ENU	Chiang, 82d	<i>Ubx^{28030.C4A}</i>		like <i>Ubx¹¹</i>
Ubx⁴⁰	X ray		<i>Ubx^{19709.40Z}</i>	normal	Like <i>Ubx</i> df over <i>bx^{34e}</i> like weak <i>Ubx</i> over ss <i>bx^d</i>
Ubx⁴³	X ray	Lewis, 73f	<i>Ubx^{19409.43}</i>	<i>Df(3R)89D-E1-2 (?)</i>	
Ubx⁵¹	EMS	Bacher, 65j	<i>Ubx^{16160.51P}</i>		vw
Ubx⁵⁶	ENU	Chiang, 81j	<i>Ubx²⁶⁹⁵⁶</i>		
Ubx⁵⁸	spont	Lewis, 67i	<i>Ubx^{17358U}</i>		
Ubx⁵⁹			<i>Ubx^{3858.59}</i>		
Ubx^{61d}		H. Gloor, 61d			
Ubx⁶⁵	X ray	Smit, 72i	<i>Ubx^{19286.65S}</i>		
Ubx^{66e}	EMS	Bacher, 66e	<i>Ubx^{16516.115}</i> <i>R(Ubx)</i>	normal	
Ubx^{67b}	X ray	Bacher, 67b	<i>Ubx^{16800.75Z}</i>	<i>In(3R)87F-88A;89E</i>	
Ubx⁸⁰	EMS	Bacher, 66h	<i>Ubx^{16800.88B}</i>		
Ubx⁸⁸	EMS	Bacher, 66h	<i>Ubx^{17756.98G}</i>		very weak <i>Ubx</i>
Ubx⁹⁸	EMS	Lewis, 68a		<i>In(3LR)80;89E1</i>	
Ubx¹⁰¹	X ray	Lewis, 47		normal	like <i>Ubx¹¹</i>
Ubx¹⁰²	EMS	Lewis, 68a	<i>Ubx^{17756.102H}</i>		lethal over <i>TM1</i>
Ubx¹⁰⁵		Lewis		<i>T(2;3)53C;89E1-2</i>	
Ubx¹⁰⁶	EMS	Lewis, 68a	<i>Ubx^{17756.106}</i>		like <i>Ubx¹¹</i>
Ubx¹⁰⁹		Lewis		<i>Df(3R)89D1-2-89E1-2</i>	
Ubx¹¹⁵		Lewis		unknown	
Ubx¹²⁵	X ray	Lewis		<i>In(3R)81;89E</i>	
Ubx¹³⁰	X ray	Lewis, 51l		<i>In(3LR)61A-C;74;89E;93B;96A</i>	
Ubx¹⁴⁵	EMS	Lewis, 68g	<i>Ubx^{18136.145}</i>		weak <i>Ubx</i>
Ubx¹⁴⁷	EMS	Lewis, 68g	<i>Ubx^{18136.147}</i>		weak <i>Ubx</i>
Ubx¹⁴⁹	EMS	Lewis, 68g	<i>Ubx^{18136.149}</i>	normal	like <i>Ubx¹¹</i> ; 7-legged over <i>bx^d</i>
Ubx¹⁵⁴	EMS	Lewis, 68a	<i>Ubx^{17756.154}</i>		like <i>Ubx¹¹</i>

allele	origin	discoverer	synonym	cytology	type
Ubx¹⁵⁹	EMS	Lewis, 68a	<i>Ubx^{17756.159}</i>		like <i>Ubx¹¹</i>
Ubx^{159A}	EMS	Lewis, 68g	<i>Ubx^{18136.159}</i>		like <i>Ubx¹¹</i> or weaker
Ubx¹⁷⁵	EMS	Lewis, 68a	<i>Ubx^{17756.175}</i>		like <i>Ubx^{1X}</i>
Ubx¹⁸⁰	EMS	Lewis, 68a	<i>Ubx^{17756.180}</i>		like <i>Ubx¹¹</i>
Ubx¹⁹⁵	EMS	Lewis, 68a	<i>Ubx^{17756.195}</i>		like <i>Ubx¹¹</i>
Ubx¹⁹⁶	X ray	R.H. Baker, 84a	<i>Ubx^{31616.196}</i>	<i>In(3LR)81;89E;90D1?</i>	x
Ubx²²³					
Ubx³¹¹					
Ubx⁵⁴⁹	X ray	R.H. Baker, 84d	<i>Ubx^{31616.549}</i>	normal	1
Ubx⁷⁵⁷	X ray	R.H. Baker, 84h	<i>Ubx^{31616.757}</i>	<i>89E;96-94;96-98;47-60</i>	x
Ubx⁷⁶⁵	X ray	R.H. Baker, 84h	<i>Ubx^{31616.765}</i>	normal	1
Ubx⁷⁸⁰	EMS	Lewis			
Ubx⁸⁴⁹	EMS	Lewis		normal	
Ubx⁸⁹⁵	X ray	R.H. Baker, 84k	<i>Ubx^{31616.895}</i>	<i>Tp(3.2)2R;89E;90A</i>	x
Ubx⁹⁶¹				<i>In(3R)89E;96</i>	
Ubx¹⁰⁶⁹	X ray	R.H. Baker, 85a	<i>Ubx^{31616.1069}</i>	<i>T(2;3)41A;89E</i>	x
Ubx¹³⁴³	X ray	R.H. Baker, 85d	<i>Ubx^{31616.1343}</i>	<i>T(Y;3)89E</i>	x
Ubx⁶⁷⁵⁴					
Ubx^A	X ray	Schalet, 59		10+ breaks	
Ubx^{AD}		Dowsett			
Ubx^{AR}		Robertson	<i>Ubx^{28729Z}</i>		
Ubx^{B1A}	EMS	Bacher, 65i	<i>Ubx^{16160.1A}</i>	normal	
Ubx^{B1Q}	EMS	Bacher, 66h	<i>Ubx^{16612.1Q}</i>		like <i>Ubx^{61d}</i>
Ubx^{B3}	EMS	Bacher, 65i	<i>Ubx^{16160.3B}</i>		like <i>Ubx¹¹</i>
Ubx^{B5}	EMS	Bacher, 65i	<i>Ubx^{16160.5C}</i>		like <i>Ubx¹¹</i>
Ubx^{B6}	EMS	Bacher, 65i	<i>Ubx^{16160.6D}</i>		weak
Ubx^{B7}	EMS	Bacher, 65i	<i>Ubx^{16160.7E}</i>		variable <i>Ubx</i>
Ubx^{B11}	ENU	Chiang, 82b	<i>Ubx^{27510.C1G}</i>		like <i>Ubx¹¹</i>
Ubx^{B17}	EMS	Bacher, 65i	<i>Ubx^{16160.17H}</i>		extreme <i>Ubx</i>
Ubx^{B18}	EMS	Bacher, 65i	<i>Ubx^{16160.18J}</i>	<i>T(2;3)21D1-2;89E</i>	
Ubx^{B18W}	EMS	Bacher, 66h	<i>Ubx^{16800.18W}</i>		
Ubx^{B19}	EMS	Bacher, 66h	<i>Ubx^{16800.19X}</i>		weak <i>Ubx</i>
Ubx^{B22}	EMS	Bacher, 65i	<i>Ubx^{16160.22}</i>	normal	like <i>Ubx¹¹</i>
Ubx^{B36}	EMS	Bacher, 65i	<i>Ubx^{16160.36}</i>	<i>T(2;3)41F;89E</i>	
Ubx^{B41}	EMS	Bacher, 65j	<i>Ubx^{16160.41N}</i>		like <i>Ubx^{161d}</i>
Ubx^{B49}	EMS	Bacher, 66h	<i>Ubx^{16800.49Y}</i>		weak <i>Ubx</i>
Ubx^{B54}	EMS	Bacher, 65j	<i>Ubx^{16160.54}</i>		like <i>Ubx¹¹</i> , but weaker
Ubx^{B57}	EMS	Bacher, 66k	<i>Ubx^{16160.57}</i>		
Ubx^{B58}	EMS	Bacher, 65k	<i>Ubx^{16160.58V}</i>		like <i>Ubx^{161d}</i>
Ubx^{B61}	EMS	Bacher, 65k	<i>Ubx^{16160.61W}</i>	normal	
Ubx^{B78}	EMS	Bacher, 66h	<i>Ubx^{16800.78A}</i>		like <i>Ubx¹¹</i> , but weaker
Ubx^{B104}	EMS	Bacher, 65k	<i>Ubx^{16160.104}</i>		like <i>Ubx¹¹</i>
Ubx^{B123}	EMS	Bacher, 65i	<i>Ubx^{16160.123}</i>		weak <i>Ubx</i>
Ubx^{B127}	EMS	Bacher, 65i	<i>Ubx^{16160.27}</i>		
Ubx^{B160}	EMS	Bacher, 66b	<i>Ubx^{16412.160L}</i>		weak <i>Ubx</i>
Ubx^{B203}	EMS	Bacher, 66a	<i>Ubx^{16160.2003}</i>	<i>89E1-2;89E3-4</i>	
Ubx^{B300}	EMS	Bacher, 66a	<i>Ubx^{16160.300}</i> , <i>Ubx^{1U}</i>	<i>In(3LR)61F-62A;89E</i>	
Ubx^{C1}					like <i>Ubx^{61d}</i>
Ubx^{C12}	ENU	Chiang, 82b	<i>Ubx^{27510.C2G}</i>		weak <i>Ubx</i>
Ubx^{C33}	ENU	Chiang, 82d	<i>Ubx^{27830.C3A}</i>		weak <i>Ubx</i>
Ubx^{C34}	ENU	Chiang, 82c	<i>Ubx^{27630.C4G}</i>		like <i>Ubx¹¹</i>
Ubx^{C37}	ENU	Chiang, 82d	<i>Ubx^{27830.C7G}</i>		like <i>Ubx^{161d}</i>
Ubx^{C38}	ENU	Chiang, 82c	<i>Ubx^{27630.C8G}</i>		weak <i>Ubx</i>
Ubx^{C43}	ENU	Chiang, 82c	<i>Ubx^{27740.C3G}</i>		Haltere winglike
Ubx^{C46}	ENU	Chiang, 82c	<i>Ubx^{27740.C6G}</i>		Haltere winglike
Ubx^{C48}	ENU	Chiang, 82c	<i>Ubx^{27740.C8G}</i>		Haltere winglike
Ubx^{C51}	ENU	Chiang, 82c	<i>Ubx^{27695.1}</i>		weak <i>Ubx</i> , very weak <i>pbx</i> effect
Ubx^{C52}	ENU	Chiang, 82b	<i>Ubx^{27475.C2A}</i>		very weak <i>Ubx</i>
Ubx^{C63}	ENU	Chiang, 82c	<i>Ubx^{27560.C3A}</i>		like <i>Ubx¹¹</i>
Ubx^{C64}	ENU	Chiang, 82d	<i>Ubx^{28060.C4A}</i>		A1 missing
Ubx^{C65}	ENU	Chiang, 82d	<i>Ubx^{28060.C5G}</i>		A1 missing
Ubx^{C82}	ENU	Chiang, 82b	<i>Ubx^{27480.C2G}</i>		Halteres anteriorly wing-like
Ubx^{C90}	ENU	Chiang, 82d	<i>Ubx²⁷⁹⁹⁰</i>		
Ubx^{C91}	ENU	Chiang, 82a	<i>Ubx^{27290.C1G}</i>		like <i>Ubx¹¹</i>
Ubx^{C92}	ENU	Chiang, 82a	<i>Ubx^{27290.C2A}</i>		like <i>Ubx¹¹</i>
Ubx^{C93}	ENU	Chiang, 82f	<i>Ubx^{28590.C3G}</i>		<i>bx</i> d mutant?
Ubx^{C95}	ENU	Chiang, 82a	<i>Ubx^{27290.C5A}</i>		like <i>Ubx¹¹</i>
Ubx^{C101}	ENU	Chiang, 82d	<i>Ubx^{27910.C1G}</i>		<i>bx</i> d mutant?
Ubx^{C104}	ENU	Chiang, 82d	<i>Ubx^{28477D}</i> <i>Ubx^{27910.C4G}</i>		mosaic?
Ubx^{D1}				<i>T(2;3)41;89E1-2</i>	
Ubx^{E10}	X ray	Crosby	<i>Ubx^{21368E.10}</i>		
Ubx^{E12}	X ray	Crosby	<i>Ubx^{21368E.12}</i>		
Ubx^{G2}	X ray	Lewis, 72i	<i>Ubx¹⁹²⁸⁶</i> , <i>Ubx^{1Uab-2}</i>	normal	

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allele	origin	discoverer	synonym	cytology	type
Ubx^J		Rasmuson	<i>Ubx</i> ^{23823J}		
Ubx^K	X ray	Kaufman	<i>Cbx</i> ^{+R1}	<i>In(3R)89E;92A</i>	
Ubx^{12.5}	X ray	Kerridge & Morata, 1982		<i>In(3R)88B;89E1-2</i>	
Ubx^{8.8}	X ray	Kerridge & Morata, 1982		normal	
Ubx^{5.12}	X ray	Kerridge & Morata, 1982		normal	
Ubx^{5.22}	X ray	Kerridge & Morata, 1982		<i>In(3R)88F;89E1-2</i>	
Ubx^{9.22}	X ray	Kerridge & Morata, 1982		normal	
Ubx^{5.2326}	X ray	Kerridge & Morata, 1982		normal	
Ubx^{6.26}	X ray	Kerridge & Morata, 1982		<i>T(2;3)59E;75C;89E1-2</i>	
Ubx^{6.28}	X ray	Kerridge & Morata, 1982		normal	
Ubx^{4.30}	X ray	Kerridge & Morata, 1982		<i>T(2;3)34;89E1-2</i>	
Ubx^{8.82}	EMS	Kerridge & Morata, 1982			
Ubx^{P18}		Lewis		<i>In(3R)81F;91F-92</i>	
Ubx^{P20}				<i>Tp(3;3)68A;68E;89E</i>	like <i>Ubx^X</i>
Ubx^{R3}	X ray	Ramey	<i>Cbx</i> ^{prev-R17.3C}		
Ubx^{R5}	X ray	Ramey	<i>Cbx</i> ^{rev-R17.5E}	<i>In(3R)89E;92A</i>	
Ubx^{R6}	X ray	Ramey	<i>Cbx</i> ^{rev-R17.6F}	<i>In(3R)87;89E</i>	
Ubx^{R10}	X ray	Ramey	<i>Cbx</i> ^{prev-R17.10K}	<i>T(2;3)41;89D-E</i>	
Ubx^{R13}	X ray	Ramey	<i>Cbx</i> ^{rev-R17.13N}	<i>Df(3R)89D1-2;89E1-2</i>	like <i>Ubx¹¹⁰⁹</i>
Ubx^{R14}	X ray	Ramey	<i>Cbx</i> ^{rev-R17.14P}	<i>In(3R)87D-E;89E</i>	
			<i>Ubx</i> ^{21987A}		
Ubx^{R16}	X ray	Ramey	<i>Cbx</i> ^{rev-R17.16R}	<i>In(3LR)80B;89E</i>	
Ubx^{R17}	X ray	Ramey	<i>Cbx</i> ^{rev-R17.17S}	<i>T(2;3)22B1-2;89E1-2</i>	
Ubx^{R20}	X ray	Ramey	<i>Cbx</i> ^{rev-R17.20V}	<i>In(3R)87B-D;89E</i>	
			<i>Ubx</i> ^{21988B}		
Ubx^{R22}	X ray	Ramey	<i>Cbx</i> ^{rev-R17.22X}	<i>T(2;3)41;89E</i>	
Ubx^{R29}	X ray	Ramey	<i>Cbx</i> ^{rev-R17.29E}	<i>Df(3R)89D;89E</i>	
Ubx^{R31}	X ray	Ramey	<i>Cbx</i> ^{rev-R17.31G}		like <i>Ubx^{161D}</i>
Ubx^{R32}	X ray	Ramey	<i>Cbx</i> ^{rev-R17.32H}	<i>Df(3R)89C;89D-E</i>	
Ubx^{R34}	X ray	Ramey	<i>Cbx</i> ^{rev-R17.34K}	<i>T(2;3)41;89E</i>	dom. <i>Ubx</i>
Ubx^{R40}	X ray	Ramey	<i>Cbx</i> ^{rev-R17.40R}	<i>T(3;4)89E;101</i>	
Ubx^{R42}	X ray	Ramey	<i>Cbx</i> ^{rev-R17.42T}	<i>In(3LR)70D;89E</i>	
Ubx^{R49}	X ray	Ramey	<i>Cbx</i> ^{rev-R17.49A}	<i>T(1;3)20F;89E</i>	<i>Cbx, bxd^{1(V)}</i>
Ubx^U	X ray	Bacher	<i>Ubx</i> ³⁰⁰	<i>In(3LR)62A2-3;89E1-2</i>	
Ubx^{R79}	X ray	Ramey		<i>In(3R)89E;98B-C</i>	
Ubx^X	X ray	Lewis		<i>T(1;3)20;89E</i>	
Ubx^{X-A}				<i>T(2;3)52A-C;89E</i>	
Ubx^{X-type}		Lewis			

bx^d: see **BXC**

bx^d: see *Ubx* in **BXC**

***Bxd: Beadexoid**

location: 1-45.

origin: Spontaneous.

discoverer: Goldschmidt.

references: 1945, Univ. Calif. (Berkeley) Publ. Zool. 49: 507, 520.

phenotype: Like a strong *Bx*. RK2.

Bxl: see *Ubx* in **BXC**

by: blistery

location: 3-48.7.

origin: Spontaneous.

discoverer: Glass, 33a.

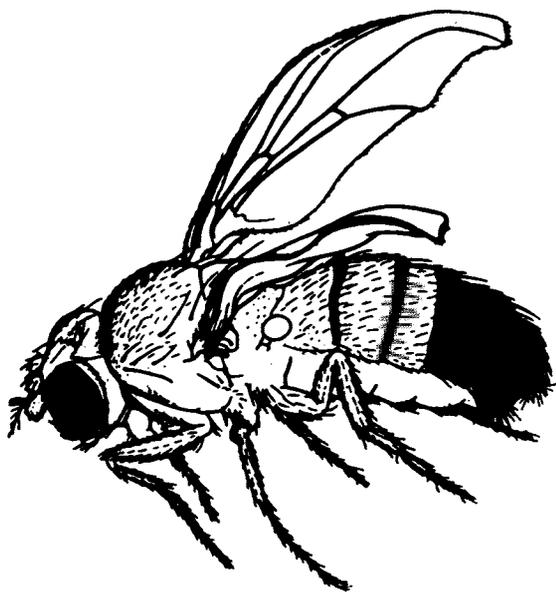
references: 1934, DIS 2: 8.

phenotype: Wings blistered in subterminal region; wing surface dusky and warped. Thorax humpy. RK1.

alleles: **by*^{46h} (CP627) like *by* but without thoracic effect.

cytology: Placed in 85D11-E3 based on its inclusion in *Df(3R)by62 = Df(3R)85D11-14;85F6* and *Df(3R)by416 = Df(3R)85D10-12;85E1-3* (Kemphues, Raff, and Kaufman, 1983, Genetics 105: 345-56).

bz: see *mal*^{bz}

**c: curved**

From Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 165.

c: curved

location: 2-75.5.

phenotype: Wings thin textured, divergent, uplifted at base, and curved downward throughout their length. RK1.

alleles:

allele	origin	discoverer	synonym	ref ^α
<i>c</i> ¹	spont	Bridges, 11124		1,3
<i>c</i> ²		Erlich, 62i	<i>c</i> ⁶²ⁱ	4
<i>c</i> ³	EMS	Davis		2
<i>c</i> ⁴	EMS	Davis		2
<i>c</i> ⁵	EMS	Davis		2
<i>c</i> ⁶	EMS	Davis		2
<i>c</i> ⁷	EMS	Davis		2

^α 1 = Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 164 (fig.); 2 = Davis and MacIntyre, 1988, Genetics 21: 755-66; 3 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 211 (fig.); 4 = Nelsen, 1967, DIS 42: 37.

cytology: Placed in 52D3-9 based on its inclusion in *Df(2R)KL9 = Df(2R)52D3;52D7-9* (Davis and MacIntyre).

C^K: see *C-K*

C-abl: see *Abl*

C-erb: see *Egfr*

***C-K: Curved of Krivshenko**

location: 2- or 3- (rearrangement).

origin: X ray induced.

discoverer: Krivshenko, 5513.

references: 1956, DIS 30: 74.

synonym: *C*^K.

phenotype: Wings are thin textured, slightly divergent, uplifted basally, and then curved downward. Homozygous lethal. RK2A.

cytology: Associated with *T(2;3)C-K = T(2;3)52;76;81;-*

86.

other information: Probably allele of *Rev*.

C-myb: see *Myb*

C-ras1: see *Ras1*

C-ras2: see *Ras2*

C-ras3: see *Ras3*

C-src1: see *Src1*

C-src2: see *Src2*

C(): Crossover suppressor

The terminology originally used for dominant suppressors of crossing over. These effects were found to be rearrangements and are so treated here. The symbol *C* in this context has been dropped except where included under synonymy.

***c(1)a: crossover suppressor for chromosome 1**

location: One factor in *X* and probably several autosomal modifiers.

origin: Spontaneous.

discoverer: Bridges, 1916.

references: Bonnier, 1923, Hereditas 4: 81-110.

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 220.

phenotype: Reduces recombination between *v* and *f* from 23 to 15% and between *w*^e and *v* from 31% to 10%. *c(1)a* was probably the cause of a secondary nondisjunction frequency of 15-30%. RK3.

other information: Validity of phenotypic description seems dubious. Probably an inversion.

C(2)R: see *In(2R)NS*

C(2;3): see *In(2L)t*

C(2L)HR: see *In(2L)t*

C(2L)T: see *In(2L)t*

c(3)G: crossover suppressor in chromosome 3 of Goben

location: 3-57.4 (1.0 to the left of *sbd*², 4.0 to the right of *cv-c*).

phenotype: Eliminates meiotic crossing over in homozygous females. First-division nondisjunction and chromosome loss frequent (30% for *X*, 2, and 3; 20% for 4); second division normal; exceptions more frequent at 25° than at 19°. Production of triploids and intersexes 300-500 times normal. Egg hatch very low. Synaptonemal complexes absent from oocytes (Meyer), and both duration of meiotic prophase and number of 16-cell cysts per germarium reduced in *c(3)G/c(3)G* females (Smith and King, 1969, Genetics 60: 335-51). Meiotic loss of ring chromosomes increased in *c(3)G* females (Sandler, 1961, Nat. Cancer Inst. Monogr. 18: 243-73). No meiotic effects in homozygous males. In *c(3)G/+* females, both intergenic recombination (Hinton, 1966, Genetics 53: 157-64) and the subset of intragenic recombination accompanied by recombination of flanking markers (Carlson, 1972, Genet. Res. 19: 129-32) increased. Somatic pairing (Semjenov, 1979, Tsitologiya 21: 1353-55) and somatic crossing over (Le Clerc, 1946, Science 103: 553-54) normal in *c(3)G* homozygotes;

however, reported to be deficient for one of two X-ray-induced reactions leading to mitotic exchange (Handle, 1974, *Molec. Gen. Genet.* 128: 233-39). Heterochromatic interchanges inducible in oocytes of homozygous females by X irradiation (Roberts, 1969, *Genetics* 47: 387-408; Würigler, Graf, Ruch, Beck, and Steiner, 1978, *Arch. Genet.* 51: 217-42). Oocytes of homozygous females show increased sensitivity to X irradiation and lack the normally observed fractionation effect (Watson, 1969, *Mutat. Res.* 8: 91-100; 1972, *Mutat. Res.* 14: 299-307); Watson (1972) also reports increased sensitivity of *c(3)G*-bearing sperm from *c(3)G/+* males to translocation induction. RK3.

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>c(3)G</i> ¹	spont	Gowen	<i>cx, c(3)G</i> ¹⁷	1, 2, 3	
<i>c(3)G</i> ⁶⁸	EMS	Sandler, 1968	<i>mei-W22</i>	3, 5	> <i>c(3)G</i> ¹
<i>c(3)G</i> ^{OR28γ}	EMS	R.F. Grell		4	< <i>c(3)G</i> ¹

^α 1 = Gowen, 1922, *Am. Naturalist* 56: 286-88; 2 = Gowen, 1933, *J. Exp. Zool.* 65: 83-106; 3 = Hall, 1972, *Genetics* 71: 367-400; 4 = McKinley, Generoso, and Grell, 1979, *Genetics* 92: s79. 5 = Sandler, 1971, *DIS* 47: 48.

^β Severity of phenotype vis-à-vis *c(3)G*.

^γ Fourteen other alleles recovered in same experiment.

cytology: Placed in region 89A2-5 on the basis of its inclusion in *Df(3R)c3G2 = Df(3R)89A2-3;89B4-5* (Hughes, Nelson, Yanuk, and Szauter).

***c(3)G*⁶⁸**

phenotype: Effects slightly more severe than in *c(3)G*. Meiotic recombination absent; first-division nondisjunction and loss high (40% for X, 2, and 3; 30% for 4). Low level of nondisjunction in second meiotic division. Oocytes of homozygous females lack synaptonemal complex (Carpenter).

***c(3)G*^{OR28}**

phenotype: Hypomorphic allele. Homozygous females show polarized reduction of recombination to 4.3% between *y* and *f* with greatest reduction distally. X-chromosome nondisjunction temperature sensitive; 16.7% at 25°; reduced to 10.2% in oocytes exposed to 17° during metaphase I and anaphase I; no X-chromosome loss at either temperature. Synaptonemal complex reduced in quantity and abnormal in structure. *c(3)G*^{OR28}/*Df(3R)sbd*¹⁰⁵ females exhibit no recombination and 32% X-chromosome nondisjunction.

C2L: see *In(2L)NS*

C3: see *In(3R)C*

C4: see *RpII*^{C4}

CIIL: see *In(2L)NS*

CIIRE: see *In(3R)C*

ca: claret

location: 3-100.7.

references: Sequeira, Nelson, and Szauter, 1989, *Genetics* 123: 511-24.

Yamamoto, Komma, Shaffer, Pirrotta, and Endow, 1989, *EMBO J.* 8: 3543-52.

phenotype: Eye color ruby. With *cn*, eye color is deep red-dish yellow; with *bw*, translucent brownish yellow (Mainx, 1938, *Z. Indukt. Abstamm. Vererbungsl.* 75: 256-76). Larval Malpighian tubes colorless (Beadle,

1937, *Genetics* 22: 587-611). Eye color autonomous when larval optic disk from *ca* is transplanted into wild type or *v*. Wild-type disk in *ca* not entirely autonomous (Beadle and Ephrussi, 1936, *Genetics* 21: 230); *ca* flies produce less *v*⁺ substance than wild type (Clancy, 1942, *Genetics* 27: 417-40). Uptake of radio-labeled kynurenine by larval Malpighian tubules greatly reduced; uptake by developing eyes nearly normal (Sullivan and Sullivan, 1975, *Biochem. Genet.* 13: 603-13). Presence of 4-O-glucosyl-N-acetyldopamine, possibly a detoxification product of dopamine, reported by Okubo (1958, *Med. J. Osaka Univ.* 9: 327-37). Slightly narrow body and pointed wing. RK1.

alleles: Some alleles are mutant for two closely spaced genes and are *ncd ca* double mutants; these alleles are designated *ca*nd in the following table.

allele	origin	discoverer	synonym	ref ^α	comments
<i>ca</i> ¹	spont	Bridges, 19112		1, 2	
<i>ca</i> ²	spont	Bridges, 32f22		2	in <i>In(3R)P</i>
<i>ca</i> ³			<i>ca</i> ^{572jlla3}		
<i>ca</i> ⁴	γ ray			8	
<i>ca</i> ⁵	γ ray			8	
<i>ca</i> ⁶	γ ray			8	
<i>ca</i> ⁷	γ ray			8	
<i>ca</i> ⁸	γ ray			8	
<i>ca</i> ⁹	γ ray			8	
<i>ca</i> ¹⁰	γ ray			8	
<i>ca</i> ¹¹	γ ray			8	
<i>ca</i> ¹²	γ ray			8	
<i>ca</i> ¹³	γ ray			8	
<i>ca</i> ¹⁴	γ ray			8	
<i>ca</i> ¹⁵	γ ray			8	
<i>ca</i> ¹⁶	γ ray			8	hypomorphic allele; Malpighian tubes pale yellow
<i>ca</i> ¹⁷	γ ray			8	
<i>ca</i> ¹⁸	γ ray			8	
<i>ca</i> ¹⁹	γ ray			8	
<i>ca</i> ²⁰	γ ray			8	
<i>ca</i> ²¹	X ray		<i>ca</i> ⁵	4	in <i>Dp(3;1)B152</i>
<i>ca</i> ²²	X ray		<i>ca</i> ¹⁵	4	in <i>Dp(3;1)B152</i>
<i>ca</i> ²³	X ray		<i>ca</i> ¹⁷	4	in <i>Dp(3;1)B152</i>
<i>ca</i> ²⁴	X ray		<i>ca</i> ^{31A}	4	in <i>Dp(3;1)B152</i>
<i>ca</i> ²⁵	X ray		<i>ca</i> ⁵⁰	4	in <i>Dp(3;1)B152</i>
<i>ca</i> ²⁶	X ray		<i>ca</i> ⁹²	4	in <i>Dp(3;1)B152</i>
<i>ca</i> ²⁷	X ray		<i>ca</i> ^{129P}	4	in <i>Dp(3;1)B152</i>
<i>ca</i> ²⁸	X ray		<i>ca</i> ^{148P}	4	in <i>Dp(3;1)B152</i>
<i>ca</i> ²⁹	X ray		<i>ca</i> ^{166P}	4	in <i>Dp(3;1)B152</i>
<i>ca</i> ³⁰	X ray		<i>ca</i> ^{R16}	4	in <i>Dp(3;1)B152</i>
<i>ca</i> ³¹	X ray		<i>ca</i> ^G	4	in <i>Dp(3;1)B152</i>
<i>ca</i> ³²	HD		<i>ca</i> ^{P1}	9	no <i>P</i> insert
<i>ca</i> ³³	HD		<i>ca</i> ^{P2}	9	no <i>P</i> insert
<i>ca</i> ³⁴	HD		<i>ca</i> ^{P6}	9	<i>P</i> insert at 99B8-10
<i>*ca</i> ³⁵	X ray	E. L. Smith, 34f	<i>bw-b</i>	2	
<i>*ca</i> ³⁶	X ray		<i>ca</i> ^G <i>bnw</i> ^G	5,6	
<i>ca</i> ^{nd1}	X ray		<i>ca</i> nd	3,7	mutant for <i>ncd</i>
<i>ca</i> ^{nd3}	γ ray		<i>ca</i> ^{nd2}	8	<i>T(2;3)44B-C;100B</i> mutant for <i>ncd</i>
<i>ca</i> ^{nd4}	γ ray		<i>ca</i> ^{nd3}	8	mutant for <i>ncd</i> ; also lethal rearranged in 99B
<i>ca</i> ^{nd5}	HD		<i>ca</i> ³⁻¹	9	mutant for <i>ncd</i> ; no <i>P</i> insert
<i>ca</i> ^{nd6}	HD		<i>ca</i> ²²⁻⁸	9	mutant for <i>ncd</i> ; no <i>P</i> insert
<i>ca</i> ^{nd7}	spont		<i>ca</i> ^{Cm}	9	mutant for <i>ncd</i>
<i>ca</i> ^{nd8}	HD		<i>ca</i> ^{P3}	9	mutant for <i>ncd</i> ; no <i>P</i> insert
<i>ca</i> ^v	X ray		<i>ca</i> ^v	2	<i>T(2;3)59D;81F;94;99C-E</i>

^α 1 = Bridges and Morgan, 1923, *Carnegie Inst. Wash. Publ. No.* 327: 219 (fig.); 2 = CP627; 3 = Davis, 1969, *Genetics* 61: 577-94; 4 = Frisardi and MacIntyre, 1984, *Mol. Gen. Genet.* 197: 403-13; 5 = Gossi and Moree, 1971, *DIS* 46: 40; 6 = Gossi, Kreisman, and Moree, *DIS* 48: 15; 7 = Lewis and Gencarella, 1952, *Genetics* 37: 600-01; 8 = Sequeira, Nelson, and Szauter, 1989, *Genetics* 123: 511-24; 9 = Yamamoto, Komma, Shaffer, Pirrotta, and Endow, 1989, *EMBO J.* 8: 3543-52.

cytology: Placed in region 99B5-9 based on its inclusion in both $Df(3R)ca46 = Df(3R)98F14;99B5-9$ and $Df(3R)L127 = Df(3R)99B5-6;99E4-F1$ (Frisardi and MacIntyre, 1984, Mol. Gen. Genet. 197: 403-13). Placed in 99B8-10 by *in situ* hybridization (Yamamoto *et al.*).

molecular biology: Region cloned in a 160-kb chromosome walk from 99C6-8 to 99B8-10 (Yamamoto *et al.*). A 7.4 kb mRNA detected on Northern blots with sequence from this region is the putative *ca* transcript; it is reduced or absent in ca^1 , ca^{34} , ca^{nd1} , and ca^{nd7} , but present in *ncd* RNA. The 7.4-kb RNA is transcribed from left to right and its 5' end is very close to that of a transcription unit on the opposite strand that generates a 2.2-kb mRNA thought to encode the *ncd* product. All five *ncd ca* chromosomes tested have the same or similar 2.6-kb deletions for the 5' region shared by the two transcription units; the *P* element insert of ca^{34} resides in this segment.

***ca^v*: claret-variegated**

origin: X ray induced.

discoverer: E. B. Lewis.

phenotype: ca^v/ca slightly variegated. Can be confused with wild type. ca^v/ca^{nd1} females produce normal progeny. Homozygous lethal. RK3A.

cytology: Associated with $T(2;3)ca^v = T(2;3)59D;81F;94;99C-E$ (Craymer, 1980, DIS 55: 199).

***cab*: cabbage (J.C. Hall)**

location: 1-(not localized).

origin: Induced by ethyl methanesulfonate.

discoverer: Sziber.

references: Aceves-Piña and Quinn, 1979, Science 206: 93-96.

phenotype: Blocked or impaired in learning, with respect to several types of conditioning tests used on groups of flies or on individuals, including those involving electric shocks (Booker and Quinn, 1981, Proc. Nat. Acad. Sci. USA 78: 3940-49) or exposure to flies (mated females, immature males) that in wild type induce poor courtship subsequently (Gailey, Jackson, and Siegel, 1982, Genetics 102: 771-82). *cab* flies may be generally debilitated, and tests involving habituation or sensitization to sugar stimuli gave unreliable results (Duerr and Quinn, 1982, Proc. Nat. Acad. Sci. USA 79: 3646-50).

***cac*: cacophony (J.C. Hall)**

location: 1-36.6.

origin: Induced by ethyl methanesulfonate.

references: Schilcher, 1976, Behav. Biol. 17: 187-96. 1977, Behav. Genet. 7: 251-59.

Hall, Siegel, Tompkins, and Kyriacou, 1980, Stadler Genet. Symp. 12: 43-82.

Kulkarni and Hall, 1987, Genetics 115: 461-75.

Wheeler, Kulkarni, Gailey, and Hall, 1989, Behav. Genet. 19: 503-28.

phenotype: Males court abnormally with poor mating success and aberrant courtship song, which includes pulses of tone that are polycyclic, rather than monocyclic or tricyclic, within wild-type pulses and have increased amplitude. Some *cac* pulses, however, are quasi-normal in their cycle numbers (Wheeler *et al.*, 1989); these, as well as many of the polycyclic pulses, have essentially normal

intra-pulse frequencies; other *cac* pulses exhibit anomalous modulations, and these show multiple peaks in the intra-pulse spectra, unlike wild type. Mating success of wingless mutant males is still worse than that of wingless wild-type males, which is correlated with genetic separability of song abnormalities from deficit in mating performance. Female courtship appears to be unaffected by *cac*; but general locomotor activity of males or females is subnormal. *cac* is recessive for generally abnormal behavior and for courtship song defects in *tra/tra* flies (Kulkarni, Hall, and Schilcher).

cytology: Placed in 11A2 based on the failure of $Df(1)HF368 = Df(1)11A2;11A9$ to complement *cac* in $Df(1)HF368/cac$; *tra/tra* flies, and the normal courtship song of *cac* males carrying $Dp(1;3)v^{+74c} = Dp(1;3)9E2;11B1-2$.

other information: $l(1)11Aa$ fails to complement both *cac* and *nbaA*; however *cac* and *nbaA* complement, and *cac* flies exhibit normal visual behavior and visual system physiology (Kulkarni and Hall, 1987). Chromosome rearrangements with breakpoints in 11A and which are lethal in combination with $l(1)11Aa$ also fail to complement *cac* and *nbaA* [$ln(1)A78$, $ln(1)A97$, $ln(1)N66$, and $Tr(1;1)A101$]. Complementation tests for *cac* carried out in diplo-X individuals transformed into phenotypic males by *tra*.

***cact*: cactus (T. Schüpbach)**

location: 2-52 (between *b* and *pr*).

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

phenotype: Maternal-effect lethal mutant; embryos from homozygous mothers appear "ventralized"; at differentiation they form only a narrow strip of dorsal cuticle, whereas ventral setal belts are expanded and encircle in irregular fashion most of the embryonic periphery. At gastrulation the germband hardly extends at all, and the posterior midgut invaginates close to the posterior pole. Cephalic furrow is more pronounced than in wildtype.

alleles:

allele	origin	synonym	comments
<i>cact</i> 1	EMS	<i>cact</i> ⁹⁹	zygotic lethal
<i>cact</i> 2	EMS	<i>cact</i> ^{D12}	zygotic lethal
<i>cact</i> 3	EMS	<i>cact</i> ^{D13}	zygotic lethal
<i>cact</i> 4	EMS	<i>cact</i> ^{G8}	zygotic lethal
<i>cact</i> 5	EMS	<i>cact</i> ^{S1}	zygotic lethal
<i>cact</i> 6	EMS	<i>cact</i> ^{U7}	zygotic lethal
<i>cact</i> 7	EMS	<i>cact</i> ^{A2}	very strong allele
<i>cact</i> 8	EMS	<i>cact</i> ^{F11}	very strong allele
<i>cact</i> 9	EMS	<i>cact</i> ^{III G}	very strong allele
<i>cact</i> 10	EMS	<i>cact</i> ^{H4}	very strong allele
<i>cact</i> 11	EMS	<i>cact</i> ^{O9}	very strong allele
<i>cact</i> 12	EMS	<i>cact</i> ^{O11}	very strong allele
<i>cact</i> 13	EMS	<i>cact</i> ^{PD}	very strong allele
<i>cact</i> 14	EMS	<i>cact</i> ^{SG}	very strong allele
<i>cact</i> 15	EMS	<i>cact</i> ^{H8}	weak allele; temperature sensitive
<i>cact</i> 16	EMS	<i>cact</i> ^{HE}	weak allele; temperature sensitive
<i>cact</i> 17	EMS	<i>cact</i> ^{Q6}	weak allele; temperature sensitive
<i>cact</i> 18	EMS	<i>cact</i> ^{P2}	weak allele; temperature sensitive
<i>cact</i> 19	P	<i>cact</i> ^{UK}	
<i>cact</i> 20	P	<i>cact</i> ^{UL}	
<i>cact</i> 21	P	<i>cact</i> ^{UW}	
<i>cact</i> 22	P	<i>cact</i> ^{VQ}	

cytology: Placed in 35E6-36A9 between the right break of $Df(2L)osp29 = Df(2L)35B1-3;35E6$ and the left break of

Df(2L)H20 = Df(2L)36A8-9;36E1-2.

cad: caudal

location: 2-{55}.

references: Mlodzik, Fjose, and Gehring, 1985, EMBO J. 4: 2961-69 (fig.).

Levine, Harding, Wedeen, Doyle, Hoey, and Radomska, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 209-33 (fig.).

Macdonald and Struhl, 1986, Nature 324: 537-456 (fig.).
Hoey, Doyle, Harding, Wedeen, and Levine, 1986, Proc. Nat. Acad. Sci. USA 83: 4809-13.

Mlodzik and Gehring, 1987, Cell 48: 465-78.

phenotype: Homozygous lethal; homozygous *cad* embryos from *cad*⁺ moths develop into nearly normal first-instar larvae, which, however, lack cuticular structures of the external terminalia, e.g., the anal tuft, parts of the anal pads and the terminal sense organs. Distribution of *cad*⁺ gene product in such embryos indistinguishable from that in their *cad*⁺ and +/+ sibs, evidently maternally derived. *cad*⁺ offspring from *cad/cad* oogenic cysts indistinguishable from those from *cad*⁺ cysts; larvae lack portions of the eighth abdominal segment and sometimes parts of the fourth and less frequently other even-numbered abdominal segments; frequently they develop into normal adults. *cad/cad* embryos from homozygous *cad* cysts display variably abnormal segmentation; head and anterior thorax normal; many posterior segments deleted with T2 and odd-numbered abdominal segments more resistant to deletion; most of eighth abdominal segment and terminalia (but not some parts of posterior spiracles) deleted; replaced by small plates of sclerotized cuticle resembling mouth hooks. Maternal germ-line transcription of the *cad*⁺ gene demonstrated by the detection of transcript in oocytes and nurse cells; transcripts uniformly distributed in early embryos; in the syncytial blastoderm transcripts disappear from the anterior end of the embryo and an anterior-posterior gradient develops with heaviest concentration posteriorly. The cellular blastoderm shows a band of hybridization three to five cells wide encircling the embryo .13 to .19 of the distance from posterior to anterior end; label is internalized during gastrulation and is seen in hind gut, midgut, and Malpighian tubules. Immunocytochemical observations detect no *cad* polypeptide until the stage 5 or 6 embryo. In the syncytial blastoderm there is dramatic accumulation of *cad* polypeptide in the same antero-posterior gradient as observed for transcript; the polypeptide localizes strongly to nuclei during interphase. The same gradient develops over time in unfertilized eggs. Subsequent collapse of the gradient into a posteriorly disposed circumferential band follows the behavior of transcript. Embryos produced by homozygous *BicD* or *bcd* females display an uniform distribution of *cad*⁺ product, which subsequently becomes restricted to symmetrically disposed anterior and posterior rings of *cad* polypeptide. During gastrulation *cad*⁺ polypeptide persists in a position suggesting a fifteenth parasegment, in the posterior midgut and Malpighian tubules, in six narrow bands at double segment intervals, in pairs of neuromeres initially in parasegments 1-14, later in thorax and anterior abdomen, and in portions of the genital disc, possibly precursors of the analia. In third instar larvae transcripts are also found in germ cells of both sexes and in the

presumptive hind gut and analia of the genital disc. The *cad* gene product can increase the level of *ftz* transcription in the posterior half of the embryo by interacting with multiple copies of a TTTATG consensus sequence located in the zebra-stripe element of the *ftz* promoter (Dearolf, Topol, and Parker, 1989, Nature 341: 340-42).

alleles: Four ethyl-methanesulfonate-induced alleles described by Macdonald and Struhl: *cad*¹, *cad*², *cad*³, and *cad*⁴.

cytology: Localized to 38E5-6 by *in situ* hybridization.

molecular biology: Locus initially identified and gene isolated on basis of homeobox homology. Genomic clones isolated and sequenced; two exons separated by an intervening sequence of 10.5 kb. The homeobox begins 14 base pairs from the beginning of the 3' exon. 2.4 kb maternal mRNA detected in 0-4 hr embryos and a 2.6 kb zygotic mRNA at cellular blastoderm. mRNA's differ in both initiation and termination sites; exons identical in the two. The polypeptide is characterized by a number of homopolymeric repeats: 10/10 His/Ala, 6/7 His, 10/12 Ser, 15/20 gly/val and 20/27 Asn in the 5' exon and 11/11 Arg in the 3' exon.

***cal: coal**

location: 3-59.5.

origin: Spontaneous.

discoverer: Grout, 47120.

references: Ives, 1948, DIS 22: 53.

phenotype: Black body color similar to *e*⁴. Viability reduced slightly. RK2.

Cal: Calmodulin

location: 2-{67}.

discoverer: Yamanaka.

references: Yamanaka, Sangstad, Hanson-Painton, McCarthy, and Tobin, 1987, Nucleic Acids Res. 15: 3335-48.

Smith, Doyle, Maune, Munjaal, and Beckingham, 1987, J. Mol. Biol. 196: 471-85.

phenotype: The structural gene of the calcium-binding protein, calmodulin (148 amino acids, 17000 daltons).

cytology: Located in region 49A by *in situ* hybridization (E.B. Lewis).

molecular biology: Genomic clone isolated from library using electric eel cDNA probe. Gene comprises four exons separated by three introns of 3400 to 4300 base pairs in length, exon 1 consists of 5' untranslated region and the initiator ATG; exon 2 encodes amino-acid residues 1 to 58.3; exon 3 residues 58.3 to 139.3 and exon 4 residues 139.3 to 148 plus the 3' untranslated region. Sequence highly conserved among eukaryotic species. Transcripts of 1.65 and 1.9 kb found at intermediate levels in embryos, high levels in larvae, and low levels in adults.

Calcium dependent protein kinase: see *Pkc*

Calmodulin: see *Cal*

calyx bulging: see *cxb*

camel: see *iab5* under *BXC*

canoe: see *cno*

canopy wing: see *cpw*

capu: cappucino (*T. Schüpbach*)

location: 2-8.

origin: Induced by ethyl methanesulfonate.

references: Manseau and Schüpbach, 1989, *Genes Dev.* 3: 1437-52.Schüpbach and Wieschaus, 1989, *Genetics* 121: 101-17.

phenotype: Maternal-effect lethal. Homozygous females lay eggs which sometimes (5-10%) have a "pointed cap" (cappucino) of dorsal appendage material sitting over the anterior end of the egg, instead of two distinct dorsal appendages. Such eggs are similar to eggs formed by the female-sterile mutation *fs(1)K10* but the extent of dorsal appendage material on *capu* eggs is much more variable than that of *fs(1)K10* eggs. No polar granules are found in such eggs. Mutant females produce embryos lacking polar granules, pole cells, and normal abdominal segmentation. In combination with *Bic-D*, however, abdominal segmentation does develop in the anterior half of the embryo. Improper localization of abdominal determinants also indicated by the lack of posterior localization of *vasa* protein. Cellularization of the blastoderm irregularly defective, with nuclei of different sizes and densities. Resemble embryos formed by other grandchildless-knirps-like mutations, such as *vasa* or *tudor*, but in addition, some of the embryos from *capu* also appear dorsitized. Mosaic studies demonstrate germ-line function of *capu*.

alleles: Four, *capu*¹ to *capu*⁴ isolated as *RK*, *G7*, *H3*, and *H8*, respectively.

car: carnation

location: 1-62.5.

origin: X ray induced.

discoverer: Patterson, 28c20.

references: 1934, *DIS* 1: 31.

phenotype: Eye color dark ruby. Body shape and proportions seem rounded. With *st*, eye color is yellow-brown; with *bw*, brownish yellow to brown (Mainx, 1938, *Z. Indukt. Abstamm. Vererbungsl.* 75: 256-76). Malpighian tubes pale yellow in mature larva (Beadle, 1937, *Genetics* 22: 587-611) but hard to distinguish from wild type before third instar. Eye color autonomous in transplant into wild-type host (Beadle and Ephrussi, 1936, *Genetics* 21: 230). *car dor* is pupal lethal and shows reduced recovery in gynandromorphs with male parts *car dor* (Nash, 1971, *DIS* 47: 73). *car lt* also lethal, dies as larva when mother *car;lt/+* or *car/+;lt* and as pupa when mother *car/+;lt/+* (Nickla, 1977, *Nature* 268: 638-39); lethal focus domineering; fate maps to ventral nervous system (Nickla, Lilly, and McCarthy, 1980, *Experientia* 36: 402-05). Brain histology abnormal [McCarthy and Nickla, 1980, *Experientia* 36: 1361-63 (fig.)].

alleles: **car*² and *car*²⁶⁻⁴⁸ (CP627).

cytology: Shown to lie in doublet 18D1-2 by deficiency analysis (J.I. Valencia).

caramel: see *cml***cardinal:** see *cd***carmine:** see *cm***carminoid:** see *cmd***carnation:** see *car***Casein kinase:** see *CklI***cast: ojos castaños**

location: 1-36.

origin: Spontaneous in dysgenic cross.

references: Valdé del Rio and Costas, 1982, *DIS* 58: 210.

phenotype: Eye color chestnut brown in both sexes. *cast*-bearing strain reverts with low frequency and generates mutations at other loci as well.

Cat: Catalase

location: 3-47.0 (assuming that strain differences in catalase level represent allelic differences at the *Cat* locus).

references: Lubinski and Bewley, 1977, *Genetics* 86: s39. 1979, *Genetics* 91: 723-42.

Bewley, Nahmias, and Cook, 1983, *Dev. Genet.* 4: 49-60.

Mackay and Bewley, 1989, *Genetics* 122: 643-52.

phenotype: The structural gene for catalase [CAT(EC 1.11.1.6)]. Two peaks of activity, the smaller in late third instar larvae just prior to puparium formation and the larger during metamorphosis; coincident with the two major peaks of ecdysone titer. High specific activity in larval Malpighian tubules, gut, and fat body; higher in adult abdomen than in thorax or head. Purification and characterization of enzyme by Nahmias and Bewley (1984, *Comp. Biochem. Physiol.* 77B: 355-64). Amorphic and hypomorphic mutants are hemizygous viable on normal medium; however those with the lowest levels of catalase activity exhibit severely reduced viability (i.e., less than 2% normal levels). All mutants show increased sensitivity to the presence of hydrogen peroxide in the medium.

alleles: No electromorphs detected among 50 inbred laboratory strains. Bewley, MacKay, and Cook (1986, *Genetics* 113: 919-38) describe two strains that differ from the majority of strains in their levels of catalase; one exhibits a reduction in activity to 0.47 X normal, with a disproportionate reduction during third instar, and the other displays a 1.5 fold increase in activity; the differences are attributable to differences in the rates of enzyme synthesis. The difference maps between *st* and *cu* at 3-47.0, which is within or near the 75D-F interval within which *Cat* resides. Thus these activity differences may be attributable to allelic variation at the *Cat* locus.

allele	origin	ref ^α	comments
<i>Cat</i> ^{+lo}	spont	1	47% wild type activity
<i>Cat</i> ^{+hi}	spont	1	150% wild type activity
<i>Cat</i> ⁿ¹	EMS	2	amorphic allele; no CRM
<i>Cat</i> ⁿ²	EMS	2	hypomorphic allele; low CRM
<i>Cat</i> ⁿ³	EMS	2	hypomorphic allele; low CRM
<i>Cat</i> ⁿ⁴	EMS	2	amorphic allele; no CRM
<i>Cat</i> ⁿ⁵	EMS	2	hypomorphic allele; low CRM
<i>Cat</i> ⁿ⁶	EMS	2	hypomorphic allele; low CRM

^α 1 = Bewley, Mackay and Cook, 1986, *Genetics* 113: 919-38. 2 = Mackay and Bewley, 1989, *Genetics* 122: 643-52.

cytology: Located in region 75D1-F1 based on specific activity of segmental hyperploid produced from *T(Y;3)L131 = T(Y;3)75D* and *T(Y;3)B132 = T(Y;3)76A*, and the segmental hypoploid produced from *T(Y;3)L131* and *T(Y;3)L14 = T(Y;2)76B*, and on *Df(3L)Cat = Df(3L)75B8;75F1*.

other information: *Cat*ⁿ⁶ complements *Cat*ⁿ, partially

complements *Cat*ⁿ² and *Cat*ⁿ³, and displays nearly wild-type activity levels in combination with *Cat*⁺. CAT is a tetrameric enzyme (Nahmias and Bewley).

Cat: see *Cha* and *spa*^{*Cat*}

Catalase: see *Cat*

cau: cauliflower

location: 1-4.2.

origin: Induced by ethyl methanesulfonate.

references: Eberl and Hilliker, 1988, Genetics 118: 109-20.

phenotype: Hemizygous lethal; midgut poorly developed; dense yolk plug; poor cuticle differentiation.

cytology: Located in 2A2-B18.

caudal: see *cad*

cauliflower: see *cau*

***cb: club**

location: 1-16.5.

origin: Spontaneous.

discoverer: Morgan, 13e.

references: Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. No. 237: 69 (fig.).

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet 2: 78 (fig.).

phenotype: Wings unexpanded in about half the flies. Sternopleural bristles absent from all flies. RK3.

cb8: see *ogre*

***Cb: Curled blistered**

location: 1-13.

origin: Spontaneous.

discoverer: Villee, 40b.

references: 1945, DIS 19: 47.

phenotype: Heterozygous or homozygous *Cb* give curled and blistered wings only in presence of homozygous *px*^{*Cb*}. RK3.

cbd: central body defect (J.C. Hall)

location: 1-41.

origin: Induced by ethyl methanesulfonate.

references: Heisenberg, Borst, Wagner, and Byers, 1985, J. Neurogenet. 2: 1-30.

phenotype: Entire central complex of brain (with exception of protocerebral bridge) dissociated into two fiber masses of variable shape; some of dorsal brain's mushroom body lobes reduced in size; inter-hemispheric commissure of brain reduced in size (in white pupae); abnormal learning in adult and larvae tests using olfactory stimuli; subnormal locomotor activity.

alleles: *cbd*¹, *cbd*², and *cbd*³ (formerly *cbd*^{KS96}, *cbd*^{KS171}, and *cbd*^{KS188}).

***cbf: clubfoot**

location: 1-45.

origin: X ray induced.

discoverer: Cantor, 46d20.

references: 1946, DIS 20: 64.

phenotype: Leg segments greatly shortened; abnormally shaped tarsi and metathoracic legs. Wings slightly warped, wide in center, and tapering at ends. All flies emerging show both wing and leg effects but expression variable. Only about 3% of *cbf* flies eclose. RK3.

other information: Not tested for allelism to *pl* (1-47.9).

Cbx: see *BXC*

cc: see *svr*

ccb: central complex broad (J.C. Hall)

location: 1-56.

origin: Induced by ethyl methanesulfonate.

references: Heisenberg, Borst, Wagner, and Byers, 1985, J. Neurogenet. 2: 1-30.

phenotype: Ellipsoid body and central body of central brain abnormally flat and broad; learning abnormal in tests using olfactory stimuli.

alleles: *ccb*¹ and *ccb*² (formerly *ccb*^{KS127} and *ccb*^{KS145}).

ccd: central complex deranged (J. Hall)

location: 1-15.

origin: Induced by ethyl methanesulfonate.

references: Heisenberg, Borst, Wagner, and Byers, 1985, J. Neurogenet. 2: 1-30.

phenotype: Ellipsoid body of central brain abnormally flat and broad; fiber number in inter-hemispheric commissure of brain reduced (in white pupae); abnormal larval and adult learning in tests using olfactory stimuli; activity in open-field locomotor test reduced.

ccw: concave wing

location: 1-23.4.

origin: Induced by L-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 68.

phenotype: Wings shorter and narrower with L3 and L4 shifted toward each other, occasionally truncated. Wing membrane depressed in center into slight concavity, giving slight scooped effect. Not easily classified. RK3.

other information: One allele induced by L-p-N,N-di-(2-chloroethyl)amino-phenylalanine.

cd: cardinal

location: 3-75.7.

origin: Spontaneous.

discoverer: Johnson, 19k24.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 217 (fig.).

phenotype: Eye color yellowish vermilion, changing toward wild type with age. Drosoperin content of eyes about 85% normal (Gearhart and MacIntyre, 1970, Anal. Biochem. 37: 21-25). Phenoxazinone synthetase activity about 39% wild type; accumulates 3-hydroxykinurenine (Phillips, Forrest, and Kulkarni, 1973, Genetics 73: 45-56; Sullivan, Grillo, and Kitos, 1974, J. Exp. Zool. 188: 225-34). Ocelli white, showing no effect of age. Accumulates a blue-fluorescent compound termed cardinalic acid (xantherenic acid 8-O-β-O-glucoside) (Ferre, Mensua, and Jacobson, 1985, DIS 61: 71). Eye color autonomous in transplant of larval optic disk into wild type, *ca*, *cn*, *st*, or *v* larval host (Beadle and Ephrussi, 1936, Genetics 21: 230). Larval Malpighian tubes bright yellow; not distinguishable from wild type. RK2.

alleles: *cd*³ [persistent allele in Amherst population (Ives, 1970, Evolution 24: 507-18)]; *cd*⁷⁹ⁱ and *cd*^{81d} isolated from a natural population by Najera (1985, DIS 61: 215) and **cd*⁶³ (CP627).

cytology: Placed in 94A-E based on segmental hypoploid

from $T(Y;3)D100 = T(Y;3)94A$ and $T(Y;3)B27 = T(Y;3)94E$ (Jones, 1971, DIS 47: 90).

cd^{wo}: cardinal-white ocelli

discoverer: Bridges, 12f21.

synonym: *wo*.

references: 1920, Biol. Bull. 38: 231-36.

Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 66.

phenotype: Ocelli colorless. Eye color wild type. Modifies w^e to a lighter and less yellow tone. cd/cd^{wo} has normal eye color but white ocelli (Jones, 1971, DIS 47: 90). RK2.

Cd: see *Cu*

Ce: Cell

origin: Spontaneous.

location: 4- (not located).

discoverer: Green.

references: 1952, DIS 26: 63.

phenotype: Ocelli reduced or absent; ocellar and scutellar bristles absent; interocellar microchaetae disrupted but frontals normal; postverticals short, thick, often with an adventitious pair between the normally placed postvertical bristles. Wing veins L3 and L4 converge, giving wing phenotype much like *fu* although wing phenotype variable. Homozygous lethal; lethality occurs during embryonic period (Hochman). Shown to be a segment polarity gene; homozygous embryos exhibit loss of the naked cuticle in the posterior half of each segment plus the anterior margin of the adjacent segment; most dorsal pattern elements also eliminated, leaving a lawn of fine hairs. Engrailed-antibody staining fails to detect a subset of CNS neurons in *Ce* homozygotes that are normally stained in wild-type embryos (Patel, Schafer, Goodman, and Holmgren, 1989, Genes Dev. 3: 890-904). No maternal requirement of Ce^+ for either oogenesis or embryonic phenotype (Orenic, Chidsey, and Holmgren, 1987, Dev. Biol. 124: 50-56). Lethal phenotype of *Ce* not complemented by *l(4)I7* or by induced revertants of the wing phenotype of ci^D (Orenic *et al.*); pupal lethal in combination with ci^D [Hochman, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 902-28]. RK3.

alleles: *l(4)I7* renamed Ce^1 : Cell recessive; homozygous and heterozygous with Ce^2 has the same phenotype as Ce^2/Ce^2 . *Scn* may also be allelic to *Ce* (Hochman).

allele	origin	discoverer	ref ^α	comments
* Ce^1		Glass, 39a28	1, 2	
Ce^2	spont	Green	1, 3	
* Ce^3	X ray	Green, 59c11	1, 4	
Ce^{r1}	X ray		1, 5, 6	no dominant phenotype
Ce^{r2}	X ray		1, 5, 6	no dominant phenotype

^α 1 = CP627; 2 = Glass, 1939, DIS 12: 47; 3 = Green, 1952, DIS 26: 63; 4 = Green, 1959, DIS 33: 94; 5 = Hochman, Goor, and Green, 1964, Genetica 35: 109-26; 6 = Orenic, Chidsey, and Holmgren, 1987, Dev. Biol. 124: 50-56.

cytology: Placed in salivary chromosome region 101E through 102B16, based on the inclusion of both Ce^1 and Ce^2 within *Df(4)M = Df(4)101E-F;102B6-17*.

other information: Based on (a) position, (b) homozygous phenotype, and (c) complementation results, *Ce* and ci^D

postulated to be part of a single complex (Orenic *et al.*).

ceb: central brain deranged (J.C. Hall)

location: 1-23.

origin: Induced by ethyl methanesulfonate.

discoverer: Heisenberg.

phenotype: Extra lobe, in adult brain, of approximately 400 coiled Kenyon-cell fibers next to calyx of mushroom bodies; olfactory learning in adults slightly reduced; memory is normal.

cytology: Located in 7F1-8A5.

Cec: Cecropin

location: 3-{101}.

references: Kylsten, Samakovlis, and Hultmark, 1990, EMBO J. 9: 217-24.

phenotype: Three structural genes for three very similar cecropin proteins (cecropin *A1*, *A2*, and *B*), bacteriotoxic proteins that are induced in adults in response to bacterial infection. All three cecropin genes are coordinately induced by injection of bacteria, although the *B* gene is expressed at a much lower level. Levels appreciable within one hour, peak at 6 hours and have generally returned to base line by 24 hours. Cecropins also induced by ingested bacteria.

cytology: Placed in 99E by *in situ* hybridization.

molecular biology: Isolated from genomic library using homologous cloned sequence from *Sarcophaga peregrina*. Two neighboring clones found to encode three functional cecropin genes and two apparent pseudogenes, the order being *A1*, $\Psi 1$, *A2*, $\Psi 2$, and *B*. Each gene is interrupted by a short intron in the coding sequence; *CecA1* and *CecA2* encode the same peptide and differ from *Sarcophaga* sarcotoxin 1A at five residues in the signal sequence; *CecB* differs from *A1* and *A2* by ten amino acid replacements, five in the signal sequence. cDNA's from all three genes recovered indicating that all are transcribed. *CecA1* and *CecA2* are transcribed off of the same strand and *CecB* is transcribed off the other.

cel: celibate (J.C. Hall)

location: 1-48.5.

origin: Induced by ethyl methanesulfonate.

discoverer: Lindsley.

references: Hall, Siegel, Tompkins, and Kyriacou, 1980, Stadler Genet. Symp. 12: 43-82.

phenotype: Males court females vigorously but rarely attempt to copulate and even less frequently achieve genital contact; females apparently unaffected by the mutation.

central body defect: see *cbd*

central brain deranged: see *ceb*

central complex broad: see *ccb*

central complex deranged: see *ccd*

***cf: cleft**

location: 1-65.6.

origin: Spontaneous.

discoverer: Bridges, 14j28.

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 55 (fig.).

phenotype: Wings smaller and somewhat spread. L3 split just beyond first crossvein; extra crossveins and branches.

Gap in L4 beyond second crossvein. Males sterile. Viability good. RK2.

cf: see *cff*

Cf: see *Px^{Cf}*

cf1: Chorion factor 1

location: 1-{0.5}.

references: Shea, King, Conboy, Mariani, and Kafatos, 1990, *Genes Dev.* 4: 1128-40.

phenotype: Encodes a protein that binds the promoter region of *Cp15*.

cytology: Placed in 2C4-8 by *in situ* hybridization.

molecular biology: Gene cloned and sequenced.

cf2

location: 2-{13}.

references: Shea, King, Conboy, Mariani, and Kafatos, 1990, *Genes Dev.* 4: 1128-40.

phenotype: Encodes a protein that binds the promoter region of *Cp15*.

cytology: Placed in 25A5-8 by *in situ* hybridization.

molecular biology: Gene cloned and sequenced.

Cf-3: see *Dl^{Cf-3}*

***cff*: control of female fertility**

location: 1-39.09 (0.09 unit to the right of *dnc*).

origin: Spontaneous.

synonym: *cf* (preoccupied).

references: Salz and Kiger, 1984, *Genetics* 108: 377-92.

phenotype: *cff* is a specific cis-acting enhancer of the female-sterile phenotype of *dnc¹*, without effect on phosphodiesterase activity. The number of progeny per female measured as 0 in *dnc¹ cff/dnc¹ cff*, 2.3 in *dnc¹ cff/dnc^{M14} cff⁺*, and 30 in *dnc¹ cff⁺/dnc^{M14} cff*.

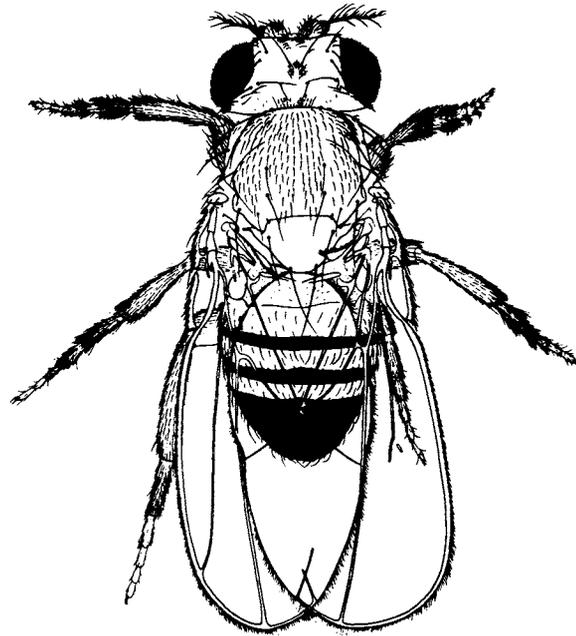
***cg*: comb gap**

location: 2-71.1.

origin: Spontaneous.

discoverer: Bridges, 25k16.

phenotype: Sex combs of male extremely large. Number of sex comb teeth increased from 10 to 18; number of bractless bristles increased; width of distal portion of basitarsus increased 1.6 fold; total number of bristles on basitarsus increased in both sexes (Datta and Mukherjee, 1968, *Proc. XII Int. Congr. Genet.* 1: 146; 1971, *Genetics* 68: 269-86). Some distortion and shortening of legs. Wings show gap in vein L4 between posterior crossvein and margin. Wings slightly curved. Effects result from a combination of overgrowth and irregular folding of imaginal rudiments during the pupal period. Strong exaggeration in compound homozygotes with genes such as *d*, *fj*, *ds*, and *ss^a*. Double heterozygote for *cg* and *ci* often shows gap in L4 (Waddington, 1952, *J. Genet.* 51: 243-58). Double heterozygote *en cg/++* has slight degree of L4 interruption and thinning at low temperature. Triple heterozygote *en cg/++; ci/+* has L4 interruption in half the flies (House, 1961, *Genetics* 46: 871). *ci^W* interacts strongly with *cg*. *cg/+; ci^W/+* resembles *ci^W/ci^W* (House, 1953, *Genetics* 38: 669-70). Sex-comb phenotype enhanced by *sn* or *Pc/Scx* (Datta and Mukherjee, 1968, *Proc. XII Int. Congr. Genet.* 1: 146). Females sterile. Oogenesis highly irregular (Beatty, 1949, *Proc. Roy. Soc. Edinburgh B* 63: 249-70). RK2.



cg: comb gap

From Bridges and Brehme, 1944, Carnegie Inst. Washington Publ. No. 552: 40.

***Cg*: Collagen**

Fifteen of thirty clones, isolated from a genomic library using a chicken-derived pro α 2(I) collagen clone, define at least ten different sequences that can be differentiated by (1) their positions in the genome, (2) their restriction maps, (3) the numbers and sizes of mRNA's with which they hybridize on Northern blots, and (4) the developmental profiles of the various mRNA's (LeParco, Cecchini, Knibiehler, and Mirre, 1986, *Biol. Cell* 56: 217-26). The genes tabulated below are differentiated according to their estimated position on the polytene map based on *in situ* hybridization. Two of these loci (*Cg19-20* and *Cg25C*) had been identified previously and are described in more detail following the table.

locus	genetic location	synonym	mRNA sizes (kb)
<i>Cg9E</i>	1-{32}		1.3, 6.3
<i>Cg10AB</i>	1-{34}		1.3, 2.3
<i>Cg16-17</i>	1-{58}		0.7, 1.6, 2.7
<i>Cg19-20</i>	1-{65}		3.8, 4.3
<i>Cg25C</i>	2-{15}	<i>DCg1</i>	6.4
<i>Cg42DE</i>	2-{57}		
<i>Cg63-64</i>	3-{15}		1.6
<i>Cg98F99A</i>	3-{101}		
<i>Cg-C^α</i>			

^α At least five separable sequences in the chromocenter; the probe that identifies these sequences does not contain rDNA sequences.

Cg19-20

location: 1-{65}.

references: Natzle, Monson, and McCarthy, 1982, *Nature* 296: 368-71.

phenotype: Encodes a type IV collagen, the major component of basement membrane. Transcripts accumulate during ecdysis in wandering hematocytes, indicating that hematocytes contribute to extra cellular matrix deposition

(Knibiehler, Mirre, Cecchini, and LeParco, 1987, Dev. Biol. 196: 243-47).

cytology: *In situ* hybridization ³HcRNA from pDCg2 to polytene chromosomes identifies complementary sequences in region 19E-20B.

molecular biology: Identified by means of a genomic clone (pDCg2) selected from a *Drosophila melanogaster* λ library using a cDNA probe for chicken pro α 2(I) collagen. Developmental profile of RNA hybridizing to pDCg2 shows peak during larval stage; little hybridization at other stages.

Cg25C

location: 2-{15}.

synonym: *DCgI*.

references: Natzle, Monson, and McCarthy, 1982, Nature 296: 368-71.

Monson, Natzle, Friedman, and McCarthy, 1982, Proc. Nat. Acad. Sci. USA 79: 1761-65.

LeParco, Knibiehler, Cecchini, and Mirre, 1986, Exp. Cell. Res. 163: 405-12 (Fig.).

phenotype: Structural gene for a type IV basement-membrane collagen. Expression first detected following germ-band shortening in cells of both somatic and visceral mesodermal origin, specifically in mesoblasts (hemocytes) and fat-body cells (Mirre, Cecchini, LeParco, and Knibiehler, 1988, Development 102: 369-76). Translation takes place in embryonic mesoblasts and larval fat-body cells, and is deposited extracellularly in basement membranes surrounding skeletal and visceral muscles (LeParco, Bevic, Knibiehler, Mirre, and Cecchini, 1989, Insect Biochem. 19: 789-802).

cytology: Placed in 25C by *in situ* hybridization.

molecular biology: Identified by means of a genomic clone selected from a *Drosophila melanogaster* λ library using a cDNA probe for chicken pro α 2(I) collagen. *Drosophila* probe hybridizes to a 6.4 kb mRNA which shows peak during larval stages preceded by a small peak in late embryonic stages; cDNA and genomic sequence studies (Cecchini, Knibiehler, Mirre, and LeParco, 1987, Eur. J. Biochem. 165: 587-93; Blumberg, MacKrell, Olson, Kurkinen, Monson, Natzle, and Fessler, 1987, J. Biol. Chem. 262: 5947-50; Blumberg, MacKrell, and Fessler, 1988, J. Biol. Chem. 263: 18328-37) reveal that the gene comprises nine relatively large exons interrupted by eight relatively small introns (unlike mammalian homologues, which have numerous small exons separated by large introns). The complete cDNA sequence encodes an 1775 amino-acid protein including a signal peptide. Both N-terminal and C-terminal nonhelical domains involved in intermolecular junctional complexes share homology with mammalian type IV collagens, whereas the triple helical region shows little homology. The triple helical region comprises repeating sequences of Gly X Y tripeptides typical of such regions; similarly to other type IV collagens, the Y residues of the Gly X Y tripeptides show preference for lysines and prolines; furthermore, the positions of 11/22 interruptions of the tripeptide-repeat sequences in the *Drosophila* protein correspond nearly exactly to those of such nonhelical segments of the mammalian triple-helical domain, although there is no conservation of length or sequence of such interruptions. The C-terminal nonhelical domain displays 58% amino-acid identity and 76% similarity to its counterparts in human

and mouse type IV collagen. In all three sequences this region is composed of two homologous halves, A and B; all six segments each contain six completely conserved cysteins. Intraspecific homology between the A and B segments is considerably less than interspecific homology between A segments or B segments, indicating an ancient origin of the duplication.

cgd: congested

location: 1-40 (37.6 and 41.7 in two determinations).

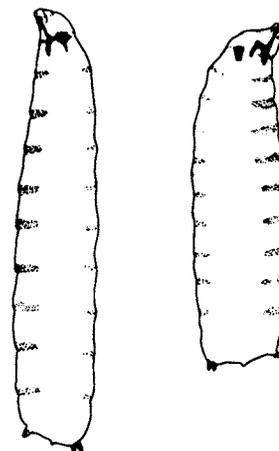
origin: Induced by ethyl methanesulfonate.

references: Eberl and Hilliker, 1988, Genetics 118: 109-20 (fig.).

phenotype: Hemizygous lethal; first instar larvae unable to escape egg shell. Gut often poorly differentiated; cuticle phenotype variable - some normal, some with abnormal (e.g. fused) segments. Frequent holes in cuticle or absence of regions of cuticle.

alleles: Two.

cytology: Placed in 11B12-12C7.



ch: chubby

Left: wild-type larva. Right: *chubby* larva. From Dobzhansky and Duncan, 1933, Wilhelm Roux' Arch. Entwicklungsmech. Organ. 130: 109-30.

ch: chubby

location: 2-73.8 (based on mapping of *ch*^{76F}; Rizki and Rizki, 1981, J. Hered. 72: 78-81).

origin: Spontaneous.

discoverer: Bridges, 17j26.

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 222.

phenotype: Adults, pupae, and larvae thickset and short. Difficult to distinguish from wild type. Chubby larvae shorter than wild type at hatching [Dobzhansky and Duncan, 1933, Wilhelm Roux's Arch. Entwicklungsmech. Organ. 130: 109-30 (fig.)]. RK3.

alleles: *ch*^{76F}, spontaneous (Rizki and Rizki, 1981).

Ch^V: see *Kg*^V

**ch-b: chilblained-b*

location: 1-23.8.

discoverer: Moriwaki, 39e22.

references: 1939, DIS 12: 50.

phenotype: Tarsi conglutinated. RK3.

cha: chaff (*T. Schüpbach*)

location: 2-84.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1989, *Genetics* 121: 101-17.

phenotype: Maternal-effect lethal mutant; embryos from homozygous mothers form a fragmented cuticle with variable holes and head defects.

alleles: *cha*^{HB} = *cha*¹.

Cha: Choline acetyltransferase (*J.C. Hall*)

location: 3-64.6.

synonym: *Cat*.

references: Hall, Greenspan, and Kankel, 1979, *Soc. Neurosci. Symp.* 4: 1-42.

Greenspan, 1980, *J. Comp. Phys.* 137: 83-92.

phenotype: Probable structural gene for choline acetyltransferase [ChAT, acetyl CoA-choline-O-acetyltransferase (EC 2.3.1.6)], which has been purified and whose molecular weight approximates 67 kilodaltons (Slemmon, Salvaterra, Crawford and Roberts, 1982, *J. Biol. Chem.* 257: 3847-52); monoclonal antibodies prepared and inhibit enzyme (Crawford, Slemmon, and Salvaterra, 1982, *J. Biol. Chem.* 257: 3853-56); homozygotes and hemizygotes for either of the original two non-conditionally mutant alleles, *Cha*¹¹ or *Cha*¹², show no detectable enzymatic activity as late embryos, which is when the mutants die; other non-conditional lethals at the locus not tested for lethal stage or ChAT activity; either of two temperature sensitive alleles, *Cha*^{ts1} or *Cha*^{ts2}, causes a variety of phenotypic defects when hemizygous or homozygous: reduced viability at 25° and death at 29° (*Cha*^{ts1}), reduced viability at 18° and death at 22° or above (*Cha*^{ts2}) plus gradual but reversible decline of enzymatic activity and of acetylcholine levels following transfer of low-temperature reared *Cha*^{ts} adults to 29-30° or above (Greenspan, 1980; Salvaterra and McCaman, 1985, *J. Neurosci.* 5: 903-10); correlated with decrements in these biochemical parameters (reversible on lowering temperature), are heat-induced abnormalities of general mobility, male courtship ability, plus electroretinogram (Greenspan, 1980), landing response (Gorczyca and Hall, 1985, *Neurosci. Abstr.* 11: 512), and of several elements of physiological responses made by thoracic indirect flight muscles following stimulation of giant fiber pathway, implying that certain interneuronal synapses in this pathway are cholinergic (Gorczyca and Hall, 1984, *J. Neurogenet.* 1: 289-313); whereas wild type exhibits two molecular forms of ChAT activity after isoelectric focusing, homogenates of *Cha*^{ts1} lead to a single form, and of *Cha*^{ts2} to two forms shifted to higher-than-normal pI (Salvaterra and McCaman, 1985); these two conditional *Cha* mutations also cause ChAT activity to have accentuated thermolability in vitro (Greenspan, 1980; Salvaterra and McCaman, 1985). Immunohistochemical staining of ChAT reveals wide distribution in CNS; this staining is strong in *Cha*^{ts1} at permissive temperature but diminishes after *in-vivo* heat treatment (Gorczyca and Hall, 1987, *J. Neurosci.* 7: 1361-69); *Cha*^{ts2} staining is poor even at permissive temperature and diminishes after heating the flies (Gorczyca and Hall, 1987). *In situ* hybridization to tissue sections detected transcriptional activity in most neuronal elements of the

cell-rich areas of the cortical regions of the cerebrum and optic lobes; however, some cells in the lamina including the amacrine neurons showed no label. Highest expression is seen in laminar monopolar-cell region, and the cerebral cortical rind, and to a lesser degree, over cortical cells of medulla-lobula, the antennal sensory neurons, and the reticular-cell layer of the compound eye (Barber, Sugihara, Lee, Vaughn, and Salvaterra, 1989, *J. Comp. Neurol.* 280: 533-43). Immunolocalization of ChAT in the adult cephalic ganglion reveals weak staining in the lamina corresponding to the synaptic layer of photoreceptor cells 1-6 of the ommatidia, three layers of strong reaction corresponding to the synaptic layers in the medulla, and labeling of four layers in the lobula (Ikeda and Salvaterra, 1989, *J. Comp. Neurol.* 280: 283-90). In *Cha*^{ts1} staining seen at 19° but disappears by 120 hours after shift to 30°; *Cha*^{ts2} shows reduced staining at 19° and none after 80 hours at 30°.

alleles:

allele	origin	synonym	ref ^α	cytology
<i>Cha</i> ¹¹	EMS	<i>Cha</i> ¹⁹	1, 2	+ β
<i>Cha</i> ¹²	EMS	<i>Cha</i> ¹²⁸	1, 2	
<i>Cha</i> ¹³	X ray		3	<i>T(H;3)91C</i>
<i>Cha</i> ¹⁴	γ ray		3	<i>In(3R)91C;93B-D</i>
<i>Cha</i> ¹⁵	X ray		3	+
<i>Cha</i> ¹⁶	γ ray		3	+ γ
<i>Cha</i> ¹⁷	γ ray		3	<i>Tp(2;3)41,91C</i>
<i>Cha</i> ¹⁸	γ ray		3	+
<i>Cha</i> ¹⁹	EMS		3	
<i>Cha</i> ¹¹⁰	EMS		3	
<i>Cha</i> ¹¹¹	EMS		3	
<i>Cha</i> ¹¹²	EMS		3	
<i>Cha</i> ¹¹³	EMS		3	
<i>Cha</i> ¹¹⁴	EMS		3	
<i>Cha</i> ^{ts1}	EMS	<i>Cha</i> ¹¹⁶	1, 2	
<i>Cha</i> ^{ts2}	EMS	<i>Cha</i> ¹⁴³	1, 2	

^α 1 = Hall, Greenspan, and Kankel, 1979, *Soc. Neurosci. Symp.* 4: 1-42; 2 = Greenspan, 1980, *J. Comp. Physiol.* 137: 83-92; 3 = Myers and Gelbart.

^β Fails to complement *l(3)91Ca*, *l(3)91Cb*, *Cha*, and *l(3)91Cd*.

^γ Fails to complement both *Cha* and *l(3)91Cb*.

cytology: Placed in 91C7-D2 based on common breakpoints of *Cha*¹ rearrangements and on its absence from *Df(3R)Cha9 = Df(3R)91C7;92A8-9* and from *Df(3R)Cha12 = Df(3R)91A;91D1-2*.

molecular biology: 3' end of coding sequence recovered as a cDNA clone prepared from mRNA isolated from adult heads; sequence determined (Itoh, Slemmon, Hawke, Williamson, Morita, Itakura, Roberts, Shively, Crawford, and Salvaterra, 1986, *Proc. Nat. Acad. Sci. USA* 83: 4081-85). The *Drosophila* enzyme produced from complete *Cha* cDNA expressed in *Xenopus* oocytes, leads to active Chat (McCaman, Carbini, Maines, and Salvaterra, 1988, *Mol. Brain Res.* 3: 107-14).

chaeta: see *cht*

chaetelle: see *chl*

chaff: see *cha*

chal: chalice (*T. Schüpbach*)

location: 2-37.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus.

phenotype: Female sterile; homozygous females show abnormalities in late oogenesis. Follicle cells do not

migrate centripetally between nurse cells and oocyte. They do synthesize a chorion which remains open ended (like a chalice, or cup). Such eggs are usually not laid and remain unfertilized.

alleles: *chal*¹ to *chal*³ isolated as *WP*, *HC*, and *QC*, respectively. *chal*² produces eggs of normal morphology which remains unfertilized.

chaoptic: see *chp*

***che: cherub**

location: 2-62.0.

origin: Ultraviolet induced.

discoverer: Meyer, 48g.

references: Meyer and Edmondson, 1951, DIS 25: 71.

phenotype: Wings short, papery, and downcurved with short, broad alulae. Males sterile. Homozygotes short lived; balanced stock *cn che bw spl In(2L+2R)Cy, al² Cy cn² L⁴ sp²* has a generation time 30% longer than normal. RK3.

alleles: **che*² and **che*³ in CP627.

chic: chickadee (T. Schüpbach)

location: 2-23.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus.

phenotype: Female sterile; nurse cell contents are not transported into the egg, and homozygous females lay tiny eggs, which remain unfertilized.

alleles: *chic*¹ to *chic*³ recovered as *WC*, *WF*, and *WK*.

chif: chiffon (T. Schüpbach)

location: 2-53 (between *b* and *pr*).

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus.

phenotype: Female sterile, homozygous females lay eggs which have thin, fragile chorions and remain unfertilized.

alleles: *chif*¹ to *chif*⁴, recovered as *WD*, *QV*, *QW*, and *WF* respectively.

cytology: Placed in 35E6-A9 between the right break of *Df(2L)osp29 = Df(2L)35B1-3;35E6* and the left break of *Df(2L)H20 = Df(2L)36A8-9;36E1-2*.

chilblained-b: see *ch-b*

chl: chaetelle

location: 2-60.8.

discoverer: Bridges, 33a4.

references: Beatty, 1949, Proc. Roy. Soc. Edinburgh B 63: 249-70.

phenotype: Bristles very small. Wing venation slightly plexuslike, exaggerates *px* when combined with it. Body size small. Rotated genitalia in many males. Blunt-tipped abdomen. Females infertile, but ovary and oocytes appear normal. RK2.

chlorotic: see *svr*

cho: chocolate

location: 1-5.5 [right of *ec*; (Lefevre, 1970, DIS 45: 40)].

origin: X ray induced.

discoverer: Weigle, 1955.

references: Sturtevant, 1955, DIS 29: 75.

phenotype: Eye color brown with whitish highlights. Paler than *se*, less purplish than *pn*. In *cho*² 2-amino-4-hydroxypterine absent; isoxanthopterin II present in excess (McKay, 1972, DIS 48: 62). Malpighian tubes of

larvae and adults contain brown pigment like *red*. Larvae easily distinguished from wild type. Malpighian tube color autonomous in mosaic larvae [Falk, Orevi, and Menzel, 1973, Nature (London) New Biol. 246: 19-20]. Brown pigment of Malpighian tubes absent when *cho* is combined with *v*, *cn*, or *st*, mutations which prevent formation of brown eye pigment. Eye color of *cho v* is yellowish, but *cho g* cannot be distinguished from *g*. Separability, viability, and fertility excellent. RK1.

alleles: *cho*² (CP627).

cytology: Placed in 3F on the basis of being included in *Df(1)GA102 = Df(1)3D5;3F7-8*, but not *Df(1)w64d = Df(1)3C2;3E8*.

Choline acetyltransferase: see *Cha*

Chorion factor: see *Cf*

Chorion protein: see *Cp*

chp: chaoptic

location: 3-{103}.

references: Fujita, Zipursky, Benzer, Ferrus, and Shotwell, 1982, Proc. Nat. Acad. Sci. USA 79: 728-33.

Zipursky, Venkatesh, Teplow, and Benzer, 1984, Cell 36: 15-26.

Zipursky, Venkatesh, and Benzer, 1985, Proc. Nat. Acad. Sci. USA 82: 1855-59.

VanVactor, Krantz, Reinke, and Zipursky, 1988, Cell 52: 281-90.

Reinke, Krantz, Yen, and Zipursky, 1988, Cell 52: 291-301.

phenotype: The structural gene encoding chaoptin, a 160 kd glycoprotein localized to the extracellular membrane surface of photoreceptor cells and their axonal projections to the optic ganglia. Protein first identified by a monoclonal antibody (MAb24B10). Antigen first appears behind the morphogenetic furrow during differentiation of the eye imaginal disk. Defective alleles, *chp*¹ and *chp*², recognized by reduced levels of antigen. Rhabdomeres of mutant photoreceptor cells highly deranged or nearly absent in homozygotes and hemizygotes for *chp*¹ or *chp*², respectively; also close apposition of adjacent retinular cells partially disrupted. Gene product postulated to play role in cell surface interactions necessary for correct alignment of the microvilli that form the rhabdomeres.

cytology: Located in 100B6-9 by *in situ* hybridization, uncovered by *Df(3R)til-e = Df(3R)10A5;100C1* but not by *Df(3R)ca48 = Df(3R)98F14;100B7-8*.

molecular biology: Genomic clone isolated using synthetic oligonucleotide probe; clone hybridizes to a 4.3 kb polyadenylated transcript in head-derived RNA, which is absent from body RNA. Same sequence isolated by Levy and Manning (1982, Dev. Biol. 94: 465-76) using poly-A+ head RNA. Both cDNA and genomic sequences determined; eleven introns, eight of which are in the translated region, varying from 509 (intron 1) to 49 (intron 6) base pairs. Two CATT boxes but no TATA box found 5' to the transcription initiation site. Open reading frame with conceptual amino sequence of 1134 amino acids; first 29 amino acids have features of signal sequences; otherwise there is no membrane spanning domain, although chaoptin shown to be an integral membrane protein. Mature polypeptide of 127 kd with fourteen potential N-linked glycosylation sites. 90% of the

sequence comprises 41 tandemly arranged repeats of average residue number 24, with periodically disposed hydrophobic amino acids separated by hydrophilic ones; repeats occur in three large blocks separated by short unique regions; homologous to repeats occurring in three other known polypeptides including the Toll gene product in *Drosophila*.

***chr: chromosome**

location: 1- (not located).
origin: Spontaneous.
discoverer: Bridges, 13115.
references: Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. No. 237: 74.
phenotype: Body color brownish yellow or tan. Abdominal bands clear yellow. RK3.

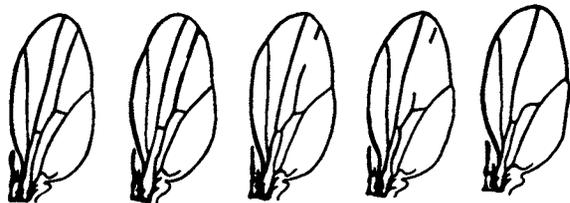
cht: chaeta

location: 2-65.6 (located in relation to *po*, *sca*, and *vg*).
origin: Isolated from a line selected for high scutellar-bristle number.
references: MacBean, McKenzie, and Parsons, 1971, Theor. Appl. Genet. 41: 227-35.
phenotype: *cht/cht* averages 1.64 scutellar bristles more than *cht/+* (6.04 vs. 4.40).

chubby: see ch

chy: chunky

location: 2- (between 8 and 28).
origin: Spontaneous.
discoverer: Bridges, 38b10.
phenotype: Body short and heavy set. Wings shorter than wild type. Difficult to classify. RK3.



ci: cubitus interruptus

Wings showing from no interruption (extreme left) to complete absence (extreme right) of the cubital vein. From Stern and Kodani, 1955, Genetics 40: 343-73.

ci: cubitus interruptus

location: 4-0 [most proximal mutant in 4 (Sturtevant, 1951, Proc. Nat. Acad. Sci. USA 37: 405-7)].
phenotype: Vein L4 shows one or more gaps both distal and proximal to posterior crossvein, generally nonterminal. Anterior crossvein shortened or absent. Other gaps and scattered branch veins in region of crossveins. At 19°, nearly all flies have a mutant phenotype; at 25°, there is slight overlap with wild type; at 30°, virtually all flies are wild type. Dosage effect such that *ci*0 haplo-4's are more extreme than *ci/ci* diplo-4's, which are more extreme than *ci/ci/ci* triplo-4's. Suppressed by *su(Hw)*² (Kotarski). For interactions of *ci* with *en*, *H*, *ve*, and *cg*, see House (1953, Genetics 38: 199-214, 309-27; 1955, Anat. Record 122: 471; 1959, Genetics, 44: 516; 1961, Genetics, 46: 871). Expression of *ci* sensitive to genetic background; selection possible for more and less extreme phenotypes (House and Yeatts, 1962, Genetics

47: 960); contribution of chromosome 2 more important than that of 3 (House and Pernaveau, 1971, Genetics 68: s29). Phenotypic effect visible in prepupa by absence of the longer longitudinal vein. RK1 at 19° and higher rank with higher temperatures.

alleles: Two types of alleles: mutant alleles tabulated below and described in more detail following the table; wild-type isoalleles described separately.

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>ci</i> ¹	spont	Terentieva, 30b		1, 5, 6	gypsy insert
<i>ci</i> ³⁶	spont	Curry, 36l		2	4.0 kb insert
<i>ci</i> ⁵⁷	X ray	Hochman, 57g		3	800 bp deletion
<i>ci</i> ^D	X ray	Ruch, 32a18		1	molecular rearrangement
<i>ci</i> ^{D-G}		Gloor		4	
<i>ci</i> ^W	spont	Wallace, 36d20	<i>It: Interrupted</i>	2	4.0 kb insert ^γ
^α					
1 = Bridges, 1935, Biol. Zh. (Moscow), 4: 401-20; 2 = CP627; 3 = Hochman, 1971, Genetics 67: 235-52; 4 = Scharloo, 1963, DIS 38: 32; 5 = Terentieva, 1931, Eksperim. Biol. 7: 187-90 (fig.); 6 = Tiniakov and Terentieva, 1933, Genetics 18: 117-20 (fig.).					
^β					
Molecular results from Kotarski.					
^γ					
Different from the insert in <i>ci</i> ³⁶ .					

cytology: Placed in salivary chromosome region 101F2-102A5, on the basis of its inclusion in *Df(4)M*^{63a} = *Df(4)101F2-102A1;102A2-5*.

molecular biology: Region cloned and restriction mapped by Locke and Kotarski; all molecular lesions indicated in the table occur in the same 5.8 kb *Bgl*I fragment. This fragment hybridizes to a 2.0 kb RNA that shows peak concentrations in late pupae.

other information: The expression of *ci*⁺ can be altered in direction of *ci* by certain chromosome rearrangements that have one break in vicinity of *ci* locus. Rearranged fourth chromosomes carrying a mutant allele of *ci*, *R(ci)*, may also show altered expression of gene [Stern and Kodani, 1955, Genetics 40: 343-73 (fig.)]. *R(ci)* and *R(ci*⁺) terminology not retained here; interaction with *ci* included in descriptions of aberrations involving chromosome 4.

***ci⁺²: cubitus interruptus-wild-type isoallele**

origin: On fourth chromosome carrying *ey*².
references: Stern and Schaeffer, 1943, Proc. Nat. Acad. Sci. USA 29: 361-67.

phenotype: Homozygote wild type at 14° and 26°. *ci*⁺²/*Df(4)M* wild type at 26°; shows some thinning and interruption of L4 at 14°. *ci*⁺²/*ci* wild type at 26°; at 14°, fewer flies show thinning or interruption of L4 than *ci*^{+C}/*ci*. *ci*⁺²/*ci*^W shows significantly greater amount of thinning and interruption of L4 than *ci*^{+C}/*ci*^W. RK3.

alleles: *ci* isoalleles superscripted *+2, +3, and *+C described by Stern and Schaeffer (1943), +5 by Hochman (1961, Evolution 15: 239-46). Relative strengths are +C > +5 > +3 > +2.

ci^D: cubitus interruptus-Dominant

phenotype: Wings show interruptions of L4 in two places: proximal to and distal to anterior crossvein. L5 also shows distal interruption. L3 and L5 thick. Considerable plexus effect and knotting of veins. Wings broader, warped or concave upward, regularly extended, and bent backward. Alula fused with and in same plane as blade of wing. Black dried haemolymph from axillary spiracle. Slight scalloping of inner wing margin, with

hairs and tufts. Direction and extent of temperature effects depends on genetic background (Scharloo). In general, no overlapping of wild type. Inviabile in combination with *Ax/Ax* or *Ax/Y* (House and Lutes, 1975, Genetics 80: 542-43). *H/+* inhibits scalloping of *ci^D* but greatly enhances L4 interruption (House, 1959, Genetics 44: 516). Fully dominant in triplo-4's (Sturtevant, 1936, Genetics 21: 448). Two doses of *ci^D* reduce survival of triplo-4 flies (Parker, 1969, Mutat. Res. 7: 393-407). Homozygotes lethal in embryo (Hochman, 1971, Genetics 67: 235-52). Embryonic segment polarity disrupted; anterior portions of segments with their denticle belts duplicated in mirror-image fashion; posterior portions missing; each segment almost entirely covered with denticles (Nüsslein-Volhard and Wieschaus, 1980, Nature 287: 795-801). Fine hairs eliminated from dorsal abdominal segments; replaced with clear cuticle and socketed denticles (Orenic, Chidsey, and Holmgren, 1987, Dev. Biol. 124: 50-56). Embryonic CNS relatively normal (Patel, Schafer, Goodman, and Holmgren, 1989, Genes Dev. 3: 890-904). Genotype of oocyte with respect to *ci^D* without effect on phenotype of progeny (Orenic *et al.*). *ci^D/l(4)102ABc* dies as embryo, *ci^D/l(4)102ABb* as embryo or larva; in *ci^D/Ce²* death usually delayed until pupal stage (Hochman, 1971). Survival of *ci/ci^D/Df(4)M101-63a* argues for nonallelism or pseudoallelism of *ci* and *ci^D* (Hochman, 1971). RK1.

alleles: *ci^{D-G}*, a less severe derivative of *ci^D*, recovered as a recombinant between *ci^D* and *spa^{pol}* (CP627). *ci^{D-l}* is a revertant of the *ci* phenotype in combination with either \pm or *ci*, which has retained the lethal phenotype (Puro). Ten similar revertants induced by γ rays isolated by Orenic *et al.*; three were cytologically normal and seven were translocations with breakpoints in or near the chromocenter. *ci^{Drv}* [*ci^{D-l}* of Haavisto (see Puro, 1982, DIS 58: 205)] reverted for *ci* phenotype but not for recessive lethality; complements *ci*.

cytology: Salivary study by Bridges revealed no chromosomal aberration.

other information: Not allelic with *ci*, at least with respect to its lethality, since *ci^D/Df(4)M101-63a* survives, whereas *ci/Df(4)M101-63a* is mutant (Hochman, 1965, DIS 40: 60). Based on location, phenotype, and complementation, *ci^D* and *Ce* postulated to be part of a single complex (Orenic *et al.*).

ci^W: cubitus interruptus of Wallace

phenotype: Homozygote is extreme *ci* type. Wings sometimes almost twice normal width, arlike, and virtually lack veins. Often present is a well-organized pattern of venation in which the posterior crossvein flows smoothly into L5. Legs lumpy, sex combs larger than normal, antennae enlarged, eyes smaller, and extra bristles present. Heterozygote shows gap in L4 in 80% of flies. *ci^W* enhanced by *H*, *en*, and *Cy* (House, 1953, Genetics 38: 669-70; 1959, Genetics 44: 516) and by *Tp(4;Y)* (Benner, 1972, Genetics 71: s4). Temperature effect described by House (1955, Genetics 40: 576). RK2.

cin: cinnamon

location: 1-0 (1.7×10^{-3} unit to the left of *y*; Padilla and Nash, 1977, Mol. Gen. Genet. 155: 171-77).

references: Baker, 1973, Dev. Biol. 33: 429-40.

phenotype: *cin/Y* and *cin/cin* embryos of *cin/cin* mothers almost completely lethal; rare surviving adults have

reddish-brown eyes; both survival and normal eye pigmentation affected by the presence of *cin⁺* in either the mother or the zygote; however, *cin* progeny of *cin⁺* mothers exhibit mutant phenotype when raised on 0.02% allopurinol (Padilla and Nash, 1977, Mol. Gen. Genet. 155: 171-77). Survival of the double mutant *cin³ v* is also sensitive to allopurinol (Bentley and Williamson, 1982, DIS 58: 23). Mosaic and transplantation analysis (Nissani and Fellingner, 1978, Dev. Biol. 65: 117-27) indicate that the maternal effect of *cin⁺* on eye color requires the presence of *cin⁺* in the oocyte, whereas *cin⁺* in ovarian mesoderm contributes to the maternal effect on viability. Affected progeny lack activity for four different molybdenum hydroxylases—all known to require a molybdenum-containing cofactor for catalytic function. These are aldehyde oxidase (Bentley and Williamson, 1982, Can. J. Genet. Cytol. 24: 1-9), pyridoxal oxidase, xanthine dehydrogenase (Browder and Williamson, 1976, Dev. Biol. 53: 241-49), and sulfite oxidase (Bogaart and Bernini, 1981, Biochem. Genet. 19: 929-46). Inactivity of xanthine dehydrogenase accounts for the eye color phenotype. Level of low-molecular-weight molybdenum cofactor severely reduced. *cin* progeny of *cin* mothers CRM negative for pyridoxal oxidase (Warner, Watts, and Finnerty, 1980, Mol. Gen. Genet. 180: 449-53). Aldehyde oxidase CRM found in hemolymph of *cin¹* (100%) and *cin⁹* (60%) larvae and in extracts of *cin⁹* (32%) adults. Both alleles show about 70% normal quantities of xanthine dehydrogenase CRM (Browder, Wilkes, and Tucker, 1982, Biochem. Genet. 20: 111-24). Eye color expression nonautonomous in gynandromorphs (Padilla and Nash, 1977).

alleles: All alleles induced by ethyl methanesulfonate except the spontaneous *cin²* and perhaps *cin^B*.

allele	ref ^{α}	phenotype
<i>cin 1</i>	1	
<i>cin 2</i>	3	temperature sensitive
<i>cin 3</i>	6	
<i>cin 3N</i>	7	female sterile
<i>cin 4</i>	2	
<i>cin 4N</i>	7	β
<i>cin 5</i>	2	
<i>cin 6</i>	2	
<i>cin 7</i>	2	
<i>cin 8</i>	2	
<i>cin 9</i>	2	β
<i>cin 10</i>	2	γ
<i>cin 11</i>	2	γ
<i>cin 12</i>	2	
<i>cin 13</i>	2	
<i>cin 14</i>	2	
<i>cin 15</i>	2	β
<i>cin B</i>	4	
<i>cin lkδ</i>	5	

α 1 = Baker, 1973, Dev. Biol. 33: 429-40; 2 = Bentley and Williamson, 1979, Can. J. Genet. Cytol. 21: 457-71; 3 = Courtright, 1976, Genetics 83: s17; 4 = Courtright, unpublished; 5 = Hickey and Singh, 1982, DIS 58: 74-76; 6 = Mohler, 1977, Genetics 85: 259-72; 7 = Padilla and Nash, 1977, Mol. Gen. Genet. 155: 171-77.

β Males carrying this allele produce *cin⁺* sons in crosses to *C(1)RM*, *cin y* females.

γ Males carrying this allele produce *cin* sons in crosses to *C(1)RM*, *cin y* females. Males carrying other alleles produce no sons from such crosses.

δ *cin lk* considered to be a hypomorphic or leaky allele of *cin*; it produces low levels of aldehyde oxidase and xanthine dehydrogenase activity and is genetically very close to yellow; however, *cin¹/cin lk* are wildtype both with respect to eye color and enzyme

activity and may not therefore be an allele.

cytology: Duplication analysis by Padilla and Nash place *cin* distal to *arth* and proximal to *l(1)J1*.

other information: Four complementation groups described by Baker; a fifth observed by Warner.

cinnabar: see *cn*

cinnamon: see *cin*

ck: crinkled (M. Ashburner)

location: 2-51.0 (estimated).

discoverer: Bridges, 30c30.

synonym: *l(2)br27*, *l(2)35Ca*, D group of O'Donnell *et al.* (1977).

references: O'Donnell, Mandel, Kraus, and Sofer, 1977, *Genetics* 86: 553-66.

Ashburner, Tsubota, and Woodruff, 1982, *Genetics* 102: 401-20.

Gubb, Shelton, Roote, McGill, and Ashburner, 1984, *Chromosoma* 91: 54-64 (fig.).

phenotype: Recessive lethal or semilethal. *ck* embryos exhibit denticles thickset and forked; hairs basally fused; sensory hairs blunt [Nüsslein-Volhard, Wieschaus, and Klüding, (fig.)]. Hemizygous survivors, or survivors among heteroallelic combinations that partially complement for viability, have stubbly chaetae, multiple trichomes, and feathery aristae (Gubb *et al.*). Variable expression of a wavy, crinkled wing phenotype. Cell viable in clones of adult epidermis; bristle and hair phenotypes autonomous; useful as a cell marker in *2L* for clonal analysis (Struhl).

alleles: 13 named alleles, all but *ck*¹ induced by ethyl methanesulfonate. Four additional EMS-induced alleles, including one weak allele, isolated by Nüsslein-Volhard, Wieschaus, and Klüding (1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82).

allele	isolation #	ref ^α	phenotypic class ^β
<i>ck</i> ¹			
<i>ck</i> ²	BMW13	1, 2	2
<i>ck</i> ³	BMW14	1, 2	3
<i>ck</i> ⁴	BMW26	1, 2	1
<i>ck</i> ⁵	HG22	1, 2	1
<i>ck</i> ⁶	HG29	1, 2	1
<i>ck</i> ⁷	HG32	1, 2	2
<i>ck</i> ⁸	64-106	2	1
<i>ck</i> ⁹	64-1564	2	1
<i>ck</i> ¹⁰	GM12	2, 3	3
<i>ck</i> ¹¹	CH11	3, 4	?
<i>ck</i> ¹²	CH39	3, 4	3
<i>ck</i> ¹³	CH52	3, 4	2

^α 1 = Ashburner, Tsubota, and Woodruff, 1982, *Genetics* 102: 401-20; 2 = Gubb, Shelton, Roote, McGill, and Ashburner, 1984, *Chromosoma* 91: 54-64; 3 = Maroni (unpublished); 4 = O'Donnell, Mandel, Kraus, and Sofer, 1977, *Genetics* 86: 553-66.

^β Class 1 survival at 1% or less, class 2 at 5%, and class 3 greater than 10% expected value.

cytology: Placed in 35B10-C1 based on its inclusion in *Df(2L)64j = Df(2L)34D1-2;35B9-C1 and Df(2L)fr30 = Df(2L)34C6-7;35B9-C1*, but not in *Df(2L)AR-R1 = Df(2L)35A3-4;35B9-C1*.

other information: Allelism of *l(2)35Ca* and *ck* inferred from similarities in phenotype and map position. Insertion TE35B between 35B9 and 35C1 results in a class 2 *ck* allele (Gubb *et al.*).

CkII α: Casein kinase II, α subunit

location: 3-{47}.

references: Saxena, Padmanabha, and Glover, 1987, *Mol. Cell. Biol.* 7: 3409-17.

phenotype: The structural gene for the catalytic subunit of casein kinase II (CKII). CKII is a cyclic-nucleotide-independent, Ca²⁺ and calmodulin-insensitive protein kinase. It phosphorylates serine and threonine residues of a broad range of nuclear and non-nuclear proteins with functions in development, cell division, and oncogenesis. It exists as a 130,000 molecular weight α₂β₂ tetramer. Purification of the *Drosophila* enzyme described by Glover, Shelton, and Brutlag (1983, *J. Biol. Chem.* 258: 3258-65).

molecular biology: cDNA sequence isolated by antibody screening of a λgt11 expression library. Nucleotide sequence shows an open reading frame encoding a 336-amino-acid polypeptide with strong homology to calf thymus CKIIα and homology to the yeast homologue; also shows weak homology to the yeast *Cdc28* gene. *Drosophila CkII α* and β can rescue casein kinase II deficient yeast (Glover).

cytology: Placed in 80A by *in situ* hybridization (Glover).

CkII β: Casein kinase II, β subunit

location: 1-{36}.

references: Saxena, Padmanabha, and Glover, 1987, *Mol. Cell. Biol.* 7: 3409-17.

phenotype: The structural gene for the autophosphorylated, regulatory β subunit of casein kinase II (CKII).

molecular biology: cDNA sequence isolated by antibody screening of a λgt11 expression library. Nucleotide sequence shows an open reading frame encoding a 215-amino-acid polypeptide with strong homology to calf thymus CKIIβ and homology to the yeast homologue. Genomic sequence reveals five introns in the protein-coding region and at least one more upstream from the initiating ATG (Saxena, Carney, Bruley, and Glover, 1989, *Genetics* 122: s25). *Drosophila CkII α* and β can rescue casein kinase II deficient yeast (Glover).

cytology: Placed in 10E by *in situ* hybridization (Glover).

cl: clot

location: 2-18.83 (Kotarski, Pickert, and MacIntyre, 1983, *Genetics* 105: 371-86).

origin: Spontaneous.

discoverer: Bridges, 27a3.

phenotype: Eye color dark maroon to sepialike with age, is less extreme than *sepia*. Severely deficient in one of three enzyme activities required for the conversion of dihydroneopterin triphosphate to pyrimidiazepine, a precursor of the drosopterins (Wiederrecht, Paton, and Brown, 1984, *J. Biol. Chem.* 259: 2195-2200). Claimed to accumulate 2-amino-4-hydroxypteridine, biopterin, sepiapterin, and xanthopterin (Nikla, 1972, *Can. J. Genet. Cytol.* 14: 105-11). Eye color autonomous when larval optic disk is transplanted into wild-type host (Beadle and Ephrussi, 1936, *Genetics* 21: 230). Larval Malpighian tubes pale yellow, distinguishable from wild type (Brehme and Demerec, 1942, *Growth* 6: 351-56). RK1.

alleles: *cl*² (CP627). *cl*³ and *cl*⁴ induced by ethyl methanesulfonate in *CyO* (Kotarski, Pickert, and MacIntyre, 1981, *DIS* 56: 191); *cl*^{CA1} associated with *T(1;2)cl*^{CA1} (Ashburner, Faithfull, Littlewood, Richards, Smith, and Velissariou, 1980, *DIS* 56: 186).

cytology: Placed in salivary chromosome bands 25E1-2 (Ashburner and Velissariou, 1980, DIS 55: 196).

claret: see *ca*

****cld*: cloudy**

location: 2-96 to -101.

origin: γ ray induced.

discoverer: Wallbrunn, 61j6.

references: 1964, DIS 39: 59.

phenotype: Wings opaque from fluid between upper and lower membranes; occasionally, fluid forms small blisters. Males sterile; females highly infertile. RK2.

cleft: see *cf*

clf: see *wtw*^{*clf*}

***cli*: clift (C. Nüsslein-Volhard)**

location: 2-17.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82 (fig.).

phenotype: Embryonic lethal. Defect in head involution. No segmental movements. Shows abdominal transformations in combination with *Pc*-like mutants.

alleles: Two.

****Cli*: Clipped wings**

location: 1- (to the left of *f*).

discoverer: Agol.

references: 1936, DIS 5: 7.

phenotype: Dominant wing mutant (no description given). Viable in male and homozygous female. RK3.

Clift: see *Cli*

clip wing: see *dp*^{*o2*}

clipped: see *cp*

Clipped wings: see *Cli*

Clipt: see *Cpt*

***Clk*: Clock (J.C. Hall)**

location: 1-1.4 (0.015 map units to the right of *per*^{*o1*}).

origin: Induced by ethyl methanesulfonate.

discoverer: Konopka and Orr.

references: Dushay, Konopka, Orr, Greenacre, Kyriacou, Rosbash and Hall, 1990, Genetics 125: 557-78.

synonym: *Clk-6*.

phenotype: The normal 24 hour period of the circadian rhythms of adult locomotor activity and of eclosion are shortened by approximately 1.5 hours. *Clk*+ heterozygotes have a period phenotype intermediate between wild-type and mutant homozygotes. The period of the phase-resetting response of the activity rhythm is shortened by 1-2 hours per cycle; adults entrain well to 12 hour light: twelve hour dark cycles, but evening peak of locomotor activity is advanced relative to wild-type; courtship song rhythms of mutant males have essentially normal one-minute periods. Temperature compensation of circadian period is normal (Q_{10} approximately equal to 1.0), in tests of locomotor activity rhythms.

cytology: Series of deletions and duplications running from X distal tip to white; all failed to uncover and ameliorate effects of *Clk*.

other information: *Clk* is close enough to *per*^{*o1*} mutation so that it could be allele of the period gene; however, *Clk/per*^{*o1*} indistinguishable from *Clk*+.

****clm*: clumpy marginals**

location: 1-32.6.

origin: Induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3205).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 68.

phenotype: Irregularly bent marginal hairs, especially on posterior border of wings. Bristles stiff and frequently bent or split. Viability and fertility of males good. Homozygous females reduced in viability and fertility. RK2.

other information: One allele each induced by 2-chloroethyl methanesulfonate and DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine.

***clo*: close down (T. Schüpbach)**

location: 2-77.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus.

phenotype: Female-sterile; homozygous females contain egg chamber which degenerate before yolk uptake. Occasionally normal egg chambers are found which take up yolk and form eggs. Such eggs often have fused dorsal appendages and remain unfertilized.

alleles: *clo*^{*1*} to *clo*^{*3*} isolated as *WG*, *WK*, and *WU*.

Clock: see *Clk*

close down: see *clo*

clot: see *cl*

cloudy: see *cld*

cloven thorax: see *clv*

club: see *cb*

clubfoot: see *cbf*

club wing: see *clw*

clumpy marginals: see *clm*

***clt*: cricketlet**

location: 2-96.

origin: Induced by ethyl methanesulfonate.

references: Shirras and Bownes, 1989, Proc. Nat. Acad. Sci. USA 86: 4559-63.

phenotype: Yolk protein synthesis reduced approximately twenty fold in homozygous females; such females are also defective in the synthesis of larval-serum protein 2 (LSP2) although LSP2 synthesis in larvae is unaffected. Adult fat body normal in morphology and in other secretory functions. Larval fat body persists into adult stage; oocyte development arrested in previtellogenic stage. Methoprene, a juvenile hormone analogue, which stimulates fat-body synthesis of yolk and vitellogenesis in starved wild-type females, has no effect in *clt* females; however, ovarian transplant experiments indicate that *clt* females have sufficient JH concentrations to promote oogenesis. Ecdysone, on the other hand, stimulates LSP2 and yolk-protein synthesis in *clt* females, but has no effect on larval-fat-body regression or oogenesis.

***clv-1: cloven thorax no. 1**

location: 1-0.0.
origin: X ray induced.
discoverer: Muller, 19h.
references: 1935, DIS 3: 29.
phenotype: Thorax often has long cleft; partially dominant. Semilethal at low temperature; viable at high temperature. RK3.

***clv-2**

location: 1-42.0.
origin: X ray induced.
discoverer: Muller, 26111.
references: 1935, DIS 3: 29.
phenotype: Thorax has longitudinal cleft, sometimes half thorax. One wing often reduced or like *vg*. Partially dominant. Semilethal. RK3.
alleles: **clv-2*^{52b} completely recessive (CP627). **clv-2*^D [formerly *Clv*³ (Maroni, 1968, DIS 43: 60)] is a fully dominant allele associated with *T(1;2)clv-2*^D.
cytology: Placed in 11A7-8 based on association of *clv-2*^D with *T(1;2)11A7-8;27E2-3* (Kirschbaum).

*Clv*³: see *clv-2*^D

***clw*: club wing**

location: 1-21 (same as *sn*; inferred from instability correlated with that of *sn*^{49s}).
origin: Presumably induced by *P*-factor in chromosome isolated from natural population; arose simultaneously with *sn*^{49s}.
references: Golubovsky and Zakharov, 1979, Genetika (Moscow) 15: 1599-1609.
 1980, DIS 55: 49-55.
 Zakharov and Golubovsky, 1980, Genetika (Moscow) 16: 1603-12.
 Zakharov and Yurchenko, 1984, Genetika (Moscow) 20: 266-73.
 Yurchenko, Zakharov, and Golubovsky, 1984, Mol. Gen. Genet. 194: 279-85.
phenotype: Wings fail to expand; retain pupal conformation. Penetrance low, 11% in males and 0.6% in homozygous females; inversely related to temperature; penetrance in *XO* males less than in *XY* males. Unstable, *clw sn*^{49s} can mutate to wild type, either unstable (*i.e.*, can regenerate *clw sn*^{49s}) or stable, or to a stable *clw*⁺ with an unstable *sn* phenotype of intermediate strength. Interpreted as owing to an insertional sequence between *clw* and *sn*.
cytology: Placed in 7D based on its presumed contiguity with *sn*. Since it may have been carried in by a transposon, there may not be a normal allele at this position in wild-type chromosomes.
other information: May be allelic to *cb*.

***cm*: carmine**

location: 1-18.9.
origin: Spontaneous.
discoverer: Mohr, 27d27.
references: 1927, Z. Indukt. Abstamm. Vererbungsl. 45: 403-5.
phenotype: Eye color translucent dark ruby. With *st*, eye color deep orange; with brown, slightly lighter than *bw* alone. Larval Malpighian tubes very pale yellow. RK1.

alleles:

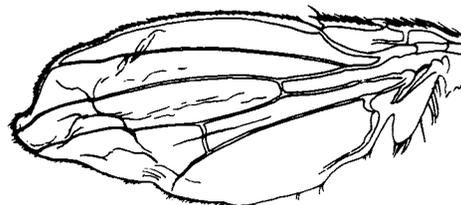
allele	origin	discoverer	synonym	ref ^α	comments
<i>cm</i> ¹	spont	Mohr, 27d27		4	
<i>cm</i> ²	mustard	Sobels, 571	<i>cm</i> ²⁸⁻⁴	5	
<i>cm</i> ³	X ray	Valentin, 67j	<i>cm</i> ^{67j}	6	
<i>cm</i> ⁴		Gerasimova	<i>cm</i> ^{MR1}	1, 2	unstable allele ^β
<i>cm</i> ⁵	EMS	Lefevre	<i>cm</i> ^{VEM119}	3	

^α 1 = Gerasimova, 1983, DIS 59: 38; 2 = Gerasimova, and Ilyin, 1984, DIS 60: 111-12; 3 = Lefevre, and Watkins, 1986, Genetics 113: 869-95; 4 = Mohr, 1927, Z. Indukt. Abstamm. Vererbungsl. 45: 403-05; 5 = Sobels, 1958, DIS 32: 84; 6 = Valentin, 1971, DIS 46: 40.

^β Arose in a stock carrying a transposable element in *ct* and reverts spontaneously; inserted element neither *P* nor *gypsy* (their *mdg4*).

cytology: Located in 6E5-6 (CP627; Lefevre, 1981, Genetics 99: 461-80).

cm: see *cmp*



***Cm*: Crimp**

Edith M. Wallace, unpublished.

****Cm*: Crimp**

location: 3-43.5.
origin: Spontaneous.
discoverer: Bridges, 28a28.
phenotype: Heterozygote has crimped wings ruffled on rear edge. Classification good in first 4 days' hatch, then *Cm* overlaps wild type progressively. Better at 25° than at 19°. Homozygous lethal. RK2 as lethal; RK3 as dominant.

***Cma*: Comma**

location: 3-57.5 (between *jvl* and *sbd*).
origin: X ray induced.
discoverer: Lewis, 1971.
references: Craymer, 1980, DIS 55: 197.
phenotype: *Cma*⁺ has a pair of comma-like depressions at the anterior edge of the dorsal mesothorax similar to those of some *dp* alleles, intumed dorsocentral bristles, a *sr*-like phenotype, and a soft-appearing cuticle. *Cma*⁺ imparts dominance to the vortex effects of *dp*, *dp*^v, *dp*^{ov}, and *dp*^{olv} alleles. *Cma*⁺ flies flightless, have abnormal myofibrils of indirect flight muscles (Mogami). Homozygous lethal. RK1.
cytology: Location in 88C-F inferred from its map position and its survival in combination with *Df(3R)sbd*¹⁰⁵ = *Df(3R)88F9-89A1;89B4-5* and the failure of a synthetic deficiency to the left that includes 88C to include *jvl* (Craymer).

cmd: carminoid

location: 3-68.2.

origin: Spontaneous.

references: Mostashfi and Koliantz, 1970, DIS 45: 34.

phenotype: Eye color resembles that of *cm*.*cMdh*: see *Mdh1**cMhc-1*: see *Mhc-c***cml: caramel**location: 2-71.2 (said to complement *sf* and *ake*).

origin: Spontaneous.

references: Ribó, 1968, DIS 43: 59.

phenotype: Eye color brownish orange at eclosion, darkens with age but not as dark as *se*. Produces orange eye in combination with *cn*. RK1.**cmp: crumpled**

location: 3-93.

origin: Spontaneous.

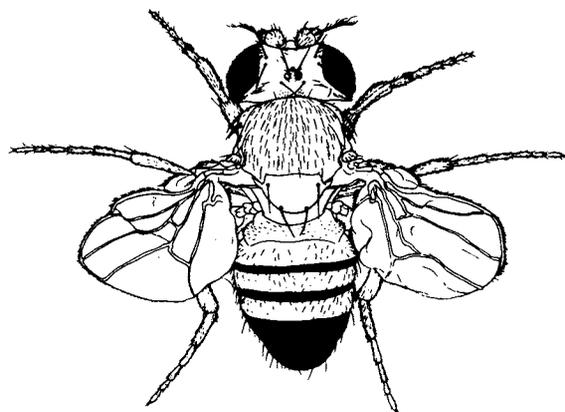
discoverer: Bridges, 22d2.

synonym: *cm*.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 247.

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 223.

phenotype: Wings about two-thirds normal size and greatly crumpled or blistered. Marginal hairs irregularly clumped. Legs irregularly shortened and gnarled. Bristles somewhat short and thick. Posterior scutellars slightly divergent. Branches of arista bent anteriorly near middle, with apices parallel to main axes of arista. Viability and fertility may be low. RK3.

**cmp: crumpled**

Edith M. Wallace, unpublished.

***cmt: comet**location: 3-57.2 (based on 35 *cu-sr* recombinants).

origin: Induced by ethyl methanesulfonate.

references: García-Bellido and Dapena, 1973, DIS 50: 179.

1974, Mol. Gen. Genet. 128: 117-30 (fig.).

phenotype: Homozygous lethal; however, clones of homozygous cells survive in cuticle. Wing cells like *mwh* but with fewer trichomes per cell and smaller angles between trichomes. Less distinct in tergites but accom-

panied by loss of pigment.

cn: cinnabar

location: 2-57.5.

origin: Spontaneous.

discoverer: Clausen, 20i8.

references: 1924, J. Exp. Zool. 38: 423-36.

phenotype: Eye color bright red, like *v* or *st*. Ocelli colorless. Eye color darkens with age, but ocelli remain colorless. Larval Malpighian tubes pale yellow (Beadle, 1937, Genetics 22: 587-611). *cn* defective in ommochrome synthesis; blocks conversion of kynurenine to 3-hydroxykynurenine, which has been identified as the *cn*⁺ hormone (Butenandt, Weidel, and Schlossberger, 1949, Z. Naturforsch. 4b: 242-44). *cn* homozygotes devoid of kynurenine 3-hydroxylase activity (Ghosh and Forrest, 1967, Genetics 55: 423-31). Enzyme activity proportional to the number of doses of *cn*⁺; *cn*⁺ therefore concluded to be the structural gene for kynurenine 3-hydroxylase (EC 1.14.13.9) (Sullivan, Kitos, and Sullivan, 1973, Genetics 75: 651-61). Also *cn* defective in the uptake of kynurenine by eye discs and Malpighian tubules, where it is normally converted to 3-hydroxykynurenine (Sullivan, Grillo, Kitos, 1974, J. Exp. Zool. 188: 225-34). Nonautonomous in development of pigment of transplanted eye disks (Beadle and Ephrussi, 1936, Genetics 21: 230); however, ethyl methanesulfonate-induced mutants recovered as mosaics in homozygous *red* background (Paton and Sullivan, 1978, Biochem. Genet. 16: 855-65); furthermore, even though *st* has no detectable 3-hydroxykynurenine, it is able to rescue *cn* in transplants (Phillips, Simmons, and Bowman, 1970, Biochem. Genet. 4: 481-87). Enzyme activity developmentally regulated with a peak of activity in early third instar and a five-fold higher peak in the second half of pupal development (Sullivan *et al.*). Heterozygotes in all pairwise combinations of 13 ethyl methanesulfonate-induced alleles exhibit mutant phenotype (Paton and Sullivan, 1978). RK1.**alleles:**

allele	origin	ref ^α	comments
<i>cn</i> ¹		3, 4	
<i>cn</i> ²		3, 4	hypomorph in <i>In(2R)Cy</i>
<i>cn</i> ^{2P}		5	amorphic derivative of <i>cn</i> ² in <i>In(2LR)CyO</i>
<i>cn</i> ³		3	amorphic allele
<i>cn</i> ⁴			amorphic allele in <i>In(2LR)bw</i> ^{VI}
<i>cn</i> ^{4P-30P}	EMS	7	<i>cn</i> ^{12P} and three other alleles hypomorphic
<i>cn</i> ^{31P}	EMS	7	amorphic derivative of <i>cn</i> ² in <i>In(2LR)SM1</i>
<i>cn</i> ^{35k}		3	hypomorphic allele
<i>cn</i> ^{36c}		3	hypomorphic allele
<i>cn</i> ^{38j}		6	hypomorphic allele
<i>cn</i> ⁷⁸	spont	2	amorphic allele in <i>In(2LR)Gla</i>
<i>cn</i> ^{80c}	γ ray		amorphic allele
<i>cn</i> ^{lvi}	X ray	1	amorphic allele
<i>cn</i> ^{rbr}		8	detail follows
<i>cn</i> ^s		3, 4	

^α 1 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186; 2 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193; 3 = CP552; 4 = CP627; 5 = Craymer, 1980, DIS 55: 197; 6 = Ives, 1968, DIS 43: 64; 7 = Paton and Sullivan, 1978, Biochem Genet. 16: 855-65; 8 = Valadé del Rio, 1974, DIS 51: 22.

cytology: Placed in 43E3-14 by deficiency analysis (Zacharopoulou, Yannopoulos, and Stamatis, 1981, DIS 56: 166-67); restricted to 43E6-14 by inclusion in

Df(2R)CA53 = Df(2R)46E6;44B6.

cn^{rbr}

phenotype: An unstable amorphic allele that reverts with frequency of 3×10^{-3} (Valadé del Rio, 1974, DIS 51: 22); also induces reversions of *cn*¹ and *cn*² alleles on homologous chromosome in heterozygotes; some of the revertants themselves mutate back to *cn*^{rbr} (Valadé del Rio, 1982, *Experientia* 38: 790-92).

cno: canoe

location: 3-49.
origin: Induced by ethyl methanesulfonate.
references: Jürgens, Wieschhaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95 (fig.).
phenotype: Homozygous lethal; dorsal surface of embryo open.
alleles: Fourteen; four weak and one temperature sensitive.

Co-3A: see *I(2)S3a*

Co-7: see *I(2)S7*

Co-122: see *mle*

Coa: Coarse

location: 3-7.7.
discoverer: E.H. Grell, 1969.
references: R.F. Grell, 1978, *Genetics* 89: 65-77.

coal: see *cal*

Coarse: see *Coa*

coatless: see *ctl*

***coc: collapsed ocelli**

location: 1-61.5.
origin: Induced by D-1:6-dimethanesulfonyl mannitol (CB. 2511).
discoverer: Fahmy, 1960.
references: 1964, DIS 39: 58.
phenotype: Ocelli small and flat. Anterior ocellar hairs frequently missing. Other slight alterations in body size and wing shape. RK3.
cytology: Placed in salivary region 18A4 through 18B8 on the basis of its inclusion within the deficiency carrying the left end of *In(1)y*⁴ = *In(1)1A8-B1;18A3-4* and the right end of *In(1)sc*⁹ = *In(1)1B2-3;18B8-9* (Norton and Valencia, 1965, DIS 40: 40).

coi: coitus interruptus (J.C. Hall)

location: 1-22.1.
origin: Induced by ethyl methanesulfonate.
discoverer: Lindsley.
references: Hall, Siegel, Tompkins, and Kyriacou, 1980, *Stadler Genet. Symp.* 12: 43-82.
phenotype: Males court females with slightly reduced vigor and mating success but mate frequently; duration of such copulations average 60% of normal 20 min; high proportion of mutant males also have abnormal sperm, e.g., nonmotile; females apparently unaffected by the mutation.

Coi: see *Cu*

Coiled: see *Cu*

Coiledex: see *Cu*

coitus interruptus: see *coi*

Collagen: see *Cg*

collapsed ocelli: see *coc*

com: compressed

location: 3-48.5.
origin: Spontaneous.
discoverer: Bridges, 18k27.
references: Bridges and Morgan, 1923, *Carnegie Inst. Washington Publ. No. 327*: 193.
phenotype: Head flattened ventrally. Eyes small, displaced. Vibrissae tufted. Aristae crumpled. Humeral patches elevated. Wings droopy. Poor viability and fertility. RK3.

com: see *comt*

comatose: see *comt*

***com-d: compressed-dilapidator**

location: 3-68.5.
origin: Spontaneous.
discoverer: Bridges, 19c8.
phenotype: Flies small, pale, weak, with defective legs and wings. RK3.

comb gap: see *cg*

comet: see *cmt*

Comma: see *Cma*

Compensatory Response: see *CR*

compensatory response: see *Xh-cr*

compressed: see *com*

compressed-dilapidator: see *com-d*

comt: comatose (J.C. Hall)

location: 1-39.2.
origin: Induced by ethyl methanesulfonate.
synonym: *com*, preoccupied.
references: Siddiqi and Benzer, 1976, *Proc. Nat. Acad. Sci. USA* 73: 3253-57.
phenotype: Larvae or adults become paralyzed when exposed to high temperatures, but this requires many seconds for induction; recovery from immobility on lowering of temperature is slow and directly correlated with duration of paralysis; most of the *comt* alleles do not induce relatively rapid paralysis until 38° (Siddiqi and Benzer, 1976), but two (*com*¹⁰¹, *com*¹⁰²) cause pass-out at 29° (Homyk, Szidonya, and Suzuki, 1980, *Mol. Gen. Genet.* 177: 553-65); the mutations induce graded decrease and recovery of function of synapses at neuromuscular junctions as temperature is raised and lowered (Siddiqi and Benzer, 1976); several alleles are cold sensitive for paralysis as well as heat sensitive (Siddiqi and Benzer, 1976). ERG normal (Homyk and Pye, 1989, *J. Neurogenet.* 5: 37-48).

alleles:

allele	isolation number	discoverer
<i>comt</i> ¹	ST53	Siddiqi and Benzer
<i>comt</i> ²	101	Homyk <i>et al.</i>
<i>comt</i> ³	102	Homyk <i>et al.</i>
<i>comt</i> ⁴	ST17	Siddiqi and Benzer
<i>comt</i> ⁵	ST47	Siddiqi and Benzer

allele	isolation number	discoverer
<i>comt</i> ⁶	TP7	Siddiqi

***con: condensed**

location: 1-27.1.

origin: Spontaneous.

discoverer: Bridges, 36d11.

references: 1937, DIS 7: 6.

phenotype: Thorax and abdomen shortened; abdomen dilated, exposing ventral skin to side view. Eyes slightly roughened, occasionally kidney shaped, and somewhat dark. Wings short and bluntly rounded with crossveins closer together than normal. Bristles shortened and somewhat fine at 19°, stubby at 25°. Postscutellars semi-erect and crossed; posterior verticals shortened or missing. Male entirely sterile. Viability 50% wild type. RK2.

cytology: Salivary chromosome studies (Demerec, Kaufmann, Fano, Sutton, and Sansome, 1942, Year Book - Carnegie Inst. Washington 41: 191) show locus to lie between 7C4-5 and 8C1-2. Further restricted to 8A through 8C2 on the basis of its genetic location to the right of *oc* at 8A.

concave wing: see *ccw*

concertina: see *cta*

condensed: see *con*

Confluens: see *Co*

Confluent: see *Cf*

Confluent-3: see *Dl*^{cf-3}

congested: see *cgd*

contorted: see *ctt*

Contrabithorax: see *Cbx*

Contrabithorax: see *Cbx* under *BXC*

contrast blind: see *l(1)ogre*

control of female fertility: see *cff*

convex wing: see *cvw*

cop: copper

location: 1-43.3.

origin: Induced by D-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3026).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 68.

phenotype: Brownish red eye color. Best classification in newly emerged flies. Occasionally, wings show cutaway inner margins. Excellent viability and fertility in both sexes. RK2.

other information: Two alleles induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine.

Cor: Corroded eye

location: 3- (not located).

origin: X ray induced.

discoverer: Muller.

references: 1946, DIS 20: 66.

phenotype: *Cor*⁺ shows slight irregular flecking of eye. In combination with *v*, expression enhanced, producing

patchy diminution in color, especially near posterior margin of eye, giving impression that color was washed or eaten away, especially from deeper layers; regions of surface often blackened. Homozygote not described. RK2.

corkscrew: see *csw*

corrugated wing: see *corr*

****corr: corrugated wing***

location: 2-36.

origin: Spontaneous.

discoverer: Mayeda, 61g.

references: 1963, DIS 38: 31.

phenotype: Wings wrinkled and wavy, reduced to three-fourths normal size. Whole wing corrugated at 20°, only posterior third at 25°. Good classification. RK2.

Corroded eye: see *Cor*

corrugated wing: see *corr*

cort: cortex (T. Schüpbach)

location: 2-49.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

phenotype: Maternal-effect lethal mutant; homozygous females lay eggs in which a narrow halo of clear cytoplasm appears in the cortex around the time when nuclei would be expected to migrate to the periphery, but no cellularization takes place.

alleles: Two, *cort*¹ and *cort*², isolated as *QH* and *RH*.

cos: costa (P. Simpson)

location: 2-57.4.

synonym: *cos2*.

references: Whittle, 1976, Dev. Biol. 51: 257-68.

Grau and Simpson, 1987, Dev. Biol. 122: 186-200 (fig.).

Simpson and Grau, 1987, Dev. Biol. 122: 201-09 (fig.).

phenotype: Homozygotes, hemizygotes, and heteroallelic heterozygotes for class I alleles die as embryos with a normal cuticular phenotype. When derived from homozygous ovarian clones, however, abnormal deletion-duplication patterns in each segment are observed; posterior rows of abdominal denticles are replaced by mirror-image duplications of the anterior rows, including the segmental boundary, which is thereby duplicated; thoracic denticle belts missing; *cos*¹ especially severely affected in this regard, including loss of entire segments. Heterozygotes derived from homozygous ovarian clones show slightly abnormal segmental patterning. Flies heterozygous for class I alleles or *cos* deficiencies and at the same time heterozygous for *Cos* alleles display pattern duplications of wings and halteres. Class II alleles are hemizygous lethal but homozygous viable. They display wing and haltere duplications indistinguishable from those found for *cos*¹/*Cos*¹ heterozygotes as described by Whittle. Flies heterozygous for class III alleles and *Cos* show moderately reduced viability and occasional wing duplications; flies homozygous for class III alleles show reduced viability and have wild-type phenotype, but enhance the pattern duplications associated with *Cos*⁺; hemizygotes for class III alleles or heterozygotes with class I alleles are lethal or nearly so in the presence of *Cos*, the survivors invariably exhibiting pattern duplications of wings and halteres.

alleles: Severity of class I and II alleles estimated as follows: $cos^3 > cos^2 > cos^1 = cos^4 = cos^5 > cos^6 > cos^7$ Similarly for class III alleles: $cos^{V5} \geq cos^{V4} = cos^{V1} \geq cos^{V3} \geq cos^{V2}$.

allele	origin	discoverer	comments
<i>cos</i> ¹ α	EMS	Whittle	class I allele
<i>cos</i> ²	X ray	Grau & Simpson	class I allele
<i>cos</i> ³	X ray	Grau & Simpson	class I allele
<i>cos</i> ⁴	EMS	Grau & Simpson	class I allele
<i>cos</i> ⁵	EMS	Grau & Simpson	class I allele
<i>cos</i> ⁶	EMS	Grau & Simpson	class I allele
<i>cos</i> ⁷	EMS	Grau & Simpson	class II allele
<i>cos</i> ^{V1} β		Simpson	class III allele
<i>cos</i> ^{V2} β		Simpson	class III allele
<i>cos</i> ^{V3} β		Simpson	class III allele
<i>cos</i> ^{V4} β		Simpson	class III allele
<i>cos</i> ^{V5}	EMS	Grau & Simpson	class III allele

α Synonym: *cos2*^{W1}.

β Found to exist in various laboratory stocks.

cytology: Placed in 43B2-C3 on the basis of its inclusion in the region of overlap of *Df(2R)pk78k = Df(2r)42E3;43C3* and *Df(2R)ST1 = Df(2R)43B2-C1;43E1-8*.

Cos: Costal (P. Simpson)

location: 2-67.2.

synonym: *Epa: Epalette; Cos1*.

references: Whittle, 1976, Dev. Biol. 51: 257-68.

Grau and Simpson, 1987, Dev. Biol. 122: 186-200 (fig.).

Simpson and Grau, 1987, Dev. Biol. 122: 201-09 (fig.).

phenotype: Mirror-image duplications of wings and halteres; duplications of wing begin in mid-costal region and may include entire anterior compartment; posterior compartments unaffected, even when transformed into anterior compartments in *en* homozygotes. Penetrance of *Cos* higher when maternally than when paternally inherited. Class I alleles are homozygous viable and display pattern duplications in both heterozygotes and homozygotes. Penetrance and expressivity variable and may overlap wild type. Class II alleles are lethal when homozygous or in heteroallelic combination with one another; they sometimes display pattern duplications in heterozygotes. Class III alleles are homozygous lethal and have no dominant phenotype. Flies heterozygous for alleles of any of the three classes display the phenotype when simultaneously heterozygous for lethal alleles or deletions of *cos*; the heterozygous expression of *Cos* is dependent on the number of *cos*⁺ alleles present, with the severity of wing duplication decreasing as the number of *cos*⁺ alleles increases from one to three. Flies heterozygous for *Cos* and homozygous or hemizygous for some viable alleles of *cos* die as pharate adults and display pattern duplications in the anterior compartment of every body segment. Homozygotes of class II and III alleles die as embryos with abnormal cuticular patterns; failure to develop of variable extents of the anterior end of the embryo, *i.e.* head or head and thorax, as well as mirror image duplications of anterior denticle belts of abdominal segments, or more frequently simply disturbed denticle polarity. Embryos that are simultaneously homozygous for *cos*^V alleles and *Cos*³ are more severely affected, with some exhibiting a bicaudal phenotype. *cos*^{V1} *Cos*²/*Df(2R)CA58* flies survive poorly, but show pattern duplications in the anterior compartments of all seg-

ments. Revertants of *Cos* are viable and wild type in phenotype; as they are presumably null alleles, *Cos* mutations are presumed to be gain of function alleles.

alleles:

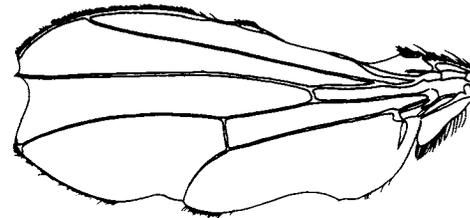
allele	origin	discoverer	synonym	ref	α	comments
<i>Cos</i> ¹	EMS	Whittle, 1976	<i>Cos1</i> ^{W1}	2		class III allele
<i>Cos</i> ²	EMS	Simpson		1		class II allele
<i>Cos</i> ³	X ray	Simpson		1		class II allele
<i>Cos</i> ⁴	EMS	Grau & Simpson		1		class III allele
<i>Cos</i> ⁵	X ray	Grau & Simpson				class II allele
<i>Cos</i> ⁶	X ray	Grau & Simpson				class III allele
<i>Cos</i> ⁷	X ray	Grau & Simpson				class III allele
<i>Cos</i> ⁸	EMS	Mariol		1		class II allele
<i>Cos</i> ⁹	EMS	Nüsslein-Volhard		1		class II allele
<i>Cos</i> ¹⁰	γ ray	Harrington	<i>Epa; Cos1</i> ^{AI}	1		class I allele

α 1 = Grau and Simpson, 1987, Dev. Biol. 122: 186-200; 2 = Whittle, 1976, Dev. Biol. 51: 257-68

cytology: Placed in 49F13-50A3 based on its inclusion in *Df(2R)vg-B = Df(2R)49D3-4;50A2-3* but not *Df(2R)vg104 = Df(2R)49C4;49F13* (Lasko and Pardue, 1988, Genetics 120: 495-502).

cos2: see *cos*

costakink: see *csk*



cp: clipped

Edith M. Wallace, unpublished.

cp: clipped

location: 3-45.3.

discoverer: Mainx, 34g.

references: 1936, Z. Indukt. Abstamm. Vererbungslehre 71: 303-4 (fig.).

Pollitzer, 1937, DIS 8: 91.

phenotype: Wing margins snipped, most often along marginal vein. At 19°, character slighter but completely penetrant. Shows partial dominance in combination with Minutes (Sinclair and Suzuki, 1979, Genetics 91: s117; V. Walker). Most stocks carry closely linked modifiers; has reduced viability when phenotype is good (Craymer). RK2.

alleles: *cp*^{SS305} and *cp*^{SS307} X ray induced (Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95).

cytology: Placed between the breakpoints of *T(Y;3)L131 = T(Y;3)75D4-5* (V. Walker) and *T(2;3)spy = T(2;3)33D4-E3;79A4-B1* (Puro, 1983, Hereditas 62: 414-18).

Cp15: Chorion protein

location: 3-{26.5} (based on *in situ* hybridization).

Located between *Cp18* and *Cp19*.

synonym: *S15*: Shell.

references: Spradling, Digan, Mahowald, Scott, and Craig, 1980, Cell 19: 905-14.

Spradling, 1981, Cell 27: 193-201.

Griffin-Shea, Thireos, and Kafatos, 1982, Dev. Biol. 91: 325-36.

Levine and Spradling, 1985, Chromosoma 92: 136-42.

Wong, Pustell, Spoerel, and Kafatos, 1985, Chromosoma 92: 124-35.

phenotype: The second of four chorion-protein genes in a 6-kb sequence; encodes S15-1, a chorion protein estimated at 15,000 daltons by Waring and Mahowald (1979, Cell 16: 599-607) and 9700 daltons by Petri, Wyman, and Kafatos (1976, Dev. Biol. 49: 185-99). Temporal and spatial distribution of expression described by Park and Spradling (1987, Genes Dev. 1: 497-509).

alleles: Probably the locus for which Yannoni and Petri (1980, Wilhelm Roux's Arch. Entwicklungsmech. Organ. 189: 17-24) detected electrophoretic variants by isoelectric focusing.

cytology: Localized to 66D11-15 by *in situ* hybridization. Less than 1 kb from *Cp18*.

molecular biology: Gene cloned and sequenced (Levine and Spradling; Wong *et al.*). Transcription unit estimated to be 515 nucleotides by Levine and Spradling and 590 by Wong *et al.*; contains a 71 nucleotide intron four codons downstream from the translation initiation site. Direction of transcription the same as for *Cp16*, *Cp18* and *Cp19*. The conceptual polypeptide, which contains a signal peptide and a putative signal-peptide cleavage site, contains 115 residues and the mature chorion protein 97 residues for a calculated molecular weight of 9934 (Wong *et al.*) or 9467 (Levine and Spradling). Flanking sequences examined for putative regulatory sequences. Located in a chromosome region that is amplified approximately 60 fold as a series of nested bidirectional replication forks spanning approximately 100 kb in the follicle cells surrounding maturing oocytes; amplification of the entire region under the control of *ACE3*, a *cis*-acting sequence between 615 and 187 base pairs upstream from *Cp18* (Kalfayan, Levine, Orr-Weaver, Parks, Wakimoto, deCicco, and Spradling, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 527-35), and at least four amplification-enhancing regions (AERs) located one base pair downstream from each of the four second-chromosome chorion-protein genes (Delidakis and Kafatos, 1989, EMBO J. 8: 891-901).

Cp16

location: 3-{26.5} (based on sequence proximity to *Cp15* and *Cp18*).

synonym: *S16*.

references: Griffin-Shea, Thireos, and Kafatos, 1982, Dev. Biol. 91: 325-36.

phenotype: The fourth of four chorion-protein genes in a 6-kb sequence; encodes S16-1, a 16,000-dalton chorion protein. Temporal and spatial distribution of expression described by Park and Spradling (1987, Genes Dev. 1: 497-509).

cytology: Placed in 66D11-15 based on sequence proximity to *Cp15* and *Cp18*. Approximately 1 kb from *Cp19*.

molecular biology: Identified on genomic clone selected with cDNA probe for *Cp15*. S1 nuclease digest of DNA-mRNA hybrid indicates presence of small intron toward one end of gene. Located in a chromosome region that is amplified approximately 60 fold as a series of nested bidirectional replication forks spanning approxi-

mately 100 kb in the follicle cells surrounding maturing oocytes; amplification of the entire region under the control of *ACE3*, a *cis*-acting sequence between 615 and 187 base pairs upstream from *Cp18* (Kalfayan, Levine, Orr-Weaver, Parks, Wakimoto, deCicco, and Spradling, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 527-35), and at least four amplification-enhancing regions (AERs) located one base pair downstream from each of the four second-chromosome chorion-protein genes (Delidakis and Kafatos, 1989, EMBO J. 8: 891-901). Transcriptionally most active during stages 13 and 14.

Cp18

location: 3-{26.5} (based on *in situ* localization).

synonym: *S18*.

references: Spradling, Digan, Mahowald, Scott, and Craig, 1980, Cell 19: 905-14.

Spradling, 1981, Cell 27: 193-201.

Griffin-Shea, Thireos, and Kafatos, 1982, Dev. Biol. 91: 325-36.

Levine and Spradling, 1985, Chromosoma 92: 136-42.

Wong, Pustell, Spoerel, and Kafatos, 1985, Chromosoma 92: 124-35.

phenotype: The first of four chorion-protein genes in a 6-kb sequence; encodes S18-1 estimated at 18,000 daltons by Waring and Mahowald (1979, Cell 16: 599-607) and 15,600 by Petri, Wyman, and Kafatos (1976, Dev. Biol. 49: 185-99). Temporal and spatial distribution of expression described by Park and Spradling (1987, Genes Dev. 1: 497-509).

cytology: Localized to 66D11-15 by *in situ* hybridization. Less than 1 kb from *Cp15*.

molecular biology: Gene cloned and sequenced (Levine and Spradling; Wong *et al.*). Transcription unit estimated to be 650 nucleotides by Levine and Spradling and 825 by Wong *et al.*; contains a 176 nucleotide intron five codons downstream from the translation initiation site. Direction of transcription the same as for *Cp15*, *Cp16* and *Cp19*. The conceptual polypeptide, which contains a signal peptide and a putative signal-peptide cleavage site, contains 172 residues and the mature chorion protein 156 residues for a calculated molecular weight of 15517 (Wong *et al.*) or 15027 (Levine and Spradling). Flanking sequences examined for putative regulatory sequences. Located in a chromosome region that is amplified approximately 60 fold as a series of nested bidirectional replication forks spanning approximately 100 kb in the follicle cells surrounding maturing oocytes; amplification of the entire region under the control of *ACE3*, a *cis*-acting sequence between 615 and 187 base pairs upstream from *Cp18* (Kalfayan, Levine, Orr-Weaver, Parks, Wakimoto, deCicco, and Spradling, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 527-35), and at least four amplification-enhancing regions (AERs) located one base pair downstream from each of the four second-chromosome chorion-protein genes (Delidakis and Kafatos, 1989, EMBO J. 8: 891-901).

Cp19

location: 3-{26.5} (based on sequence proximity to *Cp15* and *Cp18*).

synonym: *S19*.

references: Griffin-Shea, Thireos, and Kafatos, 1982, Dev. Biol. 91: 325-36.

Wong, Pustell, Spoerel, and Kafatos, 1985, Chromosoma

92: 124-35.

phenotype: The third of four chorion-protein genes in a 6-kb sequence; encodes S19-1, a chorion protein estimated at 19,000 daltons by Waring and Mahowald (1979, Cell 16: 599-607) and 17,500 by Petri, Wyman, and Kafatos (1976, Dev. Biol. 49: 185-99). Temporal and spatial distribution of expression described by Park and Spradling (1987, Genes Dev. 1: 497-509).

cytology: Placed in 66D11-15 based on sequence proximity to *Cp15* and *Cp18*. Approximately 1 kb from *Cp16* on one side and less than 1 kb from *Cp15* on the other.

molecular biology: Gene cloned and sequenced (Wong *et al.*). Transcription unit estimated to be 742 nucleotides contains a 89 nucleotide intron five codons downstream from the translation initiation site. Direction of transcription the same as for *Cp15*, *Cp16* and *Cp18*. The conceptual polypeptide, which contains a signal peptide and a putative signal-peptide cleavage site, contains 173 and the mature chorion protein 156 residues for a calculated molecular weight of 16722 daltons. Flanking sequences examined for putative regulatory sequences. Located in a chromosome region that is amplified approximately 60 fold as a series of nested bidirectional replication forks spanning approximately 100 kb in the follicle cells surrounding maturing oocytes; amplification of the entire region under the control of *ACE3*, a *cis*-acting sequence between 615 and 187 base pairs upstream from *Cp18* (Kalfayan, Levine, Orr-Weaver, Parks, Wakimoto, deCicco, and Spradling, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 527-35), and at least four amplification-enhancing regions (AERs) located one base pair downstream from each of the four second-chromosome chorion-protein genes (Delidakis and Kafatos, 1989, EMBO J. 8: 891-901).

alleles: Electrophoretic variants identified by isoelectric focusing (Yannoni and Petri, 1981, Wilhelm Roux's Arch. Dev. Biol. 190: 301-03).

Cp36

location: 1-{23} (based on *in situ* localization).

synonym: *S36*.

references: Spradling and Mahowald, 1979, Cell 16: 589-98.

Spradling, Waring, and Mahowald, 1979, Cell 16: 609-16.

Spradling and Mahowald, 1980, Proc. Nat. Acad. Sci. USA 77: 1096-1100.

Spradling, 1981, Cell 27: 193-201.

phenotype: The structural gene for s36-1, a 36,000-dalton chorion protein. Synthesis primarily during stages 12 and 13 (Waring and Mahowald, 1979, Cell 16: 599-607) and during stages 11 and 12 (Petri, Wyman, and Kafatos, 1976, Dev. Biol. 49: 185-99). Approximately 1,000 daltons cleaved from primary translation product to yield mature protein (Spradling *et al.*, 1980). Temporal and spatial distribution of expression described by Park and Spradling (1987, Genes Dev. 1: 497-509).

alleles: Two-dimensional gels reveal electrophoretic variants, which we designate *Cp36^a* and *Cp36^b*. Null allele, *Cp36^{nl}* [formerly *fs(1)cor-36*] produces no 36,000-dalton chorion protein (Digan, Spradling, Waring, and Mahowald, 1979, ICN-UCLA Symposium 14: 171-81).

cytology: Localized to 7F1-2 by *in situ* hybridization. Less than 1 kb distal to *Cp38*.

molecular biology: Resides on the same transcription unit as *Cp38*; separated by 1414 bp; gene 1100 bp in length and contains a small 5' intron (Wakimoto); nascent transcripts contain a ribonucleoprotein particle at each splice junction (Olsheim, Miller, and Beyer, 1985, Cell 43: 143-51). Abundantly transcribed producing polyadenylated mRNA in follicle cells surrounding stage-12 and -13 oocytes. Abundance markedly reduced in *In(1)oc = In(1)7F1-2;8A1-2* homozygotes. Located in a chromosome region that is amplified approximately 14-16 fold as a series of nested bidirectional replication forks spanning approximately 100 kb in the follicle cells surrounding maturing oocytes; amplification of the entire region under the control of *ACE3*, a *cis*-acting sequence between 654 and 266 base pairs upstream from *Cp38* (Wakimoto). Amplification disrupted by *In(1)oc = In(1)7F1-2;8A1-2*; female sterility results in homozygotes. Electron-microscope characterization of transcription unit, including length, polymerase II density, and discrete termination site by Olsheim, Miller, and Beyer (1986, EMBO J. 5: 3591-96).

Cp38

location: 1-{23} (based on *in situ* localization).

synonym: *S38*.

references: Spradling and Mahowald, 1979, Cell 16: 589-98.

Spradling, Waring, and Mahowald, 1979, Cell 16: 609-16.

Spradling, Digan, Mahowald, Scott, and Craig, 1980, Cell 19: 905-14.

Spradling and Mahowald, 1980, Proc. Nat. Acad. Sci. USA 77: 1096-1100.

Spradling, 1981, Cell 27: 193-201.

phenotype: Structural gene for s38-1, a 38,000-dalton chorion protein. Synthesis primarily during stages 12 and 13 (Waring and Mahowald, 1979, Cell 16: 599-607) and during stages 11 and 12 (Petri, Wyman, and Kafatos, 1976, Dev. Biol. 49: 185-199). 2,000 to 3,000 daltons cleaved from primary translation product to yield mature protein (Spradling *et al.*, 1980). Temporal and spatial distribution of expression described by Park and Spradling (1987, Genes Dev. 1: 497-509).

alleles: Isoelectric focusing resolves two electrophoretic forms of s38-1 which may be designated *Cp38^a* and *Cp38^b*.

cytology: Localized to 7F1-2 by *in situ* hybridization. About 1 kb proximal to *Cp36*.

molecular biology: Resides on the same transcription unit as *Cp36*; separated from it by 1414 bp; gene 1516 bp in length and contains a small 5' intron (Wakimoto); nascent transcripts contain a ribonucleoprotein particle at each splice junction (Olsheim, Miller, and Beyer, 1985, Cell 43: 143-51). Abundantly transcribed producing polyadenylated mRNA in follicle cells surrounding stage-12 and -13 oocytes. Abundance markedly reduced in *In(1)oc = In(1)7F1-2;8A1-2* homozygotes. cDNA and approximately 200 kb of genomic sequences in the region of *Cp38* cloned and restriction mapped. Direction of transcription is toward the centromere. Located in a chromosome region that is amplified approximately 14-16 fold as a series of nested bidirectional replication forks spanning approximately 100 kb in the follicle cells surrounding maturing oocytes; amplification of the entire

region under the control of *ACE3*, a *cis*-acting sequence between 654 and 266 base pairs upstream from *Cp38* (Wakimoto). Amplification disrupted by *In(1)oc* = *In(1)7F1-2;8A1-2*; female sterility results in homozygotes. Electron-microscope characterization of transcription unit, including length, polymerase II density, and discrete termination site by Olsheim, Miller, and Beyer (1986, EMBO J. 5: 3591-96).

Cp70

location: 1-{0}.

references: Yannoni and Petri, 1984, Dev. Biol. 102: 504-08.

phenotype: The structural gene for a 70-dalton minor component of the chorion.

alleles: Two electrophoretic alleles known.

cytology: Placed in 2B3-6 based on its inclusion in *Df(1)S39* = *Df(1)1E1-2;2B5-6*, but not in *Df(1)sta* = *Df(1)1E1-2;2B3-4* (Peterson and Petri, 1986, DIS 63: 158).

***cpl: cupola**

location: 1-0.0 (no crossing over with *sc* in 584 males).

origin: Induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1953.

references: 1959, DIS 33: 83-84.

phenotype: Small, inviable fly. Wings shorter and curved to form canopy over abdomen with tips converging toward mid-dorsal line. Head and eyes slightly deformed. Abdominal tergites abnormal; from irregular pigmentation to absence or gross deformation of the sixth and seventh tergites. Males sterile. RK3.

***Cpt: Clipt**

location: 2-43.7.

origin: Spontaneous.

discoverer: Sturtevant, 26b18.

phenotype: Bristles short, like those of *Sb*. Homozygous lethal. Male sterile. RK1.

***cpw: canopy wing**

location: 1-2.5 [not between *w* and *vt* (Lefevre and Green, 1972, Chromosoma 36: 391-412)].

origin: Induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 69.

phenotype: Wings short and very broad; longitudinal veins frequently do not reach wing margin and often diverge. Eyes large and slightly rough. Head bristles reduced in number (ocellars most frequently affected). Thorax broad, one or more bristles occasionally absent; hairs more widely separated, with noticeable hairless areas. Males sterile. Viability 40% wild type. RK3.

cq: see *rk*⁴

***cr: crisp**

location: 1- (not located).

discoverer: Agol.

references: 1936, DIS 5: 7.

phenotype: Bristles like forked. RK2.

other information: Not an allele of *f* or *sn*.

CR: Compensatory Response (K. Tartof)

location: 1-{66}. *CR* is located in the centric heterochromatin of the *X* chromosome distal to the tandem array of ribosomal RNA genes (rDNA). It lies between the heterochromatic breakpoints of *sc*⁴ (distally) and *w*^{md} and *rst*³ (proximally).

origin: Absence or dysfunction of this otherwise wild-type locus is detected by deletions or rearrangements of the heterochromatic interval indicated above.

references: Tartof, 1971, Science 171: 294-97.

Spear and Gall, 1973, Proc. Nat. Acad. Sci. USA 70: 1359-63.

Procunier and Tartof, 1978, Genetics 88: 67-79.

synonym: *cr*⁺.

phenotype: In females there are about 250 tandemly arrayed rRNA genes in each of the two *X* chromosome. However, when only a single *X* is present, as in *X/O* males or *X/In(1)sc*^{4L}*sc*^{8R} females, there are approximately 400 rRNA genes per *X*. This phenomenon, referred to as "compensation", results from the disproportionate replication of rDNA in somatic cells of adults and is controlled by the *CR* locus. *CR* is novel in that it exhibits both *trans* and contiguous-*cis* acting properties. It acts in *trans* to detect the presence of its partner locus in the homologue, and if that partner locus is absent, it acts in *cis* to drive the disproportionate replication of those rRNA genes that are contiguous with it. The observed disproportionate rDNA replication may occur primarily in polytene nuclei. *CR* is not required for the "magnification" or "reduction" of germ line rDNA.

cytology: Placed in 20F based on its location between the proximal breakpoints of *In(1)sc*⁴ to the left and *In(2)rst*³ and *In(1)w*^{md} to the right. This places *CR* in h26-29 on the heterochromatic map of Gatti and Pimpinelli.

***Cr-2: Cream in chromosome 2**

location: 2- (not located).

origin: Spontaneous.

discoverer: Bridges, 13i15.

references: 1919, J. Exp. Zool. 28: 337-84.

Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 239 (fig.).

phenotype: Specific dilutor of *w*^e. *w*^e; *Cr-2/Cr-2* has a pale-cream eye color. *w*^e; *Cr-2/+* has eye color between eosin and cream. RK3.

***cr-3: cream in chromosome 3**

location: 3-36.5.

origin: Spontaneous.

discoverer: E. M. Wallace, 14b27.

references: Bridges, 1919, J. Exp. Zool. 28: 337-84.

Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 112 (fig.).

phenotype: Homozygote has slightly diluted eye color. Eye color of *w*^e; *cr-3* cream. Larval Malpighian tubes of *w*^e; *cr-3* white, those of *cr-3* bright yellow (Brehme and Demerec, 1942, Growth 6: 351-56). RK3.

***cr-a: cream-a**

location: Autosomal, not located.

origin: Spontaneous.

discoverer: Bridges, 13g15.

references: 1916, Genetics 1: 147.

1919, J. Expt. Zool. 28: 337-84.

phenotype: Strong specific dilutor of *w*^e. RK3.

***cr-b**

location: 2-24.
origin: Spontaneous.
discoverer: Bridges, 14c10.
references: 1916, *Genetics* 1: 149.
 1919, *J. Exp. Zool.* 28: 337-84.
 Bridges and Morgan, 1919, *Carnegie Inst. Washington Publ. No. 278*: 245 (fig.).
phenotype: Specific dilutor of w^e . RK3.

***cr-c**

location: 2- (near *S*).
origin: Spontaneous.
discoverer: Bridges, 16g13.
phenotype: Weak specific dilutor of w^e . RK3.

cra: crack (C. Nüsslein-Volhard)

location: 2-77.
origin: Induced by ethyl methanesulfonate.
references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 267-82.
phenotype: Embryonic lethal. Defect in head involution. Shows abdominal transformations in combination with *Pc*-like mutants.
alleles: Two.

cramped: see *crm*

cramped-like: see *crm*²

crb: crumbs

location: 3-82.
origin: Induced by ethyl methanesulfonate.
references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95 (fig.).
phenotype: Homozygous lethal; many small holes in cuticle.
alleles: Six; one weak.
cytology: Placed in 95E-96A; covered by Y^{P_3D} element from $T(Y;3)HI73 = T(Y;3)Xhy^+$; 95E but not $T(Y;3)G73 = T(Y;3)YL;96A$.
molecular biology: Tentatively identified as a putative neurogenic gene based on hybridization of a cDNA clone with partial homology to *N* and *Dl* to 95F (Knust, Dietrich, Tepass, Bremer, Weigel, Vässin, and Campos-Ortega, 1987, *EMBO J.* 6: 761-66).

CRB: see $T(1;4)A1$

crc: cryptocephal

location: 2-55.
origin: Spontaneous.
discoverer: Hadorn, 1942.
synonym: $l(2)crc$.
references: Hadorn and Gloor, 1943, *Rev. Suisse Zool.* 50: 256-61.
 Gloor, 1945, *Arch. Julius Klaus-Stift. Vererbungsforsch. Sozialanthropol. Rassenhyg.* 20: 209-56.
 Fristrom, 1965, *Genetics* 52: 297-318.
 Sparrow and Chadfield, 1985, *Dev. Genet.* 5: 103-14 (fig.)
phenotype: Homozygotes frequently die as larvae; failure to eliminate chitinous mouthparts at the larval molt frequently results in larvae with supernumerary mouth parts; homozygotes undergo pupation but rarely eclose from puparia. Imaginal head is not everted from thorax.

Except for slightly reduced eyes and shortened legs, wings, and thoracic bristles, the head and thorax are fully differentiated; frequently fail to pass gas bubble to anterior retro-opercular position, thus failing to provide space for head eversion. Head eversion is inhibited by integument being more rigid than normal. Mutant integument contains more glucosamine than normal; Sparrow and Chaddfield unable to confirm (1982, *Dev. Genet.* 3: 235-45). Feeding glucosamine to wild-type larvae produces a phenocopy very similar to $l(2)crc$. Abdomen often shows no differentiation, and internal organ development is arrested at pupal stage. Lethality suppressed by $su(crc)l$ (Sparrow, 1981, *Genet. Res.* 38: 297-314). RK3.

cytology: Placed in 39C2-D12 based on its inclusion in $Df(2L)TW12 = Df(2L)37E2-F4;39D12$ but not in $Df(2L)TW1 = Df(2L)38A7-B1;39C2-3$ or $Df(2L)TW2 = Df(2L)37D2-E1;38E6-9$ (Wright, Hodgetts, and Sherald, 1976, *Genetics* 84: 267-85).

cream: see *cr*

Cream: see *Cr-2*

Cream: see bw^{V291}

cream underscored: see *cru*

creased: see *cs*

creeper: see rk^4

crib: cribble (T. Schüpbach)

location: 2-{57}.
origin: Induced by ethyl methanesulfonate.
references: Schüpbach and Wieschaus.
phenotype: Maternal-effect lethal mutant; Embryos from homozygous females cellularize irregularly at the blastoderm stage. Further development is very abnormal; at final differentiation embryos form only fragmented pieces of cuticle.
alleles: $crib^{HD} = crib^1, crib^{PA}, crib^{RS}$
cytology: Placed in 42C1-43F8, since uncovered by $Df(2R)pk78s = Df(2R)42C1-7;43F5-8$.

cricketlet: see *clt*

Crimp: see *Cm*

crinkled: see *ck*

***crip: cripple**

location: 2- (between *pr* and *cn*).
discoverer: Komai, 1924.
references: 1926, *Genetics* 11: 280-93.
 1927, *Mem. Coll. Sci. Univ. Kyoto, Ser. B* 2: 211-57.
phenotype: Middle and hind legs twisted and shortened. Thirty percent penetrance. RK3.

cripA: crippled-A (J.C. Hall)

location: 1-1.8.
origin: Induced by ethyl methanesulfonate.
references: Homyk and Sheppard, 1977, *Genetics* 87: 95-104.
phenotype: Adults walk slowly in uncoordinated manner, dragging one or more legs; legs weak and unable to support normal posture; flies weak; males court feebly and move wings up and down slowly instead of vibrating them.

cytology: Proximal to 2B11 owing to its not being deleted by *Df(1)RA19 = Df(1)IE3-4;2B11-12* nor carried by the terminal duplication $y^2Y67g = Dp(1;Y)2B6-7$ (Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbertova, Kramers, and Zhimulev, 1982, DIS 58: 184-90).

cripB (J.C. Hall)

location: 1-8.8.

origin: Induced by ethyl methanesulfonate.

references: Homyk, Szidonya, and Suzuki, 1980, Mol. Gen. Genet. 177: 553-65.

phenotype: Adults walk in uncoordinated manner; normal jumping difficult to induce; legs move to abnormal position during tethered flight; roughened eye phenotype.

cripC (J.C. Hall)

location: 1-57 (approximate).

origin: Induced by ethyl methanesulfonate.

references: Homyk, Szidonya, and Suzuki, 1980, Mol. and Gen. Genet. 177: 553-65.

phenotype: Running, climbing, jumping are uncoordinated and generally abnormal; all such defects seemingly caused by stiffness of legs (nonbending at tibia-tarsal joints); mutant males court in uncoordinated manner with abnormal wing display, e.g., extension of both wings simultaneously; wing beat abnormal in tethered flight, as is position of legs.

crisp: see *cr*

***crk: crooked setae**

location: 1-60.1.

origin: Induced by D-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3026).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 69.

phenotype: Bristles thin and slightly shortened; occasional missing scutellar. Acrostichals deranged. Abdominal hairs of female frequently missing; tergites occasionally abnormal. Classification difficult. Viability and fertility good. RK3.

other information: One allele induced by L-p-N,N-di-(2-chloroethyl)amino-phenylalanine.

***crm: cramped**

location: 1-1.48 (based on *crm^{sa}* mapping 0.02 unit to the left of *w*).

origin: Induced by P32.

discoverer: Bateman.

synonym: *sta^P: stubarista* from P32; *cramped-like* (*crm²*).

references: 1951, DIS 25: 78. 1953, DIS 27: 55.

phenotype: Antennae swollen, arista short, and branches fewer than in wild type. Wings slightly divergent and in some alleles nicked. Males have small extra sex combs on second tarsal segment of prothoracic legs and less frequently on basitarsal segments of mesothoracic and rarely metathoracic legs. *crm^{sa};Pc/+* males have large sex combs on all six basitarsi and small sex combs on all second tarsal segments in many flies. One or both post-ventricles lacking and other bristles occasionally missing or doubled. Body slightly darkened. Fertility of both males and females reduced, females more severely than males. Strength of sterilizing effect allele dependent. Females generally sterile; surviving *crm¹* females have

rudimentary ovaries with no oocytes beyond stage 7 (Shannon, Kaufman, Shen, and Judd, 1972, DIS 48: 92); males of *crm²* and *crm⁷* partially fertile, other alleles male sterile. Viability variably reduced depending on crowding in culture; *crm⁴*, *crm⁷* are semilethal alleles, *crm⁶* die in late third instar; survivors eclose on days 11-13; rarely survive in combination with a deficiency (Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56; Shannon *et al.*, 1972).

alleles: Independently discovered and named mutants with the same phenotype and map position have been designated *l(1)zw⁹*, *sa*: sparse arista, and *swa*: swollen antennae. These are now designated alleles of *crm*. Designated alleles are tabulated below.

allele	origin	discoverer	synonym	ref ^α	comments
<i>crm¹</i>	32 P	Bateman	<i>sta^P</i>	1	
<i>*crm²</i>	CB3025	Fahmy, 1953		1	
<i>crm³</i>	β	X ray	<i>l(1)3C1^{65g}</i>	2	in <i>ln(1)w^{m51bL_wmJR}</i>
<i>crm⁴</i>	β	X ray	<i>l(1)3C1^{65l}</i>	2	in <i>ln(1)w^{m51bL_wmJR}</i>
<i>crm⁵</i>	X ray		<i>l(1)zw⁹l⁴</i>	6	
<i>crm⁶</i>	X ray		<i>l(1)zw⁹lk17</i>	6	
<i>crm⁷</i>	X ray		<i>l(1)zw⁹lk18</i>	6	
<i>crm⁸</i>	spont	Schalet	<i>l(1)17-15</i> <i>l(1)zw⁹S1</i>		
<i>crm⁹</i>	EMS	Lefevre	<i>l(1)DA670</i>	3	
<i>crm¹⁰</i>	EMS	Lefevre	<i>l(1)VA54</i>	3	
<i>crm^{sa}</i>	spont		<i>sa</i>	4, 5	
<i>crm^{sa2}</i>	spont		<i>sa²</i>	4, 5	
<i>crm^{swa}</i>	CB1506 β	Fahmy, 1956	<i>swa</i>	1	

^α 1 = CP627; 2 = Gersh, 1967, Genetics 56: 309-19; 3 = Lefevre; 4 = Rayle, 1968, DIS 43: 62; 5 = Rayle and Green, 1969, Genetica 39: 497-507; 6 = Shannon, Kaufman, Shen, and Judd, 1972, DIS 48: 92.

^β Allelism inferred from missing-bristle phenotype of escapers; could be allelic to *l(1)3Bf* instead.

cytology: Placed in the left edge of or to the left of 3C1 based on the failure of *Df(1)wr-J1 = Df(1)3A1-2;3C2-3* but not *Df(1)X2 = Df(1)2F6-3A1;3B5-C1* to complement *crm¹* (Judd, Shen, and Kaufman, *ibid.*) and the failure of *Df(1)w70e7 = Df(1)3B5-C1;3C1-2* [Sorsa, Green, and Beermann, 1973, Nature (London) New Biol. 245: 34-37] to delete *crm*.

crn: crooked neck

location: 1-{0.95}.

synonym: *l(1)2Fa*.

references: Gvozdev, Gerasimova, Kovalev, and Ananiev, 1977, DIS 52: 67-68.

Perrimon, Engstrom, and Mahowald, 1984, Genetics 111: 23-41 (fig.).

phenotype: Hemizygous embryos from heterozygous mothers show twisted phenotype; *crn³/Df(1)64c18* and *crn⁴/Df(1)64c18* exhibit a similar but more extreme phenotype, suggesting that they are hypomorphic. Both alleles lethal in female germ-line clones.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>crn¹</i>	X ray	Lefevre	<i>l(1)A35</i>	4	
<i>crn²</i>	X ray	Lefevre	<i>l(1)HC208</i>	4	
<i>crn³</i>	X ray	Lefevre	<i>l(1)RC63</i>	2, 4, 6	2.5 kb insert at coordinate +66
<i>crn⁴</i>	EMS	Lefevre	<i>l(1)EA130</i>	5, 6	heterochromatic rearrangement
<i>crn⁵</i>	EMS	Gvozdev	<i>l(1)N7²</i>	1	
<i>crn⁶</i>	EMS	Gvozdev	<i>l(1)N7⁸</i>	1	
<i>crn⁷</i>	EMS	Gvozdev	<i>l(1)N7³⁰</i>	1	

allele	origin	discoverer	synonym	ref ^α	comments
<i>crn</i> ⁸	EMS	Gvozdev	<i>l(1)N7</i> ⁵¹	1	
<i>crn</i> ⁹	EMS	Gvozdev	<i>l(1)N7</i> ⁵⁹	1	
<i>crn</i> ¹⁰	EMS	Gvozdev	<i>l(1)N7</i> ⁶⁶	1	
<i>crn</i> ¹¹	EMS	Gvozdev	<i>l(1)N7</i> ⁷⁶	1	
<i>crn</i> ¹²	EMS	Gvozdev	<i>l(1)N7</i> ⁸³	1	
<i>crn</i> ¹³	EMS	Gvozdev	<i>l(1)N7</i> ⁸⁵	1	
<i>crn</i> ¹⁴	EMS	Gvozdev	<i>l(1)N7</i> ⁹¹	1	
<i>crn</i> ¹⁵	EMS	Gvozdev	<i>l(1)N7</i> ⁹²	1	
<i>crn</i> ¹⁶	γ ray	Gvozdev	<i>l(1)N7</i> ¹⁰⁶	1	
<i>crn</i> ¹⁷	HMS β		<i>l(1)HM23</i>	3	
<i>crn</i> ¹⁸	HMS		<i>l(1)HM27</i>	3	
<i>crn</i> ¹⁹	HMS		<i>l(1)HM433</i>	3	

^α 1 = Gvozdev, Gerassimova, Kovalef, and Ananiev, 1977, DIS 52: 67-68; 2 = Haenlin, Steller, Pirrotta, and Mohier, 1985, Cell 40: 827-37; 3 = Kramers, Schalet, Paradi, and Huiser-Hoogteyling, 1983, Mutation Res. 107: 187-201; 4 = Lefevre, 1981, Genetics 99: 461-80; 5 = Lefevre; 6 = Perrimon, Engstrom, and Mahowald, 1984, Genetics 108: 559-72.

^β HMS = hycanthon methanesulfonate.

Failure of HMS-induced mutants to complement both *crn*³ and *crn*⁷ established allelism among the three series.

cytology: Placed in 2F1 based on its inclusion in *Df(1)278.4B1 = Df(1)2E3-F3;3A5-B4* but not *Df(1)2F1-3A4*.

molecular biology: Localized by transformation to a subclone of a component cosmid of a 200 kb walk initiated from microdissected polytene chromosomes. *crn* 65-70 kb to the right of the arbitrarily chosen initiation point of the walk; probes from the region identify a 2.1 kb transcript on Northern blots (Haenlin, Steller, Pirrotta, and Mohier, 1985, Cell 40: 827-37).

Crn: Crown

location: 3-46.8 (0.1 map unit to left of *in*).

origin: Induced by ethyl methanesulfonate.

discoverer: E.H. Grell

phenotype: Dark trident pattern in heterozygote. Homozygous lethal.

cro: see *ptg*³

crooked: see *fw*^c

crooked neck: see *crn*

crooked setae: see *crk*

Crossover suppressor: see *C()*

crossveinless: see *cv*

crown: see *ptg*³

crs: cru sterile

location: 2- (between *px* and *bw*).

discoverer: Muller.

references: 1951, DIS 25: 119. 1955, DIS 29: 146.

phenotype: Male sterile. RK2.

cytology: Located between 58E3 and 59A2 on basis of sterility in combination with *Df(2R)P + Dp(2;Y)bw⁺ = Df(2R)58E3-F1;60D14-E2 + Dp(2;Y)Y^L;58F1-59A2;60D14-E2* (Muller, 1955).

other information: Male sterility formerly associated with but separable from *cru*.

crt: crumpled tips

location: 1-40.3 (7.3 units from *v*, based on 3035 flies).

origin: Induced by triethylenemelamine (CB. 1246).

discoverer: Fahmy, 1952.

references: 1959, DIS 33: 84.

phenotype: Wing tips frequently shriveled, pleated, or crumpled and often turned up or down. Wings vary from completely unexpanded to wild type. Viability and fertility good in both sexes. Failure of *crt*² to produce homozygous germ-line clones reported by Perrimon, Engstrom, and Mahowald (1989, Genetics 121: 333-52). RK2.

alleles: *crt*², induced by ethyl nitrosourea by Scott (PhD Thesis, 1987, University of California, San Diego), considered to be allelic based on similarity in phenotype and genetic position. Twelve other alleles: One each induced by X rays, triethylenemelamine, 2-fluoroethyl methane-sulfonate, and L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine; two induced by *p*-N,N-di-(2-chloroethyl)-amino-phenylethylamine; three each induced by S-2-chloroethylcysteine, and DL-*p*-N,N-di-(2-chloroethyl)-amino-phenylalanine.

cytology: Placed in region 11F2 through 12A2 based on its inclusion in *Df(1)C246 = Df(1)11D1-2;12A1-2* but not *Df(1)wy2 = Df(1)11A6-12;11F2-4* (Scott).

cru: cream underscored

location: 2-52.5.

origin: Spontaneous.

discoverer: Bridges, 20a5.

phenotype: Specific dilutor of *w^e* and *P*. Slight dominant but used as a recessive. Originally thought to be male sterile, but this was caused by a factor in *2R*, *crs*. Larval Malpighian tubes of *w^e*; *cru* colorless; those of +; *cru* bright yellow (Brehme and Demerec, 1942, Growth 6: 351-56). RK3.

cytology: Not uncovered by *Df(2L)64j = Df(2L)34D1-2;35B9-C1*, *Df(2L)75c = Df(2L)35A1-2;35D4-7*, or *Df(2L)fn1 = Df(2L)34F4-35A1;35D5-7*. Presumably proximal to 35D5 (Woodruff and Ashburner, 1979, Genetics 92: 117-32).

crumbs: see *crb*

crumpled: see *cmp*

crumpled tips: see *crt*

crumpled wings: see *cwl*

cru sterile: see *crs*

cryptocephal: see *crc*

cs: creased

location: 1-56.

origin: X ray induced.

discoverer: K. C. Atwood, 41i.

references: 1942, DIS 16: 47.

phenotype: Wings longitudinally creased in first posterior cell from distal end of L3 virtually to anterior crossvein. Fertility and viability good. RK1.

alleles: *cs*⁵³ (CP627).

csk: costakink

location: 1-33.0.

phenotype: Most alleles are lethal; originally described on basis of a single surviving semilethal allele which

displayed smaller eyes, with wings slightly reduced in size and abnormally held, with the costal vein frequently kinked near L2. Not fully penetrant; male viability and fertility good; female fertility reduced to about 50% wild type. Survivors of other semilethal or nearly lethal alleles display similar phenotype (Zhimulev, Pokholkova, Bgatov, Umbetova, Solovjeva, Khudyakov, and Belyaeva, 1987, Biol. Zentralbl. 106: 699-720). Lethal allele *csk*³³, functions in homozygous germ-line clones, but has no maternal effect (Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-52). Fate maps, as a bilateral domineering mutant, to a broad region of the neurogenic ectoderm posterior to the metathoracic ganglion; in addition a nonautonomous focus in the wing disc gives rise to crumpled turned up wings (Bgatov, Zharkikh, and Zhimulev, 1984, Mol. Gen. Genet. 196: 110-16).

allele	origin	discoverer	synonym	ref ^α	comments
<i>*csk</i> ¹	TEM	Fahmy, 1953		1	viable allele
<i>*csk</i> ²	X ray	Fahmy, 1953		1	viable allele
<i>csk</i> ³	EMS	Belyaeva	<i>l(1)162</i>	6, 7, 8	heat-sensitive semilethal
<i>csk</i> ⁴	EMS	Belyaeva	<i>l(1)166</i>	6, 7, 8	heat-sensitive semilethal
<i>csk</i> ⁵	EMS	Belyaeva	<i>l(1)E109</i>	6, 7, 8	heat-sensitive semilethal
<i>csk</i> ⁶	EMS	Belyaeva	<i>l(1)E115</i>	6, 7, 8	
<i>csk</i> ⁷	EMS	Belyaeva	<i>l(1)E128</i>	6, 7, 8	
<i>csk</i> ⁸	EMS	Belyaeva	<i>l(1)E143</i>	6, 7, 8	
<i>csk</i> ⁹	EMS	Belyaeva	<i>l(1)E147</i>	6, 7, 8	
<i>csk</i> ¹⁰		Pokholkova	<i>l(1)F57</i>	9	heat-sensitive semilethal
<i>csk</i> ¹¹		Pokholkova	<i>l(1)F69</i>	9	heat-sensitive semilethal
<i>csk</i> ¹²		Pokholkova	<i>l(1)F97</i>	9	heat-sensitive semilethal
<i>csk</i> ¹³		Pokholkova	<i>l(1)F98</i>	9	heat-sensitive semilethal
<i>csk</i> ¹⁴		Pokholkova	<i>l(1)F101</i>	9	heat-sensitive semilethal
<i>csk</i> ¹⁵		Pokholkova	<i>l(1)F124</i>	9	heat-sensitive semilethal
<i>csk</i> ¹⁶		Pokholkova	<i>l(1)F128</i>	9	heat-sensitive semilethal
<i>csk</i>		Pokholkova	<i>l(1)F305</i>	9	
<i>csk</i>		Pokholkova	<i>l(1)F404</i>	9	
<i>csk</i>		Pokholkova	<i>l(1)F407</i>	9	
<i>csk</i>		Pokholkova	<i>l(1)F411</i>	9	
<i>csk</i>		Pokholkova	<i>l(1)F436</i>	9	heat sensitive semilethal
<i>csk</i> ¹⁷	EMS	Pokholkova	<i>l(1)G50</i>	6, 7, 8	
<i>csk</i> ¹⁸	EMS	Pokholkova	<i>l(1)G92</i>	6, 7, 8	heat sensitive semilethal
<i>csk</i> ¹⁹	EMS	Pokholkova	<i>l(1)G99</i>	6, 7, 8	
<i>csk</i> ²⁰	EMS	Pokholkova	<i>l(1)G130</i>	6, 7, 8	
<i>csk</i> ^{21Y}	EMS	Pokholkova	<i>l(1)dpG1</i>	6, 7, 8	on <i>v</i> ⁺ <i>Yy</i> ⁺
<i>csk</i> ²²	EMS	Khudyakov	<i>l(1)J20</i>	6, 7, 8	
<i>csk</i> ²³	EMS		<i>l(1)v88</i>	2	larval lethal
<i>csk</i> ²⁴	EMS		<i>l(1)v301</i>	2	
<i>csk</i> ²⁵	EMS		<i>l(1)v318</i>	2	
<i>csk</i> ^{26Y}	EMS		<i>l(1)v370</i>	2	on <i>v</i> ⁺ <i>B</i> ^{S-} <i>Yy</i> ⁺
<i>csk</i> ²⁷	X ray	Lefevre	<i>l(1)GA54</i>	3	
<i>csk</i> ²⁸	X ray	Lefevre	<i>l(1)HF319</i>	3	hetab, <i>sev</i> mutant
<i>csk</i> ²⁹	X ray	Lefevre	<i>l(1)L68</i>	3, 5	
			<i>l(1)RC68</i>		
<i>csk</i> ³⁰	X ray	Lefevre	<i>l(1)N43</i>	3	euchab
<i>csk</i> ³¹	EMS	Lefevre	<i>l(1)DA651</i>	4	
<i>csk</i> ³²	EMS	Lefevre	<i>l(1)DC835</i>	4	
<i>csk</i> ³³	EMS	Lefevre	<i>l(1)DF923</i>	4	P/NME
<i>csk</i> ³⁴	EMS	Lefevre	<i>l(1)EA46</i>	4	
<i>csk</i> ³⁵	EMS	Lefevre	<i>l(1)VA28</i>	4	
<i>csk</i> ³⁶	EMS	Lefevre	<i>l(1)VE710</i>	4	
<i>csk</i> ³⁷	EMS	Lefevre	<i>l(1)VE793</i>	4	

^α 1 = Fahmy, 1958, DIS 32: 69; 2 = Geer, Lischwe, and Murphy, 1983, J. Exp. Zool. 225: 107-18; 3 = Lefevre, 1981, Genetics 99: 161-80; 4 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 5 = Lefevre and Wiedenheft, 1974, DIS 51: 83; 6 = Zhimulev, Belyaeva, Pokholkova, Kotchneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baricheva, Aizenzon, Kramers, and Eeken, 1981, DIS 56: 192-96; 7 = Zhimulev *et al.*, 1981, Chromosoma 82: 25; 8 = Zhimulev, Belyaeva, Pokholkova, Kochneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baricheva, Aizenzon, Kramers, Eeken, 1982, DIS 58: 210-14; 9 = Zhimulev, Pokholkova, Bgatov, Umbetova, Solovjeva, Khudyakov, and Belyaeva, 1987,

Biol. Zentralbl. 106: 699-720.

cytology: Allelism inferred from observation that both samples of alleles map to the region of overlap between *Df(1)v-L1* and *Df(1)v-L2*. Survivors carrying semilethal alleles exhibit *csk* phenotype. Some surviving heteroallelic combinations display late eclosion and small pale bristles.

csw: corkscrew

location: 1-{0.55}.

synonym: 1(1)2Db.

references: Perrimon, Engstrom, and Mahowald, 1985, Genetics 111: 23-41 (fig.).

phenotype: Hemizygotes die at the larval-pupal transition; dissected third-instar larvae display small imaginal disks. Larval neuroblast cells show low mitotic index, but chromosome morphology appears normal. Homozygous germ-line clones produce inviable embryos with U-shaped or twisted phenotypes, which are unaffected by paternal genotype or developmental temperature. *csw*⁶ is viable and fertile with rough eyes.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>csw</i> ¹	X ray	Lefevre	<i>l(1)C114</i>	2, 4	euchromatic rearrangement
<i>csw</i> ²	X ray	Lefevre	<i>l(1)GA113</i>	2	<i>T(1;2)2C;49A</i> (complex)
<i>csw</i> ³	X ray	Lefevre	<i>l(1)KC16</i>	2	<i>T(1;2)2D5;52F</i>
<i>csw</i> ⁴	X ray	Lefevre	<i>l(1)SS3</i>	2	<i>T(1;2;3)2D5;57E;86B</i>
<i>csw</i> ⁵	EMS	Lefevre	<i>l(1)VA199</i>	3, 4	
<i>csw</i> ⁶			<i>l(1)15086</i>		viable and fertile
<i>csw</i> ⁷	spont	Schalet	<i>l(1)19-106</i>		
<i>csw</i> ⁸	X ray			1	
<i>csw</i> ⁹	X ray			1	
<i>csw</i> ¹⁰	X ray			1	
<i>csw</i> ¹¹	EMS			1	
<i>csw</i> ¹²	EMS			1	
<i>csw</i> ¹³	EMS			1	viable

^α 1 = Dura, Randsholt, Deatrck, Erk, Santamaria, Freeman, Freeman, Weddell, and Brock, 1987, Cell 51: 829-39; 2 = Lefevre, 1981, Genetics 99: 461-80; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 4 = Perrimon, Engstrom, and Mahowald, 1985, Genetics 111: 23-41.

cytology: Placed in 2D3-4 based on its inclusion in *Df(1)pn38 = Df(1)2D3-4;2E3* but not *Df(1)Pgd-kz = Df(1)2D3-4;2F5*.

ct: cut

location: 1-20.0.

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 35, 223 (fig.).

Lefevre and Johnson, 1973, Genetics 74: 633-45.

Johnson and Judd, 1979, Genetics 92: 485-502.

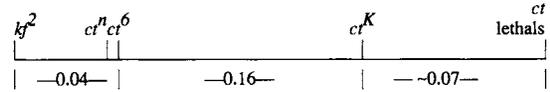
Jack, 1985, Cell 42: 869-76.

phenotype: *ct* mutations fall into three nonoverlapping phenotypic classes: kinked femur, cut wings, and lethal. Kinked-femur mutants are small with slightly dark, dull, red eye color; femurs kinked; wings seldom expand following eclosion, or when they do expand they are opaque and abnormal in shape; flies seem unable to move normally and die on the food soon after eclosion. Cut-wing mutants variably affect wing shape and head capsule development; phenotypic effects include incised wing margins with the tips usually cut to points, missing or ventrally displaced vibrissae, deformed antennae, *e.g.*, flattened and embedded with arista concave forward,

smaller kidney-shaped eyes, warped abdominal bands, and fine bristles. Most lethal alleles survive as clones of homozygous epidermal cells (Demerec). Developmental study of *ct*⁶ by Waddington [1939, Proc. Nat. Acad. Sci. USA 25: 299-308; 1940, J. Genet. 41: 75-139 (fig.)] shows wing bud narrower than wild type as early as just after eversion of wing in early pupa. Cell death observed in prepupal wing bud (D. Fristrom, 1969, Mol. Gen. Genet. 103: 363-79). Clones of *ct*⁶ cells in internal areas of wing blade normal in size; marginal clones much reduced in size indicating cell death. Homozygous clones in either dorsal or ventral membrane must reach margin in order to produce incision, 100/127 marginal clones unassociated with gaps; when gaps are produced, they affect both wing surfaces even though clone confined to a single surface. Both dorsal and ventral chaetal elements at the edges of such gaps may show the markers of such clones (Santamaria and Garcia-Bellido, 1975, Wilhelm Roux's Arch. Entwicklungsmech. Org. 178: 233-45). Lethal alleles fall into three groups, based on their complementation characteristics: cutless, group I, and group II. Lethal alleles *ct*^{C145}, *ct*^{JA124}, and *ct*¹⁴⁹ exhibit polyphasic lethality from late embryo to pharate adult (Johnson and Judd, 1979). Lethal embryos characterized by posterior defects in spiracles; no Keilin's organs, and abnormal maxillary complex (Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307). Group II and to a slightly lesser degree group I lethals fail to differentiate external sensory neurons in the peripheral nervous system; the presumptive external sensory neurons of the embryonic peripheral nervous system and their support cells are transformed into chordotonal neurons with their support cells; the transformed organs are chordotonal both in morphology and antigenic specificity. Same effect seen in the adult sensory organs in mosaics; embryonic effect differs from that seen in adults in that embryos lack peripheral sensory structures, e.g., Keilin's organs, whereas such structures persist, though reduced in size, in adult tissue. The numbers and positions of peripheral neurons is normal. CNS structure and function appear normal. No discernable effect of absence of *ct* function in the maternal germ line. Effect of *ct* mutations on PNS differentiation cell autonomous. [Bodmer, Barbel, Sheperd, Jack, Jan, and Jan, 1987, Cell 51: 293-307 (fig.)] Antibodies to *ct* protein specifically bind to nuclei of presumptive external sensory organ cells including those of the antennamaxillary organ and external sensory organs in spiracles, but not to nuclei of chordotonal organs; antibody staining also seen in some neurons with multiple dendritic arborizations and in cells lining the Malpighian tubules (Blochlinger, Bodmer, Jack, Jan, and Jan, 1988, Nature 333: 629-35).

Kinked-femur, cut-wing, and cutless alleles are mutually complementing: group I lethals complement kinked-femur but not cut-wing alleles; and group II lethals are noncomplementing; all combinations of lethal alleles are lethal. The different phenotypic classes of alleles occupy discrete and separate regions of the complex, with the order from left to right being, kinked femur, cut wing, group I lethals, and group II lethals; cutless alleles have not been mapped. Kinked femur, cut, and group-I-lethal mutations are associated with chromosome aberrations or insertions of transposable elements,

whereas group II lethals appear to be point mutations. *ct*⁶, *ct*^{68E} (= *ct*^{67s2}), *ct*^{78a}, and *ct*^K suppressed by *su(Hw)*²; *dvr*² enhances *ct*⁶ and inhibits its complete suppression by *su(Hw)*²; *su(Hw)*^{2/+} shows slight dominant suppression of wing phenotype of *ct*^K (Lee, 1973, Aust. J. Biol. Sci. 26: 903-09). *ct*⁶ and *ct*^K strongly enhanced by *su(s)*; *su(s)* *ct*^K lethal (Johnson) but rescued by *su(Hw)*^{2/+} (Craymer). *ct*⁶ the most commonly used allele.



Genetic fine structure map of the cut locus

alleles: *ct* is among the most mutable X-linked genes; several large-scale mutagenesis experiments have yielded many alleles, the majority of which are lost. The majority of induced *ct* alleles are lethal, or associated with chromosome rearrangements, or both. X-ray-induced alleles superscripted *2a2, *2a3, *2c1, *3a2, *3b1, 4b1, *4c1, *6a1, *7a1, *7a2, *7b2, *7c1, *7c2, *9b1, *9b2, *10a1, *10b1, *10c1, *11a, *12a1, *12a2, *12c1, *12c2, *13a2, *13b1, *14a1, *14a2, *14a3, *14b1, *14b2, *14c1, 15b1, and *15b4 described by Hannah (Proc. Int. Congr. Genet. 8th, 1949, 588-89; 1971, Mol. Gen. Genet. 113: 191-203; CP627). Of a similar set of 28 alleles with superscripts between *268-1 and 268-42 induced with X rays by Demerec or Hoover (CP552 and 627), only *Df(1)ct*²⁶⁸⁻⁴² persists. Early alleles superscripted *2 through *21 (CP552 and 627) are lost with the exception of *ct*⁶. Other early alleles are superscripted *34a, *34b, *36b, *41c23, *41i30 (CP552 and 627), *43aH1, 46l, *50e, *62a, *62f, *do-vg, *n4, *n36, and *So (CP627). Information on *ct* alleles that persist or that have been described since CP627 and on *kf*: kinked femur, which is a member of the cut complex, is tabulated below. *ct*^{cl}: cut-cutless complements various non-lethal *ct* alleles but fails to complement lethal alleles except *ct*^{HA46} (Lefevre and Leeds, 1983, Genetics 104: s45-46). A derivative of *ct*⁶ (termed *Uc*: Unstable chromosome) studied by Lim (1979, Genetics 93: 681-701) and *ct*^{MR2} derived from a hybrid dysgenic cross involving *MR-h12* on chromosome 2 by Gerasimova (1981, Mol. Gen. Genet. 184: 544-547) are highly unstable. The instability is manifest as increased incidence of lethal mutations, many of which are associated with chromosome aberrations broken in 6F (Lim), high reversion frequencies often accompanied by mutation at other loci. (Gerasimova, 1983, Mol. Gen. Genet. 190: 390-93; Gerasimova and Ilyin, 1984, DIS 60: 111-112) to either stable, unstable (most frequent, →10⁻⁴ mutations to *ct*), or superunstable (least frequent, → around 50% *ct* mutants) alleles, and to other *ct* alleles, which in turn may be unstable or superunstable. In both instances the *gypsy* sequence [called *L* by Lim, Simmons, Raymond, Cox, Doll, and Culbert (1983, Proc. Nat. Acad. Sci. USA 80: 6624-27) and *mdg4* by Gerasimova, Ilyin, Mizrokhi, Semjonova, and Gregoriev (1984, Mol. Gen. Genet. 193: 488-92)] is inserted into the *ct* locus. The *gypsy* sequence appears to be mobilized and transposed into other parts of the *ct* locus to produce new *ct* alleles or, when *ct* undergoes reversion, to other posi-

allele	origin	discoverer	ref ^α	phenotype ^β	comments	DNA coordinates ^γ
ct 1	spont	Bridges, 15j12	3	wa		
ct 6	gypsy	Bridges, 20c20	2, 3, 10, 11	wav	cytology normal	-1.1 to -0.5
ct 7						
ct 43aH1	X ray		3, 23	1	<i>In(1)4B1-4;7B4-C1 +</i> <i>In(1)10D5-6;20B-C</i>	
ct 461	X ray		3, 10, 22	w		B104 insert at -5.6
ct 53d	EMS		10	w		0.5 kb deletion in -6.8 to -4.8
ct 68E			13			
ct 71g	X ray	Johnson	10, 14	fw	<i>In(1)dl-49</i>	ζ
ct 71j	X ray	Johnson	10, 14	w	<i>In(1)dl-49</i>	
ct 78a	gypsy		10	wa		-1.1 to -0.5
ct 81/1	gypsy		10, 21	w		0 to +19.3
ct 149	EMS	Johnson	10, 11	1	cytology normal	
ct 152	EMS	Johnson	10, 11	1	cytology normal	
ct 161	EMS	Johnson	10, 11	1	cytology normal	
ct 268-42	X ray		3	1	<i>T(1;3)7B;80</i>	
ct cl δ	X ray		15	1		
ct cl1 δ	X ray	Lefevre	17	δ		
ct cl2 δ	EMS	Lefevre	17	δ		
ct C75	X ray	Lefevre	11		<i>T(1;2)7B3-4;35C</i>	
ct C145	X ray	Lefevre	2, 10, 11		cytology normal	
ct DA639	EMS	Lefevre	17			
ct db1	DEB		1	1		
ct db2	DEB		1	1	molecular lesion 130 to 150 kb	
ct db3	DEB		1	1		
ct db4	DEB		1	1		
ct db5	DEB		1	1		
ct db6	DEB		1	1	molecular lesion 130 to 150 kb	
ct db7	DEB		1	1	1 kb deletion in 130 to 150 kb	
ct db8	DEB		1	1		
ct db9	DEB		1	1		
ct db10	DEB		1	1		
ct db11	DEB		1	1		
ct db12	DEB		1	1		
ct db13	DEB		1	1		
ct EA2	EMS	Lefevre	17			
ct EA127	EMS	Lefevre	17			
ct EC234	EMS	Lefevre	17			
ct EF404	EMS	Lefevre	17			
ct F928	EMS	Lefevre	17			
ct GA83	X ray	Lefevre	16		<i>In(1)7A1;7B3</i>	
ct GA86	X ray	Lefevre	16		<i>T(1;2)7B3;42A-B</i>	
ct GE253	X ray	Lefevre	16		<i>In(1)7E3;7E-F1</i>	
ct HA46	X ray	Lefevre	15	1	<i>In(1)7B1-2;8D5</i>	break at -32 kb
ct HA79	X ray	Lefevre	16		<i>In(1)A7-8;7B3-4</i>	
ct HC211	X ray	Lefevre	16			
ct HC265	X ray	Lefevre	16		<i>In(1)1A2-3;7B3-4</i>	
ct HF357	X ray	Lefevre	16		<i>Tp(1;3)7B2;20;75A</i>	
ct J1	X ray	Johnson	14	1	<i>In(1)dl-49 +</i> <i>T(1;2)7B3-4;60E</i>	
ct J2	X ray	Johnson	14	1	<i>In(1)dl-49 +</i> <i>T(1;3)7B3-4;96A</i>	
ct J3	X ray	Johnson	14	1	<i>In(1)dl-49 +</i>	
ct J5	X ray	Johnson	14	1	<i>In(1)dl-49 +</i> <i>T(1;2)7B3-4;40</i>	
*ct J7	X ray	Johnson	14	1 ⁺	<i>In(1)dl-49 +</i> <i>In(1)7B3-4;7D22</i>	
ct J8	X ray	Johnson	14	1	<i>In(1)dl-49 +</i> <i>T(1;3)4D1;7B3-4;92A</i>	
*ct J9	X ray	Johnson	14	1	<i>In(1)dl-49 +</i> <i>T(1;3)7B3-4;86D-E</i>	
ct J10	X ray	Johnson	14	1	<i>In(1)dl-49 +</i>	
ct J11	X ray	Johnson	14	1	<i>In(1)dl-49 +</i> <i>T(1;3)7B3-4;95F</i>	
ct J12	X ray	Johnson	14	1	<i>In(1)dl-49 +</i> <i>In(1)7B3-4;7D4</i>	
ct J13	X ray	Johnson	14	1	<i>In(1)dl-49 +</i> <i>In(1)5A;7B3-4</i>	
*ct J14	X ray	Johnson	14	1	<i>In(1)dl-49</i>	
ct J15	X ray	Johnson	14	1	<i>In(1)dl-49</i>	
ct J16	X ray	Johnson	14	1	<i>In(1)dl-49</i>	
ct JA11	X ray	Lefevre	16		<i>In(1)7B;7E5</i>	
ct JA109	X ray	Lefevre	16	1		
ct JA120	X ray	Lefevre	16		<i>In(1)7B3;11A3-5</i>	

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	ref ^α	phenotype ^β	comments	DNA coordinates ^γ
ct JA124	X ray	Lefevre	10, 11, 16	1	cytology normal	
ct JA134	X ray	Lefevre	16		<i>T(1;2)7B3-4;32F-33A</i>	
ct JC20	X ray	Lefevre	2, 10, 11, 16	1 ⁺ fuwavb	cytology normal	ζ
ct K	gypsy	Krivshenko	2, 3, 10, 11	wbll ⁵	cytology normal	+70.8 to +73.3
ct J	EMS		20	1	<i>ln(1)dl-49</i>	
ct L1	gypsy	Lim	2, 10, 18	1		+70.8 to +73.3
ct L5	gypsy	Lim	10, 18	1		+70.8 to +73.3
ct L7	gypsy	Lim	10, 18	1		+70.8 to +73.3
ct L10	gypsy	Lim	10	wv		-1.1 to -0.5
ct L13	gypsy	Lim	10	1		
ct L18	gypsy	Neuwald	10	wv		0 to +19.3
ct L20	gypsy	Neuwald	10	wv		0 to +19.3
ct L23	gypsy	Lim	10	1		
ct L25	gypsy	Lim	10	1		
ct L27	gypsy?	Lim	10	1		
ct L30	gypsy	Lim	10	wv		0 to +19.3
ct L31	gypsy?	Lim	10	1		
ct L32	gypsy	Lim	10	wv		+19.3
ct L33	gypsy?	Lim	10	1		
ct L34	gypsy	Lim	10	wv		0 to +19.3
ct L35	gypsy	Lim	2, 10	1		+70.8 to +73.3
ct L36	gypsy	Lim		1		
ct L37	gypsy	Lim	10	1		-1.1 to -0.5
ct L39	gypsy	Lim	2, 10	1		+40
ct L41	gypsy	Lim	10	1		+70.8 to +73.3
ct L44	gypsy	Lim	10	wv		0 to +19.3
ct L45	gypsy	Lim	10	1		+70.8 to +73.3
ct L47	gypsy	Lim	10	1		+70.8 to +73.3
ct L49	gypsy	Lim	10	1		+70.8 to +73.3
ct L51	gypsy	Lim	10	1		
ct L53	gypsy	Lim	10	1		+70.8 to +73.3
ct L54	X ray	Lefevre	16		<i>T(312)96A;7B3-4</i>	
ct L55	gypsy	Lim	10	1		
ct L57	gypsy	Lim	10	1		
ct L59	gypsy	Lim	2, 10	1		36
ct L60	gypsy	Lim	10	wv		0 to +19.3
ct L61	gypsy	Lim	10	1		+70.8 to +73.3
ct L62	gypsy	Lim	10	wv		0 to +19.3
ct L64	gypsy	Lim	10	w		-1.1 to -0.5
ct L65	spont	Lim	2, 10	1		+70.8 to +73.3
ct L67	gypsy	Lim	10	1		
ct L149	EMS		11	1		
ct L857	gypsy	Lim	10	1		
ct IS1	<i>copia?</i>	Schalet	10	1		+140 to +142.5
ct IS2	spont	Schalet	2, 10	1		
ct MR2	gypsy		5, 7, 19			0
ct MR2a	gypsy		8			
ct MR4	gypsy		5, 7	wl(u)		
ct MR7	gypsy		8			
ct MR8	gypsy		8			
ct MR9	gypsy		8			
ct MR10	gypsy		8			
ct MR10a	gypsy		8			
ct MR11	gypsy		8			
ct MR12	gypsy		8			
ct MR13	gypsy		8			
ct MR14	gypsy		8			
ct MR15	gypsy		8			
ct MR16	gypsy		8			
ct MR17	gypsy		8			
ct MR115	gypsy		7	l(u)		
ct MR11	<i>burdock</i>		26	1	<i>ct MR2</i> derivative	<i>burdock</i> at +2 kb
ct MR18	<i>burdock</i>		26	1	<i>ct MR2</i> derivative	<i>burdock</i> at +2 kb
ct MR137	<i>burdock</i>		7, 26	l(su)	<i>ct MR2</i> derivative	<i>burdock</i> at +2 kb
ct MR1A1	<i>burdock</i>		26	1	<i>ct MR2</i> derivative	<i>burdock</i> at +2 kb
ct MR1A12	<i>burdock</i>		26	1	<i>ct MR2</i> derivative	<i>burdock</i> at +2 kb
ct MR1B1	<i>burdock</i>		26	1	<i>ct MR2</i> derivative	<i>burdock</i> at +2 kb
ct MR1E1	<i>burdock</i>		26	1	<i>ct MR2</i> derivative	<i>burdock</i> at +2 kb
ct MR1K1	<i>Hercules</i>		26	1	<i>ct MR2</i> derivative	<i>burdock</i> at +2 kb
	in gypsy +					ε
ct MRIL2	<i>burdock</i>				<i>ct MR2</i> derivative	<i>burdock</i> at +2 kb
ct MRIL46	<i>burdock</i>		26	1	<i>ct MR2</i> derivative	<i>burdock</i> at +2 kb
ct MRILD1	<i>Hercules</i>		26	1	<i>ct MR2</i> derivative	ε
	in gypsy +					
	<i>burdock</i>					<i>burdock</i> at +2 kb

allele	origin	discoverer	ref ^α	phenotype ^β	comments	DNA coordinates ^γ
ct MRILM1	<i>burdock</i>		26		<i>ct MR2</i> derivative	<i>burdock</i> at +2 kb
ct MRn1	<i>gypsy</i>		6, 7	w(su)		
ct MRn2	<i>gypsy</i>		6, 7			
ct MRn3	<i>gypsy</i>		6, 7			
ct MRP	<i>roo</i>		26		<i>ct MRpN19</i> derivative	<i>gypsy</i> lost; <i>roo</i> at -5 kb
ct MRpN1	<i>gypsy</i>		7			
ct MRpN1a	<i>jocky</i> in <i>gypsy</i>		8, 26		<i>ct MR2</i> derivative	ε
ct MRpN1b	<i>gypsy</i>		8			
ct MRpN1c	<i>gypsy</i>		8			
ct MRpN6	<i>gypsy</i>		7	w(u)		
ct MRpN7	<i>jocky</i> in <i>gypsy</i>		26		<i>ct MR2</i> derivative	ε
ct MRpN10	<i>jocky</i> in <i>gypsy</i>		5, 7, 19, 26	w(u)	<i>ct MR2</i> derivative	ε
ct MRpN16	<i>gypsy</i>		7	w(u)		
ct MRpN17	<i>jocky</i> in <i>gypsy</i>		8, 26	w(u)	<i>ct MR2</i> derivative	ε
ct MRpN19	<i>jocky</i> in <i>gypsy</i>		7, 26	w(u)	<i>ct MR2</i> derivative	ε
ct MRpN20	<i>jocky</i> in <i>gypsy</i>		26		<i>ct MR2</i> derivative	ε <i>roo</i> LTR at -5 kb
ct MRpN20a	<i>jocky</i> in <i>gypsy</i>		26		<i>ct MR2</i> derivative	ε
ct MRpN22	<i>jocky</i> in <i>gypsy</i>		26		<i>ct MR2</i> derivative	ε
ct MRpN23	<i>jocky</i> in <i>gypsy</i>		26		<i>ct MR2</i> derivative	ε
ct MRpN24	<i>gypsy</i>		8			
ct MRpN25	<i>gypsy</i>		8			
ct MRpN26	<i>gypsy</i>		8			
ct MRpN30	<i>jocky</i> in <i>gypsy</i>		26		<i>ct MR2</i> derivative	ε
ct MRwR	<i>gypsy</i>		7	w	cytology normal	
ct MRwR1	<i>gypsy</i>		8			
ct n	<i>roo</i>	Ives, 32c3	3, 10, 11	w	cytology normal	-6.8 to -4.8
ct ns	<i>copia</i>	Schalet	10, 11	w	<i>In(1)dl-49</i>	-6.8 to -4.8
ct RA4	X ray	Lefevre	16	l	<i>T(1;2)7B3-4;32E1-2</i>	
ct RC26	X ray	Lefevre	10, 11, 16	l	cytology normal	
ct S					<i>In(1)FM7</i>	
ct S6	X ray	Lefevre	16		<i>In(1)7B;8C1-2</i>	
ct S20	X ray	Lefevre	16		<i>T(1;3)7B3-4;84;</i> complex	
ct S69	X ray	Lefevre	16, 26		<i>In(1)7B;8C1-2</i>	break at +93 kb
ct tuh	spont		9		cytology normal	
ct VA109	EMS	Lefevre	17			
ct VA123	EMS	Lefevre	17			
ct VE698	EMS	Lefevre	17			
ct W1	EMS		25	l		
ct W2	EMS		25	l		
ct W3	EMS		25	l		
ct XM31	EMS	Wieschaus	2	l		
ct YE118	EMS	Wieschaus	2	l		
kt¹	CB3007	Fahmy, 1954	4			
kt²			5, 11, 24	f	Basc	ζ
kt^{3δ}	EMS	Lefevre	15	f	<i>T(1;2)7B1-2;2L</i>	
kt MR1	spont		26	f	<i>ct MRpN19</i> derivative	same as <i>kt²</i>
kt MR2	spont		26	f	<i>ct MRpN19</i> derivative	same as <i>kt²</i>
kt MR3	spont		26	f	<i>ct MRpN19</i> derivative	same as <i>kt²</i>

^α 1 = Blochlinger, Bodmer, Jack, Jan, and Jan, 1988, Nature 333: 629-35; 2 = Bodmer, Barbel, Sheperd, Jack, Jan, and Jan, 1987, Cell 51: 293-307; 3 = CP627; 4 = Fahmy, 1959, DIS 33: 87; 5 = Gerasimova, 1981, Mol. Gen. Genet. 184: 544-47; 6 = Gerasimova, 1983, Mol. Gen. Genet. 190: 390-93; 7 = Gerasimova, Ilyn, Mizvokhi, Semjonova, and Gregoriev, 1984, Mol. Gen. Genet. 193: 488-92; 8 = Gerasimova, Matjunina, Mizrokhii, and Georgiev, 1985, EMBO J. 4: 3773-79; 9 = Kuhn and Walker, 1980, DIS 55: 207; 10 = Jack, 1985, Cell 42: 869-76; 11 = Johnson and Judd, 1979, Genetics 92: 485-502; 12 = Krivshenko, 1956, DIS 30: 74; 13 = Lee, 1980, DIS 48: 18; 14 = Lefevre and Johnson, 1973, Genetics 74: 633-45; 15 = Lefevre and Leeds, 1983, Genetics 104: s45-46; 16 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 17 = Lefevre; 18 = Lim, Simmons, Raymond, Cox, Doll, and Culbert, 1983, Proc. Nat. Acad. Sci. USA 80: 6624-27; 19 = Mizrokhii, Obolenkova, Priimägi, Ilyin, Gerasimova and Georgiev, 1985, EMBO J. 4: 3781-87; 20 = Muller; 21 = Muskovitch; 22 = Poulson and King, 1948, DIS 22: 54; 23 = Valencia, 1966, DIS 41: 58; 24 = Whitney and Lucchesi, 1972, DIS 49: 35; 25 = Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307; 26 = Tchurikov, Gerasimova, Johnson, Barbakar, Kenzior, and Georgiev, 1989, Mol. Gen. Genet. 219: 241-48.

^β f = kinked femur; u = unexpanded wings; w = cut wings; a = deformed antennae; v = displaced or missing vibrissae; b = fine bristles; l = lethal; (u) = unstable; (su) = superunstable.

- γ Coordinate system of Jack; origin at site of *gypsy* insertion of *ct*⁶. Coordinates used by Tchurikov *et al.* place *ct*⁶ insertion at -118 kb; here converted to those of Jack by the addition of 118 kb.
- δ *ct*^{cl} = cut-cutless; *ct*^{cl1} and *ct*^{cl2} recovered as *l(1)VA39* and *l(1)VE829* respectively; isolation number of *kf*³ = VE614.
- ε Derivatives of *ct*^{MR2} in which an additional transposable element is inserted in the left end of the *gypsy* element associated with *ct*^{MR2}: *ct*^{MRpN1a}, *ct*^{MRpN7}, *ct*^{MRpN17}, *ct*^{MRpN19}, and *ct*^{MRpN26} have *jockey* inserts in one position and in one orientation; *ct*^{MRpN10}, *ct*^{MRpN20A}, *ct*^{MRpN22}, *ct*^{MRpN23}, and *ct*^{MRpN30} have *jockey* inserts in a slightly different position and in the opposite orientation; *ct*^{MRIK1}, and *ct*^{MRILD1} have *Hercules* inserts in different sites in the same region.
- ζ Additional information detailed below.

tions on the chromosomes where new mutants arise. Other types of transposing elements also appear to be mobilized in these two situations (*e.g.* Gerasimova, Matyunina, Ilyin, and Georgiev, 1984, *Mol. Gen. Genet.* 194: 517-22).

In the accompanying table of alleles, those superscripted *L* were recovered by Lim and colleagues; *L* followed by an even number designates a cut-wing allele, and *L* followed by an odd number a group-I lethal allele; alleles designated as weak cut wings have more nearly entire wing margins and lack head-capsule defects. Alleles superscripted *MR* are derived, either primarily or secondarily from hybrid-dysgenesis crosses by Gerasimova and her colleagues; those designated by *MR* followed by a number display the strong *ct*⁶-like wing phenotype; those designated *MRpN* are less extreme with multiple small incisions in the wing margin. *ct*^{MR2} can revert or partially revert by excision of, or insertion of other elements (*e.g.* *Jockey* or *Hercules*), into the *gypsy* insert responsible for *ct*^{MR2} (Leigh, Brown, Ross, Alphey, Flavell, and Gerasimova, 1989, *Mol. Gen. Genet.* 218: 203-13; Tchurikov, Gerasimova, Johnson, Barbakar, Kenzior, and Georgiev, 1989, *Mol. Gen. Genet.* 219: 241-48). Unstable revertants are able to generate new *ct* derivatives, retain the *gypsy* LTR of *ct*^{MR2} (Mizrokhi, Obolenkova, Priimägi, Ilyin, Gerasimova and Georgiev, 1985, *EMBO J.* 4: 3781-87).

ctology: Placed in 7B1-2 by *in situ* hybridization.

molecular biology: Two hundred kilobases of DNA from the cut region cloned and restriction mapped (Jack, 1985, *Cell* 42: 869-76). Coordinate 0 is the site of the *gypsy* insert in *ct*⁶; coordinates 0 to -60 extend to the left and 0 to +140 to the right; all sequences hybridize to 7B1-2. Kinked-femur effects map in the -60 to -14 kilobase region, and cut-wing effects, many of which are known to result from *gypsy* inserts, to -6 to +19.3. Group I lethals cluster in the +71 to +73.5 region, and most of them contain *gypsy* inserts; group II mutants do not contain *gypsy* inserts and are thought to lie to the right of coordinate +100. 8,217 base pairs of cDNA clones sequenced; transcribed from over 70 kb of genomic sequence from coordinates approximately 80 to 150, which contain lethal I and lethal II regions. Transcription is from left to right. Sequence reveals long open reading frame encoding a putative protein of 2,175 amino acids with molecular mass of 240 kd. Principle among the features of the amino-acid sequence is a 60-residue homeodomain homologue, which although the most divergent of *Drosophila* homeobox sequences shows identity at the nine residues that are invariant in all homeodomains. In addition there are three 60-residue repeats showing 55-68% identity to each other which are not homologous to repeats found in other proteins; there are also four stretches of polyglutamate aspartate and a number of runs rich in single amino acids (Blochlinger *et al.*).

ct^{71g}

molecular biology: A molecular inversion with one break-point between DNA coordinates -35.8 and 33.2 and the other between +4.1 and +4.8.

ct^{JC20}

molecular biology: A molecular deletion beginning between coordinates -1.7 and +1.0 and extending to the left beyond -60, the extent of the cloned region. Deletes material of both kinked-femur and cut-wing regions of the complex.

ct^k

phenotype: Classified as a group-I lethal because homozygotes show reduced viability, and only 10% of fewer heterozygotes with other lethal alleles survive; survivors have weakly cut wings, as well as fine bristles, and enlarged and deformed humeral callus, not seen in other cut-wing mutants.

ct^{cl}: cut-cutless

references: Leeds and Lefevre, 1983, *Genetics* 104: s45-46.

phenotype: Acts as a lethal allele in combination with deficiencies for *ct*; phenotype normal in combination with viable *ct* alleles. Homozygotes have reduced viability and show thoracic protuberances (Schalet). Heterozygotes of *ct*^{cl} with lethal alleles of *ct* die or eclose in small numbers; an exception is *ct*^{cl/ct}^{HA46}, which exhibits normal survival.

ct^{IS1} (A. Schalet)

phenotype: Lethal; a member of cut Lethal II group, maps proximal to *ct*^{C145} (Jack, 1985). Not suppressed by *su(Hw)*² (Schalet). *ct*^{IS1/+} males, frequently show thoracic protuberances (Schalet).

ct^{IS2} (A. Schalet)

phenotype: Almost complete lethal; survival less than 1%. Not suppressed by *su(Hw)*²; cut-cutless type of mutant in that *ct*^{IS2} fails to complement lethal alleles, *e.g.* *ct*^{IS1}, but complements *kf*², *ct*⁶ and *ct*^S. Rare surviving males and females are fertile with normal wings, but usually show thoracic protuberances in the region of the presutural and notoplural bristles as are also seen in heterozygotes of *ct*^{IS1}, *Df(1)ct*^{J4} or *Df(1)ct*^{J6}.

*kf*²

molecular biology: An approximately 18 kilobase deletion extending from a point between coordinates -35.8 and -33.2 to a point between -18.5 and -14.0.

cta: concertina (T. Schüpbach)

location: 2-{54.8}.

references: Schüpbach and Wieschaus, 1989, *Genetics* 121: 101-17.

phenotype: Maternal-effect lethal, female sterile: Embryos from homozygous mothers gastrulate abnormally; no posterior midgut formed; the germ band does not advance to the dorsal side of the embryo, but as it

expands, it is thrown into a series of folds at the ventral side of the embryo. Anterior midgut appears to develop normally. The phenotype is reminiscent of the zygotic embryonic lethal mutation folded gastrulation (*fog*). At final differentiation, the embryos from *cta* mothers have a fairly normal array of thoracic and abdominal segments with morphologically normal denticle belts. Defective head skeleton seen in larvae.

alleles:

allele	origin	synonym
<i>cta</i> ¹	EMS	<i>cta</i> ^{WU}
<i>cta</i> ²	EMS	<i>cta</i> ^{PC}
<i>cta</i> ³	EMS	<i>cta</i> ^{PG}
<i>cta</i> ⁴	EMS	<i>cta</i> ^{QB}
<i>cta</i> ⁵	EMS	<i>cta</i> ^{RC}

cytology: In heterochromatic region of 2L, since uncovered by *Df(2L)PR31*.

***ctl*: coatless**

location: 1-25.8.

origin: Induced by ethyl methanesulfonate.

references: Eberl and Hilliker, 1988, *Genetics* 118: 109-20.

phenotype: Hemizygous lethal; distinct yolk plug indicating poorly developed gut; variably incomplete Malpighian tubules; poorly differentiated cuticle; mouthparts abnormal.

***ctt*: contorted**

location: 1-0.3.

origin: Induced by ethyl methanesulfonate (CB. 1528).

discoverer: Fahmy, 1956.

references: 1959, *DIS* 33: 84.

phenotype: Wings shorter than normal and abnormally shaped, frequently curved either convexly or concavely. Eyes rough and slightly altered in shape. Bristles thinner and straggly; orbitals frequently reduced or absent. Male genitalia frequently slightly twisted and abnormal. Males fertile; females sterile. RK2.

***cu*: curled**

location: 3-50.0.

origin: Spontaneous.

discoverer: Morgan, 15115.

references: Morgan and Bridges, 1923, *Carnegie Inst. Washington Publ. No. 327*: 152 (fig.).

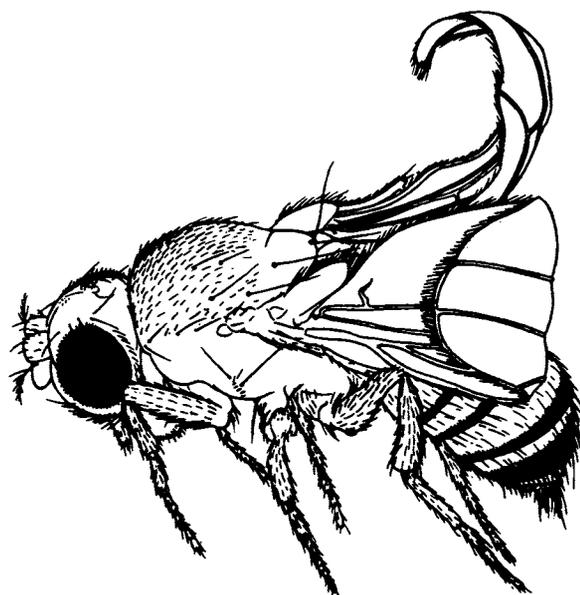
Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 215 (fig.), 223.

Whittinghill, 1937, *DIS* 7: 22.

phenotype: Wings curved upward throughout length and slightly divergent. Body color dark. Postscutellars erect and crossed. Good nutrition of larvae enhances curled character as does high temperature in last day of pupal life. (Nozawa, 1956, *Jpn. J. Genet.* 31: 321-26). RK1.

alleles: **cu*^{100.69}, **cu*^{100.384}, and **cu*^{300.215} (CP627); *cu*^{5J} associated with *In(3R)84F11-12;86D1-E2* (Ashburner, 1981, *DIS* 56: 190).

cytology: Located in 86D1-4 based on the absence of *cu*⁺ from *Df(3R)M86D = Df(3R)86D1;86D4* (Ashburner, 1981, *DIS* 56: 189).



***cu*: curled**

From Bridges and Morgan, 1923, *Carnegie Inst. Washington Publ. No. 327*: 152.

****Cu*: Curl**

location: 2-54.6 (0.1 unit to the right of *pr*).

origin: X ray induced.

discoverer: Bateman, 1954.

synonym: *Cd*: *Coildex*; *Coi*: *Coiled*.

references: 1955, *DIS* 29: 69.

phenotype: Similar to *Cy* but wing curvature more extreme; wings opaque and greyish. Anterior margin of wing invaginated at point where L1 meets wing margin. When expression is weakest, it appears only as a slight wave in the wing margin. In 10-15% of the flies, wings also curve downward over flanks before curling upward. In *y*; *Cu*⁵⁴ flies, curvature reduced to a shallow spoon. *Cu*⁵⁴ epistatic to *Cy*. RK2.

alleles:

allele	origin	discoverer	synonym	ref ^α	homozygote phenotype
<i>*Cu</i> ¹	spont.	Ives		5	viable
<i>Cu</i> ²	EMS	Grell			viable, does not complement <i>stw</i>
<i>Cu</i> ³		Ish-Horowicz, 1976	<i>Cd</i> ²	2	lethal
<i>*Cu</i> ⁵⁴	X ray	Bateman, 1954	<i>Cd</i>	2	lethal
<i>Cu</i> ⁵⁷	X ray	Carlson, 57g	<i>Coi</i>	3	viable; complements <i>stw</i> ^β
<i>Cu</i> ⁷⁵	X ray	Ashburner	<i>Coi</i> ⁷⁵	1	viable
<i>*Cu</i> ^A	spont.	Fernandez Gianotti	<i>Ac</i>	4	viable

^α 1 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, *DIS* 55: 193-95; 2 = Bateman, 1955, *DIS* 29: 69; 3 = Carlson, 1960, *DIS* 34: 48; 4 = Fernandez Gianotti, 1948, *DIS* 22: 53; 5 = Ives, 1948, *DIS* 22: 53.

^β Viability and fertility of *Cu*⁵⁷ excellent; phenotype extreme.

other information: Allelism of *Cd*, *Coi*, and *Cu* inferred from similarity of phenotype and of genetic position (54.6-56.6). *Cd* (= *Cu*⁵⁴) and *Coi* (= *Cu*⁵⁷) declared to be allelic based on renewed mapping of *Coi* just to the right rather than to the left of *pr* in agreement with the published position of *Cd* (Ashburner and Woodruff, 1979, *Genetics* 92: 117-32). Both *Cu*⁵⁷ and *Cu*⁷⁵ suppressed by *Dp(2;2)Adh3* (Ashburner and Woodruff). *Cu* and

*Cu*⁵⁷ shown to be in 2R based on their being recoverable in *C(2R)* but not *C(2L)* compounds (E.H. Grell).

Cu-3: Curl in chromosome 3

location: 3-66.0.
origin: Spontaneous.
discoverer: Erickson and Meyer, 51c.
synonym: *Cur*: *Curl* preoccupied.
references: Meyer, 1952, DIS 26: 66.
phenotype: Heterozygote has curly wings with parchment-like texture resembling *Cy*. Homozygous lethal. RK2.

cu-X: curled-X

location: 1- (not located but not allelic to *cx*).
origin: Spontaneous in *In(1)dl-49+B^{MI}*, *y sc v*.
discoverer: Krivshenko, 57j29.
references: 1956, DIS 32: 80.
phenotype: Males have wings that are bent upwards and diverge slightly. *cu-X* is never expressed in females. It represents a mutation whose phenotypic expression is sex limited. Expressed equally well in males with and without a Y chromosome. RK2.

cubitus interruptus: see ci

cuff: cutoff (T. Schüpbach)

location: 2-61.
origin: Induced by ethyl methanesulfonate.
references: Schüpbach and Wieschaus.
phenotype: female-sterile; homozygous females often have underdeveloped ovaries which seem to lack germ cells altogether. In some females, a small number of developing egg chambers is found; these never develop beyond the first few stages of oogenesis.
alleles: *cuff*¹ to *cuff*³ isolated as *WL*, *QQ*, and *WM* respectively.

cui: curvi

location: 2-23.4 (1.4 to the right of *Sp* and 0.5 to the right of *lys*).
origin: Spontaneous.
discoverer: Nicoletti.
synonym: *curved*.
references: 1957, DIS 31: 84.
phenotype: Distal half of wing curved upward. Viability and expressivity very good. RK1.

cup: cup (T. Schüpbach)

location: 2-23.
origin: Induced by ethyl methanesulfonate.
references: Schüpbach and Wieschaus.
phenotype: Female-sterile; homozygous females show abnormalities in late oogenesis. Follicle cells do not migrate centripetally between nurse cells and oocyte. They do synthesize a chorion which remains open ended (like a chalice, or cup). Such eggs are usually not laid and remain unfertilized.
alleles: Thirty seven alleles; *cup*¹ to *cup*³⁷ (isolated as *WQ*, *PB1*, *PB53*, *PF21*, *PF63*, *PH69*, *PL11*, *PN14*, *PS73*, *PV36*, *PW3*, *PV76*, *QD57*, *QJ9*, *QJ36*, *QJ65*, *QK4*, *QK12*, *QK24*, *QL54*, *QM66*, *QR55*, *QR56*, *QV7*, *QV50*, *QW45*, *RC11*, *RG6*, *RH13*, *RH24*, *R177*, *RM12*, *RN61*, *RS2*, *RS42*, *RS50*, and *RU45* respectively).

cupola: see cpl

cur: curvoid

location: 3-30.
origin: Spontaneous.
discoverer: Bridges, 33c14.
phenotype: Wings divergent and curved down. Resembles *c*. Viability erratic. RK3.

Cur: see *Cu-3*

Curl: see *Cu*

curled: see *cu*

Curled blistered: see *Cb*

curlex: see *cx*

Curly: see *Cy*

Curlyoid: see *Cyd*

curved: see *cr*

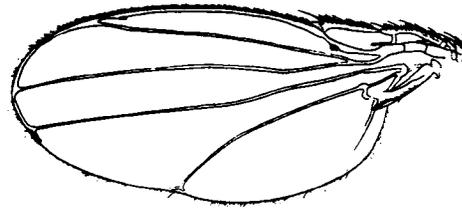
curved: see *cui*

Curved of Krivshenko: see *C-K*

curvi: see *cui*

curvoid: see *cur*

cut: see *ct*



cv: crossveinless

From Weinstein, A., 1920, Proc. Nat. Acad. Sci. USA 6: 625-39.

cut off: see *cuff*

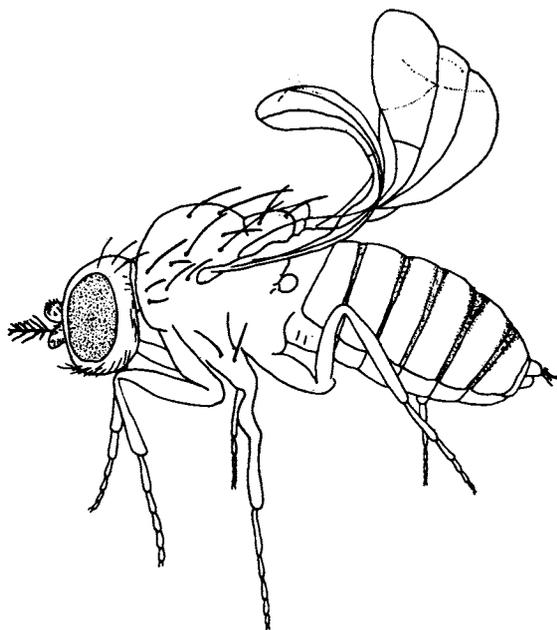
cv: crossveinless

location: 1-13.7.
origin: Spontaneous.
discoverer: Bridges, 1912.
references: 1920, Proc. Nat. Acad. Sci. USA 6: 660-63. Weinstein, 1920, Proc. Nat. Acad. Sci. USA 6: 625-39 (fig.).
phenotype: Crossveins absent or traces only present. Veins L3 and L4 slightly delta at tips. Classifiable in unexpanded wings. Wing effects due to excessive contraction in the pupal period, obliterating the cavity which should normally remain between the epithelia to form the vein (Waddington, 1940, J. Genet. 41: 75-139). RK1.
alleles: **cv*⁶⁸ induced by ethyl methanesulfonate (Hayman and Maddern, 1969, DIS 44: 50).
cytology: Located in polytene region 5B [Lefevre, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 31-66].

cv-2: crossveinless on chromosome 2**location:** 2-96.2.**origin:** Spontaneous.**discoverer:** Nicoletti, 62j.**phenotype:** Anterior and posterior crossveins absent. RK1.**alleles:** Found segregating in several natural populations (Boyer, Parris, and Milkman, 1973, *Genetics* 75: 169-75).**cytology:** Salivary chromosomes normal.***cv-b: crossveinless-b****location:** 3-65.**origin:** Spontaneous.**discoverer:** Bridges, 24k8.**phenotype:** Crossveins reduced or absent. May overlap wild type. RK3.**cv-c: crossveinless-c****location:** 3-54.1 (0.5 units to the right of *red*, Craymer).**origin:** Spontaneous.**discoverer:** Stern, 25g13.**references:** 1934, DIS 1: 35-36.**phenotype:** Posterior crossvein usually absent or greatly reduced. Anterior crossvein usually present but often detached. Eye flattened or with vertical shallow furrow. Legs weak, especially tarsal joints. Occasionally overlaps wild type. RK2.**cytology:** Placed in 88B-C based on its inclusion in the synthetic deficiency with *3R* proximal derived from $T(Y;3)P102 = T(Y;3)87B2-3$ and *3R* distal derived from $T(3;4)P86 = T(3;4)88B-C;101$ (Bernstein) as well as in the duplication from $T(1;3)O5 = T(1;3)4F2-3;62B-C;88A-C;92C-D$ (Lindsley and Grell, 1958, DIS 32: 136).**cv-d: crossveinless-d****location:** 3-65.**origin:** Appeared among progeny of ether-treated flies.**discoverer:** Duncan, 34c.**references:** 1935, DIS 4: 7.**phenotype:** Posterior crossvein absent or reduced to an oblique fragment or bar parallel to L5. Anterior crossvein sometimes detached. RK2.**other information:** Possibly an allele of *cv-b*.**cve: crossveinless effect**

This is a term used by Milkman for alleles anywhere in the genome which affect the formation of crossveins. Not strictly a gene locus designation.

cvl-5: crossveinless like**location:** 3-48.1 [based on 55 crossovers between *st* and *cu* (Thompson, 1967, DIS 42: 41)].**origin:** The major component in a line selected by Mohler (1965, *Genetics* 51: 641-51) for crossveinless-like phenotype.**references:** Thompson, 1967, *Genetics* 56: 13-22.**phenotype:** Removes crossveins. Complex response to heat shock described by Thompson (*ibid*).**cvl-6: crossveinless like 6****location:** 1-59.1 (2.4 map units to the right of *f*).**origin:** Major component of a line selected by Mohler for crossveinless-like phenotype.**references:** Mohler, 1965, *Genetics* 51: 641-51.**phenotype:** Removes crossveins. Developmental sequence differs from that of *cv* and *cvl-5*; longitudinal veins form in time and manner expected of wild type; *cvl-6* differs in that the wings begin vein development with a joining of the wing surfaces through the crossvein region; partial repair is accomplished by a secondary developmental sequence, including withdrawal of cell processes joining the wing surfaces at this position and subsequent reorganization of a crossvein around this gap (Mohler and Swedberg, 1964, *Genetics* 50: 1403-19).***cvw: convex wing****location:** 1-58.2.**origin:** Induced by D-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3026).**discoverer:** Fahmy, 1953.**references:** 1958, DIS 32: 69.**phenotype:** Wings slightly shortened and arched convexly. Variable and may overlap wild type. Tergites in some females have serrated edges or are grossly deformed. Viability and fertility good in both sexes. RK2.**cwi: crumpled wings****location:** 1-{0.5}.**references:** Zhimulev, Belyaeva, Fomina, Protopopov, and Bolshakov, 1986, *Chromosoma* 94: 492-504.**phenotype:** Crumpled wings?**cytology:** Placed in 2B7-10.**cx: curlex****location:** 1-13.6.**origin:** Spontaneous.**discoverer:** R. L. King, 1927.**phenotype:** Wings bent upward for posterior two-thirds of length; anterior one-third warped; margin kinked. Wings not spread. RK2.**cytology:** Placed in polytene region 5A-B by Lefevre.*cx*: see *c(3)G***cx^{tg}: curlex-twisted genitalia****origin:** Spontaneous.**discoverer:** Curry, 37c19.**phenotype:** Wings always divergent, usually 45° from axis. Basal one-third of wing wavy but less so than in *cx*; posterior two-thirds of wing curled slightly upward or downward. Genitalia of nearly all males rotated, usually 45° counterclockwise. Flies dwarfish. Viability irregular. Male sterile. RK2.*cx-b*: see *wy*²**cxb: calyx bulging (J.C. Hall)****location:** 2-73.**origin:** Induced by ethyl methanesulfonate.**synonym:** *cxb*^{N71}.**references:** Heisenberg, Borst, Wagner, and Byers, 1985, *J. Neurogenet.* 2: 1-30.**phenotype:** Calyces of mushroom bodies in dorsal brain of adult are enlarged and have abnormal shape; peduncles and lobes (projecting from these calyces) small or missing; ellipsoid body of central brain also aberrant in morphology; learning abnormal, in tests using olfactory stimuli.*CxD*: see *In(3LR)CxD**CxF,D*: see *In(3LR)DcxF*



Cy: Curly

From L. Ward, 1923, *Genetics* 8: 276-300.

Cy: Curly

location: 2-6.1 (removed from *In(2L)Cy* and located by Tinderholt).

origin: Spontaneous.

discoverer: L. Ward, 20c.

references: 1923, *Genetics* 8: 276-300 (fig.).

phenotype: Wings curled upward; rarely overlaps wild type at 25°, but frequently overlaps at 19°. Curvature caused by the unequal contraction of the upper and lower epithelia during the drying period following emergence from the pupa case (Waddington, 1940, *J. Genet.* 41: 75-139). Flightless but thoracic innervation normal (Chiarodo, Reing, and Saranchak, 1971, *J. Expt. Zool.* 178: 325-30). Expression decreased by larval crowding, increased by increased temperature during pupal development (Nozawa, 1956, *Jpn. J. Genet.* 31: 161-71). Classifiable in single dose in triploids. Usually homozygous lethal but may emerge as dwarf with more extreme wing character. Effective lethal phase late embryo-early larva (Kidwell, 1972, *J. Hered.* 63: 100). RK1A.

alleles: *Cy^M*: Curly of Meyerowitz; *Cy^{rv76}*, X-ray-induced reversion of *Cy* in *CyO* (Wright, Hodgetts, and Serald, 1976, *Genetics* 84: 267-85).

cytology: Placed in 22F4-23B2 on the basis of its being lethal in combination with *Df(2L)DTD2 = Df(2L)22D4-5;23B1-2* but not *Df(2L)DTD48 = Df(2L)22E2-4;22F-23A1* (Spencer, Hoffman, and Gelbart, 1982, *Cell* 28: 451-61). Ordinarily inseparable from *In(2L)Cy = In(2L)22D1-2;33F5-34A1*, although it was separated by Tinderholt (1961, *DIS* 35: 47).

other information: *Cy* removed from *In(2L)Cy* still causes a local reduction in crossing over in the *ed-cl* region (Sederoff).

Cyclic AMP dependent protein kinase: see *Pka*

Cyclic GMP dependent protein kinase: see *Pkg*

Cyclic AMP Phosphodiesterase-2: see *dnc*

CycA: Cyclin A

location: 3-{37}.

synonym: *l(3)68Ea; l(3)rsg11*.

references: Whitfield, González, Sánchez-Herrero, and Glover, 1989, *Nature* 338: 337-40.

Lehner and O'Farrell, 1989, *Cell* 56: 957-68.

phenotype: Encodes cyclin, a molecule involved in the cell cycle; the pattern of transcription reflects this. Maternal message uniformly distributed in newly laid egg, but becomes more concentrated in the cortex prior to peripheral nuclear migration. At the time of cellularization of the blastoderm, zygotic message is produced. Cyclin A accumulates in the interphase cytoplasm of cellularized embryos, but relocates to the nuclear region early in prophase and is completely degraded during metaphase. A functional cyclin A gene is required for continued cell division after exhaustion of maternally provided cyclin A. Message confined to dividing tissues in brain and imaginal disks in larvae.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>CycA¹</i>	EMS		<i>l(3)70-2</i>	2	
<i>CycA²</i>	EMS		<i>l(3)107.43</i>	2	
<i>CycA³</i>	EMS		<i>l(3)183</i>	2, 3	molecular deletion
<i>CycA⁴</i>	EMS		<i>l(3)V4-4</i>	1, 2	
<i>CycA⁵</i>	P	Jan	<i>neo114</i>	3	P insert present

^α 1 = Akam, Roberts, Richards, and Ashburner, 1978, *Cell* 13: 215-25; 2 = Hoogwerf, Akam, and Roberts; 3 = Lehner and O'Farrell, 1989, *Cell* 56: 957-68.

cytology: Placed in 68E by *in situ* hybridization; included in *Df(3L)vin3 = Df(3L)68C5-6;68D6* but not *Df(3L)vin2 = Df(3L)67F2-3;68E3-4*.

molecular biology: Sequence isolated from a genomic library using a synthetic oligonucleotide based on conserved sequences of known cyclin genes. Detects a 2.3 kb transcript that is abundant in pre-cellularization embryos and in adult females; a 2.5 kb zygotically derived transcript appears after cellularization (Akam *et al.*). These transcripts are 2.5 and 3.0 kb respectively according to Lehner and O'Farrell. Akam *et al.* report three other transcripts detectable in pupae, of which one persists into adulthood. Lehner and O'Farrell report the sequence of a cDNA that contains a long open reading frame that encodes a 491-amino-acid protein, which displays 50% amino-acid identity to clam cyclin A and 34% identity to clam cyclin B.

CycB: Cyclin B

location: 2-{100}.

references: Whitfield, González, Sánchez-Herrero, and Glover, 1989, *Nature* 338: 337-40.

phenotype: Encodes cyclin, a molecule involved in the cell cycle; the pattern of transcription reflects this. Maternal message uniformly distributed in newly laid egg, but becomes concentrated at the posterior pole at the time of polar-nucleus migration; also evident in the cortex of the syncytial blastoderm. Larval message concentrated in the testis.

cytology: Placed in 59A by *in situ* hybridization.

molecular biology: Sequence isolated from a genomic library using a synthetic oligonucleotide based on conserved sequences of known cyclin genes. Detects a 2.3

kb transcript that is abundant in pre-cellularization embryos and in adult females; a 2 kb transcript is found in adult males.

Cyclin A: see *CycA*

Cyclin B: see *CycB*

Cyd: Curlyoid

location: 3- (rearrangement).

discoverer: Jollos.

references: Curry, 1939, DIS 12: 46.

phenotype: Wings curled upward in heterozygote, only slightly so in some flies; wings slightly reduced in size and dusky. *In(3R)P,Cyd/+* has 2- to 3-day developmental delay (Craymer). Homozygous lethal. RK3.

cytology: Associated with *In(3R)P*; no other aberration present (Craymer).

cyl: see *rk^{cyl}*

Cyt-b: Cytochrome-b homologue

location: 2-{52}.

synonym: *TU36B*.

references: Levin, Boychuk, Croniger, Kazzaz, and Rozek, 1989, Nucleic Acids Res. 17: 6349-67.

phenotype: Encodes a cytochrome-b homologue that is expressed specifically in muscles; expression is concordant with that of *Mhc*.

cytology: Placed in 36B based on its juxtaposition to *Mhc*.

molecular biology: cDNA's cloned and sequenced; gene shown to have one intron. mRNA transcribed off of

opposite strand from *Mhc*; poly(A) addition sites 99 nucleotides apart. Contains an open reading frame of 1242 nucleotides able to encode a polypeptide of 414 amino acids of 47 kd. Sequence shows 17.6% identity and 41% homology with yeast cytochrome-b2.

Cyt-c1 and Cyt-c2: Cytochrome c

location: 2-{52}.

origin: Isolated from a Charon-4 library using mouse cytochrome-c gene sequence as a probe.

references: Limbach and Wu, 1985, Nucleic Acids Res. 13: 631-44.

phenotype: Apparently the structural genes for two cytochrome-c type enzymes, which differ from each other in 32/107 amino-acid residues. *Cyt-c1* (sequence D3) expressed at constant, but relatively low, level throughout development; *Cyt-c2* (sequence D4) expressed at varying, but relatively high, levels throughout development. Amino acid sequence also given in Handbook of Biochemistry and Molecular Biology: Proteins (Fasman, ed.) CRC Press, Cleveland, Vol. 3, pp. 282-83.

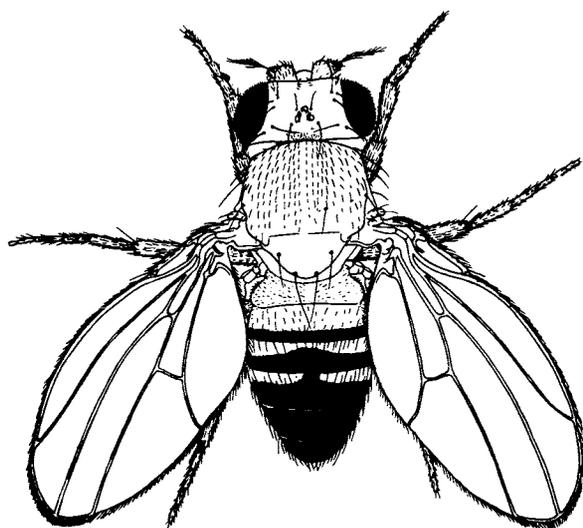
cytology: Placed in 36A10-11 by *in situ* hybridization.

molecular biology: Sequences located within a 4 kb region of DNA. Sequence analysis indicates that both can encode functional cytochrome-c proteins. *Cyt-c* genomic clones also isolated, using *Manduca sexta* cDNA clone as probe, and sequenced by Swanson, Zieminn, Miller, Garber, and Margoliash (1985, Proc. Nat. Acad. Sci. USA 82: 1964-68).

d: dachs

location: 2-31.0.
origin: Spontaneous.
discoverer: Morgan and Bridges, 12k22.
references: Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 216 (fig.).
 Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 212 (fig.), 223.
phenotype: Similar to *ff*. Tarsi four jointed instead of five jointed owing to failure of joint formation between second and third tarsal segments (Tokunaga and Gerhart, 1976, Genetics 83: s76); joint remnants sometimes persist beneath the cuticle (Held, Duarte, and Derakhshanian, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 145-57). Legs short and held close to body. Femur, tibia, and tarsae foreshortened [Tokunaga and Gerhart; Mikuta and Mglinetz, 1979, Genetika (Moscow) 15: 624-32]. Probability of failure of joint formation proportional to degree of foreshortening of second tarsal segment (Tokunaga and Gerhart). Joint failure phenotype can extend into *d/+* tissue adjacent to *d/d* clones (Tokunaga and Gerhart). Number of longitudinal rows of bristles in second basitarsus unaffected, but number of bristles per row reduced (Held, 1979, Wilhelm Roux's Arch. Dev. Biol. 187: 105-27). Leg effects enhanced by *ss^a* and *ss^{aB}* (Villem, 1945, Genetics 30: 26-27). Wings smaller than wild type, narrowed, with L2 and L3 joined near anterior crossvein; distance between crossveins smaller and crossveins sometimes absent. Angle between L2 and L5 greater than normal. Eyes small and rough. Posterior scutellar bristles erect. Viability erratic. Frequently sterile. Enhances transforming effects of *Antp^{Ns}* (Mikuta and Mglinetz, 1979) and *pb*; labial legs in *d*; *pb* flies show failure of joint formation [Kaurov, 1978, Genetika (Moscow) 14: 306-12]. RK2.

d¹: see *Df(2L)d*



D: Dichaete

From Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 127.

D: see *Lvp-D*

D: Dichaete

location: 3-40.7.
origin: Spontaneous.
discoverer: Bridges, 15a3.
references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 127 (fig.).
phenotype: Wings extended uniformly at 45° from body axis and elevated 30° above (occasionally sharply down-cast and dragging). Alulae missing. Dorsocentrals and some other bristles reduced in number (Sturtevant, 1918, Carnegie Inst. Washington Publ. No. 264; Plunkett, 1926, J. Exp. Zool. 46: 181-244). Head often deformed or split in postvertical region. Halteres turned down. Homozygous lethal. Nearly lethal in combination with *ey^D* (Sobels, Kruijt, and Spronk, 1951, DIS 25: 128). Partially suppressed by *sc* alleles that remove postverticals (*sc*, *sc⁴*, *sc⁶*, *sc⁷*) but not by others (*sc²*, *sc⁵*) (Sturtevant). Classifiable in triploids. RK2A.
alleles: *D³* and *D^E* less severe derivatives of *D* (CP627). *D⁴* X ray induced, associated with *T(2;3)21D;70-71* (Craymer, 1980, DIS 55: 197).
cytology: Inseparable from *In(3L)D = In(3L)69D3-E1;70C13-D1* (Bridges in Morgan, Bridges, and Schultz, 1937, Year Book - Carnegie Inst. Washington 36: 301). Tentatively placed in 70C13-D1 based on breakpoint common to *In(3L)D* and *T(2;3)D⁴*.

da: daughterless (C. Cronmiller and T.W. Cline)

location: 2-41.5.
origin: Spontaneous.
discoverer: Bell.
references: Bell, 1954, Genetics 39: 958-59.
 Colainne and Bell, 1968, DIS 43: 155.
 Sandler, 1972, Genetics 70: 261-74.
 Mange and Sandler, 1973, Genetics 73: 73-86.
 Mason, 1973, DIS 50: 93.
 Sandler, 1975, Israel. J. Med. Sci. 11: 1124-34.
 Cline, 1976, Genetics 84: 723-42.
 Bownes, Cline, and Schneiderman, 1977, Wilhelm Roux's Arch. Dev. Biol. 181: 279-84.
 Sandler, 1977, Genetics 86: 567-82.
 Watanabe and Yamada, 1977, Jpn. J. Genet. 52: 9-14.
 Cline, 1978, Genetics 90: 683-98.
 Picologlou, Bell, and Rogler, 1978, Genetika 48: 201-06.
 Cline, 1980, Genetics 96: 903-26.
 Cline, 1983a, Dev. Biol. 95: 260-74.
 Cline, 1983b, Genetics 104: s16-17.
 Cline, 1984, Genetics 107: 231-77.
 Cronmiller and Cline, 1986, Dev. Genet. 7: 205-21.
 Cronmiller and Cline, 1987, Cell 48: 479-87.
 Gergen, 1987, Genetics 117: 477-85.
 Muir and Bell, 1987, Genetika 72: 43-54.
 Brand and Campos-Ortega, 1988, Wilhelm Roux's Arch. Dev. Biol. 197: 457-70.
 Caudy, Grell, Dambly-Chaudière, Ghysen, Jan, and Jan, 1988a, Genes and Dev. 2: 843-52.
 Caudy, Vässin, Brand, Tuma, Jan, and Jan, 1988b, Cell 55: 1061-67.
 Cronmiller, Schedl, and Cline, 1988, Genes and Dev. 2: 1666-76.
 Dambly-Chaudière, Ghysen, Jan, and Jan, 1988, Wilhelm Roux's Arch. Dev. Biol. 197: 419-23.
 Murre, McCaw, and Baltimore, 1989a, Cell 56: 777-83.
 Murre, McCaw, Vässin, Caudy, Jan, Jan, Cabrera,

Buskin, Hauschka, Lassar, Weintraub, and Baltimore, 1989b, Cell 58: 537-44.

phenotype: da^+ performs multiple roles during development. Maternally supplied da^+ is required in female embryos as a positive activator of the gene, Sex-lethal (*Sxl*), the key binary switch gene for the sex determination pathway. Also, da^+ expression is required in the somatic gonad of adult females for proper egg membrane formation, and hence for the survival of all progeny regardless of their sex. Embryonic expression of da^+ is required in both sexes for the formation of the peripheral nervous system (PNS) and parts of the central nervous system (CNS). And, during larval and/or pupal stages, da^+ may be required for the growth and/or differentiation of cells that form the adult cuticle.

Amorphic alleles (da^2 , da^3 , da^5 , etc.) are recessive lethals, with a lethal period which is predominantly embryonic (Cronmiller and Cline, 1987; Caudy *et al.*, 1988a). In addition, the hypomorphic allele, da^1 (originally called *da*), is hemizygous [$da^1/Dff(2L)da^-$] lethal (Mange and Sandler, 1973), and da^1 homozygotes die when they undergo the first half of embryonic development at 29° (Cline, 1976). Death appears to be a consequence of dosage compensation defects (Cline, 1983a; Gergen, 1987). Viability of da^1 homozygotes is improved by the presence of extra X or Y heterochromatin in either the parental female or her progeny (Sandler, 1972; Mason, 1973). Temperature-sensitive lethality of the da^1 zygotic lethal effect is not affected by the *Sxl* genotype (Cline, 1980). da^+ is not required in the germline, since $da^-(da^2/da^3)$ pole cells produce fertile gametes; however, mitotic recombination failed to yield significant $da^-(da^2$ or $da^3)$ somatic clones, suggesting da^+ may be essential during epidermal development (Cronmiller and Cline, 1987). Embryos, homozygous for lethal *da* alleles, have a reduced CNS, lack all peripheral neurons, and have no external sensory structures (Caudy *et al.*, 1988a). Adult flies heterozygous for a deletion of the achaete-scute (*ASC*) genes and simultaneously heterozygous for *Df(da)* (also $da^2/+$ or $da^5/+$) exhibit characteristic bristle defects (Dambly-Chaudière *et al.*, 1988). Hemizygosity for da^+ reduces the number of supernumerary bristles in *Hw* mutants (Dambly-Chaudière *et al.*, 1988).

In addition to its zygotic phenotype, da^1 exhibits two separable maternal effects. There is a female-specific maternal effect: At 22° and 25°, homozygous da^1 females produce no daughters, while at 18°, they produce approximately 20% as many daughters as sons (Cline, 1976). At 29°, da^1 displays a sex-nonspecific maternal effect. Homozygous females are reversibly sterile; they lay eggs that show little or no development (Cline, 1976). Sterility of da^1 females at high temperature results from a defect in the somatic gonad rather than in the germline, since da^- germ cells in wild-type ovaries produce normal eggs which support full viability of sons (Cronmiller and Cline, 1987).

The female-specific maternal effect has a temperature-sensitive period which includes the last 60 hr of oogenesis and the first 3 hr of development (Cline, 1976). This maternal effect is also observed in crosses of da^1 females to *D. simulans* males (Watanabe and Yamada, 1977). The female-lethal maternal effect is autonomous to the germline, as demonstrated by transplantation of

da^1 , or da^2/da^3 pole cells into + hosts (Cline, 1983b; Cronmiller and Cline, 1987). Female zygotes from da^1 mothers at 25° die as embryos. Such lethal female embryos show consistent abnormalities in midgut formation, and in about 50% of the abnormal embryos, shortening of the germ band fails, while anus and posterior spiracles open on the dorsal surface behind the head segments (Counce). Female embryos from da^1 mothers also show consistent defects in the CNS, which is either reduced in width or shows abrupt bends or twists; abnormally formed gut often extends into the CNS (Caudy *et al.*, 1988a). The majority of daughters of da^1 mothers surviving at 18° are morphologically abnormal, often missing structures from one or more imaginal discs or abdominal histoblasts, and frequently with duplication of structures (Cline, 1976).

Though it was reported that daughters of homozygous da^1 females could be rescued by cytoplasmic injection (Bownes *et al.*, 1977), the apparent rescue was subsequently found to result from nonspecific effects that may have slowed the early development of females who are on the threshold of surviving (Cline, 1984; see also Muir and Bell, 1987). Gynandromorphs can survive the lethal maternal effects, but there is no localized lethal focus. Diplo-X tissue develops abnormally alongside normally developing haplo-X tissue. Survival of the mosaics and their average fraction of diplo-X tissue increases with decreasing temperature (Cline, 1976).

The da^1 maternal effect masculinizes escaper daughters that are homozygous for *mle* (Cline, 1984) and masculinizes triploid intersex (XXAAA) progeny (Cline, 1983a). Females heterozygous for *Sxl* alleles that lead to male development develop as sterile males, mosaic intersexes, or sterile females (depending on the *Sxl* allele), when produced by da^1 mothers (Cline, 1984). The da^1 female lethal maternal effect is unaffected by *tra* or *dsx* (Bell, 1954; Colainne and Bell, 1968). However, daughters of da^1/da^1 mothers are almost fully rescued by a single zygotic dose of *Sxl^{MI}* and to a limited degree by a duplication for *Sxl⁺* (Cline, 1978). Conversely, zygotic *Sxl⁻* enhances the *da* maternal effect. Females with reduced *Sxl* dose (*Sxl⁻/+*) fail to survive from da^1/da^1 mothers at the semipermissive 18° (Cline, 1978). A strong dominant *da* maternal effect [$da^1/+$, *Df(2L)da/+*, or $da^2/+$ mothers] is observed when female progeny are doubly heterozygous for *Sxl⁻* and *sis-a⁻* (Cline, 1986, Genetics 113: 641-63; Cronmiller and Cline, 1986, 1987). The maternal effect of da^1 is made semidominant also by *E(da)* (*cis* or *trans* to da^1) in the mother (Mange and Sandler, 1973; see also Cline, 1980). The zygotic da^+ dose itself does not affect expression of *Sxl⁺* sex determination function (Cronmiller and Cline, 1986).

alleles:

allele	origin	synonym	ref. ^α	comments	molecular data
da^1	spont		1	(= <i>da</i>); viable; temperature sensitive hypomorph; strong daughterless maternal effect above 22°; lethal with <i>Df</i> or amorphic alleles; lethal at 29°	900 bp insertion of middle repetitive DNA in untranslated leader
da^2	EMS		2	apparent amorph; recessive lethal; lethal	normal Southern

allele	origin	synonym	ref ^α	comments	molecular data
<i>da</i> ³	EMS		2	with <i>da</i> ¹ , <i>Df</i> , or other lethal alleles apparent amorph; recessive lethal; lethal with <i>da</i> ¹ , <i>Df</i> , or other lethal alleles	normal Southern
<i>da</i> ⁴	HD	<i>da</i> ^{A73}	6	recessive lethal	500 bp deletion
<i>da</i> ⁵	EMS	<i>da</i> ^{11B31}	3	recessive lethal; lethal with <i>da</i> ¹ , <i>Df</i> or other lethal alleles	normal Southern
<i>da</i> ⁶	EMS		5	apparent amorph	
<i>da</i> ⁷	HD	<i>da</i> ^{Pa}	4	viabile; reduced viability with <i>da</i> ¹ ; partially complements <i>da</i> ¹ female lethal maternal effect; lethal with <i>da</i> ² , <i>da</i> ³ , <i>Df</i> , or <i>da</i> ⁵	<i>P</i> element insertion in untranslated leader
<i>da</i> ⁸	X ray	<i>da</i> ^{X9.3}	6	recessive lethal	5' end rearrangement
<i>da</i> ⁹	X ray	<i>da</i> ^{X80}	5, 6	(= <i>In(2L)da</i> ⁹); recessive lethal	inversion breakpoint within gene
<i>da</i> ¹⁰	X ray	<i>da</i> ^{X136}	6	recessive lethal	4 kb deletion
<i>da</i> ¹¹	X ray	<i>da</i> ^{X251}	6	(= <i>T(2;3)da</i> ¹¹); recessive lethal	translocation; breakpoint within gene

^α 1 = Bell, 1954, *Genetics* 39: 958-59; 2 = Cronmiller and Cline, 1987, *Cell* 48: 479-87; 3 = Caudy, Grell, Dambly-Chaudière, Ghysen, Jan, and Jan, 1988, *Genes Dev.* 2: 843-52; 4 = Cronmiller, Schedl, and Cline, 1988, *Genes Dev.* 2: 1666-76; 5 = Brand and Campos-Ortega, 1988, *Roux's Arch. Dev. Biol.* 197: 457-70; 6 = Caudy, Vässin, Brand, Tuma, Jan, and Jan, 1988, *Cell* 55: 1061-67.

cytology: Placed in 31E based on localization by *in situ* hybridization of *da*⁷ *P*-element insertion (Cronmiller, Schedl, and Cline, 1988).

molecular biology: The genomic region was cloned by transposon tagging from *da*⁷ (Cronmiller, Schedl, and Cline, 1988) and by chromosome walking (Caudy *et al.*, 1988b). Approximately 30-kb walks were restriction mapped; the *da* transcription unit was localized by the demonstration of altered transcripts on Northern blots of *da*¹ and *da*⁷ RNAs and by mapping rearrangements of several mutant alleles within a 6-kb genomic region. Two transcripts are detected by Northern analysis, 3.2 and 3.4-3.7 kb; both transcripts are present in male and female adults and at all stages of development [including unfertilized eggs (Cronmiller)], although the smaller transcript is enriched in unfertilized eggs and in 0-2.5 hr embryos. cDNA sequence analyses revealed a single intron, approximately 1.5 kb, located upstream of the initiating AUG. The inferred amino acid sequence predicts a protein of 710 aa, with a molecular weight of 74,000 daltons. From the amino acid sequence, the *da* protein is a member of the Helix-Loop-Helix family of DNA binding proteins. Within this family, the most extensive sequence similarity exists between *da* and the human kE2 enhancer binding protein, E12, where the two proteins are 78% identical over 93 amino acids (Murre *et al.*, 1989a). *In vitro*, *da* protein and E12 form heterodimers and bind to DNA (Murre *et al.*, 1989b). The predicted *da* protein also includes two short (27 aa) regions of similarity to the *bcd* protein, one being the His-Pro repeat that is also present in *prd*.

da: see *dar*

Dab: Disabled

location: 3-[44].

synonym: *dab*.

references: Gertler, Bennett, Clark, and Hoffmann, 1989, *Cell* 58: 103-13.

phenotype: A dominant enhancer of the lethal phenotype of genotypes deficient in *Abl* function. *Abl*¹/*Abl*⁻, which ordinarily survive to late pupal or adult stages, die as late embryos or early larvae with abnormal CNS development when heterozygous for *Dab* mutants or deficiencies; such *Dab*⁻ or *Dab*⁻ heterozygotes are normal in the presence of *Abl*⁺. Double mutant embryos, *Abl*⁻ *Dab*⁻, are lethal and have few or no proper longitudinal or commissural axons in the CNS. Homozygous *Dab*⁻ in the presence of *Abl*⁺ results in reduced survival with intermediate levels of CNS disruption.

alleles: Two ethyl-methanesulfonate-induced alleles, *Dab*¹ and *Dab*², isolated as *M2* and *M29*, mimic the effects of *Dab* deficiencies; not allelic to *l(3)73Bc*.

cytology: Placed in 73B2-3 based on the enhanced *Abl* phenotype of *Abl*¹¹/*Df(3L)st100.62* = *Df(3L)72F3-7;73B3* but not *Abl*¹¹/*Df(3L)st-j7* = *Df(3L)73A1-2;73B1-2* (Henkemeyer, Gertler, Goodman, and Hoffmann, 1987, *Cell* 51: 821-28).

dachs: see *d*

dachsous: see *ds*

dal: daughterless abnormal oocyte like

alleles: 2-44 (not separated from *abo*).

references: Sandler, 1977, *Genetics* 86: 567-82.

phenotype: External phenotype normal. Shows reduced survival in heterozygous combination with deficiency. Maternal effect embryonic lethal with relative survival of sons less than that of daughters. Maternal effect more severe at 25° than at 19°. Maternal effect reduced by presence of *Y* or *Xh* in zygotes. Presence of *dal*⁺ allele increases survival of offspring of *abo* mothers.

cytology: Placed in 31F-32E based on inclusion in *Df(2L)J-der-39* = *Df(2L)31A-B;32E* but not *Df(2L)J27* = *Df(2L)31B-D;31F* or *Df(2L)Mdh-1J* = *Df(2L)30D-F;31F*.

other information: Complements *abo*, *hup*, and *wd*².

***dar: darky**

location: 1-0 (no crossovers with *sc* in 547 flies).

origin: X ray induced.

discoverer: Fahmy, 1956.

synonym: *da*; preoccupied.

references: 1959, *DIS* 33: 84.

phenotype: Small, heavily melanized flies. Sometimes wings curl upward. Male sterile; viability about 15% wild type; late eclosing. RK2.

***dark: darkener of white-eosin**

location: Autosomal.

discoverer: Bridges, 13i23.

references: 1916, *Genetics* 1: 148.

1919, *J. Exp. Zool.* 28: 347.

phenotype: Specific partial suppressor of *w*^e. RK3.

dark: see *dk*

dark body: see *db*

dark bubbly: see *dkb*

dark carmine: see *dcm*

dark eye: see *dke*

dark eye: see *sf*^{32e}

dark hairy margins: see *dhm*

dark red brown: see *drb*

Darkened eye: see *Dke*

Darkener of apricot: see *Doa*

darkener of white-eosin: see *dark*

darker legs: see *dki*

darker legs: see *thi*^d

darky: see *dar*

dash: discs absent, small, or homeotic (A. Shearn)

location: 3-47.6.

discoverer: Shearn.

references: Shearn, Rice, Garen, and Gehring, 1971, Proc. Nat. Acad. Sci. USA 68: 2594-98.

phenotype: Homozygotes lethal at prepupal-pupal stage; imaginal discs described as homeotic, with the second leg and the wing discs described as like engrailed. Disc and cell autonomous.

alleles:

allele	synonym
<i>dash 1</i>	<i>dash III-10</i>
<i>dash 2</i>	<i>dash XVI-18</i>
<i>dash 3</i>	<i>dash GTK117</i>
<i>dash 4</i>	<i>dash GTN402</i>
<i>dash 5</i>	<i>dash RD317</i>
<i>dash 6</i>	<i>dash RE418</i>
<i>dash 7</i>	<i>dash RF327</i>
<i>dash 8</i>	<i>dash RF605</i>
<i>dash 9</i>	<i>dash RL031</i>
<i>dash 10</i>	<i>dash RZ426</i>
<i>dash 11</i>	<i>dash RZ606</i>
<i>dash 12</i>	<i>dash SP1017</i>
<i>dash 13</i>	<i>dash VD11</i>
<i>dash 14</i>	<i>dash V234</i>
<i>dash 15</i>	<i>dash VD238</i>
<i>dash 16</i>	<i>dash VE238</i>
<i>dash 17</i>	<i>dash jVF101</i>
<i>dash 18</i>	<i>dash VS356</i>
<i>dash 19</i>	<i>dash VS393</i>
<i>dash 20</i>	<i>dash VT367</i>
<i>dash 21</i>	<i>dash jVU215</i>
<i>dash 22</i>	<i>dash VV183</i>
<i>dash 23</i>	<i>dash VZ406</i>
<i>dash 24</i>	<i>dash WA160</i>

Dash: see *Abl*

Dat: Dopamine-N-acetyltransferase

location: 2-107 (within 0.17 unit to the left of *sp*).

references: Maranda and Hodgetts, 1977, Insect Biochem. 7: 33-43.

Huntley, 1978, Ph.D. Thesis, University of Virginia.

Marsh and Wright, 1980, Dev. Biol. 80: 379-87.

phenotype: The structural gene for dopa N-acetylase (EC 3.2.1.5). Molecular weight 2.9×10^4 daltons. Biochemical characterization by Maranda and Hodgetts (1977). Marsh and Wright (1980) argue from developmental profile that enzyme level not coordinately con-

trolled with that of dopa decarboxylase and not hormonally regulated.

alleles: *Dat*^{lo} specifies an enzyme with low activity and relative thermolability. *Dat*⁺/*Dat*^{lo} enzyme level intermediate between those of the two homozygous genotypes.

cytology: Placed in 60B1-10 based on the failure of *Df(2R)Px = Df(2R)60B8-10;60D1-2*, which includes *sp*, to delete the locus and on dosage effects of segmental aneuploidy constructed using *T(Y;2)A160 = T(Y;2)60B-C* and *T(Y;2)H137 = T(Y;2)60D* (Huntley, 1978).

daughterless: see *da*

daughterless abnormal oocyte like: see *dal*

db: dark body

location: 3-45.

references: Chovnick and Talsma, 1966, DIS 41: 58.

phenotype: Body color darker than normal. Male rarely survives, dies in late pupal stage as pharate imago.

Female weakly fertile. RK2.

alleles:

allele	origin	discoverer	comments
<i>db 1</i>	spont		male lethal, female viable
<i>db 2</i>	ENU	Belote	both sexes lethal
<i>db 4</i>	ENU	Belote	both sexes viable
<i>db 5</i>	ENU	Belote	male lethal, female viable
<i>db 83e</i>	X ray	Belote	male lethal, female viable
<i>db 83l</i>	X ray	Belote	male lethal, female viable

*db*⁴ viable in heterozygous combination with *db*¹, *db*⁵, and deficiencies for *db*; however male lethal in combination with *db*² (Belote). *db*² complements other lethals in vicinity and may be an antimorphic allele (Belote).

cytology: Placed in 73C2-D1 by deficiency analysis (Belote).

other information: Possibly allelic to *du*.

db1: dichaeete-beadex-lethal

location: 1-23.0.

origin: I-factor induced.

references: Ellison, 1981, Mol. Gen. Genet. 183: 123-29.

phenotype: Produces a phenotype resembling the double mutant *Bx/+; D/+* at 20°; lethal at 25°.

other information: Reverts at frequency between 10^{-3} and 10^{-2} .

DCgl: see *Cg25C*

dcm: dark carmine

location: 1- (between *y* and *w*).

origin: Spontaneous.

references: Aguado, Galán-Estella, and González-Gulián, 1988, DIS 67: 109.

phenotype: Eye color dark carmine. *dcm;st* eyes orange.

Dcx: see *In(3LR)CxD*

DcxF: see *In(3LR)DcxF*

***dd: displaced**

location: 1-24.3.

discoverer: Bridges, 31d7.

phenotype: Antennae sunken into shortened head; eyes also deformed. Females often sterile. RK2.

cytology: Locus lies between 7C4 and 8C2 (Demerec, Kaufmann, Fano, Sutton, and Sansome, 1942, Year Book

- Carnegie Inst. Washington 41: 191). Further restricted to 7F through 8C2 on the basis of its genetic location to the right of *oc* which is in 7F-8A.

*dd*³: see *ddl*

Ddc: Dopa decarboxylase (T.R.F. Wright and J. Hirsh)

location: 2-53.9+ (.025 centimorgans to the right of *hk* at 53.9 and .002 centimorgans to the right of *amd*).

discoverer: Wright, 1974.

references: Wright, Bewley, and Sherald, 1976, *Genetics* 84: 287-310.

Wright, Beerman, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, *Chromosoma* 83: 45-58.

Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, *Mol. Gen. Genet.* 188: 18-26.

phenotype: Structural gene for dopa decarboxylase [DDC, 3-4-dihydroxy-L-phenylalanine-carboxylase (EC 4.1.28)] which catalyzes the decarboxylation of dopa to dopamine (Lunan and Mitchell, 1969, *Arch. Biochem. Biophys.* 132: 450-56) and 5-hydroxytryptophan to serotonin (5-hydroxytryptamine) but not tyrosine to tyramine (Livingstone and Tempel, 1983, *Nature* 303: 67-70). Native DDC isolated from mature larvae is a homodimer with subunit molecular weight 54 kd (Clark, Pass, Venkataraman, and Hodgetts, 1978, *Mol. Gen. Genet.* 162: 287-97). Distinct DDC isoforms are generated in the CNS and hypoderm by alternate splicing of the *Ddc* primary transcript; the CNS isoform differs by the addition of 35 amino acids at the amino terminus (Morgan, Johnson, and Hirsh, 1986, *EMBO J.* 5: 3335-42). The predicted subunit molecular weights of these are 57.1 and 53.4 kd, respectively. DDC requires pyridoxal-5-phosphate for activity and is strongly inhibited by heavy-metal ions and the sulfhydryl reagent, N-ethylmaleimide. Initial velocity constants determined by Black and Smarrelli (1986, *Biochim. Biophys. Acta* 870: 31-40). The dopamine produced by DDC is necessary to effect sclerotization of the cuticle, being further metabolized both to N-acetyldopamine and N-β-alanyldopamine, which after oxidation to their respective quinones, crosslink cuticular proteins. Thus in adults and white prepupae more than 90% of the DDC activity is located in the epidermis (Lunan and Mitchell, 1969; Scholnick, Morgan, and Hirsh, 1983, *Cell* 34: 37-45). Some DDC activity (~5%) is found in the central nervous system of white prepupae and adults where it produces the neurotransmitters dopamine and serotonin [Wright, 1977, *Amer. Zool.* 17: 707-21; Livingstone and Tempel, 1983; White and Vallés, 1985, *Molecular Basis of Neural Development* (Edelman, Gell, and Cowan (eds.). John Wiley and Sons, N.Y., pp 547-63)]. The limited amounts found in the ovaries (Wright, Steward, Bentley and Adler, 1981, *Dev. Genet.* 2: 223-35) and proventriculus (Wright and Wright, *Proc. Int. Congr. Genet.*, 15th, 1978, Part I, p. 615) are localized in associated neural ganglia (Konrad and Marsh, 1987, *Dev. Biol.* 122: 172-85). Five peaks of DDC activity evident during development: at the end of embryogenesis, the two larval molts, pupariation, and eclosion (Marsh and Wright, 1980, *Dev. Biol.* 80: 379-87; Kraminsky, Clark, Estelle, Gietz, Sage, O'Conner, and Hodgetts, 1980, *Proc. Nat. Acad. Sci. USA* 77: 4175-79). The largest peak, which occurs at pupariation, is induced by a coincident ecdysone peak of the

molting larvae (Marsh and Wright, 1980) and has been shown to be attributable to a rapid increase in translatable DDC mRNA following administration of 20-OH-ecdysone (Kraminsky *et al.*, 1980). Ecdysone induces *Ddc* expression in the mature larval epidermis within two to four hrs (Kraminsky, *et al.*, 1980; Clark, Doctor, Frisstrom, and Hodgetts, 1986, *Dev. Biol.* 114: 141-50). Since cycloheximide addition is sufficient to largely abolish this induction, it appears that this response is an indirect action of ecdysone. A different response of *Ddc* to ecdysone occurs in cultured imaginal discs; *Ddc* induction occurs only subsequent to withdrawal of the hormone (Clark *et al.*, 1986).

Most mutations in *Ddc* are homozygous or hemizygous lethal. The effective lethal phases of the first eight lethal alleles, *Ddc*^{nl}-*Ddc*^{ns}, were almost identical. As hemizygotes over *Df(2L)TW130* almost all mortality is late embryonic with actively moving larvae, exhibiting unpigmented cephalopharyngeal apparatuses and denticle belts, unable to hatch. When homozygous there is a fairly uniform shift in effective lethal phases with mean mortalities from all eight alleles in the cross of *Ddc*^{nl}/*CyO* × *Ddc*^{nl}/*cn bw* being 13.6% embryonic, 14.1% larval, and 4.8% pupal (Wright and Wright, 1978). Many larvae hemizygous for lethal alleles, or homozygous deficient for *Ddc*, when mechanically released from the egg membranes, continue development to the 3rd larval instar and to the pharate adult stage.

Genotypes which produce individuals with drastically reduced DDC activities (~0.5-5% of wild type) exhibit an "escaper" phenotype characterized by incomplete pigmentation and sclerotization of the cuticle; developmental time can be prolonged for as many as four or five days; puparia are easily scored showing melanization at each end of the greenish-gray pupa case; adults often die or get stuck in the food within 24 hr of eclosion; macrochaetae may be very thin, long, and straw-colored or colorless; the whole body remains light, *i.e.*, doesn't take on its normal pigmentation; abdominal markings are apparent but do not darken; upon aging a few hours wing axillae become melanized similar to the phenotype of *sp*, leg joints also become melanized perhaps due to the phenoloxidase wound reaction brought on by ruptures of weakened cuticle; flies walk on tibiae rather than tarsi, but leg movements appear to be coordinated (Wright, Bewley, and Sherald, 1976). Genotypes that produce flies exhibiting the "escaper" phenotype include heteroallelic intragenic complementing heterozygotes with less than 5% of the expected number of survivors (Wright, Bewley, and Sherald, 1976), hemizygotes of the *ts* allele *Ddc*^{ts2} raised continuously at 22° or 25°, or homozygotes for *Ddc*^{ts1} or *Ddc*^{ts2} exposed to the restrictive temperature 30° for 24- or 48-hour pulses at the end of the pupal stage (Wright).

Ddc temperature-sensitive mutants have been reported to show reduced learning after a three-day period at the restrictive temperature (Tempel, Livingstone, and Quinn, 1984, *Proc. Nat. Acad. Sci. USA* 81: 3577-81). However, these results cannot presently be reproduced by other investigators (see Tully, 1987, *Trends in Neurosci.* 10: 330-35; Hirsh, 1989, *Dev. Genet.* 10: 232-38). It is possible that this lack of reproducibility is due to the accumulation of genetic modifiers. In homozygous deficient larvae normally-serotonin-containing neurons

lack immunologically detectable serotonin but display normal levels of uptake of exogenously supplied serotonin (Valles and White, *J. Neurosci.* 6: 1482-91).

Further studies of these *Ddc*⁻ larvae, on which catecholamine histofluorescence studies were performed, revealed novel neuronal subsets lighting up, which become fluorogenic earlier than the wild-type-like neurons in the mutant CNS (Budnik, Martin-Morris, and White, 1986, *J. Neurosci.* 6: 1482-91). Certain serotonin-containing nerve fibers in developing larvae are still able to reach their normal targets in *Ddc*⁻ animals (which therefore are intrinsically serotonin-minus), but there is anomalous extra branching associated with the incoming fibers (Budnik, Wu, and White, 1989, *J. Neurosci.* 9: 2866-77). *Ddc* mosaics generated by crossing a transduced *Ddc*⁺ insert into *R(1)w^{VC}* (Gailey, Bordne, Valles, Hall, and White, 1987, *Genetics* 115: 305-11). Such adult mosaics used to reveal no absolute requirement of DDC in any particular portion of epidermis or CNS, but there was low recovery of gynandromorphs with large *Ddc*⁻ patches. Larval mosaics show that DDC-positive neurons always contain serotonin, but some serotonin-positive cells (which were near DDC⁺) have no detectable enzyme protein; hence, the serotonin phenotype can be nonautonomous (Valles and White, 1990).

In addition to the naturally occurring alleles, *Ddc*^{RE}, *Ddc*^{RS}, and *Ddc*⁺⁴, which are described separately, three surveys of natural populations for *Ddc* variants have been reported. Estelle and Hodgetts (1984, *Mol. Gen. Genet.* 195: 434-41) measured DDC levels in 109 strains isogenic for second chromosomes isolated independently by Bewley (1978, *Biochem. Genet.* 16: 769-75) from collections at Raleigh, NC, Bloomington, IN., and Webster Groves, MO. (WGM). Two (WGM) strains (including *Ddc*⁺⁴) had increased activities and two had reduced activities when compared with a Canton-S control. Marsh and Wright report DDC activities from twelve different wild-type strains maintained in laboratories for many years. Relative to Oregon-R (*Ddc*^C) females, they ranged from a low of 68% for Urbana males to a high for Canton-S females (180%) and males (130%) with most strains with activities between Oregon-R and Canton-S. Aquadro, Jennings, Bland, Laurie-Ahlberg, and Langley (1984, *Genetics* 107: s3) surveyed forty-six second chromosome lines isolated from five natural populations for restriction fragment variations in the 80kb region surrounding *Ddc* and for adult DDC activity. No consistent pattern of association between level of DDC activity and restriction site haplotype was apparent although the lines showed a two-fold variation in DDC activity. Two lines with 5kb and 1.5kb inserts within an intron and at the 5' end of *Ddc* showed normal adult DDC activities.

The temperature-sensitive periods causing lethality for *Ddc*^{ts2} homozygotes are primarily during embryogenesis and late in the third larval instar. Heat shocks, 30° for 24 or 48 hr, during metamorphosis do not increase lethality significantly but produce adults with the extreme "escaper" phenotype. DDC in extracts from adult *Ddc*^{ts1} and *Ddc*^{ts2} homozygotes is significantly more thermolabile than that from wild-type controls. DDC from *Ddc*^{ts1/+} heterozygotes is much less labile showing a biphasic inactivation curve. *Ddc*^{ts2/+} DDC is no more thermolabile than wild-type DDC (Wright, unpublished

data).

Genotypes with reduced levels of DDC activity, e.g. *Ddc*ⁿ⁵/*Ddc*ⁿ⁸ and *Ddc*ⁿ¹/*Ddc*ⁿ⁸ with less than 4% DDC activity, are not more sensitive to dietary alpha methyl dopa nor are genotypes with increased levels of DDC activity more resistant (Marsh and Wright, 1986, *Genetics* 112: 249-65). In fact, the reverse may be true: reduced DDC, more resistant; increased DDC, more sensitive.

alleles: The numbered superscripts of many of the alleles were previously preceded by n to indicate null; the n's have been removed, but the numbers retained.

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>Ddc</i> ¹	EMS	Wright	<i>l(2)E7</i>	5	32.6
<i>Ddc</i> ²	EMS	Wright	<i>l(2)E57</i>	5	53.3
<i>Ddc</i> ³	EMS	Wright	<i>l(2)E61</i>	5	51.7
<i>Ddc</i> ⁴	EMS	Wright	<i>l(2)E126</i>	5	31.1
<i>Ddc</i> ⁵	EMS	Wright	<i>l(2)E135</i>	5	33.3
<i>Ddc</i> ⁶	EMS	Wright	<i>l(2)E17</i>	5	28.4
<i>Ddc</i> ⁷	EMS	Wright	<i>l(2)E140</i>	5	43.1
<i>Ddc</i> ⁸	EMS	Wright	<i>l(2)E143</i>	5	39.6
<i>Ddc</i> ⁹	EMS	Wright	<i>l(2)214</i>	5	51.2
<i>Ddc</i> ¹⁰	EMS	Wright	<i>l(2)247r</i>	5	33.7
<i>Ddc</i> ¹¹	EMS + F	Wright	<i>l(2)315</i>	5	48.1
<i>Ddc</i> ¹²	EMS + F	Wright	<i>l(2)332</i>	5	49.4
<i>Ddc</i> ¹³	EMS + F	Wright	<i>l(2)340</i>	5	42.2
<i>Ddc</i> ¹⁴	EMS + F	Wright	<i>l(2)348</i>	5	39.2
<i>Ddc</i> ¹⁵	EMS + F	Wright	<i>l(2)3543</i>	5	53.0
<i>Ddc</i> ¹⁶	EMS + F	Wright	<i>l(2)405</i>	5	32.0
<i>Ddc</i> ¹⁷	γ ray	Wright	<i>l(2)esc4</i>	5	53.5
<i>Ddc</i> ¹⁸	γ ray	Wright	<i>l(2)esc6</i>	5	50.5
<i>Ddc</i> ¹⁹	EMS	Wright	<i>l(2)605</i>	5	50.5
<i>Ddc</i> ²⁰	EMS	Wright	<i>l(2)617</i>	5	31.0
<i>Ddc</i> ²¹	EMS	Wright	<i>l(2)628</i>	5	50.0
<i>Ddc</i> ²²	EMS	Wright	<i>l(2)634</i>	5	37.4
<i>Ddc</i> ²³	EMS	Wright	<i>l(2)637</i>	5	30.7
<i>Ddc</i> ²⁴	EMS	Nüsslein-Volhard		5	39
<i>Ddc</i> ²⁵	EMS	Nüsslein-Volhard		5	42
<i>Ddc</i> ²⁶	EMS	Nüsslein-Volhard		5	54
<i>Ddc</i> ²⁷	γ ray	Cecil	<i>l(2)esc7</i>	5	44 2.2kb deletion in <i>Ddc</i>
<i>Ddc</i> ²⁸	γ ray	Cecil	<i>l(2)esc8</i>	5	43
<i>Ddc</i> ²⁹	γ ray	Cecil	<i>l(2)esc9</i>	5	39
<i>Ddc</i> ³⁰	EMS + γ ray	Wright	<i>l(2)7411</i>	5	30.5
<i>Ddc</i> ³¹	EMS + γ ray	Wright	<i>l(2)7422</i>	5	37.5
<i>Ddc</i> ³²	EMS + γ ray	Wright	<i>l(2)7423</i>	5	23.5
<i>Ddc</i> ³³	EMS + γ ray	Wright	<i>l(2)7426</i>	5	42.7
<i>Ddc</i> ³⁴	EMS + γ ray	Wright	<i>l(2)7443</i>	5	78.5
<i>Ddc</i> ³⁵	DEB	Cecil	<i>l(2)esc10</i>	5	29
<i>Ddc</i> ³⁶	EMS	Brittnacher	<i>l(2)L3</i>	5	37.5
<i>Ddc</i> ³⁷	EMS	Brittnacher	<i>l(2)L6</i>	5	24.0
<i>Ddc</i> ³⁸	EMS	Brittnacher	<i>l(2)L8</i>	5	50.0
<i>Ddc</i> ³⁹	EMS	Brittnacher	<i>l(2)L15</i>	5	29.0
<i>Ddc</i> ⁴⁰	DEB	Cecil	<i>l(2)esc11</i>	5	55
<i>Ddc</i> ⁴¹	DEB	Cecil	<i>l(2)esc12</i>	5	49
<i>Ddc</i> ⁴²	DEB	Cecil	<i>l(2)esc13</i>	5	60
<i>Ddc</i> ⁴³	EMS	Wright	<i>l(2)620</i>	5	57.8; chromosome also lethal over <i>l(2)37Ca</i>
<i>Ddc</i> ^C	spont	Wright		3, 4	100 ^Y
<i>Ddc</i> ^{DE1}	EMS	Wright		1	γ
<i>Ddc</i> ^{lo1}	EMS	Wright	<i>l(2)651</i>	5	76.5
<i>Ddc</i> ^{RE}	spont	Sherald		3, 4	hypomorph ^Y
<i>Ddc</i> ^{RS}	spont	Sherald		3, 4	158 hypermorph ^Y
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allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>Ddc</i> ^{ts1}	EMS	Wright	<i>l(2)248</i>	5	65.5
<i>Ddc</i> ^{ts2}	EMS + F	Wright	<i>l(2)308</i>	5	52.3
<i>Ddc</i> ^{ts3}	EMS	Wright	<i>l(2)607</i>	5	52.5
<i>Ddc</i> ^{ts4}	EMS	Wright	<i>l(2)652</i>	5	43.7
<i>Ddc</i> ^{ts5}	EMS	Wright	<i>l(2)633</i>	5	65.4
<i>Ddc</i> ⁺⁴	spont	Estelle		2	γ

^α 1 = Bishop and Wright, 1987, Genetics 115: 477-91; 2 = Estelle and Hodgetts, 1984, Mol. Gen. Genet. 195: 434-41; 3 = Marsh and Wright, 1986, Genetics 112: 249-65; 4 = Sherald and Wright, 1974, Mol. Gen. Genet. 133: 25-26; 5 = Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26.

^β Specific DDC activity of newly eclosed adult *CyO/Ddc* heterozygotes expressed as a percentage of appropriate *CyO+* controls.

^γ Expanded description below.

cytology: *In situ* hybridization with cloned *Ddc* placed the *Ddc* gene in or very close to 37C1-2 (Hirsh and Davidson, 1981, Mol. Cell Biol. 1: 475-85). The distal breakpoint of *Df(2L)VA17* breaks in 37C1,2 deleting the proximal part of the band (Wright *et al.*, 1981), and at the DNA level this breakpoint is located in the first intron of *Ddc* deleting the 5' (proximal) exon (Gilbert, Hirsh, and Wright, 1984, Genetics 106: 679-94). The smallest deficiency that completely deletes *Ddc* is *Df(2L)TW130*.

molecular biology: *Ddc* was cloned by a two-step screen, first screening for genes encoding hypodermally expressed mRNA's and then screening these clones by *in situ* hybridization (Hirsh and Davidson, 1981, Mol. Cell Biol. 1: 475-85). The sequence of *Ddc* and flanking DNA has been determined by the Hirsh and Marsh laboratories, as shown in the Genbank entries DRODDC and DRODDCG, respectively. (Primary references: Morgan *et al.*, 1986; Eveleth, Geitz, Spencer, Nargan, Hodgetts, and Marsh, 1986, EMBO J. 5: 2663-72). Whereas these entries are in general agreement, there are several differences, including several that affect the predicted reading frames. The open reading frame given in DRODDC shows conservation with the periwinkle tryptophan hydroxylase sequence (Genbank entry CTRTPDC) in several regions, where the DRODDCG sequence indicates alternate reading frames. *Ddc* encompasses four exons, labelled A, B, C, and D, of which B, C, and D are protein encoding. The major mRNA encoded by *Ddc* is in the hypoderm, a 2.1-kb mRNA containing all four exons. As mentioned above, these mRNA's encode different DDC isoforms. A different pattern of *Ddc* exons is predicted in the exon B-C region by Eveleth *et al.*, but these predictions are not consistent with the functional expression analyses in Morgan *et al.* (1986).

Ddc and *amd* share high levels of sequence identity; their 3' ends are separated by a sequence of 2 kb, which contains another transcription unit (Eveleth and Marsh, 1986, Nucleic Acids Res. 114: 6169-83; Black, Pentz, and Wright, 1987, Mol. Gen. Genet. 209: 306-12).

Using the same 7.5 kb *Pst* restriction enzyme fragment that straddles the *Ddc* gene but using different *P* element vector constructs, both Scholnick, Morgan, and Hirsh (1983, Cell 34: 37-45) and Marsh, Gibbs, and Timmons (1985, Mol. Gen. Genet. 198: 393-403) have effected germline transformation of *Ddc*⁺ DNA which rescues *Ddc* mutant homo- and hemizygotes. All except two of the total of 16 transformed strains examined showed

approximately normal levels of DDC activity along with normal tissue and temporal expression of the transposed *Ddc* genes. One strain had the expected level of DDC activity at pupariation but unexpectedly low levels in both sexes of newly emerged adults, and the other strain gave elevated DDC activities at all stages (Marsh, Gibbs, and Timmons, 1985). Of the two X-linked transformants, one was dosage compensated (Scholnick, Morgan and Hirsh, 1983) and the other was not (Marsh, Gibbs and Timmons, 1985).

P-element germline integration (Scholnick *et al.*, 1983; Marsh *et al.*, 1985) initially defined essential *Ddc* regulatory sequences to lie within 2.5 kb of 5' and 1 kb of 3' flanking DNA. These studies were extended by analyses of deletions (Hirsh, Morgan, and Scholnick, 1986, Mol. Cell Biol. 6: 4548-57; Scholnick, Bray, Morgan, McCormick, and Hirsh, 1986, Science 234: 998-1002) and by the detection of promoter sequences conserved between the *Ddc* genes from *D. melanogaster* and *D. virilis* (Bray and Hirsh, 1986, EMBO J. 5: 2305-11). These data indicate that sequences farther than 98 base pairs from the RNA transcription start site are not required for normal temporal regulation of *Ddc* expression in the hypoderm and implicate sequences between -106 and -38 as being necessary for this regulation. This region contains a number of sequence elements conserved between the two evolutionarily related *Ddc* genes. In a wild-type larval CNS (Beall and Hirsh, 1987, Genes Dev. 1: 510-20; Konrad and Marsh, 1987, Dev. Biol. 122: 172-85), *Ddc* is expressed in approximately 85 serotonergic neurons, and in another approximately 45 neurons previously identified as containing catecholamines (Budnick, Martin-Morris, and White, 1986, J. Neuroscience 6: 3682-91) which are presumably dopaminergic neurons. In addition, there is a low level of expression in a network consisting of a subset of glial cells (Beall and Hirsh, 1987). All *cis* elements necessary for neuronal expression of *Ddc* are contained within the 2,200 base pairs of *Ddc* 5' flanking sequences (Bray, Johnson, Hirsh, Heberlein, and Tjian, 1988, EMBO J. 7: 177-88). At least two separate regions are required for normal expression of *Ddc* in the CNS: a distal CNS-enhancer region, extending from ~1000 to 1600 upstream of the transcription start point (Beall and Hirsh, 1987; Johnson and Hirsh, 1989, Genes Dev. 3: 676-86) and a small proximal element I, at -60 (Scholnick *et al.*, 1986; Bray *et al.*, 1988; Bray, Burke, Brown, and Hirsh, 1989, Genes Dev. 3: 1130-45). The distal enhancer contains most if not all of the information controlling cell specificity of *Ddc* expression in the CNS (Johnson and Hirsh, 1989) whereas element I is required for *Ddc* expression in all *Ddc*-expressing neurons. The protein NTF (Neurogenic-element-binding transcription factor) binds to the CNS-specific element I in the proximal promoter (Bray *et al.*, 1988, 1989), and a number of binding factors are found within the distal enhancer (Johnson and Hirsh, 1989). NTF and the factor cf1a, which binds to dopamine-cell-specific regulatory elements in the distal enhancer, have been cloned (Bray *et al.*, 1989; Dynlacht, Attardi, Admon, Freeman, and Tjian, 1989, Genes Dev. 3: 1677-88; Johnson and Hirsh, 1990, Nature 343: 467-70). cf1a encodes a POU homeodomain protein.

Ddc^C: Dopa decarboxylase-C

origin: Made isogenic by homozygosing single first, second, and third chromosomes from the Oregon-R6 isogenic strain from Yale University.

phenotype: DDC activity and resistance to dietary alpha methyl dopa in the normal range for Oregon-R derived stocks. This strain was put through the same genetic manipulations as *Ddc^{RE}* and *Ddc^{RS}* so it could serve as a valid control for those strains.

cytology: Normal.

Ddc^{DE1}: Dopa decarboxylase Differential Expression 1

discoverer: K. Wade.

references: Bishop and Wright, 1987, Genetics 115: 477-91.

phenotype: Hemizygous adults (9% of expected eclose) exhibit an extreme "escaper" phenotype (see *Ddc* above) except macrochaetae are normally pigmented suggesting that *Ddc^{DE1}* is differentially active in the epidermis *vis-a-vis* the bristle-forming cells. Pupa cases of *Ddc^{DE1}* homo- and hemizygotes are wild type. DDC activity in newly eclosed adult *Ddc^{DE1}* homozygotes is $4.4 \pm 0.2\%$ and in hemizygotes is $0.6 \pm 0.1\%$ of wild-type controls. Homozygous late embryos have $4.8 \pm 2.3\%$ activity. In striking contrast homozygous white prepupae have $46.5 \pm 2.8\%$ DDC activity. However, central nervous systems dissected from these *Ddc^{DE1}* homozygous white prepupae show a tissue specific difference having $4.8 \pm 2.3\%$ DDC activity compared to wild-type CNS. Specific DDC activity in *Ddc^{DE1}* homozygotes ranges significantly more than two times DDC levels in *Ddc^{DE1}/Df(2L)TW130* hemizygotes.

DDC from *Ddc^{DE1}* homozygotes, crawling third instar larvae and adults, is less thermostable *in vitro* in comparison to controls. Late *Ddc^{DE1}/Ddc^{DE1}* embryos (16-20 hr) have no detectable mature 2.0 kb *Ddc* RNA and have reduced levels of the 2.3 kb RNA. The precise reason for the differential expression has yet to be established but is not due to position effect variegation (Bishop and Wright, 1987, Genetics 115: 477-91). *Ddc^{DE1}* phenotype rescued by a 7.5kb transformant of *Ddc⁺* DNA.

Ddc^{lo1}: Dopa Decarboxylase low-1

discoverer: T.R.F. Wright.

references: Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26;

phenotype: Some hemizygous adults exhibit the incomplete sclerotization "escaper" phenotype. Not temperature sensitive: hemizygotes being equally viable at 18°, 25°, and 30° (40-56% of expected). DDC from *Ddc^{lo1}* hemizygotes is not more thermolabile *in vitro* than that from wild type. Heterozygous *Ddc^{lo1}/CyO* have about 77% wild type specific DDC activity and *Ddc^{lo1}* homozygotes have 15-30% activity.

cytology: No visible aberration. No detectable difference in the DNA (>50bp).

Ddc^{RE}: Dopa decarboxylase-R^E

origin: Natural variant; Canton-S-like on the basis of restriction enzyme site polymorphisms. Originally reported as being EMS-induced in Oregon-R but probably a Canton-S contaminant. Made coisogenic with *Ddc^C* strain.

discoverer: Sherald.

references: Marsh and Wright, 1986, Genetics 112: 249-65.

phenotype: Dual phenotype of elevated DDC activity and increased resistance to dietary alpha methyl dopa relative to Oregon-R derived controls (*Ddc^C*). Specific DDC activity of newly eclosed adults 158% and DDC crossreacting material (CRM) 156% of the *Ddc^C* control. LD₅₀ for alpha methyl dopa is ~0.4 mM vs. ~0.2 mM for the *Ddc^C* control. Gene dosage studies with *Ddc⁺* and *l(2)amd⁺* demonstrate that increased resistance to alpha methyl dopa is not the result of increased DDC activity. Thus, the dual phenotype is inferred to arise from a coordinated increase in *Ddc⁺* activity and *l(2)amd⁺* activity produced either by accumulated changes in a genetic element (or elements) in the close proximity to the *Ddc* and *amd* genes.

cytology: No visible difference apparent: not a duplication at either the cytological level or at the DNA level.

Ddc^{RS}: Dopa decarboxylase-R^S

origin: Variant isolated from a *sp² bs²* stock. Made coisogenic with *Ddc^C* strain.

discoverer: Sherald.

phenotype: Dual phenotype of elevated DDC activity and increased resistance to dietary and methyl dopa relative to Oregon-R derived controls (*Ddc^C*). Specific DDC activity of newly eclosed adults 141% and DDC crossreacting material (CRM) 137% of the *Ddc^C* control. Interpretation of phenotype identical to that for *Ddc^{RE}*.

cytology: No visible difference apparent: not a duplication at either the cytological level or at the DNA level.

Ddc⁺⁴: Dopa decarboxylase +4

origin: Natural variant in a second chromosome extracted from a Webster Groves, Missouri (WGM) population and made isogenic using marked balancer chromosomes (Bewley, 1978, Biochem. Genet. 16: 769-75).

references: Estelle and Hodgetts, 1984, Mol. Gen. Genet. 195: 434-41.

phenotype: No visible phenotype: *Ddc⁺⁴* overproduces DDC activity at embryonic hatching, the second to third instar molt, and at adult eclosion relative to a Canton-S control: 141%, 150%, and 118% respectively; in contrast, underproduces DDC at pupariation: 50%. These temporal differences are found in epidermis but not in neural tissues where DDC activities are normal. DDC CRM at pupariation and adult eclosion are 49% and 140% respectively of Canton-S CRM. No difference was found in the electrophoretic mobility of non-denatured and denatured DDC molecules. DDC mRNA is 140%, 52%, and 148% of Canton-S at embryonic hatching, pupariation, and adult eclosion respectively indicating that the temporal phenotype is reflected in mRNA levels.

molecular biology: *Ddc⁺⁴* DNA was cloned and examined by acrylamide gel electrophoresis of restriction fragments. Six small restriction length polymorphisms and one restriction site polymorphism exist between *Ddc⁺⁴* and Canton-S DNA. Five of these differences occur in the 5' untranslated leader sequence of the DDC mRNA or in the 4.5 kb of DNA upstream of the transcription start site. Some DNA sequence data have been acquired (Spencer and Hodgetts, unpublished data).

ddd: defective dorsal discs (A. Shearn)

location: 3-18.0.

origin: Induced by ethyl methanesulfonate.

references: Shearn, Rice, Garen, and Gehring, 1971, Proc. Nat. Acad. Sci. USA 68: 2594-98.

Wurst, Hersperger, and Shearn, 1984, Dev. Biol. 106: 147-55.

Simcox, Wurst, Hersperger, and Shearn, 1987, Dev. Biol. 122: 559-67.

phenotype: Homozygous larvae perish between the first larval instar and the prepupal stage; dorsal thoracic imaginal discs, *i.e.* of the pronotum, mesonotum, and metanotum reduced to 3% or less of normal size; all other imaginal discs develop normally. Mutant larvae support the growth of wild-type wing discs; mutant wing discs show very little development in wild-type larval. Mutant cells develop normally in wing discs that contain mixtures of mutant and wild-type cells, as produced by nuclear or cellular transplantation into blastoderms or by somatic exchange. Mutant leg discs transplanted into wild-type hosts can transdetermine to wing development. Studies of temperature-sensitive genotypes indicate that *ddd*⁺ product is not required for normal wing development during embryogenesis. No evidence for a maternal effect in either conditional mutants raised under permissive conditions and switched to restrictive temperatures or in germ-line-transplants of mutant cells into wild-type hosts.

alleles: Thirteen alleles induced by ethyl methanesulfonate by Shearn.

allele	synonym	comments
<i>ddd</i> ¹	<i>l(3)LGA</i>	
<i>ddd</i> ²	<i>l(3)RD310</i>	
<i>ddd</i> ³	<i>l(3)RG436</i>	L3-P lethal
<i>ddd</i> ⁴	<i>l(3)RY507</i>	embryonic lethal
<i>ddd</i> ⁵	<i>l(3)SA519</i>	cold-sensitive allele
<i>ddd</i> ⁶	<i>l(3)SI134</i>	
<i>ddd</i> ⁷	<i>l(3)UH5</i>	
<i>ddd</i> ⁸	<i>l(3)UH64</i>	
<i>ddd</i> ⁹	<i>l(3)VJ449</i>	
<i>ddd</i> ¹⁰	<i>l(3)VK97</i>	
<i>ddd</i> ¹¹	<i>l(3)VU288</i>	
<i>ddd</i> ¹²	<i>l(3)VW100</i>	
<i>ddd</i> ¹³	<i>l(3)WB240</i>	heat-sensitive allele

cytology: Placed in 64D-E by segmental aneuploidy.

***ddl: displacedlike**

location: 1-27.2.

origin: Induced by triethylenemelamine.

discoverer: Fahmy, 1953.

synonym: *dd*³.

references: 1959, DIS 33: 84.

phenotype: Frontal region with antennae sunken into shortened head. Eyes deformed. Thoracic bristles stiff and slightly shortened. Wings frequently misheld. Males sterile; viability slightly reduced. RK2.

alleles: One X-ray-induced allele.

***de: deacon**

location: 1-56.

origin: X ray induced.

discoverer: Muller, 26112.

references: 1935, DIS 3: 29.

phenotype: Body and wings narrow and rectangular. Eyes slightly flattened with oblique cast. RK3.

other information: Possibly an allele of *sl* (1-53.5).

***De: Dented**

location: 2- (between *dp* and *b*).

origin: X ray induced.

discoverer: Belgovsky, 36c.

references: 1937, DIS 8: 7.

phenotype: In heterozygote, most flies show one or two indentations on thorax at front. Homozygote has two smaller, sharper dents. Wings often raised. RK3.

deacon: see *de*

deadlock: see *del*

dec-1: defective chorion 1

location: 1-20.7 (based on 69 *ct-oc* and 93 *ct-sn* recombinants; combined data of Lineruth *et al.*).

references: Gans, Audit and Masson, 1975, Genetics 81: 683-704.

Komitopoulou, Gans, Margaritis, Kafatos, and Masson, 1983, Genetics 105: 897-920.

Lineruth and Lambertsson, 1985, Wilhelm Roux's Arch. Dev. Biol. 194: 436-39.

Lineruth, Lambertsson, and Lindberg, 1985, Mol. Gen. Genet. 201: 375-78.

Lineruth and Lambertsson, 1986, Mol. Gen. Genet. 205: 213-16.

Bauer and Waring, 1987, Dev. Biol. 121: 349-58.

phenotype: Females homozygous for *dec-1*² produce few eggs; chorion thin and fragile, often lost during ovoposition; dorsal appendages much reduced, often absent; chorion appears vacuolated and permits uptake of neutral red. Mosaic studies with *dec-1*² demonstrate that it is a somatically active gene [Wieschaus, Audit, and Masson, 1981, Dev. Biol. 92-103 (fig.)]. Mature egg shell shows lack of organization within the endochorion and accumulation of electron dense material in the vitelline membrane of stage-14 egg chambers. No abnormalities detected in stage-10 oocytes. Three protein products of gene detected; the primary translation product of 130 kd found in stage-10 follicles; this appears to be quickly processed into an 85-kd product, which is in turn processed into a 67-kd protein in stage 13 and 14 egg shells (Bauer and Waring); these products measured as 92, 82, and 76 kd, respectively, by Lineruth and Lambertsson (1985). All three proteins absent in *dec-1* mutants, and they vary coordinately in molecular weight in natural variant alleles.

alleles: Three molecular-weight variants found among strains from natural populations; each variant affects all three forms of the gene product. The majority of strains are as characterized: strains labeled Israel and Krasnodar produce slightly smaller polypeptides, and strains labeled Alma Ata, Frunze, and Shahrinay produce polypeptides that are even smaller (Lineruth, Lambertsson, and Lindberg, 1985, Mol. Gen. Genet. 201: 375-78). These wild-type alleles are designated *dec-1*⁺¹, *dec-1*⁺², and *dec-1*⁺³ respectively.

allele	origin	discoverer	synonym	ref	uα	comments
<i>dec-1</i> ¹	EMS		<i>fs(1)A267</i>	2		
<i>dec-1</i> ²	EMS		<i>fs(1)A384</i>	2, 4		protein products absent
<i>dec-1</i> ³	EMS		<i>fs(1)A1336</i>	2		
<i>dec-1</i> ⁴	EMS		<i>fs(1)A1501</i>	2, 4		proteins 25 kd larger than normal; complements <i>Df(1)ct</i> ^{4b1} , but not

allele	origin	discoverer	synonym	ref	uc ^α	comments
<i>dec-1</i> mutants						
<i>dec-1</i> ⁵	EMS		<i>fs(1)K467</i>	3		
<i>dec-1</i> ⁶	EMS		<i>fs(1)K718</i>	3		
<i>dec-1</i> ⁷	EMS		<i>fs(1)K1090</i>	3		
<i>dec-1</i> ⁸	EMS		<i>fs(1)K1124</i>	3		
<i>dec-1</i> ⁹	EMS		<i>fs(1)K1232</i>	3		
<i>dec-1</i> ¹⁰	EMS		<i>fs(1)K1511</i>	3		
<i>dec-1</i> ¹¹	EMS		<i>11-549</i>	5		
<i>dec-1</i> ¹²	EMS		<i>12-365</i>	5		
<i>dec-1</i> ¹³	EMS		<i>12-403</i>	4,5		protein products absent
<i>dec-1</i> ¹⁴	EMS		<i>12-1873</i>	5		
<i>dec-1</i> ¹⁵	EMS		<i>12-2514</i>	5		
<i>dec-1</i> ¹⁶	EMS		<i>12-3512</i>	5		
<i>dec-1</i> ¹⁷	EMS		<i>12-3907</i>	5		
<i>dec-1</i> ¹⁸	EMS		<i>12-4860</i>	5		
<i>dec-1</i> ¹⁹	EMS		<i>13-453</i>	5		
<i>dec-1</i> ²⁰	EMS		<i>14-963</i>	5		
<i>dec-1</i> ²¹	EMS		<i>13C-57</i>	5		
<i>dec-1</i> ²²	EMS		<i>13C-73</i>	4,5		protein products absent
<i>dec-1</i> ²³	EMS		<i>14A-114</i>	5		
<i>dec-1</i> ²⁴	EMS		<i>14E19</i>	5		
<i>dec-1</i> ²⁵	EMS	Vyse	<i>fs(1)A15</i>	5		
<i>dec-1</i> ²⁶	EMS	L. Girton	<i>fs(1)952</i>	5		
<i>dec-1</i> ²⁷	EMS		<i>fs(1)107</i>	1		
<i>dec-1</i> ²⁸	EMS		<i>fs(1)400</i>	1		
<i>dec-1</i> ²⁹	EMS		<i>fs(1)410</i>	1,4		protein products absent
<i>dec-1</i> ³⁰	EMS		<i>fs(1)764</i>	1		1.6-kb insert in 3.2 to 3.6 kb
<i>dec-1</i> ³¹	EMS		<i>fs(1)1195</i>	1		
<i>dec-1</i> ³²	EMS		<i>fs(1)2325</i>	1		

^α 1 = Bauer and Waring, 1987, Dev. Biol. 121: 349-58; 2 = Gans, Audit, and Masson, 1975, Genetics 81: 683-704; 3 = Komitopoulou, Gans, Margaritis, Kafatos, and Masson, 1983, Genetics 105: 897-920; 4 = Lineruth and Lambertsson, 1986, Mol. Gen. Genet. 205: 213-16; 5 = Mohler and Carroll, 1984, DIS 60: 236-41.

cytology: Placed in 7C1-3 based on its inclusion in *Df(1)ct^{4bl} = Df(1)7B2-4;7C3-4* but not in *Df(1)ct²⁶⁸⁻⁴² = Df(1)7A5-6;7B8-C1*.

molecular biology: Region isolated in a chromosome walk from the proximal breakpoint of *Df(1)ct^{4bl}* (Hawley and Waring, 1988, Genes Dev. 2: 341-49); coordinate 0 placed at *EcoRI* site just proximal to above breakpoint with positive values extending to the left. Sequence from 0 to 6.6 kb (which includes *ct^{4bl}* breakpoint) is complementary to an abundant poly(A)⁺ RNA of 4.0 kb from stage-8 to stage-11 follicles and a less abundant one of 5.8 kb from stage-11 and stage-12 follicles. cDNA's indicate that both messages have a common transcription start site, share several small introns, and that transcription is from right to left. A segment is apparently spliced out of a large exon present in the 5.8-kb message to generate the 4.0-kb message, which contains sufficient coding capacity to encode the 130 kd protein. *Df(1)ct^{4bl}* removes the 3' end of the transcript beginning at approximately the site of the 4.0-kb RNA-specific splice donor. Preliminary sequence determination indicates a N-terminal signal sequence. A 200-base-pair deletion between coordinates 1.9 and 2.7 differentiates the Shahri-nau from the Canton-S wild-type allele and accounts for the molecular weight differences between their protein products.

dec-2

location: 1-23.1.

discoverer: D. Mohler.

synonym: *fs(1)C2*.

phenotype: Homozygous females rarely ovoposit. Eggs have thin chorion with thin respiratory appendages.

cytology: Placed in 7E10-8A5.

decapentaplegic: see *dpp*

deep orange: see *dor*

defective: see *df*

defective chorion 1: see *dec-1*

defective dorsal disc: see *ddd*

defective in phototaxis plasticity: see *dipp*

Defective in phototaxis plasticity: see *Dipp*

deflected wing: see *dfw*

Deformed: see *Dfd*

deformed antennae: see *dfa*

deformed eye: see *dfe*

deformed tergites: see *dft*

deformed wings: see *dwg*

degenerated spermatheca: see *dg-a*

del: deadlock (T. Schüpbach)

location: 2-{54}.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus.

phenotype: Female-sterile; homozygous *del¹* females often have underdeveloped ovaries which seem to lack germ cells altogether. In some females a small number of developing egg chambers are found. These never develop beyond the first few stages of oogenesis. Females homozygous for *del²* and *del³* have normal numbers of developing egg chambers of all stages in their ovaries. The eggs produced by the females very frequently have fused dorsal appendages, or lack dorsal appendages altogether, and remain unfertilized.

alleles: *del¹* to *del⁴* are isolated as *WK*, *HN*, *PS*, and *WH*, respectively.

cytology: Placed in 37F5-39F1, since uncovered by *Df(2L)TW65 = Df(2L)37F5-38A1;39E2-F1*.

Delayed recovery: see *Dly*

Delta: see *DI*

delta vein: see *thv^d*

delta wing: see *dta*

deltex: see *dx*

deltoid veins: see *dlv*

den: denervated

location: 2- {39}.

references: de la Concha, Dietrich, Weigel, and Campos-Ortega, 1988, Genetics 118: 499-508.

genetics: Loss-of-function of *den⁺* results in considerable neural hypoplasia (Brand and Campos-Ortega).

cytology: Located in 31B-D.

Dented: see *De*

Deoxyribonuclease-1: see *DNase-1*

***dep: depressed**

location: 1-18.
discoverer: Bridges, 13d.
references: Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. No. 237: 67 (fig.).
phenotype: Wings turned down at tips, flat from side to side. Somewhat variable but does not overlap wild type. RK2.

depilated: see *dpt*

***depl: depressedlike**

location: 1-23.
origin: Recovered among progeny of flies treated with Janus green.
discoverer: Muller, 28e20.
synonym: *dep^r: depressed-roof*.
references: 1935, DIS 3: 29.
phenotype: Wings droop at sides. Flies dark and weak; bristles fine. Viability variable, about 20% wild type. RK3.

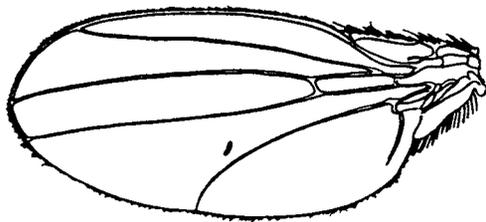
depressed: see *dep*

depressedlike: see *depl*

***der: deranged**

location: 1-57.2.
origin: Induced by triethylenemelamine.
discoverer: Fahmy, 1953.
references: 1958, DIS 32: 69.
phenotype: Thoracic hairs deranged, many point toward midline. Wings usually obliquely upheld and twisted, bringing inner margins together. Overlaps wild type. Good viability in both sexes, but female fertility reduced. RK3.

DER: see *Egfr*



det: detached

From Bridges and Brehme, 1944, Carnegie Inst. Washington Publ. No. 552: 54.

det: detached

location: 3-72.5.
origin: Spontaneous.
discoverer: Nichols-Skoog, 35k27.
phenotype: Posterior crossveins detached from longitudinals at one or both ends and may be absent. Wings occasionally folded back under or folded flat at middle. Eyes sometimes rough and bulging. Wings slightly spread. Bristles tend to break; scutellars occasionally doubled. RK3.

Detached: see *Dt*

dev: devenir

location: 3-40.9.
origin: Induced by ethyl methanesulfonate.
discoverer: Kennison, 1983.
references: Kennison and Tamkun, 1988, Proc. Nat. Acad. Sci. USA 85: 8136-40.
phenotype: Isolated as a dominant suppressor of *Pc* mutations. Associated with recessive larval lethality. Lowered survival and loss of humeral bristles in combination with *D³* may indicate allelism with *Dichaete*, but it is difficult to ascertain.
alleles: Two alleles induced by ethyl methanesulfonate.
cytology: Placed in 70C2-D3 based on its inclusion within *Df(3L)fz-CAL5 = Df(3L)70C2;70D3* and exclusion from *Df(3L)fz-M21 = Df(3L)70D2-3;71E4-5*.

***df: defective**

location: 1-32.5.
origin: Spontaneous.
discoverer: Bridges, 1513.
phenotype: Head bristles around ocelli missing. Viability poor. RK3.

***dfa: deformed antennae**

location: 1-13.9.
origin: Induced by 2-chloroethyl methanesulfonate.
discoverer: Fahmy, 1956.
references: 1959, DIS 33: 84.
phenotype: Wings short, broad, either convex or concave, and abnormally held. Eyes small, dark, and rough. Bristles short, stiff, and occasionally bent. Trident pattern more pigmented. Abnormal antennae and arista. Males viable and fertile. Females sterile. RK2.

Dfd: see *ANTC*

***dfi: deformed eye**

location: 3- (near *D*).
origin: Recovered among descendants of heat-treated flies.
discoverer: Ives, 32c.
synonym: *rough III*.
references: Plough and Ives, 1934, DIS 1: 34. 1935, Genetics 20: 42-69.
phenotype: Eyes roughish, reduced, and misshapen. Overlaps wild type. Female sterile; poorly viable. RK3.

deflected wing: see *dfw*

deformed antennae: see *dfa*

***dft: deformed tergites**

location: 1-33.7.
origin: Induced by 2-chloroethyl methanesulfonate.
discoverer: Fahmy, 1956.
references: 1959, DIS 33: 84.
phenotype: Small fly with small, slightly rough eyes. Wings slightly divergent or upheld, abnormally shaped with occasional incision of the inner margin. Bristles slightly thinner and shorter with one or both postscutellars frequently absent, and a dorsocentral occasionally missing. Abdominal segmentation deformed to various degrees; abdominal hairs fewer and deranged. Males poorly fertile; viability about 50% wild type. RK2.

degenerated spermatheca: see *dg-a*

dfw: deflected wing

location: 1-21.6.

origin: Induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine.

discoverer: Fahmy, 1955.

references: 1959, DIS 33: 84.

phenotype: Wings slightly divergent and upheld to various degrees, often twisted on their axes. Inner margins frequently incised; occasionally, wing membranes separated by fluid. Eyes slightly smaller. Males viable and fertile. Females sterile; viability reduced. RK2.

alleles: One X-ray-induced allele.

dg: Don Giovanni (J.C. Hall)

location: 1- (between *y* and *cv*).

origin: Spontaneous in Canton-S stock.

discoverer: Gailey.

references: Gailey, Jackson, and Siegel, 1984, Genetics 106: 613-23.

Gailey and Siegel, 1989, Anim. Behav. 38: 163-89.

phenotype: Males not conditioned by courtship of fertilized females, apparently because they fail to elicit the appropriate cues from them (Gailey and Siegel, 1989); this means, further, that after a *dg* male courts a fertilized female, a wild-type male will not show the usual depressed courtship of this female; yet if the fertilized female is first courted by a wild-type male, a *dg* male will exhibit depressed courtship of her; females fertilized by *dg* males do not effectively modify subsequent courtships directed at them by any male type (Gailey and Siegel, 1989). *dg* males can, in general, learn, *i.e.* also in artificial test situations such as those using shocks and odorants (Siegel, Hall, Gailey, and Kyriacou, 1984, Behav. Genet. 14: 425-52) or involving females to which quinine is applied (Ackerman and Siegel, 1986, J. Neurogenet. 3: 111-23).

***dg-a: degenerated spermatheca**

location: 3-75.5.

origin: Spontaneous.

discoverer: Collins, 21a.

references: Wexelsen, 1928, Genetics 13: 389-400 (fig.).

phenotype: Adult females show degeneration and pigmentation of epithelial cells of spermathecae 24 hr or more after eclosion. Viability and fertility good. Penetrance 100%. RK3.

dgl: double glazed

location: 2-{51}.

references: McGill, Chia, Karp, and Ashburner, 1988, Genetics 119: 647-61.

cytology: Placed in 34C4-35C5 based on its inclusion in the *Sco* transposition.

dh: see *eg*²

***dhm: dark hairy margins**

alleles: 3-43.2 (proximal to *th*).

origin: Spontaneous.

references: Barker and Hollingdale, 1970, DIS 45: 39.

phenotype: Wings darker than wild type, have hairy margins and thick veins; increased numbers of abdominal and scutellars bristles. Viability and fertility good.

other information: *l(3)DTS5* may be allelic to *dhm* (Ashburner).

Dhod: Dihydroorotate dehydrogenase

alleles: 3-48.0 (no recombinants with *p*^P among 11,618 offspring of heterozygous mothers).

references: Rawls, Chambers, and Cohen, 1981, Biochem. Genet. 19: 115-27.

Porter and Rawls, 1984, Mol. Gen. Genet. 193: 27-32.

phenotype: Likely to be the structural gene for dehydroorotate dehydrogenase, which catalyzes the fourth enzymatic step of *de novo* pyrimidine biosynthesis [DHODEHase (EC 1.3.3.1)]. Homozygotes for null alleles display less than 3% normal levels of enzyme activity; heterozygotes have half-normal levels; flies with three normal alleles show increased levels. Enzyme appears to be monomeric; no interallelic complementation among null alleles. Enzyme activity located in outer surface of inner mitochondrial membrane in other species; also mitochondrial in *Drosophila*. Homozygotes for null alleles exhibit wing truncation, irregular lengths and distribution of hairs on wing margin, deformed posterior legs, and female sterility, as seen in *r* and *r-l*. Viability normal. Phenotype suppressed, but enzyme level not restored, by *su(r)*. Null mutants suppress eye mottling characteristic of *r-l* in doubly mutant genotypes.

alleles: A naturally occurring variant designated *Dhod*^{low} produces but 25% of normal DHODEHase activity. Twelve EMS-induced null or nearly null alleles are designated *Dhod*¹ through *Dhod*¹². *Dhod*² and *Dhod*¹² are hypomorphic, exhibiting weak wing effects and female fertility. In addition, *Dhod*¹³ and *Dhod*¹⁴ were X ray induced by Vincent (originally designated *Dhod*^{V7} and *Dhod*^{V10}); the former was associated with *In(3R)Dhod*¹³ = *In(3R)85A;89B*. *Dhod*¹⁵ induced by hybrid dysgenesis as *Dhod*^{C2}.

cytology: Placed in 85A4-5 based on its inclusion in the region of overlap of *Df(3R)V2* = *Df(3R)84E6-9;85A3-5* and *Df(3R)G1* = *Df(3R)85A4-5;85A6-11*; coordinates -49 to +19 (Jones and Rawls, 1988, Genetics 120: 733-42).

molecular biology: Included in a 145-kb walk carried out by Jones and Rawls (0 coordinate chosen as the proximal end of the initiating lambda clone, with positive values to the right). A putative 0.8-kb *P* insertion associated with *Dhod*¹⁵ located between coordinates 14.4 and 14.9 kb; the inversion associated with *Dhod*¹³ is broken at 14 kb. Genomic sequence hybridizes to a 1.5-kb transcript that is most abundant in early embryos, moderately abundant in mid embryos, mid pupae and adults; lowest in mid-larval stages; some stages display a 1.6-kb transcript as well; transcribed from right to left. Partial sequence shows homology to bacterial DHODEHase in the 3' end but no discernable homology in the 5' end (Jones, Kirkpatrick, and Rawls, 1989, Mol. Gen. Genet. 219: 397-403).

***di: dimorphos**

location: 1- (near spindle attachment).

origin: Spontaneous.

discoverer: Harnly, 32d10.

references: 1935, J. Exp. Zool. 72: 75-99 (fig.). 1940, DIS 13: 49.

phenotype: Specific lengthener of *vg* wings, especially in males (*di*; *vg* female much like *vg*). At higher temperatures, eyes small and rough, and wings of both sexes approach wild type. RK2 in *vg* male.

dib: disembodied**location:** 3-12.**origin:** Induced by ethyl methanesulfonate.**references:** Jürgens, Wieschaus, Nüsslein-Volhard and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95 (fig.).**phenotype:** Homozygous lethal; no differentiation of cuticle and head skeleton.**alleles:** Three. *dib*¹, *dib*², and *dib*³, isolated as *10F*, *10K*, and *10L*.**cytology:** Placed in 62D-64C; uncovered by terminally-deficient segregant from *T(2;3)D11 = T(2;3)?;64B-C* but not covered by *Dp(3;Y)H141 = Dp(3;Y)61B;62D*.*dibro*: see *fr*^{di}**dic: dicephalic****location:** 3-46.0.**origin:** Spontaneous.**references:** Lohs-Schardin, 1982, Wilhelm Roux's Arch. Dev. Biol. 191: 28-36 (fig.).**phenotype:** A semidominant maternal-effect mutant with low penetrance. Homozygous *dic* females and, to a lesser extent, *dic/+* heterozygotes exhibit variable numbers of abnormal follicles and abnormal oocytes. During vitellogenesis, the oocyte occupies a position in the middle of or lateral to the nurse cells rather than posterior to them; the fifteen nurse cells distribute themselves into two clusters, one at each end of the follicle, instead of remaining together anteriorly, as normally occurs. The larger cluster tends to be located anteriorly. One or several nurse cells may become detached from remainder of cyst [Frey, Sander, and Gutzeit, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 388-93 (fig.)]. Pole-cell transplantation experiments show that the *dic* phenotype results if either the germ line or the soma or both are homozygous for *dic* (Frey and Gutzeit, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 527-31). Chorionic respiratory processes irregular in shape and frequently point toward oviduct rather than germarium, as usually occurs. Eggs of *dic* females form micropyle at both ends, and they are less uniform in size and more irregular in shape than eggs from normal females. Embryonic development restricted to eggs of nearly normal shape. Blastoderm appears normal but no pole cells form. Head furrows and head lobes seen at both ends of embryo. Embryos secreting larval cuticle display (1) anterior denticle belt patterns at both ends, (2) a single abnormal polarity gradient, or (3) posterior pattern elements at both ends; segment showing polarity switch varies. The relative frequencies of these three classes agree with hypothesis that the two ends of the egg develop independently with P (anterior development) = 0.9 for both ends.**Dichaete:** see *D***dichaete-beadex-lethal:** see *dbl***Difl: Drosophila-interferon-like-protein****location:** 3-{27}.**discoverer:** Garen.**phenotype:** Homologous to mouse $\alpha 2$ interferon.**cytology:** Localized to 67A8-10 by *in situ* hybridization.*dihedral*: see *eg*²**Dihydroorotate dehydrogenase:** see *Dhod***dil: dilute****location:** 2-57.**origin:** Spontaneous.**discoverer:** Bridges, 32f22.**phenotype:** Dilutes *bw* to pale yellowish brown and *we*^e, *w*^{e2}, and *w*^{bl} to paler grades. RK3.***dil-3: dilute in chromosome 3****location:** 3- (not located).**discoverer:** Bridges, 1519.**references:** Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 151.**phenotype:** Eye color like maroon, overlaps wild type. RK3.***dil-w^a: dilutor of white-apricot****location:** 3- (not located).**discoverer:** Weinstein.**references:** Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 218.**phenotype:** Lightens *w*^a. RK3.**dilute:** see *dil***dilute in chromosome 3:** see *dil-3**dilute ocelli*: see *po*²*Dilute-1*: see *bw*^{V291}*Dilute-2*: see *bw*^{V30k1}*Dilute-3*: see *bw*^{V30k10}*Dilute-4*: see *bw*^{V30k12}*Dilute-5*: see *bw*^{V30k13}*Dilute-6*: see *bw*^{V30k18}**dilutor of white-apricot:** see *dil-w^a***diminutive:** see *dm***Dimethylnitrosoamine demethylase:** see *Dmnd***dimorphos:** see *di****Din: Dinty****location:** Unknown; associated with a rearrangement.**origin:** X ray induced.**discoverer:** Braver, 55a.**references:** 1955, DIS 29: 70.

Pollock, 1963, DIS 38: 50,

phenotype: In male and heterozygous female, central portion of vein L2 interrupted. Posterior supra-alar bristles absent in 95-99% of females and 97-99.5% of males. Anterior postalar bristles absent in 6-11% of females and 2-6% of males. Wings divergent. Viable and fertile in male and heterozygous female; homozygous lethal. RK2.**cytology:** Associated with *T(1;2;3)Din = T(1;3)3C;63A + T(2;3)39D-73A*.*Dint-1*: see *wg***Dinty:** see *Din*

Dip-A: Dipeptidase-A

location: 2-55.2 (based on 74 *pr-cn* recombinants).

origin: Naturally occurring polymorphism.

references: Voelker and Langley, 1978, *Genetica* 49: 233-36.

Ohnishi and Voelker, 1981, *Biochem. Genet.* 19: 75-85.

phenotype: Structural gene for dipeptidase A [DIP-A (EC 3.4.14.?)], a dimeric molecule based on the formation of hybrid enzymes in heterozygotes for electrophoretic variants. Enzyme molecular weight estimated at approximately 117,000 daltons by gel filtration (Leigh Brown). Substrate specificities determined by Laurie-Ahlberg (1982, *Biochem. Genet.* 20: 407-24). The only *Drosophila* peptidase with glycyl-L-isoleucine-ase activity. Occurs at high levels in the larval midgut.

alleles: In order of increasing anodal migration in 12% starch gel electrophoresis designated *Dip-A*², *Dip-A*⁴, and *Dip-A*⁶. Null alleles superscripted *nNC1*, *nNC2*, *nGB1*, *nGB2*, and *nGB3* recovered from natural populations (Voelker, Langley, Leigh Brown, Ohnishi, Dickson, Montgomery, and Smith, 1980, *Proc. Nat. Acad. Sci. USA* 77: 1091-95). *Dip-A*^{nGB2}/*Dip-A*^{nGB3} shows partial complementation producing *Dip-A*² migration rate (Barkhart, Montgomery, Langley, and Voelker, 1984, *Genetics* 107: 295-306).

cytology: Placed in 41A at heterochromatic-euchromatic junction of 2R based on its inclusion in *Df(2R)M-S2*⁴, in which no loss of euchromatic bands can be detected; the locus is not included in *Df(2)M-S2*⁸ or *Df(2)M-S2*¹⁰, the latter of which has been shown to be deficient for the majority of 2R heterochromatin.

Dip-B: Dipeptidase B

location: 3-53.6 (Laurie-Ahlberg, 1982).

references: Ohnishi and Voelker, 1981, *Biochem. Genet.* 19: 75-85.

phenotype: Structural gene for dipeptidase B [DIP-B (EC 3.4.14.?)], which is virtually monomorphic in natural populations but which differs in electrophoretic mobility from dipeptidase-B variants in *D. simulans*. Substrate specificities determined by Laurie-Ahlberg (1982, *Biochem. Genet.* 20: 407-24).

alleles: *Dip-B*⁴ is the common allele; *Dip-B*⁶, a slow variant, and *Dip-B*^{10NC25}, a nearly null allele, isolated from natural populations (Laurie-Ahlberg, 1982).

cytology: Localized to 87F13-88C2 based on the absence of *D. melanogaster* enzyme from hybrids between *D. melanogaster* and *D. simulans* when the *melanogaster* genome carries *Df(3R)red*³¹ = *Df(3R)87F12-14;88C1-3*.

Dip-C: Dipeptidase-C

location: 3-{50-51}.

references: Ohnishi and Voelker, 1981, *Biochem. Genet.* 19: 75-85.

phenotype: Structural gene for dipeptidase C [DIP-C (EC 3.4.14.?)], for which no genetic variants are known but which differs in electrophoretic mobility from dipeptidase-C in *D. simulans*. Specific for dipeptides with carboxy proline (Laurie-Ahlberg, 1982, *Biochem. Genet.* 20: 407-24).

cytology: Localized to 87B6-9 based on the presence of the *D. melanogaster* enzyme in hybrids between *D. melanogaster* and *D. simulans* when the *melanogaster* genome carries *Df(3R)T45* = *Df(3R)86;87B5-6* but not

when it carries *Df(3R)E-079* = *Df(3R)86F1-2;87B8-10*. None of the three lethal complementation groups (*e*¹⁸⁰ and ²⁸⁰*e*²²⁷, and *e*⁰⁷⁸) also defined by those two deficiencies is associated with *Dip-C* function.

Dipeptidase-A: see *Dip-A*

Diphenol oxidase: see *Dox*

Dipp: Defective in phototaxis plasticity
(J.C. Hall)

location: 1-not mapped.

references: Willmund and Ewing, 1982, *Anim. Behav.* 30: 209-15.

Willmund, Emanns, Eusmann, and Ross, 1984, *J. Insect Physiol.* 30: 431-36.

phenotype: Fails to display long-lasting modification of visual responses (*e.g.*, light/dark choices) that are usually induced by exposure to strong blue light; prolonged depolarizing afterpotential (PDA) of photoreceptor cells, usually induced by such exposure, decays rapidly in the dark, unlike wild type, in which PDA is stable; in optomotor behavior, *Dipp*¹ still shows some responses to moving stimuli after PDA induced, whereas *Dipp*², as does wild type, has such responses switched off by induction of PDA.

alleles: Possibly two mutant alleles, *Dipp*¹ and *Dipp*².

other information: The two mutations here do not complement, given their dominance; yet, they have not been mapped meiotically, so they could be non-allelic.

dipp-: defective in phototaxis plasticity
(J.C. Hall)

origin: Induced by ethyl methanesulfonate.

references: Willmund, Emanns, Eusmann, and Ross, 1984, *J. Insect Physiol.* 30: 431-36.

Speck, Mutz, Ohl, and Spatz, 1984, *J. Insect Physiol.* 30: 437-40.

phenotype: A series of sex-linked non-complementing, mutants with similar phenotypes. Homozygotes fail to display long-lasting decrement in visual response (*i.e.*, light-dark choices) that are usually induced by exposure to strong blue light; prolonged depolarizing afterpotentials of photo receptors occur normally; other aspects of physiological phenotype described in individual entries, which follow.

dipp-3

location: 1-(not localized; said not to be mappable to a single X-chromosome interval).

phenotype: Photoreceptors show subnormal sensitivity, in electroretinogram recordings, after blue light exposure followed by dark adaptation. ERG responses to flickering light (400 or 600 nm) suggest that the two central photoreceptors in each facet are abnormal in their response (Speck, Mutz, Ohl, and Spatz, 1984, *J. Insect Physiol.* 30: 437-40). Fast phototaxis responses show subnormal sensitivity after some kinds of pretreatment.

dipp-4

location: 1-(*v*-centromere).

phenotype: Photoreceptors show somewhat aberrantly high sensitivity, in electro-retinogram recordings, to various wave lengths of light. ERG light-on transient smaller than normal (Speck, Mutz, Ohl, and Spatz, 1984, *J. Insect Physiol.* 30: 4337-40). Fast phototaxis responses show

subnormal sensitivity after exposure to blue light followed by dark adaptation.

dipp-5

location: 1-(*cv-f*).

phenotype: ERG response to flickering light normal (Speck, Mutz, Ohl, and Spatz, 1984, *J. Insect Physiol.* 30: 437-40). Fast phototaxis responses show subnormal sensitivity after exposure to blue light followed by dark adaptation.

dipp-6

location: 1-(not localized; no recombinants from *y cho cv v f/dipp-6* exhibit normal phenotypes as if several factors responsible for mutant behavior of *dipp-6* strain.

phenotype: Photoreceptors show subnormal sensitivity, in electroretinogram recordings, after blue light exposure followed by dark adaptation. Fast phototaxis generally defective. Mutant females exhibit defective mating kinetics (Willmund and Ewing, 1982, *Anim. Behav.* 30: 209-15).

dipp-7

location: 1-(*cv-v*).

phenotype: Photoreceptors show subnormal blue-green light sensitivity, in electroretinogram recordings, after blue light exposure followed by dark adaptation. Fast phototaxis generally defective.

dipp-8

location: 1-(*cv-f*).

phenotype: Fast phototaxis generally defective.

Dipr: see *rn*

***dis: distorted eye**

location: 1-23.

origin: Recovered among progeny of natural-gas-treated fly.

discoverer: Mickey, 49b5.

references: 1951, *DIS* 25: 74.

phenotype: Whole or part of eye roughened. Sometimes bristles absent or doubled. Wings may be roughened with nicked margins and plexus veins. Expressivity variable. RK3.

cytology: Salivary chromosomes appear normal.

Disabled: see *Dab*

discless: see *dsl*

disco: disconnected (J.C. Hall and H. Steller)

locus: 1-53.1.

discoverer: K. Fischbach and M. Heisenberg.

references: Steller, Fischbach, and Rubin, 1987, *Cell* 50: 1139-53 (fig.).

Dushay, Rosbash, and Hall, 1989, *J. Biol. Rhythms* 4: 1-27.

Zerr, Hall, Rosbash, and Sibicki, 1990, *J. Neurosci.* 10: 2749-62.

phenotype: Compound eyes are disconnected from optic ganglia in most mutant individuals, but approximately 5-10% have superficially normal eye-brain connections. Photoreceptor cells initially present but degenerate progressively with age; axons of photoreceptor cells which are still present form plexus beneath the eye. Focus of gene function fate maps to a point well anterior to the focus of either the eye or optic lobe (Fischbach).

Adult defect arises as consequence of a defect in the larval visual nerve (Bolwig's nerve) which fails to connect with its target cells in the central nervous system; subsequently, owing to loss of the pioneer function of Bolwig's nerve, reticular axons fail to innervate their target cells in the developing optic lobes leading to massive degeneration of the optic ganglia during the early pupal stages (Steller and Rubin). Slight disarray of embryonic peripheral nervous system detectable; occasional errant neurons seen. Developing CNS appears normal, but adult brain is abnormal in that certain lateral neurons, which normally express *per* are either absent or do not express *per* (Zerr *et al.*). Larval reacts normally to all stimuli except light. All alleles display significantly reduced viability; death occurs in late pupal stages as pharate adults. In tests of circadian rhythms, eclosion and adult locomotor activity are essentially arrhythmic (Dushay *et al.*). Cyclical expression of *per* protein, which normally occurs in eyes and brain, is fairly robust in *disco*, whether eyes connected to the brain or not (Zerr *et al.*); hence this eye rhythm may be autonomous, *i.e.*, given absence of CNS neuronal staining in mutant adults.

alleles: Four ethyl-methanesulfonate-induced alleles: *disco*¹ (Fischbach) and *disco*², *disco*³ and *disco*⁴ (Steller); all alleles show reduced viability.

cytology: Placed in 14B3-4 on the basis of its being included in *Df(1)82c3k = Df(1)14B3-4;14E* but not *Df(1)80g7d = Df(1)14B3-4;14C6-8*; not covered by *Dp(1;2)r⁺75c = Dp(1;2)14B13;15A9;35D-E*. *Dp(1;4)r⁺* and *Dp(1;4)80g7d* cover *disco*-induced arrhythmicity (Dushay *et al.*).

other information: Not allelic to *bss* or *eas*, two closely linked behavioral mutants in 14B4-13; order = *disco eas bss*.

Discolored: see *bw*^{v2}

Disconnected: see *disco*

Discs absent, small, or homeotic: see *dash*

Discs large: see *dlg1*

Disembodied: see *dib*

Dishevelled: see *dsh*

Dispersed: see *dsp*

Displaced: see *dd*

Displacedlike: see *ddl*

Disrupted: see *dsr*

diss: see *nonA*

Distal into proximal: see *rn*

Distalless: see *Ba*

Distorted eye: see *dis*

Disturbed segmentation: see *dss*

Divergent: see *dv*

Divergent wings: see *dvw*

Divers: see *dvr*

***dk: dark**

location: 3- (not located).
discoverer: Clausen, 20g.
references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 235.
 Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 223.
phenotype: Eye color maroon. Overlaps wild type. RK3.

***dkb: dark bubbly**

location: 2- (to the left of *vg*).
discoverer: Bridges, 38d25.
phenotype: Thorax has dark bubbly longitudinal streak. RK3.

dke: dark eye

location: 2-73.
origin: Spontaneous.
discoverer: Bridges, 38c11.
phenotype: Eye color soft, dull, and dark, like *sf*. *sfi dke* is wild type. RK2.

Dke: Darkened eye

location: 2- (not located).
origin: X ray induced.
discoverer: Hendrix, 1963.
references: 1964, DIS 39: 58.
phenotype: In heterozygotes, eye facets roughened with black-spotted pigmentation, varying from light spotting near margin of eye to heavy pigmentation covering one-half of the eye. A bleached area sometimes appears adjacent to the pigmentation. Effect usually symmetrical. Homozygous lethal. RK3.
cytology: Salivary chromosomes appear normal (Peacock).

***dki: darker legs**

origin: Induced by L-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).
discoverer: Fahmy, 1954.
synonym: *thl-d: thick legs darker*.
references: 1959, DIS 33: 85.
phenotype: Extra pigment in body and legs. Legs slightly shortened, especially in female. Wings small and divergent. Eye shape altered. Viability good in both sexes; female fertility reduced. RK3.

dl: dorsal

location: 2-52.9.
synonym: *mat(2)dorsal*.
references: Nüsslein-Volhard, 1979, Symp. Soc. Dev. Biol. 37: 185-211 (fig.).
 1979, INSERM Symp. 10: 69-82 (fig.).
 Nüsslein-Vollard, Lohs-Schardin, Sander, and Cremer, 1980, Nature 283: 474-76.
 Anderson and Nüsslein-Volhard, 1984, Pattern Formation (Malacinski and Bryant, eds.). Macmillan, New York, pp. 264-89 (fig.).
 Steward and Nüsslein-Volhard, 1986, Genetics 113: 665-78.
phenotype: Embryos produced by homozygous *dl* females form normal cellular blastoderm but at gastrulation develop into yolk-filled tube of dorsal hypoderm. Hair pattern of cuticle characteristic of dorsal hypoderm; ventral structures, such as denticle belts, lacking. Normally, dorsal infoldings occupy entire circumference of embryo. Evidence of anterior-posterior differentiation includes

possible mouth armature structures anteriorly, small spiracles posteriorly, and orientation of hairs. The periodicity of stripes of *ftz* expression in pre gastrulation embryos, as revealed by antibody staining, displays the pattern normally characteristic of the dorsum circumferentially in embryos produced by *dl* females (Carroll, Winslow, Twombly, and Scott, 1987, Development 99: 327-32. Embryos produced by *dll/dl* and *dll/Df(2L)TW137* indistinguishable, suggesting *dl* to be amorphic. Penetrance complete; expression constant. Embryos of *dl*² females lack all structures normally derived from the ventral half of the egg, including mesoderm, endodermal gut, ventral nervous system, and ventral hypoderm.

*dl*¹ and to a lesser extent *dl*², females produce embryos with reduced capacity for neurogenesis in response to an absence of *dl* function (Campos-Ortega, 1983, Wilhelm Roux's Arch. Dev. Biol. 192: 317-26). *dl* germ line dependent; homozygous germ-line clones produce dorsalized embryos (Schüpbach and Wieschaus, 1986, Dev. Biol. 113: 443-48).

Embryos of *dll*+ females produced at 29° develop into comparatively normal-looking larvae; they mainly lack internal organs, such as mesoderm and parts of the anterior and posterior gut; often ventral hypoderm including denticle belts reduced; phenotype sensitive to genetic background. At 22°, *dll*+ females produce normal embryos. Developmental fate of ventrally located cells on cellular blastoderm apparently shifted to that of more dorsally located cells. The phenotype of embryos produced by *dll/dl* females partially rescued by the injection of wild-type cytoplasm but not RNA (Santamaria and Nüsslein-Volhard, 1983, EMBO J. 2: 1695-99; Anderson and Nüsslein-Volhard, 1944, Nature 34: 225-27).

Developmental profiles show transcript to be present only in ovaries and pre-cellular-blastoderm stages of embryogenesis. *In situ* hybridization indicates that ovarian transcript accumulates in nurse cells from stage 5 to 11; number of transcripts per genome equivalent in these polytene cells remains low and constant until stage 10, at which time there is a dramatic increase in the relative numbers of transcripts. After a lag of one or two nuclear divisions, transcript begins to accumulate in the oocyte; by stage 12 there is little detectable transcript in the nurse cells. It appears as though the nurse-cell transcript is transferred to the oocyte and thus to the embryo; transcript seems to be uniformly distributed in stage 14 oocytes (Steward, Ambrosio, and Schedl).

dl protein is uniformly distributed throughout cytoplasm of early embryo; in the syncytial blastoderm a gradient of expression is achieved by the graded transport of *dl* protein into nuclei, with the highest nuclear concentrations found ventrally; protein remains cytoplasmic dorsally. Maternal dorsalizing mutants prevent nuclear localization and ventralized embryos show dorsal as well as ventral nuclear localization (Steward, Zusman, Huang, and Schedl, 1988, Cell 55: 487-95; Rushlow, Han, Manley, and Levine, 1989, Cell 59: 1165-77; Steward, 1989, Cell 59: 11179-88; Roth, Stein, and Nüsslein-Volhard, 1989, Cell 59: 1189-1202).

alleles:

allele	origin	synonym	ref ^α	comments
<i>dl</i> ¹	EMS		1	

allele	origin	synonym	ref ^α	comments
<i>dl</i> ²	EMS		2	hypomorphic allele; temperature sensitive
<i>dl</i> ³	EMS		7	
<i>dl</i> ⁴	EMS		7	
<i>dl</i> ⁵	EMS		5	hypomorphic allele
<i>dl</i> ⁶	EMS		5	
<i>dl</i> ⁷	EMS			
<i>dl</i> ⁸	EMS		5	
<i>dl</i> ⁹	EMS	<i>dl</i> ¹⁰³	7	dominant allele
<i>dl</i> ¹⁰	EMS	<i>dl</i> ¹⁶⁰	7	dominant allele
<i>dl</i> ¹¹	EMS	<i>dl</i> ⁷⁶⁰⁷	7	dominant allele
<i>dl</i> ¹²	X ray	<i>dl</i> ^H	4, 5	<i>In(2L)36C;37B</i>
<i>dl</i> ¹³	X ray	<i>dl</i> ^T	4, 5	<i>In(2L)21E-F;36C</i>
<i>dl</i> ¹⁴	EMS	<i>dl</i> ^{B10}	7	dominant allele
<i>dl</i> ¹⁵	X ray	<i>dl</i> ¹⁵	5	
<i>dl</i> ¹⁶	EMS	<i>dl</i> ^{O11}	7	dominant allele
<i>dl</i> ¹⁷	EMS	<i>dl</i> ^{PZ}	3, 7	temperature sensitive
<i>dl</i> ¹⁸	EMS	<i>dl</i> ^{QD}	3, 7	hypomorphic allele
<i>dl</i> ¹⁹	EMS	<i>dl</i> ^{QF}	3, 7	hypomorphic allele
<i>dl</i> ²⁰	EMS	<i>dl</i> ^{Q1}	3	hypomorphic allele
<i>dl</i> ²¹	EMS	<i>dl</i> ^{SC}	3, 7	hypomorphic allele
<i>dl</i> ²²	EMS	<i>dl</i> ^{SG}	3, 7	hypomorphic allele
<i>dl</i> ²³	EMS	<i>dl</i> ^{U5}	7	dominant allele
<i>dl</i> ²⁴	EMS	<i>dl</i> ^{D4}	6	dominant allele
<i>dl</i> ²⁵	EMS	<i>dl</i> ^{D5}	6	dominant allele
<i>dl</i> ²⁶	EMS	<i>dl</i> ^{D6}	6	dominant allele
<i>dl</i> ²⁷	EMS	<i>dl</i> ^{D7}	6	dominant allele

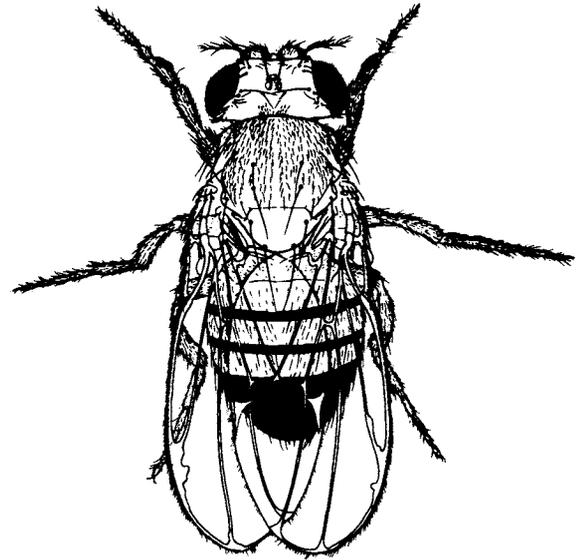
^α 1 = Nüsslein-Volhard, 1979, Symp. Soc. Dev. Biol. 37: 185-211; 2 = Nüsslein-Volhard, 1979, INSERM Symp. 10: 69-82; 3 = Schüpbach and Wieschaus, 1989, Genetics 121: 101-17; 4 = Steward, McNally, and Schedl, 1984, Nature 311: 262-65; 5 = Steward and Nüsslein-Volhard, 1986, Genetics 113: 665-78; 6 = Szabad, Erdélyi, Hoffmann, Szidonya, and Wright, 1989, Genetics 122: 823-35; 7 = Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

cytology: Placed in 36C2-D1 based on its inclusion in *Df(2L)TW137 = Df(2L)36C2-4;37B9-C1* but not *Df(2L)VA18 = Df(2L)36C4-D1;36F-37A*. Further confined to 36C by breakpoints common to *In(1)dl^H* and *In(1)dl^T*.

molecular biology: Genomic clone isolated by Steward, McNally, and Schedl (1984, Nature 311: 262-65). A 14 kb transcription unit encodes a 2.8 kb poly-adenylated transcript; transcription proceeds from right to left; transcript contains two and probably three introns, a 1 kb intron down stream from a 5 kb intron. A 2.3 HindIII-SstI restriction fragment from the 3' exon contains the breakpoints of both *In(2)dl^H* and *In(2L)dl^L* (Steward, Ambrosio, and Schedl, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 223-38). The cDNA sequence indicates that the dorsal protein has 678 amino acids and a molecular weight of 75,450 daltons; the NH₂ terminal portion of the conceptual amino-acid sequence reveals a high level of homology with the *c-rel* proto-oncogene. The C-terminal portion has regions in common with *N* and *en* (Steward, 1987, Science 238: 692-94; 1989, Cell 59: 1179-88; Rushlow, Han, Manley, and Levine, 1989, Cell 59: 1165-77).

other information: Expression of *twi* prevented in embryos produced by *dl* females (Thisse, Stoetzel, El Messal, and Perrin-Schmidt, 1987, Genes Dev. 1: 709-15).

dl: see *dpl*



DI: Delta

From Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 197.

DI: Delta

location: 3-66.2.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 197-201 (fig.).

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 75 (fig.).

Vässin and Campos-Ortega, 1987, Genetics 116: 433-45. Alton, Fechtel, Terry, Miekle, and Muskavitch, 1988, Genetics 118: 235-45.

phenotype: A haplo-insufficient member of the group of neurogenic genes originally described on the basis of its dominant phenotype. Several classes of alleles designated by Vässin and Campos-Ortega based on the phenotype of heterozygous adults: Amorphic and strong hypomorphic alleles display wing veins widened at their junctions with the margin to form delta-like structures; in addition, they show irregular thickening of vein 2, and wings frequently held in divergent attitude; fusion of ommatidia may give rise to disruptions in regular hexagonal array of eye facets; ocelli are slightly enlarged; additional bristles are present on head, thorax, and abdomen; homozygotes die as embryos. Rare antimorphic alleles display the above phenotype in exaggerated form with irregular widening of all longitudinal wing veins, enlarged deltas, regularly divergent wings, smaller rougher eyes, larger and often fused ocelli, and further increase in the numbers of extra bristles; in addition, tarsal joints 2 to 4, but not 5 are fused; homozygotes are embryonic lethals. Rare recessive alleles show low levels of survival as homozygotes or trans heterozygotes with more severe alleles; survivors usually display a less extreme version of the phenotype exhibited by heterozygotes for amorphic alleles; however, some combinations are wild type in appearance and others (e.g., the antimorphs) are lethal. The embryonic lethality of homozygotes displays the typical neurogenic phenotype with neural hyperplasia accompanied by epidermal aplasia; most or all cells of the neurogenic ecto-

derm recruited into the neurogenic pathway. Transplantation of homozygous *Dl* pole cells demonstrate *Dl* expression during oogenesis (Dietrich and Campos-Ortega, 1984, J. Neurogenet. 1: 315-32). *Dl* classed as non-autonomous in that single cells from the neurogenic ectoderm of *Dl⁻* embryos are capable of giving rise to both neural and epidermal derivatives when transplanted into the neurogenic region of wild-type embryos, suggesting that *Dl⁻* cells are capable of responding normally to information from neighboring cells (Technau and Campos-Ortega, 1987, Proc. Nat. Acad. Sci. USA 84: 4500-04). Transcription in cellular blastoderm seen in the ventrolateral neurogenic ectoderm, with a ventral-to-dorsal gradient of expression, corresponding to the gradient of neurogenic capabilities of the neurogenic ectoderm. During gastrulation a metameric pattern of expression appears, disappears, and reappears; as development proceeds complicated spatial and temporal specificities of expression ensue (Vässin *et al.*, 1987).

Interactions with other neurogenic mutations complex; *Dl* mutations suppress the *spl*-enhancing effect of *E(spl)* (Shepard, Boverman, and Muskavitch, 1988, Genetics 122: 429-38) and the expression of *Ax* (Sirén and Portin, 1989, Genet. Res. 54: 23-26); severe alleles fail to survive in heterozygotes with *E(spl)* loss-of-function alleles [Lehmann, Dietrich, Jiménez, and Campos-Ortega, 1981, Wilhelm Roux's Arch. Dev. Biol. 190: 226-29 (fig.)] especially when *E(spl)* is maternally inherited. Expression of *Dl/+* observed to be partially suppressed by duplications for *E(spl)⁺* (Vässin, Vielmetter, and Campos-Ortega, 1985, J. Neurogenet. 2: 291-308), yet, de la Concha, Dietrich, Weigel, and Campos-Ortega (1988, Genetics 118: 499-508) report that extra doses of *E(spl)⁺* enhance the neurogenic phenotype of *Dl⁻*. *Dl/+* and *Dl⁻* phenotypes are suppressed by heterozygous and homozygous deficiencies for *H*, respectively. For example, *H⁻* is able to suppress the phenotypic effects of *Dl^{9P}*, either in *Dl^{9P}/+* or in *Dl^{9P}/Dl^{9P}* genotypes; *Dl^{9P}/Dl^{9P}* is cell lethal in both the eye and the cuticle; *Dl^{9P} H²/Dl^{9P} H²* cells, on the other hand, develop nearly normally (Dietrich and Campos-Ortega, 1984). Expression of *Dl* enhanced by duplications for *N⁺* or *H⁺*, and three doses of *Dl⁺* enhance expression of *N⁻* and *neu⁻*, but reduce the severity of the *mam⁻* phenotype. de la Concha, *et al.* have incorporated many of these observations into a model of neurogenic-gene interaction. *Dl* alleles interact synergistically with certain Minutes, producing extreme phenotypes and drastically lowered viability (Schultz, 1929, Genetics 14: 366-419); *Dl^{Of}* enhances *spa^{Cat}* (Tsukamoto, 1956, DIS 30: 79).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>Dl¹</i>	spont	Bridges, 18k30		3, 4	
<i>*Dl²</i>	spont	Bridges, 20h13		3	
<i>Dl³</i>	spont	Bridges, 24110		1, 3, 4	
<i>*Dl⁴</i>	spont	Bridges, 26g28		4	
<i>Dl⁵</i>	X ray	R.L. King, 32d		1, 3, 4	
<i>Dl^{5F}</i>	EMS	Nüsslein-Volhard	5F102	1, 6, 7, 9	
<i>*Dl⁶</i>	X ray	Schultz, 33a5		3, 4	
<i>Dl^{6B}</i>	EMS	Nüsslein-Volhard	6B37	1, 6, 7, 9	temperature-sensitive ^β
<i>Dl^{6L}</i>	EMS			7	
<i>Dl⁷</i>	X ray	Schultz, 33a7		3, 4	
<i>*Dl^{7P}</i>		Panshin, 1935		3, 4	T(3,4)

allele	origin	discoverer	synonym	ref ^α	comments
<i>Dl⁸</i>				3	
<i>Dl⁹</i>				1, 3, 5	moderate allele
<i>Dl^{9D}</i>	EMS	Nüsslein-Volhard	9D27	1, 6, 7, 9	extreme allele
<i>Dl^{9K}</i>	EMS	Nüsslein-Volhard	9K23	1, 6, 7, 9	weak allele
<i>Dl^{9M}</i>	EMS	Nüsslein-Volhard	9M46	1, 6, 7	extreme allele
<i>Dl^{9P}</i>	EMS	Nüsslein-Volhard	9P39	1, 6, 7	extreme allele
<i>Dl^{9Q}</i>	EMS	Nüsslein-Volhard		1, 7	temperature-sensitive
<i>Dl¹⁰</i>	spont	Curry, 37k30		3	weak allele
<i>Dl^{10G}</i>	EMS			7	
<i>Dl¹¹</i>	spont	Bridges, 36f11		1, 3, 5	moderate allele
<i>Dl¹³</i>	spont	Bridges, 38d4		1, 3, 5	weak allele
<i>Dl¹⁴</i>	spont	Curry, 40d6		1, 3, 5	weak allele
<i>Dl³³</i>	X ray	Oliver, 33a3		3	
<i>*Dl^{55k}</i>	EMS	Clark		4	
<i>Dl^B</i>		Schultz, 1933		3, 4, 9	<i>In(3R)90B1-2;92A1-2</i>
<i>Dl^{B29}</i>	EMS			8	
<i>Dl^{B107}</i>	EMS	Vässin		9	antimorphic allele ^β
<i>Dl^{BE21}</i>	EMS	Muskavitch		1	moderate allele
<i>Dl^{BE23}</i>	EMS	Muskavitch		1	weak allele
<i>Dl^{BE24}</i>	EMS	Muskavitch		1	weak allele
<i>Dl^{BE26}</i>	EMS	Muskavitch		1	recessive hypomorph
<i>Dl^{BE30}</i>	EMS	Muskavitch		1, 2	weak allele
<i>Dl^{BE31}</i>	EMS	Alton		1, 2	recessive hypomorph
<i>Dl^{BE32}</i>	EMS	Alton		1, 2	weak allele
<i>Dl^{BE33}</i>	EMS	Alton		1, 2	recessive hypomorph
<i>Dl^{BE34}</i>	EMS	Alton		1, 2	recessive hypomorph
<i>Dl^{BE35}</i>	EMS	Alton		1, 2	recessive hypomorph
<i>Dl^{BE36}</i>	EMS	Alton		1, 2	recessive hypomorph
<i>Dl^{BE37}</i>	EMS	Alton		1, 2	recessive hypomorph
<i>Dl^{BE38}</i>	EMS	Alton		1, 2	weak allele
<i>Dl^{BE39}</i>	EMS	Alton		1, 2	weak allele
<i>Dl^{BX1}</i>	X ray	Muskavitch		1	weak allele
<i>Dl^{BX2}</i>	X ray	Muskavitch		1	strong allele deletes locus
<i>Dl^{BX3}</i>	X ray	Muskavitch		1	strong allele
<i>Dl^{BX4}</i>	X ray	Muskavitch		1	moderate allele
<i>Dl^{BX5}</i>	X ray	Muskavitch		1	moderate allele
<i>Dl^{BX6}</i>	X ray	Muskavitch		1	moderate allele
<i>Dl^{BX7}</i>	X ray	Muskavitch		1	weak allele
<i>Dl^{BX8}</i>	X ray	Muskavitch		1	recessive hypomorph
<i>Dl^{BX9}</i>	X ray	Muskavitch		1, 5a	moderate allele
<i>Dl^{BX10}</i>	X ray	Muskavitch		1	strong allele; 114.6 to 123.1 kb
<i>Dl^{BX11}</i>	X ray	Muskavitch		1, 5a	strong allele
<i>Dl^{BX13}</i>	X ray	Muskavitch		1	strong allele
<i>Dl^{BX14}</i>	X ray	Muskavitch		1	moderate allele; 96.0 to 103.7 kb
<i>Dl^{BX32}</i>	X ray	Terry		1, 5a	moderate allele
<i>Dl^{BX35}</i>	X ray	Terry		1	moderate allele; partially deletes locus
<i>Dl^{BX38}</i>	X ray	Terry		1	moderate allele; partially deletes locus
<i>Dl^{BX39}</i>	X ray	Terry		1	strong allele
<i>Dl^{BX40}</i>	X ray	Terry		1, 5a	strong allele; 114.6 to 123.1 kb
<i>Dl^{BX41}</i>	X ray	Terry		1, 5a	moderate allele; 108.9 to 114.6 kb
<i>Dl^{BX43}</i>	X ray	Terry		1, 5a	moderate allele
<i>Dl^{BX44}</i>	X ray	Terry		1	moderate allele
<i>Dl^{BX45}</i>	X ray	Terry		1, 5a	moderate allele; 96.0 to 103.7 kb
<i>Dl^{BX46}</i>	X ray	Terry		1, 5a	recessive hypomorph; 95.7 to 108.9 kb
<i>Dl^{CE1}</i>	EMS	Muskavitch		1	strong allele
<i>Dl^{CE2}</i>	EMS	Muskavitch		1	moderate allele
<i>Dl^{CE3}</i>	EMS	Muskavitch		1	moderate allele
<i>Dl^{CE4}</i>	EMS	Muskavitch		1	weak allele
<i>Dl^{CE5}</i>	EMS	Muskavitch		1	strong allele
<i>Dl^{CE6}</i>	EMS	Muskavitch		1	strong allele
<i>Dl^{CE7}</i>	EMS	Muskavitch		1	moderate allele
<i>Dl^{CE8}</i>	EMS	Muskavitch		1	weak allele

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	synonym	ref ^α	comments
<i>DI CE9</i>	EMS	Muskavitch		1	moderate allele
<i>DI CE10</i>	EMS	Muskavitch		1	moderate allele
<i>DI CE11</i>	EMS	Muskavitch		1	weak allele
<i>DI CE12</i>	EMS	Muskavitch		1	strong allele
<i>DI CE13</i>	EMS	Muskavitch		1	moderate allele
<i>DI CE14</i>	EMS	Muskavitch		1	moderate allele
<i>DI CE15</i>	EMS	Muskavitch		1	weak allele
<i>DI CE16</i>	EMS	Muskavitch		1	moderate allele
<i>DI CE21</i>	EMS	Muskavitch		1	moderate allele
<i>DI CE23</i>	EMS	Muskavitch		1, 5a	strong allele; 114.6 to 123.1 kb
<i>DI CE33</i>	EMS	Muskavitch		1, 5a	strong allele; 114.6 to 123.1 kb
<i>DI CE34</i>	EMS	Muskavitch		1, 2	weak allele
<i>DI CE37</i>	EMS	Muskavitch		1	moderate allele
<i>DI CE43</i>	EMS	Muskavitch		1, 5a	recessive hypomorph; 98.6 to 101.6 kb
<i>*DI Cf-3</i>	spont	Imaizumi	<i>Cf-3</i>	3, 4	
<i>DI CS20</i>	spont	Muskavitch		1	moderate allele; 95.5 to 96.8 kb
<i>DI E50-2</i>				6, 9	antimorphic allele
<i>DI FE1</i>	EMS	Jiménez		6, 9	extreme allele
<i>DI FE2</i>	EMS	Jiménez		6, 9	extreme allele
<i>DI FE3</i>	EMS	Jiménez		6, 9	extreme allele
<i>DI FE4</i>	EMS	Lehman		6, 9	weak allele
<i>DI FE7</i>	EMS	Jiménez		6, 9	extreme allele
<i>DI FE9</i>	EMS	Jiménez		6, 9	extreme allele
<i>DI FE17</i>	EMS	Jiménez		6, 9	extreme allele
<i>DI FE26</i>	EMS	Jiménez		6, 9	extreme allele
<i>DI FE27</i>	EMS	Vässin		9	severe allele
<i>DI FE28</i>	EMS	Vässin		9	severe allele
<i>DI FE29</i>	EMS	Vässin		9	severe allele
<i>DI FE30</i>	EMS	Vässin		9	antimorphic allele
<i>DI FE31</i>	EMS	Vässin		9	severe allele
<i>DI FE32</i>	EMS	Vässin		9	antimorphic allele
<i>DI FE33</i>	EMS	Vässin		9	intermediate allele
<i>DI FE35</i>	EMS	Vässin		9	intermediate allele
<i>DI FE36</i>	EMS	Vässin		9	weak-to-severe allele
<i>DI FE37</i>	EMS	Vässin		9	weak-to-severe allele
<i>DI FE38</i>	EMS	Vässin		9	severe allele
<i>DI FE39</i>	EMS	Vässin		9	intermediate-to-severe allele
<i>DI FE40</i>	EMS	Vässin		9	severe allele
<i>DI FE41</i>	EMS	Vässin		9	intermediate-to-severe allele
<i>DI FE43</i>	EMS	Vässin		9	weak-to-intermediate allele
<i>DI FE45</i>	EMS	Vässin		9	weak-to-intermediate allele
<i>DI FE46</i>	EMS	Vässin		9	intermediate allele
<i>DI FE47</i>	EMS	Vässin		9	intermediate allele
<i>DI FX1</i>	X ray	Jiménez		6, 9	extreme allele
<i>DI FX4</i>	X ray	Campos-Ortega		6, 9	extreme allele
<i>DI FX5</i>	X ray	Campos-Ortega		6, 9	extreme allele
<i>DI FX6</i>	EMS	Lehman		6, 9	extreme allele
<i>*DI FX7</i>	EMS	Lehman		6, 9	extreme allele
<i>DI FX8</i>	X ray	Lehman		6, 9	extreme allele
<i>DI H22</i>	spont	Tanaka 35d28		3	
<i>DI HD9</i>	HD	Artavanis-Tsakonas		1, 5a	recessive hypomorph 98.6 to 101.6 kb
<i>DI HD40</i>	HD	Artavanis-Tsakonas		1, 5a	recessive hypomorph 95.7 to 108.9 kb
<i>DI HD62</i>	HD	Artavanis-Tsakonas		1, 5a	moderate allele 95.5 to 96.8 kb
<i>DI HD82</i>	HD	Artavanis-Tsakonas		1, 5a	weak allele 98.6 to 101.6 kb
<i>DI I79</i>	EMS		<i>IL79N</i>	6, 7, 9	
<i>DI I113</i>	X ray	Schrons	<i>III3</i>	8, 9	<i>Tp(3;3)88F5-9;</i> <i>91A3-8;92A1-2</i>
<i>DI KE1</i>	EMS	Vässin		9	intermediate allele
<i>DI KE2</i>	EMS	Vässin		9	weak-to-intermediate allele
<i>DI KE3</i>	EMS	Vässin		9	severe-to-intermediate allele

allele	origin	discoverer	synonym	ref ^α	comments
<i>DI KE4</i>	EMS	Vässin		9	intermediate allele
<i>DI KE6</i>	EMS	Vässin		9	weak allele
<i>DI KE7</i>	EMS	Vässin		9	severe allele
<i>DI KE8</i>	EMS	Vässin		9	severe allele
<i>DI KE12</i>	EMS	Vässin		9	intermediate allele
<i>DI KE13</i>	EMS	Vässin		9	intermediate allele
<i>DI KE14</i>	EMS	Vässin		9	weak-to-intermediate allele
<i>DI KE15</i>	EMS	Vässin		9	severe allele
<i>DI KE48</i>	EMS	Vässin			
<i>DI KE49</i>	EMS	Vässin			
<i>DI KE50</i>	EMS	Vässin			
<i>DI KE50-2</i>	EMS	Jürgens			extreme allele
<i>DI KE51</i>	EMS	Vässin			
<i>DI KE52</i>	EMS	Vässin			
<i>DI KE53</i>	EMS	Vässin			
<i>DI KE54</i>	EMS	Vässin			
<i>DI KE55</i>	EMS	Vässin			
<i>DI KE56</i>	EMS	Vässin			
<i>DI KE57</i>	EMS	Vässin			
<i>DI KE58</i>	EMS	Vässin			
<i>DI KE59</i>	EMS	Vässin			
<i>DI KE60</i>	EMS	Vässin			
<i>DI KE61</i>	EMS	Vässin			
<i>DI KX1</i>	X ray	Vässin		9	severe allele
<i>DI KX2</i>	X ray	Vässin		8, 9	severe allele
<i>DI KX3</i>	X ray	Vässin		8, 9	severe allele
<i>DI KX6</i>	X ray	Vässin		8, 9	severe allele
<i>DI KX7</i>	X ray	Vässin		8, 9	severe allele
<i>DI KX8</i>	X ray	Vässin		9	severe allele
<i>DI KX13</i>	X ray	Vässin		9	severe allele
<i>DI KX14</i>	X ray	Vässin		8	
<i>DI KX15</i>	X ray	Vässin		8, 9	severe allele
<i>DI KX19</i>	X ray	Vässin		8, 9	severe allele
<i>DI KX22</i>	X ray	Vässin		9	severe allele
<i>*DI I</i>	spont	Bridges, 38c10	<i>l(3)DI</i>	3, 4	
<i>*DI M</i>	spont	L.V. Morgan, 36k		3	
<i>*DI N</i>	spont	Kikkawa		3	
<i>*DI Of</i>	spont	Tsukamoto, 1956		4	
<i>DI P-B55</i>	P			8	
<i>DI P-N</i>	P			8	
<i>DI r</i>	spont	L.V. Morgan	<i>*DI⁶</i>	3	
<i>DI roe</i>				6, 9	extreme allele
<i>DI roex</i>	X ray	Jürgens	<i>roXM3</i>	6, 9	extreme allele
<i>DI ru</i>	EMS		<i>ru-41</i>	6	extreme allele
<i>DI Sp</i>	spont	Knust		8, 9	
<i>DI vi1 β</i>	EMS	Vässin	<i>via1</i>	9	recessive hypomorph β
<i>DI vi2 β</i>	EMS	Vässin	<i>via2</i>	9	recessive hypomorph β
<i>DI vi3 β</i>	EMS	Vässin	<i>via3</i>	9	recessive hypomorph β
<i>DI X</i>		Stern, 1932		1, 3, 5	extreme allele
<i>DI X8</i>				9	extreme allele
<i>DI X18 γ</i>		Jürgens		6, 9	extreme allele
<i>DI XT</i>			<i>XTÜ1</i>	6	extreme allele

^α 1 = Alton, Fechtel, Kopczynski, Shepard, Kooh, and Muskavitch, 1989, *Dev. Genet.* 10: 261-72; 2 = Alton, Fechtel, Terry, Mickle, and Muskavitch, 1987, *Genetics* 118: 235-45; 3 = CP552; 4 = CP627; 5 = Craymer, 1980, *DIS* 55: 197; 5a = Kopczynski, Alton, Fechtel, Kooh, and Muskavitch, 1988, *Genes Dev.* 2: 1723-35; 6 = Lehmann, Jiménez, Dietrich, and Campos-Ortega, 1983, *Wilhelm Roux's Arch. Dev. Biol.* 192: 62-74; 7 = Tearle and Nüsslein-Volhard, 1987, *DIS* 66: 209-26; 8 = Vässin, Bremer, Knust, and Campos-Ortega, 1987, *EMBO J.* 6: 3431-40; 9 = Vässin and Campos-Ortega, 1987, *Genetics* 116: 433-45.

β Phenotypes described more fully below.

γ Associated with *T(2;3)*.

cytology: Placed in 92A2 based on its inclusion in both *Df(3R)Bxd¹¹⁰ = Df(3R)91D;92A2* and *Df(3R)DI-KX12 = Df(3R)92A2;92A4*.

molecular biology: Two chromosomal walks carried out in the region (Vässin, Bremer, Knust, and Campos-Ortega, 1987, *EMBO J.* 6: 3431-40; Kopczynski, Alton, Fechtel, Kooh, and Muskavitch, 1988, *Genes Dev.* 2: 1723-35);

coordinate 0 of the Kopczynski *et al.* walk is at approximately -20 kb on the Vässin *et al.* walk; in both coordinate systems, positive values extend to the left. Lesions associated with *Dl* mutations found between 75 and 100 kb on the Vässin *et al.* coordinates and between 95 and 127 kb on that of Kopczynski *et al.*; probes from this region identify two major transcripts of 4.5 or 4.6 and 5.4 kb; in addition, Kopczynski *et al.* report four minor transcripts. Probing of cDNA with genomic restriction fragments indicate that both major transcripts comprise four exons spread over 25 kb of chromosomal DNA. The smaller transcript is found in ovaries and in 0-2 hour embryos and is presumably maternal in origin; the larger one accumulates maximally in 2-6 hour embryos. Conceptual amino acid sequence indicates a protein of 832 amino acids which contains a signal sequence (residues 1-25), an extracellular domain, which included nine EGF-like repeats (residues 26-595), a transmembrane domain (residues 596-617), and an intracellular domain (residues 618-832); it also contains five consensus asparagine-linked glycosylation sites at residues 98, 137, 167, 421, and 649 (Kopczynski *et al.*).

Dl^{6B}

phenotype: Like *Dl*¹ except that severity of phenotype in homozygous embryos temperature sensitive. At 18° there is patchy neuralization of cephalic and ventral ectoderm; expression more severe at 25° and extreme at 29°. Temperature-sensitive period between pole-cell formation and mesodermal segmentation. Clone of ommatidia homozygous for *Dl*^{6B}, normal when reared under permissive conditions; in flies raised at 29°C, however, ommatidial pattern severely disturbed, producing scarring of the eye surface; ommatidia appear larger than normal and interommatidial bristles missing; homozygous mutant facets contain more than a normal complement of retinula cells-up to 13; cytodifferentiation apparently normal. Cuticular clones exhibit elaboration of extra bristles at bristle-forming sites [Dietrich and Campos-Ortega, 1984, *J. Neurogenet.* 1: 315-32 (fig.)].

Dl^{B107}

phenotype: The most severe antimorphic allele (Vässin and Campos-Ortega, 1987). All components of the phenotype of heterozygosity for a *Dl* deletion are present in a drastically increased manner in heterozygotes for *Dl*^{B107} (or for *Dl*^{FE30} or *Dl*^{FE32}). All wing veins are irregularly widened, veins 3 and 5 being broadened along their whole lengths (same for vein 2 in *Dl*^{FE30} and *Dl*^{FE32}) and are occasionally incised posteriorly; the deltas formed at the wing margins are larger and the wings are held spread with complete penetrance. The eyes are smaller and rougher. There is also a severe disturbance of the normal bristle pattern on the head, thorax, and abdomen owing to a further increase in the number of bristles. The ocelli are larger and often fused together, thus forming a half circle. Finally, tarsal segments 2 to 4 are fused, but segment 5 is never found to be affected.

Dl^{vi}: Delta viable

phenotype: Three alleles survive as homozygotes (Vässin, and Campos-Ortega, 1987). Slight delta-like thickenings at posterior tips of wing veins 2, 3, 4 and 5; roughening of eye. *Dl*^{vi} homozygotes also show shortening and frequent fusion of tarsal segments. A few homozygous

embryos fail to hatch, showing patchy neuralization in cephalic and ventral territories. *Dl*^{vi/+} normal. *Trans* heterozygotes with dominant alleles show extreme wing, eye, and tarsal abnormalities; *Dl*^{vi} lethal in combination with *Dl*^{F30}, *Dl*^{F32}, *Dl*^{E50-2}, and *Dl*^{B107} (Vässin, and Campos-Ortega, 1987).

dlg1: discs large

location: 1-34.82.

synonym: *l(1)10Bf*.

references: Stewart, Murphy, and Fristrom, 1972, *Dev. Biol.* 27: 71-83.

Gateff, 1978, *Biol. Rev.* 53: 123-68.

1978, *Science* 200: 1448-59.

Kiss, Szabad, and Major, 1978, *Mol. Gen. Genet.* 164: 77-83.

Perrimon, 1988, *Dev. Biol.* 127: 392-407.

Woods and Bryant, 1989, *Dev. Biol.* 134: 222-35.

phenotype: Late larval lethal; prolonged larval stages with bloated larvae attempting pupariation around day 15; some cuticular tanning, but no adult cuticular structures formed. During early larval development, the imaginal discs are smaller than those of normal larvae of the same age and are misshapen, but as the larvae continue to survive after the normal pupariation time, the discs continue to grow. They become large, amorphous, and solid, containing three times the normal numbers of cells at ten days of age; they also experience substantial cell death. By eleven days, wing and haltere discs may fuse; also first and second leg discs fuse with ventral ganglion of the CNS; great enlargement of optic lobes of brain also takes place. Discs, but not brains, transplanted into adults grow rapidly displaying invasive growth; they do not differentiate when transplanted into larvae for metamorphosis. Homozygous tissues do not survive, nor do gynandromorphs (one with male abdominal tissue vs. 66 expected). It is possible to produce homozygous germ-line clones (Perrimon). *dlg1* embryos generated from such clones display defects in morphogenesis and neurogenesis; most tissues are defective; partial rescue achieved by a paternal *dlg1*⁺ contribution, in the form of either a normal X or a v⁺Y.

alleles:

allele	origin	discoverer	synonym	ref	α	comments
<i>dlg1</i> ¹	X ray	Lefevre	<i>l(1)L11</i>	4, 6		<i>dlg</i> ¹ / <i>dlg</i> ² lethal at 18°
<i>dlg1</i> ²	X ray	Lefevre	<i>l(1)HF321</i>	5, 6		temperature sensitive; 5.5 kb insert near 5' end β
<i>dlg1</i> ³	X ray	Lefevre	<i>l(1)N17</i>	5		<i>ln(1)10B10-11;10D5-6</i>
<i>dlg1</i> ⁴	X ray	Lefevre	<i>l(1)RA16</i>	5, 6		<i>dlg</i> ⁴ / <i>dlg</i> ² lethal at 18°
<i>dlg1</i> ⁵	EMS	Geer	<i>l(1)v55</i>	2, 6		complements <i>dlg1</i> ² , <i>dlg1</i> ⁷ , and <i>dlg1</i> ¹³ ; late pupal lethal
<i>dlg1</i> ⁶	EMS	Geer	<i>l(1)v59</i>	2		
<i>dlg1</i> ⁷	EMS		<i>l(1)d.lg.-1</i> <i>X1-2</i>	6, 7		null allele
<i>dlg1</i> ⁸	EMS	Gateff	<i>l(1)bwn</i>	1		late pupal lethal
<i>dlg1</i> ⁹	EMS		<i>l(1),pr.2</i>	3		
<i>dlg1</i> ¹⁰	ENU	Voelker	<i>l(1)M15</i>	8		
<i>dlg1</i> ¹¹	ENU	Voelker	<i>l(1)M24</i>	8		
<i>dlg1</i> ¹²	ENU	Voelker	<i>l(1)M30</i>	8		
<i>dlg1</i> ¹³	ENU	Voelker	<i>l(1)M35</i>	6, 8		complements <i>dlg1</i> ¹⁸
<i>dlg1</i> ¹⁴	ENU	Voelker	<i>l(1)M52</i>	6, 8		<i>dlg1</i> ⁴ / <i>dlg</i> ² lethal at 18°
<i>dlg1</i> ¹⁵	X ray		<i>l(1)G3</i>	9		
<i>dlg1</i> ¹⁶			<i>l(1)G6</i>	9		
<i>dlg1</i> ¹⁷			<i>l(1)M1</i>	9		
<i>dlg1</i> ¹⁸	EMS	Perrimon	<i>l(1)1P20</i>	6		complements <i>dlg1</i> ² and <i>dlg1</i> ¹³ ; late pupal lethal

allele	origin	discoverer	synonym	ref. ^α	comments
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<i>dlg1</i>¹⁹	DEB	Perrimon	<i>l(1)565</i>	6	<i>dlg</i> ¹⁹ / <i>dlg1</i> ² lethal at 18°
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^α 1 = Gateff, 1977, DIS 52: 4-5; 2 = Geer, Lischwe, and Murphy, 1983, J. Exp. Zool. 225: 107-18; 3 = Kiss, Szabad, and Major, 1978, Mol. Gen. Genet. 164: 77-83; 4 = Lefevre, 1971, Genetics 67: 497-513; 5 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 6 = Perrimon, 1988, Dev. Biol. 127: 392-407; 7 = Stewart, Murphy, and Fristrom, 1972, Dev. Biol. 27: 71-83; 8 = Voelker, Wisely, Huang, and Gyurkovics, 1985, Mol. Gen. Genet. 201: 437-45; 9 = Zhimulev, Pokholkova, Bgatov, Umbertova, Solovjeva, Khudyakov, and Belyaeva, 1987, Biol. Zentralbl. 106: 699-720.

^β More complete description following molecular biology.

cytology: Placed in 10B8 based on its inclusion in *Df(1)DA622 = Df(1)10B8;10D2* but not *Df(1)m13 = Df(1)10B8;11A3-5*.

molecular biology: Region contained in a 60-kb walk initiated from the junction fragment of *Df(1)m13*. A 5.1 kb putative *dlg1* cDNA clone isolated (Woods and Bryant); it hybridizes to genomic fragments extending over 25 kb; antisense probe detects five different mRNA species in Northern blots; these species altered or absent in various *dlg1* mutants; the direction of transcription is from left to right. Late third instar larvae have transcripts to 6.0 and 5.5 kb; they are reduced in two-day pupae and a 1.9 kb transcript appears; it disappears in three-day pupae and the 6.0 and 5.5-kb species become more abundant and a 5.0-kb species appears. Adult females show five distinct mRNA species of 6.0, 5.5, 5.0, 4.0 (ovary specific), and 1.9 kb; only the two largest bands are seen in adult males. 0-4 hour embryos contain the 5.0 and at lower level the 5.5 kb transcripts; at 6-12 h these levels reduced and the 6.0 kb RNA appears; all species are barely detectable at end of embryogenesis; 5.5 and 6.0-kb species appear in the first larval instar becoming abundant in the third instar.

*dlg1*²

phenotype: Homozygotes fail to survive when raised at 25°; when raised at 18° however, 37% survival attained; hemizygous females show 65-70% survival at 18°, but are also lethal at 25°. Survival at 18° nearly complete in heterozygotes with *dlg1*⁵ and *dlg1*¹⁸, and survival at 25° is 4% and 95% respectively. Embryos produced by matings between surviving *dlg1*² flies raised at 18° show but 4% hatchability at 18°, and such crosses are completely sterile at 25°; however in crosses of homozygous *dlg1*+2 females to *dlg1*⁺ males, rescue of heterozygous daughters nearly complete at either 18° or 25° and is somewhat reduced at 29°. Abnormal embryos produced by *dlg1*² females show failure of dorsal closure, and head involution. The maternal effect of *dlg1* is germ-line specific, since embryos produced by homozygous clones in heterozygous mothers are indistinguishable from those produced by homozygous mothers (Perrimon, 1988, Dev. Biol. 127: 392-407).

dlg2

location: 1-24.9.

origin: Induced by ethyl methanesulfonate.

synonym: *l(1)d.lg.-2*.

references: Gateff, 1978, Biol. Rev. 53: 123-68. 1978, Science 200: 1448-59.

phenotype: Imaginal disc neoplasm with invasive mode of growth. Brains enlarged, but do not display neoplastic

growth.

Dll: see *Ba*

dlmd: dorsal-longitudinal-muscle-defective (J.C. Hall)

location: 3-18 to 19 (approximately 8 cM. to left of *h*).

origin: Induced by ethyl methanesulfonate.

references: Wang, Keng, Hsu, and Tan, 1989, J. Neurogenet. 6: 27-39.

phenotype: Flightless; wings held in normal position; walking, jumping, overall viability said to be normal; dorsal-longitudinal muscles (DLMs) degenerate, usually observed most readily at periphery of fibers; in degenerative areas, muscle Z lines disappear. DLM degeneration begins in late pupae, becoming more pronounced in newly eclosed adults (this progressive atrophy quantified for four of the alleles in homozygous condition: 3, 14, 23, 26); physiology of giant-fiber nerve pathway (which in part ends at DLMs) is essentially normal, though muscle-cell resting potential in mutant flies is lower than normal, and membrane excitability is accentuated.

alleles: *dlmd*²³, *dlmd*³⁰, *dlmd*¹⁷, *dlmd*²⁶, *dlmd*¹⁴, *dlmd*³, listed in order of decreasing penetrance for flightlessness, which ranges from 80-100%; also *dlmd*²³/+ almost thoroughly flightless; other alleles, over +, lead to 50-70% adults unable to fly.

other information: *dlmd*¹⁷ mutation was the only one mapped meiotically; others said to be allelic by lack of *inter se* complementations in flight tests; *dlmd*²³ seems not knowable as allelic, given its nearly complete dominance.

dlv: deltoid veins

location: 1-25.9.

origin: Induced by S-2-chloroethylcysteine.

discoverer: Fahmy, 1957.

references: 1959, DIS 33: 85.

phenotype: Wings small, abnormal, with margin occasionally incised, and frequently either divergent or slightly upheld. Extra venation, especially at junctions between longitudinal and costal veins, giving Delta-like formations. In extreme cases, wings grossly deformed and blistered. Excess melanization throughout body. Eyes dark, small, and slightly rough. Total body size reduced. Both sexes viable and fertile. RK1.

alleles: One allele induced by S-2-chloroethylcysteine.

Dly: Delayed recovery (W.B. McCrady)

location: 3-52.7 (2.7 units to right of *cu*).

origin: Natural population.

discoverer: McCrady, 1959.

references: McCrady and Sulerud, 1964, Genetics 50: 509-26.

Clark, McCrady, and Fielding, 1979, Genetics 92: 503-10.

phenotype: Recovery from CO₂ exposure for 15 min at 14°C of most *Dly/Dly* flies occurs between one and four hr, although many never recover, compared to less than 15 min for wild-type. *Dly*/+ flies recover between 15 min and one hr post-exposure. Percent of flies recovering decreases with increasing temperatures at time of exposure to CO₂ and with length of exposure. Exposure of *Dly/Dly* flies to 28° during development rescues them but not their *Dly*-bearing progeny from delayed recovery. *Dly* strains carry cytoplasmic factors which can transmit

CO₂ sensitivity by injection. Cytoplasmically transmissible factor reduced by successive backcrossing, but *Dly* strains remain CO₂ sensitive. *Dly* strains show partial resistance to suprainfection by sigma virus.

dm: diminutive

location: 1-4.0.

origin: Spontaneous by insertion of gypsy (Modolell, Bender, and Meselson, 1983, Proc. Nat. Acad. Sci. USA 80: 1678-82).

discoverer: Nichols-Skoog, 33j9.

references: 1935, DIS 3: 10.

phenotype: Bristles and body small and slender. Viability excellent. Females sterile. Oocyte cyst development abnormal; nurse-cell nuclei swell in stage 7, subsequently becoming pycnotic (King and Burnett, 1957, Growth 21: 263-80); cuboidal follicle cells covering developing oocyte in stage 8 fail to show normal growth and degenerate rather than becoming columnar (King and Vanoucek, 1960, Growth 24: 333-38). Chambers degenerate around stage 8; phenotype of homozygous *dm* less severe than that of *dml Df(1)dm* (King and Vanoucek). RK1.

alleles: **dm*²⁶⁴⁻⁵⁸ (CP627); *dp*^{ls}.

cytology: 3D5 (Lefevre, 1981, Genetics 99: 461-80).

dmd: see *pch*

Dmnd: Dimethylnitrosamine demethylase

location: 2-64.7.

references: Waters, Shigemi, Simms, and Nix, 1984, Biochem. Biophys. Res. Comm. 123: 907-13.

phenotype: Different strains differ with respect to presence or absence of this activity.

cytology: Placed in 48A-49D.

Dmras64B: see *Ras2*

dn: doughnut

location: 3-50.

origin: Spontaneous.

references: Wallbrunn, 1942, DIS 16: 54. Wright, 1946, DIS 20: 68.

phenotype: Eye of *se dn* has unpigmented spot (in middle or toward posterior) at emergence from puparium. Spot gradually darkens; after 2 days, eyes appear sepia. Difficult to detect with wild-type eye color, appears as slightly lighter red spot, which disappears after 2 days. Viability low; many die as pupae at 25°. Viability nearly normal at 17° but character not detectable. Both sexes highly infertile; testes about one-third normal length. Spermathecae very small. External genitalia of both sexes often abnormal. RK3.

DNase-1: Deoxyribonuclease-1

location: 3-61.8 (based on 83 *bx-sr* recombinants).

references: Grell, 1976, Genetics 83: s28-29.

Detwiler and MacIntyre, 1978, Biochem. Genet. 16: 1113-34.

phenotype: The structural gene for DNase 1, one of an estimated 7 or more DNase species (Boyd, 1964, Biophys. Biochem. Acta 171: 103-12). Active on native DNA at acid pH (optimum = 4.8) and in the presence of EDTA. Heterozygotes for electrophoretic alleles show two bands on gels indicating monomeric nature of enzyme. Activity of paternally inherited allele demonstr-

able in 0- to 1-hr embryos. Activity fluctuates during embryogenesis, is low during larval stages, rises precipitously just before pupation, and falls after eclosion (Detwiler and MacIntyre, 1978). DNase-1 activity isolated with lysosomal fraction of embryo homogenates (Detwiler and MacIntyre, 1980, Insect Biochem. 10: 255-63). Flies homozygous for hypomorphic allele are small with reduced viability; maturation of ovaries delayed for several days after eclosion; recombination normal (Grell, 1976). Homozygotes for the hypomorphic allele *DNase1^{lmn}* accumulate low-molecular-weight, low-thermal-stability DNA, especially in the ovaries; Feulgen-positive vesicles visible in maturing oocytes near chorionic appendages (Stone, Dower, Hauseman, Cseko and Sederoff, 1983, Can. J. Genet. Cytol. 25: 129-38).

alleles: Naturally occurring alleles designated *F*, *I*, and *S* by Grell are probably same as those subsequently designated *A*, *B*, and *C* by Detwiler and MacIntyre (1980); we adopt *F*, *I*, and *S*. Young adult flies homozygous for *DNase1^F* or *DNase1^I* exhibit two bands on gels; *DNase1^S* homozygotes show one (Grell, 1976). *DNase1^{lo}* (Grell, 1976) induced by ethyl methanesulfonate is hypomorphic. Also mentioned by Detwiler and MacIntyre (1978, 1980) are three hypomorphic alleles isolated by J. Stone, including *DNase^{lmh}*, and eight null alleles, including *DNase1ⁿ³²⁴*, isolated by Detwiler.

cytology: Placed between 90C2 and 90F based on the absence of any electrophoretic allele on *Df(3R)P14 = Df(3R)90C2-D1;91A2-3* or on the *Y^{P3D}* element of *T(Y;3)B116 = T(Y;3)YL;90E* (Detwiler and MacIntyre, 1978).

other information: Hyperploidy for 30F-35BC and 67C-70C as well as 88C-91B caused greater than 50% increase in acid deoxyribonuclease activity (MacIntyre, 1974, Isozyme Bull. 7: 23).

DNase2

location: 3-45.9 (between *st* and *in*).

references: Grell, 1976, Genetics 83: s28-29.

phenotype: The structural gene for a deoxyribonuclease that is active on heat-denatured DNA at pH 8.5. Causes the appearance of two bands on polyacrylamide gels. Null allele without detectable phenotypic effect.

alleles: A naturally occurring null allele designated *DNase2^{nl}* removes both bands.

other information: There is evidence for two other alkaline deoxyribonuclease genes. One produces a band on denatured substrate at pH 8.5 and is localized to *3L* on the basis of different mobilities in *D. melanogaster* and *D. simulans* (Grell, 1976). The other is located in 97F-100F based on increased alkaline DNase activity in hyperploids for that region (MacIntyre, 1974, Isozyme Bull. 7: 23); segmental aneuploidy failed to identify the other two regions as affecting DNase levels (MacIntyre, 1974).

dnc: dunce (R. Davis; J.C. Hall)

location: 1-3.9 [about five-eighth the recombinational distance from *w* to *ec* (Salz, and Kiger, 1984, Genetics 108: 377-92); however, as it is adjacent to *dm* (1-4.0), it is assigned a map position of 3.9].

origin: Induced by ethyl methanesulfonate (all alleles except *dnc^{CK}*, which was X ray induced).

discoverer: Byers.

- references:** Dudai, Jan, Byers, Quinn, and Benzer, 1976, Proc. Nat. Acad. Sci. USA 73: 1684-86.
 Byers, Davis, and Kiger, 1981, Nature 289: 79-81.
 Davis and Kiger, 1981, J. Cell Biol. 90: 101-07.
 Salz, Davis, and Kiger, 1982, Genetics 100: 587-96.
 Davis and Kauvar, 1983, Adv. Cyclic Nucleotide and Protein Phosphorylation Res. 16: 393-402.
 Salz and Kiger, 1984, Genetics 108: 377-92.

phenotype: Gene encodes a cAMP-specific phosphodiesterase. Mutants blocked or impaired in learning, with respect to several of the conditioning tests used on groups of flies or larvae or on individual adults, e.g., those involving odors and electric shocks or sugars (Aceves-Piña and Quinn, 1979, Science 206: 93-96; Tempel, Bonini, Dawson, and Quinn, 1983, Proc. Nat. Acad. Sci. USA 80: 1482-86; see also Aceves-Piña, Booker, Duerr, Livingstone, Quinn, Smith, Sziber, Tempel, and Tully, 1983, Cold Spring Harbor Symp. Quant. Biol. 48: 831-40), visual stimuli (Folkers, 1982, J. Insect Physiol. 28: 535-39), or various elements of courtship (with respect to tests on mutant males or females, summarized by Hall, 1984, Dev. Genet. 4: 355-78); also, *dnc*^{M14} males have reduced reproductive fitness after exposure to and courtship of immature females, i.e. when mutant males are put, post training, with mixed female/young male populations (Gailey, Siegel, and Hall, 1985, Genetics 111: 795-804). *dnc* females display an increased frequency of mating; it is suggested that this could account for their 50% normal longevity (Bellen and Kiger, 1987, Genetics 115: 153-60). *dnc* males are not conditioned to avoid virgin females by sequestration with such females in the presence of quinine; wild type males are (Ackerman and Siegel, 1986, J. Neurogenet. 3: 111-23). *dnc* mutants apparently learn normally, or nearly so, in certain experiments but have abnormally short memory (e.g., Dudai, 1979, J. Comp. Physiol. 130: 271-75; Mariath, 1985, J. Insect Physiol. 31: 779-87); more specifically, modifications of original shock-odor testing system reveal that *dnc* is defective in short term memory, with long-term memory similar to wild type (Tully and Quinn, 1985, J. Comp. Physiol. 157: 263-77); *dnc/+* females also memory deficient (Dudai, 1983, Proc. Nat. Acad. Sci. USA 80: 5445-48). Whereas *dnc* adults seem normal in several general behaviors (Dudai *et al.*, 1976), the mutant displays aberrant "centrophobic" behavior [*i.e.*, transient avoidance of the center of an arena displayed by normal adults (Gotz and Biesinger, 1985, J. Comp. Physiol. 156: 319-27, 329-37)]. Mosaic studies suggest that the focus of *dnc/+* function is the brain, even though some learning responses demonstrated by headless flies (Aceves-Piña, *et al.*). Sensory fatigue associated with an adult mechanosensory neuron, as measured by bristle stimulation, occurs more rapidly than normal in *dnc* (allele not specified) (Corfas and Dudai, 1990, J. Neurosci. 10: 491-99). There is an increased number of mushroom-body axonal fibers in young adults expressing *dnc*¹ or *dnc*^{M11}, which, unlike wild type, decreases over the next few days (Balling, Technau, and Heisenberg, 1987, J. Neurogenet. 4: 65-73). Mutations or deletions of the locus reduce or eliminate one form of cyclic AMP phosphodiesterase (EC 3.1.4.17) activity; caffeine, an inhibitor of this enzyme, decreases visual learning performance of normal adults and of *dnc*¹ as well, suggesting that the biochemical effects of the drug and the mutation

are not identical (Folkers and Spatz, 1984, J. Insect Physiol. 30: 957-65). The effects of *dnc* mutations on heat stability or K_m of this activity indicate that the locus codes for this enzyme (Kauvar, 1982, J. Neurosci. 2: 1347-58; Davis and Kauvar); *dnc* variants also lead to increased levels of cyclic AMP (summarized by Davis and Kauvar, 1983), more specifically, such that most of the excess is in free (vs. bound) nucleotide; both fractions exist in whole-fly homogenates (Friedrich, Solti, Gyurkovicz, 1984, J. Cell. Biochem. 26: 197-203). *dnc*^{M11} affects the level of phosphorylation of the regulatory subunit of the cyclic-AMP-dependent kinase (Dévay, Pintér, Yalcin, and Friedrich, 1986, Neurosci. 18: 193-203). Levels of regulatory subunit of cAMP-dependent protein kinase tend to be higher than normal in *dnc*¹ and *dnc*² (Muller and Spatz, 1989, J. Neurogenet. 6: 95-114). *dnc*^{M11} flies exhibit increased levels of expression of *copia* (Yun and Davis, 1989, Nucleic Acids Res. 17: 8313-26).

dnc alleles cause varying degrees of female sterility; oocytes of females homozygous for amorphic alleles rarely reach maturity, and for the most part are not oviposited; 90% of the few that are oviposited are fragile, lacking a chorion. That this phenotype is somatic in origin is demonstrated by the observation that homozygous germline clones produce morphologically normal eggs; some of these eggs undergo a few abortive nuclear divisions, but they never reach an identifiable stage of oogenesis. The maternal-effect lethality partially suppressible by *rut*, which reduces adenylate cyclase activity. The earliest defect seen in the embryos produced by *dnc rut* females occurs soon after fertilization and affects DNA replication and mitosis, prevents nuclear migration, and leads to large polyploid nuclei; a later defect prevents cleavage nuclei from migrating into, or dividing in, the posterior region of the egg, affecting the developmental behavior or fate of blastoderm cells. The few surviving offspring of double-mutant females show frequent developmental abnormalities of the second and third thoracic, and the first five abdominal segments; these include deficiencies, duplications, and transformation of structures; some 15% of the daughters of such females lack one or both ovaries (Livingstone, Sziber, and Quinn, 1984, Cell 37: 205-15; Bellen, Gregory, Olsson, and Kiger, 1987, Dev. Biol. 121: 432-44; Bellen and Kiger, 1988, Roux's Arch. Dev. Biol. 197: 258-68).

alleles:

allele	origin	discoverer	synonym	phenotype
<i>dnc</i> ¹	EMS	Byers	<i>dnc</i> ^{DB38}	hypomorph
<i>dnc</i> ²	EMS	Byers	<i>dnc</i> ^{DB}	hypomorph
<i>dnc</i> ^{CK α}	X ray			hypomorph
<i>dnc</i> ^{M11 β}	EMS	Mohler	<i>fs(1)M42</i>	amorph
<i>dnc</i> ^{M14}	EMS	Mohler	<i>fs(1)M42</i>	amorph
<i>dnc</i> ^{ML}	EMS			amorph

α Associated with *T(1;3)dnc*^{CK} = *T(1;3)3C-D4;63C*.
 β *dnc*^{M11} maps 0.04 map unit to the left of the other *dnc* alleles. Also, two molecularly defined "iso-alleles", resulting from transposon insertions at the locus that may have accompanied intragenic recombination events, have been characterized at high resolution (Pittler and Davis, 1987, Mol. Gen. Genet. 208: 325-28).

cytology: Placed in 3C11-D4, with exons 1 and 2 at 3C11 and exons 3-13 in 3D4 (Chen, Malone, Beckendorf, and Davis, 1987, Nature 329: 721-24). Claimed to be uncovered by *Df(1)N64i16* = *Df(1)3C2-3;3D4-5*, but not

Df(1)N64j15 = *Df(1)3C3-5;3D3-4* (Kiger and Golanty, 1977, *Genetics* 85: 609-22).

molecular biology: Both cDNA and genomic DNA cloned and sequenced (Davis and Davidson, 1984, *Mol. Cell. Biol.* 4: 358-67; Chen *et al.*). Genomic DNA at the locus identified in part by intragenic recombination events and following RFLPs as markers (see Pittler, Salz, and Davis, 1987, *Mol. Gen. Genet.* 208: 315-24 for further information on such recombination and putative gene conversion events). Gene comprises 13 exons, with exons 1 and 2 being separated from the remainder by an intron of 79 kb; this intron contains at least two and perhaps four genes, including *Pigl* (transcribed from the opposite strand) and *Sgs4* plus another developmentally regulated transcript (transcribed off the same strand as *dnc*); *sam* may also be encoded within the 79 kb intron (Chen, Malone, Beckendorf, and Davis, 1987, *Nature* 329: 721-24). A probe from the region of exon 6 identifies polyadenylated RNA's of 9.5, 7.4, 7.2, 7.0, 5.4, and 4.2 kb in RNA from adult flies; exons 1 and 2 share homology with the 9.5 kb mRNA and at least one of about 7.2 kb. The 5.4 kb species found in all developmental stages; the 9.6-, 7.4-, 7.2-, and 7.0-kb species found in late embryos and beyond; the 4.5-kb species found in early embryos and adults but not elsewhere (Davis and Davidson, 1986, *Mol. Cell. Biol.* 6: 1464-70). cDNA clones representing part of transcript contains an open reading frame that could define a 40,000 dalton polypeptide; the deduced amino acid sequence exhibits greater than 50% sequence identity with bovine Ca²⁺/calmodulin-dependent cyclic nucleotide phosphodiesterase; lesser and more restricted regions of homology seen with cAMP-dependent protein kinase and *Aplysia californica* egg-laying hormone precursor. Genomic clones reveal the presence of five introns in the sequence containing this open reading frame (Chen, Denome, and Davis, 1986, *Proc. Nat. Acad. Sci. USA* 83: 9313-17).

other information: *Df(1)N71h24/Df(1)dm75e19* females survive, even though they are completely deleted for some five bands, including 3D4; such females are defective in learning and are sterile. *dnc*^{M11}-induced sterility partly suppressed by *rut* (Livingstone *et al.*, 1984), and this phenotype of the double mutant used to select two new purported *rut* alleles, one of which (*rut*²) also suppresses learning defects associated with *dnc*^{M14} (Feany, 1990, *Proc. Nat. Acad. Sci. USA* 87: 2795-99).

do: see *po*²

Doa: Darkener of apricot

location: 3-99.

references: Rabinow and Birchler, 1989, *EMBO J.* 8: 879-89.

phenotype: Homozygous larval lethal, but in heterozygotes causes the *copia* insertion allele *w*^a and some of its revertants to produce more pigment than usual; not only in the eye, but in the ocelli, testis sheath, and Malpighian tubules as well; enhances *w*^{sp55} resulting in less than normal pigmentation; other tested alleles not affected. Flies carrying three copies of the wild-type allele of *Doa* lighten *w*^a and darken *w*^{sp55}. *Doa* also darkens *z w*^a in homozygous females. Heterozygotes for *Doa*¹ or *Doa*² and *Doa*⁶ or *Doa*⁷ survive rarely, and when they do, *w*^a is darkened to nearly wild-type color; *w*^{sp55} reacts in the

opposite manner, becoming nearly white; eyes of escapers have disarranged facets. Males of these heteroallelic combinations involving *Doa*⁶ are sterile despite having motile sperm; females and both sexes involving *Doa*⁷ are weakly fertile.

alleles:

allele	origin	synonym	comments
<i>Doa</i> ¹	HD	<i>Doa</i> ^{HD1}	
<i>Doa</i> ²	HD	<i>Doa</i> ^{HD2}	
<i>Doa</i> ³	EMS	<i>Doa</i> ^{EMS1}	
<i>Doa</i> ⁴	EMS	<i>Doa</i> ^{EMS2}	
<i>Doa</i> ⁵	EMS	<i>Doa</i> ^{EMS3}	
<i>Doa</i> ⁶	γ ray	<i>Doa</i> ^{CC}	<i>Tp(3;2)50B;84F;98C-D + Tp(3;3)80-81;98F1-4;100F</i>
<i>Doa</i> ⁷	X ray	<i>Doa</i> ¹⁰⁵	<i>ln(3LR)? + T(2;3)?;98F1-4</i>
<i>Doa</i> ⁸	X ray	<i>Doa</i> ^{V-53}	<i>Tp(1;3)14D;17F;98F1-4</i>
<i>Doa</i> ⁹	X ray	<i>Doa</i> ¹⁻⁵	

cytology: Placed in 98F1-4 based on the breakpoint common to three *Doa* rearrangements.

Don Giovanni: see *dg*

doomed: see *pch*

Dopa decarboxylase: see *Ddc*

Dopamine-N-acetyltransferase: see *Dat*

dor: deep orange

location: 1-0.3.

origin: X ray induced.

discoverer: E.D. King.

references: Merrell, 1947, *Am. Naturalist* 81: 399-400.

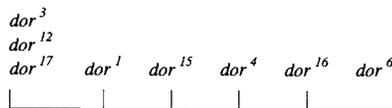
Counce, 1956, *Z. Indukt. Abstamm. Vererbungsleh.* 87: 443-61 (fig.).

Bischoff and Lucchesi, 1971, *Genetics* 69: 453-66.

phenotype: *dor* mutants affect a number of developmental processes; severity of effect increases with increasing developmental temperature. Eye color orange, shade depending on allele and temperature. *dor* reduces eye pigmentation in combination with either *cn* or *v* or with *bw*, indicating reduction in both drospterins and xanthommatin. Biochemical analyses show xanthommatin and five drospterins to be reduced to different degrees in *dor*; levels, but not relative proportions, change according to temperature of development (Counce, 1957, *Experientia* 13: 354; Puckett and Petty, 1980, *Biochem. Genet.* 18: 1221-28). Reciprocal transplantation experiments show that eye color is autonomous (Hadorn and Counce). *dor* females produce no progeny in crosses to *dor* males at 25°, although some allelic combinations able to produce progeny at 18°; and a few *dor*/+ daughters are produced in crosses to + males. Germ-line clones homozygous for the lethal allele *dor*²⁸ produce collapsed eggs (Perriman, Egstrom, and Mahowald, 1989, *Genetics* 121: 333-52). The lethal embryos produced by *dor* mothers reach gastrulation or beyond (Hildreth and Lucchesi, 1967, *Dev. Biol.* 15: 536-52; Counce, 1969, *DIS* 44: 101-82). Maternal effect shown to be germ line autonomous by both ovarian (Garen and Gehring, 1972, *Proc. Nat. Acad. Sci. USA* 69: 2982-85) and pole-cell transplantation (Marsh, van Deusen, Wieschaus, and Gehring, 1977, *Dev. Biol.* 56: 195-99). Maternal lethal effect rescuable by injections into preblastoderm embryos of cytoplasm from

unfertilized eggs of normal females (Garen and Gehring, 1972); *dor*⁺ substance present during early stages of vitellogenesis but not detected in yolk of cellular blastoderm embryos (Marsh *et al.*, 1977). Abnormalities of *dor* cells in culture eliminated by extracts of normal post- but not pregastrulation embryos (Kuroda, 1977, Dev. Growth Differ. 19: 57-66). *dor* males show variable extents of gonadal dysgenesis depending on culture conditions and genotypic background; abnormalities range from failure of testes to attach to genital ducts to failure of one attached testis to elongate (Lucchesi, Counce, and Hildreth, 1968, J. Exp. Zool. 168: 437-50). Viability and longevity of *dor* homozygotes and hemizygotes variably reduced depending on allele and temperature; *dor*¹ larvae develop melanotic pseudotumors (Stark, 1918, J. Exp. Zool. 27: 509-29; Oftedal, 1953, Z. Indukt. Abstamm. Vererbungsl. 85: 408-22) and midgut occlusion (Russell, 1940, J. Exp. Zool. 84: 363-79), dying in late third instar (Bischoff and Lucchesi, 1971). *dor/dor*¹ lethal at 29° (Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelova, and Zhimulev, 1980, Chromosoma 81: 281-306). *dor* in combination with *ry*, *ry*² (Lucchesi, 1968, Genetics 59: 37-44) and *car* (Nash, 1971, DIS 47: 73) causes lethality in pupal stage. Recovery of gynandromorphs with *dor car* male sectors less than in controls; bilateral gynandromorphs not observed, but distribution of male tissue resembles that of control (Grell, 1976). *dor* behaves as a semilethal in combination with *pd* and with *cn bw* (Lucchesi, 1968).

alleles: Mutants can be arranged in a linear sequence of increasing strengths; heterozygotes between different pairs of alleles show intermediate phenotypes; no evidence of interallelic complementation (Bischoff and Lucchesi, 1971). Recombinational mapping by Bischoff (1973, DIS 50: 172) established the order illustrated.



Genetic fine structure map of the **deep orange** locus.

allele	origin	discoverer	synonym	ref ^α	comments
<i>dor</i> ¹	X ray	E. D. King		12	viable, male sterile; eyes orange at 25°
<i>dor</i> ²			<i>dor</i> ^{17A}		
<i>dor</i> ³	spont	Hildreth, 61e	<i>dor</i> ^{61e}	7	viable, male sterile; eyes pale orange at 25°
<i>dor</i> ⁴	EMS	Lefevre	<i>dor</i> ^{66g}	9	viable, male fertile; eyes dark orange at 25°
<i>dor</i> ⁵	EMS		<i>dor</i> ^{69e}	5	viable, male fertile; eyes red-orange at 25°
<i>dor</i> ⁶	EMS		<i>dor</i> ^{69L1}	5	viable, male sterile; eyes orange at 25°
<i>*dor</i> ⁷	EMS		<i>dor</i> ^B		
<i>dor</i> ⁸	spont	Bridges	<i>l(1)7, dor</i> ¹	6, 12	lethal
<i>dor</i> ⁹	EMS	Alikhanian	<i>l(1)76, dor</i> ¹²	2	lethal
<i>dor</i> ¹⁰	EMS	H. Lewis	<i>dor</i> ¹³	11	lethal
<i>dor</i> ¹¹	EMS		<i>dor</i> ^{169B}	5	lethal
<i>dor</i> ¹²	EMS		<i>dor</i> ^{169F}	5	lethal
<i>dor</i> ¹³	EMS		<i>dor</i> ^{169J}	5	lethal

allele	origin	discoverer	synonym	ref ^α	comments
<i>dor</i> ¹⁴	EMS		<i>dor</i> ^{169K3}	5	lethal
<i>dor</i> ¹⁵	EMS		<i>dor</i> ^{169L1}	5	lethal
<i>dor</i> ¹⁶	EMS		<i>dor</i> ^{169L2}	5	lethal
<i>dor</i> ¹⁷	EMS		<i>dor</i> ^{169L3}	5	lethal
<i>dor</i> ¹⁸	EMS		<i>dor</i> ^{169L4}	5	lethal
<i>dor</i> ¹⁹	EMS		<i>l(1)181</i>	1, 3, 4	lethal
<i>dor</i> ²⁰	EMS		<i>l(1)128</i>	1, 3, 4	lethal
<i>dor</i> ²¹	EMS		<i>l(1)141</i>	1, 3, 4	lethal
<i>dor</i> ²²	EMS		<i>l(1)187</i>	1, 3, 4	lethal ^β
<i>dor</i> ²³	EMS		<i>l(1)257</i>	1, 3, 4	lethal
<i>dor</i> ²⁴	EMS	Pak	<i>l(1)470</i>	1, 3, 4	lethal
<i>dor</i> ²⁵	X ray	Lefevre	<i>l(1)A44</i>	10	lethal
<i>dor</i> ²⁶	X ray	Lefevre	<i>l(1)HC221</i>	10	lethal;
					<i>T(1;2)2B11;22B</i>
<i>dor</i> ²⁷	X ray	Lefevre	<i>l(1)RA17</i>	10	lethal
<i>dor</i> ²⁸	EMS	Lefevre	<i>l(1)VE915</i>	8	lethal

^α 1 = Aizenzon and Belyaeva, 1982, DIS 58: 3-7; 2 = Ardashnikov, 1941, Dokl. Akad. Nauk SSSR 30: 344-46; 3 = Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90; 4 = Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelove, and Zhimulev, 1980, Chromosoma, 81: 281-306; 5 = Bischoff and Lucchesi, 1971, Genetics 69: 453-66; 6 = Bridges, 1916, Genetics 1: 149; 7 = Hildreth, 1963, DIS 37: 48; 8 = Lefevre; 9 = Lefevre, 1970, DIS 45: 32; 10 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 11 = Lewis, 1954, J. Exp. Zool. 126: 235-75; 12 = Merrell, 1947, Am. Nat. 81: 399-400.

^β When the salivary glands of homozygotes for *dor*²², an allele that causes death in the third larval instar with no signs of ecdysone induction, were incubated with ecdysterone, the development of puffs was restored (Biyasheva, Belyaeva, and Zhimulev, 1985, Chromosoma 92: 351-56).

cytology: Tentatively placed in 2B11-12 by Lefevre [1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp.31-66; 1981, Genetics 99: 461-80]. However, 2B4-8 (Rayle and Hoar, 1969, DIS 44: 69) and 2B6-7 (Belyaeva *et al.*, 1980) have also been proposed.

dorsal: see *dl*

dorsal-longitudinal-muscle-defective: see *dlmd*

***double: double**

location: 1-0.

origin: Spontaneous.

discoverer: Bridges, 1918.

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 224.

phenotype: Postvertical bristles doubled. Wings very small. Viability somewhat low. RK3.

Double Bar: see *BB*

double glazed: see *dgl*

Double Infrabar: see *BⁱBⁱ*

double sex: see *dsx*

Doubler: see *Dp(1;1)B^SRMG*

doughnut: see *dn*

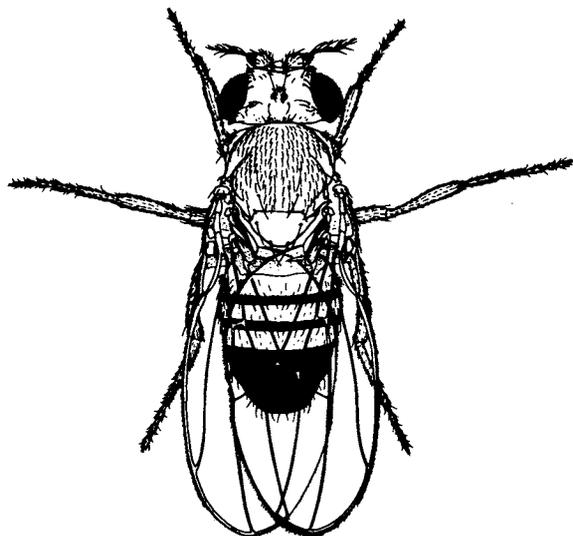
dow: downy

location: 1-8.0.

origin: Spontaneous.

discoverer: Bridges, 36c28.

phenotype: Bristles very short and slender, nearly as small as *ss*. Males entirely sterile; testis shape normal. Sper-



dow: downy

From Bridges and Brehme, 1944, Carnegie Inst. Washington Publ. No. 552: 64.

miogenesis arrested shortly after meiosis; nebenkern formation abnormal; spermatids degenerate after slight elongation [Kiefer, 1973, Genetic Mechanisms of Development (F.H. Ruddle, ed.). Academic Press, New York, London, and San Francisco, pp. 47-102]. Spermatocyte nuclei exhibit reduced amounts of Y-chromosome-dependent structures (Kiefer, 1973). Viability good. RK2.

Dox-A1: Diphenol oxidase A1 subunit

location: 2-{80.6}.

references: Deng and Rizki, 1988, Genome 30, Suppl. 1: 192.

phenotype: Enzyme detectable in embryos at ten hours and remains throughout development and adult life. A1 band more intense than those of subunits A2 and A3.

alleles: Monomorphic in *D. melanogaster*, but two bands present in *D. melanogaster* X *D. simulans* hybrids.

cytology: Placed in 55A based on its inclusion in *Df(2R)PC4 = Df(2R)55A;55F* and *Df(2R)Pcl11B = Df(2R)54F6-55A1;55C1-3* but not *Df(2R)Pcl-W5 = Df(2R)55A-B;55C*. as detected in *D. melanogaster* X *D. simulans* hybrids. *Bc* and *Phox* occupy the same cytological interval.

Dox-A2: Diphenol oxidase A2 subunit

location: 2-53.9.

synonym: *l(2)37Bf⁺*.

references: Pentz, Black, and Wright, 1986, Genetics 112: 823-41.

Pentz and Wright, 1986, Genetics 112: 843-59.

Wright, 1987, Adv. Genet. 24: 127-222.

phenotype: The structural gene for the A2 component of phenol oxidase, which utilized diphenol substrates. Developmentally regulated; levels low until just prior to pupariation; dramatic rise to maximum at puparium formation, followed by reduced, but still high, levels throughout pupal stage, again decreasing at eclosion (Geiger and Mitchell, 1966, J. Insect Physiol. 12: 755-

65). Heterozygotes for lethal alleles show reduced diphenol oxidase activity; homozygotes die as first instar larvae; dead larvae do not turn black. One homozygous *Dox-A2¹* escaper reaching pharate adult stage was liberated from the pupa case; it was chalk white, devoid of any cuticular pigment; cell lethal; no homozygous clones observed for any of the lethal alleles.

alleles: Most alleles are larval lethals; *Dox-A2^{mfs1}* homozygotes are also lethal, but hemizygotes display 60% normal adult viability and, although diphenol oxidase activity is normal, females and males are sterile.

allele	origin	discoverer	synonym	ref ^α	comments
<i>Dox-A2¹</i>	EMS + γ ray	Wright	<i>l(2)7402</i>	1	100-bp intragenic deletion
<i>Dox-A2²</i>	EMS + γ ray	Wright	<i>l(2)7409</i>	1	1.1-kb intragenic deletion
<i>Dox-A2³</i>	EMS + γ ray	Wright	<i>l(2)7416</i>	1	
<i>Dox-A2^{mfs1}</i>	EMS + γ ray	Wright	<i>mfs(2)7601</i>	2	Hemizygote viability = 60% of expected. Male and female sterile. Homozygous lethal.
<i>Dox-A2⁵</i>	EMS + γ ray	Cecil	<i>l(2)B8</i>		

^α 1 = Pentz, Black, and Wright, 1986, Genetics 112: 823-41; 2 = Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26.

cytology: Localized to a region between 14.3 and 16.8 kilobases long in 37B10-13 between the right-hand breaks of *Df(2L)hk-UC1* and *Df(2L)OD15*.

molecular biology: Genomic clone isolated in a chromosome walk through the *Ddc* region. The 1.7 kb transcription unit is processed to an 1.66 kb mRNA which encodes a basic protein of 56 kD made up of 494 amino acids. A single mRNA is present at varying levels throughout development.

Dox3

location: 2- [placed between *rdo* and *M(2)36F* by Rizki; however, placed to the right of *pr* by Pentz and Wright].

discoverer: Rizki.

references: Rizki and Rizki, 1985, Genetics 110: s98.

Rizki, Rizki and Bellotti, 1985, Mol. Gen. Genet. 201: 7-13.

phenotype: Apparently the structural gene of the A₃ component of phenol oxidase.

alleles: Electrophoretic variants exist.

cytology: Placed in 36D1-E4 based on its location in the region common to *Df(2L)H20 = Df(2L)36A89;36E3-4* and *Df(2L)M-HS5 = Df(2L)36D1-E1;36F1-37A1*. However, placed to the right of 37D1-2 by Pentz and Wright based on the failure of the following overlapping deficiencies to delete it: *Df(2L)H20 = Df(2L)36A7-10;36E4-F1*, *Df(2L)TW203 = Df(2L)36E4-F1;37B9-C1*, and *Df(2L)TW130 = Df(2L)37B9-C1;37D1-2* (Wright, 1987, Adv. Genet. 24: 127-222).

dp: dumpy

location: 2-13.0.

references: Carlson, 1959, Genetics 44: 347-73 (fig.).

Southin and Carlson, 1962, Genetics 47: 1017-26 (fig.).

Grace, 1980, Genetics 94: 647-62.

phenotype: *dp* alleles have variable effects on wing length and shape and on the thoracic cuticle. Presence of wing phenotype indicated by *o* = oblique in the allelic designation and of thoracic phenotype by *v* = vortex or *cm* = comma. The wing effect is an oblique truncation affecting the margins of the first and second posterior cells in

weak alleles and reducing wings to approximately half normal length in extreme genotypes, where the truncation is more nearly perpendicular to the long axis of wing. Margins remain intact; angle between veins L2 and L5 increased, and intercrosvein distance decreased. Phenotype resembles rudimentary. Thoracic phenotype comprises five types of hypodermal irregularities: first vortices, second vortices, commas, pre-episternal pits, and posterior invagination; all five have the form of pits, eruptions, or raised pits of the cuticle (Metcalf, 1970, Genetics 65: 627-54). First vortices are hypodermal pits or eruptions located posterolaterally on the scutum; they disrupt the acrostichal rows, resulting in surrounding whorls of microchaete. Second vortices are located anterolaterally on the scutum and resemble first vortices morphologically. Commas are comma-shaped depressions at the anterior margin of the scutum. The pre-episternal pit is in the pre-episternal plate immediately anterior to the sternopleural chaetae, which sometimes exhibit disturbed orientation (Metcalf, 1969, DIS 44: 91). The posterior invagination occurs between the laterotergite plate and the metanotum (Metcalf, 1969); different alleles exhibit different combinations of these traits. Musculature attached to disturbed regions of the cuticle often degenerates (Metcalf, 1970). Some alleles show reduced body size and small weak legs (*dp^h*, *dp^{obw}* females, *dp^{olv}/dp^{ov}*). Phenotypic expression enhanced by increased temperature during development; wing and thorax effects show dominance when heterozygotes exposed to increased temperatures at 12-16 and 8-10 hr of pupal life, respectively (Blanc and Child, 1940, Physiol. Zool. 13: 65-72). Normal larvae fed 6-azauracil produce adults with oblique phenocopies; 6-azauracil feeding suppresses *dp* (Rizki and Rizki, 1965, Science 150: 222-23), *su(r)* enhances the oblique phenotype (Stroman, 1974, Hereditas 78: 157-68). The four genotypes studied [*dp^{o2}*, *dp^{ovN}*, *dp^{v2}*, and *dp^v*; *e(dp^v)*] show increased orotate phosphoribosyl transferase activity during third larval instar and enhanced incorporation of labeled glucose into chitin (Blass and Hunt, 1980, Mol. Gen. Genet. 178: 437-42). Many alleles are lethal when homozygous; they are identified by *l* in the allelic designation. Lethal stages vary among alleles, e.g., *dp^{olv}* is embryonic lethal; *dp^{lv1}*, and *dp^{lv1}* die at the egg-larval boundary; *dp^{lm}* homozygotes die primarily at larval ecdysis between the first and second larval instar with some death at hatching and at ecdysis of second-instar larvae; *dp^{obm}* homozygotes die mostly at hatching but a few die during first and second larval instars (Metcalf, 1971, Genet. Res. 17: 173-83).

In *Me/+* heterozygotes, many *dp* alleles show a dominant oblique effect when heterozygous for *dp⁺* and *dp*; *dp^v* is an exception (Carlson, 1959). *dp^v* homozygotes normal; show thoracic phenotype only if third chromosomes homozygous for *e(dp^v)*.

alleles: One, two, or three of the phenotypic attributes of *dp* (*o*, *l*, or *v*) may be expressed in an allele, and alleles are classified according to which attributes they exhibit; the information is generally included in the superscript designation; the order *olv* is roughly in accord with map order. As a rule, the heteroallelic combination of any two alleles exhibits only phenotypic features common to the two alleles [e.g., *dp^o/dp^v* is normal, and *dp^{ov}/dp^{lv}* exhibits thoracic but not wing abnormalities (Carlson,

1959)]. Partial complementations between some pairs of *olv* and *l* or *lv* alleles are exceptions to this rule (Carlson, 1959; Grace, 1980). Identified alleles are tabulated below.

allele	origin ^α	synonym	ref ^β
o alleles			
<i>dp^o</i>	spont		1
<i>dp^{o2}</i>			5
<i>dp^{o3}</i>			
<i>*dp^{o33}</i>			
<i>dp^{oA2}</i>			
<i>dp^{oA4}</i>			
<i>*dp^{o50C}</i>		<i>dp^{50C}</i>	1
<i>dp^{o51e}</i>	UV		1
<i>dp^{obm}</i>	X ray		1
<i>*dp^{obw}</i>	spont	<i>dp^{bw}</i>	1
<i>dp^{odef}</i>	spont	<i>dp^{def}</i>	1
<i>dp^{oDG6}</i>			5
<i>dp^{oDG33}</i>			5
<i>dp^{oDG41}</i>			5
<i>*dp^{oDG53l}</i>			5
<i>dp^{oDG56}</i>			5
<i>dp^{oem2}</i>	EMS		6
<i>dp^{oem9}</i>	EMS		6
<i>dp^{oem10}</i>	EMS		6
<i>dp^{oem11}</i>	EMS		6
<i>dp^{oh13}</i>	X ray		10
<i>dp^{osz66}</i>	EMS		10
<i>*dp^{ou}</i>	UV		1
l alleles			
<i>dp^{l20}</i>			
<i>dp^{IDG82}</i>	NMU		4, 5
<i>dp^{IDG83}</i>	NMU		4, 5
<i>dp^{IDG85}</i>			
<i>dp^{IDG91}</i>			
<i>dp^{lb1}</i>	EMS		10
<i>dp^{lh40}</i>	X ray		10
<i>dp^{lh41}</i>	X ray		10
<i>dp^{lm}</i>	UV	<i>dp^{IHM52f}</i>	5
<i>dp^{lMi}</i>	spont		
<i>dp^{IM57f}</i>			8
<i>dp^{lsz31}</i>	EMS		10
<i>dp^{lsz46}</i>	EMS		10
v alleles			
<i>*dp^{cm}</i>	spont		1
<i>dv^v</i>	spont		1
<i>dp^{v2}</i>	spont		1
<i>dp^{v7}</i>	EMS		
<i>dp^{vem3}</i>	EMS		6
<i>dp^{vM}</i>	spont		1
<i>dp^{vsz4}</i>	EMS		10
<i>dp^{vW}</i>	spont		1
ol alleles			
<i>dp^{ol}</i>	spont	<i>dp^L</i>	1
<i>dp^{ol4}</i>			
<i>dp^{olAS}</i>		<i>dp^{LS}</i> <i>dp^{LSch}</i>	1
<i>dp^{olb20}</i>	X ray		10
<i>dp^{olb27}</i>	EMS		10
<i>dp^{olb42}</i>	EMS		10
<i>dp^{olCS19}</i>	ICR170		
<i>dp^{olCS41}</i>	ICR170		
<i>dp^{olCS50}</i>	ICR170		
<i>dp^{olCS80}</i>	ICR170		3
<i>dp^{olCS109}</i>			3
<i>dp^{olDG9}</i>	NMU		4
<i>dp^{olcm1}</i>	EMS		6

allele	origin α	synonym	ref β
<i>dp olem8</i>	EMS		6
<i>dp olem9</i>	EMS		6
<i>dp oih6</i>	X ray		10
<i>dp oih8γ</i>	X ray		10
<i>dp oih9γ</i>	X ray		10
<i>dp oih10γ</i>	X ray		10
<i>dp oih12γ</i>	X ray		10
<i>dp oih20γ</i>	X ray		10
<i>dp oih21γ</i>	X ray		10
<i>dp oIM</i>	UV	<i>dp LM</i> <i>dp T51b</i>	1
<i>dp oIsz24γ</i>	X ray		10
<i>dp oIXAS1</i>			
ov alleles			
<i>dp cm2</i>	spont		1
<i>dp ov1</i>	spont	<i>dp ovCB1</i>	1, 9
<i>dp ov7</i>			
<i>dp ov51f</i>		<i>dp 51f</i>	
<i>dp ov51h</i>	spont		1
<i>*dp ov52b</i>	spont		1, 9
<i>dp ovDG2</i>			5
<i>dp ovDG27</i>			
<i>dp ovDG30</i>			5
<i>dp ovDG35</i>			5
<i>dp ovDG37</i>			5
<i>dp ovDG55</i>			
<i>dp ovDG61</i>			
<i>dp ovdr</i>	spont	<i>dp dr</i>	1
<i>dp ovh</i>		<i>dp h</i>	9
<i>dp ovJ96δ</i>	γ ray		7
<i>dp ovN</i>	X ray	<i>dp Nov</i>	1
lv alleles			
<i>dp lv1</i>	spont	<i>dp lvCB1</i> <i>dp tx</i> <i>dp 2</i>	1, 5
<i>dp lv2</i>			
<i>dp lv44</i>	X ray		
<i>dp lv51e</i>	UV	<i>dp TH51e</i>	
<i>dp lvb21</i>	EMS		10
<i>dp lvCS24</i>	ICR170		
<i>dp lvCS35</i>	ICR170		3
<i>dp lvCS36</i>	ICR170		
<i>dp lvCS38</i>	ICR170		
<i>dp lvCS42</i>			5
<i>dp lvCS43</i>	ICR170		
<i>dp lvCS51</i>	ICR170		
<i>dp lvCS58</i>	ICR170		
<i>dp lvCS67</i>	ICR170		
<i>dp lvCS78</i>	ICR170		3
<i>dp lvCS81</i>	ICR170		
<i>dp lvCS105</i>	ICR170		
<i>dp lvDG1</i>			5
<i>dp lvDG3</i>			4
<i>dp lvDG5</i>	NMU		4
<i>dp lvDG7</i>	NMU		5
<i>dp lvDG10</i>			
<i>dp lvDG12</i>			
<i>dp lvDG13</i>			
<i>dp lvDG14</i>			
<i>dp lvDG16</i>			
<i>dp lvDG23</i>			
<i>dp lvDG24I</i>			
<i>dp lvDG38</i>			5
<i>dp lvDG39</i>			
<i>dp lvDG49</i>			5
<i>dp lvDG50</i>			5
<i>dp lvDG51</i>			5
<i>dp lvDG57</i>			5
<i>dp lvDG58</i>			5
<i>dp lvDG65</i>			
<i>dp lvDG66</i>	NMU		

allele	origin α	synonym	ref β
<i>dp lvDG67</i>			
<i>dp lvDG72</i>			
<i>dp lvDG78</i>			
<i>dp lvDG81</i>	NMU		
<i>dp lvem1</i>	EMS		6
<i>dp lvem2</i>	EMS		6
<i>dp lvem4</i>	EMS		6
<i>dp lvh37</i>	X ray		10
<i>dp lvi</i>	spont	<i>dp Th</i> <i>dp ThI</i> <i>dp tXl</i>	1
<i>dp lvx1</i>	X ray		8
<i>dp lvx2</i>	X ray		8
<i>dp lvx3</i>	X ray		8
<i>dp lvx4</i>	X ray		8
<i>dp lvx5</i>	X ray		8
<i>dp lvx18</i>	X ray		8
olv alleles			
<i>dp D</i>	X ray		1
<i>dp olv</i>		<i>dp T</i>	1
<i>*dp olv2</i>		<i>dp T2</i>	1
<i>*dp olv54d</i>	UV		1
<i>*dp olv55b</i>	spont	<i>dp T55b</i>	1
<i>*dp olv55c</i>	UV	<i>dp T55C</i>	1
<i>*dp olv57g</i>	UV	<i>dp T57g</i>	1
<i>dp olv101</i>			
<i>dp olv6m</i>	X ray		1
<i>dp olva22ϵ</i>	EMS		10
<i>dp olvb12</i>	EMS		10
<i>dp olvCG112</i>	ICR170		
<i>dp olvCS3</i>	ICR170		
<i>dp olvCS8</i>	ICR170		
<i>dp olvCS24</i>	ICR170		
<i>dp olvCS28</i>	ICR170		
<i>dp olvCS39</i>	ICR170		
<i>dp olvCS69</i>	ICR170		3, 5
<i>dp olvCS93</i>	ICR170		
<i>dp olvCS95</i>	ICR170		
<i>dp olvCS100</i>	ICR170		
<i>dp olvCS103</i>	ICR170		5
<i>dp olvCS108</i>	ICR170		5
<i>dp olvCS112</i>	ICR170		
<i>*dp olvD</i>	spont	<i>dp TD</i>	1
<i>dp olvDG10</i>			
<i>dp olvDG20</i>	NMU		4
<i>dp olvDG27</i>	NMU		4, 5
<i>dp olvDG32</i>			
<i>dp olvDG34</i>			
<i>dp olvDG44</i>			
<i>dp olvDG45</i>			
<i>dp olvDG46</i>			
<i>dp olvDG59</i>			
<i>dp olvDG61</i>	NMU		4, 5
<i>dp olvDG62</i>	NMU		4
<i>dp olvDG63</i>			
<i>dp olvDG72</i>			
<i>dp olvDG75</i>			
<i>dp olvem4</i>	EMS		6
<i>dp olvem5</i>	EMS		6
<i>dp olvem6</i>	EMS		6
<i>dp olvem7</i>	EMS		6
<i>dp olvem8</i>	EMS		6
<i>dp olvem9</i>	EMS		6
<i>dp olvem10</i>	EMS		6
<i>dp olvem11</i>	EMS		6
<i>dp olvh2</i>	X ray		10
<i>dp olvh3</i>	X ray		10
<i>dp olvh4</i>	X ray		10
<i>dp olvh5</i>	X ray		10
<i>dp olvh7γ</i>	X ray		10
<i>dp olvh11</i>	X ray		10
<i>dp olvh14</i>	X ray		10
<i>dp olvh17γ</i>	X ray		10

allele	origin ^α	synonym	ref ^β
<i>dp olv18</i>	X ray		10
<i>dp olv22</i> ^γ	X ray		10
<i>dp olv26</i> ^γ	X ray		10
<i>dp olv27</i>	X ray		10
<i>dp olv29</i>	X ray		10
* <i>dp olvH</i>	spont	<i>dp H</i>	1
<i>dp olvHC3</i>			
<i>dp olvHC39</i>	NMU		4, 5
<i>dp olvHC59</i>			5
<i>dp olvHC69</i>			
<i>dp olvHC234</i>			
* <i>dp olvM</i>	spont	<i>dp TO</i>	1
<i>dp olvM516</i>			5
* <i>dp olvP</i>	heat	<i>dp TP</i>	1
<i>dp olvR</i>	X ray	<i>dp Rf</i>	1
<i>dp olvS</i>	neutrons	<i>dp TSch</i>	1
* <i>dp olvSn</i>	spont	<i>dp Ts</i>	1
<i>dp olvsz28</i>	EMS		10
<i>dp olvW</i>	spont	<i>dp TW</i>	1
<i>dp olv1</i>	X ray		8
<i>dp olv2</i>	X ray		8
<i>dp olv3</i>	X ray		8
<i>dp olv4</i>	X ray		8
<i>dp olv5</i>	X ray		8

dominant alleles

dp D3
dp D4

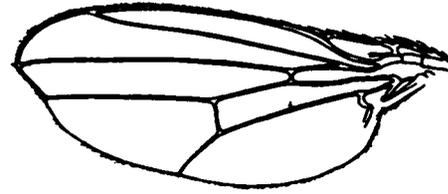
unclassified alleles

**dp 49* X ray 1
 **dp 53* spont 1
 **dp 61d* X ray 1
dp CA2 X ray 11
dp CA5 X ray 11
dp CA6 X ray 11
 **dp G* spont 1
dp w1 1
dp w2 1
dp w3 1
dp w8 1
dp w18 1

^α UV = ultraviolet; EMS = ethyl methanesulfonate; NMU = nitrosomethylurea; ICR-170 = 2-methoxy-6-dichloro-9-3(3-[ethyl-2-chloroethyl]aminopropylamino)acridine dihydrochloride.
^β 1 = CP627; 2 = Carlson and Southin, 1962, Genetics 47: 321-36; 3 = Grace, 1966, DIS 41: 83; 4 = Grace, 1970, Mut. Res. 10: 489-96; 5 = Grace, 1980, Genetics 94: 647-62; 6 = Jenkins, 1970, DIS 45: 38; 7 = Kotarski, Pickert, and MacIntyre, 1983, Genetics 105: 371-86; 8 = Meyer, 1970, DIS 45: 147; 9 = Sederoff, 1967, Nature 216: 1348-49; 10 = Szidonya and Reuter, 1988, Genet. Res. 51: 197-208; 11 = Velissariou and Ashburner, 1980, Chromosoma 77: 13-27.
^γ Also display dominant suppression of variegation presumably owing to effect on *Su(var)2-3*.
^δ Associated with *T(Y;2,4)J96*; variegated-type position effect.
^ε Shows a fully penetrant oblique phenotype; the few homozygous survivors show strong oblique-vortex phenotype with blistered wings; lethal in combination with other lethal *dp* alleles.

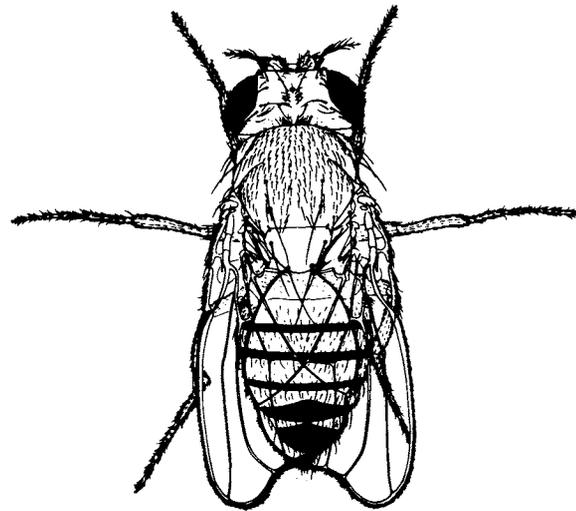
cytology: 25A1-2 (Velissariou and Ashburner, 1980, Chromosoma 77: 13-27) or 25A3-4 (Roberts and Broderick, 1982, Genetics 102: 74-89). *T(Y;2,4)J96* = *T(Y;2,4)25A2-3* shows Y-suppressed *dp* position effect (Kotarski, Pickert, and MacIntyre, 1983, Genetics 105: 371-86).

other information: Recombination between alleles belonging to the same or different phenotypic classes allows construction of genetic map with clusters of



dp^o: dumpy-oblique

From Bridges and Brehme, 1944, Carnegie Inst. Washington Publ. No. 552: 65.



dp^{ov}: dumpy-oblique vortex

Edith Wallace, unpublished.

discontinuously distributed alleles; *o* and *olv* alleles found in several clusters, other phenotypic classes confined to a single cluster.

Dp: see *Dr*

***dpl*: duplicated legs**

location: Not located.

origin: Derived from ethyl-methanesulfonate-treated flies.

synonym: *dl*.

references: Mglinetz, 1979, Ontogenez 10: 602-08.

Mglinetz and Ivanov, 1980, Ontogenez 11: 277-85.

phenotype: Some 6.7% of the flies of this strain show duplicated tarsi and in some instances tibiae; primarily involves mesothoracic legs. Distribution of bristles on leg and its duplicate agree with polar coordinate model (French, Bryant, and Bryant, 1976, Science 193: 969-81) of regeneration and duplication initiated in region of cell death. Phenotype resembles that of heat-pulsed *su(f)^{ls726}*. Produces duplicated antennal legs in combination with *ss^{ak}*, and both leg and its duplicate four jointed in combination with *ff* (Mglinetz, 1980, Ontogenez 11: 542-44). No genetic analysis reported.

dpp: decapentaplegic (W.M. Gelbart)

location: 2-4.0.

discoverer: E. Novitski (original *ho* mutation). R. Tung and W. Gelbart (original multi-disk *dpp* mutation).

synonym: *ho*, *blk*, *shv*, *DPP-C*, *Hin-d*, *Tg*.

references: Spencer, Hoffmann, and Gelbart, 1982, Cell 28: 451-61 (fig).

Segal and Gelbart, 1985, Genetics 109: 119-43 (fig).

Irish and Gelbart, 1987, Genes Dev. 1: 868-879 (fig).

St. Johnson, Hoffman, Blackman, Segal, Grimalia, Padgett, Irick, and Gelbart, 1990, Genes Dev. 4: 1114-27.

phenotype: *dpp* is a complex locus affecting numerous developmental events. Mutations fall into three major genetic and phenotypic groupings: called shortvein (***shv***), Haplo-insufficiency (***Hin***) and imaginal disk-specific (***disk***). Each group maps to a different region of the *dpp* gene. *Hin*-region mutations have two distinguishing features: they are defective in normal dorsal-ventral patterning of the embryo, and they generally fail to complement mutations of the *shv* and *disk* types. *shv*-region mutations all show recessive defects in longitudinal wing vein formation. *disk*-region mutations exhibit pattern deletions in the adult epidermal derivatives of the imaginal disks. The phenotypes of most *shv/disk* heterozygotes suggest partial or full complementation of the *shv* and *disk* lesions. Within each of the three major groupings, several phenotypic classes of alleles have been identified. Complementation between certain combinations of *dpp* alleles is transvection sensitive (Gelbart, 1982, Proc. Nat. Acad. Sci. USA 79: 2636-40).

The genetic properties of the several classes of *dpp* mutations are outlined below. For a given class, the prototypical recessive phenotypes are inferred from examinations of trans heterozygotes for two different alleles of that class. This procedure obviates possible complications due to the frequent association of *dpp* mutations with gross chromosomal rearrangements. Particular allelic combinations may deviate from the prototypical descriptions.

***Hin*-region**

***emb*:** Embryonic lethal mutation. Homozygous viable, but recessive lethal in combination with *hin-r* alleles, and, in the latter background, exhibits the same weakly ventralized phenotype as *hin-r* homozygotes. Completely complements all *shv*- and *disk*-region mutations. The sole *emb* allele is associated with a small deletion in *Hin*-region.

***Hin*:** Haplo-insufficient mutations. *Hin/+* heterozygotes exhibit dominant embryonic lethality with the same weakly ventralized phenotype as *hin-r* homozygotes. Dominant lethality is rescued by duplication of *dpp*^{*Hin+*}. Homozygotes are defective in gastrulation and die as embryos with completely ventralized cuticle. In general, *Hin* alleles do not complement any other *dpp* mutations. However, *Hin* alleles associated with small deletions or point mutations exhibit transvection effects in heterozygotes with small deletions or insertions in the *shv* and *disk*-regions. *Hin* mutations are considered the null alleles of the *dpp* gene. *Hin* alleles are associated with breakpoints, small deletions or point mutations in the *Hin*-region.

***Hin-Df*:** Haplo-insufficient mutations which behave identically to breakpoint *Hin* mutations, except that *Hin-Df*

lesions are gross deletions removing the entire *dpp* gene and adjacent vital loci.

***hin-r*:** Recessive mutations behaving as milder versions of the *Hin* lesions. In homozygotes, *hin-r* mutations exhibit embryonic lethality with weak ventralization effects (identical to *emb/hin-r* or *Hin/+* heterozygotes). All *hin-r* mutations engender temperature-sensitive mutant phenotypes when heterozygous with *shv*- and *disk*-region mutations. Phenotypes elicited in heterozygotes with small deletions, or insertions in the *shv* and *disk* regions are transvection sensitive. All *hin-r* mutations are cytologically normal and show no alterations in their restriction maps. Some have been associated with point mutations in the *Hin*-region.

***shv*-region**

***shv-lc*:** Recessive larval-lethal shortvein alleles which complement all *disk*-region mutations. Exhibit mutant phenotypes in heterozygotes with all *shv*, *Hin*, and *hin-r* mutations. Mutations generally associated with rearrangement breakpoints.

***shv-lnc*:** Recessive larval-lethal shortvein alleles which do not complement *disk*-region mutations. Also exhibit mutant phenotypes in heterozygotes with all *shv*, *Hin*, and *hin-r* mutations. Mutations generally associated with rearrangement breakpoints.

***shv-p*:** Recessive shortvein alleles surviving at least to pharate adult. Only two alleles are known; one (*s11*) is adult viable; exhibits strong venation defects, and variable head capsule defects, including loss of palps, and misarranged vibrissae. Allelic to all *shv*, *Hin*, and *hin-r* mutations. Complement all *disk*-region mutations. Both alleles are associated with rearrangement breakpoints.

***shv-w*:** Recessive viable and fertile shortvein alleles exhibiting only venation defects. Associated with small deletions of the *shv*-region. Venation phenotype allelic to all *shv*, *Hin*, and *hin-r* mutations. *shv-w/Hin*, and *shv-w/hin-r* mutant phenotypes are transvection sensitive. Only two alleles are known; both are associated with small deletions in the *shv*-region.

***Tg*:** A dominant gain-of-function allele in which the tegula on the wing appears duplicated. *Tg/+* wings are held out and down. Distinct in phenotype from heldout (*d-ho*) homozygotes. *Tg* completely complements all *dpp* mutations. The dominant effects of *Tg* can be reverted by superimposing *shv*, *Hin*, or *hin-r* mutations on the *Tg* chromosome. The one *Tg* allele is associated with a rearrangement breakpoint in or near the *shv*-region.

***disk*-region**

***disk-blk*:** Recessive viable and fertile allele in which the only mutant phenotype is loss of 80-90% of ommatidia in eye; hence this allele was designated *blink* by Sparrow (unpublished). Exhibits mutant eye phenotypes in heterozygotes with *disk-III*, *disk-V*, *Hin*, and *hin-r* mutations. Can exhibit transvection effects. The one *disk-blk* allele is associated with a small deletion within the *disk*-region.

***disk-ho*:** Recessive viable and fertile alleles in which the only mutant phenotypes are heldout wings and loss of the Sc25 on the dorsal base of the wing. Heldout phenotype displayed in heterozygotes with all *disk*-region mutations except *d-blk*, and with *Hin* and *hin-r* mutations. Can exhibit transvection effects. In addition to the one mutant allele listed here, which is associated with a small deletion within the *disk*-region, several cytologically normal *disk-ho* alleles have been associated with mobilization of

hobo mobile elements residing in the *disk*-region.

disk-II: Recessive viable alleles. Homozygotes exhibit reductions in wing blade, haltere and male genitalia. Elicit mutant phenotypes in heterozygotes with all *disk*-region mutations except *d-blk*, and with *Hin* and *hin-r* mutations. Mildest class of *disk*-region alleles associated with rearrangement breakpoints.

disk-III: Recessive viable alleles. Homozygotes exhibit multiple pattern abnormalities in epidermis of head, thorax, and terminalia. Structures absent or reduced include labial palps, arista, eye, wing blade, capitellum of haltere, tarsal claws, male terminalia, and female analia. Elicit mutant phenotypes in heterozygotes with all *disk*-region mutations, and with *Hin* and *hin-r* mutations. Intermediate class of *disk*-region alleles associated with rearrangement breakpoints.

disk-V: Recessive early pupal lethal alleles. Homozygous larvae have greatly reduced imaginal disks. Elicit mutant phenotypes in heterozygotes with all *disk*-region mutations and with *Hin* and *hin-r* mutations. Most severe class of *disk*-region alleles associated with rearrangement breakpoints.

t: Recessive larval-lethal alleles. Allelic to all *disk*, *Hin*, and *hin-r* mutations. The only two known alleles of this class behave identically to *disk-V* lesions, except for the earlier recessive lethal period. Tentatively classified as part of the *disk*-region. These two mutations are associated with breakpoints which map between the two tRNA^{tyr} genes residing at the *Hin*—*disk-V* boundary. Hence the *t* designation is used to describe these alleles.

alleles:

allele	class	origin	cytology
dpp d-blk	<i>d-blk</i>	<i>hobo</i>	+
dpp d-ho	<i>d-ho</i>	spont	+
*dpp d-ho40	<i>d-III</i>	X ray	<i>ln(2L)21D4-E1;22E2-3</i>
dpp d1	<i>d-III</i>	<i>hobo</i>	<i>ln(2L)22E2-3;22F2-3</i>
dpp d2	<i>d-III</i>	X ray	<i>ln(2L)22F1-2;28B</i>
*dpp d3	<i>d-III</i>	X ray	<i>T(2;3)22E3-F1;85B-D</i>
dpp d5	<i>d-III</i>	X ray	+
dpp d6	<i>d-III</i>	X ray	<i>ln(2L)22F1-3;24F2-6</i>
dpp d7	<i>d-III</i>	X ray	<i>T(2;3)22F1-2;80F</i>
dpp d8	<i>d-III</i>	X ray	<i>ln(2L)22A1-2;22F1-2</i>
dpp d9	<i>d-III</i>	X ray	<i>T(2;3;4)22F2-23A1;41A;57E-F;64F;80B;101</i>
dpp d10	<i>d-III</i>	X ray	<i>ln(2L)22E4-F1;23E2-4</i>
dpp d11	<i>d-III</i>	X ray	<i>Tp(2;2)22F1-2;29C;32C-D;39B</i>
dpp d12	<i>d-V</i>	X ray	<i>ln(2L)22E2-3;22F2-3</i>
dpp d13	<i>d-V</i>	X ray	<i>Tp(2;2)22F1-2;24C1-2;37F;40</i>
dpp d14	<i>d-V</i>	X ray	<i>Df(2L)22E4-F2;22F3-23A1</i>
dpp d15	<i>d-III</i>	X ray	<i>T(1;2)20;22F1-2</i>
dpp d16	<i>d-III</i>	X ray	<i>T(Y;2)Y;22F1-2</i>
dpp d17	<i>d-III</i>	X ray	<i>ln(2L)22F1-3;27E</i>
dpp d18	<i>d-III</i>	X ray	<i>ln(2L)22F1-2;36C4-6</i>
dpp d19	<i>d-III</i>	X ray	<i>Df(2L)22F2-3;22F3-4</i>
dpp d20	<i>d-III</i>	X ray	<i>T(2;3)22F1-3;64D</i>
dpp d21	<i>d-V</i>	X ray	<i>Tp(2;2)22A2-3;22F1-2;52F</i>
dpp d22	<i>d-III</i>	X ray	<i>T(2;3)22F1-3;67E</i>
dpp d23	<i>d-III</i>	X ray	<i>ln(2LR)22F1-2;41A</i>
dpp d25	<i>d-V</i>	X ray	<i>T(2;3)21F;22F1-3;72A-B;80F</i>
dpp d26	<i>d-V</i>	X ray	<i>ln(2LR)22F1-2;47A1-4</i>
dpp d28	<i>d-II</i>	EMS	<i>T(2;3)22F2-3;86E15-18</i>
dpp d29	<i>d-V</i>	X ray	<i>T(2;3)22F2-3;87D1-2</i>
dpp d30	<i>d-?</i> α	X ray	+
dpp d31	<i>d-III</i>	EMS	<i>ln(2LR)22F1-3;41C-D</i>
dpp d33	<i>d-V</i>	P	<i>Df(2L)22F1-2;23A1-2</i>
dpp d35	<i>d-V</i>	X ray	<i>ln(2LR)22F1-2;42A2-8</i>
dpp d36	<i>d-III</i>	X ray	<i>ln(2LR)22F1-3;35E</i>
dpp d41	<i>d-III</i>	γ ray	<i>ln(2LR)22F2-4;54F</i>
dpp d42	<i>d-V</i>	γ ray	<i>ln(2L)22F1-3;22F3-4</i>

allele	class	origin	cytology
dpp d44	<i>d-V</i>	γ ray	<i>T(2;3)22F1-2;80C</i>
dpp d49	<i>d-V</i>	γ ray	<i>T(1;2)XS(?);22F</i>
dpp d50	<i>d-II</i>	γ ray	<i>ln(2LR)22F2-3;27C</i>
dpp d52	<i>d-V</i>	γ ray	<i>T(2;3)22F1-2;86A-B</i>
dpp d54	<i>d-II</i>	P	+
dpp d55	<i>d-III</i>	P	+
dpp d60	<i>d-III</i>	γ ray	<i>ln(2L)21E;22F</i>
dpp d65	<i>d-V</i>	γ ray	<i>ln(2L)22F;34C;40</i>
dpp d66	<i>d-II</i>	γ ray	<i>Dp(3;2)78F;80F;21E;22F1-2</i>
dpp d67	<i>d-V</i>	γ ray	<i>ln(2LR)21F;22F</i>
dpp d68	<i>d-III</i>	γ ray	<i>ln(2LR)22F1-2;23D;51D</i>
dpp d70	<i>d-V</i>	γ ray	<i>Tp(2;2)21D;22F;21A</i>
dpp d71	<i>d-III</i>	γ ray	<i>T(2;3)22F1-2;101</i>
dpp d72	<i>d-V</i>	γ ray	<i>Tp(2;3)22F1-2;34B;81F</i>
dpp d73	<i>d-III</i>	γ ray	<i>T(2;4)22F1-2;57A;101F</i>
dpp d74	<i>d-III</i>	γ ray	<i>Tp(2;2)26A-B;29D-E;22F1-2</i>
dpp d75	<i>d-V</i>	γ ray	<i>ln(2LR)22F1-2;58D</i>
dpp d76	<i>d-III</i>	γ ray	<i>Tp(3;2) heterochromatin into 22F1-2</i>
dpp d77	<i>d-V</i>	γ ray	<i>T(2;3)22F1-2;80F</i>
dpp d78	<i>d-V</i>	γ ray	<i>T(2;3)22F1-2;95A1-2</i>
dpp d79	<i>d-V</i>	γ ray	<i>Df(2L)22F1-2;22F4-23A1</i>
dpp d80	<i>d-III</i>	γ ray	<i>Dp(3;2)85D;86E into 22F1-2</i>
dpp d81	<i>d-V</i>	γ ray	<i>T(2;3;4)22F1-2;30C;80F;101A-F</i>
dpp d82	<i>d-III</i>	γ ray	+
dpp e87	<i>emb</i>	EMS	+
dpp H32	<i>Hin-Df</i>	X ray	<i>Df(2L)22E3-F1;23A1-2</i>
dpp H34	<i>Hin-Df</i>	X ray	<i>Df(2L)22E2-3;23A2-4</i>
dpp H37	<i>Hin</i>	X ray	+
dpp H38	<i>Hin-Df</i>	γ ray	<i>Df(2L)22A1-2;22F3</i>
dpp H39	<i>Hin-Df</i>	γ ray	<i>Df(2L)21E1-2;23A2-4</i>
dpp H40	<i>Hin-Df</i>	γ ray	<i>Df(2L)22E1;23A1</i>
dpp H43	<i>Hin-Df</i>	γ ray	<i>Df(2L)22B1-2;23A3-β1</i>
dpp H45	<i>Hin-Df</i>	γ ray	<i>ln(2LR)22F1-3;52Fβ</i>
dpp H46	<i>Hin-Df</i>	γ ray	<i>Df(2L)22F1-2;22F2-3(?)</i>
dpp H47	<i>Hin</i>	γ ray	+
dpp H48	<i>Hin</i>	γ ray	+
dpp H51	<i>Hin-Df</i>	γ ray	<i>Df(2L)21F;23B1-2</i>
dpp H53	<i>Hin-Df</i>	γ ray	<i>Df(2L)22A1-2;23A3-7</i>
dpp H57	<i>Hin</i>	γ ray	+
dpp H59	<i>Hin-Df</i>	γ ray	<i>Df(2L)22A;23A</i>
dpp H61	<i>Hin</i>	γ ray	+
dpp H62	<i>Hin-Df</i>	γ ray	<i>Df(2L)22B;22F</i>
dpp H84	<i>Hin-Df</i>	γ ray	<i>Df(2L)22B;23A</i>
dpp H85	<i>Hin-Df</i>	γ ray	<i>Df(2L)22E2-F1;23A</i>
dpp H86	<i>Hin</i>	γ ray	<i>ln(2L)22F;26C;35D-E</i>
dpp H88	<i>Hin</i>	EMS	+
dpp hr4	<i>hin-r</i>	EMS	+
dpp hr27	<i>hin-r</i>	EMS	+
dpp hr56	<i>hin-r</i>	γ ray	+
dpp hr89	<i>hin-r</i>	EMS	+
dpp hr90	<i>hin-r</i>	EMS	+
dpp s1	<i>shv-w</i>	spont	+
dpp s2	<i>shv-lc</i>	X ray	<i>T(2;3)22F1-2;64E1-2</i>
dpp s3	<i>shv-lc</i>	X ray	<i>T(3;2)22F1-2;40C;82A;92A5-8</i>
dpp s4	<i>shv-lnc</i>	X ray	<i>ln(2L)21B1-C1;22F1-2</i>
dpp s5	<i>shv-lnc</i>	X ray	<i>ln(2L)21E1-2;22F1-2</i>
dpp s6	<i>shv-w</i>	X ray	+
dpp s7	<i>shv-lc</i>	X ray	<i>Tp(2)24E1-2;25A1-2 into 22F1-2</i>
dpp s8	<i>shv-lnc</i>	EMS	+
dpp s9	<i>shv-lc</i>	γ ray	<i>T(2;4)22F1-2;101</i>
dpp s10	<i>shv-lc</i>	γ ray	+
dpp s11	<i>shv-p</i>	γ ray	<i>ln(2L)22F1-2;31C-D</i>
dpp s12	<i>shv-lc</i>	γ ray	<i>ln(2L)22F1-2;24A</i>
dpp s13	<i>shv-lnc</i>	γ ray	<i>T(2;3)22F1-2;93B8-10</i>
dpp s14	<i>shv-lnc</i>	γ ray	<i>T(2;4)22F1-3;1VS</i>
dpp s15	<i>shv-lc</i>	γ ray	<i>ln(2LR)22F1-2;59B</i>
dpp s17	<i>shv-lc</i>	γ ray	<i>ln(2LR)22F1-2;41A</i>
dpp s18	<i>shv-lnc</i>	γ ray	<i>T(2;3)22F1-2;88E1-4</i>
dpp s19	<i>shv-lnc</i>	γ ray	<i>T(2;3)22F1-2;35B1-2;97B</i>
dpp s20	<i>shv-lc</i>	γ ray	<i>ln(2L)22B1-2;22F1-2</i>
dpp s21	<i>shv-lnc</i>	γ ray	<i>ln(2L)22A1-3;22F1-2</i>
dpp s22	<i>shv-p</i>	γ ray	<i>ln(2L)22F1-2;35C-D</i>
dpp s23	<i>shv-lc</i>	γ ray	+
dpp s24	<i>shv-lc</i>	γ ray	<i>T(2;3)22F1-2;59D;80F?;81F?;87C;88D;94D</i>
dpp s25	<i>shv-lnc</i>	γ ray	<i>T(2;3)22F1-2;88E1-4</i>
dpp s26	<i>shv-lc</i>	γ ray	<i>T(1;2)1D;22F1-2</i>

allele	class	origin	cytology
dpp <i>s27</i>	<i>shv-lc</i>	γ ray	+
dpp <i>s28</i>	<i>shv-lc</i>	γ ray	<i>Tp(2;?)22F1-2;24A1-2</i> → chromocenter
dpp <i>Tg</i>	<i>Tg</i>	X ray	<i>In(2L)21C;22F</i>
dpp <i>t24</i>	<i>t</i>	X ray	<i>In(2LR)22F1-2;58B</i>
dpp <i>t63</i>	<i>t</i>	γ ray	<i>In(2L)22F;39C-D</i>
dpp <i>58</i>	unknown	γ ray	+
dpp <i>64</i>	unknown	γ ray	Not done

α This mutation was induced upon *dpp*^{hr4}, complicating its mutant classification.

β Broken in 22F of the normal distal 2L copy of *dpp* and in 22C of the *Dp(2;2)dpp*^{d21} insertion into 52F.

cytology: Localized to 22F1-2 based on common breakpoints of numerous mutant rearrangements and *in situ* hybridization of cloned *dpp* DNA.

molecular biology: These phenotypic classes fall into discrete regions of the *dpp* map. The order of these groups on the chromosome from (distal to proximal) is:

[*shv-w, shv-p*] *shv-lc shv-lnc* [*Hin, hin-r*] *t disk-V disk-III disk-II disk-ho*

Several alternatively initiated and alternatively spliced transcripts encode a single 588-amino-acid long polypeptide which is a member of the transforming growth factor-β family of secreted factors (Padgett, St. Johnston, and Gelbart, 1987, Nature 325: 81-84). The open reading frame encoding the *dpp* polypeptide is contained in two exons located entirely within the *Hin*-region. Transcripts are localized dorsally at blastoderm in the dorsal ectodermal region during early germ band elongation and in two stripes, one dorsal and one lateral during late germ band extension and germ band shortening. Also expressed in two blocks in the embryonic visceral mesoderm (St. Johnston and Gelbart, 1987, EMBO J. 6: 2785-91). For proper ontogeny of normal *disk*-region functions *dpp* is required and expressed just anterior to the A/P compartment boundary in imaginal disks (Posakony *et al.*, 1990, submitted).

dpt: depilated

location: 3- (not mapped).

origin: Spontaneous.

references: DeAngelis, 1972, DIS 49: 39.

phenotype: Loss of thoracic and head bristles during first week of adult life. Hairs unaffected. Penetrance 100%; viability excellent.

***dpy: dumpoidy**

location: 3- (right arm near 90).

origin: Spontaneous.

discoverer: Villee, 40a.

phenotype: Wings obliquely truncated and reduced in length; marginal vein intact. No vortices or whorls of bristles on thorax. Suppressed by *Cy* and *Gla*, made dominant by *Me*. Overlaps wild type slightly. RK2.

dq: see *L*^{dq}

***dr: droopy**

location: 2-71.2 (to the left of Lobe).

origin: Ultraviolet induced.

discoverer: Meyer and Edmondson, 49c.

references: 1949, DIS 23: 60.

phenotype: Wings spread wide apart and droop down-

ward, like *c*, often crumple and drag in food. Alula broad and short. Viability at hatching fair; females tend to die before males. Penetrance 100%. Fertility good. RK2.

other information: Not allelic to *c*.

dr: see *drw*

Dr: Drop

location: 3-99.2.

phenotype: Heterozygotes have extremely reduced numbers of facets, 1-10 in the case of *Dr*¹ and *Dr*^A; ~30 facets coalesce to give shiny, dark red appearance in *Dr*^{Mio}; five to seven rhabdomeres per ommatidium section; some rhabdomeres fuse. Developing eye disc reduced in size; morphogenetic furrow uneven; few clusters of presumptive photoreceptor cells; arrangement disturbed; microvillar caps blurred and diffuse (Renfranz and Benzer, 1989, Dev. Biol. 136: 411-29).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
Dr ¹	X ray	Krivshenko, 54c25	<i>Dp</i>	2	homozygous lethal
*Dr ^A	X ray	Abrahamson, 60d28		1	homozygous viable
*Dr ^L	X ray	Lewis			<i>In(3R)89C;95D-96B1 + T(2;3)44;89F-90A β</i>
Dr ^{Mio}	NMS	Sobels, 57j22	<i>Mio</i>	4	homozygous lethal
Dr ^{We}	spont	Muller	<i>We</i>	3	homozygous lethal
α	<i>1</i> = Abrahamson and Siegel, 1960, DIS 34: 48; <i>2</i> = Krivshenko, 1954, DIS 28: 75; <i>3</i> = Muller, 1965, DIS 40: 36; <i>4</i> = Sobels, 1958, DIS 32: 84.				
β	Rearrangement probably unrelated to mutation.				

Dr: see *Rsp*

Dras: see *Ras*

drb: dark red brown

location: 3-47.7 (may be rearrangement; *st-p* crossing over 50% of normal).

origin: Spontaneous.

discoverer: Rosin, 48b.

references: 1951, DIS 25: 75.

phenotype: Eye color dark red-brown at 18° and dark red at 28°. *drb*⁺ darker than wild type at 18° but not at 28°. RK2(A).

drd: drop dead (J.C. Hall)

location: 1- (between *v* and *f*).

origin: Induced by ethyl methanesulfonate.

references: Hotta and Benzer, 1972, Nature (London) 240: 527-35.

Hotta and Benzer, 1973, Genetic Mechanisms of Development (F.H. Ruddle, ed.). Academic Press, New York, London, and San Francisco, pp. 129-67 (fig.).

Homyk, Sinclair, Wong, and Grigliatti, 1986, Genetics 113: 367-89.

phenotype: After normal development and normal behavior of young adults, flies begin to walk in uncoordinated manner and rapidly die (all dead by approximately ten days posteclosion); before death, there are gross holes in brain; mosaic experiment suggests primary defect is in brain. Second allele (*drd*²) causes similar effects with rapid death of population 9-11 days post eclosion plus reduced female fertility.

alleles: *drd*¹ (Hotta and Benzer), *drd*² (Homyk *et al.*).

other information: Not allelic to any of six adult-lethal (*adl*) genes mapping between *v* and *f*.

drg: dregs (*T. Schüpbach*)

location: 2-38.
origin: Induced by ethyl methanesulfonate.
references: Schüpbach and Wieschaus.
phenotype: Maternal-effect lethal mutant; embryos from homozygous mothers form a fragmented cuticle with variable holes and head defects.
alleles: *drg*¹ isolated as *HC*.

Drl: see *so*^D
droop wings: see *drp*

droopy: see *dr*
droopy wing: see *drw*

Drop: see *Dr*
drop dead: see *drd*

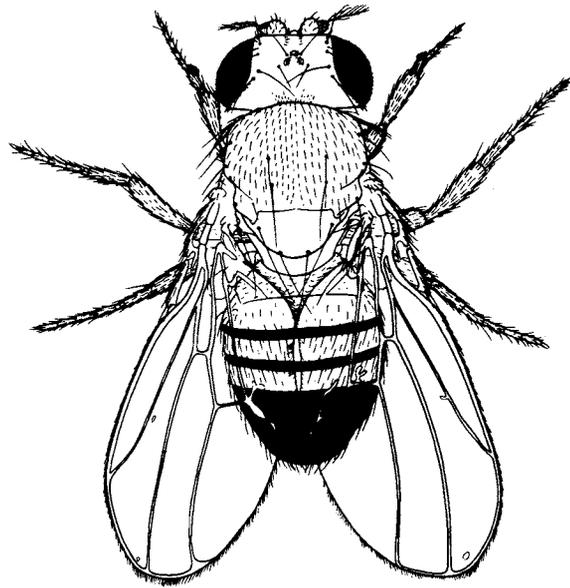
Droplet: see *so*^D
Drosophila-interferon-like-protein: see *Difl*
Drosulfakinin: see *Dsk*

***drp: droop wings**
location: 1- (rearrangement).
origin: Spontaneous.
discoverer: Ives, 48f.
references: 1949, DIS 23: 58.
cytology: Associated with *In(1)drp = In(1)12B;20B*.

***drw: droopy wing**
location: 1-52.3.
origin: Induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine.
discoverer: Fahmy, 1953.
synonym: Symbol originally *dr*, which was preoccupied.
references: 1958, DIS 32: 69.
phenotype: Small fly with drooping wings. Chitin of abdomen irregularly ridged and pigmented. Hairs deranged. Males infertile; viability 10% wild type. RK3.

ds: dachsous
location: 2-0.3.
phenotype: Wings shorter, blunter, and broader; crossveins uniformly very close together. Abdomen and legs chunky. Slight dominance of close crossveins. Stronger alleles may exhibit enhanced dominance, reduced viability, female sterility, delayed emergence, widely spaced scutellar bristles, and erect costal bristles. Strong interaction with *d*, *ffj*, and *cg*; double homozygotes often have excessive growth of thoracic parts and sometimes conversion of one organ into another {e.g., hypertrophy of notum, duplication of wings and antennae, and transformations of tarsus to arista, eye to antenna or palpus, or wing to notum [Waddington, 1943, J. Genet. 45: 29-43 (fig.); 1962, New Patterns in Genetics and Development, Columbia University Press, pp. 208-15 (fig.)]}. Tarsal shortening enhanced by homozygous *ss*^a and *ss*^{ab} (Villee, 1945, Genetics 30: 26-27). RK1.

allele	origin	discoverer	ref ^α	cytology
<i>ds</i> ¹	spont	Bridges, 17k12	3, 10, 8	
<i>*ds</i> ²	spont	Bridges, 25d2	3, 10	
<i>*ds</i> ³	spont	Bridges, 25k5	3, 10	
<i>ds</i> ^{7L}	EMS		9	
<i>ds</i> ¹⁴	X ray		7	<i>T(2;3)21D3;50B1-2;87B</i>



ds: dachsous
 Edith M. Wallace, unpublished.

allele	origin	discoverer	ref ^α	cytology
<i>ds</i> ^{33K}	spont	Bridges, 33k28	3	<i>In(2LR)21C8-D1;60D1-2</i>
<i>ds</i> ^{38K}	spont	Waddington, 38k	3, 5	
<i>*ds</i> ^{41B}	spont		3, 6	
<i>*ds</i> ^{51a}	UV		3	
<i>*ds</i> ^{53h}	UV		3	
<i>ds</i> ⁵⁵	X ray	Craymer	4	<i>In(2L)21D23;36C</i>
<i>*ds</i> ^d	spont	Sturtevant, 17b9	2, 3	
<i>*ds</i> ^{d2}	spont	Bridges	3	
<i>ds</i> ^{D4}	X ray	Sigmund, 1978	4	<i>T(2;3)21D;70-71</i>
<i>ds</i> ^{H3-1}	EMS		9	
<i>ds</i> ^{L1-1}	EMS		9	
<i>ds</i> ^{N1-1}	EMS		9	
<i>ds</i> ^{S2-1}	EMS		9	
<i>ds</i> ^{Wβ}	spont	Bridges, 29d24	1, 3	

^α 1 = Bridges, 1935, DIS 3: 10; 2 = Bridges and Morgan, 1919, Carnegie Pub. 278: 294; 3 = CP627; 4 = Craymer, 1980, DIS 55: 198; 5 = Curry, 1939, DIS 12: 45; 6 = Hinton and Bliven, 1942, DIS 16: 48; 7 = Korochkina and Golubovsky, 1978, DIS 53: 197-200; 8 = Mohr, 1929, Z. Indukt. Abstamm. Vererbungsl. 50: 113-200 (fig.); 9 = Roberts, Brock, Rudden, and Evans-Roberts, 1985, Genetics 109: 145-56; 10 = Stern and Bridges, 1926, Genetics 11: 511.
^β *ds* = dachsous-wide classified as RK1 as heterozygote.

cytology: Analysis by E.B. Lewis (1945, Genetics 30: 137-66) indicates that *ds* is located in 21D1-2 or possibly slightly to the left in the last band of 21C. Placed in 21C8-D1 by deficiency analysis [Golubovsky, Kulakov, and Korochkina, 1978, Genetika (Moscow) 14: 294-305] and in 21D1-2 by deficiency analysis by Roberts *et al.*

dsh: dishevelled
location: 1-34.05.
references: Perrimon and Mahowald, 1987, Dev. Biol. 119: 587-600 (fig.).
 Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-52.
phenotype: Most alleles lethal, but *dsh*¹, although poorly viable as homozygous females or hemizygous males,

appears to be fully viable when heterozygous to a deficiency for the region, suggesting that the reduced viability is unrelated to *dsh*. *dsh*¹ flies have deranged thoracic hairs, divergent and blistered wings, and ellipsoid eyes. Leg bristles, hairs, and bracts display high frequencies of abnormal polarity; extra joints or joint primordia found frequently in the first and second tarsal joints of the first and second pairs of legs; of 270 ectopic joints, 268 displayed inverted polarity [Held, Duarte, and Derakhshanian, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 145-57 (fig.)]. Males and females fertile, males weakly so. Homozygotes and hemizygotes for the lethal alleles die as second- to early-third-instar larvae when derived from heterozygous mothers; when derived from homozygous germ-line clones, on the other hand, embryos with segment-polarity defects result; only ventral cuticle is present, covered with a lawn of setae; lack dorsal cuticle, posterior spiracles and filzkörper material. At six to seven hours maxillary and labial segments appear to be missing and parasegmental boundaries do not form; cell death apparent in vicinity of tracheal pits, which subsequently fuse; segmental boundaries fail to form; organization of central nervous system seems normal. Loss of cells of posterior segment compartments leads to discontinued production of *en*⁺ product. Viability of *dsh*¹/*dsh*³, for example, when derived from homozygous *dsh*³ oogenic clones, is normal, indicating that *dsh*¹ is wild type for the early function.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>dsh</i> ¹	MMS	Fahmy, 1956		1	
<i>dsh</i> ²	CB. 3026	Fahmy		1	
<i>dsh</i> ³	EMS		<i>l(1)v26</i>	2	L/MER
<i>dsh</i> ⁴	EMS	Lefevre	<i>l(1)VA153</i>		
<i>dsh</i> ⁵	ENU	Voelker	<i>l(1)M12</i>	4	
<i>dsh</i> ⁶	ENU	Voelker	<i>l(1)M20</i>	4	
<i>dsh</i> ⁷	EMS		<i>dsh</i> ^{9PP3}	3	
<i>dsh</i> ⁸	EMS		<i>dsh</i> ^{9PP6}	3	

^α 1 = Fahmy, 1959, DIS 33: 85; 2 = Geer, Lischwe, and Murphy, 1983, J. Exp. Zool. 225: 107-18; 3 = Perrimon and Mahowald, 1987, Dev. Biol. 119: 587-600; 4 = Voelker, Wisely, Huang, and Gyurkovics, 1985, Mol. Gen. Genet. 201: 437-45.

cytology: Placed in 10B3-8 based on its inclusion in *Df(1)N71 = Df(1)10B5;10D4* but not in *Df(1)DA622 = Df(1)10B8;10D2*.

Dsk: Drosulfakinin

location: 3-{47}.

references: Nichols, Schnewly, and Dixon, 1988, J. Biol. Chem. 263: 12167-70.

phenotype: Encodes a Drosophila homologue of the vertebrate neuropeptide, cholecystokinin (CKK), which the authors dub drosulfakinin (DSK). *In situ* hybridization of antisense probes detects *Dsk* transcripts in regions of the adult brain, specifically the protocerebrum.

cytology: Placed in 81F by *in situ* hybridization.

molecular biology: Genomic clones isolated with synthetic probe based on the highly conserved C-terminal pentapeptide of vertebrate CKK and gastrin and cockroach leucosulfakinin peptides. cDNA clones corresponding to one class of recovered genomic clones detect a single poly(A)⁺ RNA of about 800 nucleotides on Northern blots. cDNA sequence indicates a protein product of 128 amino acids with a potential signal

sequence (residues 2-18) and two drosulfakinins, DSK-I (residues 101-110) and DSK-II (residues 113-127); these as well as a third sequence, DSK-0 (residues 63-70), are flanked by consensus pro-hormone processing sites and contain C-terminal glycyl residues, potential amidation sites. DSK-I and DSK-II sequences display identity with five of eight C-terminal residues of vertebrate cholecystokinin. DSK-0 represents a novel peptide.

dsl: discless

location: 1-35.

synonym: *l(1)dsl*.

references: Kiss, Bencze, Fekete, Fodor, Gausz, Maróy, Szabad, and Szidonya, 1976, Theor. Appl. Genet. 48: 217-26.

phenotype: Larval lethal; no disc tissue detected either by dissection of mature larvae or by serial sectioning of young larvae. *dsl* tissue does not survive in gynandromorphs nor in homozygous ovarian clones. No mitotic figures found in larval brain cells (Gatti and Baker, 1989, Genes Dev. 3: 438-53).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)dsl</i> ¹	X ray		<i>l(1)discless</i>	1, 5	L3/L
<i>l(1)dsl</i> ²	X ray	Lefevre	<i>l(1)L22</i>	2	
<i>l(1)dsl</i> ³	X ray	Lefevre	<i>l(1)C154</i>	3, 5	L/L
<i>l(1)dsl</i> ⁴	EMS	Lefevre	<i>l(1)DF936</i>	4	
<i>l(1)dsl</i> ⁵	EMS	Voelker	<i>l(1)B1</i>	6	

^α 1 = Kiss, Bencze, Fekete, Fodor, Gausz, Maróy, Szabad, and Szidonya, 1976, Theor. Appl. Genet. 48: 217-26; 2 = Lefevre, 1971, Genetics 67: 497-513; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 5 = Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14; 6 = Voelker, Wisely, Huang, and Gyurkovics, 1985, Mol. Gen. Genet. 201: 437-45.

cytology: Placed in 10C3-D4 based on its inclusion in *Df(1)N71 = Df(1)10B5;10D4*, but not *Df(1)v-N48 = Df(1)9F;10C3-5*.

dsp: dispersed

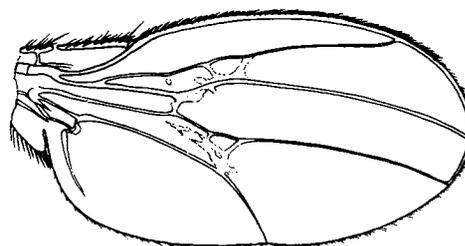
location: 1-55.6.

origin: Induced by ethyl methanesulfonate.

references: Eberl and Hilliker, 1988, Genetics 118: 109-20.

phenotype: Recessive lethal; embryonic cuticle normal; Malpighian tubules dispersed.

cytology: Placed in 15A4-F2 based on its being covered by *Dp(1;3)if⁺71b = Dp(1;3)15A4;16C2-3;80-81* and mapping genetically to the left of *f* at 15F1-2.

**dsr: disrupted**

From Bridges and Brehme, 1944, Carnegie Inst. Washington Publ. No. 552: 71.

dsr: disrupted**location:** 2-90.**origin:** Spontaneous.**discoverer:** Curry, 38a28.**phenotype:** Wings have plexus of extra and doubled veins at anterior and posterior crossveins and at L3 and L4. L3 and L4 spread wide apart. Wing slightly wider and warped. At 25°, overlaps wild type; at 19°, no overlap but viability reduced to 60% wild type. RK3.*Dsrc*: see *C-src*****dss: disturbed segmentation*****location:** 1-27.3.**origin:** Spontaneous.**discoverer:** Fahmy, 1954.**references:** 1959, DIS 33: 85.**phenotype:** Extremely abnormal abdomen with segmentation grossly deformed, very few hairs, and disturbed pigmentation. Occasionally some bristles shortened. Eyes reduced in size and sometimes abnormal in shape. Males fertile; viability about 10% wild type. Females sterile. RK3.***dsx: double sex (B. J. Taylor)*****location:** 3-48.1.**references:** Hildreth, 1965, *Genetics* 51: 659-78.Baker and Ridge, 1980, *Genetics* 94: 383-423.Baker and Belote, 1983, *Ann. Rev. Genet.* 17: 345-93.Nöthiger, Leuthold, Andersen, Gerschwiler, Gruter, Keller, Leist, Roost, and Schmid, 1987, *Genet. Res.* 50: 113-23.Baker and Wolfner, 1988, *Genes Dev.* 2: 477-89.Burtis and Baker, 1989, *Cell* 56: 997-1010.**phenotype:** The *dsx* gene regulates sexual differentiation of somatic tissues. Null alleles convert chromosomally male and female flies into sterile intersexes of similar phenotype. Dominant alleles (e.g., *dsx^D*, *dsx^M*, *dsx^T*) transform females into intersexes when heterozygous with a normal allele, and into phenotypic males when homozygous or heterozygous with a *dsx*-null allele or deficiency, but they have no effect in males. Most alleles at *dsx* affect both sexes; however, some alleles affect only one sex. The recessive allele *dsx^{II}* converts males into intersexes and is complemented by dominant *dsx* alleles and recessive alleles that affect only females (*dsx²²*) (Baker and Ridge; Nöthiger *et al.*, 1987). Double-mutant combinations of *dsx* null mutations with loss-of-function alleles at *tra*, *tra2*, and *ix* result in a doublesex phenotype (Mukherjee and Hildreth, 1971, *Genetica* 42: 338-52; Baker and Ridge; Nöthiger *et al.*, 1987). Double-mutant combinations of *dsx^{D/+}* with null alleles of *tra* and *tra2* convert females into phenotypic males or with *ix* into more male-like intersexes (Baker and Ridge; Nöthiger *et al.*, 1987). The dose of *dsx* alleles can alter the phenotype; triploid female flies *dsx^{D/+/+}* are sterile and with a weak external *dsx* phenotype (Gowen and Fung, 1957; Nöthiger *et al.*, 1987); diploid female flies that are *dsx^{D/+}*, but also carry a *dsx⁺* duplication *Tp(3;Y)P92* are sterile but female in appearance (Nöthiger *et al.*, 1987). Germline sexual differentiation is not dependent on *dsx⁺* function; only the chromosomal constitution determines the sex of transplanted *dsx^{M/+}*, *dsx^I*, *dsx^{D/+}*, and *dsx^{D/dsx^I}* germ cells (Nöthiger, Roost, and Schüpbach, 1980, DIS 55: 118; Schüpbach,1982, *Dev. Biol.* 89: 117-27). The *dsx⁺* gene does not appear to encode any vital functions (Baker and Ridge). The normal body size differences between male and female flies is maintained in *dsx*-null mutants (Hildreth) and in females heterozygous for *dsx^{D/+}*, *dsx^{D/dsx^I}* (Fung and Gowen, 1957; Baker and Ridge) and *dsx^{M/+}* (Nöthiger *et al.*, 1987). The sexcomb bristles on the prothoracic basitarsus in both sexes of *dsx*-null homozygotes (Hildreth; Mukherjee, and Hildreth; Baker and Ridge) and female *dsx^{D/dsx^I}* (Nöthiger *et al.*, 1987) are intermediate in number, morphology, and position compared with the sexcomb bristles in normal males and the transverse row bristles in normal females. The central sexcomb bristle is retained in *dsx*-null mutants (Hildreth). In *dsx*-null mutants, the pigmentation of the fifth tergite is intermediate between the completely pigmented male and the posteriorly pigmented female tergite, whereas the sixth tergite is darkly pigmented (Hildreth; Baker, and Ridge). Female flies that are *dsx^{D/+}* or *dsx^{M/+}* are similar to *dsx* homozygotes (Fung and Gowen, 1957; Duncan and Kaufman, 1975, *Genetics* 80: 733-52; Baker and Ridge; Nöthiger *et al.*, 1987). Male *dsx* flies have a seventh tergite and sternite with bristles (Hildreth; Baker, and Ridge). Female flies heterozygous for dominant alleles and either *dsx^I* or *dsx* deficiencies have the male number of tergites and sternites with the male pattern of pigmentation (Duncan and Kaufman, 1975; Baker and Ridge; Nöthiger *et al.*, 1980; Nöthiger *et al.*, 1987). By clonal analysis, the action of *dsx* has been shown to be cell autonomous in the differentiation of the sexcombs and pigmentation of the abdominal tergites; *dsx⁺* is required until the end of the larval period for the proper sexual differentiation of the sexcombs and into the pupal period, close to the time of the termination of divisions of the abdominal histoblasts, for proper sexual differentiation of the abdominal histoblasts and for proper sexual differentiation of the abdomen (Baker and Ridge). Both male and female genitalia are formed in *dsx* null mutant flies and in female flies heterozygous for dominant alleles (Fung and Gowen, 1957; Hildreth, 1965; Epper, 1981, *Dev. Biol.* 88: 104-14; Nöthiger *et al.*, 1987); a second penis differentiates with a reduced aedeagus and parameres within the female vaginal area (Hildreth). In *dsx^{D/+}* females, the development of the female genitalia and second penis are very similar to that of *dsx*-null flies, whereas in *dsx^{M/+}* females the female genitalia are more severely reduced (Gowen and Fung, 1975; Baker and Ridge; Nöthiger *et al.*, 1980; Epper, 1981; Nöthiger *et al.*, 1987). Male genitalia from *dsx* null flies and females heterozygous for *dsx* dominant alleles contain all elements except a basal apodeme but other external structures such as the penis and accessory elements are reduced and not as well formed (Fung and Gowen, 1957; Hildreth; Epper, 1981). The internal duct systems develop but can vary between dual female and male ducts and a single poorly differentiated duct (Hildreth); a similar range of phenotypes for the internal ducts is found in *dsx^{D/+}* (Fung and Gowen, 1957) and *dsx^{M/+}* (Nöthiger *et al.*, 1980). Based on fate mapping and analysis of the morphogenesis of the *dsx^{D/+}* genital disc, the female genitalia and second penis are generated from the female genital primordium, and the male genitalia from the male genital primordium and the production of both types of genitalia in *dsx* flies results from derepression of both

genital primordia (Epper, 1981; Epper, 1983, Wilhelm Roux's Arch. Dev. Biol. 192: 280-84). The intersexual analia differentiate as lateral plates, which are smaller than normal male lateral anal plates and do not have the ventral anal plate found in females (Hildreth; Baker, and Ridge; Epper, 1981). The bristle pattern is rather male-like but with one clearly identifiable long dorsal bristle that is female (Epper, 1981). The gonads are often rudimentary, but occasionally female *dsx*-null as well as *dsx^{D/+}* flies have well developed ovaries and eggs (Hildreth, Fung, and Gowen, 1957; Schüpbach, 1982; Bownes, Dempster, and Blair, 1983, J. Embryol. Exp. Morph. 75: 241-57) whereas in male *dsx*-null flies, the gonads are poorly developed and no sperm are formed (Hildreth; Schüpbach, 1982). Female and male *dsx* flies and female *dsx^{D/+}* flies make yolk protein but in amounts less than for normal females (Postelthwait, Bownes, and Jowett, 1980, Dev. Biol. 79: 379-87; Ota, Fukunaga, Kawabe, and Oishi, 1981, Genetics 99: 429-41). In *dsx^{D/+}* females less yolk protein synthesis occurs compared to normal females; little or no yolk protein mRNA is made in the rudimentary gonads, but measurements from adult flies show that from an initially low level yolk protein transcripts increase to nearly wild-type levels but without efficient conversion into protein (Bownes, Dempster, and Blair, 1983). The male-specific transcripts 316, 355a, and 355b, made by the male accessory gland, are also produced in male flies rendered intersexual by the mutation *dsx¹¹*, which does not affect female flies; in addition, females that are *dsx^{D/+}* or *dsx^{M/+}* express the male-specific transcripts, although at reduced levels (Chapman and Wolfner, 1988, Dev. Biol. 126: 195-202). Females homozygous for *dsx* do not exhibit male courtship behaviors (McRobert and Tompkins, 1985, Genetics 111: 89-96; B. Taylor, unpublished). These *dsx* females do not make 7, 11 dienes, and 7 monoenes compared to normal females (Jallon, 1984, Behav. Genet. 14: 441-78); they elicit less courtship than *dsx⁺* females from control males (McRobert and Tompkins, 1985). Female flies that are *dsx^{D/+}* or *dsx^{M/+}* also fail to express male courtship behaviors (Duncan and Kaufman, 1975; B. Taylor, unpublished). Males homozygous for *dsx* court but show reduced levels of courtship directed toward females and young males and greater-than-normal levels of courtship directed at mature males (McRobert and Tompkins, 1985). Mature *dsx* males make some pheromonal substances (Jallon, 1984) and elicit more courtship than *dsx⁺* males from control males (McRobert and Tompkins, 1985). Female flies homozygous for *dsx*-null alleles or mutant for *dsx* dominant alleles do not make the male-specific abdominal muscle unlike their male siblings (B. Taylor, unpublished).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>dsx¹</i>	spont	Hildreth		7	strong allele;
<i>dsx²</i>	EMS		<i>dsx^{10R}</i>	9	XX and XY equally affected hypomorph;
<i>dsx³</i>	EMS		<i>dsx³¹</i>	9	XX more affected than XY strong allele;
<i>dsx⁴</i>			<i>dsx^{34a}</i>		affects both sexes
<i>dsx⁵</i>	EMS		<i>dsx⁴⁰</i>	3	
<i>dsx⁶</i>	EMS		<i>dsx⁵³</i>	9	hypomorph;
<i>dsx⁷</i>		Garen	<i>dsx¹⁰⁶⁻¹</i>		XX more affected than XY

allele	origin	discoverer	synonym	ref ^α	comments
<i>dsx⁸</i>		Garen	<i>dsx¹²⁴⁻²</i>		
<i>dsx⁹</i>		Garen	<i>dsx¹²⁷</i>		
<i>dsx¹⁰</i>		Garen	<i>dsx¹²⁸⁻¹</i>		
<i>dsx¹¹</i>		Garen	<i>dsx^m</i> , <i>dsx¹³⁶</i>	1, 9	affects XY not XX
<i>dsx¹²</i>			<i>dsx⁵⁰¹</i>	9	affects XX much more than XY
<i>dsx¹³</i>			<i>dsx⁵⁸⁴</i>	9	weak hypomorph; affects XX much more than XY; (XY; <i>dsx¹²</i> / <i>dsx¹³</i> males fertile)
<i>dsx¹⁴</i>	spont	Puro	<i>ix-3</i> , <i>dsx⁶⁰¹</i>	11	
<i>dsx¹⁵</i>	EMS	Duncan	<i>dsx^{D100.41}</i>		
<i>dsx¹⁶</i>	EMS	Duncan	<i>dsx^{D100.42}</i>		
<i>dsx¹⁷</i>	EMS	Duncan	<i>dsx^{D100.71}</i>		
<i>dsx¹⁸</i>	EMS	Hazelrigg	<i>dsx^{EFH47}</i>		
<i>dsx¹⁹</i>	EMS	Hazelrigg	<i>dsx^{EFH48}</i>		
<i>dsx²⁰</i>	EMS	Hazelrigg	<i>dsx^{EFH55}</i>	1	strongly affects XX; only weakly XY
<i>dsx²¹</i>	EMS	Kaufman	<i>dsx^{EFK43}</i>		
<i>dsx²²</i>	EMS		<i>dsx^f</i>	9	affects XX; XY = normal fertile male
<i>dsx²³</i>	X ray		<i>dsx^{D+R3}</i>	2, 3, 5	<i>dsx^D</i> revertant;
<i>dsx²⁴</i>	X ray		<i>dsx^{D+R4}</i>	2, 5	<i>In(3R)84E1-2;85D22-E1 dsx^D</i> revertant;
<i>dsx²⁵</i>	X ray	Kaufman	<i>dsx^{D+R6}</i>		cytology normal <i>dsx^D</i> revertant;
<i>dsx²⁶</i>	X ray	Kaufman	<i>dsx^{D+R7}</i>	2, 3	cytology normal <i>dsx^D</i> revertant;
<i>dsx²⁷</i>	X ray	Kaufman	<i>dsx^{D+R9}</i>	2	<i>In(3R)84E1-2;92C3-6 dsx^D</i> revertant;
<i>dsx²⁸</i>	EMS		<i>dsx^{D+RH1}</i>	9	<i>In(3R)84E1-2;92C3-6 dsx^D</i> revertant; weakly fertile some dominant phenotype remains
<i>dsx²⁹</i>	EMS		<i>dsx^{D+RH2}</i>	9	<i>dsx^D</i> revertant
<i>dsx³⁰</i>	X ray		<i>dsx^{M+R1}</i>	2	<i>dsx^M</i> revertant; <i>T(2;3)28F;32F;84A1-2;84D11-E2;88B1-2 21-28F 84D11-84A2 32F-60; 61-84A1 28F-32F 88B1-84E2;88B2-100</i>
<i>dsx³¹</i>	X ray		<i>dsx^{M+R4}</i>	2, 3	<i>dsx^M</i> revertant;
<i>dsx³²</i>	X ray		<i>dsx^{M+R5}</i>	2, 3	<i>In(3R)84D9-10;84E1-2 dsx^M</i> revertant;
<i>dsx³³</i>	X ray		<i>dsx^{M+R12}</i>	2, 3	<i>T(2;3)59D1-2;84E1-2 dsx^M</i> revertant;
<i>dsx³⁴</i>	X ray		<i>dsx^{M+R13}</i>	2	<i>T(1;3)18D3-5;84E1-2; het; complex dsx^M</i> revertant;
<i>dsx³⁵</i>	X ray		<i>dsx^{M+R18}</i>	2, 3	<i>In(3LR)71F12;84E1-2 dsx^M</i> revertant;
<i>dsx³⁶</i>	X ray		<i>dsx^{M+R23}</i>	2, 3	<i>In(3R)84A;84E1-2 dsx^M</i> revertant;
<i>dsx³⁷</i>	X ray		<i>dsx^{M+R30}</i>	2, 3	<i>In(3LR)76A5-7;84E1-2 dsx^M</i> revertant;
<i>dsx³⁸</i>	X ray		<i>dsx^{M+R35}</i>	2, 3	cytology normal <i>dsx^M</i> revertant; complex rearrangement; possibly <i>In(3L)66C;80F + In(3R)81;98</i>
<i>dsx³⁹</i>	X ray		<i>dsx^{M+R38}</i>	3	<i>dsx^M</i> revertant
<i>dsx⁴⁰</i>	X ray		<i>dsx^{M+R39}</i>	2	<i>dsx^M</i> revertant; cytology normal
<i>dsx⁴¹</i>	X ray		<i>dsx^{M+R40}</i>	2	<i>dsx^M</i> revertant; cytology normal
<i>dsx⁴²</i>	X ray		<i>dsx^{M+R41}</i>	2	<i>dsx^M</i> revertant; 1-3 100A-67 100B-100; 20-3 67-64 101-102; 61-64 101 (tentative)
<i>dsx⁴³</i>	X ray		<i>dsx^{M+R45}</i>	1	<i>dsx^M</i> revertant; cytology normal
<i>dsx⁴⁴</i>	X ray		<i>dsx^{M+R46}</i>	2, 3	<i>dsx^M</i> revertant;
<i>dsx⁴⁵</i>	X ray		<i>dsx^{M+R47}</i>	2, 3	<i>T(1;3)20F;84D13-E8 dsx^M</i> revertant;
<i>dsx⁴⁶</i>	X ray		<i>dsx^{T+R}</i>	9	<i>T(2;3)58A1-2;84E1-2 dsx^T</i> revertant

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	synonym	ref ^α	comments
<i>dsx^D</i>	spont	Gowen 1940	<i>Hr, tra^D</i>	3, 4, 6, 12	dominant allele; affects XX only; <i>roo</i> -like insertion
<i>dsx^{Dw}</i>	EMS			9	dominant; XX → weak <i>dsx</i> phenotype
<i>dsx^M</i>	spont	Mischaikow 58i	<i>Mas, dsx^{Mas}</i>	3, 8, 10, 12	dominant allele; affects XX only <i>F</i> -like insertion
<i>dsx^S</i>	X ray	Brandt- Rosequist		3, 12	dominant allele; small deletion in female exon
<i>dsx^{Sw}</i>					
<i>dsx^T</i>	spont	Gehring		9	insertion

^α 1 = Baker and Belote, 1983, Ann. Rev. Genet. 17: 345-97; 2 = Baker, Hoff, Kaufman, and Hazelrigg, 1990, Genetics in press; 3 = Baker and Wolfner, 1988, Genes Dev. 2: 477-9; 4 = Denell and Jackson, 1972, DIS 48: 44-45; 5 = Duncan and Kaufman, 1975, Genetics 80: 733-52; 6 = Gowen and Fung, 1957, Heredity 11: 397-402; 7 = Hildreth, 1965, Genetics 51: 659-78; 8 = Mischaikow, 1959, DIS 33: 99; 9 = Nöhiger, Leuthold, Andersen, Gerschwiler, Gruter, Keller, Leist, Roost, and Schmid, 1987, Genet. Res. 50: 113-23; 10 = Nöhiger, Roost, and Schüpbach, 1980, DIS 55: 118; 11 = Puro, 1964, DIS 39: 64-65; 12 = Nagoshi and Baker, 1990, Genes Dev. 4: 89-97.

cytology: Placed in 84E1-2 based on breakpoints common to rearrangements associated with revertants of dominant alleles; located between the distal breakpoint of *Df(3R)Antp17 = Df(3R)84B1-2;84D11-12* (Baker et al., 1990) and the third chromosome breakpoint of *T(2;3)Es = T(2;3)48A3-4;84E3-4*.

molecular biology: Analysis of transcripts from the *dsx* region shows both male- and female-specific transcripts (Baker and Wolfner, 1988). Male-specific transcripts of approximately 3.8 and 2.8 kb are present in larvae and adults, with an additional 0.7 kb transcript in adult males (Baker and Wolfner, 1988; Burtis and Baker, 1990, Cell 56: 997-1010). The female-specific transcript around 3.5 kb is present in larvae and adults (Baker and Wolfner; Burtis and Baker). Based on mapping and sequencing of cDNA clones there are three common 5' exons, one female specific 3' exon, and two male specific 3' exons. The conceptual proteins encoded by the male and female *dsx* mRNA's contain 549 and 427 amino acids respectively (Burtis and Baker). Male and female *dsx* transcripts also differ with respect to the 3' polyadenylation site (Burtis and Baker). Within the female exon are six repeats of thirteen nucleotides found 3' to the acceptor site for the female exon (Burtis and Baker). Four dominant mutations (*dsx^D*, *dsx^M*, *dsx^S*, and *dsx^T*) have been mapped and sequenced and found to have lesions 3' to the acceptor site in the female exon but do not disrupt the intron or the acceptor site (Baker and Wolfner; Nagoshi and Baker, 1990, Genes Dev. 4: 89-97). Three of the dominant mutations (*dsx^D*, *dsx^M*, and *dsx^T*) are insertions of repetitive elements and one, *dsx^S*, is a small deletion. Using sex-specific probes, female flies heterozygous for a dominant allele have been shown to make both male- and female-specific *dsx* mRNA's, whereas females carrying a dominant allele in combination with a *dsx* deficiency make only the male-specific mRNA (Nagoshi and Baker). Chromosomally female flies that are homozygous for the hypomorphic *Sxl*²⁵⁹³ or *tra* and *tra2* null mutations produce only the male-specific *dsx* transcript whereas *ix* mutant females make female-specific *dsx* transcript of normal size and abundance (Nagoshi and Baker). Male flies that overexpress a *Hsp-tra* cDNA that

produces only the female-specific *tra* mRNA make the female-specific *dsx* mRNA and are transformed into phenotypic females (McKeown, Belote, and Boggs, 1988, Cell 53: 887-95).

***Dt: Detached**

location: 2-10.

discoverer: Bridges, 17e11.

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 224.

phenotype: Vein L2 fails to reach margin in 60% of flies. Homozygote not known. RK3.

other information: Bridges considered this a possible effect of *S* or requiring *S* as an enhancer as it was found in a *S* stock and apparently was never separated from *S*.

***dta: delta wing**

location: 1- (rearrangement).

origin: Induced by triethylenemelamine.

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 69.

phenotype: Wings widely outspread, frequently drooping in homozygous female. Viability good; female sterile. RK2A.

cytology: Associated with *In(1)dta = In(1)6B2-3;15E7-F2*.

dTKR: see *Tkr*

dtv: see *thv^d*

***du: dunkel**

location: 3-47.

origin: Spontaneous.

discoverer: Hadorn, 49e15.

references: Hadorn and Fritz, 1950, Arch. Julius Klaus-Stift. Vererbungsforsch. Sozialanthropol. Rassenhyg. 25: 504-8.

phenotype: Body color dark, sootylike. Wings blistered. Viability almost normal at 25°, greatly reduced at 18°. Males fertile; females sterile. Ovaries and eggs normal size and morphology. Insemination of females normal (motile sperm in spermathecae and receptaculum). Either eggs from *du* females not fertilized or zygotes die before blastoderm formation. *du* ovaries behave autonomously as implants in normal hosts, and wild-type ovaries are fertile in *du* hosts. RK2 at 25°.

other information: Not an allele of *by* or *cu*; possibly an allele of *db* (3-44.8)

dumpoidy: see *dpv*

dummy: see *dp*

dunce: see *dnc*

dunkel: see *du*

duplicated legs: see *dpl*

dusky: see *dy*

dusky body: see *dwb*

***dv: divergent**

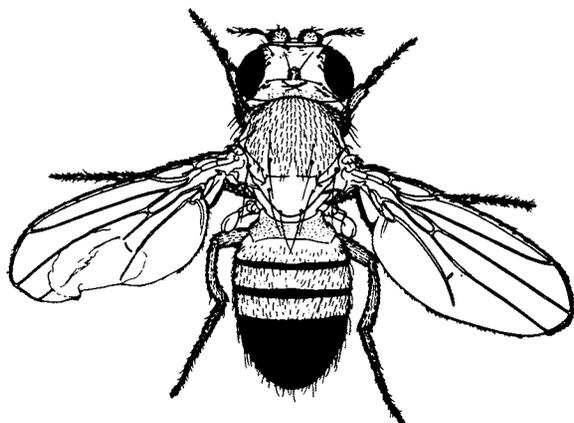
location: 3-20.0.

origin: Spontaneous.

discoverer: Bridges, 17f13.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 182 (fig.).

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet.

**dv: divergent**

From Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 182.

2: 58 (fig.).

Mohr, 1937, DIS 8: 12.

phenotype: Wings spread, smaller, and have slight venation disturbances. Both sexes rather infertile. *dv/Df(3L)Vn* progeny of homozygous *dv* mothers practically lethal, although the same genotype from other crosses survives (Mohr and Mossige, 1943, Avh. Nor. Vidensk.-Akad. Oslo, Mat.-Naturvidensk. Kl. No. 7: 1-51). RK2.

alleles: *dv*² described as *aea* by Valade del Rio (1983, DIS 59: 161); same phenotype and map position as *dv*.

cytology: Salivary chromosome locus placed between 64C12 and 65E1 on basis of its inclusion in *Df(3L)Vn = Df(3L)64C12-D1;65D2-E1* (Mohr, 1938, Avh. Nor. Vidensk.-Akad. Oslo, Mat.-Naturvidensk. Kl. No. 4: 1-7).

dv2

location: 2-71.

origin: Spontaneous.

references: Aguado, Galán-Estella, and González-Gulián, 1988, DIS 67: 109.

phenotype: Wings adopt a divergent orientation with respect to the longitudinal-axis of the body when at rest.

dvr: divers

location: 1-28.1 (located using *dvr*²).

phenotype: Has shorter, darker wings; postscutellars bowed in; body size small; sterility high; semilethal. In combination with yellow-bodied *y* alleles, gives strongly curled wings with slight outward twist; with *f*, gives crumpled wings, with *sc*, almost lethal. RK3.

allele	origin	discoverer	ref ^α
<i>dvr</i> ¹	iodine ^β	Sacharov, 1932	1, 3, 5
<i>dvr</i> ²	spont	Curry, 37k17	1
<i>dvr</i> ⁸⁴	heat ^γ	Green, 84k24	
<i>dvr</i> ^s	spont	Muller	2

^α 1 = CP627; 2 = Muller, 1946, DIS 20: 67; 3 = Sacharov, 1936, Biol. Zh. 5: 537-40 (fig.); 4 = Sacharov, 1937, DIS 8: 81.

^β Recovered among progeny of iodine-treated male.

^γ Recovered among progeny of female exposed to 37° as first instar larva.

cytology: Placed to the right of 8D8-9 (Demerec, Kaufmann, Fano, Sutton, and Sansome, 1942, Year Book - Carnegie Inst. Washington 41: 191).

***dvr*²**

phenotype: Practically wild type. With *y*², wings tightly curled; with *y*, wings spirally curled. RK2 with *y*.

***dvr*⁸⁴**

phenotype: Produces divers phenotype when hemizygous in *y*² or *In(1)y*^{3PLsc}^{8R} (i.e., *y*^{-ac}), but not *y*⁺ males; *y*^{2 dvr}^{84/y}^{59b}*y* males are *y*⁺ and *dvr* in phenotype. Homozygous females that are *y*² or *y*²; *tra* exhibit normal phenotype.

***dvr*^s: divers-subliminal**

phenotype: Wild type either alone, heterozygous to *dvr*², or in combination with *y*. *y dvr*^{s/y dvr}², on the other hand, has wings distinctly curly or wavy, usually as in typical Cy, but other effects noted in *dvr* flies not evident. RK3.

****dvw*: divergent wings**

location: 1-13.3.

origin: Induced by D-p-N,N-di-(2-chloroethyl)amino-phenylalanine.

discoverer: Fahmy, 1953.

references: 1959, DIS 33: 85.

phenotype: Sex-limited character. Males late hatching; wings divergent, occasionally upheld with inner margins frequently cut away to various degrees. Bristles short and stiff. Homozygous females normal. RK1 in males.

****dw*: dwarf**

location: 3-50.

origin: Spontaneous.

discoverer: Bridges, 13k12.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 101.

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 58 (fig.).

phenotype: Body weight 76% that of heterozygous sibs. Females usually sterile (3 of 63 gave a few offspring). RK3.

***dw-24E*: dwarf in salivary chromosome section 24E**

location: 2-13.

synonym: *dw-24F*.

references: 1941, DIS 14: 49.

phenotype: Body small; abdomen narrow and misshapen. Body surface dull, as if not properly dried. Eyes dull in color and smallish. Wings close textured, small, and tend to droop; crossveins close. Bristles slender. Low viability and fertility. RK3.

alleles:

allele	origin	discoverer	synonym	ref ^α
<i>dw24E</i> ¹	spont	Curry, 39k	<i>dw24F</i>	1
<i>dw24E</i> ²	EMS		<i>dw24F</i> ^{a23}	2
<i>dw24E</i> ³	EMS		<i>dw24F</i> ^{h3}	2

^α 1 = Curry, 1941, DIS 14: 49. 2 = Szidonya and Reuter, 1988, Genet. Res. 51: 197-208.

cytology: Placed in 24E2-F1 based on its inclusion in *Df(2L)dp-h25 = Df(2L)24E2-3;25B2-5* but not in *Df(2L)dp-h19 = Dp(2L)24E5-F1;24F7-25A1* (Szidonya and Reuter, 1988, Genet. Res. 51: 197-208).

***dw-b: dwarf-b**

location: 3-12.
origin: Spontaneous.
discoverer: Bridges, 20b5.
references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 182, 228, 231 (fig.).
phenotype: Flies about 70% as heavy as wild type. RK3.

***dw-sc: dwarf with scute**

location: 1-0.7.
origin: Spontaneous (arose with *sc* and separated).
discoverer: Bridges, 16a22.
phenotype: Small body. Viability erratic. RK3.

dwarf unexpanded: see *dwu*

dwarfex: see *dwx*

dwarfish: see *dwh*

dwarfoid: see *dwf*

dwarp: see *svr*

***dwf: dwarfoid**

location: 1-13.3.
origin: Induced by L-p-N,N-di-(2-chloroethyl)amino-phenylalanine.
discoverer: Fahmy, 1955.
references: 1959, DIS 33: 85.
phenotype: Flies small. Males fertile; viability about 50% wild type. Homozygous females show extreme expression. Fertility and viability low. RK2.

dwg: deformed wings

location: 1-1.47.
phenotype: Most alleles lethal, dying during the first larval instar; the semilethal allele, *dwg*⁷, ceases development from the second instar on, with a few individuals surviving to adulthood. Lethal alleles (*dwg*⁴, *dwg*⁵, *dwg*⁶, and *dwg*⁸) can be recovered as males in combination with variegating duplications, *Dp(1;3)N*²⁶⁴⁻⁵⁸, *Dp(1;3)w*^{m49a}, and *Dp(1;4)w*^{m65g}; have rough reduced dark eyes, sparse vibrissae, often missing orbital, ocellar, and vertical bristles, thickened wing veins, and incised inner wing margins; such males with a *Y* are fertile; *XO* males are not recovered at 25°. Of these alleles only *dwg*⁷ survives as *XO* tissue in gynandromorphs; homozygous germ-line clones in females fail to produce eggs (Garcia-Bellido and Robbins, 1983, Genetics 103: 235-47). *dwg*¹ recorded as having broad, round tipped wings, with occasional marginal incisions and sometimes grossly deformed in shape and venation; extremely fine bristles, and small occasionally rough eyes. Surviving males with any allele are sterile.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>dwg</i> ¹	CB. 3007	Fahmy, 1954		2	
<i>*dwg</i> ²	CB. 3025	Fahmy, 1953	<i>ves</i>	1	
<i>*dwg</i> ³	CB. 3025	Fahmy, 1953	<i>ves</i>	1	
<i>dwg</i> ⁴	NNG	Kaufman	<i>l(1)zw5</i> ^{20z}	3	
<i>dwg</i> ⁵	NNG	Kaufman	<i>l(1)zw5</i> ^{26j}	3	
<i>dwg</i> ⁶	NNG	Kaufman	<i>l(1)zw5</i> ³⁴ⁱ	3	
<i>dwg</i> ⁷	X ray	Alexander	<i>l(1)zw5</i> ⁸²⁷	3	semilethal
<i>dwg</i> ⁸	X ray	Judd	<i>l(1)zw5</i> ¹¹	3	
<i>dwg</i> ⁹	EMS		<i>l(1)zw5</i> ^{e32}	6	
<i>dwg</i> ¹⁰	EMS		<i>l(1)zw5</i> ^{e55}	6	
<i>dwg</i> ¹¹	EMS		<i>l(1)zw5</i> ^{e93}	6	

allele	origin	discoverer	synonym	ref ^α	comments
<i>dwg</i> ¹²	TEM		<i>l(1)zw5</i> ²²³	6	
<i>dwg</i> ¹³	MMS		<i>l(1)zw5</i> ^{m10}	7	
<i>dwg</i> ¹⁴	MMS		<i>l(1)zw5</i> ^{m21}	7	
<i>dwg</i> ¹⁵	MMS		<i>l(1)zw5</i> ^{m24}	7	
<i>dwg</i> ¹⁶	MMS		<i>l(1)zw5</i> ^{m27}	7	
<i>dwg</i> ¹⁷	MMS		<i>l(1)zw5</i> ^{m35}	7	
<i>dwg</i> ¹⁸	MMS		<i>l(1)zw5</i> ^{m36}	7	
<i>dwg</i> ¹⁹	MMS		<i>l(1)zw5</i> ^{m52}	7	
<i>dwg</i> ²⁰	MMS		<i>l(1)zw5</i> ^{m54}	7	
<i>dwg</i> ²¹	MMS		<i>l(1)zw5</i> ^{m63}	7	
<i>dwg</i> ²²	MMS		<i>l(1)zw5</i> ^{m84}	7	
<i>dwg</i> ²³	MMS		<i>l(1)zw5</i> ^{m103}	7	
<i>dwg</i> ²⁴	MMS		<i>l(1)zw5</i> ^{m112}	7	
<i>dwg</i> ²⁵	X ray	Lefevre	<i>l(1)A67</i>	4	
<i>dwg</i> ²⁶	X ray	Lefevre	<i>l(1)GA8</i>	4	
<i>dwg</i> ²⁷	X ray	Lefevre	<i>l(1)HC275</i>	4	
<i>dwg</i> ²⁸	X ray	Lefevre	<i>l(1)JA275</i>	4	
<i>dwg</i> ²⁹	EMS	Lefevre	<i>l(1)VA360</i>	5	
<i>dwg</i> ³⁰	X ray	Lefevre	<i>l(1)VE784</i>	4	
<i>dwg</i> ³¹	spont	Schalet	<i>l(1)20-96</i> <i>l(1)zw5</i> ^{S1}		
<i>dwg</i> ³²	<i>mei-9</i> ^b	Schalet	<i>l(1)zw5</i> ^{S2M}		
<i>dwg</i> ³³	<i>mei-9</i>	Schalet	<i>l(1)zw5</i> ^{S3M}		

^α 1 = Fahmy, 1958, DIS 32: 77; 2 = Fahmy, 1959, DIS 33: 85; 3 = Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56; 4 = Lefevre, 1981, Genetics 99: 461-80; 5 = Lefevre; 6 = Lim and Snyder, 1974, Genet. Res. 24: 1-10; 7 = Liu and Lim, 1975, Genetics 79: 601-11.

^β Spontaneous in the paternal X chromosome of a cross between wild-type males and *mei-9* females, such that the F₁ females were *dwg/mei-9*.

cytology: Placed in 3B5 by Judd, Shen, and Kaufman (1972, Genetics 71: 139-56).

other information: Allelism of *dwg*, *l(1)zw5*, and *ves* inferred from similarity in genetic location and phenotype (Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56).

dwh: dwarfish

location: 3- (not located).
origin: Spontaneous.
discoverer: Bridges, 30d16.
phenotype: Small body. Wings disproportionately broad; eyes irregularly knobby and somewhat dull in color; legs weak and slightly crippled. RK3.

dwp: see *svr*

***dwu: dwarf unexpanded**

location: 1-58.3.
origin: Induced by 2-chloroethyl methanesulfonate.
discoverer: Fahmy, 1956.
references: 1959, DIS 33: 85.
phenotype: Extremely inviable dwarf; wings frequently fail to expand completely. Males fertile if they survive to breed. RK3.

dwx: dwarfex

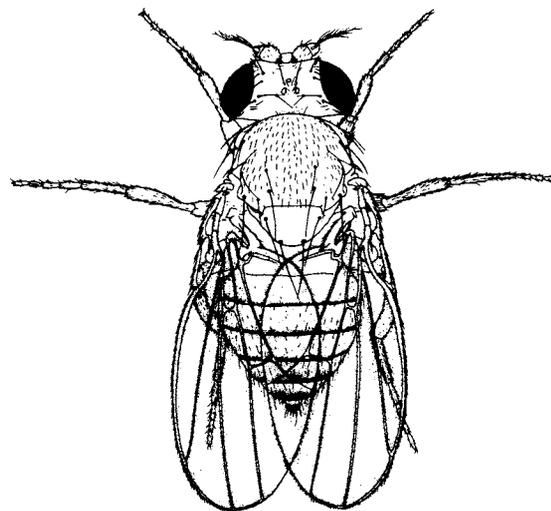
location: 1-33.2.
discoverer: Bridges, 33c31.
phenotype: Body small. Wing texture coarse; marginal hairs slightly disarranged. Classification sometimes difficult. RK3.

dwx^{mn}: see *mn*

dx: deltex**location:** 1-17.0.**origin:** Spontaneous.**discoverer:** Bridges, 22h26.**references:** Morgan, Bridges, and Schultz, 1931, Year Book - Carnegie Inst. Washington 30: 410.**phenotype:** Veins show thickenings and terminal deltas; resembles *Dl* in third chromosomes except fully viable, fertile, and easily classified. Nearly suppressed by *su(dx)*, *Su(dx)*, and *Su(dx)*². RK2.**alleles:** An allele called *dx^{fl}* recovered from natural population by Berg (see Golubovsky, 1983, DIS 59: 42-43) is actually a double mutant also carrying a *P*-factor induced *Sxl^f* allele (Belote). Another allele *dx^{SIM}* was obtained by Schalet.**cytology:** Demerec and Sutton show locus to be between 6A3-4 and 6F10-11 (Demerec, Kaufmann, Fano, Sutton, and Sansome, 1942, Year Book - Carnegie Inst. Washington 41: 191).**dxst: deltex-sterile****origin:** Spontaneous change of *dx* to *dxst*.**discoverer:** Bridges, 31a3.**phenotype:** Veins heavy, confluent, and dilated at junctions; strong deltas at tips. Wings spread wide; margins and tips snipped and nicked. Ocelli sometimes fused with disturbance of hairs and bristles in the region. Acrostichals irregular. Male sterile. Less abnormal phenotype and fertile with *Su(dx)*. RK2.**dy: dusky****location:** 1-36.2 (to the right of *m*).**origin:** Spontaneous.**discoverer:** Bridges, 1611.**references:** Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 35 (fig.), 224.

Slatis and Willermet, 1954, Genetics 39: 45-58.

Dorn and Burdick, 1962, Genetics 47: 503-18.

phenotype: Wings smaller than normal but of nearly wild-type shape, dusky in color. Cell expansion inhibited in prepupal as well as pupal period (Waddington, 1940, J. Genet. 41: 75-139). Partially suppressed by *su(f)* (Dudick, Wright, and Brothers, 1974, Genetics 76: 487-510). RK1.**alleles:** **dy²*, **dy³*, **dy^{31d}*, **dy^{58k}*, **dy^{60k}*, **dy^{61a}*, **dy^{62b}*, **dy²⁸⁶⁻⁹*, **dy^{ala}* (CP627); **dy⁷³* (Green, 1975, Mutat. Res. 29: 77-84) presumably induced by insertion of a transposable element; apparently reverted coincidentally with appearance of the mutable *m* allele *m^{fl}*.**cytology:** Located at 10E2 (Lefevre, 1981, Genetics 99: 461-80).**other information:** The mutant *And*, a rhythmic variant, has a *dy* phenotype and some newly isolated *dy*'s exhibit aberrant (long-period) circadian rhythms.***dyb: dusky body****location:** 1-44.6.**origin:** Induced by ethyl methanesulfonate.**discoverer:** Fahmy, 1958.**references:** 1959, DIS 33: 85.**phenotype:** Dusky body color and browner eyes. Eye and wing shapes slightly altered. Males viable and fertile; females sterile. RK2.**dy: dusky**

Edith M. Wallace, unpublished.

e: ebony

location: 3-70.7.

discoverer: E. M. Wallace, 12b15.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 50 (fig.).

phenotype: Body color varies from shining black to slightly darker than wild type, depending on allele. Puparia much lighter than wild type. Classifiable throughout larva period by darkened color of spiracle sheaths (Brehme, 1941, Proc. Nat. Acad. Sci. USA 27: 254-61). Viability lowered to about 80% wild type. Heterozygotes for dark alleles have slightly darker body color than normal. For interaction with other body color mutants, see Waddington (1941, Proc. Zool. Soc. London, Ser. A 111: 173-80). Postulated to encode β -alanyl dopamine synthetase, a 90-kd enzyme that requires ATP and $MgCl_2$ to catalyze the formation of N- β -alanyl dopamine from β alanine and dopamine; β alanyl dopamine absent from newly eclosed *e* flies (Wright, 1987, Adv. Genet. 24: 127-222); β alanine and dopamine accumulate in pupae and pharate adults, dopamine to twice normal levels, in *e* and *e¹¹* homozygotes; levels return to normal in older adults (Hodgetts, 1972, J. Insect Physiol. 18: 937-47; Hodgetts and Konopka, 1973, J. Insect Physiol. 19: 1211-20). Unable to utilize β alanine in tanning of puparium. Labeled β alanine or uracil injected into *e* pupae remains in hemocoel, not incorporated into pupal case as in +; light-colored pupa result; *e*/+ intermediate in these respects. Only newly emerged + adults incorporate uracil or β alanine into cuticle; *e* flies and older + flies do not; β alanine toxic to the latter two types (Jacobs, 1968, Biochem. Genet. 1: 267-75). Defect in tanning leads to spongy cuticle which responds to β alanine administration (Jacobs, 1980, Biochem. Genet. 18: 65-76). Phenylthiocarbamide inhibits development of *e¹¹* homozygotes more than wild type; reverse is true for inhibition by silver chloride; heterozygotes intermediate in both cases. Mixtures of the two inhibitors affect heterozygotes to a greater extent (Kroman and Parsons, 1960, Nature 186: 411-12). Electroretinograms of *e* flies abnormal; lamina potential reduced or absent (Hotta and Benzer, 1969, Nature 222: 354-56). Threshold for phototaxis 200-fold higher than that for wild type; high sensitivity (retinulae 1-6) optomotor threshold 500 times normal and high acuity (retinulae 7 and 8) optomotor threshold ten times normal [Heisenberg, 1972, Information Processing in the Visual Systems of Arthropods (R. Wehner, ed.). Springer-Verlag, Berlin, Heidelberg, and New York, pp. 265-68]. *e* flies more sensitive to polarized light than wild type (Heisenberg, 1972). Abnormal distribution of uptake of ³H-GABA by the lamina ganglionaris described by Campos-Ortega (Cell Tissue Res. 147: 415-31). Reduced mating success compared to wild type (Rendel, 1951, Evolution 5: 226-30). Courtship frequently aborts owing to mismounting by male [Crossley and Zuill, 1970, Nature (London) 225: 1064-65]; relative mating success increased in dark (Kyriacou, 1981, Anim. Behav. 29: 462-71) but not according to Crossley (1970, DIS 45: 170). Courtship shows deficiency in wing vibration; low proportion of sine song and long intrapulse interval; *e*/+ outsings +/+ (Kyriacou, Burnet, and Connolly, 1978, Anim. Behav. 26: 1195-1206). RK1.

alleles: Changes at *e* not known to be associated with

deficiency for the locus tabulated below. *e¹* inseparable from *In(3R)C*; stocks labeled as carrying *e*, but not *In(3R)C*, for the most part carry *e⁴* (Craymer).

allele	origin	ref ^α	phenotype	cytology
<i>e¹</i>	spont	1, 2	dark	<i>In(3R)C</i>
<i>e⁴</i>	spont	1, 2	dark	
<i>e¹¹</i>	spont	2, 5	dark	
<i>e^{60h}</i>	spont	2, 3	intermediate	
<i>e^{100.265}</i>	X ray	2, 8	dark	<i>In(3R)93B5-6;95E</i>
<i>e^{100.307}</i>	X ray	2, 8		
<i>e^{300.96}</i>	X ray	2, 8	dark	<i>In(3R)89F-90A;99B2-4</i>
<i>e^{AFA}</i>	X ray	4		<i>In(3R)86C;96D1-6</i>
<i>e^{AT5}</i>	EMS	4		
<i>e^{AT8}</i>	EMS	4		
<i>e^{D8}</i>	X ray	4		<i>T(2;3)40-41;93D1-6</i>
<i>e^{D12}</i>	X ray	4		<i>In(3R)92E12-13;93D1-6</i>
<i>e^{F5}</i>	X ray	4		
<i>e^{F6β}</i>	X ray	4		
<i>e^s</i>	spont	1, 2	intermediate	
<i>est</i>	spont	2, 7, 8	intermediate	
<i>e^{Ug}</i>	spont	2, 9	light	
<i>e^x</i>				

^α 1 = Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. 327: 59, 99, 184; 2 = CP627; 3 = Ives, 1965, DIS 40: 55; 4 = Scalenghe and Ritossa, 1976, Atti Accad. Lincei 13: 439-528; 5 = Stern, 1926, Z. Indukt. Abstamm. Vererbungsl. 41: 198-215; 6 = Villee, 1941, DIS 14: 40; 7 = Villee, 1942, Univ. Calif. Berkeley, Publ. Zool. 49: 137; 8 = Ward and Alexander, 1957, Genetics 42: 42-54; 9 = Zurcher, 1963, Genetics 34: 1-33 (fig.).

^β Probably independent of *e* mutation.

cytology: Placed in 93D2-6 by D'Alessandro, Ritossa, and Scalenghe (1977, DIS 52: 46) based on its being the position of the breakpoint common to a number of *e* rearrangements. Probably at the left end of this region based on *Df(3R)e671 = Df(3R)93B5-7;93D3* (Korge, 1972, DIS 48: 20) and *Df(3R)e-H5 = Df(3R)93B10-13;93D2-3* (Henikoff, 1980, DIS 55: 61-62).

E74: Ecdysone-inducible gene encoded in region 74 (W.A. Segraves)

location: 3-{45}.

synonym: *Eip74EF*.

references: Burtis, 1985, PhD Thesis, Stanford University. Janknecht, Taube, Lüdecke and Pongs, 1989, Nucleic Acids Res. 17: 4455-64.

Burtis, Thummel, Jones, Karim and Hogness, 1990, Cell 61: 85-99.

Thummel, Burtis and Hogness, 1990, Cell 61: 101-11.

phenotype: Encodes an ecdysone-inducible gene associated with the early puff at 74E-F. Periodic expression of the gene follows, by approximately one hour, pulses of ecdysone occurring during development; two periods of expression occur independently of ecdysone pulses, one at the end of embryogenesis and one at the end of pupal development. Return to base line levels of expression requires protein synthesis and presumably results from repression by ecdysone-induced gene products; *in situ* hybridization to larvae detects transcription of *E74* in most tissues, both imaginal and strictly larval.

alleles: Two mutant alleles selected by Burtis (1985); one dies in the pharate-adult stage, whereas the other is a prepupal lethal, showing defects in larval shortening and cuticular tanning; a low frequency of defective prepupae

reach the pharate adult stage.

allele	origin	discoverer	synonym	comments
E74¹	X ray	Burtis, 1985	<i>E74^{x1001}</i>	pharate adult lethal; <i>T(2;3)29A-C;74E-F</i> ^α
E74²	EMS	Burtis, 1985	<i>E74^{e3109}</i>	prepupal lethal

^α Broken in first intron of the long transcript.

cytology: Placed in early ecdysone-inducible puff at 74E-F by *in situ* hybridization.

molecular biology: The gene is 6 kb in length; three different transcripts produced by transcription from right to left. The long transcript comprises eight exons and is dispersed over all 6 kb; the mature message encodes a protein of 829 amino-acids and 87.1 kd. The other two transcripts are shorter and are initiated from promoters 300 base pairs apart in the fifth of seven introns of the long transcript; they have four exons, the last three being the same as those of the long transcript; thus the proteins share their C-terminal sequences but have different N-terminal ends; both short transcripts encode the same polypeptide of 833 amino acids and 94.7 kd. The sequences indicate a polypeptide with an acidic N terminus and a basic C terminus separated by a region of homopolymeric repeats. A segment of 84 amino acids in the C-terminal end shows 50% identity with a similarly situated sequence in the protein encoded by the human *c-ets-2* oncogene. Thummel *et al.* demonstrate that *E74* is transcribed at 1.1 kb per minute such that an hour is required from the time of ecdysone stimulation until the first mature transcripts are produced.

E75 (W.A. Segraves)

location: 3-{46}.

synonym: *Eip75*.

references: Segraves, 1988, PhD Thesis, Stanford University.

Feigl, Gram, and Pongs, 1989, *Nucleic Acids Res.* 17: 7167-78.

Segraves and Hogness, 1990, *Genes Dev.* 4: 204-19.

phenotype: Encodes an ecdysone-inducible gene associated with the early puff at polytene position 75B. Periodic expression occurs in response to ecdysone pulses occurring at specific times during development; regression of puff and reduction of *E75* expression in third instar does not occur in the presence of cycloheximide, and presumably depends on ecdysone-induced gene products.

alleles: 28 alleles recovered by Segraves (1988), falling into two groups, one displaying early lethality (early larval for the combinations examined) and one displaying pharate-adult lethality.

allele	origin	synonym	comments
E75¹	X ray	<i>E75^{x37}</i>	pharate-adult lethal over <i>E75⁴</i> ; 60 kb deletion ^α
E75²	X ray	<i>E75^{x44}</i>	early lethal; <i>In(3L)75B3-5;75F3-5</i> ^β
E75³	X ray	<i>E75^{x46}</i>	early lethal
E75⁴	X ray	<i>E75^{x48}</i>	105-kb deletion ^γ
E75⁵	X ray	<i>E75^{x51}</i>	larval-pupal lethal
E75⁶	EMS	<i>E75^{e101}</i>	early lethal
E75⁷	EMS	<i>E75^{e102}</i>	pupal lethal
E75⁸	EMS	<i>E75^{e109}</i>	early lethal
E75⁹	EMS	<i>E75^{e149}</i>	early lethal ^δ
E75¹⁰	EMS	<i>E75^{e165}</i>	early lethal
E75¹¹	EMS	<i>E75^{e172}</i>	early lethal

allele	origin	synonym	comments
E75¹²	EMS	<i>E75^{e174}</i>	pharate-adult lethal
E75¹³	EMS	<i>E75^{e197}</i>	pharate-adult lethal
E75¹⁴	EMS	<i>E75^{e198}</i>	early lethal ^δ
E75¹⁵	EMS	<i>E75^{e202}</i>	early lethal
E75¹⁶	EMS	<i>E75^{e203}</i>	early lethal
E75¹⁷	EMS	<i>E75^{e207}</i>	early lethal
E75¹⁸	EMS	<i>E75^{e213}</i>	early lethal
E75¹⁹	EMS	<i>E75^{e264}</i>	early lethal
E75²⁰	EMS	<i>E75^{e273}</i>	pharate-adult lethal
E75²¹	EMS	<i>E75^{e281}</i>	early lethal
E75²²	EMS	<i>E75^{e283}</i>	pharate-adult lethal
E75²³	EMS	<i>E75^{e295}</i>	early lethal ^δ
E75²⁴	EMS	<i>E75^{e304}</i>	early lethal
E75²⁵	EMS	<i>E75^{e316}</i>	early lethal
E75²⁶	EMS	<i>E75^{e341}</i>	early lethal
E75²⁷	EMS	<i>E75^{e355}</i>	early lethal
E75²⁸	EMS	<i>E75^{e397}</i>	early lethal

^α Breaks in the long transcription unit immediately downstream from the first exon.

^β Breaks immediately downstream from the first exon of the *E75 A* transcription unit; chromosome has an associated, possibly complex, inversion from 67B to the base of 3R.

^γ Removes the entire *E75* gene and eliminates the 75B puff.

^δ Lost.

cytology: Placed in early ecdysone-inducible puff at 75B by *in situ* hybridization.

molecular biology: 350 kb from 75B region cloned and used to identify two overlapping ecdysone-responsive transcription units; *E75 A* and *E75 B*. The *E75 A* unit contains six exons spread over 50 kb, and gives rise to mRNA's of 4.9 or 5.7 kb by use of alternate polyadenylation sites within the final exon. The *E75 B* unit begins within the second intron of *E75 A* and contains five exons, the final four of which are shared with *E75 A*, and produces mRNA's of 5.2 and 6.0 kb. The polypeptides encoded by the two thus contain different N termini and a common C terminus. The *E75 A* polypeptide contains 266 unique and 971 shared amino acids for a molecular weight of 132 kd; the *E75 B* polypeptide contains 422 + 971 amino acids and has a molecular weight of 151 kd. The conceptual amino acid sequence exhibits striking homology to the steroid receptor superfamily of proteins. The homology is restricted to the DNA-binding and hormone-binding domains of the proteins. The two polypeptides differ most significantly in that the *E75 A* protein contains both of the characteristic zinc fingers of the steroid-receptor-like DNA-binding domain, whereas the *E75 B* protein contains only one. A third type of *E75* cDNA has been reported, which closely resembles the *E75 A* mRNA except for the substitution of novel sequences for the first *E75 A* exon (Feigl *et al.*, 1989). This would result in the production of an *E75* protein with the same DNA-binding and hormone-binding domains as the *E75 A* protein, but with a different amino terminus. These novel sequences are transcribed from an exon 50 kb upstream of the *E75 A* promoter, which defines the > 100 kb *E75 C* transcription unit (Segraves, unpublished).

E(2)Bic: see *E(Bic)*

E(A^{53g}): Enhancer of abnormal abdomen-53g

location: 2-6 (between *al* and *dp*).

origin: Spontaneous.

references: Thalmann, 1974, *DIS* 51: 22.

phenotype: Extreme intensification of *A53g* phenotype;

flies nearly denuded of tergites. Does not enhance *S*, and *E(S)*, which maps in same region, does not enhance *A53g*.

E(Arp): see *Ba*¹⁰

***E(B)*: Enhancer of Bar**

location: 1-57.3.

origin: Spontaneous.

discoverer: Bonnier and Nordenskiöld.

synonym: *i*; *I*: *Intensifier of Bar*; *Eb*: *Exaggeration of Bar*.

references: 1942, DIS 16: 47.

Bonnier, Nordenskiöld, and Bågman, 1943, *Hereditas* 29: 113-33 (fig.).

Rasmuson, 1948, *Proc. Intern. Congr. Genet.*, 8th. pp. 645-46.

phenotype: *E(B)* heterozygous with any *B* allele, including *B*⁺, produces flies similar in phenotype to homozygotes for that allele. *B* +/+ *E(B)* eyes have 80-90 facets, but *B E(B)*/++ eyes have only 40. Homozygous lethal. RK2(A).

cytology: Salivary chromosomes appear normal, but there is occasional indication of deficiency for faint bands 16A5 and 6.

other information: Reduces *B-fu* crossing over about 40%.

E(Bic)

location: 2-64.0.

origin: Induced by ethyl methanesulfonate.

synonym: *E(2)Bic*.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, *Roux's Arch. Dev. Biol.* 193: 267-82.

Mohler and Wieschaus, 1986, *Genetics* 112: 803-22.

phenotype: An incompletely penetrant dominant female-sterile mutation; heterozygous females produce 60% apparently unfertilized eggs. In heterozygotes with *Bic-C* or *Bic-D*, *E(Bic)* causes a substantial increase in the incidence of double-abdomen embryos, but *E(Bic)*/+ females produce normal embryos.

e(bx): see *z^{ebx}*

***E(bx)*: Enhancer of bithorax**

location: 3- (to the left of R).

origin: X ray induced.

discoverer: E. B. Lewis.

synonym: *En-bx*.

phenotype: Enhances expression of *bx^{34e}*, *bx³*, and *Ubx*/+. Lethal homozygous. RK2.

***E(da)*: Enhancer of daughterless**

location: 2-55 (inferred from rearrangement breakpoint).

origin: X ray induced.

references: Mange and Sandler, 1973, *Genetics* 73: 73-86.

phenotype: One dose of *E(da)* in *da*/+ females, in either coupling or repulsion with *da*, reduces the number of daughters among their progeny; without effect in *da*/+ males. *da E(da)*/*SMI* females are daughterless. Effect enhanced by reduced heterochromatic content of progeny (Mange and Sandler, 1973). Maternal effect of *E(da)* strong at 29° but absent at 17° (Cline 1980, *Genetics* 96: 903-26). *Sxl^{fl}*/+; *da E(da)*/+ females produce no *Sxl^{fl}*/+ daughters at 29° (Cline, 1980). *E(da)*/*abo* females produce normal sex ratios; show zygotic

suppression of the maternal effect of homozygous *abo* (Mange and Sandler, 1973).

cytology: Associated with *T(2;3)E(da) = T(2;3)41,66C*. Sandler and Mange (1973) suppose *E(da)* to be a consequence of the break in 2R.

***e(dp^v)*: enhancer of dumpy-vortex**

location: 3-40.4.

origin: Spontaneous.

discoverer: Bridges, 16h7.

synonym: *vo-3*: *vortex in chromosome 3*.

references: 1919, Bridges and Mohr, *Genetics* 4: 283-306 (fig.).

1923, Bridges and Morgan, *Carnegie Inst. Washington Publ. No. 327*: 168.

1925, Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 41-43 (fig.).

phenotype: Normal. In combination with *dp^v*, produces one or two pairs of pits or volcano-like protrusions on thorax; hairs and bristles arranged in whorls. RK3.

****E(f)*: Enhancer of forked**

location: 2-86.5.

origin: X ray induced.

discoverer: Belgovsky, 37c4.

synonym: *1-f*: *Intensifier of forked*.

references: 1937, DIS 8: 7.

1938, *Izv. Akad. Nauk SSSR, Ser. Biol.* 1017-36.

1940, DIS 13: 52.

1944, *Zh. Obshch. Biol.* 5: 325-56.

phenotype: Homozygote has short, twisted bristles intermediate between *f* and *Bl*; postscutellars often pale; viability and fertility reduced. Heterozygote is wild type. *fl* +; *E(f)*/+ slightly more extreme than *f. flf*; *E(f)*/+ has an extreme forked phenotype and hairs are forked. *flf*; *E(f)*/*E(f)* rarely survives. RK3.

cytology: Salivary chromosomes normal.

***e(fa^{swb})*: enhancer of facet-strawberry**

location: 1-1.5 (no recombination with *w^a*).

origin: Spontaneous.

discoverer: Keppy.

synonym: *e(fa^{swb}) fa^{swb}* which was originally designated *fa^{swb-h}*: *facet-strawberry-hairy*.

references: Welshons and Welsons, 1986, *Genetics* 113: 337-54.

phenotype: By itself *e(fa^{swb})* is wild type in phenotype; when in *cis* with *fa^{swb}*, either in homozygous or hemizygous condition, it produces narrow, rough, glossy eyes, bowed tibiae on the metathoracic legs, and a proliferation of microchaetae. *e(fa^{swb}) fa^{swb}/fa^g* have glossy phenotype; *fa^{swb}/fa^g* have facet phenotype.

cytology: Postulated to be an inversion of bands 3C2 and 3 on the basis of its elimination of *w^a-rst* recombination and its normal appearing polytene banding pattern.

****e(g)*: enhancer of garnet**

location: 1-5.9.

discoverer: Payne and Denny, 1921.

synonym: *m(g)*: *modifier of garnet*.

references: 1921, *Am. Naturalist* 55: 377-81.

phenotype: Apparently wild type but, in combination with *g*, produces a more orange eye than *g* alone. RK3.

E(H): see *Su(H)*

***E(lz)*: Enhancer of lozenge**

location: 3-64.0 (between *sr* and *Dl*).
origin: Induced by ethyl methanesulfonate.
discoverer: Grell.
phenotype: Dominant enhancer of lozenge; no effect observed in absence of *lz*. Homozygous viable.

****E(M99E)*: Enhancer of Minute (3) 99E**

location: 3- (near spindle attachment).
origin: Spontaneous.
discoverer: Bridges.
synonym: *E(M3g)*.
phenotype: Specific intensifier of shortness of bristles of *M(3)99E*. RK3.

****e(N⁸)*: enhancer of Notch-8**

location: 3- (not located).
origin: Spontaneous.
discoverer: Mohr, 181.
references: 1923, Z. Indukt. Abstamm. Vererbungsl. 32: 108-232 (fig.).
phenotype: Produces slight nicking of wings. Enhances *N⁸*. RK3.

***E(Pc)*: Enhancer of Polycomb (*R. Denell*)**

location: 2-61.9 (0.1 cM to the left of *en*).
origin: Induced by ethyl methanesulfonate.
synonym: *l(2)28-28-12*.
references: Russel and Eberlein, 1979, Genetics 91: s109. Sato, Russel, and Denell, 1983, Genetics 105: 357-70. Sato, Hayes, and Denell, 1984, Dev. Genet. 4: 185-98.
phenotype: Although *E(Pc)* heterozygotes are cuticularly normal as larvae and adults, the mutation acts zygotically as a dominant enhancer of the adult homeotic syndrome of flies heterozygous for Polycomb mutations or *Df(3L)Pc-MK*. The locus is haplo-abnormal, as heterozygous deficiencies of the *E(Pc)* locus have an equivalent effect. Heterozygosity for *E(Pc)* or a deficiency also enhances the adult homeotic syndrome of Polycomblike mutation or deficiency heterozygotes, and renders the normally recessive mutations *esc* and *l(4)29* slightly pseudodominant, but has no effect on phenotypes associated with *Antp^{Ns}*, *Antp^{73b}*, *Antp^{Scx}*, *Antp^{EfW15}*, *Scr^{Msc}*, *Cbx*, *Ubx*, *bx^{34e}* or *abx bx³ pbx* heterozygotes. *E(Pc)* is a recessive lethal mutation; homozygotes and hemizygotes die as late embryos or larvae, which appear cuticularly normal. *E(Pc)* has dominant maternal as well as zygotic effect on the severity of the embryonic homeotic syndrome of *Pc³* or *Df(3L)Pc-MK* homozygotes. Zygotic homozygosity for *E(Pc)* also enhances the embryonic effects of *Pcl^{W4}*, *Pcl^{W5}*, and *Pcl^{W6}*, and *E(Pc)*; *l(4)29* doubly homozygous embryos from heterozygous mothers show incomplete head involution, presumably due to a cryptic homeotic effect.

cytology: Placed in 48A3-6 based on its inclusion in *Df(2R)en30 = Df(2R)48A3-4;48C6-8* and *Df(2R)en-A = Df(2R)47D3-4;48A5-6*.

E(Pc)-a

location: 3- (near *Dl*).
discoverer: E.B. Lewis and Chang.
references: Eberlein, 1984, Genetics 107: s26.
phenotype: Homozygous lethal; heterozygotes show partial transformation of legs 2 and 3 to first leg and occasionally of first and fourth abdominal segments to second and fifth, respectively. Phenotype enhanced by combina-

tion with *Pc* or *E(Pc)*.

****e(S)*: enhancer of Star**

location: 3- (between 0 and 10; perhaps an allele of *ru* or *R*).
origin: Spontaneous.
discoverer: Bridges, 16k18.
synonym: *S-i*: intensifier of *Star*.
references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 175 (fig.).
phenotype: By itself, homozygous *e(S)* has normal eyes. *S/+*; *e(S)/e(S)* has eyes smaller and rougher than *S/+*, although overlapping somewhat; abdomen bulbous; body color darkish. RK3.

***E(S)*: Enhancer of Star**

location: 2-6 [claimed to lie between left break of *In(2L)Cy* and locus of *Cy*].
discoverer: Bridges, 30a27.
phenotype: *E(S)/+* normal, *E(S)/E(S)* gives slight roughening of eye. *E(S)/+* strongly reduces size and increases roughness of *S/+* and *S²/+* eyes; imparts dominance to *ast/+*, *ast²/+*, *ast³/+*, and *ast⁴/+* (Lewis, 1945, Genetics 30: 137-66). *S/+ + E(S)* occasionally emerges as a late-eclosing giant. RK3A.
cytology: Arose in *In(2L)Cy = In(2L)22D1-2;33F5-34A1*. Placed in 22D3-E1 on the basis of enhancement of *S* by a duplication for the interval (Gelbart).

***E(sd)*: Enhancer of scalloped**

location: Autosomal.
origin: Spontaneous.
discoverer: R. M. Valencia, 1963.
references: 1965, DIS 40: 37.
phenotype: Almost completely removes wings of *sd^{SP}*; not tested with other alleles of *sd*. No interaction with *Bx* or *Bx^r*. RK2.

***E(Sd)*: Enhancer of Segregation Distorter**

location: 2-55.
origin: Found on *SD* chromosomes.
references: Ganetzky, 1977, Genetics 86: 321-55.
phenotype: In the presence of *E(SD)*, either in coupling or repulsion, *Sd* characterized by *k* values of $>.95$. Deletion of *E(SD)* reduces *k* values to .65-.75. Non-*SD* chromosomes carry either no allele or a nonenhancing allele. *E(Sd)* in the absence of *Sd* able to cause differential recovery of *Rsp⁺* and *Rsp⁻* chromosomes (Sharp; Temin).
cytology: Located in proximal heterochromatin of 2L. Deleted by three deficiencies for *lt*, *Df(2L)ESD1*, *Df(2L)ESD3*, and *Df(2L)ESD36*.

***e(sei)*: enhancer of seizure**

location: 3-39.0.
origin: Spontaneous.
references: Kasbekar, Nelson, and Hall, 1987, Genetics 116: 423-31.
phenotype: In the homozygous condition, it lowers the temperature threshold of two temperature-sensitive alleles of *sei*. *sei^{ts2}*, which is a dominant paralytic at 40°, a recessive paralytic at 38° and nonparalytic at 35°, becomes a dominant paralytic at 38° and displays a recessive hyperactive uncoordinated phenotype at 35° in the presence of homozygous, but not heterozygous *e(sei)*. Homozygous *sei^{ts1}* which is normal at 35° becomes

paralyzed at that temperature in the presence of homozygous *e(sei)*. Flies homozygous for *e(sei)* by itself, when their container is tapped lightly, become hyperactive and unable to crawl up the sides of the container to the top. *e(sei)* flies exhibit spontaneous neuronally stimulated firing of the dorsal longitudinal muscle and greatly enhances such firing of *sei*^{ts2}. Also *e(sei)* does not affect saxitoxin binding by membrane extracts as do *sei* mutations.

cytology: Placed in 69A4-B5 based on its inclusion in *Df(3L)vin7 = Df(3L)68C8-11;69B4-5* but not *Df(3L)vin6 = Df(3L)68C8-11;69A4-5* or *Df(3L)vin5 = Df(3L)68A2-3;69A1-3*.

***E(spl)*: Enhancer of split**

location: 3-89.

references: Welshons, 1956, DIS 30: 157-58.

Von Halle, 1965, DIS 40: 60.

Lehmann, Jiménez, Dietrich, and Campos-Ortega, 1983, Wilhelm Roux's Arch. Dev. Biol. 192: 62-74 (fig.).

Vässin, Wielmetter, and Campos-Ortega, 1985, J. Neurogenet. 2: 291-308.

Knust, Bremer, Vässin, Ziemer, Tepass, and Campos-Ortega, 1987, Dev. Biol. 122: 262-73 (fig.).

de la Concha, Dietrich, Weigel, and Campos-Ortega, 1988, Genetics 118: 499-508.

Ziemer, Tietze, Knust, and Campos-Ortega, 1988, Genetics 119: 63-74.

phenotype: Locus involved in the differentiation of the neural ectoderm into neuroblasts and epidermoblasts. Increased levels of gene product favor epidermal differentiation, whereas decreased levels favor neuronal differentiation. Locus originally identified by the split-enhancing feature of a dominant gain-of-function mutation. Loss of function mutations are lethals and are described separately. A weak hypomorphic allele described as *gro* (*groucho*) is also described below. *E(spl)* causes *spl/+* to display a split phenotype and elicits a more extreme phenotype in *spl/spl* and *spl/Y*. The *spl*-enhancing effect of *E(spl)*¹ is suppressed in flies heterozygous for *Dl* (Shepard, Boverman, and Muskavitch, 1989, Genetics 122: 429-38). With respect to enhancement, *+/+/+ < +/E(spl) < E(spl)/+/+ < E(spl)/E(spl)*, in accord with expectations from a hypermorphic allele; duplication for *E(spl)*⁺ achieved with *Dp(3;3)M95A⁺16*. In the absence of *spl*, *E(spl)* causes slight roughening of the eyes; furthermore, depending on parental constitution, varying percentages of embryos display defects in central- and peripheral-nervous-system development and irregular cuticular defects. A fraction of these fail to develop; percentages vary from 25% neural hypoplasia and 8% embryonic mortality in crosses between homozygous *E(spl)* parents to 100% death and 78% neural hypoplasia when both parents are *E(spl)/Dp(3;3)M95A⁺*. Both of these effects are sensitive to maternal genotype. That *E(spl)* is not simply a hypermorph is indicated by the fact that although *+/Df(3R)Espl* is viable, *E(spl)/Df(3R)Espl* is virtually lethal, especially when the deficiency is maternally inherited. Embryos homozygous for loss-of-function alleles vary in phenotype from weak to very strong neural hyperplasia, with concomitant aplasia of the epidermal sheath. Heterozygotes for stronger hypomorphic alleles may show terminal thickening of wing veins L4 and L5,

and may have adventitious vein segments in the posterior wing membrane. Double heterozygotes for *E(spl)* loss-of-function alleles and either *N* or *Dl* are lethal. In the adult, increasing levels of *E(spl)* function result in increasing levels of split enhancement and in decreased numbers of sensilla as measured by the number of costal bristles on the wing. Conversely, decreased *E(spl)* function results in larger eyes and more sensilla plus ectopic sensory neurons appearing in the wing blade, especially along the posterior margin. Hemizygoty for *E(spl)*⁺ completely suppresses *spl*. Three doses of *E(spl)*⁺ increase the severity of the effects of the absence of function of *Dl*, reduce the severity of the absence of function of *N*, *neu* and *mam* and are without effect on the phenotype of *bib*⁻; conversely, absence of function of *E(spl)* is not affected by hyperploidy for any of the neurogenic loci or by loss of *H* function; from this De la Concha *et al.* infer that *E(spl)* is positively controlled by *N* and negatively controlled by *H* and *Dl*. Unlike the results using other neurogenic mutants, single vitally stained cells taken from the neurogenic ectoderm of *E(spl)*⁻ embryos and transplanted into wild-type host embryos fail to give rise to clones containing epidermal cells; only neuronal elements are produced. This observation is interpreted to indicate that the *E(spl)*⁺ product serves a receptor rather than a signalling function (Technau and Campos-Ortega, 1987, Proc. Nat. Acad. Sci. USA 84: 4500-04).

alleles: One true mutant allele is the original dominant *E(spl)* and it is likely a double mutant; a number of recessive lethals recovered either as reversions of *E(spl)*, designated *E(spl)*^{rv} and mostly associated with chromosome rearrangements, or as mutations designated *E(spl)*^r that fail to complement *Df(3R)Espl-3*, a cytologically visible deficiency that contains more than eleven transcription units. These lethals display variable levels of neural hyperplasia and are treated as alleles in the literature; however interactions among them in *trans* heterozygotes lead to complex complementation patterns, and in general, the mutations have not been associated with particular transcription units.

allele	origin	discoverer	synonym	ref ^α	comments
<i>E(spl)</i>¹	spont	Green	<i>E(spl)</i> ^D	1, 4, 5	unique dominant allele; 0.7-kb deletion in 12 to 14 kb; 5-kb insertion at 16.5 kb
<i>E(spl)</i>²	spont	Grell,64k	<i>gro: groucho</i>		viable hypomorphic allele?
<i>E(spl)</i>^{r3}	EMS		<i>E(spl)</i> ^{A4}	5	
<i>E(spl)</i>^{r4}	EMS		<i>E(spl)</i> ^{B7}	5	
<i>E(spl)</i>^{r5}	EMS		<i>E(spl)</i> ^{B12}	5	
<i>E(spl)</i>^{r6}	EMS		<i>E(spl)</i> ^{B37}	5	
<i>E(spl)</i>^{r7}	EMS		<i>E(spl)</i> ^{B48}	5	
<i>E(spl)</i>^{r8}	EMS		<i>E(spl)</i> ^{B88}	5	
<i>E(spl)</i>^{r9}	EMS		<i>E(spl)</i> ^{B93}	5	
<i>E(spl)</i>^{r10}	EMS		<i>E(spl)</i> ^{B94}	5	
<i>E(spl)</i>^{r11}	EMS		<i>E(spl)</i> ^{B95}	5	
<i>E(spl)</i>^{r12}	EMS		<i>E(spl)</i> ^{B102}	5	
<i>E(spl)</i>^{r13}	EMS		<i>E(spl)</i> ^{B115}	5	
<i>E(spl)</i>^{r14}	EMS		<i>E(spl)</i> ^{B128}	5	
<i>E(spl)</i>^{r15}	X ray	Muskavitch	<i>E(spl)</i> ^{BX21}	3	<i>T(2;3)40-41;96F9-10</i>
<i>E(spl)</i>^{r16}	X ray	Muskavitch	<i>E(spl)</i> ^{BX22}	3	~14-kb deletion; 4 to 17.5 kb ~14-kb inversion; breaks in -10.5 to -7.6 and 4 kb
<i>E(spl)</i>^{r17}	EMS	Preiss	<i>E(spl)</i> ^{E28}	3	
<i>E(spl)</i>^{r18}	EMS	Preiss	<i>E(spl)</i> ^{E48}	3	
<i>E(spl)</i>^{r19}	EMS	Preiss	<i>E(spl)</i> ^{E73}	3	
<i>E(spl)</i>^{r20}	EMS	Preiss	<i>E(spl)</i> ^{E75}	3	
<i>E(spl)</i>^{r21}	EMS	Preiss	<i>E(spl)</i> ^{E77}	3	

allele	origin	discoverer	synonym	ref ^α	comments
<i>E(spl)</i> ^{r22}	EMS	Preiss	<i>E(spl)</i> <i>E107</i>	3	
<i>E(spl)</i> ^{r23}	X ray		<i>E(spl)</i> <i>L5</i>	2, 5	
<i>E(spl)</i> ^{r24}	X ray		<i>E(spl)</i> <i>L9</i>	5	
<i>E(spl)</i> ^{r25}	X ray	Tietz	<i>E(spl)</i> <i>L11</i>	2, 5	<i>T(3;4)96F7-11;102B-C</i> ; breakpoint in -7 to -5 kb
<i>E(spl)</i> ^{r26}	X ray		<i>l(gro)</i> <i>X115</i>	3	
<i>E(spl)</i> ^{rv27}	X ray	Knust and Ziemer	<i>E(spl)</i> <i>R-A7.1</i>	1, 2, 5	34-kb deletion; -15 to +19 kb
<i>E(spl)</i> ^{rv28}	X ray	Knust and Ziemer	<i>E(spl)</i> <i>R-C1.4</i>	1, 2, 5	<i>T(3;4)96F12-14;102E-F</i> ; breakpoint in m3 at -5 to -6 kb
<i>E(spl)</i> ^{rv29}	X ray		<i>E(spl)</i> <i>R-F4.4</i>	2, 5	lesion in -15.5 to -11.5 kb
<i>E(spl)</i> ^{rv30}	X ray		<i>E(spl)</i> <i>R-F6.2</i>	2, 5	lesion in 19 to 20.5 kb
<i>E(spl)</i> ^{rv31}	X ray		<i>E(spl)</i> <i>R-H2.1</i>	5	
<i>E(spl)</i> ^{rv32}	X ray	Bremer	<i>E(spl)</i> <i>R14.8</i>	1, 2, 5	<i>T(3;4)96F8-9</i> ; <i>100F5;102B-D</i> breakpoint at ~-65 kb

^α 1 = Knust, Bremer, Vässin, Ziemer, Tepass and Campos-Ortega, 1987, Dev. Biol. 122: 262-73; 2 = Knust, Tietze and Campos-Ortega, 1987, EMBO J. 6: 4113-23; 3 = Preiss, Hartley and Artavanis-Tsakonas, 1988, EMBO J. 7: 3917-27; 4 = Welshons, 1956, DIS 30: 157-58; 5 = Ziemer, Tietze, Knust and Campos-Ortega, 1988, Genetics 119: 63-74.

cytology: Placed in 96F11-14 based on its inclusion in *Df(3R)ro82b = Df(3R)96F11-14;97F3-11* but not *Df(3R)Tl-Q = Df(3R)97A1;97D* (Preiss et al.).

molecular biology: Region isolated in two independent walks both initiated from the same cloned sequence: a 150-kb walk (Knust, Tietze and Campos-Ortega, 1987, EMBO J. 6: 4113-23) with an *EcoRI* site just to the right of the initiating clone designated as coordinate 0 and positive values to the right and an 80-kb walk (Preiss, Hartley and Artavanis-Tsakonas, 1988, EMBO J. 7: 3917-27) with a *Xho* site just to the right of the initiating clone designated as the 0 coordinate and positive values to the left. The *Xho* site appears to be approximately 2.5 kb to the left of the *EcoRI* site; accordingly changing the sign and subtracting 2.5 kb from the Preiss et al. coordinates approximates the Knust et al. coordinates which are used here. Lesions of twelve different mutations identified within 36 kb from -15 to +21 kb of the walk. At least 11 transcripts detected in this region; they are numbered m1 through m11 in order from proximal to distal; coordinate 0 is between transcription units m4 and m5. All of these transcription units are expressed at some level during embryogenesis and several are expressed maternally as well. *In situ* hybridization of cDNA or genomic probes for nine of the eleven transcribed regions to developing embryos reveal complex temporal and spatial patterns of expression; four of the transcripts m4, m5, m7 and m8 exhibit virtually identical expression patterns, which accord with expectation for neurogenic genes; transcript first appears in late blastoderm, most intensely the neurogenic ectoderm, a ventrolateral stripe several cells wide on either side of the embryo. They are also expressed in several other neural primordia such as the stomatogastric nervous system and the optic lobes; transcripts subsequently become restricted to epidermal primordia; m9 and m10 are strongly and ubiquitously expressed in the embryo as well as maternally. Among the mutants with lesions within the 36-kb region are *gro* which appears to be an allele of *E(spl)*, and *Pr* alleles which do not. Sequence determined for m4, m5, m7 and m8 (Klämbt, Knust, Tietze and Campos-Ortega, 1989, EMBO J. 8: 203-10); m5, m7

and m8 are highly homologous to one another and share significant homology with proteins of the helix-loop-helix variety such as those encoded by *da*, *emc*, T3, T4, T5 and T8 of *ASC* and vertebrate *myc*; m4 is completely different from the other three and from known sequences in databases. The genomic sequences of all four lack introns and all encode hydrophilic polypeptides; specific information on each is as follows: m4 is a 1.1-kd mRNA that encodes a 152 (or 141 depending on start codon) amino-acid, 19-kd hydrophilic polypeptide with pI = 5.5; m5 is 1.0 kb and encodes a 19.9-kd polypeptide of 178 amino-acids, pI = 10.16; m7 is an 1.5 kb mRNA encoding a 186 amino-acid, 20.5-kd polypeptide, pI = 10.2 and m8, which is 1.0 kd in length, encodes a polypeptide of 179 amino acids and molecular weight of 19.7 kd, pI approximately equal to 10. The genomic sequence of m8 is modified in *E(spl)*; there is an 84-nucleotide deletion upstream from the putative m8 promoter plus a 483-nucleotide deletion that removes the last 56 codons from the message; the next 27 base pairs are in frame and append nine ectopic amino-acids to the truncated polypeptide; these are followed by a 60-base-pair insertion the first three nucleotides of which are a stop codon; then there is a ten-nucleotide deletion followed by wild-type sequence. Klämbt et al. have shown that the abnormal m8 region is responsible for the *E(spl)* phenotype; transformation with a genomic segment containing m8 and little else leads to genotypes with strong split suppressing effects. Transformation with a segment including the transcription unit for m9 and m10 is able to rescue lethality of seven presumed lethal point mutations of *E(spl)*, of *E(spl)Df(3R)Espl* and the phenotype of *Df(3R)Esplgro*; it does not, however, fully rescue the neural hyperplasia associated with some molecular deficiencies (Preiss et al., 1989). The genomic sequence corresponding to the transcription unit has been sequenced; the two transcripts share a common uninterrupted open reading frame, differing only in the length of their 3' untranslated region. The conceptual amino-acid sequence contains 719 residues with predicted molecular weight of 78,928 daltons; there is no evidence of either a signal sequence or transmembrane regions; three clusters of residues rich in proline seen in the amino-terminal half of the molecule in residues 3-19, 120-208 and 273-421, the carboxy-terminal half contains four or five repeating motifs of 43 to 47 residues. In database searches, the 340 carboxy-terminal amino-acids display 39% homology and 21% identity to β transducin, the G protein involved in signal transduction in the retina; homology is also detected to several other β -G proteins and to the yeast cell-cycle gene, *CDC4*.

*E(spl)*²

synonym: *gro*: *groucho*.

phenotype: Homozygotes have clumps of extra bristles above each eye which give impression of bushy eyebrows; also extra bristles on the humerus. Top of head tends to be malformed; ocelli often enlarged and run together. In selected stocks, penetrance approaches 100%, but is low in unselected stocks. Concluded to be an allele of *E(spl)* on the basis of the visible phenotype of heterozygotes with lethal presumed point mutations at *E(spl)*; however, it does not cause neural hypertrophy and is wild type in combination with *E(spl)*¹.

molecular biology: The chromosome containing the *gro* mutation contains two inserts, one of ~4 kb between coordinates -4.8 and -3.8 kb and a much smaller one at approximately +11.5 detected by Preiss *et al.*. The larger of the two is independent of the *gro* mutation since *Df(3R)Pr4*, which deletes the region into which the segment is inserted, does not uncover *gro*; a similar argument for the independence of the insert at 11.5 can be made since *gro* is rescued by transformation with a segment from 12.7 to 23.1 (Preiss *et al.*).

e(tu-K): enhancer of tumor K

location: 3- (not located).

origin: Spontaneous.

discoverer: Burnet and Sang.

references: 1964, Genetics 49: 223-35.

phenotype: Homozygote produces a significant increase in the penetrance of *tu-K* in both untreated flies and those treated in ways known to increase tumor incidence in *tu-K*. RK3.

E(tuh-1): Enhancer of tumorous head-1

location: 3- (not mapped; near *BXC*).

references: Kuhn and Dorgan, 1974, Genetics 77: s37.

phenotype: Increases penetrance by 8% and expressivity by 91% for the tumorous-head trait. Acts in *trans* to *iab8,9^{tuh3}* to increase maternal effect of *tuh1* on transformation of head to abdominal structures (Kuhn, and Packert, 1988, Dev. Biol. 125: 8-18).

E(var)7: Enhancer of variegation

location: 2- (not located).

origin: X ray induced.

discoverer: Schultz.

phenotype: *E(var)7*⁺ has no phenotype of its own but enhances variegation, e.g., *w^{m4}* is made much lighter and variegation for *rst* appears in males. Variegated position effects do not respond uniformly to *E(var)7*. RK2(A).

cytology: May be small abnormality in 25A (Schultz).

E(var)21

discoverer: Schultz.

references: Hadorn, Gsell, and Schultz, 1970, Proc. Nat. Acad. Sci. USA 65: 633-37.

phenotype: Produces large patches of yellow bristles in combination with *E(var)7* in *y/y⁺Y* males.

E(var)25F to 100C-F

A series of enhancers of variegation inferred from the effects of segmental deficiencies and duplications on the phenotype of *In(1)w^{m4}*. All but *E(var)33A-D*, which is a triplo enhancer, are haplo enhancers. Three other triplo enhancers have been associated with haplo suppressors of variegation: *Su(var)2-5 = Su(var)205*, *Su(var)7* and *Su(var)9*.

locus	genetic location	cytological location	included in	excluded from	ref ^α	comments
<i>E(var)25F</i>	2-[17]	25F2-4	<i>Df(2L)cl2</i>	<i>Df(2L)cl-h1</i>	2, 3, 5	
<i>E(var)26A</i>	2-21	25F4-26A1	<i>Df(2L)Gpdh78</i>		2, 3, 5	
<i>E(var)28A-B</i>	2-[22]	28E5-28B1	<i>Df(2L)TE28A-1</i>	<i>Df(2L)Gpdh75w</i>	5	
<i>E(var)29B-D</i>	2-[30]	29A2-D1	<i>Df(2L)TE29Aa36</i>		5	
<i>E(var)33A-D</i>	2-[45]	33A1-D5			5	triplo enhancer; adjacent to <i>Su(var)</i>
<i>E(var)36A-E</i>	2-[52]	36A8-E1	<i>Df(2L)H20</i>	<i>Df(2L)M36F-S5</i>	5	
<i>E(var)43A</i>	2-[56]	43A2-B1	<i>Df(2R)43A</i>		5	

locus	genetic location	cytological location	included in	excluded from	ref ^α	comments
<i>E(var)55</i>	2-[86]	55A-F	<i>Df(2R)PC4</i>		5	
<i>E(var)87F</i>	3-[54]	87E11-F14	<i>Df(3R)GE41</i>	<i>Df(3R)IC4a</i> <i>Df(3L)red41</i>	5	= <i>e(var)3-3</i>

E(var)100C-F 3-103.6 100C5-F5 *Df(3R)E45*

1, 4

^α 1 = Locke, Kotarski, and Tartof, 1988, Genetics 120: 818-98; 2 = Szidonya and Reuter, 1988, DIS 67: 77-79; 3 = Szidonya and Reuter, 1988, Genet. Res. 51: 197-208; 4 = Tartof, Bishop, Jones, Hobbs, and Locke, 1989, Dev. Genet. 10: 162-76; 5 = Wustmann, Szidonya, Taubert, and Reuter, 1989, Mol. Gen. Genet. 217: 520-27.

E(var)300 series

Three ethyl-methanesulfonate-induced dominant enhancers of variegation (Sinclair, Mottus, and Grigliatti, 1983, Mol. Gen. Genet. 191: 326-33). Cause substantial reduction in eye pigment of *In(1)w^{m4}*-bearing flies; less to no effect on *w^{m57b}*, *w^{mj}*, and *w^{mMc}*; all three enhance *bw^{VDe2}* and *Sb^V* (Sinclair, Lloyd, and Grigliatti, 1989, Mol. Gen. Genet. 216: 328-33).

locus	location
<i>E(var)301</i>	3-49.3
<i>E(var)302</i>	3-36.6
<i>E(var)303</i>	3-34.7

E(var)c101

location: 3-54 [based on 3.7% recombination with 89C2-3 breakpoint of *T(2;3)ap^{Xa}* (Reuter, Werner and Hoffmann, 1982, Chromosoma 85: 539-51)].

origin: Spontaneous in *T(2;3)ap^{Xa}*.

references: Reuter and Wolff, 1981, Mol. Gen. Genet. 182: 516-19.

phenotype: Produces white eyes with occasional single red facets in combination with *w^{m4h}*.

E(w^a): Enhancer of white-apricot

location: 2-105.1 (4.6 units to the right of *px*; to the left of *sp*).

origin: Spontaneous.

discoverer: Scandlyn.

references: Von Halle, 1969, DIS 44: 119.

phenotype: Heterozygote dilutes *w^a* to pale yellow. Homozygote with *w^a* is white. Enhances *w^{a1}* and *w^{a4}*. Also enhances *w^{Sp55}* and *w^h* (Birchler, 1986, Genetics 113: s47) but not *w^{a2}*, *w^{a3}*, *w^{bf}*, *w^{Bwx}*, *w^{cf}*, *w^{ch}*, *w^{co}*, *w^{col}*, *w^e*, or *w^{sp}*; enhances phenotype of *w^{a1}/w^{bf}*. No effect on eye color in presence of *w⁺*; and no interaction with other eye-color mutants. Enhancement dominant to two doses of the normal allele in *w^a/bw⁺Y;E(w^a)/+*. Homozygous males sterile; homozygous females partially fertile. RK3.

cytology: Placed to the left of 60B10 based on the *E(w^a)/+*-like phenotype of *E(w^a)/Df(2L)Px* [= *Df(2R)60B8-10:60D1-2*].

***e(w^e): enhancer of white-eosin**

location: 1-32.

origin: Spontaneous.

discoverer: Green, 55b21.

synonym: *en-w^e*.

references: 1957, DIS 31: 81.

1959, Heredity 13: 303-15.

phenotype: Enhances certain intermediate alleles of *w*;

e.g., w^{aE} , w^e , w^{e2} , w^h , and w^{X16} ; three of these shown to have transposable element *DOC* inserted at the same place; two have *pogo* (w^e and w^{e2}) and one has *roo* (w^h) inserted in the *DOC* element. Produces a nearly white eye color. No effect on w^a , w^{a2} , w^{a3} , w^{a4} , w^{bf} , w^{ch2} , w^{co} , w^{col} , or w^{sat} . Suppresses *lz^K* and *gypsy* containing mutants *lz¹*, *f¹*, *f⁵*, and *bx^{34e}* (Rutledge, Mortin, Schwarz, Thierry-Mieg, and Meselson, 1988, Genetics 119: 391-97). $e(w^e)$ flies occasionally have *px*-like venation or shortened wings, or both. Homozygous females sterile. RK2.

alleles: A second spontaneous allele with similar phenotype and map position recovered by Schalet.

$e(w^{8d})$: see *su(f)*

***e(y)*: enhancer of yellow**

A series of sex-linked recessive mutations that modify the expression of y^2 , a *gypsy*-induced allele; these mutations do not modify the expression of other *gypsy*-induced mutations. Recovered from hybrid dysgenic crosses in which the transposable element, *Stalker* plus, in some cases, *P* were mobilized. The new mutations fall into six complementation groups; they were localized by recombinational analysis and by *in situ* hybridization using the transposable sequences as probes; (Georgiev and Gerasimova, 1989, Mol. Gen. Genet. 220: 121-26). y^2 suppressed by *su(Hw)*²; however, the effects of all *e(y)* mutations on bristle color persist in the presence of *su(Hw)*².

locus	genetic location	cytological location	comments ^α
<i>e(y)1</i>	1-57.9	16B	head and thorax bristles yellow; microchaetae yellow; female sterile
<i>e(y)2</i>	1-36.2	10C	head and thorax bristles yellow; microchaetae yellow; short stocky body; wings separated; eyes small; facets altered; bristles shortened
<i>e(y)3</i>	1-62.2	18C-D	head and thorax bristles yellow; microchaetae yellow; thin bristles; female sterile; reduced viability
<i>e(y)4</i>	1-58.1	16B	thorax macro- and microchaetae yellow; causes yellow bristles on y^+ flies; males but not females have short thin bristles
<i>e(y)5</i>	1-59.2	17C	y^+ flies have shortened, thin, yellow, transparent macro- and microchaetae on thorax and abdomen; female sterile
<i>e(y)6</i>	1-37.2	10F	y^+ flies have pale yellow body, wings, and bristles; male sterile; reduced viability

^α Effect on phenotype of y^2 unless otherwise indicated; no effect on phenotype of y^+ unless so indicated.

alleles: Independent recovery of mutations at several of the loci recorded; those with *P*-element inserts generally unstable, reverting to wild type or in some cases generating novel alleles; those with *Stalker* inserts stable.

allele	origin
<i>e(y)1¹</i>	<i>Stalker</i>
<i>e(y)1²</i>	<i>Stalker</i>
<i>e(y)2¹</i>	<i>Stalker</i>
<i>e(y)3¹</i>	<i>P</i>
<i>e(y)3²</i>	<i>P</i>
<i>e(y)3³</i>	<i>P</i>
<i>e(y)3⁴</i>	<i>Stalker</i>

allele	origin
<i>e(y)4¹</i>	<i>P</i>
<i>e(y)5¹</i>	<i>P</i>
<i>e(y)5²</i>	<i>P</i>
<i>e(y)5³</i>	<i>P</i>
<i>e(y)6¹</i>	<i>P</i>
<i>e(y)6²</i>	<i>P</i>
<i>e(y)6³</i>	<i>P</i>

***E(z)*: Enhancer of zeste**

location: 3-34.0.

synonym: *l(3)1902*; *pco*; *polycombeotic*.

references: Kalisch and Rasmuson, 1974, Hereditas 78: 97-104.

Shearn, Hersperger, and Hersperger, 1978, Genetics 89: 341-53.

Shearn, Hersperger, Hersperger, Pentz, and Denker, 1978, Genetics 89: 355-70.

Wu, Jones, Lasko, and Gelbart, 1989, Mol. Gen. Genet. 218: 559-64.

Phillips and Shearn, 1990, Genetics 125: 91-101.

Jones and Gelbart, 1990, in press.

phenotype: Locus named after original gain-of-function allele $E(z)^1$ (Kalisch and Rasmuson); subsequently designated *polycombeotic* (*pco*) (by Phillips and Shearn) based on phenotype of lethal homozygotes. Loss of function alleles recovered as (a) recessive lethal mutations (b) reversions of $E(z)^1$ and (c) reversions of the antimorphic allele, $E(z)^{59}$. Reduction of $E(z)^+$ activity leads to suppression of the *z* eye color, whereas gain-of-function alleles are dominant enhancers of *zeste* eye color [i.e., $z w^+/Y$; $E(z)^1/+$ males have brownish eyes as do $z w^+/z^+ w^+$; $E(z)^1/+$ females]. $E(z)^{59}$ an antimorphic allele, is a dominant suppressor of *z* [i.e. $z w^+$; $E(z)^{59}/+$ females have orange eyes]. Hemizygoty for $E(z)^+$ produces a very mild suppression of the *z* eye color. No effects on eye color in z^+ or z^a backgrounds, and effects on eye color not specific to a particular *w* allele. Reduction of $E(z)^+$ activity also allows ectopic expression of the segment identity genes of the Antennapedia and bithorax gene complexes, resulting in homeotic transformations. This latter effect defines $E(z)$ as a Polycomb-group locus. $E(z)^{61}$ displays temperature-sensitive suppression of *z* eye color and homeotic phenotypes. At 22°, $z w^{ts}/Y$; $E(z)^{61}/Df(3L)E(z)^-$ males have orange eyes and no homeotic transformations. At 29°, such males have wild-type red eyes and die as pharate adults with strong homeotic transformations of the mesothoracic and metathoracic legs toward the prothoracic state. Embryos produced by $E(z)^{61}$ homozygous females at 29°C die with homeotic transformations of most segments toward the eighth abdominal segment. Even two copies of paternally contributed $E(z)^+$ does not rescue viability of these embryos. Complete lack of zygotically produced $E(z)^+$ results in early pupal lethality and small imaginal disks. Larval brain squashes from individuals homozygous for an amorphic allele reveal a very low mitotic index; metaphase chromosomes irregularly condensed and fragmented (Gatti and Baker, 1989, Genes Dev. 3: 438-53).

alleles: With the exception of the first four alleles, which are gain-of-function dominant suppressors of *zeste*, and $E(z)^{60}$, which is an antimorphic dominant suppressor of

zeste, all alleles listed below are presumably hypomorphic and amorphic alleles, which can act as dominant suppressors of zeste and are homozygous lethal.

allele	origin	synonym	ref ^α	comments
<i>E(z) 1</i>	EMS		2, 5	gain-of-function allele
<i>E(z) 2</i>	EMS		2	gain-of-function allele
<i>E(z) 3</i>	EMS		2	gain-of-function allele
<i>E(z) 4</i>	EMS		2	gain-of-function allele
<i>E(z) 5</i>	NNG	<i>l(3)1902</i>	3, 4	
<i>E(z) 6</i>	EMS	<i>l(3)GST311</i>	3	
<i>E(z) 7</i>	EMS	<i>l(3)MK436</i>	3	
<i>E(z) 8</i>	EMS	<i>l(3)MM130</i>	3	
<i>E(z) 9</i>	EMS	<i>l(3)MM634</i>	3	
<i>E(z) 10</i>	EMS	<i>l(3)MM701</i>	3	
<i>E(z) 11</i> β	EMS	<i>l(3)MR127</i>	3, 4	unconditional female sterile
<i>E(z) 12</i>	EMS	<i>l(3)MY939</i>	3, 4	
<i>E(z) 13</i>	EMS	<i>l(3)MZ1007</i>	3, 4	
<i>E(z) 14</i>	EMS	<i>l(3)NH006</i>	3, 4	
<i>E(z) 15</i>	EMS	<i>l(3)NU808</i>	3, 4	
<i>E(z) 16</i>	EMS	<i>l(3)NV931</i>	3, 4	
<i>E(z) 17</i>	EMS	<i>l(3)NW522</i>	3, 4	
<i>E(z) 18</i>	EMS	<i>l(3)NY721</i>	3	
<i>E(z) 19</i>	EMS	<i>l(3)OA001</i>	3	
<i>E(z) 20</i>	EMS	<i>l(3)OD522</i>	3	
<i>E(z) 21</i>	EMS	<i>l(3)OP813</i>	3	
<i>E(z) 22</i>	EMS	<i>l(3)OQ626</i>	3	
<i>E(z) 23</i>	EMS	<i>l(3)OR030</i>	3	
<i>E(z) 24</i>	EMS	<i>l(3)OS628</i>	3	
<i>E(z) 25</i>	EMS	<i>l(3)OT502</i>	3	
<i>E(z) 26</i>	EMS	<i>l(3)OU511</i>	3	
<i>E(z) 27</i>	EMS	<i>l(3)OW105</i>	3	
<i>E(z) 28</i> β	EMS	<i>l(3)OX736</i>	3	
<i>E(z) 29</i>	EMS	<i>l(3)OZ440</i>	3	
<i>E(z) 30</i>	EMS	<i>l(3)OZ1340</i>	3	
<i>E(z) 31</i>	EMS	<i>l(3)PB901</i>	3	
<i>E(z) 32</i>	EMS	<i>l(3)PC025</i>	3	
<i>E(z) 33</i>	EMS	<i>l(3)PI1517</i>	3	
<i>E(z) 34</i>	EMS	<i>l(3)PK911</i>	3	
<i>E(z) 35</i>	EMS	<i>l(3)PQ1129</i>	3	
<i>E(z) 36</i>	EMS	<i>l(3)PU620</i>	3	
<i>E(z) 37</i>	EMS	<i>l(3)PW111</i>	3	
<i>E(z) 38</i>	EMS	<i>l(3)PX338</i>	3	
<i>E(z) 39</i>	EMS	<i>l(3)PX811</i>	3	
<i>E(z) 40</i>	EMS	<i>l(3)QH527</i>	3	
<i>E(z) 41</i>	EMS	<i>l(3)QN916</i>	3	
<i>E(z) 42</i>	EMS	<i>l(3)QO703</i>	3	
<i>E(z) 43</i>	EMS	<i>l(3)QO1407</i>	3	
<i>E(z) 44</i>	EMS	<i>l(3)QW206</i>	3	
<i>E(z) 45</i>	EMS	<i>l(3)RC318</i>	3	
<i>E(z) 46</i>	EMS	<i>l(3)RF306</i>	3	
<i>E(z) 47</i>	EMS	<i>l(3)SD121</i>	3	
<i>E(z) 48</i>	EMS	<i>l(3)SO230</i>	3	
<i>E(z) 49</i>	EMS	<i>l(3)UO573</i>	3	
<i>E(z) 50</i>	EMS	<i>l(3)US53</i>	3	
<i>E(z) 51</i>	EMS	<i>l(3)VL151</i>	3	
<i>E(z) 52</i>	EMS	<i>l(3)VM7</i>	3	
<i>E(z) 53</i>	EMS	<i>l(3)VS528</i>	3	
<i>E(z) 54</i>	EMS	<i>l(3)VT244</i>	3	
<i>E(z) 55</i>	EMS	<i>l(3)VT329</i>	3	
<i>E(z) 56</i>	EMS	<i>l(3)VY141</i>	3	
<i>E(z) 57</i>	EMS	<i>l(3)VZ416</i>	3	
<i>E(z) 58</i>	EMS	<i>l(3)WA86</i>	3	
<i>E(z) 59</i>	EMS	<i>l(3)WB68</i>	3	
<i>E(z) 60</i>	EMS	<i>E(z) S1</i> <i>Su(z) 301</i>	5	antimorphic allele
<i>E(z) 61</i>	EMS	<i>E(z) S2</i>	1, 5	cytology normal
<i>E(z) 62</i>	EMS	<i>E(z) S3</i>	1	cytology normal
<i>E(z) 63</i>	EMS	<i>E(z) S4</i>	1	cytology normal
<i>E(z) 64</i>	EMS	<i>E(z) S5</i>	1	cytology normal
<i>E(z) 65</i>	γ ray of <i>E(z) 1</i>	<i>E(z) IR1</i>	1	<i>T(2;3)21C1-2;67E3-4</i>
<i>E(z) 66</i>	γ ray of <i>E(z) 1</i>	<i>E(z) IR4</i>	1	cytology normal
<i>E(z) 67</i>	γ ray of <i>E(z) 1</i>	<i>E(z) IR5</i>	1	cytology normal
<i>E(z) 68</i>	γ ray of <i>E(z) 1</i>	<i>E(z) IR6</i>	1	<i>ln(3L)64E-F;75C-76B</i>
<i>E(z) 69</i>	γ ray of <i>E(z) 1</i>	<i>E(z) IR7</i>	1	cytology normal
<i>E(z) 70</i>	γ ray of <i>E(z) 1</i>	<i>E(z) IR8</i>	1	cytology normal
<i>E(z) 71</i>	γ ray of <i>E(z) 1</i>	<i>E(z) IR10</i>	1	cytology normal

allele	origin	synonym	ref ^α	comments
<i>E(z) 72</i>	γ ray of <i>E(z) 1</i>	<i>E(z) IR11</i>	1	cytology normal
<i>E(z) 73</i>	γ ray of <i>E(z) 1</i>	<i>E(z) IR14</i>	1	cytology normal
<i>E(z) 74</i>	γ ray of <i>E(z) 60</i>	<i>E(z) SIR2</i>	1	cytology normal
<i>E(z) 75</i>	γ ray of <i>E(z) 60</i>	<i>E(z) SIR3</i>	1	cytology normal

^α 1 = Jones and Gelbart, 1990, in press. 2 = Kalisch and Rasmuson, 1974, *Hereditas* 78: 97-103. 3 = Shearn, Hersperger, and Hersperger, 1978, *Genetics* 89: 341-53. 4 = Shearn, Hersperger, Hersperger, Pentz, and Denker, 1978, *Genetics* 89: 355-70. 5 = Wu, Jones, Lasko, and Gelbart, 1989, *Mol. Gen. Genet.* 218: 559-64.

^β Heat sensitive alleles.

cytology: Placed in 67E3-4 based on its inclusion in the region of overlap of *Df(3L)Ez6 = Df(3L)67E1-2;67E3-5* and *Df(3L)Ez3 = Df(3L)67E3-4;67E6-7*, as well as on the breakpoint to *T(2;3)E(z) 65 = T(2;3)21C1-2;67E3-4*.

molecular biology: Region cloned by Jones. P-element mediated germline transformation of 9 kb fragment of DNA rescues all mutant phenotypes. Single 2.5-kb transcript expressed throughout development but most abundant in maternal mRNA. Approximately 1 kb proximal and in opposite transcriptional orientation to *hay* transcription unit.

ea: easter

location: 3-57 (between *jvl* and *sbd*).

references: Anderson and Nüsslein-Volhard, 1984, *Nature* 311: 223-27 (fig.).

1986, *Symp. Soc. Dev. Biol.* 44: 177-94.

Gametogenesis and the Early Embryo (J. Gall, ed.). Alan R. Liss, New York, pp. 177-94.

Chasan and Anderson, 1989, *Cell* 56: 391-400.

phenotype: Maternal effect lethal; both recessive loss-of-function and dominant gain-of-function alleles female sterile. Embryos produced by females homozygous for recessive alleles are dorsalized; lack ventral and lateral elements similar to embryos produced by *dll/dl* females; dorsal folds extend around circumference of embryo; lateral head fold and ventral furrow fail to form; germ band does not extend, and cuticle forms a tube with dorsal characteristics throughout. Degree of dorsalization proportional to level of *ea* expression as demonstrated by hypomorphic and temperature-sensitive alleles. Temperature sensitive period as demonstrated by *ea*¹⁴ from the time of pole-cell formation to gastrulation; developmental Northern blots demonstrate the presence of transcript in the ovaries and increased levels during the first four hours of embryonic development followed by a marked decline between hours four and six. Partial rescue of mutant phenotype achieved by injection of cytoplasm or poly-A+ RNA from wild-type embryos or embryos from females homozygous for other dorsal-group mutants; spatial source of donor cytoplasm unimportant; degree of rescue reduced with distance from site of injection; complete rescue producing viable and fertile adults achievable with injection of purified *ea*⁺ mRNA. *rw*, a zygotically active member of the dorsal group of genes, is not expressed in such embryos (Thisse, Stoetzel, El Messal, and Perrin-Schmidt, 1987, *Genes Dev.* 1: 709-15); furthermore strong alleles allow *zen* expression ventrally where it is not normally observed (Rushlow, Frasch, Doyle, and Levine, 1987, *Nature* 330: 583-86); periodicity of *ftz* stripes slightly disrupted in embryos from *ea* mothers (Carroll, Winslow, Twombly, and Scott, 1987, *Development* 99: 327-32). Females

heterozygous for fully penetrant dominant alleles are sterile; the offspring of *ea*^{D3}/+ females display ventralization or lateralization as indicated by lateral extension of the denticle bands; patches or rings of denticles may encircle the embryo; however, the mesoderm, the most ventral pattern element, is not increased; dorsal structures such as filzkörper, antennal and maxillary sense organs are absent; field of dorsal hairs greatly reduced. *ea*^{D2}/+ females are lateralized, producing embryos that lack presumptive mesoderm and have no ventral furrow, show dorsoventrally symmetrical folding at gastrulation with the lateral head fold encircling the embryo, and do not undergo germ band extension. Injection of *ea*-deficient embryos with 200 times the quantity of mRNA required for complete rescue does not cause ventralization, indicating that gain of function alleles are not merely hypomorphic in nature.

alleles: The only dorsal-group gene, other than *Tl*, that mutates at substantial frequency to dominant alleles; dominant alleles designated *ea*^D.

allele	origin	synonym	ref ^α	comments
ea 1	EMS		3	strong allele
ea 2	EMS		3	
ea 3	EMS		3	
ea 4	EMS		3	
ea 5	EMS		3	
ea 6	EMS		3	
ea 7	EMS		3	
ea 8	EMS		3	
ea 9	EMS		3	
ea 10	EMS		3	
ea 11	EMS		3	
ea 12	EMS	<i>ea</i> ¹¹¹	3	weak allele
ea 13	EMS	<i>ea</i> ¹²⁵	3	
ea 14	EMS	<i>ea</i> ⁸¹⁸	3	temperature sensitive allele
ea 15	X ray	<i>ea</i> ^{D288RX1}	3	<i>ea</i> ^{D2} revertant
ea 16	P	<i>ea</i> ^{83IRPA}	1	<i>ea</i> ^{D3} revertant
ea 17	P	<i>ea</i> ^{83IRP1}	1	<i>ea</i> ^{D3} revertant
ea 18	P	<i>ea</i> ^{83IRPN}	1	<i>ea</i> ^{D3} revertant
ea 19	P	<i>ea</i> ^{83IRPX}	1	<i>ea</i> ^{D3} revertant
ea D1	EMS	<i>ea</i> ^{5.13}	1, 3	→ 9 EMS revertants → 2 X ray revertants
ea D2	EMS	<i>ea</i> ^{D288}	3	
ea D3	EMS	<i>ea</i> ⁸³¹	1, 3	→ 1 X ray revertant → 4 P revertants
ea D4	EMS	<i>ea</i> ^{84b}	1, 3	incomplete penetrance
ea D5	EMS	<i>ea</i> ^{84e}	1	incomplete penetrance
ea D6	EMS	<i>ea</i> ^{125.3}	1	→ 11 EMS revertants
ea D7	EMS	<i>ea</i> ^{D12a}	2	
ea D8	EMS	<i>ea</i> ^{D20n}	2	
ea D9	EMS	<i>ea</i> ^{D4102}	2	
ea D10	EMS	<i>ea</i> ^{D5022}	2	

^α 1 = Chasan and Anderson, 1989, Cell 56: 391-400; 2 = Erdélyi and Szabad, 1989, Genetics 122: 111-27; 3 = Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

cytology: Placed in 88F2 by *in situ* hybridization (Chasan and Anderson).

molecular biology: Locus identified in a chromosome walk by transposon tagging; restriction fragment harboring sites of P insertion recognizes mRNA of 1.5 kb. Shown to be the *ea* message by rescuing embryos of *ea* mothers by injection of hybrid selected message. cDNA selected from 0-3 hr embryo library by above restriction fragment shown to be full length by rescue of embryos with *in vitro* transcription product. Sequence of cDNA indicates a polypeptide of 392 amino-acids, 43,070 daltons, and containing a putative signal sequence. The N-terminal 127 residues show no homology with any

known protein; the remaining residues, however, share homology with many extracellular serine proteases of the trypsin superfamily; in conserved segments of the catalytic domain there is 50% amino-acid identity, and over the entire catalytic domain, 30% identity.

eag: ether-a-go-go (J.C. Hall)

location: 1-50.0 (1-45.3 according to Homyk and Grigliatti, 1983, Dev. Genet. 4: 77-97).

origin: All alleles induced by ethyl methanesulfonate.

references: Kaplan, 1969, DIS 44: 45.

Kaplan and Trout, 1969, Genetics 61: 399-409.

Ganetzky and Wu, 1983, J. Neurogenet. 1: 17-28.

phenotype: Abnormal leg shaking under ether anaesthesia; aberrant, repetitive firing of action potentials in larval nerves; potassium currents abnormal in larval muscles (Wu, Ganetzky, Haugland and Lin, 1983, Science 220: 1076-1078). *eag Sh* double mutants display greatly increased level of spontaneous neuronal activity and extreme behavioral phenotypes (Burg and Wu, 1989, Dev. Biol. 131: 505-14).

alleles: *eag*¹; *eag*^{4PM}, weak behavioral abnormalities but is abnormal physiologically; *eag*¹⁰¹, hyperactive, flies and jumps well when raised at 22°, flies poorly or not at all when raised at 29°. (Homyk, Szidonya, and Suzuki, 1980, Mol. Gen. Genet. 177: 553-65); TSP during pupal stage (Homyk and Grigliatti, 1983, Dev. Genet. 4: 77-97). *eag*¹⁰², also causes accentuated jumping ability (Homyk *et al.*).

cytology: Located in 12F6-13A4 based on uncovering by *Df(1)RK2 = Df(1)12D2-E1;13A2-5*, by *Df(1)RK3 = Df(1)12E2-6;13A6-11*, by *Df(1)RK4 = Df(1)12F5-6;13A9-B1*; and by *Df(1)RK5 = Df(1)12E10-11;13A8-B1*; not uncovered by *Df(1)KA9 = Df(1)12E2-3;12F5-13A1* (Kreber and Ganetzky).

molecular biology: Region very close to, and likely to include, the locus cloned by Drysdale and Ganetzky (1985, Neurosci. Abstr. 11: 788).

other information: *eag* mutations interact synergistically with Shaker mutations, *i.e.*, double mutants are severely abnormal both behaviorally and physiologically (Ganetzky and Wu, 1983).

eagle: see *eg*

early: see *eay*

early: see *lds*

early C: see *elyC*

early-A: see *aur*

eas: easily shocked (J.C. Hall)

location: 1-53.5.

origin: Induced by ethyl methanesulfonate.

synonym: *PC80, RH11*.

references: Benzer, 1971, J. Amer. Med. Assoc. 218: 1015-22.

Ganetzky and Wu, 1982, Genetics 100: 597-614.

phenotype: Brief paralysis following exposure to mechanical shock; this phenotype suppressed by *nap*^{ts} mutation at its permissive temperature; release of neurotransmitter at larval neuromuscular junction is apparently normal.

alleles: Two mutant alleles: *eas*¹ and *eas*².

cytology: Placed in 14B5-18 based on its inclusion in *Df(1)81h246 = Df(1)14B15-18;14E* but not in

Df(1)82a2y = Df(1)14B5-18;15A3-4 (Steller).

other information: Separable by recombination from *bss* (which causes similar phenotype), to which *eas* is closely linked.

easter: see *ea*

eay: *early* (*T. Schüpbach*)

location: 2- {86}.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1989, *Genetics* 121: 101-17.

phenotype: Maternal-effect lethal or female-sterile mutant; homozygous females lay eggs which show no visible signs of development when observed under transmitted light in a stereo microscope; defective in fertilization or very early embryonic development.

alleles: Seven alleles *eay*¹ through *eay*⁷, originally designated *OP*, *AWG*, *AWH*, *HL*, *PI*, *PQ*, and *SJ* respectively.

cytology: Placed in 55A-F since uncovered by *Df(2R)PC4 = Df(2R)55A;55F*.

eb: see *s^{eb}*

eb: *extra bristles*

location: 2-32.

origin: Spontaneous.

references: Mostashfi and Koliantz, 1969, *DIS* 44: 51.

phenotype: Increased bristle number on mesonotum and scutellum; average of two extra scutellar and two extra mesonotal bristles.

Eb: see *E(B)*

ebonized: see *s^{eb}*

ebo: *ellipsoid body open* (*J.C. Hall*)

location: 1-0.6.

origin: Induced by ethyl methanesulfonate.

references: Heisenberg, Borst, Wagner, and Byers, 1984, *J. Neurogenet.* 2: 1-30.

phenotype: Ellipsoid body of central brain abnormally flat and broad; learning normal in tests using olfactory stimuli.

ebony: see *e*

ec: *echinus*

location: 1-5.5.

origin: Spontaneous.

discoverer: Bridges, 1916.

phenotype: Eyes large and bulging. Eye surface rough; facets large. Wings rather short and broad. Body thick-set. Tends to remove dorsocentrals (posterior more than anterior) and posterior notopleurals; may also add dorsocentrals anterior to anterior dorsocentrals whether or not posterior bristles removed (Sturtevant). Reduces number of mesopleural hairs in *h* homozygotes (Sturtevant, 1969, *Dev. Biol.* 21: 48-61). *ec* is visible in *+ec/ec* triploids (Gersh). RK1.

cytology: 3E8 or F1 [Lefevre, 1976, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, p. 59].

ecd: *ecdysone*

location: 3-1.5.

references: Garen, Kauvar, and Lepesant, 1977, *Proc. Nat. Acad. Sci. USA* 74: 5099-103.

Redfern and Bownes, 1983, *Mol. Gen. Genet.* 189: 432-40.

phenotype: Temperature-sensitive recessive lethal. At 29°, the nine-fold increase in β-ecdysone content during embryogenesis occurs normally in *ecd* homozygotes. Accompanied by normal increases in dopa-decarboxylase and dopamine acetyltransferase activity (Marsh and Wright, 1980, *Dev. Biol.* 80: 379-87). The four-fold increase during the first larval instar is reduced to 40% of normal; the additional twelve-fold increase normally occurring at pupariation eliminated. Embryonic development of *ecd* at 29° normal, but first larval molt delayed and death usually occurs by end of second instar; shift down to 20° at mid second instar produces full yield of adult progeny. Larvae shifted from 20° to 29° midway through third instar fail to pupate and survive as larvae for up to 3 weeks; ring gland, salivary glands, and brain of nonpupariating larvae are smaller than wild type; such ring glands cultured *in vitro* secrete ecdysone at lower than normal levels. Effects of 29° on third instar reversible by ecdysone feeding or by shift down to 20° within 3-5 days of shift up; after that imaginal disks cannot differentiate; *ecd* imaginal disks develop normally at 29° when implanted in a wild-type host. A heat pulse during larval development results in cell death with consequent abnormalities in emerging adults. At 29°, third instar larvae exhibit abnormally low dopadecarboxylase activity (Kraminsky, Clark, Estelle, Gietz, Sage, O'Connor, and Hodgetts, 1980, *Proc. Nat. Acad. Sci. USA* 77: 4175-79); Marsh and Wright) and high urate oxidase activity (Krase and Friedman, 1979, *Genetics* 91: s62-63); ecdysone feeding effects normal levels. Transfer to restrictive conditions at the beginning of the pupal stage leads to death as pharate adults and to the elimination of mechanosensory chaetae; Non-sensory chaetae and other sensilla not affected. Chaetae loss is autonomous as seen in homozygous clones produced by somatic exchange and in *ecd* discs passed through metamorphosis in wild type hosts under restrictive conditions (Sliter, 1989, *Development*, 106: 347-54). Sensitivity to restrictive temperature disappears in 24 hour pupae. *ecd* fails to block midpupal increase in ecdysone titer (Marsh and Wright). Shifting newly emerged *ecd* adults to 29° results in drastically reduced ecdysone titers and sterilizes both males and females; reversible by shift back to 20°; temperature-sensitive periods and therefore the times that ecdysone is required for embryonic development, chorion formation, and vitellogenesis are 1-2 days before oviposition, 24 hr prior to oviposition, and prior to stage 7, respectively (Audit-Lamour and Bussin, 1981, *J. Insect Physiol.* 27: 829-37).

alleles: In addition to *ecd*¹ described above, *ecd*², a non-conditional, ethyl-methanesulfonate-induced allele recovered by Sliter, Henrich, Tucker, and Gilbert (1989, *Genetics* 123: 327-36).

cytology: Placed in 62C4-D5 based on its inclusion in *Df(3L)R-R2 = Df(3L)62B2-4;62D3-5* but not *Df(3L)R-G5 = Df(3L)62A10-B1;62C4-D1* (Sliter *et al.*).

Ecdysone-dependent gene: see *Edg*

Ecdysone-inducible: see **E74**

Ecdysteroid-inducible polypeptide encoded in 55BD: see **Eip55BD**

echinoid: see **ed**

echinus: see **ec**

ecl: **echinus like**

location: 1- {8} [to the left of *fl(1)302*].

origin: Induced by ethyl methanesulfonate.

references: Steinmann-Zwicky, 1988, EMBO J. 7: 3889-98.

phenotype: Like *ec*.

cytology: Placed in 4C5-E1 based on its inclusion in *Df(1)rb13 = Df(1)4C5-6;4D3-E1*. Not included in *Df(1)rb23* or *Df(1)rb34* both of which overlap the left end of of *Df(1)rb13* and include *rb* and *peb* as does *Df(1)rb13*.

Eco: Extra combs

location: 2-84.

discoverer: Duncan.

phenotype: Sex combs formed on second and third pairs of legs; middle leg transformed to first leg; fifth and sixth tergites transformed to sixth and seventh, respectively. Clonal analysis indicates late activity.

alleles: Hypomorphic and amorphic alleles and deletions have been identified.

ecs: see **BRC**

ed: echinoid

location: 2-11.0.

origin: Spontaneous.

discoverer: Bridges, 31a16.

phenotype: Eyes large and rough. Easily classified, although not as extreme as *ec*. *Su(S)/ed* is echinoid in phenotype (Gelbart). RK1.

cytology: Placed in 24D3-4 on the basis of its inclusion in the region of overlap of *Df(2L)ed-Szl = Df(2L)24A3-4;24D3-4* and *Df(2L)M24F-11 = Df(2L)24D3-4;25A2-3* (Szidonya and Reuter, 1988, DIS 67: 77-79; 1987, Genet. Res. 210: 429-36).

Edg: Ecdysone-dependent gene

Four genes identified in a differential screen of a genomic library with cDNA isolated from imaginal discs pulsed with 20-hydroxyecdysone, 20-HE (Fechtler, Natzle, Brown, and Fristrom, 1988, Genetics 120: 465-74). All of these genes accumulate transcripts in imaginal disc tissue, beginning at six to nine hours, following a six-hour 20-HE pulse, but not in the absence or continual presence (except *Edg64CD*) of 20-HE; mRNA's of all four enriched in membrane-bound polysome fractions of imaginal discs. Translation product from hybrid selected mRNA of *Edg78E*, but not the other three, reacts with polyclonal antibodies prepared against purified pupal cuticle proteins.

locus	genetic location	cytological location	mRNA on Northern
Edg42A	2-{55}	42A	3.0 kb
Edg64CD	3-{19}	64C-D	5.4, 5.0, 3.9, 3.9 kb
Edg78E	3-{47}	78E	0.6 kb
Edg84A	3-{47}	84A1	0.9 kb

locus	genetic location	cytological location	mRNA on Northern
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ee: extra eye (W.K. Baker and J.C. Hall)

location: 2-18.0 (between *dp* and *spd*).

origin: Spontaneous.

discoverer: Aaverhoff.

references: Marcey and Stark, 1985, Dev. Biol. 107: 180-97.

Baker, Marcey and McElwain, 1985, Genetics 111: 67-88.

phenotype: Flies from extra eye stocks show pattern duplications of head morphology in varying degrees; small duplications may appear as a few extra orbital bristles, whereas extreme expression involves well-formed, supernumerary, compound eyes on the vertex with associated orbital structures; ectopic eye is a mirror-image partial duplication of the ipsilateral eye. Occasionally, ocelli may be duplicated and rarely the antennae; a line of mirror-image symmetry often can be recognized between normal structures and their duplicated counterparts; structural deficiencies, particularly in the occipital region, are common. Penetrance is temperature sensitive (72% at 29° and 25°, 43% at 19°); temperature-sensitive periods in mid-embryogenesis and mid-first instar. There is no evidence of cell death in eye-antenna imaginal disc; first evidence of disc hyperplasia in late third instar. Fine-structure anatomy and physiology of photoreceptors in extra eyes appear normal; electroretinograms suggest that photoreceptors in extra eyes can make functional synaptic connections, although neuroanatomical studies show receptor-central-nervous-system innervations only rarely; the rare supernumerary antennae, however, have receptor cells whose axons innervate the ventral brain.

cytology: No recombination has been observed between *ee* and a *P*-element insertion at 26D1-2.

other information: *ee* is incompletely penetrant, being influenced profoundly by apparent enhancer mutations on 1, 2, and 3; it behaves as a partial dominant when appropriate enhancers are made homozygous; *P* elements also serve as enhancers (Marcey). Penetrance is temperature-sensitive, being higher at 25° than at 19°; temperature-sensitive periods occur at mid-late embryogenesis and at early first instar.

***ef: elfin**

location: 1- (rearrangement).

origin: Induced by triethylenemelamine (CB. 1246).

discoverer: Fahmy, 1952.

references: 1959, DIS 33: 86.

phenotype: Small fly with slightly excess melanization.

Wings proportionally smaller, slightly altered in shape, and warped. Abdominal tergites often broken and abnormally pigmented. Males viable but sterile. RK3A.

cytology: Associated with *T(1;2)ef = T(1;2)14C8-D1;2R*.

Ef1α1: Elongation factor 1α #1

location: 2-{64}.

synonym: *F1*.

references: Walldorf, Hovemann, and Bautz, 1985, Proc. Nat. Acad. Sci. USA 82: 5795-99.

Hovemann, Richter, Walldorf, and Cziepluch, 1988, Nucleic Acids Res. 16: 3175-94.

Shepherd, Walldorf, Hug, and Gehring, 1986, Proc. Nat. Acad. Sci. USA 86: 7520-21.

phenotype: Encodes an α unit of the heterotrimeric protein, elongation factor 1, which promotes the binding of aminoacyl-tRNA to ribosomes. Originally isolated on the basis of its displaying 5 to 10 fold overexpression in females versus males. *Ef1 α 1* is expressed throughout development. The rate of protein synthesis in *Drosophila* declines with age; this has been attributed to impaired binding of aminoacyl-tRNA to ribosomes, which is promoted by elongation-factor 1 (EF1); transcription of elongation factor-1 mRNA shown to decrease with age; by fifteen days following emergence the level of EF1 mRNA is but a few percent that of newly emerged adults (Webster and Webster, 1984, *Mech. Ageing Dev.* 24: 335-42). Transformation with *Ef1 α 1* under the control of heat shock promoter increased the mean life span from 38.2 to 45.1 days at 25° and from 21.1 to 29.8 days at 29° (Shepherd *et al.*).

cytology: Placed in 48D by *in situ* hybridization.

molecular biology: Investigations of genomic and cDNA clones indicate a gene with 60-base pair untranslated minixon separated from the main body of the gene by an 1.3-kb intron. The single open reading frame encodes a polypeptide of 463 amino-acids; the sequence is highly conserved; it displays 90% homology with *EF1 α 1* and not much less with *EF1 α* from the brine shrimp and humans, indicating early separation of *Ef1 α 1* and *Ef1 α 2* during the evolution of *Drosophila*.

Ef1 α 2

location: 3-{102}.

synonym: *F2*.

references: Walldorf, Hovemann, and Bautz, 1985, *Proc. Nat. Acad. Sci. USA* 82: 5795-99.
Hovemann, Richter, Walldorf, and Cziepluch, 1988, *Nucleic Acids Res.* 16: 3175-94.

phenotype: Encodes an α unit of the heterotrimeric protein, elongation factor 1; isolated by homology with *Ef1 α 1*. Expressed primarily in pupae; low levels of mRNA detectable in third-instar larvae and adults.

cytology: Placed in 100E by *in situ* hybridization.

molecular biology: Gene comprises five exons separated by introns of 1.24, 0.45, 0.45 and 0.08 kb. Encodes a polypeptide one amino-acid shorter than that of *EF1 α 1* and 90% homologous thereto.

Ef2

location: 2-{54}.

references: Grinblat, Brown, and Kafatos, 1989, *Nucleic Acids Res.* 17: 7303-14.

phenotype: Encodes elongation factor 2 (EF2), which catalyzes the GTP-dependent translocation of tRNA from the aminoacyl to the peptidyl site of the ribosome; also catalyzes hydrolysis of GTP. Expression becomes detectable by four hours of development and persists into adulthood.

cytology: Placed in 39E-F by *in situ* hybridization.

molecular biology: Clone selected from embryonic cDNA library probed sequence from *mys*, which encodes the *Drosophila* homologue of β integrin, or position specific antigen (PS3); comparison of the sequence with the published sequences, however, revealed close nucleotide homology with hamster EF2. Longest open reading frame encodes an 844-amino-acid polypeptide of about 95 kd. Shows 85% identity with hamster EF2 except between residues 240 and 274 and between 90 and 100; displays

sequence similarity to other GTP binding proteins including EF1 α 1 in three domains in the N-terminal third, probably involved in GTP binding and GTPase activity; another segment in the N-terminal end and two in the carboxy-terminal end are shared only among elongation factors. The genomic sequence comprises four exons, the first of which is short and largely untranslated; the second contains the GTP-binding domains and one elongation factor specific domain, and the fourth most of the remaining shared domains. Northern blots detect a single mRNA of 3.1 kb.

eg: eagle

location: 3-47.3.

phenotype: Wings extended. Hairs on thorax somewhat disarranged. Dark pattern on thorax. Viability varies among alleles; *eg*² females sterile. RK2.

alleles:

allele	origin	discoverer	ref ^{α}	comments
<i>eg</i> ¹		Morgan, 1930	1	
<i>eg</i> ²		Bridges, 33j16	1	
<i>eg</i> ^{57c}	spontaneous	Nicoletti, 57c	1, 2	
<i>eg</i> ^{spy}	X ray	Puro, 1961	3	<i>T(2;3)33D4-E3;79A4-B1</i>

^{α} 1 = CP627; 2 = Nicoletti, 1957, *DIS* 31: 84; 3 = Puro and Arajärvi, 1969, *Hereditas* 62: 414-18.

cytology: Placed in 79A4-B1 based on the third-chromosome breakpoint of *T(2;3)eg^{spy} = T(2;3)33D4-E3;79A4-B1*.

egalitarian: see *egl*

Egfr: Epidermal growth factor receptor homologue

location: 2-100.

synonym: *C-erb*; *DER*.

references: Livneh, Glazer, Segal, Schlessinger, and Shilo, 1985, *Cell* 40: 599-607.
Wadsworth, Vincent, and Bilodeau-Wentworth, 1985, *Nature* 314: 178-80.
Lev, Shilo, and Kimchie, 1985, *Dev. Biol.* 110: 499-502.
Schejter, Segal, Glazer, and Shilo, 1986, *Cell* 46: 1091-1101.
Price, Clifford, and Schüpbach, 1989, *Cell* 56: 1085-92.
Schejter and Shilo, 1989, *Cell* 56: 1093-1104.

phenotype: Encodes the *Drosophila* homolog of epidermal growth factor receptor protein. Mutations with three different phenotypes and described under three different names shown to be alleles of *Egfr*. *Elp* (Ellipse) is a dominant eye shape and texture mutant; *flb* (faint little ball) is an embryonic lethal causing dorsalized embryos, and *top* (torpedo) is a maternal-effect lethal causing ventralized embryos; each of these classes is described in detail at the end of the entry; *in situ* hybridization with transcript-specific probes reveals uniform distribution of transcript during embryogenesis; in larvae, hybridization confined to mitotic tissues and not seen in cells with polytene chromosomes (Kammermeyer and Wadsworth, 1987, *Development* 100: 201-10). Transcript concentrated in cells of the central nervous system and gonial cells in adults.

alleles: Price *et al.* have subsumed the embryonic lethal alleles (*flb*) under the symbol for the maternal-effect-lethal alleles (*top*). We further consolidate both along with the dominant visible alleles (*Elp*) under the symbol

Egfr as *Egfr^f*, *Egfr^t*, and *Egfr^E* alleles respectively.

allele	origin	synonym	ref ^α	comments
<i>Egfr^{E1}</i>	spont	<i>Elp¹</i>	2, 3	hypermorph
<i>*Egfr^{E2}</i>	X ray	<i>Elp²</i>	1	<i>T(Y;2)57C9-DS</i>
<i>Egfr^{E3}</i>	EMS	<i>Elp^{B1}</i>	2	hypermorph
<i>Egfr^{f1}</i>	EMS	<i>flb^{1F26}</i>	4, 7	weak allele
<i>Egfr^{f2}</i>	EMS	<i>flb^{1K35}</i>	4, 7	severe allele
<i>Egfr^{f3}</i>	EMS	<i>flb^{1P02}</i>	4, 7	severe allele
<i>Egfr^{f4}</i>	EMS	<i>flb^{2C82}</i>	4, 7	intermediate allele
<i>Egfr^{f5}</i>	EMS	<i>flb^{2G31}</i>	4, 7	severe allele
<i>Egfr^{f6}</i>	EMS	<i>flb^{2L65}</i>	4, 7	severe allele
<i>Egfr^{f7}</i>	EMS	<i>flb^{2W74}</i>	4, 7	weak allele
<i>Egfr^{f8}</i>	EMS	<i>flb^{2X51}</i>	4, 7	severe allele
<i>Egfr^{f9}</i>	EMS	<i>flb^{3B41}</i>	4, 7	severe allele
<i>Egfr^{f10}</i>	EMS	<i>flb^{3B92}</i>	4, 7	severe allele
<i>Egfr^{f11}</i>	EMS	<i>flb^{3C81}</i>	4, 7	severe allele
<i>Egfr^{f12}</i>	EMS	<i>flb^{3C87}</i>	4, 7	severe allele
<i>Egfr^{f13}</i>	EMS	<i>flb^{3E07}</i>	4, 7	weak allele
<i>Egfr^{f14}</i>	EMS	<i>l(2)57DEFa¹</i>	5, 7	severe allele
<i>Egfr^{f15}</i>	EMS	<i>l(2)57DEFa²</i>	5, 7	severe allele
<i>Egfr^{f16}</i>	EMS	<i>l(2)57DEFa³</i>	5	
<i>Egfr^{f17}</i>	EMS	<i>l(2)57DEFa⁴</i>	5, 7	weak allele
<i>Egfr^{f18}</i>	EMS	<i>l(2)ES3</i>	7	weak allele
<i>Egfr^{f19}</i>	EMS	<i>l(2)ES6</i>	7	weak allele
<i>Egfr^{f20}</i>	EMS	<i>l(2)ES18</i>	7	weak allele
<i>Egfr^{f21}</i>	EMS	<i>l(2)ES45</i>	7	intermediate allele
<i>Egfr^{f22}</i>	EMS	<i>l(2)JE3</i>	7	weak allele
<i>Egfr^{f23}</i>	EMS	<i>l(2)SH3</i>	7	weak allele
<i>Egfr^{f24}</i>	EMS	<i>top^{CO}</i>	6	
<i>Egfr^{f25}</i>	EMS		6	
<i>Egfr^{f26}</i>	EMS		6	
<i>Egfr^{f27}</i>	EMS		6	
<i>Egfr^{f28}</i>	EMS		6	
<i>Egfr^{f29}</i>	EMS		6	
<i>Egfr^{f30}</i>	EMS		6	
<i>Egfr^{f31}</i>	EMS		6	
<i>Egfr^{f32}</i>	EMS		6	
<i>Egfr^{f33}</i>	EMS		6	
<i>Egfr^{f34}</i>	EMS		6	
<i>Egfr^{f35}</i>	EMS		6	
<i>Egfr^{f36}</i>	γ ray	<i>top^{10B}</i>	6	<i>ln(2R)57A;57F</i>
<i>Egfr^{f37}</i>	γ ray	<i>top^{4A}</i>	6	
<i>Egfr^{f38}</i>	γ ray	<i>top^{21B}</i>	6	
<i>Egfr^{f39}</i>	EMS	<i>Elp^{B1RB2}</i>	2	<i>Elp²</i> revertant
<i>Egfr^{f40}</i>	EMS	<i>Elp^{B1RB3}</i>	2	<i>Elp²</i> revertant
<i>Egfr^{f41}</i>	EMS	<i>Elp^{B1RB4}</i>	2	<i>Elp²</i> revertant
<i>Egfr^{f42}</i>	EMS	<i>Elp^{B1RB5}</i>	2	<i>Elp²</i> revertant
<i>Egfr^{f43}</i>	x ray	<i>Elp^{RE124}</i>	2	<i>Elp¹</i> revertant
<i>Egfr^{f11}</i>	EMS	<i>top^{QY}</i>	6, 7	maternal-effect lethal
<i>Egfr^{f12}</i>	EMS	<i>top^{CJ}</i>	6	maternal-effect lethal

^α 1 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95; 2 = Baker and Rubin, 1989, Nature (London) 150-53 (fig.); 3 = Grell, 1960, DIS 34: 50; 4 = Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-83; 5 = O'Donnell, Boswell, Reynolds, and Mackay, 1989, Genetics 121: 273-80; 6 = Price, Clifford and Schüpbach, 1989, Cell 56: 1085-92; 7 = Schejter and Shilo, 1989, Cell 56: 1093-1104.

cytology: Genomic clone hybridizes to 57F.

molecular biology: Genomic sequence isolated using a probe homologous to kinase domain of the v-erbB oncogene protein. cDNA clones isolated from 3-12 hr cDNA library. Sequence comparisons of cDNA and genomic clones reveal the presence of four 3' exons separated by three small introns; these are separated from two distant 5' exons by a long (i.e., 45 kb) intron. Three different splicing patterns in the 5' region each associated with a different 3' polyadenylation site generate three different transcripts. Conceptual amino-acid sequence suggests a 42-base-pair signal sequence followed by a 769-nucleotide extracellular putative EGF-binding domain with twelve potential N-linked glycosylation sites and 68

cystein residues, a 31-residue hydrophobic transmembrane region, and a 566-nucleotide cytoplasmic kinase domain with but 13 cysteines. The overall amino-acid homology with human epidermal-growth-factor receptor is 41% in the extracellular domain and 55% in the kinase domain. The ligand-binding domain contains three cysteine-rich regions, which share homology with each other and with two such regions present in human EGF receptor.

Egfr^E

synonym: *Elp: Ellipse*

references: Grell, 1960, DIS 34: 50.

Baker and Rubin, 1989, Nature (London) 150-53 (fig.).

phenotype: Eyes of *Egfr^{E/+}* heterozygotes rough and more oval than wild type; also display a slight disturbance of the wing-vein pattern. Homozygotes have smaller eyes with many fewer ommatidia and some regions lack them entirely; those ommatidia that are formed contain the normal number and arrangement of cells; the regions without ommatidia contain cells that resemble pigment cells and mechanosensory bristles; only about one tenth the normal number of preommatidial cell clusters differentiate behind the morphogenetic furrow. *Egfr^E* in heterozygous combination with a deficiency or null mutation for *Egfr* is normal in phenotype indicating that *Egfr^E* alleles are hypermorphic.

Egfr^f

synonym: *flb: faint little ball*

origin: Recovered as embryonic lethals (*Egfr^{f1}* to *Egfr^{f23}*) as mutations that fail to complement the female sterile effect (*Egfr^{f24}* to *Egfr^{f35}*) or the wing-vein defect (*Egfr^{f36}* to *Egfr^{f38}*) of *Egfr^t* or, as revertants of *Egfr^E* (*Egfr^{f39}* to *Egfr^{f43}*).

references: Nüsslein-Volhard, Wieschaus and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-83.

Schejter and Shilo, 1989, Cell 56: 1093-1104.

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Embryonic lethal. Embryos form a ball of dorsal hypoderm with the internal organs extruded anteriorly. Ventral cuticle absent or strongly reduced. First visible in extended-germ-band stage. Cells at the anterior and posterior ends of the embryo form clumps and slough off; very few head and gnathal cells remain. Substantial ectodermal cell death observed; germ band retraction fails to take place. Ultimately, cuticle formation produces mostly dorsal and lateral cuticular elements with but a narrow strip of denticles midventrally. Hypomorphic alleles initiate but do not complete germ-band retraction; they show intermediate phenotypes with wider denticle bands and in weak alleles some head and telson structures are formed as well. No maternal effect as shown by pole-cell transplantation.

Egfr^t

synonym: *top: torpedo*.

references: Schüpbach, 1987, Cell 49: 699-707.

Price, Clifford and Schüpbach, 1989, Cell 56: 1085-92.

phenotype: Maternal-effect lethal. Homozygous females lay eggs that are long and pointed at both ends. Such eggs often have only one fused dorsal appendage; also there is an increase in the number of follicle cells that give rise to the main body of the chorion at the expense of those ordinarily contributing to the operculum and dor-

sal appendages. *Egfr*¹ alleles are completely recessive and fully penetrant in homozygous females; the embryos never hatch. Homozygous and hemizygous adult flies exhibit incomplete fourth veins, absence of the anterior crossvein, rough eyes, loss of ocelli and ocellar bristles, and the loss of sensory bristles from the thorax. Changes in the embryonic pattern become visible at the beginning of gastrulation. Around the circumference of the embryo, 40% of the cells invaginate on the ventral side and form mesoderm; these cells become organized into two ventral furrows which are lost in later stages, and a mass of mesodermal cells fills the ventral half of the embryo. The only cuticle structure differentiated is a strip of dorsal hypoderm flanked by bands of ventral setae; lateral and ventral sides are made up of mesoderm. The head is reduced but filzkörper and spiracles are visible posteriorly. Experiments with germline mosaics produced by pole cell transplantation indicate that the mutant gives rise to ventralized eggs and embryos by interfering with processes taking place in somatic cells rather than germinal tissue. The mutant phenotype was only produced in mosaics in which wild-type germ cells were surrounded by *Egfr*¹ follicle cells and not by the reverse cell arrangement. *Egfr*¹ blocks dorsalization caused by *fs(1)K10*, but not that produced by *dl* females.

egl: egalitarian (*T. Schüpbach*)

location: 2-105.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus.

phenotype: Female sterile. Defect in cell fate detectable in anterior germarium. In contrast to wild-type, in all *egl* alleles all sixteen cells per cyst enter pachytene; subsequently all sixteen revert to nurse-cell morphology and behavior (Carpenter). Egg chambers increase in volume along the ovariole, but do not take up yolk. No chorion is formed. *BicD*¹ females simultaneously heterozygous for *egl*¹ or *egl*² fail to form double-abdomen embryos (Mohler and Wieschaus, 1986, *Genetics* 112: 803-22).

alleles: Five alleles; *egl*¹ through *egl*⁵, originally isolated as WU, RC, PB, Pr, and PV, respectively.

cytology: Placed in 59D8-60A2, since uncovered by *Df(2R)bw-S46 = Df(2R)59D8-11;60A7*, and by *Df(2R)bw-D23 = Df(2R)59D4-5;60A1-2*.

Egon: Embryonic gonad

location: 3-{47}.

origin: Isolated from genomic library using a *kni* zinc-finger probe.

references: Rothe, Nauber, and Jäckle, 1989, *EMBO J.* 8: 3087-89.

phenotype: Expression detected in embryonic gonad but not in other tissues or at other stages of development.

cytology: Placed in 79B by *in situ* hybridization.

molecular biology: Nucleotide sequence indicates an open reading frame encoding a polypeptide of 373 amino acids with a 1421-nucleotide intron between codons 26 and 27. The first 90 amino acids, which contain the zinc-finger domain, show 80% and 86% similarity to the products of *kni* and *knrl*, respectively; the three proteins share a 19-amino-acid motif, the *kni* box, just downstream from the zinc-finger domain. These polypeptides share features with vertebrate steroid hormone receptors; their putative ligand-binding domains exhibit a low level of similarity.

Eip55BD: Ecdysteroid-inducible polypeptide encoded in 55BD

location: 2-{86}.

synonym: *Eip40*.

references: Cherbas, Cherbas, Savakis, and Koehler, 1981, *Am. Zool.* 21: 743-50.

phenotype: The structural gene for a 40 kilodalton polypeptide (EIP40) whose concentration in Kc cells increases detectably beginning 30-60 min following administration of ecdysteroid hormone and reaching a maximum after 4-6 hr (Savakis, Demetri, and Cherbas, 1980, *Cell* 22: 665-74).

molecular biology: cDNA clone pKc252 isolated and used to select genomic clones from Maniatis library.

cytology: *In situ* hybridization localizes sequence to 55BD.

Eip71CD

synonym: *Eip28/29*.

location: 3-{42}.

references: Cherbas, Cherbas, Savakis, and Koehler, 1981, *Am. Zool.* 21: 743-50.

Savakis, Koehler, and Cherbas, 1984, *EMBO J.* 3: 235-43.

Cherbas, Schulz, Koehler, Savakis, and Cherbas, 1986, *J. Mol. Biol.* 189: 617-31.

Schulz, Cherbas, and Cherbas, 1986, *Proc. Nat. Acad. Sci. USA* 83: 9428-32.

phenotype: The structural gene for 28 and 29 kilodalton (as determined by gel mobility) polypeptides (EIP28, EIP29) whose concentration in Kc cells increases detectably beginning at 30-60 min and reaches a maximum at 4-6 hr following ecdysteroid administration (Savakis, Demetri, and Cherbas, 1980, *Cell* 22: 665-74). Each protein exists in three isoforms with different pI's, possibly owing to acetylation.

molecular biology: cDNA clone pKC252 isolated and used to select genomic clones. Hybrid selected mRNA translation products include both EIP28 and EIP29. Sequence analysis reveals the presence of four exons, δ , α , β , and γ from 11743 to -1627, -628 (or -640) to -435, -324 to -64, and -4 to +406 respectively. All contain coding information. Two primary transcripts are produced by alternate splicing; 75% use the consensus splice donor sequence of exon α and 25% an alternate donor site twelve bases upstream, encoding a polypeptide internally deleted for four amino acids: Tyr Lys Arg Met. Since EIP28 is two charges more basic than EIP29, it is concluded to be encoded by the longer message, which specifies a conceptual sequence of 28,218 Daltons, and EIP29 is encoded by the shorter message producing a product of 27,640 Daltons.

cytology: *In situ* hybridization localizes sequence to 71C3-D2.

Eip74EF: see *E74*

Eip75: see *E75*

el: elbow (*M. Ashburner*)

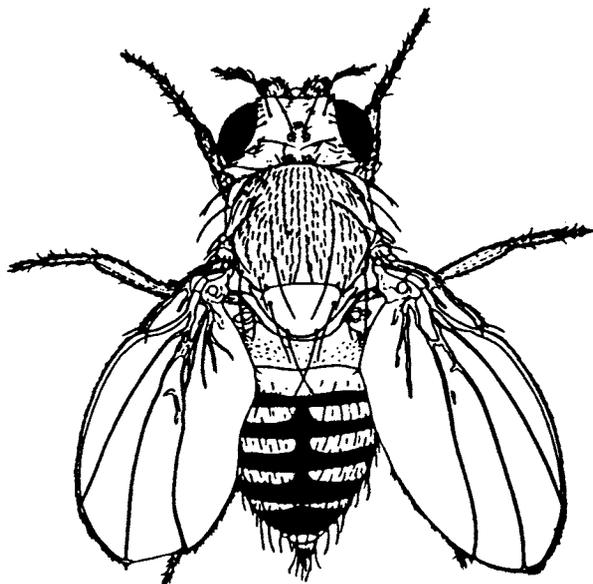
location: 2-50.0.

discoverer: E. M. Wallace, 35d1.

references: Bridges and Brehme, 1944.

phenotype: Wings extended and bent backward, often warped and shortened; sometimes blistered and nicked. Alulae reduced in size with reduced number of marginal

Temperature-sensitive allele; partially complements el^1 at 25° but not at 29°.



el: elbow

From Bridges and Brehme, 1944, Carnegie Inst. Washington Publ. No. 552: 75.

bristles—may fuse with wing blade. Venation reduced by terminal shortening of L5 and of crossveins. Halteres reduced, often to stubs. Eye size decreased (variable, even with strong alleles). Weak alleles may overlap wild type and show only a reduction in number of marginal bristles on alulae. Some alleles may be semilethal when hemizygous. Class (i) alleles enhance *Sco*, are semilethal with alleles of *l(2)35Ba* and show a weak *noc* phenotype when heterozygous with strong *noc* alleles or *noc* deletions. Class (ii) alleles do not interact with these loci. All alleles more extreme when hemizygous; strong alleles nearly apterous.

allele	origin	discoverer	ref ^α	comments
<i>el</i> ¹	spont	E. Wallace, 35dl	4	strong; class i
<i>el</i> ² β	EMS	Maroni	3	weak; class i
<i>el</i> ³ γ	EMS		2, 5	weak; class i
<i>el</i> ⁴	EMS	Harrington	1	strong; class ii
<i>el</i> ⁵	X ray			intermediate; class ii
<i>el</i> ⁶	EMS	Angel, 82f2		class ii
<i>el</i> ⁷	γ ray	Ashburner		<i>T(2;3)shv</i> ¹⁹
<i>el</i> ⁸	γ ray	Johnson		
<i>el</i> ⁹	γ ray	Johnson		<i>In(2L)34A2-3;35A3-4</i>
<i>el</i> ¹⁰	γ ray	Johnson		
<i>el</i> ¹¹	γ ray	Johnson		
<i>el</i> ¹²	γ ray	Johnson		
<i>el</i> ¹³	γ ray	Johnson		
<i>el</i> ¹⁴	γ ray	Johnson		
<i>el</i> ²¹	EMS	Johnson		
<i>el</i> ²²	EMS	Johnson		
<i>el</i> ²³	EMS	Johnson		

^α 1 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186; 2 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95; 3 = Ashburner, Tsubota, and Woodruff, 1982, Genetics 102: 401-20; 4 = CP627; 5 = Woodruff and Ashburner, 1979, Genetics 92: 133-49.

^β Formerly *el*^{GM2}; to the right of *el*¹; 0.02% recombination (Maroni); partially complements *el*¹; hemizygotes show weak expression; homozygotes exhibit < 0.5% viability.

^γ

cytology: Placed in region 35A1-3 on the basis of its inclusion in $Df(2L)fn2 = Df(2L)35A1-3;35B2-4$ but not in $Df(2L)fn3 = Df(2L)35B1;35B3-4$ or $Df(2L)b80e3 = Df(2L)34C3-4;35A4-B1$. el^4 associated with $T(Y;2)el^4$; breakpoint distal to *el*. el^5 associated with $T(Y;2)A15$.

other information: Part of *el-noc* complex.

***elav*: embryonic lethal, abnormal vision**
(J. C. Hall)

location: 1-{0}.

synonym: *l(1)EC7, fljJ, l(1)Bg*.

references: Homyk, Szidonya, and Suzuki, 1980, Mol. Gen. Genet. 177: 553-65.

Homyk and Grigliatti, 1983, Dev. Genet. 4: 77-97.

Campos, Grossman, and White, 1985, J. Neurogenet. 2: 197-218.

Homyk, Isono and Pak, 1985, J. Neurogenet. 2: 309-24.

Campos, Rosen, Robinow and White, 1987, EMBO J. 6: 425-31.

Jiménez and Campos-Ortega, 1987, J. Neurogenet. 4: 179-200.

Robinow, Campos, Yao and White, 1988, Science 242: 1570-72.

Robinow and White, 1988, Dev. Biol. 126: 294-303.

phenotype: Embryonic lethal, or in the case of viable and ostensibly hypomorphic alleles, displays poor jumping and flying ability plus aberrant visual physiology and behavior. No morphological abnormalities visible in sections of dying embryos (*elav*¹, *elav*², or *elav*³); however, whole-mount embryos show periodic interruptions in the longitudinal connectives of the CNS and missing commissures especially the posterior ones (Jiménez and Campos-Ortega). *elav*^{ts1} allows survival to adult stage at 19°-25° but viability is reduced and adults usually die soon after eclosion; viability after rearing at 30° is very low and newly emerged adults show poor coordination and die soon; this temperature-sensitive allele also causes morphological abnormalities in the brain, especially in the visual system (after postembryonic shift from 19° to 30° or even following all development at low-temperature); optic chiasma is abnormal and second order optic lobe (medulla) is rotated to aberrant position (Campos *et al.*, 1985); when *elav*^{ts1} raised at 30°, surface of eye is rough and photoreceptor layer abnormal in sections (Campos *et al.*, 1985). Another temperature-sensitive allele *elav*¹⁹ also induces abnormalities of visual system (Homyk *et al.*, 1985); rearing at 29° or high-temperature pulses delivered to pupae, raised otherwise at 20°, causes vacuolization of photoreceptors and disorganization of rhabdomeres; high-temperature rearing or pupal pulsing induces severe optic lobe defects (absence of size reduction); electroretinograms of this mutant, raised at high-temperature, are missing light-on and light-off transient spikes (also seen after low-temperature rearing) and have reduction of ERG photoreceptor potential; amplitude of this potential also deteriorates as does deep pseudopupil when adults treated at high-temperature after low-temperature rearing; mosaic analysis (Campos *et al.*, 1985) of *elav*¹ reveals autonomously induced defects in eye morphology, but no effects on other imaginal disc derivatives, and suggests both directly induced defects in optic lobe development,

as well as inductively caused CNS defects mediated through expression of this mutation in the eye (i. e., such that the visual system's ganglia are genotypically normal). Lethal "focusing" in these mosaics suggests influence of gene on derivatives of ventral blastoderm. In studies of viable alleles, *elav*¹⁹ and *elav*²⁰, both of which are temperature-sensitive, flying and jumping ability shown to be especially aberrant after rearing at 29°C; wing position also aberrant; *elav*^{ts1} most severe, including having no optomotor response when raised at high (or even low) temperature; temperature-sensitive period for aberrant wing posture in *elav*¹⁹ extends from larval to pupal period (Homyk and Grigliatti). An antibody specific to neuronal nuclei fails to stain neurons of *elav*-deficient embryos; however, the quantity of antigen does not respond to the number of *elav*⁺ genes present (Bier, Ackerman, Barbel, Jan and Jan, 1988, Science 240: 913-16). *elav* transcripts detected in all postmitotic neurons, from their birth; not seen in embryonic or larval neuroblasts. Also seen in larval eye discs, adult retinas and Johnston's organ of the antennae.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>elav</i> ¹	EMS	White		1	
<i>elav</i> ²	MMS	Lim	<i>l(1)EC7^{M3}</i>	1	
<i>elav</i> ³	MMS	Lim	<i>l(1)EC7^{M11}</i>		
<i>elav</i> ⁴	X ray	Lefevre	<i>l(1)A74</i> <i>l(1)EC7^{G3}</i> <i>elav^{G3}</i>	4	<i>ln(1)IA6-B1;IB5-9</i>
<i>elav</i> ⁵	X ray	Lefevre	<i>l(1)A138</i>	4	
<i>elav</i> ⁶	X ray	Lefevre	<i>l(1)N78</i>	4	
<i>*elav</i> ⁷	X ray	Lefevre	<i>l(1)RF57</i> <i>l(1)EC7^{G10}</i>	4	
<i>*elav</i> ⁸	EMS	Lefevre	<i>l(1)EF435</i>	5, 6	embryonic lethal no maternal effect
<i>elav</i> ⁹	EMS	Lefevre	<i>l(1)VA18</i>	5	
<i>elav</i> ¹⁰	EMS	Lefevre	<i>l(1)VA21</i>	5	
<i>elav</i> ¹¹	EMS	Lefevre	<i>l(1)VA262</i>	5	
<i>elav</i> ¹²	EMS	White	<i>elav^{MM3}</i>		leaky
<i>elav</i> ¹³	ENU	Voelker	<i>l(1)A34</i>		
<i>elav</i> ¹⁴	ENU	Voelker	<i>l(1)A54</i>		
<i>elav</i> ¹⁵	ENU	Voelker	<i>l(1)A75</i>		
<i>elav</i> ¹⁶	ENU	Voelker	<i>l(1)A129</i>		
<i>elav</i> ¹⁷	HMS		<i>l(1)HM30</i>	3	
<i>elav</i> ¹⁸	HMS		<i>l(1)HM41</i>	3	
<i>elav</i> ¹⁹	EMS	Homyk	<i>flj¹</i>	2	temperature sensitive
<i>elav</i> ²⁰	EMS	Homyk	<i>flj²</i>	2	temperature sensitive
<i>elav</i> ²¹	EMS	Rosen	<i>elav^{A34}</i>		
<i>elav</i> ²²	EMS	Rosen	<i>elav^{A54}</i>		
<i>elav</i> ²³	EMS	Rosen	<i>elav^{A75}</i>		
<i>elav</i> ²⁴	EMS	Rosen	<i>elav^{A129}</i>		
<i>elav^{ts1}</i>	EMS	White			temperature sensitive

^α 1 = Campos, Grossman, and White, 1985, J. Neurogenet. 2: 197-218; 2 = Homyk, Szidonya, and Suzuki, 1980, Mol. Gen. Genet. 177: 553-65; 3 = Kramers, Schalet, Paradi, and Huiser-Hoogteyling, 1983, Mutat. Res. 107: 187-201; 4 = Lefevre, 1981, Genetics 99: 461-80; 5 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 6 = Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14.

cytology: Placed in 1B5-9 based on the proximal breakpoint of *ln(1)elav*⁴ = *ln(1)IA6-B1;IB5-9*, which interrupts the *elav* gene.

molecular biology: Probing Northern blots with a 4.8-kb genomic subclone spanning the *ln(1)elav*⁴ breakpoint identifies 4.7, 5.4, and 6.1-kb transcripts; the 5.4-kb RNA is seen in 0-6-hr embryos and the 4.7 and 6.1-kb RNAs in 6-18-hr embryos; no transcripts seen in larvae and two very low abundance RNAs of similar size to the

embryonic RNAs seen in pupae; adult heads contain 5.4 and 6.1-kb transcripts; all three transcribed from right to left. A genomic clone encompassing the region homologous to the 4.7 and 6.1 but not all of the 5.4-kb transcript able to rescue lethality of *elav*. Sequence analysis of genomic and partial-length cDNA clones indicate an open reading frame specifying a complete polypeptide of 483 amino acids and 50.76 kd. The coding sequence contains two introns of 1289 and 2208 nucleotides. The conceptual translation product contains three repeats of a pair of sequences previously defined as RNA-binding consensus sequences, an octopeptide, RNP1, and an appropriately spaced hexapeptide, RNP2, suggesting that this protein is involved in the RNA metabolism of neurons. In addition, a palindromic sequence is seen 5' to the transcription unit and GAGA motifs are found 5' to the transcription unit, and in the second intron. Eleven copies of a putative zeste-protein-binding sequence are found in the vicinity.

elbow: see *el*

elf: *extra lamina fiber* (J.C. Hall)

location: 1- between *v* and *f*.

origin: Induced by ethyl methanesulfonate.

synonym: *H37; opm37*.

references: Heisenberg and Gotz, 1975, J. Comp. Physiol. 117: 127-62.

phenotype: An extra large fiber profile is found in each cartridge of lamina (first order optic lobe); adults exhibit defective phototaxis and optomotor responses (e.g., higher than normal light intensities needed for vigorous responses); light-off transient spike of electroretinogram weak.

other information: Morphological difference from normal may no longer exist in adult lamina (Meinertzhagen).

elfin: see *ef*

Elf-1 : see *Ntf*

Ellipse: see *Egfr^E*

ellipsoid body open: see *ebo*

elliptical rough: see *elr*

Elongation factor: see *Ef*

Elp : see *Egfr^E*

***elr:** *elliptical rough*

location: 1-25.1.

origin: X ray induced.

discoverer: Fahmy, 1956.

references: 1960, DIS 34: 49.

phenotype: Eyes slightly elliptical and rough. Wings slightly broader. Both sexes viable and fertile. RK2.

other information: Two other alleles: one induced by X rays, one by L-p-N, N-di-(2-chloroethyl)amino-phenylalanine.

elyC: *early C*

location: 3-(unmapped).

origin: Induced by ethyl methanesulfonate.

discoverer: Nüsslein-Volhard.

references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Maternal-effect lethal. Embryos produced by

homozygous females show abnormal development during early cleavage and cease development soon thereafter.

alleles: Two, *elyC*¹ and *elyC*², isolated as 043 and 230.

Embryonic gonad: see *Egon*

embryonic lethal, abnormal vision: see *elav*

***emc*: extra macrochaetae (H.M. Ellis)**

location: 3-0.

references: Botas, Moscoso del Prado, and García-Bellido, 1982, EMBO J. 1: 307-10.

Moscoso del Prado and García-Bellido, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 242-45.

García-Alonso and García-Bellido, 1988, Roux's Arch. Dev. Biol. 197: 328-38.

Ellis, Spann, and Posakony, 1990, Cell 61: 27-38.

Garrell and Modolell, 1990, Cell 61: 39-48.

phenotype: Hypomorphic alleles, or amorphic alleles when heterozygous with a hypomorphic allele, cause formation of extra macrochaetae. Severely hypomorphic combinations also cause formation of extra microchaetae. Amorphic alleles when homozygous result in embryonic lethality. Amorphic alleles when heterozygous with a normal third chromosome result in the appearance of ectopic chaetae in the occipital and premandibular regions. Extra chaetae in *emc* mutant flies appear on the head and notum (weak alleles) as well as on the wing blade, pleura, post-scutellum, tergites and legs in more severe allelic combinations. In severely hypomorphic allelic combinations extra campaniform sensilla occasionally appear on the wing blade. Reduction of wild-type *emc* function also causes the presence of extra bits of wing vein. A dominant *emc* allele (*emc*^D) causes a reduction in the number of macrochaetae on the head and notum. *emc*^D is homozygous viable; *emc*^D homozygotes are more mutant than *emc*^{D/+} individuals. The L4 and L5 wing veins are shortened in *emc*^D homozygotes but only rarely in *emc*^{D/+} heterozygotes. The insufficiency produced by *emc* can be titrated by altering the dosage of *ASC*; increased function of *ASC* produced by gain-of-function mutations (*Hw*) can be titrated by altering the dosage of *emc*⁺.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>emc</i> ¹	EMS	Ripoll		1	lethal; cytology normal
<i>emc</i> ²				1	in <i>TM2</i>
<i>emc</i> ³	X ray	Moscoso del Prado	<i>AN88</i>	5	<i>T(Y;3)61C4-9</i>
<i>emc</i> ⁴	X ray	Moscoso del Prado	<i>BB83</i>	4, 5	<i>ln(3L)61C-D;61E-F</i>
<i>emc</i> ⁵	X ray	Moscoso del Prado	<i>CC92</i>	4, 5	<i>ln(3L)61C;61C</i>
<i>emc</i> ⁶	X ray	Moscoso del Prado	<i>DT99</i>	4, 5	<i>T(2;3)34D;61C4-D</i>
<i>emc</i> ⁷	X ray	Moscoso del Prado	<i>DO96</i>	5	cytology normal
<i>emc</i> ⁸	X ray	Moscoso del Prado	<i>ES106</i>	5	<i>T(2;3)47A;61C4-D</i>
<i>emc</i> ⁹	X ray	Moscoso del Prado	<i>FX119</i>	4, 5	lethal; cytology normal
<i>emc</i> ¹⁰	X ray	Moscoso del Prado	<i>R14</i>	5	<i>ln(3L)61C3-8;80F</i>
<i>emc</i> ¹¹	X ray	Rubio	<i>emc^{pel}</i>	4	viable; cytology normal
<i>emc</i> ¹²	X ray	Ellis	<i>9-5a</i>		lethal; <i>T(2;3)52;61C</i>
<i>emc</i> ¹⁵	X ray	Ellis	<i>13-16</i>		viable; <i>ln(3L)61C;64;?</i>
<i>emc</i> ¹⁶	X ray	Ellis	<i>17-2A</i>		lethal; <i>ln(3L)61C5-6;80F</i>
<i>emc</i> ¹⁷	X ray	Ellis	<i>20-1A</i>		viable; <i>ln(3L)61C;62C</i>
<i>emc</i> ¹⁸ γ	X ray	Ellis	<i>24-4a</i>		viable; cytology normal
<i>emc</i> ¹⁹	X ray	Ellis	<i>29-2</i>		viable; <i>T(2;3)5B5-9;61C4</i>
<i>emc</i> ²⁰	EMS	Spann	<i>emc^{M7}</i>		viable; cytology normal
<i>emc</i> ²¹	EMS	Leviton	<i>emc^{M1}</i>		lethal; cytology normal
<i>emc</i> ²² δ	X ray	Ellis	<i>Ar5-5</i>		lethal; cytology normal
<i>emc</i> ²³	P ^E	Ellis	<i>emc^{P3}</i>	3	lethal; <i>P</i> insert at 61D1-2
<i>emc</i> ²⁴	P	Ellis	<i>emc^{P4}</i>	3	viable; <i>P</i> insert at 61D1-2
<i>emc</i> ²⁵	P	Ellis	<i>emc^{P6}</i>	3	viable; <i>P</i> insert at 61D1-2

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>emc</i> ²⁶	P	Ellis	<i>emc^{P7}</i>	3	lethal; <i>P</i> insert at 61D1-2
<i>emc</i> ²⁷	P	Ellis	<i>emc^{P15}</i>	3	viable; <i>P</i> insert at 61D1-2
<i>emc</i> ²⁸	P	Ellis	<i>emc^{P16}</i>	3	viable; <i>P</i> insert at 61D1-2
<i>emc</i> ²⁹	P	Ellis	<i>emc^{P17}</i>	3	viable; <i>P</i> insert at 61D1-2
<i>emc</i> ³⁰	P	Ellis	<i>emc^{P24}</i>	3	viable; <i>P</i> insert at 61D1-2
<i>emc</i> ³¹	P	Ellis	<i>emc^{P25}</i>	3	lethal; <i>P</i> insert at 61D1-2
<i>emc</i> ³²	P	Ellis	<i>emc^{P26}</i>	3	viable; <i>P</i> insert at 61D1-2
<i>emc</i> ³³ δ	P	Ellis	<i>AP1-3a</i>		viable; <i>P</i> insert at 61D1-2
<i>emc</i> ³⁴ δ	P	Ellis	<i>AP3-6</i>		lethal; <i>P</i> insert at 61D1-2
<i>emc</i> ^D	spont	Craymer	<i>Ach</i>	2	viable; cytology normal

^α 1 = Botas, Moscoso del Prado, and García-Bellido, 1982, EMBO J. 1: 307-10; 2 = Craymer, 1980, DIS 55: 197-200; 3 = Ellis, Spann, and Posakony, 1990, Cell 61: 27-38; 4 = García-Alonso and García-Bellido, 1988, Roux's Arch. Dev. Biol. 197: 328-38; 5 = Moscoso del Prado, 1985, PhD thesis, Universidad Autonoma de Madrid.

^β Viability refers to homozygotes.

^γ Hypomorphic allele; complements amorphic alleles for lethality.

^δ *emc*^D revertant.

^ε These experiments utilized Birmingham 2; delta 2-3.

cytology: Placed in 61D1-2 on the basis of *in situ* hybridization with *P*-element probes to revertible *P*-element induced *emc* null alleles.

molecular biology: The *emc* locus encodes a 2.3-kb transcript (interrupted by a single intron of 1.9-kb) that is present at all stages of development. Peak levels of transcript accumulation occur in 6-16 hour embryos and late third instar larvae and early pupae. *emc* transcripts appear in ovaries as well as female somatic tissue. Transcripts are also present in adult males. *emc* transcripts are present throughout the embryo at all stages from early cleavage through germ band extension. In third-instar larvae, *emc* transcripts are found in all tissues but are more abundant in the imaginal discs. RNA distribution is relatively homogeneous throughout the wing disc. *emc* cDNA clones contain a single long open reading frame capable of encoding a protein of approximately 22 kd. Analysis of the derived sequence of the *emc* protein shows that it is a member of the helix-loop-helix (HLH) family of proteins, but lacks the DNA binding motif characteristic of this group of proteins.

***ems*: empty spiracles**

location: 3-53.

origin: Induced by ethyl methanesulfonate.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95 (fig.). Dalton, Chadwick, and McGinnis, 1989, Genes Dev. 3: 1940-56 (fig.).

phenotype: Embryonic lethal. Embryos display loss of filzörper in the posterior spiracles; posterior ends of longitudinal tracheal trunks incomplete. Failure of head involution; many cuticular structures that normally derive from the procephalic and mandibular lobes of the head missing; embryonic antennal organs missing. *ems* protein first appears as an anterior ring around the syncytial blastoderm at a position just anterior to that of *Dfd*; it is five to six cells wide dorsally and 10-12 cells wide ventrally; at gastrulation the ring is just anterior to the cephalic furrow. As gastrulation proceeds expression becomes patchy and confined to specific groups of cells. The protein is nuclear in localization. The metameric pattern of *ems* expression commences at the beginning of germ-band extension; protein first appears in a group of

cells in each segment, which subsequently elongates and splits into two clusters of *ems*-positive cells in regions destined to give rise to tracheal pits, neuroblasts and epidermis. In the eighth abdominal segment, a large patch of *ems*-positive cells forms just posterior to the tracheal pit and presumably correspond to the primordia of the posterior spiracles and the filzkörper; a similar patch is formed anteriorly in embryos produced by *bcd* mothers. For a detailed discussion of the expression pattern see Dalton *et al.*

alleles: Five alleles induced with ethyl methanesulfonate by Jürgens *et al.*

allele	synonym	comments
<i>ems</i> ¹	7D99	
<i>ems</i> ²	9H83	amorphic allele; stop codon at residue 161
<i>ems</i> ³	9Q64	stop codon at residue 141
<i>ems</i> ⁴	10A	hypomorphic allele

cytology: Placed in 88A1-2 by *in situ* hybridization.

molecular biology: Gene isolated on basis of homology to *eve* homeo domain. Northern blots with genomic fragment detect a 2.3-kb transcript that reaches peak abundance during 6-12 hr of embryonic development, and with substantial expression throughout embryonic development as well as in the third larval instar and pupal stages. Nucleotide sequence of a 2.2-kb cDNA and a portion of genomic sequence reveals the presence of an ~300 nucleotide intron just upstream of the homeo domain. The longest open reading frame could encode a polypeptide of 494 amino-acid residues; amino acids 1-384 contain 80 proline residues; residues 99-359 contain 12% glutamines. The C-terminal end contains a 24-amino-acid segment with 62% glutamic and aspartic acid residues. The homeo-domain sequence is quite divergent from other published homeobox sequences, exhibiting less than 50% identity.

***en: engrailed* (T.Kornberg)**

location: 2-62.

references: Eker, 1929, *Hereditas* 12: 217-22.

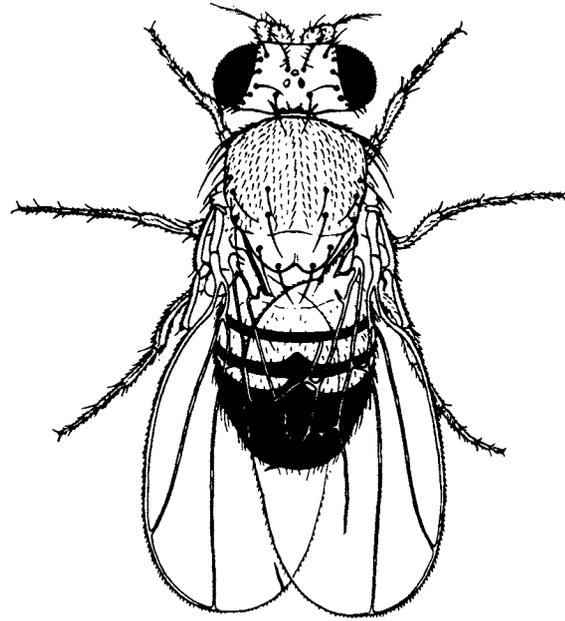
Nüsslein-Volhard and Wieschaus, 1980, *Nature* 287: 795-801.

Kornberg, 1981, *Proc. Nat. Acad. Sci. USA* 78: 1095-99.

Eberlein and Russell, 1983, *Dev. Biol.* 100: 227-37.

phenotype: Four classes of alleles, all recessive.

(1) *en*¹. Viable hemizygous and homozygous; fertile. Longitudinal cleft extends from rear border of scutellum forward, may be reduced to median nick or posterior flattening of scutellum. Wings larger, broader, and thin textured with spatulate end; venation and distribution of sensilla abnormal in posterior wing compartment. Variable duplication of anterior triple row bristles on posterior margin; alula reduced, with costal-like bristles. In males, extra sex comb often present (Brasted, 1941, *Genetics* 26: 347-73), smaller than normal, and in mirror-image position in posterior compartment. Duplications of transverse rows in female prothoracic leg, extra bristles in mesothoracic and metathoracic tarsi (García-Bellido, and Santamaria, 1972, *Genetics* 72: 87-104; Lawrence, Struhl, and Morata, 1979, *J. Embryol. Exp. Morph.* 51: 195-208). Action of *en*¹ is autonomous except for scutellar cleft (Tokunaga, 1961, *Genetics* 46: 157-76; Stern and Tokunaga, 1968, *Proc.*



en: engrailed

From Eker, 1929, *Hereditas* 12: 217-22.

Nat. Acad. Sci. USA 60: 1252-59; García-Bellido, and Santamaria, 1972, *Genetics* 72: 87-104). Clones of *en* cells of posterior compartment origin fail to respect anterior-posterior compartment border in wing disc as do *mwh* clones in wing discs of *en*¹ homozygotes (Morata and Lawrence, 1975, *Nature* 255: 614-17; Morata and Lawrence, 1976, *Dev. Biol.* 50: 321-37). *en* abnormalities are associated with posterior compartment structures only, except for scutellar cleft. Restriction of glucose-6-phosphate dehydrogenase, 6-phosphogluconate dehydrogenase (Cunningham, Smith, Makowski, and Kuhn, 1983, *Mol. Gen. Genet.* 191: 238-43) and of a protein recognized by monoclonal antibody PS2 (Brower, 1984, *Nature* 310: 496-98; 1987, *EMBO J.* 5: 2649-56) to posterior compartment of wing disc altered by *en*¹. Interaction with *ci*, *cg*, *sx* (House, 1953, *Genetics* 38: 200-15; House, 1953, *Genetics* 38: 309-27; House, 1961, *Genetics* 46: 871; Mukherjee, 1965, *Genetics* 51: 285-304; Datta and Mukherjee, 1971, *Genetics* 68: 269-86) and *fu* (Fausto-Sterling and Smith-Schiess, 1982, *EMBO J.* 1: 827-33) partially increase phenotype. No suppression by *su(Hw)*.

(2) Lethal alleles with normal cytology. Embryonic lethal. Anterior margin of each segment defective. Pair rule defects in naked cuticle of T1, T3, A2, A4, A6, A8 result in pair-wise fusion of adjacent segments. Autonomous effects in adult cuticular clones observed in posterior compartment of proboscis, thorax, abdomen, and genitalia. *en*^{lethal} clones are without effect in anterior compartments and in eye-antennal region (Kornberg, 1981, *Proc. Nat. Acad. Sci. USA* 78: 1095-99; Kornberg, 1981, *Dev. Biol.* 86: 363-72; Lawrence and Struhl, 1982, *EMBO J.* 1: 827-33). The *en*¹/*en*^{lethal} heterozygote characterized by wing abnormalities only; disruption of anterior crossvein, gap in vein IV, and occasional socketted bristles on posterior margin. In some combinations complementation is complete or nearly so (Condie and

Brower, 1989, Dev. Biol. 135: 31-42). No maternal effect (Lawrence, Johnston and Struhl, 1983, Cell 35: 27-34).

(3) Deficiencies and lethal alleles with inversion or translocation breakpoints. Embryonic lethal. Embryonic segment defects slight and variable. Alleles of this class in heterozygous combination with *en*¹ produce adults more extreme than *en*¹. For example, in *en*¹/*en*², legs are truncated, the tarsi reduced to densely bristled stumps; wings are greatly enlarged and spatulate with greater disruption of veins IV and V; higher penetrance of socketed bristles along the posterior margin. Extreme scutellar cleft. At 29°, duplication of anterior compartment bristles in mirror-image symmetry in posterior compartment of second antennal segment (Morata, Kornberg, and Lawrence, 1983, Dev. Biol. 99: 27-33).

(4) Non-lethal alleles with breakpoints. Hemizygous viable, embryos normal. Heterozygous with other allele classes, variable gaps in wing veins IV and V. Variable reductions or deletions in male and female genitalia (Epper and Sanchez, 1983, Dev. Biol. 100: 387-98). Scutellum may also be affected.

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>en</i> ¹	spont	Evang 26k7		1, 3	viable; 7 kb insert at 0 kb
<i>en</i> ²	EMS	Kornberg	<i>en</i> ^{C2}	2, 3	lethal; <i>In(2R)47A;48A</i> ; breakpoint at -0.0 to 2.7 kb
<i>en</i> ³	EMS	Kornberg	<i>en</i> ^{LA3}	2, 3	viable; <i>T(2;3)48A;96C</i> ; breakpoint at 25.3 to 34.2 kb
<i>en</i> ⁴	EMS	Kornberg	<i>en</i> ^{LA4}	2, 3	lethal
<i>en</i> ⁵	EMS	Kornberg	<i>en</i> ^{LA5}	2	
<i>en</i> ⁶	EMS	Kornberg	<i>en</i> ^{LA7}	2	
<i>en</i> ⁷	EMS	Kornberg	<i>en</i> ^{LA9}	2	
<i>en</i> ⁸	EMS	Kornberg	<i>en</i> ^{LA10}	2	
<i>en</i> ⁹	EMS	Kornberg	<i>en</i> ^{LA11}	2	
<i>en</i> ¹⁰	EMS	Kornberg	<i>en</i> ^{LA12}	2	
<i>en</i> ¹¹	EMS	Kornberg	<i>en</i> ^{LA13}	2	
<i>en</i> ¹²	EMS	Kornberg	<i>en</i> ^{LA14}	2	
<i>en</i> ¹³	EMS	Kornberg	<i>en</i> ^{LA15}	2	
<i>en</i> ¹⁴	EMS	Kornberg	<i>en</i> ^{LA16}	2	
<i>en</i> ¹⁵	EMS	Kornberg	<i>en</i> ^{LA17}	2	
<i>en</i> ¹⁶	EMS	Kornberg	<i>en</i> ^{LA18}	2	
<i>en</i> ¹⁷	γ ray	Kornberg	<i>en</i> ^{SFγ1}	2	
<i>en</i> ¹⁸	HOOH	Kornberg	<i>en</i> ^{SFH1}	2	
<i>en</i> ¹⁹	HOOH	Kornberg	<i>en</i> ^{SFH2}	2	
<i>en</i> ²⁰	HOOH	Kornberg	<i>en</i> ^{SFH3}	2	
<i>en</i> ²¹	HOOH	Kornberg	<i>en</i> ^{SFH4}	2	
<i>en</i> ²²	HOOH	Kornberg	<i>en</i> ^{SFH5}	2	
<i>en</i> ²³	HOOH	Kornberg	<i>en</i> ^{SFH6}	2	
<i>en</i> ²⁴	HOOH	Kornberg	<i>en</i> ^{SFH7}	2	
<i>en</i> ²⁵	HOOH	Kornberg	<i>en</i> ^{SFH8}	2	
<i>en</i> ²⁶	HOOH	Kornberg	<i>en</i> ^{SFH9}	2	
<i>en</i> ²⁷	HOOH	Kornberg	<i>en</i> ^{SFH10}	2	
<i>en</i> ²⁸	HOOH	Kornberg	<i>en</i> ^{SFH11}	2	
<i>en</i> ²⁹	HOOH	Kornberg	<i>en</i> ^{SFH12}	2	
<i>en</i> ³⁰	HOOH	Kornberg	<i>en</i> ^{SFH13}	2	
<i>en</i> ³¹	HOOH	Kornberg	<i>en</i> ^{SFH14}	2	lethal
<i>en</i> ³²	X ray	Kornberg	<i>en</i> ^{SFX1}	2	lethal
<i>en</i> ³³	X ray	Kornberg	<i>en</i> ^{SFX12}	2	
<i>en</i> ³⁴	X ray	Kornberg	<i>en</i> ^{SFX24}	2, 3	lethal; <i>T(2;3)48A;90C</i> ; breakpoint at 2.7 to 12.0 kb
<i>en</i> ³⁵	X ray	Kornberg	<i>en</i> ^{SFX26}	2	
<i>en</i> ³⁶	X ray	Kornberg	<i>en</i> ^{SFX29}	2	
<i>en</i> ³⁷	X ray	Kornberg	<i>en</i> ^{SFX30}	2	lethal
<i>en</i> ³⁸	X ray	Kornberg	<i>en</i> ^{SFX32}	2, 3	<i>T(Y;2)48A</i> ; breakpoint at -4.2 to -1.0 kb
<i>en</i> ³⁹	X ray	Kornberg	<i>en</i> ^{SFX33}	2	
<i>en</i> ⁴⁰	X ray	Kornberg	<i>en</i> ^{SFX34}	2	
<i>en</i> ⁴¹	X ray	Kornberg	<i>en</i> ^{SFX35}	2	
<i>en</i> ⁴²	X ray	Kornberg	<i>en</i> ^{SFX36}	2	
<i>en</i> ⁴³	X ray	Kornberg	<i>en</i> ^{SFX37}	2, 3	<i>Tp(2;3)46C;48A;80-81</i> ; breakpoints at -33.9 to -28.0 kb

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>en</i> ⁴⁴	X ray	Kornberg	<i>en</i> ^{SFX38}	2	and 2.7 to 12.0 kb
<i>en</i> ⁴⁵	X ray	Kornberg	<i>en</i> ^{SFX39}	2	
<i>en</i> ⁴⁶	X ray	Kornberg	<i>en</i> ^{SFX40}	2	
<i>en</i> ⁴⁷	X ray	Kornberg	<i>en</i> ^{SFX42}	3	lethal; <i>T(2;3)48A;65F</i> ; breakpoint at -15.2 to -10.6 kb
<i>en</i> ⁴⁸	X ray	Kornberg	<i>en</i> ^{SFX49}	3	lethal; <i>In(2R)47F;48A3-4</i> ; breakpoint at -15.2 to -10.6 kb
<i>en</i> ⁴⁹	X ray	Kornberg	<i>en</i> ^{SFX50}	3	lethal; <i>T(2;3)48A;57A;81A</i> breakpoint at -4.7 to -1.0 kb
<i>en</i> ⁵⁰	X ray	Kornberg	<i>en</i> ^{SFX52}	3	lethal; <i>T(2;3)48A;57B;88F</i> ; breakpoint at -33.9 to -28.0 kb
<i>en</i> ⁵¹	X ray	Kornberg	<i>en</i> ^{SFX61}	3	lethal; <i>T(2;3)48A;89A3;96B</i> ; breakpoint at 13 to 20.5 kb
<i>en</i> ⁵²	X ray	Kornberg	<i>en</i> ^{SFX62}	3	viable; <i>T(2;3)48A;84D</i> ; breakpoint at -15.2 to -10.6 kb
<i>en</i> ⁵³	X ray	Kornberg	<i>en</i> ^{SFX63}	3	breakpoint at -10.6 to -5.4 kb
<i>en</i> ⁵⁴	EMS		<i>en</i> ^{IIB}	5, 6	
<i>en</i> ⁵⁵	EMS		<i>en</i> ^{IIIB}	5, 6	
<i>en</i> ⁵⁶	EMS		<i>en</i> ^{IIT}	5, 6	
<i>en</i> ⁵⁷	EMS		<i>en</i> ^{IK}	5, 6	
<i>en</i> ⁵⁸	EMS		<i>en</i> ^{IM}	5, 6	
<i>en</i> ⁵⁹	EMS		<i>en</i> ^{IO}	5, 6	
<i>en</i> ^{Es}	X ray	Baker		3, 4	viable; <i>T(2;3)48A;84D</i> ; breakpoint at 25.3 to 34.2 kb

^α 1 = Eker, 1929, Hereditas 12: 217-22; 2 = Kornberg, 1981, Proc. Nat. Acad. Sci. USA 78: 1095-99; 3 = Kumer, Nakanishi, Ali, Drees, Gustavson, Theis, Kornberg, and O'Farrell, 1985, Cell 42: 309-16; 4 = Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84; 5 = Nüsslein-Volhard and Wieschaus, 1980, Nature 287: 795-801; 6 = Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

^β Origin of molecular coordinates defined as the insertion site in *en*¹; minus values to the left.

cytology: Placed in 48A3-4, based on two inversion and eleven translocation breakpoints.

molecular biology: 225 kb of genomic DNA proximal to *tRNA-Met2* at 48B5-7 (Poole, Kauvar, Drees, and Kornberg, 1985, Cell 40: 37-43) was used to locate *en* rearrangements. Gene also isolated directly from genomic library using *Ubx* homeobox probe (Fjose, McGinnis, Gehring, 1985, Nature 313: 284-89). *en*¹ is associated with a 7-kb insertion of a middle-repetitive DNA element (defined as molecular coordinate 0 with negative values to the left). Lethal breakpoints span the *en*¹ insertion from -35 kb proximally to +10 kb distally; non-lethal breakpoints are at +15 to +35 (Kunner, Nakanishi, Ali, Drees, Gustavson, Theis, Kauvar, Kornberg and O'Farrell, 1985, Cell 42: 309-16). Subclones from -20 to -12 detect transcripts of 1.4, 2.7 and 3.6 kb on Northern blots of RNA from embryos (high abundance), larvae, and pupae (low abundance), but not from adults; the abundance of the 2.7-kb species is ten times that of the other two in all stages investigated. Sequence determinations reveal a 1700-nucleotide open reading frame within which a polypeptide of 562 amino-acids and approximately 60 kd is encoded. The initial transcript contains introns of 1.1 and 0.28 kb, the latter one being 3' to the first and interrupting a homeobox encoding domain. The homeobox sequence diverges significantly from those at *ftz*, *Antp*, and *Ubx*, the latter displaying closer homology to human homeobox sequences than they do to the *en* homeo domain. In addition, in the 5' end of the gene there is a concentration of the repeating CAA and CAG as well as other single-amino-acid runs throughout the sequence (Poole, Kauvar, Drees, and Kornberg, 1985,

Cell 40: 37-43). Transcription occurs from the right to left (Drees, Ali, Soeller, Coleman, Poole, and Kornberg, 1987, EMBO J. 6: 2803-09). *en* protein binds to clusters of sequences located upstream from the *en* transcription-start site; both *en* and *ftz* gene products compete for the consensus sequence TCAATTAAT (Desplan, Theis, and O'Farrell, 1985, Nature 318: 630-35; Jaynes and O'Farrell, 1988, Nature 336: 744-49; Desplan, Theis, and O'Farrell, 1988, Cell 54: 1081-90). Shown to form stable polyprotein DNA-binding complex with other soluble proteins in the nucleus (Gay, Poole, and Kornberg, 1988, EMBO J. 4291-97). The developmental and spatial program of *en* expression determined by immunolocalization of its protein product (DiNardo, Kuner, Theis, and O'Farrell, 1985, Cell 43: 59-69; Karr, Weir, Ali, and Kornberg, 1989, Development 105: 605-12). Engrailed protein is first detectable in Western blots in the precellular blastoderm; at the initiation of cellularization a low level of protein distributed uniformly in embryo; as cellularization proceeds, the protein becomes concentrated in the second and fifth sixths of the egg length; subsequently stripes become more numerous and narrower, filling the central third of the embryo with stripes, first in even-numbered segments and then in every segment. At the onset of gastrulation; the cellular blastoderm exhibits fourteen one-cell-wide stripes of *en* expression; each stripe defines the anlagen of the posterior compartment of a metameric segment and the anterior parasegment boundary. At full germ-band extension *en* expressed in labial, maxillary, mandibular primordia, in three thoracic, and nine abdominal segments. Cell movements in head and tail regions lead to complex distributions of expressing cells. Subsets of cells in the ventral nervous system also seen to be expressing *en* at ten hours of development. As expected for a DNA-binding protein, *en* antigen accumulates in nuclei. In third-instar larvae *en* expression observed in presumptive posterior compartments of imaginal discs and in discrete cells in the ventral ganglion of the larval brain (Brower, 1987, EMBO J. 5: 2649-56). Expression of *en* positively controlled by *ftz*, *eve*, and *prd*; modulated by *h*, *odd*, *opa*, and *run* (Weir, and Kornberg, 1985, Nature 318: 433-39; Harding, Rushlow, Doyle, Hoey, and Levine, 1986, Science 233: 953-59; Howard, and Ingham, 1986, Cell 44: 947-57; DiNardo, and O'Farrell, 1987, Genes Dev. 1: 1212-25; Ingham, Baker, and Martinez-Arias, 1988, Nature 331: 73-75; Martinez-Arias, and White, 1988, Development 102: 325-28).

en-we: see *e(w^e)*

En-bx: see *E(bx)*

engrailed-related: see *inv*

enhancer: see *e()*

Enhancer: see *E()*

Enhancer-of-split mimic: see *Esm*

***Eno*: Enolase**

location: 2-{3}.

references: Bishop, and Corces, 1990, Nucleic Acids Res. 18: 191.

phenotype: Structural gene for enolase [2-phospho-D-glycerate hydrolase (EC 4.2.1.11)].

cytology: Placed in 22A by *in situ* hybridization.

molecular biology: Isolated from genomic library using cloned rat sequence. Coding sequence lacks introns; specifies a 433-amino-acid, 46,563-dalton polypeptide.

eo: extra organs

location: 1-{66}.

references: Schalet and Lefevre, 1973, Chromosoma 44: 183-202.

Schalet and Lefevre, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902.

Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31.

phenotype: Homozygous lethal; in rare surviving genotypes, a leg may be branched or completely duplicated, an antenna or arista duplicated, or triplicated; eyes and wings malformed; usually only eye effect present. Phenotype expressed in presence of *y⁺Ymal¹²⁶*. Homozygous germ-line clones are maternal-effect lethals; *eo²⁵* clones produce embryos with head defects and ventral holes; rescuable by *eo⁺* sperm. *eo¹* and *eo²⁸* clones produce embryos that are U-shaped as a result of incomplete germ-band retraction; have poor cuticle differentiation and ventral holes. *eo²⁷* clones also form ovarian tumors after approximately six days.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>eo¹</i>	3 HT	Kaplan	<i>l(1)A7</i>	6, 8, 9	larval lethal; maternal-effect lethal
<i>eo²</i>	X ray	Lifschytz	<i>l(1)B3</i>	3, 4	
<i>eo³</i>	X ray	Lifschytz	<i>l(1)B145</i>	4	
<i>eo⁴</i>			<i>l(1)E3</i>		
<i>eo⁵</i>			<i>l(1)E6</i>		
<i>eo⁶</i>	EMS	Lifschytz	<i>l(1)E54</i>	4, 8, 9	
<i>eo⁷</i>	EMS	Lifschytz	<i>l(1)M112</i>	5	on <i>y⁺Ymal⁺</i>
<i>eo⁸</i>		Himoe	<i>l(1)N30</i>		
<i>eo⁹</i>			<i>l(1)P313</i>		
<i>eo¹⁰</i>			<i>l(1)P414</i>		
<i>eo¹¹</i>	EMS	Lifschytz	<i>l(1)R-9-4</i>	4	
<i>eo¹²</i>	EMS	Lifschytz	<i>l(1)R-9-12</i>	4	
<i>eo¹³</i>	EMS	Lifschytz	<i>l(1)R-9-20</i>	4	
<i>eo¹⁴</i>	EMS	Lifschytz	<i>l(1)R-9-22</i>	4	
<i>eo¹⁵</i>	EMS	Lifschytz	<i>l(1)R-9-30</i>	4	
<i>eo¹⁶</i>	EMS	Lifschytz	<i>l(1)R-10-2</i>	4	
<i>eo¹⁷</i>	EMS	Lifschytz	<i>l(1)R-10-12</i>	4	
<i>eo¹⁸</i>	EMS	Lifschytz	<i>l(1)R-10-13</i>	4	
<i>eo¹⁹</i>	X ray	Lefevre	<i>l(1)C120</i>	1	
<i>eo²⁰</i>	X ray	Lefevre	<i>l(1)GA109</i>	1	
<i>eo²¹</i>	X ray	Lefevre	<i>l(1)KC18</i>	1	
<i>eo²²</i>	X ray	Lefevre	<i>l(1)L63</i>	1	
<i>eo²³</i>	EMS	Lefevre	<i>l(1)DA602</i>	2	
<i>eo²⁴</i>	EMS	Lefevre	<i>l(1)DC712</i>	2	
<i>eo²⁵</i>	EMS	Lefevre	<i>l(1)DC726</i>	2, 6	embryonic-to-larval lethal
<i>eo²⁶</i>	EMS	Lefevre	<i>l(1)DC801</i>	2, 6	
<i>eo²⁷</i>	EMS	Lefevre	<i>l(1)EA13</i>	2, 6	larval lethal; maternal-effect lethal
<i>eo²⁸</i>	spont	Schalet	<i>l(1)I7-44-2</i>	6, 7	pupal lethal; some escapers maternal-effect lethal
<i>eo²⁹</i>	spont	Schalet	<i>l(1)eo^{S1}</i> <i>l(1)I7-252</i> <i>l(1)eo^{S2}</i>	7	
<i>eo³⁰</i>	P	Gergen	<i>l(1)A-2</i>	10	
<i>eo³¹</i>	P	Gergen	<i>l(1)A7-1</i>	6, 10	
<i>eo³²</i>	P	Gergen	<i>l(1)S1-1</i>	10	
<i>eo³³</i>			<i>l(1)I7-36</i>	6	
<i>eo³⁴</i>			<i>l(1)I7-260</i>	6	

^α 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 3 = Lifschytz and Falk, 1968, Mut. Res. 6: 235-44; 4 = Lifschytz and Falk, 1969, Mut. Res. 8: 147-55; 5 = Lifschytz and Jakobovitz, 1978, Mol. Gen. Genet. 161: 275-84; 6 = Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31. 7 = Schalet, 1986, Mutat. Res. 163: 115-44; 8 = Schalet and Lefevre, 1973, Chromosoma 44: 183-202; 9 = Schalet and Lefevre, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902; 10 = Zusman, Coulter, and Gergen, 1985, DIS 61: 217-18.

cytology: Placed in 20A1-2 based on its inclusion in $Df(1)B12 = Df(1)I9E1;20A1-2$ and $Df(1)JC4 = Df(1)20A1;20E-F$.

Epa: see *Cos1*

Epidermal growth factor receptor homologue: see *Egfr*

eq: equational producer

location: 1- (to the right of *car*, probably heterochromatic).
origin: X ray induced.

discoverer: Schultz, 33a2.

references: Morgan, Bridges, and Schultz, 1934, Year Book - Carnegie Inst. Washington 33: 280.

phenotype: Produces 1-2% equational nondisjunction of X's in male, producing both X/X and nullo-X, nullo-Y sperm. Original *eq* male when crossed to attached-X female produced 89/289 equational exceptional X/X daughters. Claimed to generate *bb*-deficient Y chromosomes. More recent experiments (Valentin, 1984, Hereditas 101: 115-17) produce an order of magnitude fewer equational exceptions from males; none from homozygous females.

cytology: Both salivary and mitotic chromosomes appear normal.

eq1: equatorial-less (J.C. Hall)

location: 2-right arm near *bw*.

origin: Induced by ethyl methanesulfonate.

discoverer: Ransom.

references: Campos-Ortega, 1980, Current Topics in Developmental Biology 15: 347-71.

phenotype: Recessive lethal; in homozygous mutant clones in mosaic eyes, ommatidia lack one, two, or three photoreceptor cells from among two specific outer cells in a given facet (R_1, R_6) and a specific inner cell (R_7).

alleles: Two mutant alleles, *eq1*¹ and *eq1*² (published as *eq1*^{lff40} and *eq1*^{lff225}, respectively), the latter of which is more severe in its effects, often removing three photoreceptors from each facet instead of one or two.

cytology: Not uncovered by $Df(2R)bw^5$.

er: erupt

location: 3-71 (60.7 to 80.7; not an allele of *k*).

origin: Spontaneous.

discoverer: Glass, 1941.

references: 1943, DIS 17: 50.

1944, Genetics 29: 436-46.

1957, Science 126: 683-89 (fig.).

phenotype: Exhibits eruption of underlying hypodermis in center of one or both eyes. Eruption may be segmented and have hairs. Less extreme expression produces derangement of central or anterior-central facets. Eruption may occur as encroachment of chitin with bristles and hairs into anterior edge of eye. Extra tissue derived

from eye portion of eye-antennal disk (Aubele, 1968, DIS 43: 139). RK2.

other information: Alleles of at least five different strengths present in different wild stocks. Present in many wild stocks in suppressed condition.

***Er: Erect**

location: 3-50.

origin: Spontaneous.

discoverer: Neel, 41c9.

references: 1942, DIS 16: 50.

phenotype: Posterior scutellars at greater than normal angle with body, vary from slight effect to conditions in which bristles stand at right angles to scutellum. In latter case, bristles usually appear warped and twisted. Wings incompletely expanded and crinkled to varying degrees. RK3.

Erased: see *en*^{Es}

Erect: see *Er*

erect wing: see *ewg*

err: erratic (T. Schüpbach)

location: 2-40.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

phenotype: Maternal effect lethal; embryos from homozygous mothers have variable cuticular defects and holes.

alleles: *err*¹, isolated as *err*^{RE}.

cytology: Placed in 31B-32A based on lethality over $Df(2L)J27 = Df(2L)31B-D;31F-32A$.

erupt: see *er*

es: ether sensitive

location: 2- (not located).

origin: Spontaneous.

discoverer: Tinderholt.

references: Kidd, 1963, DIS 37: 49.

phenotype: Hypersensitive to diethyl ether and chloroform. Homozygotes killed by exposure to doses of these agents harmless to normal flies. Sensitivity probably affected by modifiers. A male sterility factor seems to be associated but may be separable. Viability of homozygote about 70% that of *es/SMI* and remains low in strains selected for less sensitivity. Not sensitive to carbon dioxide. RK3.

Es: see *en*^{Es}

esc: extra sex combs

location: 2-{47}.

discoverer: Slifer, 40e2.

references: 1942, J. Exp. Zool. 90: 31-40 (fig.).

Struhl, 1981, Nature (London) 293: 36-41 (fig.).

phenotype: Distal portions of second and third legs weakly transformed into first leg and wing blade into haltere; transformation partial and variable in hypomorphic allele, *esc*¹. Sex combs may be present on all six legs of male; at least one extra sex comb present in majority of males. Expression affected by culture conditions. When expressivity high, extra transverse bristle rows appear between sixth and eighth longitudinal rows of bristles, mainly on distal portion of basitarsus and tibia of second and third legs in both sexes; accompanied by shortening of affected

leg segments. Sex comb development autonomous in mosaics produced by somatic crossing over [Tokunaga and Stern, 1965, *Dev. Biol.* 11: 50-81 (fig.)]. For interactions with *Pc* and *Scx* see Hannah-Alava [1958, *Genetics* 43: 878-905 (fig.)]. Leg effects autonomous in homozygous clones of hypomorphic or amorphic alleles. Studies with amorphic alleles demonstrate that *esc* offspring of *esc*/+ mothers express the leg transformation, but *esc* zygotes produced by *esc* mothers die as newly hatched larvae that exhibit drastic homeotic transformations. All abdominal and thoracic and some head segments are transformed into eighth abdominal segments. The effects of the maternal insufficiency partially overcome by the presence in the zygote of a paternally derived *esc*⁺ allele, more so by two paternally derived *esc*⁺ alleles. Larvae with one paternal *esc*⁺ range between normal and transformed phenotype; those with two frequently develop into adults, more than half of which show patchy transformations in abdominal and thoracic segments to more posterior segments. Temperature-shift experiments with the temperature-sensitive allele, *esc*¹⁷ (i.e., on the *esc/esc* progeny of *esc*¹⁷/*esc* mothers when crossed to *esc* fathers) indicate that *esc*⁺ product is required only around the time of gastrulation, as that is the only temperature-sensitive period (Struhl and Brower, 1982, *Cell* 31: 285-92). In the presence of extreme *BXC* deficiencies (including *abdA*⁻, *AbdB*⁻, and *abdA*⁻ *AbdB*⁻) which transform the eighth abdominal segment to more anterior segments, *esc*⁺ product insufficiency in the zygote causes all segments to develop as the eighth abdominal segment would develop under the influence of the *BXC* genotype in the presence of *esc*⁺ product (Struhl, 1981; Struhl and White, 1985, *Cell* 43: 507-19). In the *esc*⁻ embryo produced by *esc*²/*esc*¹⁰ parents, *Ubx* transcription is normal early in development, but following gastrulation transcripts appear in ectodermal and mesodermal derivatives of all fourteen parasegments rather than being restricted to parasegments 6-12 as in wild-type (Struhl and Akam, 1985, *EMBO J.* 4: 3259-64). *esc*⁻ embryos display reduced levels of *Antp* protein to levels below those of wild-type in all segments of the nervous system (Carroll, Laymon, McCutcheon, Riley, and Scott, 1986, *Cell* 47: 113-22).

alleles:

alleles	origin	discoverer	synonym	ref ^α	comments
<i>esc</i> ¹	spont	Slifer, 40e2		1, 3	hypomorph
<i>esc</i> ²	spont	Strömnaes, 53f	<i>esc</i> ^D	1, 2	<i>In(2L)t</i>
<i>esc</i> ³	spont	Struhl		4	amorph in <i>CyO</i>
<i>esc</i> ⁴		Struhl		4	amorph
<i>esc</i> ⁵		Struhl		4	amorph
<i>esc</i> ⁶		Struhl		4	amorph
<i>esc</i> ⁷		Struhl		4	amorph
<i>esc</i> ⁸		Struhl		4	amorph
<i>esc</i> ⁹		Struhl		4	amorph
<i>esc</i> ¹⁰		Struhl		4	380 kb deletion
<i>esc</i> ¹¹	EMS	Struhl		5	
<i>esc</i> ¹²	EMS	Struhl		5	
<i>esc</i> ¹³	EMS	Struhl		5	
<i>esc</i> ¹⁴	EMS	Struhl		5	
<i>esc</i> ¹⁵	EMS	Struhl		5	
<i>esc</i> ¹⁶	EMS	Struhl		5	
<i>esc</i> ¹⁷	EMS	Struhl	<i>esc</i> ^{ts}	5	temperature-sensitive
<i>esc</i> ¹⁸	EMS	Struhl		5	
<i>esc</i> ¹⁹	EMS	Struhl		5	

alleles	origin	discoverer	synonym	ref ^α	comments
<i>esc</i> ²⁰	EMS	Struhl		5	
<i>esc</i> ²¹	EMS	Struhl		5	

^α 1 = CP627; 2 = Hannah and Strömnaes, 1955, *DIS* 29: 121-23; 3 = Slifer, 1942, *J. Exp. Zool.* 90: 31-40; 4 = Struhl, 1981, *Nature* (London) 293: 36-41; 5 = Struhl and Brower, 1982, *Cell* 31: 285-92.

^β Erroneously designated a dominant allele in CP627.

cytology: Placed in 33B1-2 based on *Df(2L)esc10*, a 380-bp deletion (coordinates -315 to +65) deficient for those two bands (G. Richards).

molecular biology: Region cloned by walking from sequences cloned from microdissected chromosomes (Frei, Baumgartner, Edström and Noll, 1985, *EMBO J.* 4: 979-87). Transformation experiments restrict locus to a 12-kb fragment from around coordinate -70 kb (0 = left end of a 44-kb *EcoRI* fragment from the walk; negative values to the left); the 12-kb fragment detects a single transcript of 1.8-kb, which is most abundant in follicles and 0-4 hr embryos, but is drastically reduced in later stages. Genomic sequences homologous to the transcript lie entirely within the segment from -67.8 to -64.6 kb. cDNA's identify the 3' end and a 364-base-pair intron, but not the 5' end of the transcript; transcription from right to left (Frei, Bopp, Burri, Baumgartner, Edström and Noll, 1985, *Cold Spring Harbor Symp. Quant. Biol.* 50: 127-34).

Esm: Enhancer-of-split mimic

location: 3-{81}.

references: Knust, Dietrich, Weigel, Tepass, Vässin, Bremer, and Campos-Ortega, 1987, *J. Neurogenet.* 4: 145-46).

cytology: Tentatively placed in 95F.

molecular biology: May carry *EGF*-like repeats.

Est-6: Esterase 6

location: 3-35.9 [based on 334 recombinants between *h* and *th* (Franklin, 1971, *DIS* 47: 113)].

discoverer: Wright, 61h.

synonym: *Est-D*.

references: 1963, *DIS* 37: 53.

1963, *Genetics* 48: 787-801 (fig.).

phenotype: The structural gene for the nonspecific carboxylesterase, [EST-6 (EC 3.1.1.1)]. One of the ten positively migrating esterases demonstrable with α -naphthyl acetate and Fast Blue BB after starch gel electrophoresis of homogenates. Molecular weight determinations on purified enzyme indicate that the active enzyme is a 62,000 to 65,000 dalton glycoprotein monomer (Mane, Tepper, and Richmond, 1983, *Biochem. Genet.* 21: 1019-40). Substrate specificities to different esters explored by Danford and Beardmore (1979). Specific EST-6 activity during development shows a prominent transient peak during second larval instar and shows a male-specific rise in activity beginning 36-48 hr after eclosion; males reach level double that of females (Sheehan, Richmond, and Cochrane, 1979, *Insect Biochem.* 9: 443-50); similar high levels found in male-like triploid intersexes (Aronsham and Kuzin, 1974, *Zh. Obsch.* 35: 926-33) and *X/X;tra/tra* (Johnson, 1964, *Genetics* 50: 259). High concentrations of EST-6 found in ejaculatory duct; at mating, male level depleted and female level enhanced; transfer of activity from male to

*Est-6*⁰ female detected early during copulation, prior to the transfer of sperm, indicating that *EST-6* is a component of seminal fluid (Richmond, Gilbert, Sheehan, Gromko, and Butterworth, 1980, *Science* 207: 1483-85). Enzyme levels in flies carrying different X chromosomes from natural populations in combination with constant autosomal complement vary suggesting X-linked modifiers (Tepper, Richmond, and Terry, 1981, *Genetics* 97: s103). In *D. melanogaster* X *D. simulans* hybrid males that carry a *D. melanogaster* X chromosome, the level of the *D. melanogaster* enzyme is reduced compared to that of the *D. simulans* enzyme; hybrid females have equivalent activities of the two enzymes (Korchkin, Aronshtam, and Matveeva, 1974, *Biochem. Genet.* 12: 9-24). Electrophoretic mobility reduced modestly from 1.10 to 1.08 for *Est-6*^F and from 1.0 to 0.98 for *Est-6*^S by *m(Est-6)* on the third chromosome (Cochrane and Richmond, 1979, *Biochem. Genet.* 17: 167-83).

alleles: Allelic variation in *Est-6* is expressed in electrophoretic mobility (complicated by heterogeneity for *m(Est-6)* on chromosome 3), thermal stability, and level of activity. The same alleles have been designated differently by different authors. The accompanying table attempts to define the equivalences. Some use electrophoretic mobility relative to that of *Est-6*^S = 1.00 in designating alleles. Relative mobility estimates vary within and among gels; also relations between mobilities on starch and acrylamide are nonlinear; consequently, similar but not identical estimates may not represent real differences.

allele	possible equivalence	relative mobility	thermal denaturation constant	ref ^α
<i>Est-6</i> ^{0β}				3
<i>Est-6</i> ^{0.73}		0.73		5
<i>Est-6</i> ^{0.79}		0.79		5
<i>Est-6</i> ^{0.95}	<i>Est-6</i> ^S <i>m(Est-6)</i>	0.95		2
<i>Est-6</i> ^{0.97}	<i>Est-6</i> ^S <i>m(Est-6)</i>	0.97		4
<i>Est-6</i> ^{1.07}	<i>Est-6</i> ^F <i>m(Est-6)</i>	1.07		4
<i>Est-6</i> ^{1.11}	<i>Est-6</i> ^F	1.11		4
<i>Est-6</i> ^{1.15}		1.15		1
<i>Est-6</i> ^{1.25}		1.25		2
<i>Est-6</i> ^{F1}		1.10	-0.0458	1
<i>Est-6</i> ^{F2}		1.10	-0.0811	1
<i>Est-6</i> ^{F3a}		1.10	-0.0959	1
<i>Est-6</i> ^{F36}		1.10	-0.123	1
<i>Est-6</i> ^{F4}		1.10	-0.158	1
<i>Est-6</i> ^{S1}		1.0	-0.0245	1
<i>Est-6</i> ^{S2}		1.0	-0.0615	1
<i>Est-6</i> ^{S3}		1.0	-0.0727	1
<i>Est-6</i> ^{Sloγ}		1.0?		6
<i>Est-6</i> ^{VF}		1.21		4
<i>Est-6</i> ^{VS}		0.85		4

^α 1 = Cochrane and Richmond, 1979, *Genetics* 93: 461-78; 2 = Franklin, 1971, *DIS* 47: 113; 3 = Johnson, 1964, *J. Hered.* 55: 76-8; 4 = Rodinó and Danieli, 1972, *DIS* 48: 77; 5 = Triantaphyllidis and Christodoulou, 1973, *Biochem. Genet.* 8: 383-90; 6 = Trippa, Scozzari, Costa, and Danieli, 1979, *Egypt. J. Genet.* 8: 295-302.

^β Contains insert of 412.

^γ Requires 3-4 times normal incubation time to detect activity.

cytology: Placed in 69A1-5 based on its inclusion within *Df(3L)vin-5 = Df(3L)68A3;69A1-2*, but not *Df(3L)vin-6 = Df(3L)68C8-11;69A4-5* (Akam, Roberts, Richards, and Ashburner, 1978, *Cell* 13: 215-25). Further localized to 69A by *in situ* hybridization (Oakeshott, Collet, Phillis, Nielsen, Russell, Chambers, Ross, and Richmond, 1987, *Proc. Nat. Acad. Sci. USA* 84: 3359-63).

molecular biology: Gene isolated by screening a cDNA library with oligonucleotide probes based on tryptic-peptide amino-acid sequences (Oakeshott *et al.*). The 1.85-kb cDNA clone sequence is contained within a 2.3-kb *EcoRI-BamHI* genomic fragment. One strand of the cDNA clone hybridizes to mRNA's of 1.68 and 1.83 kb. Developmental profiles of transcripts concordant with that of *EST-6*; transcripts absent in *Est-6*⁰ adults. Conceptual amino acid sequence of larger transcript suggests a signal peptide of 21 amino acids followed by 527 residues of the mature protein (59,380 daltons); potential glycosylation sites at residues -2, 21, 399, 435, and 485. Contains an eight-residue sequence with strong similarity to the consensus of active sites of nine other eukaryotic esterases.

Est-9

location: 2- (probably right arm based on apparent homology to and position of *Est-9* in *Drosophila pseudoobscura*).

references: Loukas, 1981, *DIS* 56: 85.

phenotype: The apparent structural gene of an esterase whose detection depends on the presence of 1-leucyl-β-naphthylamide in addition to α-naphthyl acetate as substrate.

alleles: *Est-9*^F and *Est-9*^S detected.

Est-A: Esterase A

location: Not located.

references: Mizianty and Case, 1972, *J. Heredity* 62: 345-47.

phenotype: The most anodally migrating esterase band; migrates considerably in advance of *EST-C*. Activity weak in standard gel system.

alleles: *Est-A* inferred from absence of band from Swedish-C wild stock; *EST-A* band present in Oregon-R. Presence and absence segregate as a single genetic character.

Est-C

location: 3-47.7 [to the left of *p* (Ohnishi and Voelker, 1979, *Jpn. J. Genet.* 54: 203-209); 0.006 from *Odh* (Mukai and Voelker, 1977, *Genetics* 86: 175-85)].

references: Beckman and Johnson, 1964, *Hereditas* 51: 212-20 (fig.).

phenotype: Specifies *EST-C* (EC 3.1.1.1), the next-to-most rapidly migrating of six anodally migrating esterases detectable with α-naphthyl acetate and Fast Blue BB following starch gel electrophoresis. Enzyme a monomer. Putative null alleles homozygous viable, consistent with failure to find lethal complementation group within the interval.

alleles: Selected from natural populations. Since they were isolated from the same population, either *Est-C*^{nNC1} and *Est-C*^{nNC4}, or *Est-C*^{nNC2} and *Est-C*^{nNC3}, or *Est-C*^{nGB1} and *Est-C*^{nGB2} could represent reisolates of the same allele.

cytology: Placed in 84D3-5 on the basis of its position between *Df(3R)Scx4 = Df(3R)84B1-2;84D3-4* and *Df(3R)D7 = Df(3R)84D3-5;84F1-2* (Cavener, Otterson, and Kaufman, 1986, *Genetics* 114: 111-23).

allele	synonym	ref ^α	relative mobility	relative activity
<i>Est-C</i> ^F	<i>Est-C</i> ⁶	1, 2	1.00	

allele	synonym	ref α	relative mobility	relative activity
<i>Est-C^{nNC1}</i>		3		null
<i>Est-C^{NC2}</i>		3	0.91?	low
<i>Est-C^{NC3}</i>		3	0.91?	low
<i>Est-C^{nNC4}</i>		3		null
<i>Est-C^{nGB1}</i>		3		null
<i>Est-C^{nGB2}</i>		3		null
<i>Est-C^S</i>	<i>Est-C⁴</i>	1, 2	0.91	
<i>Est-C^{VF}</i>	<i>Est-C⁸</i>	2, 4	1.07	
<i>Est-C^{VS}</i>	<i>Est-C²</i>	2		

α 1 = Beckman and Johnson, 1964, *Hereditas* 51: 212-20 (fig.); 2 = Johnson and Schaeffer, 1973, *Biochem. Genet.* 10: 149-63; 3 = Langley, Voelker, Leigh Brown, Ohnishi, Dickson, and Montgomery, 1982, *Genetics* 99: 151-56; 4 = Triantaphyllidis and Christadoulou, 1973, *Biochem. Genet.* 8: 383-90.

Est-D: see *Est-6*

Etd: Eye tissue determiner

location: 1-23 (approximate).

origin: Spontaneous.

references: Gethman, 1971, *Mol. Gen. Genet.* 114: 144-55.

phenotype: Removes lower half of eye.

Eth: Ether resistant (J.C. Hall)

location: 3-61.

origin: Strain produced by 29 generations of selection.

references: Ogaki, Nakashima-Tananka, and Murakami, 1967, *Jpn. J. Genet.* 42: 387-94.

Gamo, Ogaki, and Nakashima-Tanaka, 1979, *Jpn. J. Genet.* 54: 229-34.

Gamo, Nakashima-Tanaka, and Ogaki, 1980, *Jpn. J. Genet.* 55: 133-40.

phenotype: The major determinant in a selected strain that is more resistant than usual to killing effects of ether vapors administered to adults; resistance dominant to sensitivity; minor factors on chromosomes 1 and 4; *Eth*-bearing strain also relatively resistant to killing effects of chloroform or halothane; these phenotypes are dominant and semidominant, respectively; chloroform resistance due to major factor on chromosome 1 and a minor one on chromosome 2; halothane resistance caused by major factor on third chromosome (not known if same locus as that responsible for most of the ether resistance), and minor components on chromosomes 1 and 2; halothane resistance also seen with respect to anesthesia, and genetic difference for this maps to chromosome 1.

ether-a-go-go: see *eag*

ether sensitive: see *es*

Etre: see *Fs(2)Sz4*

Ets2: Ets-2 proto-oncogene homologue

location: 2-{100}.

references: Pribyl, Watson, McWilliams, Ascione, and Papas, 1988, *Dev. Biol.* 127: 45-53.

phenotype: Encodes a *Drosophila* homologue to the *ets* gene from the avian erythroblastosis retrovirus E26. mRNA constitutively present in all developmental stages, with elevated levels of expression in 0-9-hr embryos and pupae.

cytology: Placed in 58A-B by *in situ* hybridization.

molecular biology: Genomic clone isolated using restriction fragment from a *Drosophila Ets* clone previously

derived from Schneider cells. The sequence shows homology to the last two exons of chicken *ets* or mammalian *ets-2* (as does *v-ets* from E26) with an intron of 63 base pairs separating the two exons; the intron is smaller in size, but identical in position to that of *ets* genes in other species. *Ets* sequences detect a single 4.7-kb mRNA on Northern blots. Between *Drosophila Ets* and *v-ets*, there is 71% homology at the nucleotide level and 91% at the predicted amino-acid level; these figures with respect to human *ets-2* are 76% and 94%.

eve: even skipped

location: 2-58.

references: Nüsslein-Volhard and Wieschaus, 1980, *Nature (London)* 287: 795-801 (fig.).

Nüsslein-Volhard, Wieschaus, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 267-82 (fig.).

Nüsslein-Volhard, Kluding, and Jürgens, 1985, *Cold Spring Harbor Symp. Quant. Biol.* 50: 145-54 (fig.).

phenotype: Homozygous lethal; embryos homozygous for a null allele or a deficiency fail to undergo segmentation and their ventral surfaces are covered by a lawn of short denticles pointed toward the midline; denticles in the anterior region show thoracic characteristics suggesting that segmental identities persist in the absence of segmentation. All derivatives of gnathal segments are missing, such as maxillary sense organs, cirri, mouth hooks, labial sense organs and the mandibular parts of the cephalopharyngeal skeleton; the labrum, antennal sense organs, and a rudimentary skeleton are the only remains of the larval head. Posteriorly, anal plates and some sensory organs persist as do remnants of spiracles, filzkörper, and tufts. Homozygotes and hemizygotes for hypomorphic alleles display pair-rule segmentation defects. Denticle bands and adjacent naked cuticle of the prothoracic, metathoracic and even-numbered abdominal segments removed; some naked cuticle of the adjacent segment removed as well (i.e., the odd numbered parasegments are removed). Combinations of alleles with *Df(2R)eve* raised at different temperatures can achieve an array of phenotypes between these extremes. Expression of *eve* is first detected at the eleventh nuclear division following fertilization; at this stage, *eve* protein is uniformly distributed among the nuclei, both at the periphery and deep within the egg; by the thirteenth nuclear division, the anterior one-third of the embryo is devoid of detectable protein; over the next 20 minutes, the antibody staining in the posterior two-thirds of the embryo becomes concentrated in seven transverse stripes four or five cells wide separated by stripes three to four cells wide with lower levels of staining. By the time of germ-band elongation, the seven stripes have become narrowed and sharply defined and seven new weakly expressing stripes, one to two cells wide, appear between the major stripes; during germ-band elongation all stripes gradually disappear. As *eve* stripes become more sharply defined so too do *ftz* stripes, no longer overlapping *eve* stripes, but forming a complementary pattern. At the same time, a group of expressing cells appears at the posterior end of the germ band; these cells form a ring around the anal plate during germ-band shortening. Also during germ-band shortening, a specific subset of sixteen neurons in each hemisegment of the CNS expresses *eve* product as does a row of cell clusters on either side of the

dorsal midline; lateral to these clusters are curious rings of weakly staining cells; the dorsal cells do not appear to be neuronal (Frasch, Hoey, Rushlow, Doyle, and Levine, 1987, *EMBO J.* 6: 749-59; Frasnch and Levine, 1987, *Genes Dev.* 1: 981-95). In homozygous *eve*¹ embryos switched to restrictive temperature during neurogenesis, four specifically studied *eve*-expressing neurons in each hemisegment are found to persist; two of them develop normally, but two send axonal processes to abnormal destinations [Doe, Smouse, and Goodman, 1988, *Nature (London)* 333: 376-78]. Frasnch and Levine observe that segmentation-gene-mutations generally have reciprocal effects on the expression of *eve* and *ftz*, leading them to postulate that their promoters respond reciprocally to the same positional cues. *eve* concluded to be an early pair-rule gene, since its expression is modified by gap-gene mutations, but not by most other pair-rule gene mutations nor by segment-polarity gene mutations. Three pair-rule genes do influence *eve* expression: in either *eve* or *h* genotypes, *eve* expression is reduced and in *run* embryos *eve* is overexpressed (Frasch and Levine, 1987). In *eve* embryos, *en* stripes do not appear (Macdonald, Ingham, and Struhl, 1986, *Cell* 47: 721-34). *Ubx* protein is detected at high level in odd-numbered parasegments from 7 through 13 rather than in every parasegment from 6 through 12 (Martinez-Arias, and White, 1988, *Development* 102: 325-38). *ftz* stripes are disrupted in regularity of position, size, and timing (Carroll and Scott, 1986, *Cell* 45: 113-26).

alleles:

allele	origin	synonym	ref ^α	comments
<i>eve</i> ¹	EMS	ID19	1, 2, 3, 5	temperature-sensitive allele; 121 arg → his
<i>eve</i> ²	EMS	IIR59	1, 2, 3, 5	weak allele; 75 thr → ile
<i>eve</i> ³	EMS	R13	1, 4, 5	strong allele; → no <i>eve</i> protein
<i>eve</i> ⁴	X ray	3.77.17	1, 5	intermediate allele; molecular deletion → frame shift → 134 C-terminal residues replaced by 79 extraneous ones

^α 1 = Frasnch, Warrior, Tugwood, and Levine, 1988, *Genes Dev.* 2: 1824-38; 2 = Nüsslein-Volhard and Wieschaus, 1980, *Nature (London)* 287: 795-801 (fig.); 3 = Nüsslein-Volhard, Wieschaus, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 267-82 (fig.); 4 = Nüsslein-Volhard, Kluding, and Jürgens, 1985, *Cold Spring Harbor Symp. Quant. Biol.* 50: 145-54 (fig.); 5 = Tearle and Nüsslein-Volhard, 1987, *DIS* 66: 209-26.

cytology: Placed in 46C3-11 based on *Df(2R)eve* = *Df(2R)46C3-4;46C9-11*.

molecular biology: Gene isolated based on homology to known homeobox sequences (Macdonald *et al.*, 1986; Frasnch *et al.*, 1987). Probing Northern blots with the genomic sequence identifies a single RNA species of 1.4 kb whose expression is strong during early embryogenesis, persisting at declining levels until first instar. Sequence data reveals the presence of a 71-bp intron between the initiating AUG and the homeobox; the conceptual amino-acid sequence defines a basic 40-kd polypeptide of 376 amino acids with a 60-amino acid homeobox domain extending from residue 70 to 130; the homeobox sequence shows only about 50% identity with those of previously sequenced homeoboxes. Repeats of the trinucleotide GCX are found in positions 564-608 producing a polyalanine sequence, as is also seen in *en*,

N, and *cad*. Promoter fusion experiments utilizing a *LacZ* reporter gene identify promoter elements for stripe 1 (tentative), stripe 3, and stripes 2 and 7 within 8 kb of upstream sequence; in addition a target for an autoregulatory function of *eve* protein identified in this region (Goto, Macdonald, and Maniatis, 1989, *Cell* 57: 413-22; Harding, Hoey, Warrior, and Levine, 1989, *EMBO J.* 8: 1205-12). *eve* protein shown to repress *Ubx* transcription *in vitro* (Biggin and Tjian, 1988, *Cell* 58: 433-40); in DNA footprint studies *eve* protein can be shown to bind to specific sequences 5' to both *en* and *eve*, but the sequences in the two genes do not appear to have features in common [Hoey and Levine, 1988, *Nature (London)* 332: 858-61].

ewg: erect wing (J.C. Hall)

location: 1-0.0.

references: Deak, 1978, *Dev. Biol.* 66: 422-41.

Fleming, Zusman, and White, 1983, *Dev. Genet.* 3: 347-63.

Fleming, DeSimone, and White, 1989, *Mol. Cell Biol.* 9: 719-25.

phenotype: Viable allele causes wings to be held upright; wing posture phenotype shows incomplete penetrance. Dorso-ventral flight muscles often absent, especially when the mutation is heterozygous with a deficiency. Ultrastructure of tergal depressor of trochanter jump muscle normal. Flies hemizygous for lethal alleles die either just prior to hatching of the larva from the egg or immediately thereafter; mosaic analysis of certain lethal alleles suggests primary defect in developing muscles; however, *in situ* studies of transcription reveal that expression is confined to the nervous system and not the muscles (Fleming *et al.*, 1989).

alleles: Survival of lethal alleles in combination with *ewg*¹ variable and temperature-sensitive *ewg*¹¹ complements at least partially *ewg*⁴, *ewg*⁷, *ewg*¹⁰, and *ewg*¹².

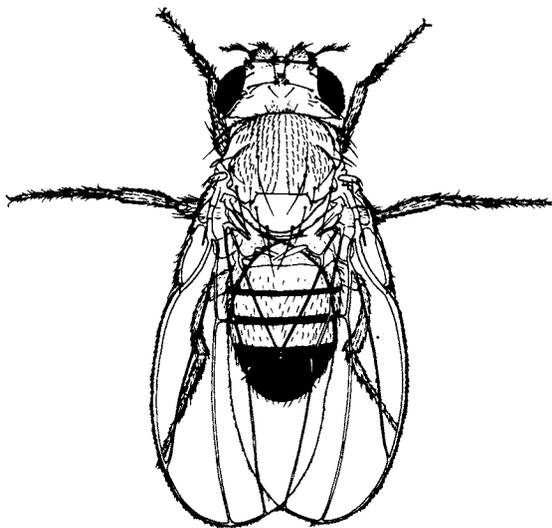
allele	origin	discoverer	synonym	ref ^α	comments
<i>ewg</i> ¹	EMS	Hanratty			viable allele
<i>ewg</i> ²	EMS	White	<i>ewg</i> ¹¹	1, 2	late embryonic lethal
<i>ewg</i> ³	EMS	White	<i>ewg</i> ¹²	1, 2	first-instar lethal
<i>ewg</i> ⁴	EMS	White	<i>ewg</i> ¹³	1, 2	late embryonic lethal
<i>ewg</i> ⁵	X ray	Lim	<i>l(1)EC3</i> , <i>ewg</i> ¹⁴	1, 2, 3	
<i>ewg</i> ⁶	EMS	Lim	<i>l(1)EC3</i> ⁶³		
<i>ewg</i> ⁷	EMS	Lim	<i>l(1)EC3</i> ⁹²		
<i>ewg</i> ⁸	EMS	Lim	<i>l(1)EC3</i> ¹¹²		
<i>ewg</i> ⁹	EMS	Lim	<i>l(1)EC3</i> ¹¹⁸		
<i>ewg</i> ¹⁰	EMS	White	<i>ewg</i> ¹⁵	1, 2	late embryonic lethal
<i>ewg</i> ¹¹	EMS	White	<i>ewg</i> ¹⁶	1, 2	
<i>ewg</i> ¹²	EMS	White	<i>ewg</i> ¹⁷	1, 2	late embryo, early larval lethal
<i>ewg</i> ¹³	X ray	Lefevre	<i>l(1)A58</i>	3	
<i>ewg</i> ¹⁴	EMS	Lefevre	<i>l(1)DA659</i>	4	
<i>ewg</i> ¹⁵	EMS	Lefevre	<i>l(1)DA678</i>	4	
<i>ewg</i> ¹⁶	EMS	Lefevre	<i>l(1)DA687</i>	4	y mutant
* <i>ewg</i> ¹⁷	EMS	Lefevre	<i>l(1)DC729</i>	4	
* <i>ewg</i> ¹⁸	EMS	Lefevre	<i>l(1)VA270</i>	4	
<i>ewg</i> ¹⁹	EMS	Fleming	<i>ewg</i> ¹¹⁸	2	viable hypoactive, flightless hypomorph; indirect flight muscles intact

^α 1 = Fleming, Zusman, and White, 1983, *Dev. Genet.* 3: 347-63; 2 = Fleming, DeSimone, and White, 1989, *Mol. Cell Biol.* 9: 719-25; 3 = Lefevre, 1981, *Genetics* 99: 461-80; 4 = Lefevre and Watkins, 1986, *Genetics* 113: 869-95.

cytology: Placed in 1A7-8 based on inclusion in *Df(1)SJ1a* = *Df(1)1A1-3;1A8-B1* but not *Df(1)SJ1d* = *Df(1)1A1-*

3;1A6-B1. To the right of *cin*, which is in the same interval and separated from *ewg* by the breakpoint of *T(1;4)cin*.

molecular biology: Region cloned by an extension of the chromosome walk of Campuzano *et al.* (Cell 40: 327-38). Gene localized to a 9.5-kb segment at approximately 140 to 150 kb on the molecular map by means of germ-line-transformant rescue of lethal alleles of *ewg*. Probing developmental Northern blots with this sequence reveals a collection of at least five transcripts ranging in size from 3.7 to 6 kb; transcription is most pronounced in six-to-twelve hour embryos with subsets of these transcripts appearing in 0-6 hr embryos and in adults (Fleming, DeSimone, and White, 1989, Mol. Cell Biol. 9: 719-25).



ex: expanded

From Stern and Bridges, 1926, Genetics 11: 503-30.

ex: expanded

location: 2-0.1.

origin: Spontaneous.

discoverer: Bridges, 17k21.

references: Stern and Bridges, 1926, Genetics 11: 514 (fig.).

phenotype: Wings extremely wide and large, sometimes curved and divergent. Effect produced in prepupal wing, probably by influence on cell division (Waddington, 1940, J. Genet. 41: 75-139). Eyes slightly reduced in size and roughish. Body large. RK2.

alleles: **ex*² (Mickey, 1950, DIS 24: 60).

cytology: Salivary chromosome location in or near 21C3 (Lewis, 1945, Genetics 30: 137-66). Between 21C2 and 8 by deficiency analysis [Golubovsky, 1979, Genetika 14: 294-305 (fig.)].

Exaggeration of Bar: see *E(B)*

exb: extreme bar

location: 3-47.4.

origin: Occurred with *TE#68*.

discoverer: Ising.

Phenotype: Very narrow eyes (smaller than *B/B*); no ocelli.

exd: extradenticle

location: 1-54.

origin: Induced by ethyl methanesulfonate.

references: Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307. Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Homozygous lethal; meso- and metathoracic segments resemble prothorax; first abdominal like posterior abdominal segments. In combination with *Pc*-like mutants shows abdominal transformations.

alleles: Two. *exd*¹ and *exd*², isolated as *XP* and *YO*.

cytology: Placed in 14A1-B1 based on inclusion in the region of overlap of *Df(1)sd72b = Df(1)13F1;14B1* and *Dp(1;4)r⁺ = Dp(1;4)14A1-2; 16A1-2; 102F2-3*.

***exi: exiguous**

location: 1-51.5.

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1956.

references: 1958, DIS 32: 70.

phenotype: Small fly with rather dusky body color. Not easily classified. Viability and fertility good in male, slightly reduced in female. RK3.

exo: exocephalon

location: 1-58.

origin: Induced by ethyl methanesulfonate.

references: Eberl and Hilliker, 1988, Genetics 118: 109-20.

phenotype: Sex-linked recessive lethal; variably differentiated cuticle; complete failure of head involution with well developed head structures on exterior of the embryo. Many embryos slightly twisted with defective ventral denticle patterns.

alleles: *exo*¹ and *exo*² isolated as *l(1)EH354* and *l(1)EH406*.

cytology: Placed in 16C2-18B11 between breakpoints of *Dp(1;3)j⁺71b = Dp(1;3)15A4;16C2-3;80-81* and *T(1;Y)B50 = T(1;Y)18B4-11*.

expanded: see *ex*

***exr: extra venation**

location: 1- (associated with *In(1)exr*).

origin: Induced by triethylenemelamine (CB. 1246).

discoverer: Fahmy, 1952.

references: 1958, DIS 32: 70.

phenotype: Eyes slightly rough and smaller than normal. Wings have irregularly distributed extra vein tissue. Males viable and fertile; females viable but sterile. RK3A.

cytology: Associated with *In(1)exr = In(1)12E8-10;15D1-3*.

***ext: extended**

location: 2- (not located).

origin: Spontaneous.

discoverer: Ströher, 1958.

references: Mainx, 1958, DIS 32: 82.

phenotype: Wings held out at about a 75° angle from body axis, are wavy, and gradually curve downward. Distal parts of wings often crumpled and folded. Halteres normal. Function of wings reduced. Viability and fertility good. RK3.

Ext: Extras

location: 1-15.2.

discoverer: Schultz, 3318.

phenotype: Heterozygous female has thickened, branched, and extra veins. Overlaps wild type. Lethal in male. RK3.

cytology: Tentatively placed in 7C-E on the basis of *Ext* phenotype of segmental deficiency (Merriam and colleagues).

ext-b: see *bat*

Ext-sct-3: see *Su(sc)*

extended: see *ext*

extended-b: see *bat*

extra bristles: see *eb*

Extra combs: see *Eco*

extra eye: see *ee*

extra lamina fiber: see *elf*

extra macrochaetae: see *emc*

extra organs: see *eo*

Extra sex comb: see *Scx*

extra sex combs: see *esc*

extra wing hairs: see *xwh*

extra venation: see *exr*

extradenticle: see *exd*

Extras: see *Ext*

extreme bar: see *exb*

exu: exuperantia (T. Schüpbach)

location: 2-93.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 302-17.

Mlodzik, DeMontrion, Hiromi, Kraus, and Gehring, 1987, Genes Dev. 1: 603-14.

Fronhöfer and Nüsslein-Volhard, 1987, Genes Dev. 1: 880-90.

Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

phenotype: Maternal-effect lethal mutant; embryos from homozygous mothers lack anterior-most head structures. Instead they form an inverted posterior midgut and proctodeal region at their anterior end. Cephalic furrow at gastrulation is shifted towards anterior. Analysis of germline clones indicates that the mutation is germline autonomous (Schüpbach and Wieschaus, 1986, Dev. Biol. 113: 443-48). Causes shift in blastoderm fate map as indicated by *ftz* expression; thoracic stripes broadened and shifted anteriorly; abdominal stripes narrowed and compressed posteriorly (Mlodzik *et al.*). Eggs produced by *exu* mothers appear to have a more uniform distribution of *bcd*⁺ product, i.e., less concentration anteriorly (Fronhöfer and Nüsslein-Volhard).

cytology: Placed in 56F-57B by segmental aneuploidy (Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26).

alleles: *exu*¹ - *exu*⁴ isolated as *PJ*, *QR*, *SB*, and *SC*, respectively.

ey: eyeless

location: 4-2.0 (located by recombination in diplo-4 triploids by Sturtevant, 1951, Proc. Nat. Acad. Sci. USA 37: 405-7).

origin: Spontaneous.

discoverer: Hoge, 14e.

references: 1915, Am. Naturalist 49: 47-49.

Bridges, 1935, Biol. Zh. 4: 401-20 (fig.).

phenotype: Eye size variably reduced depending on allele (see table); expressivity more variable for some alleles than for others. Tetragonal packing of facets and face-centered tetragonal bristle lattice (*ey*^R) in place of hexagonal array of wild type [Hartman and Hayes, 1971, J. Hered. 62: 41-43 (fig.)]; associated with a failure of the horizontal secondary pigment cell to expand to give rise to the horizontal boundaries between ommatidia [Ready, Hanson, and Benzer, 1976, Dev. Biol. 53: 217-40 (fig.)]. Some *ey*² flies show duplications of antennae or antennal segments with or without duplication of arista; extra maxillary structures also observed (Shatouri, 1963, Caryologia 16: 431-37). Optical disks reduced in size [(*ey*¹) Richards and Farrow, 1922, Proc. Oklahoma Acad. Sci. 2: 41-45; (*ey*²) Medvedev, 1935, Z. Indukt. Abstamm. Vererbungslehre 70: 55-72 (fig.); 1935, Tr. Inst. Genet. Akad. Nauk SSSR 10: 119-51; Steinberg, 1944, Proc. Nat. Acad. Sci. USA 30: 5-13; (*ey*⁴) Chen, 1929, J. Morphol. 47: 135-99]. Degenerating cells abundantly observed in the optic disks of third-instar larvae of *ey*² [Fristrom, 1969, Mol. Gen. Genet. 103: 363-79 (fig.); Ransom, 1979, J. Embryol. Exp. Morphol. 53: 225-35]. Expressivity sensitive to genetic background [(*ey*⁴) Spofford, 1956, Genetics 41: 938-59; (*ey*¹, *ey*², *ey*⁴, *ey*^K) Hunt and Burnet, 1969, Genet. Res. 13: 251-65]. Phenotype also responds to developmental temperature, larval density, and composition of medium. Eye size reported to increase with increased temperature in *e*¹ (Baron, 1935, J. Exp. Zool. 70: 461-90) and *ey*^K (Sang and Burnet, 1963, Genetics 48: 1683-99) but to decrease in *ey*^W (Meyer, 1959, DIS 33: 97). Phenotype less extreme in flies raised under crowded conditions at 18° but not 25° (Sang and Burnet, 1963; see also Chester, 1971, DIS 46: 62-63). Eye size of four alleles increased by cholesterol deprivation and decreased by dietary deficiencies in thiamine or RNA (Hunt and Burnet, 1969). Larval feeding of lactamide to *ey*² causes decreased eye size, which is of opposite sign from its effect on *B* (Grant and Rapport, 1980, DIS 55: 53); no such effect of lactamide on *ey*^K observed by Sang and Burnet (1963). *ey*² flies exhibit normal visual orientation in Y maze (Bülthoff, 1982, DIS 58: 31). *ey*², *ey*⁴, and *ey*^K in combination with *eyg* (3-35.5) results in almost complete curtailment of eye development and synthetic lethality, with the major lethal crisis at the end of the pupal stage and a minor lethal phase at pupation; rare surviving adults have brain in anterior thorax [Hunt, 1970, Genet. Res. 15: 29-34 (fig.)].

alleles: *ey*² is the most frequently used allele. Four lethal alleles, formerly *l(4)10* = *l(4)33*, assigned to the *ey* locus by Hochman (1971, Genetics 62: 235-52); they produce a low incidence of escapers with reduced eyes and but partially complement *ey*^R.

allele	discoverer	origin	ref ^α	phenotype ^β
<i>ey</i> ¹	Hoge, 14e	spontaneous	4, 8	.50-.75

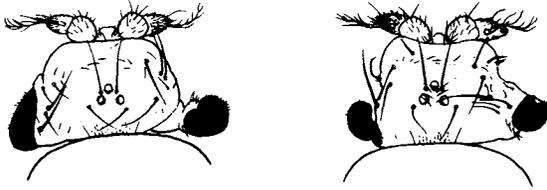
allele	discoverer	origin	ref ^α	phenotype ^β
<i>ey</i> ²	Nonidez, 1919	spontaneous	2, 4	.25-.50
<i>ey</i> ⁴	Li, 25f10	spontaneous	2, 4	.50±
* <i>ey</i> ³⁴	Gottchewski, 34g2b	cold treatment	4, 5	.25-.50
* <i>ey</i> ³⁶	Spencer, 36e25	spontaneous	4, 15	.50-.75
<i>ey</i> ⁴⁶	King, 46l	X ray	4, 9	.25±
<i>ey</i> ⁶⁷	Biggen, 67b	spontaneous	1	
<i>ey</i> ^{AD}	Das, 63a7	spontaneous	4, 13	0-1.0
<i>ey</i> ^{Dγ}	Muller, 27k	X ray	3, 4, 11	see entry
* <i>ey</i> ^{D39}	Sutton, 39k	X ray	4, 6	?-1.0
<i>ey</i> ^K		spontaneous	4, 12	.50-1.0
<i>ey</i> ^{I1}	Hochman	EMS	7	
<i>ey</i> ^{I2}	Hochman	EMS	7	
<i>ey</i> ^{I3}	Hochman	EMS	7	
<i>ey</i> ^{I4}	Hochman	spontaneous	7	
<i>ey</i> ^{opt δ}				
<i>ey</i> ^R	Sacharov, 23h13	spontaneous	4, 14	.25-.50
* <i>ey</i> ^{ta}	Datta, 63c26	spontaneous	4, 13	
* <i>ey</i> ^W	White, 59f	spontaneous	4, 10	.25±?

^α 1 = Biggen, 1967, DIS 43: 61; 2 = Bridges, 1935, Biol. Zh. 4: 401-20 (fig.); 3 = Bridges, 1935, Tr. Dinam. Razvit. 10: 463-73; 4 = CP627; 5 = Gottchewski, 1935, DIS 4: 15; 6 = Hinton, 1940, DIS 13: 49; 7 = Hochman, 1971, Genetics 67: 235-52; 8 = Hoge, 1915, Am. Nat. 49: 47-49; 9 = King and Poulson, 1948, DIS 22: 54; 10 = Meyer, 1959, DIS 33: 97; 11 = Patterson and Muller, 1930, Genetics 15: 495-577 (fig.); 12 = Sang and McDonald, 1954, J. Genet. 52: 392-412 (fig.); 13 = Sarkar, 1963, DIS 38: 28; 14 = Serebrovsky and Sacharov, 1925, Z. Eksperim. Biol. 1: 75-91; 15 = Spencer, 1937, DIS 7: 8.

^β Eye size relative to wild type.

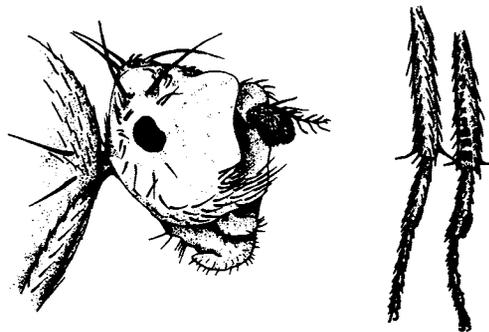
^γ See *ey*^D entry.

^δ See *Opt*^G.



*ey*⁴: eyeless-4

Edith M. Wallace, unpublished.



ey^D: eyeless-Dominant

Left: head. Right: first pair of legs.

From Patterson and Muller, 1930, Genetics 15: 495-577.

***ey*^D: eyeless-Dominant**

origin: X ray induced.

discoverer: Muller, 27k.

references: Patterson and Muller, 1930, Genetics

15: 495-577 (fig.).

Bridges, 1935, Biol. Zh. 4: 401-20.

1935, Tr. Dinam. Razvit 10: 463-73.

phenotype: Eyes of heterozygotes small, outline irregular, displaced toward top and rear. Head large, often with duplicated antennae or ocelli. Basitarsus broadened distally and incompletely separated from second tarsal segment owing to interruptions of the intersegmental membrane. Polarity of bract-bristle arrangement locally reversed in regions of membrane gaps [Poodry and Schneiderman, 1976, Wilhelm Roux's Arch. Dev. Biol. 180: 175-88 (fig.)]. 27-48 sex-comb teeth disposed in more-or-less parallel longitudinal rows in males; number of transverse-bristle rows increased in females (Stern and Tokunaga, 1967, Proc. Nat. Acad. Sci. USA 57: 658-64). Extra leg joints tend to form as mirror-image duplications proximal to the normal joint between the first and second tarsal joints (Held, Duarte, and Derakhshanian, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 145-57). Clones of *ey*⁺ tissue in *ey*^{D/+} background exhibit *ey*^{D/+} phenotype (Stern and Tokunaga, 1967), but both *ey*⁺ leg disks transplanted into *ey*^D hosts and the reciprocal transplant develop autonomously (Tokunaga, 1970, Dev. Biol. 18: 401-13). *ff ey*^D flies have but three tarsal joints (Postlethwaite and Schneiderman, 1975, Annu. Rev. Genet. 7: 381-433). Fully dominant in triplo-4 flies (Sturtevant, 1936, Genetics 21: 448). Eye size of *B; ey*^{D/+} males larger than of *B* alone. Produces extreme phenotype in combination with *D*. *D/+; ey*^{D/+} almost completely lethal (Sobels, Kruijt, and Spronk, 1951, DIS 25: 128). Homozygous lethal; two lethal crises, one during first or second larval instar and the other just prior to or during pupal stage. Cell degeneration observed in optic disks of homozygous second-instar larval (Ransom, 1979, J. Embryol. Exp. Morphol. 53: 225-35). Larvae which are unable to pupate rescuable by injection of α -ecdysone (Arking, 1969, J. Exp. Zool. 171: 285-96). Homozygotes reaching pupal stage lack adult derivatives of eye-antennal disks; adult derivatives are formed by *ey*^{D/ey^D eye-antennal disks transplanted into wild-type hosts; brain present but number of cortical cells severely reduced (Arking, Putnam, and Schubiger, 1975, J. Expt. Zool. 193: 301-12). RK2.}

cytology: Salivary chromosomes show duplication of about a dozen bands inserted into middle of fourth chromosome as a reversed repeat. Source of duplication unknown (Bridges, 1935) but suggested to be 4L (Hochman, 1971, Genetics 67: 235-52).

other information: May not be an allele of *ey*.

ey^{opt}: see *Opt*

***eya*: eyes absent**

location: 2-.

origin: Viable allele *eya*¹ spontaneous; other alleles X-ray induced or induced by ethyl methansulfonate.

references: Sved, 1986, DIS 63: 169 (fig.).

Renfranz and Benzer, 1989, Dev. Biol. 136: 411-29.

phenotype: Eye facets completely absent in strong alleles; other aspects of head development appear normal; antennae and ocelli, which also arise from eye-antenna disc, present. Eye portion of disc markedly reduced; no morphogenetic furrow or putative photoreceptor-cell clusters seen; little or no staining with monoclonal antibodies that normally stain presumptive photoreceptor cells.

alleles: More than 40 alleles obtained (Leiserson, 1990); most of them are homozygous lethal. Some alleles are temperature-sensitive.

cytology: Located in 26E. Associated with *In(2L)eya* = *In(2L)22D;34B*.

molecular biology: Gene cloned, the region involved spanning seven breakpoint alleles.

other information: Used by several groups in subtractive hybridization protocols for the isolation of eye-specific cDNA's.

***Eye: Eyeless dominant in chromosome 2**

location: 2-62.7.

origin: Probably ultraviolet induced.

discoverer: Edmondson, 51g.

synonym: *ey-II^D*.

references: 1952, DIS 26: 60.

phenotype: Eyes may be greatly reduced in size with frequent doubling of antennae. Overlaps wild type, especially in old vials. Recessive in triploids. *Eye/+*; *ey^D/+* has smaller eyes than either alone. Homozygous lethal. RK3.

eye gone: see *eyg*

eye missing: see *eym*

Eye tissue determiner: see *Etd*

eyeless: see *ey*

Eyeless dominant in chromosome 2: see *Eye*

eyelisch: see *eyh*

Eyeluf: see *Eyl*

eyes absent: see *eya*

eyes reduced: see *ld^{eyr}*

eyg: eye gone

location: 3-37.5 [between 37 and 38, to the right of *Est-6* (Roberts and Malpica, 1972, DIS 49: 40)].

origin: Spontaneous.

discoverer: Ives, 40g20.

references: 1942, DIS 16: 48.

phenotype: Eyes and head much smaller than normal. Considerable pupal mortality, probably from inability to push open pupa cases. Adults normal in viability and productivity. Character subject to genetic modifiers and possibly environmental influences. Expression varies

from complete absence of facets to formation of about 100 facets. Homozygotes show reduced larval and pupal survival (Inoue, 1980, DIS 55: 206). Lethal in combination with *ey*; phenotype described under *ey* [Hunt, 1970, Genet. Res. 15: 29-34 (fig.)]. RK2.

alleles: *eyg^{64e}*, X ray induced, slightly dominant (Mittler, 1967, DIS 42: 38).

cytology: Placed in 69C on the basis of its exclusion from *Df(3L)vin⁷* = *Df(3L)68C8-II;69B3-CI* and its mutant phenotype in combination with *In(3LR)gs^U* = *In(3LR)69B5-C4;8I* (Akam, Roberts, Richards, and Ashburner, 1978, Cell 13: 215-25).

eyh: eyelisch

location: 3-58 (0.47 units from *Sb* but order unspecified).

origin: Spontaneous.

references: Kalisch, 1980, DIS 55: 206-07.

phenotype: Eyes variably reduced. 7.5% of flies have no eyes; 0.3% of females and 5.6% of males overlap wild type. The proportion of completely eyeless flies increases dramatically and there is no overlap with wild type when *eyh* is combined with *eyg* or *ey*. RK3.

***Eyl: Eyeluf**

location: 1-18.

origin: Spontaneous.

discoverer: Marzluf.

phenotype: One or both eyes reduced in size. Expression varies from slight reduction to absence of eye. Sometimes extraneous materials protrude through eye; frequently, one or more duplicated antennae present. Penetrance incomplete; viability good. In aged and crowded cultures, both penetrance and expressivity increased. Third chromosome carries important modifiers affecting penetrance, and different wild-type and mutant stocks carry different modifiers. Penetrance lower at 18° than at 25°. RK3.

eym: eye missing

location: 3-67.9.

origin: Spontaneous.

references: Inoue, 1980, DIS 55: 206.

phenotype: Eyes almost completely absent in homozygotes. Heterozygotes with a wild-type chromosome but with *eyg* (3-37.5) show variable reduction in eye size. Homozygotes have normal larval but reduced pupal survival. Fertile. RK1.

eyr: see *ld^{eyr}*

f: forked

location: 1-56.7.

origin: Spontaneous.

discoverer: Bridges, 12k19.

references: Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. No. 237: 58 (fig.).

phenotype: Macrochaetae, microchaetae, and trichomes affected to various degrees depending on allele—short, gnarled, and bent with ends split or sharply bent. Extreme effect of f^{36a} on trichomes makes it the most useful allele as a cell marker in cuticular spots produced by somatic exchange. Treatment with methylurea causes normal bristle formation (De Marinis). Developmental studies [Lees and Waddington, 1942, Proc. Roy. Soc. (London) Ser. B 131: 87-110 (fig.); Lees and Picken, 1945, Proc. Roy. Soc. (London), Ser. B 132: 396-423 (fig.)] show nature of pupal bristle secretion is affected. Suppression by $su(f)$ and $su(Hw)^2$ allele specific. f^1 and f^5 suppressed by $su(Hw)$, $e(w^e)$, $su(f)$, and $su(pr)$, enhanced by $su(s)$ and $su(w^a)$ (Rutledge, Mortin, Schwarz, Thierry-Mieg, and Meselson, 1988, Genetics 119: 391-97). RK1.

alleles: Can be classed as weak with macrochaetae slightly bent, moderate with both macrochaetae and microchaetae clearly affected, strong with macrochaetae and microchaetae strongly gnarled, and extreme with chaetae and trichomes strongly affected. Descriptions of phenotypes are such as to make classification highly subjective. Bridges (1938, DIS 9: 46-47; see also CP552) lists 14 lost alleles of diverse origin not included here, and Belgovsky (1940, DIS 13: 47-48) lists 35 f^B alleles induced by X rays in $In(1)sc^8$ or $In(1)B^{M2}$, of which 33 are lost and not included here. Included in the table are deficiencies which have been erroneously given allelic designations in the past.

allele	origin	discoverer	ref ^α	phenotype
f^1 β	gypsy spont	Bridges, 12k19	5, 12, 13	moderate; suppressed by $su(f)$ and $su(Hw)^2$
f^3 β	spont	Bridges, 14k14	4, 5, 6	weak; not suppressed by $su(f)$
f^{3N} βγ	spont ^β	Green	5, 6, 8, 11	not suppressed by $su(f)$
f^5 β	gypsy	Bridges, 21b	4, 5, 12	strong; suppressed by $su(f)$ and $su(Hw)^2$
f^{34b}	X ray	Stone, 34b	5, 20	moderate
f^{34e}	X ray	Oliver, 34e4	5, 17	subliminal
f^{36a} β		Ives, 36a27	5	extreme; not suppressed by $su(Hw)^2$
f^{42}	spont	Anderson, 42c30	5, 18	strong
f^{51a}	X ray	Green, 51a	5, 11	extreme; not suppressed by $su(f)$
f^{56e} β	spont	Williams, 56e	5, 22	moderate
f^{67a}	X ray	Becker	1	strong; suppressed by $su(f)$ and $su(Hw)^2$
f^{68e}	EMS	Maddern, 68e11	8	moderate
f^{68i}	EMS	Maddern, 68i30	8	moderate
f^{70}				
f^{71i}	spont		21	
f^{257-4}	X ray	Demerec, 33j	5	strong
f^{257-15}	X ray	Demerec, 35a	5	lethal
f^{257-19}	X ray	Hoover, 35h	5	lethal
f^{257-22}	X ray	Demerec, 36c	5	lethal
f^{257-24}	X ray	Demerec, 36e	5	lethal
f^{257-29}	X ray	Bishop, 40i	5	slight
f^{257-30}	X ray	Bishop, 41a	5	moderate

allele	origin	discoverer	ref ^α	phenotype
f^{+ih} β	X ray	Muller	5, 14, 15, 16, 19	
f^{B15} βγ	X ray	Belgovsky, 36i	2, 3	variegated
f^{B27} βγ	X ray	Belgovsky, 36i	2, 3	variegated
f^H	spont	Hexter	5, 7	moderate; not suppressed by $su(f)$
f^K	gypsy	Kuhn	12	suppressed by $su(Hw)^2$
f^S δ				
f^{S1}	spont	Schalet		
f^{tuh}	spont	Kuhn	10	
f^U				
f^X β	X ray	Muller	5, 14, 15, 16, 19	moderate;
f^X1	gypsy X ray	Green	5, 7	suppressed by $su(f)$ moderate; not suppressed by $su(f)$
f^X2	X ray	Green	5, 7	moderate; not suppressed by $su(f)$
f^X3	X ray	Green	5, 7	moderate; not suppressed by $su(f)$
f^X4	X ray	Green	5, 7	moderate; not suppressed by $su(f)$

^α 1 = Becker, 1968, DIS 43: 59; 2 = Belgovsky, 1938, Isv. Akad. Nauk; 3 = Belgovsky, 1940, DIS 13: 47-48; 4 = Bridges, 1938, DIS 9: 46-47; 5 = CP627; 6 = Green, 1955, Proc. Nat. Acad. Sci. USA 41: 375-79; 7 = Green, 1956, Proc. Nat. Acad. Sci. USA 42: 73-77; 8 = Green, 1959, Proc. Nat. Acad. Sci. USA 45: 16-18; 9 = Hayman and Maddern, 1969, DIS 44: 50; 10 = Kuhn, 1973, DIS 50: 24; 11 = Lefevre and Green, 1959, Genetics 44: 769-76; 12 = Modolell, Bender, and Meselson, 1983, Proc. Nat. Acad. Sci. USA 80: 1678-82; 13 = Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. 237: 58 (fig.); 14 = Muller, 1946, DIS 20: 88-89; 15 = Muller, 1947, DIS 21: 71; 16 = Muller and Oster, 1957, DIS 31: 141-44; 17 = Oliver, 1939, DIS 12: 8; 18 = Oliver, 1942, DIS 16: 53; 19 = Oster, Erlich, and Muller, 1958, DIS 32: 144-45; 20 = Stone, 1934, DIS 4: 63; 21 = Thompson and Purnell, 1972, DIS 48: 16; 22 = Williams, 1956, DIS 30: 79.

^β Further descriptions below.

^γ Spontaneous derivative of f .

^δ Forked-suppressible, presumably = f^1 (Dudick, Wright, and Brothers, 1974, Genetics 76: 487-510).

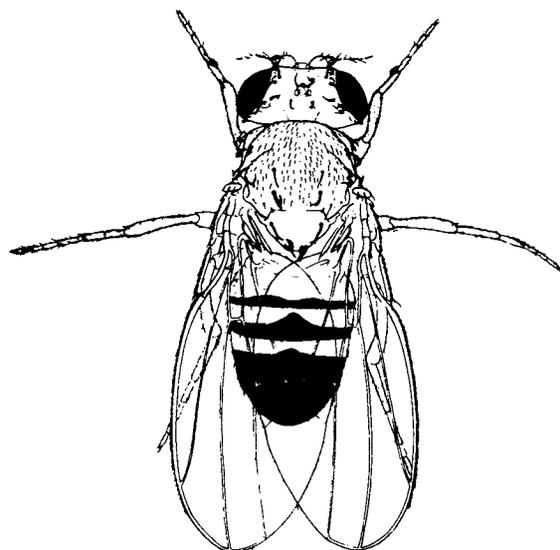
allele	cytology
f^{67a}	$Tp(1;1)12E;15E;18B;20$
f^{257-15}	$T(1;2)13E9-10;15E2-3;24F$
f^{257-19}	+
f^{257-22}	$T(1;2)4D2-3;8F;15E4-F1;39E;41F-42A$
f^{257-24}	+
f^{257-29}	$T(1;3)15F5-16A1;64$
f^{257-30}	+
f^{+ih}	+
f^X	+

cytology: Localized to 15F1-3 on the basis of $Df(1)f^{257-5} = Df(1)15E7-F1;15F2-4$. Polytene analysis completed on some alleles.

molecular biology: Region cloned using gypsy insertion into the f^1 allele (Parkhurst and Corces, 1985, Cell 41: 429-37) and f^5 (McLachlan, 1988, J. Mol. Cell. Biol. 6:1-6). f alleles from the right pseudoallelic series of Green (f , f^5 , f^{36a}) have DNA inserts within a 5.4 kb segment of DNA. The forked phenotype as well as the accumulation of RNAs can be rescued by P -element mediated germline transformation with a 6.1 kb SaII-XhoI fragment. There are four transcripts encoded within this DNA region: polyadenylated 4.3 kb, 2.4 kb, 1.9 kb, and 1.4 kb RNAs present only during mid to late pupal stages. Transcripts are reduced or missing in mutants. Transcripts in the f^1 mutant return to wild-type levels when combined with the $su(Hw)$ or $su(f)$ mutations (see McLachlan).

other information: Green (1955, Proc. Nat. Acad. Sci.

USA 41: 375-79; 1956, Proc. Nat. Acad. Sci. USA 42: 73-77) showed the forked mutants can be assigned to either of two pseudoallelic series, *f* is a member of the right series. Back mutations to *f*⁺ occur spontaneously, and their incidence is not increased by X rays (Green, 1959, Proc. Nat. Acad. Sci. USA 45: 16-18; Lefevre and Green, 1959, Genetics 44: 769-76).



f: forked

Edith M. Wallace, unpublished.

f¹

molecular biology: A member of the right hand group of alleles. Contains a gypsy insert in a 3.8 kb *EcoRI* restriction fragment (Parkhurst and Corces) and 3.2 kb from a particular *SalI* restriction site (McLachlan, 1986, J. Mol. Cell. Biol. 6: 1-6). Orientation of gypsy element opposite that of the gene.

f³

molecular biology: A member of the left hand group of alleles shown to contain an insert of 6 kb, but to the right of *f*¹, according to the orientation on the chromosome as determined by Parkhurst and Corces (McLachlan, 1986, J. Mol. Cell. Biol. 6: 1-6).

f^{3N}

phenotype: Expression similar to *f* but unlike *f*, does not respond to *su(f)*. Characterized by a high spontaneous reversion rate ($\sim 2 \times 10^{-5}$); rate may be further increased by a closely linked *cis*-dominant mutator allele, *Mu(f^{3N})*, (5 \times ; Woodruff, 1975, Genet. Res. 25: 163-77), by X rays (20 \times ; Green, 1977, Mutat. Res. 43: 305-08), and by mustard gas (Woodruff, Bowman, and Simmons, 1972, Mut. Res. 15: 86-89). RK1.

molecular biology: A member of the left hand group of alleles. Shown to contain a 2.8 kb tandem repeat, but to the right of *f*¹ according to the orientation on the chromosome as determined by Parkhurst and Corces (McLachlan). A revertant of *f^{3N}* retains the repeated sequence.

f⁵

molecular biology: A member of the right hand group of alleles; contains two gypsy inserts, one which is similar or identical to that of *f*¹ and the other which is closer to the *SalI* restriction site (1.2 kb); orientation is the same. In addition *f*⁵ contains a 4.4 kb insert in a restriction fragment to the left of that carrying the *f*¹ gypsy insert; it is located approximately 4.7 kb to the left of the reference *SalI* site (McLachlan).

f^{36a}

molecular biology: A member of the right-hand group of alleles. Contains a 3.0 kb insertion 2.5 kb to the right of the reference *SalI* site (McLachlan).

f^{56e}

molecular biology: A molecular deletion detected by Parkhurst and Corces but not by McLachlan; in the 3.8 *EcoRI* fragment studied by Parkhurst and McLachlan.

f^{+ih}: forked-wild type in heterochromatin

origin: X ray induced simultaneously with *f*^X.

synonym: *f*^m: *forked-mottled* = *f*^X*f*^{+ih}.

phenotype: *f*^{+ih} with any *f* allele has normalizing effect.

Patches of bristles and occasionally whole fly is wild type. An extra Y chromosome enhances the normalizing effect. RK2A.

cytology: Salivary chromosomes appear normal (J.I. Valencia).

molecular biology: The inserted segment contains the entire 40 kb walk of McLachlan.

other information: Apparently, *f*^{+ih} is all or part of the normal allele of *f* transposed to the proximal heterochromatin of the X chromosome, where it variegates. The sequences remaining in the original position behave as a hypomorphic allele of *f*, *f*^X. Oster, Erlich, and Muller (1957, DIS 31: 141-44) argue that the sum of sequences inserted in the heterochromatin plus those remaining behind exceeds the normal sequential content of *f*⁺.

f^{B15}

origin: X ray induced in *B*^{M2} male.

phenotype: Shows variegated expression of *f*. More extreme in combination with *E(f)*. RK2A.

cytology: Genetic data indicate that the mutation is associated with a reinversion of the *B*^{M2} inversion. *B*^{M2} phenotype reverted.

f^{B27}

origin: X ray induced in *B*^{M2} male.

phenotype: Males have mostly normal bristles, a few reduced like a Minute, rarely forked. *f*^{B27}/*f* are mosaic for forked. *f*^{B27}/*f*^{B27} females rarely survive; those that do sometimes have reduced bristles or notched wings, or both, and are sterile. More extreme in combination with *E(f)*. RK3A.

f^X: forked from X irradiation

origin: X ray induced, simultaneously with *f*^{+ih}.

synonym: *f*^m: *forked-mottled* = *f*^X*f*^{+ih}.

discoverer: Muller.

phenotype: A medium *f*. Suppressed by *su(f)*. Behaves as a hypomorphic allele (Oster, Erlich, and Muller, 1958, DIS 32: 144-45). RK1.

molecular biology: Restriction map indistinguishable from

that of *f¹* (McLachlan).

F2: see *Efl α 2*

fa: see *N*

fa^{swb-h}: see *e(fa^{swb})*

facetious: see *rg^P*

factor for male fertility: see *fmf*

fai: *faint*

location: 2-61.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82 (fig.)

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Homozygous larval lethal. Unpigmented cuticle and mouth parts.

alleles: Six, two of which are weak; *fai¹* and *fai²* (isolated as IIA and IIV) retained.

faint little ball: see *Egfr*

faint sausage: see *fas*

faintoid: see *gs*

fap: *female abdomen pattern*

location: 3-0.5 (to the left of *mwh*; Robertson and Riviera, 1972, DIS 48: 21).

origin: Spontaneous.

references: Robertson and Louw, 1966, DIS 41: 154-55 (fig.).

Robertson, Briscoe, and Louw, 1977, Genetica 47: 73-77.

phenotype: Female-specific recessive that removes black pigment from the sixth and seventh tergites when homozygous; heterozygotes exhibit intermediate phenotype at 18°.

alleles: Natural populations highly polymorphic at this locus with at least six alleles (Robertson and Riviera, 1972).

cytology: Polytene chromosomes normal (Slyzinska).

Farkas: see *Fs(3)Sz8*

fas: see *rg^P*

fas: *faint sausage*

location: 2-68.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82 (fig.)

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Embryonic lethal; embryos have poorly developed cuticle with necrotic patches and an abnormal head. First visible in extended-germ-band stage.

alleles: Four, out of which two are retained, *fas¹* and *fas²* (isolated as IC and IIA).

Fas1: *Fasciclin I*

location: 3-{58}.

origin: Isolated from *Drosophila* embryonic cDNA libraries using a fasciclin I probe from grasshopper.

references: Zinn, McAllister, and Goodman, 1988, Cell 53: 577-87 (fig.).

phenotype: Expression confined to neurons. In 12-14 h embryos high levels of fasciclin I are detectable in two commissural axon bundles (fascicles) per segment, a thick one in the posterior commissure and a thin one in the anterior commissure. In addition high expression is seen in two clusters of neuronal cell bodies per hemisegment as well as in the intersegmental and segmental nerve axons. Finally, fasciclin I is detected on the surface of all peripheral neurons. Postulated to be involved in neuron recognition during growth cone guidance. Flies deficient in *FasI* function are fully viable and CNS development apparently is normal.

cytology: Placed in 89D by *in situ* hybridization to polytene chromosomes. Deleted by *Df(3R)Ubx¹⁰⁹* = *Df(3R)89D1-2;89E1-2*.

molecular biology: Apparently full-length cDNA clone of 3.0 kb isolated and sequenced. Conceptual amino-acid sequence indicates a protein similar in size to the 638-amino-acid, 70 kd fasciclin I from grasshoppers; it is 48% homologous to the grasshopper protein. Contains a putative signal sequence, but no convincing transmembrane domain. Concluded to be an extrinsic membrane protein since it is isolated with the membrane fraction. Mature protein composed of four homologous domains of approximately 150 amino acids each, followed by 20-25 C-terminal residues.

Fas3: *Fasciclin III*

location: 2-{53}.

origin: Identified by screening a cDNA expression library with monoclonal antibodies directed against embryonic CNS and shown to identify proteins expressed on the cell surface of a restricted subset of cells of the embryo, including specific cells of the developing central nervous system.

references: Gauger, Glicksman, Saltino, Condie, Schuberger, and Brower, 1987, Development 100: 237-44.

Patel, Snow, and Goodman, 1987, Cell 48: 975-88 (fig.).

Snow, Bieber, and Goodman, 1989, Cell 59: 313-23.

phenotype: Monoclonal-antibody staining reveals a complex sequence of temporal and spatial expression of *Fas3*. It is expressed on segmentally repeated patches of cells during neurogenesis and outside the developing CNS on segmentally repeated stripes in the epidermis at the segmental grooves, on patches of epithelial cells near the stomodeal and proctodeal invaginations, on the visceral but not the somatic mesoderm, and on the luminal surface of the salivary gland epithelium; expression is highest where fasciclin-III-positive cells contact one another. After germ-band extension transiently expressed on segmentally repeated patches of neuroepithelial cells and specific underlying neuronal lineages; by the end of germ band retraction fasciclin-III is expressed in repeated stripes across all body segments. At hour 12 fasciclin is detected on a subset of three axon bundles (fascicles) in the anterior commissure and two in the posterior commissure of each segment; generally, however it's not expressed on the axons of these neurons that exit CNS, specifically on the longitudinal fascicles. Expression of *Fas3* in transfected cultured cells promotes their adhesion to each other, but not to nonexpressing cells (Snow, *et al.*). The monoclonal antibodies used in the isolation of fasciclin identify four closely related membrane glycoproteins of 80, 66, 59, and 46 kd molec-

ular weight; all depend on the presence of *Fas3*⁺ for their presence.

cytology: Placed in 36E1 by *in situ* hybridization to polytene chromosomes. In the region of overlap of *Df(2L)H20 = Df(2L)36A7-10;36E4-F1* and *Df(2L)V18 = Df(2L)36C4-D1;37C2-5*.

alleles: A null mutation, *Fas3*^{nl}, has been isolated (Elkins).

molecular biology: cDNA's cloned and sequenced; conceptual amino acid sequence indicates a mature polypeptide, following cleavage of a 20-amino-acid signal sequence, of 488 amino acids of calculated molecular mass of 53.6 kd. It comprises a putative 326-amino-acid extracellular domain, a 24-amino-acid transmembrane domain, and a 138-amino-acid intracellular domain. The extracellular domain contains four potential N-linked glycosylation sites, and the intracellular domain contains a single tyrosine residue, two potential phosphorylation sites, and an *opa*-repeat sequence (a second such sequence found in the 3' untranslated region).

fat: see *ft*

Fat body protein: see *Fbp*

faulty chaetae: see *fc*

***fb: fine bristle**

location: 1-1.0.

origin: Induced by D-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3026).

discoverer: Fahmy, 1954.

references: 1958, DIS 32: 70.

phenotype: Thin, slightly shortened bristles. Occasional scalloping of wing margins. Delayed emergence. Good viability and fertility in both sexes. RK3.

fbr: fine bristles

location: 1-3.3.

origin: Spontaneous (appears to be unstable).

references: Golubovsky, 1978, DIS 53: 122.

phenotype: Bristles reduced in length and diameter.

other information: Transmission in crosses irregular; possibly associated with a transposable element. Possibly allelic to *dm* or *slc*.

Fbp1: Fat body protein 1

location: 3-{41}.

synonym: *P1*.

references: Lepesant, Kejzlarova-Lepesant, and Garen, 1978, Proc. Nat. Acad. Sci. USA 75: 5570-74.

Lepesant, Levine, Garen, Kejzlarova-Lepesant, Rat, and Somme-Martin, 1982, J. Appl. Mol. Genet. 1: 371-83.

Paco-Larson, Nakanishi, Levine, and Garen, 1986, Dev. Genet. 7: 197-203.

Deutsch, Laval, Lepesant, Maschat, Pourrain, and Rat, 1989, Dev. Genet. 10: 220-31.

phenotype: Encodes P1 polypeptide, which accumulates in the fat body of third-instar larvae; not detectable at earlier stages or in other tissues. Expression stimulated by the increased ecdysone level characteristic of third-instar larvae; function of the protein product uncertain (Deutch *et al.*, 1989).

cytology: Located in 70D1-2 by *in situ* hybridization to the salivary chromosomes.

molecular biology: *Fbp1* cloned and its nucleotide

sequence determined (Lepesant, *et al.*, 1982); gene shows a simple molecular structure and organization, with very short introns interrupting transcribed sequences coding for unique mRNA. The putative P1 polypeptide of 1,030 amino acids has a presumptive N-terminal signal peptide; since no extracellular P1 product is detectable in the hemolymph, the signal peptide may serve to compartmentalize the protein within the fat-body cell. The sequence also contains two aspartic + asparagine stretches of 11 or 12 amino acids (Deutch *et al.*, 1989). A transcript is not detected until the second larval molt; at this time, it is restricted to the fat body (Lepesant *et al.*, 1982; Paco-Larson *et al.*, 1986). A peak in the accumulation of transcript occurs at the time of puparium formation and is followed by its disappearance after the early pupal stage; the rise and fall follows by several hours that of the larval serum proteins, which are also fat-body specific in their expression. *Fbp1* expression completely inhibited in *ecd-1*^{ts} flies shifted to restrictive temperature following the second larval molt or in *dor*^{lit87} third-instar larvae; expression rescued by administration of 20-hydroxyecdysone. Germ-line transformation experiments demonstrate that 138 but not 80 base pairs upstream from the transcription start site are sufficient to impart complete temporal, spatial, and hormonal regulatory control of transcription.

Fbp2: Fat body protein 2

location: 2-{35}.

synonym: *P6*.

references: Langer-Safer, Levine, and Ward, 1982, Proc. Nat. Acad. Sci. USA 79: 4381-85.

Lepesant, Levine, Garen, Lepesant-Kejzlarova, Rat, and Somme-Martin, 1982, J. Appl. Mol. Genet. 1: 371-83.

Paco-Larson, Nakanishi, Levine, and Garen, 1986, Dev. Genet. 7: 197-203.

Deutsch, Laval, Lepesant, Maschat, Pourrain, and Rat, 1989, Dev. Genet. 10: 220-31.

phenotype: Gene detected in the larval fat body of *Drosophila melanogaster*. Function of the protein product uncertain (Deutch *et al.*, 1989).

cytology: Located in 30B by *in situ* hybridization to the salivary chromosomes.

molecular biology: *Fbp2* cloned, and its nucleotide sequence determined (Lepesant, 1982). The gene has an unusually high methionine content (20%); conceptual amino acid sequence shows significant similarity to that of *Drosophila* alcohol dehydrogenase, which lacks methionine. A transcript is not detected until the second larval molt, when it is restricted to the fat body (Lepesant *et al.*, 1982; Paco-Larson *et al.*, 1986). As in *Fbp1*, a peak in the accumulation of transcript occurs at the time of puparium formation and is followed by its disappearance after the early pupal stage; apparently not sensitive to the removal or addition of ecdysone during the third larval instar.

Fat body protein: see *Fbp*

***fc: faulty chaetae**

location: 1-0.9.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).

discoverer: Fahmy, 1954.

references: 1958, DIS 32: 70.

phenotype: Short, thin bristles. About one-third of flies show either absence or duplication of one scutellar bristle. Viability and fertility good in both sexes. RK2.
other information: Possibly allelic to *kz*.

fch: fragile chorion

location: 3-55.
origin: Induced by ethyl methanesulfonate.
references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.
phenotype: Maternal effect; chorion very thin or nonexistent.
alleles: Two, *fch*¹ and *fch*² (isolated as 055 and 267).

fcl: foreclosed

location: 1-(between 57.8 and 59.1).
origin: Induced by ethyl methanesulfonate.
references: Eberl and Hilliker, 1988, Genetics 118: 109-20.
phenotype: Embryonic lethal; no internal organogenesis; prominent central yolk plug; dorsal closure may be incomplete; head involution partial; germ band contraction incomplete. No cuticle evident in cuticle preparations, but mouth parts and spiracles visible. Embryos exhibit considerable movement.
alleles: Two putative alleles *fcl*¹ and *fcl*² (isolated as *l(1)EH244b* and *l(1)EH523*).
cytology: Placed in 16C2-18B11 based on the failure of $Dp(1;3)f^{+71b} = Dp(1;3)15A4;16C2-3;80-81$ and $X^P Y^D B5O = X^P Y^D 18B4-11$ to cover it.

***fd: furled**

location: 1-(rearrangement).
origin: Induced by ³²P.
discoverer: Bateman, 1949.
references: 1950, DIS 24: 54.
 1951, DIS 25: 77.
phenotype: Like vestigial but with immovable mouth parts and fully extended proboscis. Dies early, perhaps owing to failure to ingest. Viability at eclosion good. RK3A.
cytology: Associated with $T(1;3)fd = T(1;3)7A;86E + In(3R)89C;96A$ (Darby).

fdg: fibrillar dysgenesis

location: 1-55.0
origin: Induced by ethyl methanesulfonate.
synonym: *l(1)fdg*.
references: Newman, and Wright, 1983, Dev. Genet. 3: 329-45 (fig.).
phenotype: Embryonic lethal at 32-30°; larval-pupal lethal at lower temperatures. Mutant embryos exhibit abnormal distribution of muscle fiber elements and organelles. Also abnormal Z-body alignment.

female abdomen pattern: see *fap*

female lethal 302: see *fl(1)302*

Female lethal: see *Sxl*

female sterile: see *fs()*

Female sterile: see *Fs()*

femaleless: see *fle*

fes: see *fs(2)B*

fes(2)K: see *fs(2)K*

ff: fluff

location: 1-57.7.
origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).
discoverer: Fahmy, 1955.
references: 1959, DIS 33: 86.
phenotype: Extremely fine short bristles. Wings slightly rounded at tips. Males and females viable and fertile; eclosion delayed. RK3.
other information: One allele induced by nitrogen-mustard.

***fft: fused filament**

location: Not located.
origin: Spontaneous.
discoverer: Robertson and Reeve.
references: 1954, DIS 28: 78.
phenotype: Chorionic filaments of eggs laid by *fft* females usually fused into a single structure. A few normal eggs also laid. Hatchability reduced and variable. RK3.

fg: see *spd*^{fg}

***fi: frail**

location: 1-53.
origin: Recovered among progeny of flies treated with Janus green.
discoverer: Muller, 28e20.
references: 1935, DIS 3: 30.
phenotype: Wings nearly as small as *m*, thin and frail. Bristles fine. Fly weak. Viability 10-30% wild type. RK3.

fiber loop: see *flo*

fibrillar dysgenesis: see *fdg*

***fil: fine lash**

location: 1-56.8.
origin: Induced by L-p-N,N-(2-chloroethyl)amino-phenylalanine (CB. 3025).
discoverer: Fahmy, 1953.
references: 1959, DIS 33: 86.
phenotype: Thin, slightly shorter bristles. Eyes reduced in size; posterior border very close to orbital bristles. Both sexes viable and fertile. RK3.
other information: Two alleles induced by ethyl methanesulfonate.

filzig: see *flz*

fin: finer

location: 1-29.6.
origin: Induced by D-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3026).
discoverer: Fahmy, 1954.
references: 1959, DIS 33: 86.
phenotype: Fly slightly smaller than normal with shorter, thinner bristles. Delayed eclosion. Males viable but sterile. RK3.

fine bristle: see *fb*

fine bristles: see *fbr*

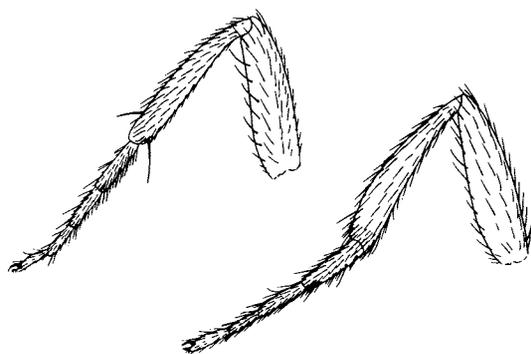
fine chaetae: see *fnc*

fine lash: see *fil*

fine macros: see *fm*

finer: see *fin*

fizzy: see *fzy*



fj: four jointed

Edith M. Wallace, unpublished.

fj: four jointed

location: 2-81.5.

origin: Spontaneous.

discoverer: Schultz, 31d1.

phenotype: Similar to *d*. Tarsi four instead of five jointed.

Legs short and stocky, owing to failure of joint formation between second and third tarsal segments (Tokunaga and Gerhart, 1976, *Genetics* 83: s76). Femur, tibia, and tarsae foreshortened [Tokunaga and Gerhart, 1976; Mikuta, 1979, *Genetika (Moscow)* 15: 624-32]. Probability of joint failure proportional to degree of shortening of second tarsal segment (Tokunaga and Gerhart, 1976). Joint failure phenotype can extend into *ff*/+ tissue adjacent to *ff*/*ff* clones (Tokunaga and Gerhart, 1976). Leg chaetae show irregularities in the relative orientation of sockets and bracts (Held, Duarte, and Derakhshanian, 1986, *Wilhelm Roux's Arch. Dev. Biol.* 195: 145-57). Enhanced by *ss^a* and *ss^{ab}* [Villem, 1945, *Genetics* 30: 26-27; Mglinetz and Ivanov, 1976, *Genetika (Moscow)* 12: 87-94] and by *pb* [Kaurov, Ivanov, and Mglinetz, 1978, *Genetika (Moscow)* 14: 306-12]; also influenced by *Antp^{Ns}* (Mikuta and Mglinetz). *ff ey^D* flies have but three tarsal joints (Postlethwait and Schneiderman, 1975, *Ann. Rev. Genet.* 7: 381-433). Development similar to that of *dachs* [Waddington, 1943, *J. Genet.* 45: 29-43 (fig.)]. Wings shorter and broader with crossveins conspicuously closer together; veins diverge at greater angle (Tokunaga, Michinomae, Sizemore, and Gerhart, 1978, *Genetics* 88: s98). Effect visible in pupal wing (Waddington, 1940, *J. Genet.* 41: 75-139). Eyes smaller, ellipsoid, coarse textured; head foreshortened. RK2.

alleles: **ff^{40e}* (Ives, 1941, *DIS* 14: 39) showed more extreme but more variable venation anomalies than *ff¹*.

cytology: Placed in 55B-C based on its inclusion in *Df(2R)11B = Df(2R)54F6-55A1;55C1-3* and *Df(2R)Pcl-w5 = Df(2R)55A-B;55C* (Deng and Rizki, 1988, *Genome* 30, Suppl. 1: 192).

Fk: see *Hex-C*

fkh: fork head

location: 3-95.

references: Jürgens and Weigel, 1988, *Wilhelm Roux's Arch. Dev. Biol.* 197: 345-54.

Weigel, Jürgens, Küttner, Seifert, and Jäckle, 1989, *Cell* 57: 645-58.

phenotype: Embryonic lethal. *fkh⁺* appears to be required in the most anterior and posterior regions of the embryo; in amorphic mutants homeotic transformation effects the appearance of post-oral head structures in these terminal domains. The ectopic head structures are sometimes associated with thoracic structures anteriorly and anterior tail structures posteriorly. Thus *fkh* mutations produce transformations directed to the center involving structures normally found in different parasegments. Anteriorly, esophagus and proventriculus, derivatives of the ectodermal stomodaeum, are absent but not the pharynx or the hypopharyngeal organ, which resides on the anterior surface of the embryo; parts of the head skeleton are distorted; other parts apparently normal. Salivary glands absent. Posteriorly, anal pads and Malpighian tubules, derivatives of the ectodermal proctodaeum, are absent; replaced by anterior tail structures and post-oral head structures; supernumerary anal sensilla and dorsal hairs also present. Anterior and posterior *fkh* domains are beyond the regions controlled by *ANTC* and *BXC*, respectively; however ectopic expression of *ANTC* and *BXC* expression can occur in the *fkh* domains of *fkh* but not *fkh⁺* embryos. No maternal effect. Homozygous *fkh* clones in adult cuticle completely normal in structure indicating no requirements for *fkh⁺* in imaginal disk development. *In situ* hybridization reveals transcript in two terminal domains shortly before blastoderm cellularization occupying 5% embryonic length anteriorly and 15% posteriorly. *fkh* protein first detected in posterior domain at the end of syncytial blastoderm and the anterior domain at the beginning of cellularization. Expression continues in the derivatives of the ectodermal stomodaeum and proctodaeum throughout gastrulation. In addition *fkh* protein is detected in the midgut, the salivary glands, the central nervous system, and the yolk cells.

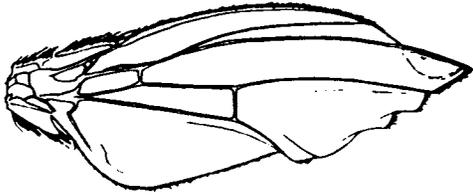
alleles:

allele	origin	ref ^α	comments
<i>fkh¹</i>	EMS	1	nonsense mutation in codon 254
<i>fkh²</i>	EMS	2	in frame deletion of codons 233-237; 17 bp deletion + 2 bp insertion
<i>fkh³</i>	EMS	2	nonsense mutation in codon 63
<i>fkh⁴</i>	X ray	2	weak allele; <i>T(2;3)38;98D2-3</i>
<i>fkh⁵</i>	X ray	2	weak allele; <i>T(Y;3;4)98D2-3;99F;101</i>
<i>fkh⁶</i>	X ray	2	out of frame deletion of codons 8-10 + 2 bp of codon 11
<i>fkh⁷</i>	X ray	2	deletion of ca. 90 kb
<i>fkh⁸</i>	X ray	2	weak allele; <i>In(3LR)80F;98D2-3</i>

^α 1 = Jürgens, Wieschaus, Nüsslein-Volhard and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95; 2 = Jürgens and Weigel, 1988, *Wilhelm Roux's Arch. Dev. Biol.* 197: 355-59.

molecular biology: Region cloned in a 120 kb walk. Identity of *fkh* gene confirmed by *P*-mediated germ-line transformation. 4.2 kb transcript observed in Northern blots; 4002 base pairs of cDNA sequence determined; direction of transcription from right to left; breakpoints of *fkh* rearrangement located downstream from the gene. Sequence reveals an uninterrupted open reading frame of

1530 base pairs, encoding a 510-residue polypeptide of calculated molecular weight of 54.3 kd; antibody staining reveals the restriction of *fkh* protein to nuclei. No significant sequence similarity to known proteins detected in computer search of databases. A second transcript, distal to fork head (*dfk*), which is transcribed from left to right, lies less than 500 base pairs to the right of *fkh*.



fl: fluted

Edith M. Wallace, unpublished.

fl: fluted

location: 3-59.7+ (Kuhn, Woods and Andrew, 1984, DIS 60: 134-35).

origin: Spontaneous.

discoverer: Redfield, 211.

phenotype: Wings creased lengthwise and dark. Overlaps wild type slightly at 25° but not at 19°. RK3.

alleles: **fl*², spontaneous, Spencer, 36d15.

Fl: see *Sxl*

fl(1)302: female lethal 302

location: 1-{9} (to the right of *ect*).

origin: Induced by ethyl methanesulfonate.

discoverer: Steinmann-Zwicky.

references: 1988, EMBO J. 7: 3889-98.

phenotype: Homozygous females die in larval or pupal stages. Male viable.

cytology: Placed in 4C5-E1 based on its inclusion in *Df(1)rb13 = Df(1)4C5-6;4D3-E1*. Not included in *Df(1)rb23* and *Df(1)rb27* which overlap the left end of *Df(1)rb13* but have not been characterized cytologically.

fla: flateye

location: 1-2.4 [no evidence for such in *w-spl* interval (Lefevre and Green, 1972, Chromosoma 36: 391-412)].

origin: Induced by L-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 70.

phenotype: Smaller fly with smaller and less curved eyes. Wings extremely variable, from normal through incised margins to crumpled vestigial stumps. Not easily classified. Viability and fertility good in males but reduced in females.

flap wing: see *flw*

flare: see *flr*

flateye: see *fla*

flb: see *Egfr*

fle: femaleless

location: 3-45 (mapped with respect to *mwh* and *red*).

origin: Spontaneous.

synonym: *file(3)100*.

references: Inouchi, Okuno, Oishi, and Watanabe, 1983, Jpn. J. Genet. 58: 525-30.

Okuno, Satou, and Oishi, 1984, Jpn. J. Genet. 59: 237-47.

phenotype: Homozygous females die; males survive. Lethal effect late. Dosage compensation normal as determined by tritiated uridine labeling of polytene chromosomes. No interaction with *tra* in *fle/tra* heterozygotes.

fli-385: flightless-385 (J.C. Hall)

location: 1-ca. 0.

origin: Induced by ethyl methanesulfonate.

references: Homyk and Sheppard, 1977, Genetics 87: 95-104.

phenotype: Adults cannot fly or even beat wings; induced simultaneously with a *fli* allele; not allelic to *fliA* or *fliB*.

fli: flightless (J.C. Hall)

A series of ethyl-methanesulfonate-induced, sex-linked mutations selected by Homyk *et al.*. All were crudely mapped; complementation tests were performed among *fli*, *fliA*, and other behavioral mutants recovered in the screens and mapping to the same interval. In this way eleven *fli* loci, designated *fliA* through *fliK*, were identified. Allelism tests with *fli* mutants not carried out.

references: Homyk and Sheppard, 1977, Genetics 87: 95-104.

Homyk, Szidonya, and Suzuki, 1980, Mol. Gen. Genet. 177: 553-65.

Homyk and Grigliatti, 1983, Dev. Genet. 4: 77-97.

Homyk and Emerson, 1988, Genetics 119: 105-21.

fliA: see *Actn*

fliB

location: 1-0.0.

phenotype: Flightless and beats wings poorly after rearing at 17°; weak flier after rearing at 22°; jumps abnormally short distances when raised at restrictive temperature. TSP late larval early pupal.

fliC

location: 1-19.9.

phenotype: Flight and other behaviors impaired; for one allele (*fliC*¹), raising at 22° leads to weak flight though normal appearance; rearing at 29° causes drooped wing position (often bilaterally asymmetrical), poor flying, weak locomotion, inadequate wing display of courting males; TSP for flightlessness in first half of pupal stage. *fliC*² allele nearly lethal at 29°; rearing at 22° leads to good survival but weak flight; both alleles have lethal maternal effect. *fliC*¹ females sterile.

alleles: Two mutant alleles: *fliC*¹ and *fliC*². ERG normal (Homyk and Pye, 1989, J. Neurogenet. 5: 37-48).

cytology: Placed in 7B2-C1; in the region of overlap of *Df(1)ct-J6 = Df(1)6E1;7C1* and *Df(1)ct4B1 = Df(1)7B2-4;7C3-4* (Homyk and Emerson, 1988, Genetics 119: 105-21).

fliD

location: 1-25.5.

phenotype: When raised at 17°, adults are weak fliers; when raised at 22°, bristles are small and adults cannot

fly and cannot jump normally; lethal when raised at 29°. ERG normal (Homyk and Pye, 1989, J. Neurogenet. 5: 37-48). TSP a restricted period in early pupa.

cytology: Placed in 8C-9B1; not deleted by *Df(1)KA14* = *Df(1)7F1-2;8C6* or *Df(1)v-L15* = *Df(1)9B1-2;10A1-2*. Females carrying *fliD* in heterozygous combination with *Df(1)C52* = *Df(1)8E;9C-D* and raised at 29° exhibit partially impaired flying and hopping ability which could indicate location of the gene at the 8E breakpoint; however, *Df(1)C52*± females also show abnormal movement after development at 29°.

fliE

location: 1-26.8.

phenotype: Flightless when raised at 17°; weak flight after rearing at 22°; jumping ability poor after rearing at restrictive temperature. TSP first half of pupal stage. ERG normal (Homyk and Pye, 1989, J. Neurogenet. 5: 37-48).

fliF

location: 1-29.1.

phenotype: Flightless, jumping ability poor; mosaic analysis suggests primary defect could be in muscles (Homyk, 1977, Genetics 87: 105-28). In *fliF*² and *fliF*³, indirect flight muscles have relatively mild abnormalities of z bands and myofibrils. Reduced amounts of at least two high-molecular-weight proteins from indirect flight muscles [Deak, Rahmi, Bellamy, Bienz, Blumer, Fenner, Golin, Ramp, Reinhardt, Dubendorfer, and Cotton, 1980, Development and Neurobiology of Drosophila (Siddiqi, Babu, Hall, and Hall, ed.). Plenum Press, New York and London, pp. 183-92; Deak, Bellamy, Bienz, Dubuis, Fenner, Gollin, Rahmi, Ramp, Reinhardt, and Cotton, 1982, J. Embryol. Exp. Morphol. 69: 61-81].

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>fliF</i> ¹	EMS	Homyk		2	
<i>fliF</i> ²	EMS		696 = <i>gmp</i> ¹	1	
<i>fliF</i> ³	EMS		628 = <i>gmp</i> ²	1	

^α 1 = Deak, Rahmi, Bellamy, Bienz, Blumer, Fenner, Golin, Ramp, Reinhardt, Dubendorfer, and Cotton, 1980, Development and Neurobiology of *Drosophila* (Siddiqi, Babu, Hall, and Hall, ed.). Plenum Press, New York and London, pp. 183-92; 2 = Homyk and Sheppard, 1977, Genetics 87: 95-104.

fliG

location: 1-32.67.

references: Zhimulev, Belyaeva, Pokolkova, Kotchneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baricheva, Aizenzon, Kramers, and Eeken, 1981, DIS 56: 192-96.

1982, DIS 58: 210-14.

Zhimulev, Pokholkova, Bgatov, Umbetova, Solovjeva, Khudyakov, and Belyaeva, 1987, Biol. Zentralbl. 106: 699-720; 1987, DIS 66: 194-97.

phenotype: Flight and other behaviors impaired. One allele, *fliG*¹, allows for weak flight; another, *fliG*², leads to almost no flight ability and weak running or climbing (Homyk, 1977, Genetics 87: 105-28); wings abnormal in appearance (intrinsically and in terms of positions in which they are held); male fertility reduced; sex combs reduced. All alleles are nearly lethal when reared at 18° and cause poor viability when raised at 25°. Not allelic to *fliC*, *fliD*, *fliE*, *fliF*, or *fliG*, or to *sdby*.

cytology: Placed in the 9F11-12 interband or in the left

edge of 9F12 based on its being uncovered by *Df(1)v-L3* = *Df(1)9F10;10A7-8* but not by *Df(1)v-M6* = *Df(1)9F11-12;10A1-2* and mapping to the left of *v* (Zhimulev *et al.*, 1981, 1987).

alleles: Four mutant alleles: *fliG*¹, *fliG*², *fliG*³ (= *fliG*^{B186}), and *fliG*⁴ (= *fliG*^{dp324}), all cold-sensitive lethals or semilethals. The first two described by Homyk and Sheppard, the latter two by Zhimulev *et al.*; *fliG*⁴ induced in *v*⁺*Yy*⁺.

fliH: see *hdp-a*

fliI

location: 1-66.5.

phenotype: Both viable and lethal alleles isolated. Viable alleles are recessive and flightless; wing position normal; jumping abilities variously impaired depending on allele. Temperature-sensitive alleles show temperature-sensitive period to be during first half of pupal stage. Rearing *fliI*¹ and *fliI*² at 17° leads to weak flight and at 22° to flightlessness. Indirect-flight-muscle fibers wavy with amorphous internal structures; myofilaments disorganized; z bands and myofibrils abnormal; thin filaments often aggregated into bundles with striations (not seen in wild type). Mosaic analysis with *fliI*⁵ suggests primary defect in thoracic muscles (Koana and Hotta, 1978, J. Embryol. Exp. Morphol. 45: 123-43). Trans heterozygotes with *sdby* are flightless. Lethal alleles die in larval or pupal stages. Maternal effect lethal; progeny produced from homozygous female germline clones of severe alleles (e.g., *fliI*¹⁴) are not rescuable by normal allele of paternal origin; abnormal folding during gastrulation; mesoderm invagination abnormal; only patches of epidermis present at later stages; embryos from clones homozygous for weaker alleles (e.g. *fliI*²²) were more nearly normal in appearance.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>fliI</i> ¹	EMS	Homyk		2	temperature sensitive
<i>fliI</i> ²	EMS	Homyk		2	temperature sensitive
<i>fliI</i> ³	EMS	Homyk		2, 9	nonconditional
<i>fliI</i> ⁴	EMS	Koana	<i>fliO</i> ¹	3	strong allele
<i>fliI</i> ⁵	EMS	Koana	<i>fliO</i> ²	3, 9	moderate allele
<i>fliI</i> ⁶	EMS	Koana	<i>fliO</i> ³	3	moderate allele
<i>fliI</i> ⁷	EMS	Koana	<i>fliO</i> ⁴	3	moderate allele
<i>fliI</i> ⁸	P		<i>l(1)D44</i>	1, 9	
<i>fliI</i> ⁹		Kaplan	<i>l(1)DCA3-19</i>		
<i>fliI</i> ¹⁰	X ray	Novitski	<i>l(1)EN3</i>	9, 10, 11	larval lethal; maternal-effect lethal
<i>fliI</i> ¹¹	EMS	Lifschytz	<i>l(1)M90</i>	8	on <i>y</i> ⁺ <i>Ymal</i> ⁺
<i>fliI</i> ¹²	EMS	Lifschytz	<i>l(1)R-9-6</i>	7	
<i>fliI</i> ¹³	EMS	Lifschytz	<i>l(1)R-10-9</i>	7	
<i>fliI</i> ¹⁴	EMS	Lifschytz	<i>l(1)W2</i>	4, 6, 7, 8, 9, 11	larval lethal; maternal-effect lethal
<i>fliI</i> ¹⁵	X ray	Lefevre	<i>l(1)A3</i>	4	
<i>fliI</i> ¹⁶	X ray	Lefevre	<i>l(1)A141</i>	4	
<i>fliI</i> ¹⁷	X ray	Lefevre	<i>l(1)GA105</i>	4	
<i>fliI</i> ¹⁸	X ray	Lefevre	<i>l(1)HC183</i>	4, 9	pupal lethal; maternal-effect lethal
<i>fliI</i> ¹⁹	X ray	Lefevre	<i>l(1)HF338</i>	4	
<i>fliI</i> ²⁰	X ray	Lefevre	<i>l(1)RA70</i>	4	
<i>fliI</i> ²¹	EMS	Lefevre	<i>l(1)DA534</i>	5, 9	pupal/adult lethal; maternal-effect lethal
<i>fliI</i> ²²	EMS	Lefevre	<i>l(1)EF498</i>	5, 9	pupal lethal; maternal-effect

allele	origin	discoverer	synonym	ref ^α	comments
<i>flil</i> ²³	EMS	Lefevre	<i>l(1)VE924</i>	5	lethal

^α 1 = Eeken, Sobels, Hyland, and Schalet, 1985, *Mutat. Res.* 150: 261-75; 2 = Homyk and Sheppard, 1977, *Genetics* 87: 95-104; 3 = Koana and Hotta, 1978, *J. Embryol. Exp. Morphol.* 45: 123-43; 4 = Lefevre, 1981, *Genetics* 99: 461-80; 5 = Lefevre and Watkins, 1986, *Genetics* 113: 869-95; 6 = Lifschytz and Falk, 1968, *Mutat. Res.* 6: 235-44; 7 = Lifschytz and Falk, 1969, *Mutat. Res.* 8: 147-55; 8 = Lifschytz and Yakobovitz, 1978, *Mol. Gen. Genet.* 161: 275-84; 9 = Perrimon, Smouse, and Miklos, 1989, *Genetics* 121: 313-31; 10 = Schalet and Lefevre, 1971, *Chromosoma* 44: 183-202; 11 = Schalet and Lefevre, 1976, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902;

cytology: Placed in the distal half of 19F based on deficiency mapping with deficiencies of unknown polytene breakpoints (Perrimon, Smouse, and Miklos, 1989, *Genetics* 121: 313-31).

flilJ: see *elav*

flIK

location: 1-30.1.

synonym: *l(1)9Fc*.

phenotype: Flying and jumping ability poor or absent; mutants tend to be generally inactive, including poor courtship; both alleles lead to stress-sensitive paralysis and are temperature sensitive, in that rearing at 29° leads to severe weakness and early death. Temperature sensitive throughout most of development. ERG normal (Homyk and Pye, 1989, *J. Neurogenet.* 5: 37-48).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>flIK</i> ¹	EMS	Homyk		1	
<i>flIK</i> ²	EMS	Homyk		1	
<i>flIK</i> ³	X ray	Lefevre	<i>l(1)A147</i>	2	<i>In(1)9F9;20</i>
<i>flIK</i> ⁴	X ray	Lefevre	<i>l(1)L49</i>	2	lethal
<i>flIK</i> ⁵	X ray	Lefevre	<i>l(1)N51</i>	2	lethal
<i>flIK</i> ⁶	EMS	Lefevre	<i>l(1)EM15</i>	2, 3	PA/NME
<i>flIK</i> ⁷	EMS	Lefevre	<i>l(1)VE686</i>	3	lethal
<i>flIK</i> ⁸	EMS	Lefevre	<i>l(1)VE828</i>	3	E/L
<i>flIK</i> ⁹	HMS	Kramers	<i>l(1)HM25</i>	4, 5	lethal

^α 1 = Homyk, Szidonya, and Suzuki, 1980, *Mol. Gen. Genet.* 177: 553-65; 2 = Lefevre, 1981, *Genetics* 99: 461-80; 3 = Lefevre and Watkins, 1986, *Genetics* 113: 869-95; 4 = Zhimulev, Belyaeva, Pokolkova, Kotchneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1981, *DIS* 56: 192-96; 5 = Zhimulev, Pokholkova, Bgatov, Semeshin, and Belyaeva, 1981, *Chromosoma* 82: 25-40.

cytology: Placed in 9F3-5 based on inclusion in *Df(1)HC133 = Df(1)9B9-10;9F2-5* but not *Df(1)ras-P14 = Df(1)9E1-2;9F3-4* (Zhimulev, Pokholkova, Bgatov, Umbetova, Solovjeva, Khudyakov, and Belyaeva, 1987, *Biol. Zentralbl.* 106: 699-720; 1987, *DIS* 66: 194-97).

***flightless*:** see *flit*

***flight defective*:** see *flit*

***flight reduced*:** see *flrd*

***flipper*:** see *flp*

****flil*: flyless**

location: 3- (not located).

origin: Spontaneous.

discoverer: Cercos, 41g15.

references: Andres, 1943, *DIS* 17: 48.

phenotype: Wings apparently normal, but fly cannot keep them spread and cannot fly more than a few inches. RK3.

***flo*: fiber loop (J.C. Hall)**

location: 2-58 (1% recombination with *cn*).

origin: Induced by ethyl methanesulfonate.

discoverer: Heisenberg and Fishbach.

synonym: *flo*^{N115}.

phenotype: Looped axon fiber present in axonal columns in distal part of medulla (second order optic lobe); nearly full penetrance (Heisenberg).

flp: see *flw*

****flp*: flipper**

location: 2-27 (based on location of *flp*^{hd}).

origin: Spontaneous.

discoverer: Mohr, 18h5.

references: Bridges and Mohr, 1919, *Genetics* 4: 304.

phenotype: Wings fail to expand, remain compact, very dark, extended, and curved slightly downward. Fly a wizened dwarf. Body surface dull and dark. Both sexes sterile. RK3.

***flp*^{hd}: flipper-hood**

origin: X ray induced.

discoverer: M. Simmons, 1972.

references: Craymer, 1980, *DIS* 55: 198.

phenotype: Like *flp* but fertile in both sexes. *flp*^{hd}/₁ + tends to have dusky, corrugated wings. RK3 as dominant; RK1 as recessive.

other information: Allelism inferred from location (27 vs 30) and phenotype.

***flr*: flare**

location: 3-38.8 (with respect to *h* and *th*).

origin: Induced by ethyl methanesulfonate.

references: Garcia-Bellido and Dapena, 1974, *Mol. Gen. Genet.* 128: 117-30.

phenotype: Chaetae and trichomes in thorax and abdomen abnormally shaped. Chaetae have rudimentary sockets, and shaft is frequently crooked and branched. Trichomes transformed into multiple short outgrowths which appear as swellings on wing cells and as rosettes in abdominal cuticle cells. Homozygotes are zygotic lethals but cell viable; homozygous clones may have reduced survival in thorax but not in abdomen. Excellent marker for homozygous *3L* clones.

alleles: Three alleles: *flr*¹, *flr*², and *flr*³. Lethal in all pairwise combinations.

***flrd*: flight reduced (J.C. Hall)**

A series of ethyl-methanesulfonate-induced, sex-linked mutants with reduced flying ability recovered by Homyk *et al.*. All were crudely mapped; complementation tests were performed among *flit*, *flrd*, and other behavioral mutants recovered in the same screens and mapping to the same interval. In this way 17 *flrd* loci, designated *flrdA* through *flrdR* (no K or Q) were identified; two others were not mappable. Allelism tests with *flit* mutants

were not performed.

references: Homyk and Sheppard, 1978, *Genetics* 87: 95-104.

Homyk, Szidonya, and Suzuki, 1980, *Mol. Gen. Genet.* 177: 553-65.

firdA

location: 1- 1.2.

phenotype: Flight weak; small amplitude of wing beats. Not allelic to *firdB* or *firdC*.

firdB

location: 1- 1.1.

phenotype: Flight weak; females may be more impaired than males; small amplitude of wing beats; jumping ability subnormal.

firdC

location: 1- 4.0.

phenotype: Flight weak; adults seem generally feeble, including poor jumping ability; 10-30% of adults fail to inflate wings normally; even when wings inflated, males seldom extend them fully when courting.

firdD

location: 1- 28.1.

phenotype: Weak flight when raised at 29°; flightless when raised at 22°; wing beats have small amplitude and wing behavior of courting males is aberrant; impaired jumping ability when raised at either temperature; mosaic analysis suggests primary defect could be in muscles (Homyk, 1977, *Genetics* 87: 105-28).

firdE

location: 1- 39.9.

phenotype: Weak flight; adults often fail to retract wings properly and walk holding one or both wings at 90° angle to body axis.

firdF

location: 1- 42.2.

phenotype: Flightless; jumping ability poor; mosaic analysis suggests primary defect could be in muscles (Homyk and Sheppard, 1977).

firdG

location: 1- 44.6.

phenotype: Weak flight; males may be more impaired than females; no wing beats; general locomotor activity weak, including poor jumping ability; adults begin dying around 10 days post eclosion; homozygous mutant females do not lay eggs (which is not necessarily due to same variant as that causing behavioral defects).

firdH

location: 1-51.0.

phenotype: Weak to very weak flight; small amplitude of wing beats in flight; weak jumping ability; all alleles lead to decreased viability, flying ability, or both when raised at higher temperatures; mutations are heat-sensitive male steriles, and at least one allele, *firdH*³, causes abnormalities of wing movements in courtship after rearing at 29°. TSP of *firdH*³ second and third larval instars (Homyk and Grigliatti, 1983, *Dev. Genet.* 4: 77-97).

alleles: Four mutant alleles: *firdH*¹, *firdH*², *firdH*³, and *firdH*⁴; *firdH*² (*firdH*^{2xk}) originally designated *firdK*.

firdJ

location: 1- 51.0.

phenotype: Very weak flight. Mosaic analysis suggests primary defect in thoracic muscles [Homyk, 1977, *Genetics* 87: 105-28 (described these as *firdI* rather than *firdJ*)].

firdI: see *l(1)adl16*

firdK: see *firdH*²

firdL

location: 1- 62.5.

phenotype: Weak flight; generally somewhat inactive.

firdM

location: 1- 23.6.

phenotype: Weak flight and jumping; adults generally somewhat inactive; some such individuals have poor landing response.

firdN

location: 1-38.0.

phenotype: Weak flight and jumping ability; more severe phenotype, including poor wing usage in male courtship, after rearing at 29°. Wings held up and out when raised at 29°. TSP for wing posture restricted to late third instar; that for flightlessness begins in second instar and extends until near end of pupal stage.

firdO

location: 1- 45.3.

phenotype: Weak to poor flying ability; *firdO*¹ is most severe of three alleles; wings tend to be held in aberrant positions, including by courting males (all alleles).

alleles: Three mutant alleles: *firdO*¹, *firdO*², and *firdO*³.

firdP

location: 1- 44.6.

phenotype: Flight and jumping ability weak; adults become uncoordinated briefly after mechanical shock.

other information: Complements *firdG* although mapping to the same position.

firdQ

location: 1- 56.7.

phenotype: Weak flying and jumping ability; wing beating in flight has small amplitude; adults generally somewhat inactive.

fird-R

location: 1- 58.6.

phenotype: Flight weak; generally somewhat inactive; some of the mutant individuals have melanotic spot under cuticle, near wing (not known to be due to same genetic change as that affecting flight).

fird-393: flight reduced-393 (J.C. Hall)

location: 1- not localized.

origin: Induced by ethyl methanesulfonate.

references: Homyk and Sheppard, 1977, *Genetics* 87: 95-104.

phenotype: Very weak flight; small amplitude of wing beats in flight; usage of wings by courting males abnormal.

other information: Position of mutation or mutations responsible for aberrant phenotypes said not to be resolvable.

flrd-397 (J.C. Hall)

location: 1- not localized.
origin: Induced by ethyl methanesulfonate.
references: Homyk and Sheppard, 1977, Genetics 87: 95-104.
phenotype: Weak flight; usage of wings by courting males abnormal.
other information: Position of mutation or mutations responsible for aberrant phenotypes said not to be resolvable.

flt: flight defective (J.C. Hall)

A series of ethyl-methanesulfonate-induced X-chromosome mutants isolated by Koana and Hotta (1978, J. Embryol. Exp. Morphol. 45: 123-43). All were crudely mapped and complementation tests performed among those mapping in the same interval. In this way 15 loci were identified and designated *fltA* through *fltO*. They were not tested for allelism with other collections of sex-linked flightless mutants such as *fli* or *flrd*; many cases of allelism between these series are likely.

fltA

location: 1- between *y* and *cho*.
phenotype: Impaired flight; jumping ability nearly normal.

fltB

location: 1- between *cho* and *cv*.
phenotype: Impaired flight; jumping ability nearly normal.

fltC

location: 1- between *cv* and *v*.
phenotype: Impaired flight, but when raised at 24°, some individuals fly normally; when raised at 29°, all are completely flightless; jumping ability nearly normal.

fltD

location: 1- between *cv* and *v*.
phenotype: Completely flightless; reduced jumping ability. Mosaic analysis suggests primary focus in thoracic muscles.
other information: Complements *flrdD*, *gmp*, and *sdby*.

fltE

location: 1- between *cv* and *v*.
phenotype: Impaired flight; jumping ability normal.

fltF

location: 1- between *cv* and *v*.
phenotype: Impaired flight; unable to take off from a surface; severely impaired jumping ability.

fltG

location: 1- between *cv* and *v*.
phenotype: Impaired flight; *fltG* has near-normal jumping ability; *fltG*² is normal in this behavior.
alleles: Two mutant alleles: *fltG*¹ and *fltG*².

fltH

location: 1- between *v* and *f*.
phenotype: Completely flightless; normal jumping ability; wavy myofibrils in flight muscles, with larger than normal diameter; thick and thin filaments sometimes disorganized with deficient Z bands; sarcomere length abnormal; mosaic analysis suggests primary defect in thoracic muscles.

fltI

location: 1- between *v* and *f*.
phenotype: Impaired flight; jumping ability nearly normal.

fltJ

location: 1- between *v* and *f*.
phenotype: Impaired flight; unable to take off from a surface; normal jumping ability.
alleles: Three mutant alleles: *fltJ*¹, *fltJ*², and *fltJ*³, the latter two weaker than the first.

fltK

location: 1- between *v* and *f*.
phenotype: Completely flightless; reduced jumping ability.

fltL

location: 1- between *v* and *f*.
phenotype: Completely flightless; normal jumping ability.

fltM

location: 1- between *f* and centromere.
phenotype: Impaired flight; jumping ability nearly normal.

fltN

location: 1- between *f* and centromere.
phenotype: Impaired flight; jumping ability nearly normal.

fltO: see *fliI*

flu: fluorouracil response

location: 3- not mapped.
origin: Naturally occurring polymorphism.
references: Duke and Glassman, 1968, Nature 220: 588-89.
 O'Byrne-Ring and Duke, 1980, Biochem. Genet. 18: 717-26.
phenotype: Resistant strain, *flu*^r, resistant to at least 0.0035% FU and shows resistance to FUDR as well. Sensitive strain, *flu*^s exhibits over 90% mortality following exposure to 0.0008% FU (6 × 10⁻⁵ M). Thymidilate synthetase levels of resistant strains four times those of sensitive strains.
alleles: Two alleles described: *flu*^r and *flu*² designating resistant and sensitive alleles.

Flu: Flutter (J.C. Hall)

location: 1-37.
origin: Induced by ethyl methanesulfonate.
references: Deak, Bellamy, Bienz, Dubuis, Fenner, Gollin, Rahmi, Ramp, Reinhardt, and Cotton, 1982, J. Embryol. Exp. Morphol. 69: 61-81.
phenotype: Flies heterozygous for one of the mutant alleles, *Flu*¹, have reduced flight ability, impaired jump response, and relatively slight disturbances in morphology of Z bands and myofibrils of indirect flight muscles; these *Flu*⁺ flies are also like Hyperkinetic (*Hk*) mutants in terms of agitated behavior after etherization; *Flu*¹/*Flu*¹ flies are more severely affected with respect to flight and *Hk*-like phenotype; *Flu*² is recessive in regard to flight and shaking defects; *Flu*¹ studied for indirect flight muscle proteins and has reduced amounts of at least two high-molecular-weight materials.
alleles: Two mutant alleles: *Flu*¹ and *Flu*².

fluff: see *ff*

fluorouracil response: see *flu*

fluted: see *fl*

Flutter: see *Flu*

flw: flap wing (J.C. Hall)

location: 1-31.

references: Deak, 1977, J. Embryol. Exp. Morphol. 40: 35-63.

Deak, Rahmi, Bellamy, Bienz, Blumer, Fenner, Gollin, Ramp, Reinhardt, Dubendorfer, and Cotton, 1980, Development and Neurobiology of *Drosophila*, (Siddiqui, Babu, Hall, and Hall, eds.). Plenum Press, New York and London, pp. 183-92.

phenotype: Wings held in lateral position, instead of dorsally, and parallel to substrate as in wild type; thorax has darkened longitudinal stripe; anterior scutellar bristles sometimes missing or doubled; eyes bulging and slightly roughened; head compressed; third antennal joint shortened; unable to fly; jumping ability poor; fibrillar muscles of thorax (dorsoventrals, dorsolongitudinals) are variably abnormal, ranging from disorganized to missing; tergal depressor of trochanter jump muscle is normal in light microscope; behavioral and muscular phenotypes affected by *flw*² may be due to primary defect in muscle primordia, concluded from mosaic experiment (Deak, 1977); at least two *flw* alleles (*flw*³ and *flw*⁴) cause reduced amount of a particular protein from indirect flight muscles (same molecule as that affected by *hdp*, *int*, *rsd*, and *up* mutations) (Deak, Bellamy, Bienz, Dubuis, Fenner, Gollin, Rahmi, Ramp, Reinhardt, and Cotton, 1982, J. Embryol. Exp. Morphol. 69: 61-81). *flw* is not allelic to *gmp*.

alleles:

allele	origin	discoverer	synonym
<i>flw</i> ¹		Waletsky, 1937	<i>flp</i>
<i>flw</i> ²		Hanratty	
<i>flw</i> ³	EMS		<i>flw</i> ⁶⁷¹
<i>flw</i> ⁴	EMS		<i>flw</i> ⁷²⁵
<i>flw</i> ⁵	EMS		<i>flw</i> ⁹⁴²

cytology: Placed in 9C on the basis of its inclusion in *Df(1)N110 = Df(1)9B3-4;9D1-2* but not *Df(1)C52 = Df(1)8E;9C-D* (Craymer and Roy, 1980, DIS 55: 200-04).

flyless: see *fll*

flz: filzig

location: 2-59.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82 (fig.)

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Embryonic lethal; denticles and hairs of larvae have abnormal fuzzy texture. Mouth hooks poorly developed.

alleles: Five, *flz*¹ and *flz*² (isolated as *IP* and *IIG*) retained.

cytology: Placed in 44F-46D by segmental aneuploidy.

***fm: fine macros**

location: 1-66.1.

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 86.

phenotype: Small fly with narrow abdomen and extremely

short, thin bristles. Males fertile; viability about 50% wild type. RK3.

other information: Possibly allelic to *lf* or *lfl*.

fmf: factor for male fertility

location: 1- 0.5 (more than 0.008 but less than 0.189 cM to right of *dor*).

origin: Locus inferred from infertility of males deficient for 2B7-12, i.e., *Df(1)st472/ly²Y67g = Df(1)2B6-8;2B11-12/Dp(1;Y)1A1;2B6-7*.

references: Aizenzon and Belyaeva, 1982, DIS 58: 3-7.

Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 190.

phenotype: Males poorly viable and sterile.

cytology: Inferred position 2B7-12.

Fmrf: FMRF amide homologous precursor

location: 2-{59}.

references: Nambu, Murphy-Erdosh, Andrews, Feistner, and Scheller, 1988, Neuron 1: 55-61.

phenotype: Encodes the precursor polypeptide that is processed to generate a nonapeptide, DPKQDFMRFamide, that shares homology with the FMRFamide neuropeptide from molluscs. Gene appears to be expressed from late embryogenesis through the adult stage (see also White, Hurteau, and Punsal, 1986, J. Comp. Neurol. 247: 430-38).

cytology: Placed in 46C by *in situ* hybridization; proximal to *Df(2R)eve1.27 = Df(2R)46C3-4;46C9-11*.

molecular biology: Clones isolated from library using an oligonucleotide probe based on the putative amino-acid sequence of the nonapeptide, which had been isolated. Gene encodes a single 1.7 kb message; cDNA clones sequenced; conceptual amino-acid sequence indicates a precursor protein of 347 amino acids; the sequence contains a putative signal sequence, five copies of DPKQDFMRF as well as ten additional peptides exhibiting various degrees of relatedness. Each peptide is flanked by single arginine cleavage sites and ends contain a carboxy-terminal glycine that can serve as the substrate for amidation.

***fnc: fine chaetae**

location: 1-34.9.

origin: Induced by S-2-chloroethylcysteine (CB. 1592).

discoverer: Fahmy, 1957.

references: 1959, DIS 33: 86.

phenotype: Extremely fine, short bristles. Body parts disproportionately reduced; reduction least marked on head and most marked on abdomen. Wings broad and slightly rounded at tips, occasionally with incisions of margin. Eyes slightly brighter red than normal. Males viable but sterile. RK3.

fo: folded

location: 1-63.

discoverer: Grossman, 1932.

references: 1934, DIS 1: 30.

phenotype: Wings remain unexpanded in a varying percentage of flies. Balancers shriveled; postscutellars bent forward. Overlaps wild type. RK3.

Fo: Forkoid

location: 2-107 (between *or* and *sp*).

origin: X ray induced.

discoverer: Mohler, 58c18.

references: 1960, DIS 34: 52.

phenotype: Heterozygote shows reduction in size of bristles and weak forking of head and posterior thoracic bristles. Using *Dp(2;3)P*, it may be shown that the expression of *+/+Fo < +/Fo < +/Fo/Fo; +/Fo/Fo* shows extreme forking of all bristles and is sterile. Homozygous lethal. *Fo* interacts with *f* alleles to produce extreme *f* bristles. RK1.

alleles: *Fo*¹ and *Fo*²; the latter induced by Angel with ethyl methanesulfonate; allelism based on phenotype and map position (1.4% to right of *bw*).

cytology: Located between 58E3 and 60B10, on basis of its inclusion in *Dp(2;3)P = Dp(2;3)58E3-F2;60D14-E2;96B5-C1* but not in *Df(2R)Px = Df(2R)60B8-10;60D1-2* (Mohler) or in the deficiency for the tip of 2R derived from *T(1;2)Bld = T(1;2)IC3-4;60B12-13* (Armentrout).

focal melanosis: see *me*

fog: folded gastrulation

location: 1-65.

references: Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307. Zusman and Wieschaus, 1985, Dev. Biol. 111: 359-71. Perrimon, Smouse, and Miklos, 1989, 121: 313-31.

phenotype: Hemizygous lethal; the cellular blastoderm apparently normal and gastrulation begins at normal time. No posterior midgut formed; germ band does not elongate onto dorsal side of embryo but it is thrown into a series of transverse ventral folds. Older embryos often twist around the longitudinal axis for one complete turn; many exhibit an anterodorsal hole through which the brain protrudes and a split in the posterior CNS and protrusion of the midgut. Hemizygous deficiency gives same phenotype. No effect of maternal genotype; homozygous germ line clones make eggs capable of supporting normal embryonic development. Fate mapping localizes *fog* lethal focus to the posterior pole most likely in presumptive posterior-midgut or proctodaeum cells.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>fog</i> ¹		Novitski	<i>l(1)114</i>	1, 3, 4, 5, 6, 8	
<i>fog</i> ²	EMS	Lifschytz	<i>l(1)M67</i>	2	on <i>y</i> ⁺ <i>Ymal</i> ⁺
<i>*fog</i> ³	X ray	Lefevre	<i>l(1)C88x</i>	1	<i>In(1)5E4-7;20</i>
<i>fog</i> ⁴	EMS		<i>fog</i> ^{4A6}	7, 8	
<i>fog</i> ⁵	EMS			7	
<i>fog</i> ⁶	EMS			7	
<i>fog</i> ⁷	EMS			7	
<i>fog</i> ⁸	EMS			7	
<i>fog</i> ⁹	EMS			7	
<i>fog</i> ¹⁰	EMS			7	
<i>fog</i> ¹¹	EMS			7	
<i>fog</i> ¹²	EMS			7	

^α 1 = Lefevre, 1981, Genetics 99: 461-80.; 2 = Lifschytz and Yakobovitz, 1978, Mol. Gen. Genet. 161: 275-84.; 3 = Perrimon, Smouse, and Miklos, 1989, 121: 313-31; 4 = Schalet and Lefevre, 1973, Chromosoma 44: 183-202; 5 = Schalet and Lefevre, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902; 6 = Schalet and Singer, 1971, DIS 46: 131-32; 7 = Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux Arch. Dev. Biol. 193: 172-86; 8 = Zusman and Wieschaus, 1985, Dev. Biol. 111: 359-71;

cytology: Localized to 20A-B.

***fol: folded wings**

location: 2-39.

origin: Spontaneous.

discoverer: Goldschmidt, 1937.

phenotype: Expanded wing folded. Overlaps wild type. RK3.

folded: see *fo*

folded gastrulation: see *fog*

folded wings: see *fol*

for: foraging

location: 2-10.

origin: Naturally occurring variants.

references: DeBelle, Hilliker, and Sokolowski, 1987, Behav. Genet. 17: 620-21. 1989, Genetics 123: 157-63.

phenotype: The major gene responsible for foraging strategy of feeding larvae. Larvae from so-called sitter strains move about 6.5 cm in five minutes while feeding on a yeasted surface, whereas those from so-called rover strains travel some 17 cm; heterozygotes are rovers. Several X-ray induced reversions of the dominant rover phenotype shown to be non-complementing lethals which map to 10 on chromosome 2, thus localizing a major behavioral locus.

alleles: Two alleles thus defined, *for*^R, the rover allele, and *for*^S, the sitter allele.

cytology: Placed in 24A3-C5 based on the lethality of the noncomplementing lethal alleles in combination with *Df(2L)ed-Sz = Df(2L)24A3-4;24D3-4* but not with *Df(2L)ed = Df(2L)24C3-5;25A1-4*.

foreclosed: see *fcl*

fork head: see *fkf*

forked: see *f*

Forkoid: see *Fo*

four jointed: see *fj*

four wheel drive: see *fwd*

fr: fringed

location: 2-80.

origin: Spontaneous.

discoverer: Bridges, 22c30.

references: 1938, DIS 9: 48.

phenotype: Wings often spread; wing margins snipped; bristles irregular and fringelike. Eyes small and rough. Midline of abdomen at slight angle to longitudinal axis of fly. Much variability in expression; safest criterion is wing margin irregularity. Viability and fertility variably reduced depending on allele. Character less extreme at low temperature.

alleles: Four spontaneous alleles recorded.

allele	discoverer	synonym	ref ^α	viability	fertility	RK
<i>*fr</i> ⁰	Bridges, 15a20		1, 2			3
<i>fr</i> ¹	Bridges, 22c30		2	16-90%	female nearly sterile	3
<i>fr</i> ²	Novitski, 37a22	<i>trm: trimmed</i>	2, 3, 5	good but erratic	female sterile	2
<i>*fr dibro</i>	Bridges, 17k19	<i>dibro</i>	2, 4	poor	sterile	3

^α 1 = Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. 278: 257 (fig.); 2 = CP627; 3 = Lewis, 1938, DIS 10: 55-56; 4 = Lynch, 1920, Genetics 4: 527-28; 5 = Novitski, 1937, DIS 8: 10, 13.

fragile chorion: see *fch*

frail: see *fi*

Frd: Freckled

location: 2-102.4 (2.1 units to the left of *bw*).

origin: X ray induced.

discoverer: M. G. Davis, 1961.

references: Erlich, 1963, DIS 37: 47.

Barigozzi, 1963, Proc. Intern. Congr. Genet., 11th., Vol. 1: 207.

1965, DIS 40: 64.

phenotype: Pupa and young fly characterized by accumulation of dark pigment; in older fly, pigment becomes concentrated in black specks scattered through body, head, and legs. Deposition of electron-dense material in nucleus and cytoplasm of fat cells begins during pupal stage; pericardial cells of adult become filled with electron-opaque granules and vacuoles (Perotti and Bairati, 1968, J. Insect Path. 10: 122-38). The occasional weak manifestation of the *Frd* phenotype transmitted from *Cy/Frd* males to their *Cy* offspring and their descendents led Barigozzi and Gira (1968, Genet. Res. 11: 141-50) to postulate an extra chromosomal component of the *Frd* phenotype. Homozygous lethal. RK2.

frh: fruh (T. Schüpbach)

location: 2-62.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach, and Wieschaus, 1989, Genetics 121: 101-17.

phenotype: Maternal-effect lethal mutant; homozygous females lay eggs which show no visible signs of development when observed under transmitted light in a stereo microscope; defective in fertilization or very early embryonic development. *frh*³ and *frh*⁶ sometimes give rise to embryos which form irregular blastoderms and develop very abnormally, producing fragmented pieces of cuticle.

alleles: Six; *frh*¹ through *frh*⁶ (isolated as *RO*, *HM*, *PH*, *PM*, *SB*, and *APL* respectively).

fringed: see *fr*

frizzled: see *fz*

fru: fruitless (J.C. Hall)

location: 3-62.0.

origin: X ray induced.

synonym: *fruity*.

references: Gill, 1963, DIS 38: 33.

1963, Am. Zool. 3: 507.

Hall, 1978, Behav. Genet. 8: 125-41.

Tompkins, Hall, and Hall, 1980, J. Insect. Physiol. 26: 689-97.

Hall, Siegel, Tompkins, and Kyriacou, 1980, Stadler Genet. Symp. 12: 43-82.

phenotype: Males court but do not mate with females; *fru* males court other males (especially when such flies also are homozygous for *fru*) and stimulate vigorous courtship on the part of *fru* or wild-type males. Mutant males

produce volatile compounds different from those generated by normal males, on criteria of gas chromatography and bioassays of behavioral effects of these compounds; females appear to be unaffected by *fru*, which is not allelic to *sr*.

cytology: Placed in 90C to 91A, based on uncovering by *Df(3R)P14*.

other information: Not allelic to *sr*.

Fructokinase: see *Hex-C*

fruh: see *frh*

frühe2: see *aur*

fruitless: see *fru*

fruity: see *fru*

fs 2.: see *fs(2)E*

fs(1)-: female sterile on chromosome 1

There have been three concerted searches for female sterile mutations on the X chromosome. The first to be reported is that of Gans, Audit, and Masson (1975, Genetics 81: 683-704), the second is by Mohler (1977, Genetics 85: 259-72), the third is by Komitopoulou, Gans, Margaritis, Kafatos, and Masson (1983, Genetics 105: 897-920). These series of mutants are designated *fs(1)A*, *fs(1)M*, and *fs(1)K* respectively. In designating loci the order of priority is *A>M1-M63>K*; e.g., all alleles of mutants first recovered (Gans *et al.*) are designated *fs(1)A-*, and the nominate allele is the one with the lowest acquisition number; alleles found by Audit are numbered, consecutively, alleles of Mohler are designated by the superscript M, and those by Komitopoulou by K. Allelism among these mutants has been incompletely tested; the number of loci is certainly lower than the tables indicate.

In addition to these extensive searches there are other female sterile mutants whose designations are more *ad hoc*.

fs(1)5: see *yl*

fs(1)5e

location: 1-37.5.

origin: Induced by ethyl methanesulfonate.

references: Perez-Chiesa, Cintron, and Morales, 1985, DIS 61: 215.

phenotype: Homozygous females sterile; males fertile. 95% of heterozygous females are fertile when reared at 25°, but only 5% are fertile when reared at 29°, or when reared at 25° but shifted for three days to 29°. Eggs produced are permeable to neutral red and burst when dechorionated in a 3% sodium hypochlorite solution.

Fs(1)10A

location: 1-33.52.

synonym: *hfs: haplo female sterile*.

references: Lefevre, 1969, Genetics 63: 589-600.

Zhimulev, Pokholkova, Bgatov, Semeshin, and Belyaeva, 1981, Chromosoma 82: 25-40.

phenotype: Locus inferred from the infertility of females heterozygous for vermilion deficiencies.

cytology: Placed in 10A8 by deficiency analysis; included in *Df(1)v^{65b} = Df(1)9F12-23;11A8-9* and *Df(1)RA37 = Df(1)10A5-6;10B17-C1* but not *Df(1)v^{L3} = Df(1)9F10;-*

10A7-8 or *Df(1)KA7 = Df(1)10A8-9;10F10*.

fs(1)11 to *fs(1)14*: see *otu*

fs(1)29: see *yl*

fs(1)42

location: 1-13.3.

origin: Induced by ethyl methanesulfonate.

discoverer: Denell, 1971.

references: Rizzo and King, 1977, *J. Morphol.* 152: 329-40 (fig.).

phenotype: Viability and longevity normal. Oogenesis in homozygous females proceeds through stage 8; chambers then degenerate. Dying follicle cells seen in chambers at all positions in ovarioles; profollicle cells also die in germaria and clusters of cystocytes delayed in achieving full complement of covering follicle cells. Egg chambers in vitellarium contain about 60% the normal number of follicle cells; these have greater lateral dimensions, and their nuclei and nucleoli are larger than normal. Follicular envelope of mutant chambers often contains gaps through which projections from cystocytes protrude.

fs(1)117: see *yl*

fs(1)205: see *yl*

fs(1)209: see *otu*

fs(1)288

origin: Induced by ethyl methanesulfonate.

references: Waring, DiOrio, and Hennen, 1983, *Dev. Biol.* 100: 452-63.

cytology: Placed in 12A6-D3 based on its inclusion in *Df(1)HA92 = Df(1)12A6-7;12D3*.

fs(1)445: see *yl*

fs(1)1001: see *otu*

fs(1)1163: see *Ypl^{ts1}*

fs(1)1304b: see *otu*

fs(1)1396: see *otu*

fs(1)1401: see *otu*

fs(1)1867

location: 1-62.9 (0.4 cM to the right of *car*).

origin: Induced with ethyl methanesulfonate.

references: Wieschaus, 1979, *Cell Lineage, Stem Cells, and Cell Determination* (Le Douarin, ed.), Elsevier/North Holland Biomedical Press, pp. 291-302. Szabad and Szydony, 1980, *Development and Neurobiology of Drosophila* (Siddiqui, Babu, Hall, and Hall, eds.), Plenum Press, New York, pp. 95-108.

phenotype: Homozygotes and hemizygotes exhibit delayed development and Minute-like or missing bristles; eclosion delayed about four days compared to heterozygous sibs; gynandromorphs delayed in development in proportion to quantity and independent of the quality of *fs(1)1867* tissue. Homozygous females sterile, never lay eggs; ovarioles develop normally until stage 10 after which degeneration and resorption occurs. Oocytes in *fs(1)1867* ovaries transplanted into wild-type hosts unable to develop, but *fs(1)1867* oocytes in wild-type ovaries do develop. *fs(1)1867* follicle cells fail to become columnar; defect concluded to be follicular.

fs(1)4077: see *otu*

***fs(1)A*: female sterile of Audit**

A series of ethyl-methanesulfonate-induced sex-linked recessive female sterile mutations first reported by Gans, Audit, and Masson (1975, *Genetics* 81: 683-704). The mutants were apportioned to the following phenotypic classes: Class I - females produce few if any eggs; Class II - females produce morphologically abnormal eggs; and Class III - females lay numerous normal appearing eggs. Class III is subdivided into IIIA for cases where fertilization by a sperm carrying the normal allele of the mutant for which the mother is homozygous rescues the eggs and produces an offspring and IIIB in which a sperm bearing a normal allele is unable to effect rescue. All classes have been described briefly by Gans *et al.*; Class II mutants have been described in more detail by Komitopoulou, Gans, Margaritis, Kafatos, and Masson [1983, *Genetics* 105: 897-920 (fig.)], and Class IIIB by Zalokar, Audit, and Erk [1975, *Dev. Biol.* 47: 419-32 (fig.)]. Information on mapping, allelism, and phenotypic class of these mutant presented in following table followed by phenotypic descriptions from above references.

<i>mutant</i>	genetic location	phenotype	allelism ^α	cytology
<i>fs(1)A59</i>		II	2A, 1K	8F-9A
<i>fs(1)A73</i>		IIIB	<i>fs(1)Ya</i>	
<i>fs(1)A97</i>	1-31	IIIB		
<i>fs(1)A99</i>		IIIA		
<i>fs(1)A107</i>		IIIA		
<i>fs(1)A116</i>		I	<i>otu</i>	
<i>fs(1)A117</i>		II		
<i>fs(1)A120</i>	1-46.9	II	6A	
<i>fs(1)A125</i>		II t.s.		
<i>fs(1)A147</i>	1-0	II	2A, 3M	1B10-1E3
<i>fs(1)A148</i>	1-48	II	<i>yl</i>	12E1-13A5
<i>fs(1)A180</i>	1-36	II t.s.		
<i>fs(1)A214</i>	1-38	IIIB	2A	
<i>fs(1)A231</i>		I	<i>otu</i>	
<i>fs(1)A248</i>		I		
<i>*fs(1)A265</i>		I	<i>otu</i>	
<i>*fs(1)A267</i>		II	<i>dec-1</i>	
<i>fs(1)A273</i>	1-65	II	1A, 1K, 1M	18E1-20A
<i>fs(1)A305</i>		II	<i>yl</i>	
<i>fs(1)A321</i>		II		
<i>fs(1)A330</i>		IIIB		
<i>fs(1)A331</i>		IIIB		
<i>fs(1)332</i>		II	<i>yl</i>	
<i>*fs(1)A336</i>		II		
<i>fs(1)A343</i>		II	<i>fs(1)A120²</i>	
<i>fs(1)A371</i>	1-0.3	II	<i>fs(1)N</i>	1E4-2B12
<i>fs(1)A379</i>		II	<i>fs(1)N</i>	
<i>fs(1)A383</i>	1-62	IIIB	2A	
<i>fs(1)A384</i>		II	<i>dec-1</i>	
<i>fs(1)A387</i>		IIIA		
<i>fs(1)A426</i>		IIIB		
<i>fs(1)A427</i>		I		
<i>fs(1)A436</i>		IIIA		
<i>fs(1)A456</i>	<i>ec-cv</i>	II		4F1-5A1
<i>fs(1)A457</i>	1-49	IIIA	1M	3A
<i>fs(1)A462</i>		I t.s.		
<i>fs(1)A473</i>	<i>ct-v</i>	II t.s.		7E-8A
<i>fs(1)A475</i>		IIIA		
<i>fs(1)A476</i>		IIIA		
<i>fs(1)A489</i>		II	<i>fs(1)A120³</i>	
<i>fs(1)A508</i>	1-18	II		7C3-D1 or 5D6-6E1
<i>fs(1)A543</i>		I t.s.		
<i>fs(1)A571</i>		II	<i>fs(1)A120⁴</i>	
<i>fs(1)A572</i>		IIIB		
<i>fs(1)A573</i>		IIIB	<i>gd</i>	
<i>fs(1)A1001</i>		I t.s.		

<i>mutant</i>	genetic location	phenotype	allelism ^α	cytology
<i>fs(1)A1010</i>		II		
<i>fs(1)A1024</i>		IIIA		
<i>fs(1)A1031</i>		IIIB	<i>fs(1)A214</i> ²	
<i>fs(1)A1038</i>		II	<i>fs(1)N</i>	
<i>fs(1)A1042</i>		IIIB		
<i>fs(1)A1057</i>		I	<i>sn</i>	
<i>fs(1)A1059</i>		II	<i>IA, IK</i>	4F1-5A2
<i>fs(1)A1061</i>		II	<i>yl</i>	
<i>fs(1)A1071</i>		IIIA	<i>r</i>	
<i>fs(1)A1074</i>		IIIA		
<i>fs(1)A1081</i>		II	<i>yl</i>	
<i>fs(1)A1103</i>		IIIB	<i>par</i>	
<i>fs(1)A1122</i>		IIIB	<i>par</i>	
<i>fs(1)A1130</i>		II	<i>yl</i>	
<i>fs(1)A1140</i>	1-62	IIIB		
<i>fs(1)A1145</i>		I t.s.	<i>fs(1)A248</i> ²	
<i>fs(1)A1162</i>		IIIB	<i>fs(1)A383</i> ²	
<i>fs(1)A1168</i>		IIIB	<i>fs(1)A457</i> ²	
<i>fs(1)A1182</i>		IIIB	<i>mh</i>	
<i>fs(1)A1186</i>		II	<i>yl</i>	
<i>fs(1)A1187</i>		IIIB		
<i>fs(1)A1190</i>		II	<i>fs(1)A120</i> ⁵	
<i>fs(1)A1198</i>		II	<i>fs(1)A120</i> ⁶	
<i>fs(1)A1203</i>		IIIB	<i>fs(1)A457</i> ³	
<i>fs(1)A1242</i>	1-26	IIIB		7E-8A
<i>fs(1)A1245</i>		I		
<i>fs(1)A1246</i>		I	<i>fs(1)A1245</i> ²	
<i>fs(1)A1248</i>		I	<i>fs(1)A1245</i> ³	
<i>fs(1)A1268</i>		II		
<i>fs(1)A1304</i>	1-19	I		
<i>fs(1)A1336</i>		II	<i>dec-1</i>	
<i>fs(1)A1361</i>		IIIA	<i>r</i>	
<i>fs(1)A1369</i>		II	<i>fs(1)A147</i> ²	
<i>fs(1)A1371</i>	1-9	IIIB	<i>3K, 1M</i>	
<i>fs(1)A1456</i>		IIIA t.s.	<i>fs(1)h</i>	
<i>fs(1)A1459</i>	v-f	IIIB	<i>2M</i>	
<i>fs(1)A1497</i>	1-13.7	IIIB	<i>swa</i>	
<i>fs(1)A1501</i>	1-20.7	II	<i>dec-1</i>	
<i>fs(1)A1502</i>		IIIB	<i>swa</i>	
<i>fs(1)A1509</i>		IIIB		
<i>fs(1)A1526</i>		IIIB		
<i>fs(1)A1528</i>		IIIB		
<i>fs(1)A1559</i>		IIIA		
<i>fs(1)A1561</i>	ct-v	II		9B-C
<i>fs(1)A1569</i>		I t.s.	<i>lz</i>	
<i>fs(1)A1578</i>	1-55	IIIB		
<i>fs(1)A1621</i>	1-11.7	I	<i>liz; stf</i>	4F1-5A1
<i>fs(1)A1630</i>		IIIA	<i>rA3</i>	

^α Mutants allelic to previously recognized or more recently renamed loci are listed as alleles of those loci. For newly recognized loci the numbers of subsequently identified alleles in the A, K, and M series are indicated.

fs(1)A59

phenotype: Eggs show limited flaccidity; chorion with slightly shortened dorsal appendages. Mosaic analysis with *fs(1)A59^{K1}* shows normal allele to function in somatic tissue (Perrimon and Gans, 1983, Dev. Biol. 100: 365-73).

fs(1)A97

phenotype: Homozygous females sterile at 29° but not 16°. Produce haploid embryos at 29° which form abnormal blastoderms.

fs(1)A99

phenotype: Cold sensitive; death occurs during larval stage; normal development at 29°.

fs(1)A107

phenotype: Development ceases late in embryogenesis.

fs(1)A117

phenotype: Embryogenesis arrested at various stages; low fecundity.

fs(1)A120

phenotype: Embryogenesis arrested at various stages; dependent on culture medium. Somatic-cell-specific function (Wieschaus, Audit, and Masson, 1981, Dev. Biol. 88: 92-103).

fs(1)A125

phenotype: Low fecundity; dorsal appendages fused; partially fertile at 16° and 23°.

fs(1)A147

phenotype: No initiation of development; fine structure of chorion normal.

fs(1)A180

phenotype: Fertile at 16° and 23°. Mosaic analysis suggests that normal allele functions in somatic tissue (Wieschaus, Audit, and Masson, 1981, Dev. Biol. 88: 92-103). Chorion ultrastructure normal (Komitopoulou, Gans, Margaritis, Kafatos and Masson, 1983, Genetics 105: 897-920).

fs(1)A214

phenotype: No evidence of cleavage; Feulgen-positive material accumulates during first two hr. No evidence that fertilization takes place regularly. (Figured by Zalokar, Audit, and Erk, 1975, Dev. Biol. 47: 419-32). *fs(1)A214*² fertile at 16° but not 29°.

fs(1)A248

phenotype: Adult phenotype normal but females retain eggs. *fs(1)A248*² females fertile at 16°.

fs(1)A273

phenotype: Chorion appendages of *fs(a)A273^{K1}* females show abnormal size and shape. About 5% of eggs produce adults. Mosaic results ambiguous (Perrimon and Gans, 1983, Dev. Biol. 100: 365-73).

fs(1)A321

phenotype: Homozygous females have short bristles; lay flaccid eggs.

fs(1)A330

phenotype: Time of developmental failure varies - at fertilization, blastoderm formation, gastrulation, or in embryo. Such development as does occur is abnormal.

fs(1)A331

phenotype: Various times of developmental arrest.

fs(1)A383

phenotype: Embryos apparently normal but few larvae hatch. Many eggs of *fs(1)A383*² females cease mitosis during first hour, forming a few polyploid nuclei; others reach blastoderm which displays a peculiar mosaicism of an area with large swollen nuclei sharply separated by a line of pycnotic nuclei from an area with nuclei having more condensed chromatin. Occasionally two such areas in succession divided by lines girdling egg. Preblastoderm mitoses diploid and synchronous.

fs(1)A387

phenotype: Embryos fail late in development; some rescue by fertilization with sperm carrying normal allele.

fs(1)A426

phenotype: Embryonic development arrested at various times during development at 29°; some adult survival at 16°.

fs(1)A427

phenotype: Phenotype of adults normal, but females retain eggs.

fs(1)A436

phenotype: Development fails at advanced stage of embryogenesis. Rescue effected by fertilization with sperm carrying normal allele.

fs(1)A456

phenotype: Mosaic analysis shows normal allele to function in somatic tissue. Chorion ultrastructure normal (Komitopoulou, Gans, Margaritis, Kafatos, and Masson, 1983, *Genetics* 100: 897-920).

fs(1)A457

phenotype: Haploid development to abnormal blastoderm or gastrula stages. Nuclei smaller than normal and their numbers in blastoderm may exceed 10,000. In at least one case a Y chromosome was seen suggesting proliferation of paternal pronucleus. Few develop to late embryonic stages.

fs(1)A462

phenotype: Adults normal but females retain eggs; partially fertile at 16°. Clones of homozygous germinal cells in heterozygous female produce oviposited eggs (Wieschhaus, Audit, and Masson, 1981, *Dev. Biol.* 88: 92-103).

fs(1)A473

phenotype: Eggs partially devoid of chorion; females fertile at 16° and 23°. Mosaic studies show gene function to be specific to soma (Wieschhaus, Audit, and Masson, 1981, *Dev. Biol.* 88: 92-103; Perrimon and Gans, 1983, *Dev. Biol.* 100: 365-73).

fs(1)A475

phenotype: Mutant females lay eggs at 23° but not at 29°. Fertilization with sperm carrying normal allele produces some progeny at 23°.

fs(1)A476

phenotype: Development arrested in late embryo; rescue occurs with sperm carrying normal allele.

fs(1)A508

phenotype: Adults display twisted bristles and delayed development. Eggs produced by homozygous females vary in size, often smaller than normal.

fs(1)A543

phenotype: Adults have short wings; homozygous females retain eggs at 29° but are fertile at 16°.

fs(1)A572

phenotype: Early blastoderm apparently normal; later at nuclear elongation nuclei appear constricted and some forced out of peripheral layer and into deeper regions of the egg. Excessive numbers of blastoderm nuclei

(>10,000), but not due to haploidy. Gastrulation appears normal; development arrested at time of first muscular contractions. Difficulties of nuclear separation in early cleavage stages observed.

fs(1)A1001

phenotype: Adults normal in appearance. Females retain eggs but are partially fertile at 16°. Mosaic results at 29° indicate mutant interferes with somatic function of normal allele.

fs(1)A1010

phenotype: Development arrested in second and third cleavage; in metaphase, excessive numbers of punctiform and filamentous chromosomes; spindles appear normal. A few escapers appear at 16°-18°; many are agametic.

fs(1)A1024

phenotype: Offspring of homozygous mothers usually die in late pupal stages; about 30% escapers have unexpanded wings and die within a few days of eclosion.

fs(1)A1042

phenotype: Development arrested in second and third cleavage; in metaphase, excessive numbers of punctiform and filamentous chromosomes; spindles appear normal. A few escapers appear at 16°-18°; many are agametic.

fs(1)A1059

phenotype: Egg development and chorion morphology of *fs(1)A1059^{K1}* females at 19° normal. At 29° chorion resembles that of previously identified chorion-protein-gene-amplification-defective mutants [*fs(1)K451* and *fs(1)K1214*]. At 29° follicle cells show at least a three-to-five-fold suppression of amplification of the chorion-protein genes on both the X and third chromosomes compared to 24° (Komitopoulou, Kouyanou, and Kafatos, 1986, *Dev. Genet.* 7: 75-80).

alleles: Although not explicitly stated, it seems that lack of complementation has been demonstrated in the following combinations: *fs(1)A1059¹/fs(1)A1059^{K1}* (Komitopoulou), *fs(1)A1059²/fs(1)A1059^{M1}*, and *fs(1)A1059^{K1}/fs(1)A1059^{M1}* (Mohler).

allele	origin	synonym	ref ^α
<i>fs(1)A1059¹</i>	EMS		1
<i>fs(1)A1059²</i>	EMS	<i>fs(1)A1371</i>	1
<i>fs(1)A1059^{K1}</i>	EMS	<i>fs(1)K575</i>	2
<i>fs(1)A1059^{M1}</i>	EMS	<i>fs(1)M60</i>	3

^α 1 = Gans, Audit and Masson, 1975, *Genetics* 81: 683-704; 2 = Komitopoulou, Gans, Margaritis, Kafatos, and Masson, 1983, *Genetics* 105: 897-920; 3 = Mohler and Carroll, 1984, *DIS* 60: 236-41;

fs(1)A1074

phenotype: At 29° eggs fail to develop; at 23° development ceases during embryogenesis or larval or even pupal stages. Some rescue at 23° by fertilization with sperm carrying the normal allele.

fs(1)A1140

phenotype: Preblastoderm formed more or less normally, but nuclei irregularly placed within periplasm; many polyploid mitoses. Gastrulation abnormal; death precedes onset of muscular movement.

fs(1)A1187

phenotype: Homozygous females fertile at 16° but not at 29°. Blastoderm abnormal containing haploid nuclei.

fs(1)A1242

phenotype: Eggs arrest after a few nuclear divisions; have polyploid groups of chromosomes or deeply stained masses of chromatin. Mosaic studies indicate germ-line function of normal allele (Perrimon and Gans, 1983, Dev. Biol. 100: 365-73).

fs(1)A1245

phenotype: Viability reduced when raised at 29° and development delayed 1-3 days depending on allele. Oogenesis normal until stage 11-12 of King [stage 9 for *fs(1)A1245*³]. Appendages of chorion absent. Mosaic analysis using *fs(1)A1245*² suggest germ line function of gene (Perrimon and Gans, 1983, Dev. Biol. 100: 365-73).

fs(1)A1304

phenotype: Most egg chambers of homozygous females degenerate after King stage 6; a few cysts escape to produce stage 14 oocytes which are retained in the ovariole. Vitellogenesis severely inhibited and uptake of protein from the hemolymph apparently impaired. (Khipple Mulligan and Rasch, 1980, J. Exp. Zool. 212: 343-54). Nucleoli of nurse cells morphologically aberrant (Khipple Mulligan and King, 1976, Int. J. Insect. Morphol. Embryol. 5: 127-35). Oocytes grow at reduced rate and become covered by multiple layers of follicle cells, which secrete abnormal egg coverings (Khipple Mulligan and King). Phenotype insensitive to ecdysone administration and pole-cell-transplantation in studies by Lamnissou and Galti-Douka (1986, Dev. Genet. 6: 239-46), (Khipple Mulligan and Rasch). Mosaic studies by Wieschaus, Audit, and Masson (1981, Dev. Biol. 88: 92-103) indicate that the gene functions in the germ line, presumably in the nurse cells.

fs(1)A1371

phenotype: Homozygous females produce few offspring at 29°; at 16° nuclei begin to degenerate in preblastoderm stage leading to irregular blastoderm and irregular gastrula. Claimed to be an allele of *fs(1)1059*^{K1} and *fs(1)1059*^{M1} by Mohler and Carroll (1984, DIS 60: 236-41) but phenotype is not concordant with such a claim.

fs(1)A1459

phenotype: Homozygous females produce progeny at 16° but not at 29°. Blastoderm develops normally at 29°, but at nuclear elongation stage, nuclei move irregularly from surface into periplasm; nuclei degenerate, cell wall formation irregular.

fs(1)A1509

phenotype: Blastoderm and early gastrulation normal; orifice of midgut remains widely separated from cephalic furrow, intervening space filled with yolky ooplasm covered with a layer of flat epithelium; area may subsequently evert exposing internal organs. Cyst-like diverticles formed within embryo. Developmental arrest at time of first muscular contractions. Primary defect is failure of germ band elongation.

fs(1)A1526

phenotype: Few nuclear divisions resulting in polyploid groups of chromosomes or in deeply stained masses of chromatin.

fs(1)A1528

phenotype: Most eggs remain unfertilized.

fs(1)A1559

phenotype: Developmental arrest in late embryo; a few larvae hatch.

fs(1)A1561

phenotype: Mosaic studies indicate somatic function of normal allele (Perrimon and Gans, 1981, Dev. Biol. 100: 365-73).

fs(1)A1578

phenotype: Most eggs remain unfertilized.

fs(1)A1621

location: 1-11.7.

origin: Induced with ethyl methanesulfonate.

synonym: *liz*; *snf*.

references: Gans, Audit, and Masson, 1975, Genetics 81: 683-704.

Gollin and King, 1981, Dev. Genet 2: 203-18.

Wieschaus, Audit, and Masson, 1981, Dev. Biol. 88: 92-103.

Perrimon and Gans, 1983, Dev. Biol. 100: 365-73.

Steinmann-Zwicky and Nöthiger, 1985, Cell 42: 877-87.

Cline, 1988, Genetics 119: 829-62

Oliver, Perrimon, and Mahowald, 1988, Genetics 120: 159-71.

Steinmann-Zwicky, 1988, EMBO J. 7: 3889-98.

phenotype: Homozygous females viable but semisterile; males normal. Young females produce ova which can be fertilized and develop normally into adults. Older females cease production of oocytes, producing at first only pseudo nurse cells and subsequently tumorous germlaria containing hundreds or thousands of cells of apparently germinal origin. Tumorigenesis takes place earlier at higher temperatures. *fs(1)A1621/Df(1)C159* hemizygous for mutant; more temperature sensitive; the germ-line phenotype more severe than in homozygote but viability seems unaffected (Gollin and King). Mosaic studies suggest that gene function is germ line autonomous (Wieschaus *et al.*; Perrimon and Gans); however, the leakiness of the mutant phenotype introduced an element of ambiguity, raising the possibility of a somatic contribution to oogenesis as well. Interactions with *Sxl* alleles studied by Cline (1989), Steinmann-Zwicky (1988), and Oliver *et al.* (1988). Fertility of *fs(1)A1621* homozygotes is rescued by the presence of *Sxl*^{M1}. *Trans* heterozygotes of *fs(1)A1621* or *Df(1)HC244* with *Sxl*^{J1} show very low viability when *fs(1)A1621* is inherited from the female; survivors are sterile and show patchy transformations to maleness, such as sex-comb bristles and pigmentation of tergites 5 and 6. Viability of heterozygotes produced by the reciprocal cross also reduced but less so; surviving heterozygotes display reduced fertility. Viability effects appear to arise mostly from a maternal effect of the gene, whereas masculinizing and sterility effects result from decreased zygotic expression. The maternal effect of *fs(1)A1621* also reduces survival of *Sxl*^{J1/+} daughters; survival is cold sensitive. The

presence of *Sxl^{M1}* rescues *fs(1)A1621/Sxl^{fl}* females; *Df(1)HC244*, *Sxl^{M1}/Sxl^{fl}* are viable and fertile without male transformations. *Sxl^{M1}* is also able to rescue *Sxl^{fl}/+* from the maternal effect of *fs(1)A1621/+*, and *fs(1)A1621* is able to rescue male viability and fertility of *Sxl^{M1}* in *fs(1)A1621 Sxl^{M1}* double mutants. In contrast to the *sis* genes, which also interact with *Sxl*, the zygotic dose of *fs(1)A1621* has little or no influence on the sexual phenotype of 2X;3A animals; moreover, *fs(1)A1621* interacts little if at all with *sis-a*. Thus although *fs(1)A1621⁺* clearly has a positive involvement in *Sxl⁺* functions, its precise placement in the sex determination hierarchy is currently unclear. It has been suggested that it is involved in the positive autoregulatory aspect of *Sxl* function that maintains female development. Females heterozygous for *fs(1)A1621* and either *ovo^{D1}*, *ovo^{D2}*, or *ovo^{D3}* have ovarian tumors (Oliver, Pauli, and Mahowald, 1990, Genetics 125: 535-50).

cytology: Placed in 4F7-12 based on its inferred inclusion in *Df(1)HC244 = Df(1)3E8;4F11-12* but not *Df(1)A113 = Df(1)3D6;4F7-8* (Steinmann-Zwicky, and Nöthiger, 1985; Cline, 1988).

fs(1)BP

location: 1-32.67.

discoverer: Bgatov.

phenotype: Adults normal; fertility lesion not characterized.

alleles: Six; *fs(1)BP¹* to *fs(1)BP⁶* isolated as *F58*, *F129*, *F403*, *F417*, *F456*, and *F469*, respectively.

cytology: Placed in 9F3-5 based on inclusion in *Df(1)HC133 = Df(1)9B9-10;9F2-5* but not *Df(1)ras-P14 = Df(1)9E1-2;9F3-4* (Zhimulev, Pokholkova, Bgatov, Umbetova, Solovjeva, Khudyakov, and Belyaeva, 1987, Biol. Zentralbl. 106: 699-720; 1987, DIS 66: 194-97).

fs(1)cor-36: see *Cp-36^{nl}*

fs(1)C

A small series of ethyl-methanesulfonate induced mutants produced and partially characterized by D. Mohler and A. Carroll (1984, DIS 60: 236-41).

mutant	location	synonym	phenotype	cytology
<i>fs(1)C1</i>		dec-1		7C1
<i>fs(1)C2</i>		dec-2		7E10-8A5
fs(1)C3	cv-v		no eggs laid; chorion thin with short appendages	8B-C
fs(1)C4	cv-v		no eggs laid; thin chorion disorganized ovaries	
fs(1)C5	cv-v		no eggs laid; thin chorion disorganized ovaries	
fs(1)C6	cv-v		no eggs laid; thin chorion disorganized ovaries	

fs(1)de: female sterile (1) defective eggs

A subset of hybrid-dysgenesis-induced, X-linked female-sterile mutations that are characterized by the production of morphologically abnormal eggs that fail to hatch [Orr, Galanopoulos, Romano, and Kafatos, 1989, Genetics 122: 847-58 (fig.); see also Galanopoulos, Orr, Szabad and Kafatos, 1989, Dev. Genet. 10: 87-97 (fig.)]. The loci are numbered from 1 through 17 with alleles of *fs(1)del* designated by lower case letters in the superscript. The X chromosomes in these lines harbor multiple P elements, thus complicating cytological localization.

No case of allelism with previously identified sex-linked female sterile mutations and one case of allelism to a known lethal mutation in the *BRC* detected. Various defects in respiratory appendages and chorionic architecture described in above references.

locus	location	number of alleles	comments
fs(1)de1	near <i>cv</i>	7	germ line dependent
fs(1)de2	<i>f-car</i>	1	
fs(1)de3	near <i>car</i>		
fs(1)de4			double mutant?
fs(1)de5	<i>f-car</i>	1	
fs(1)de6	<i>m-f</i>		
fs(1)de7	near <i>car</i>		
fs(1)de8	<i>f-car</i>	1	
fs(1)de9	near <i>car</i>	1	
fs(1)de10	<i>m-f</i>	1	germ line dependent
fs(1)de11	<i>ct-m</i>	1	
<i>fs(1)de12</i>	<i>sc-ec</i>	1	npr^{fs} ; soma dependent
fs(1)de13	<i>m-f</i>		
fs(1)de14	<i>m-f</i>		
fs(1)de15	near <i>car</i>		
fs(1)de16	<i>m-f</i>		
fs(1)de17	<i>f-car</i>	1	germ line dependent

fs(1)h: female sterile (1) homeotic

location: 1-21.65 (0.05 cM to the left of *mys*).

synonym: *l(1)7Da*; *fsh*.

references: Gans, Audit, and Masson, 1975, Genetics 81: 683-704.

Zalokar, Audit, and Erk, 1975, Dev. Biol. 47: 419-32 (fig.).

Gans, Forquignon, and Masson, 1980, Genetics 96: 887-902.

Forquignon, 1981, Wilhelm Roux's Arch. Dev. Biol. 190: 132-38.

Digan, Haynes, Mozer, Dawid, Forquignon, and Gans, 1986, Dev. Biol. 114: 161-69.

phenotype: Many mutant alleles at this locus are nonconditional lethals, but there are three temperature-sensitive hypomorphic alleles. *fs(1)h¹* is the weakest of these and many of the phenotypic studies have been carried out on it and on *Df(1)C128*, which is deficient for the locus. Temperature-sensitive genotypes display two periods of sensitivity to elevated temperature; one is during oogenesis and affects embryonic development and the other is during the pupal stage and affects adult survival. Adult survival of *fs(1)h¹* homozygotes and *fs(1)h¹/Df(1)C128* heterozygotes is normal at 20° and 25°, but 10% and 0 respectively at 28.5°. As mentioned above, the TSP for lethality is the pupal stage. *fs(1)h¹⁵* shown to be lethal in germ-line clones (Perrimon, Engstrom, and Mahowald, 1984a, Dev. Biol. 105: 404-14). Fertility of *fs(1)h* homozygotes and *fs(1)h/Df(1)C128* temperature sensitive with the former becoming completely sterile at 29° and the latter at 25°; temperature-sensitive periods for *fs(1)h/Df(1)C128* females during pregastrulation embryogenesis and perhaps late oogenesis. Eggs laid at restrictive temperatures exhibited normal preblastoderm development, although about half the nuclei are haploid; nuclear behavior during blastoderm formation disrupted and development arrested in blastoderm or early gastrulation. Larvae and adults surviving semi-permissive temperatures display homeotic anomalies; dead embryos, and newly hatched larvae frequently have missing thoracic

and anterior abdominal segments; surviving adults exhibit a sex ratio skewed in favor of males and frequently show missing halteres and less frequently third legs; effects are more severe in progeny of *fs(1)h/Df(1)C128* than homozygous *fs(1)h* females, in daughters than sons, and at 25° than 23°; small areas of homeotic transformations of anterior metathoracic to anterior mesothoracic structures also seen. The maternal effect of *fs(1)h* interacts synergistically with *Ubx*¹³⁰, *Df(3R)red*, and *trx*, but not *bx*³ or *pbx*.

alleles: Most alleles are nonconditional lethals; fine structure mapping places two nonconditional alleles tested distal to the three tested conditional alleles. Relative strengths of the mutant alleles estimated as follows: *Df(1)C128* = *fs(1)h*⁵ = *fs(1)h*⁴ > *fs(1)h*¹⁸ > *fs(1)h*³ > *fs(1)h*⁸ > *fs(1)h*¹⁷ > *fs(1)h*² > *fs(1)h*⁶ > *fs(1)h*¹, the last three being conditional.

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>fs(1)h</i> ¹	EMS		<i>fs(1)A1456</i>	3	temperature sensitive
<i>fs(1)h</i> ²	EMS			1	temperature sensitive
<i>fs(1)h</i> ³	EMS			1	
<i>fs(1)h</i> ⁴	EMS			1	8 kb insert in 38.4 to 38.9 kb
<i>fs(1)h</i> ⁵	HD		<i>l(1)R10.4</i>	1	Inversion and deletion
<i>fs(1)h</i> ⁶	EMS		<i>fs(1)M16, fsh^M</i>	6, 7	temperature sensitive
<i>fs(1)h</i> ⁷	X ray	Lefevre	<i>l(1)HF339</i>	4	<i>T(1;3)7D;75C-D1</i>
<i>fs(1)h</i> ⁸	X ray	Lefevre	<i>l(1)N72</i>	4	<i>T(1;3)7D3-4;61F</i> ; breakpoint in 43.1 to 46.8 kb
<i>fs(1)h</i> ⁹	X ray	Lefevre	<i>l(1)RA50</i>	4	<i>ln(1)7D3;18E5-F1</i>
<i>fs(1)h</i> ¹⁰	X ray	Lefevre	<i>l(1)RF29</i>	4	<i>T(1;2;3)3D3-4;7D3-6;41;93F</i>
<i>fs(1)h</i> ¹¹	EMS	Lefevre	<i>l(1)DA548</i>	5	
<i>fs(1)h</i> ¹²	EMS	Lefevre	<i>l(1)DF911</i>	5	
<i>fs(1)h</i> ¹³	EMS	Lefevre	<i>l(1)EF422</i>	5	
<i>fs(1)h</i> ¹⁴	EMS	Lefevre	<i>l(1)EF491</i>	5	<i>ln(1)7C9-D1;20</i>
<i>fs(1)h</i> ¹⁵	EMS	Lefevre	<i>l(1)VA75</i>	5	
<i>fs(1)h</i> ¹⁶	EMS	Lefevre	<i>l(1)VE728</i>	5	
<i>fs(1)h</i> ¹⁷	spont	Schalet	<i>l(1)16-18, fsh^S</i>	8	copied-like insert in 45.4 to 47.8 kb
<i>fs(1)h</i> ¹⁸	HD		<i>l(1)26/62A, fsh^E</i>	2	two copies of 37 to 47 kb in tandem separated by 0.8 kb

^α 1 = Digan, Haynes, Mozer, Dawid, Forquignoni, and Gans, 1986, Dev. Biol. 114: 161-69; 2 = Eeken, Sobels, Hyland, and Schalet, 1985, Mutat. Res. 150: 261-75; 3 = Gans, Audit, and Masson, 1975, Genetics 81: 683-704; 4 = Lefevre, 1981, Genetics 99: 461-80; 5 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 6 = Mohler, 1975, Genetics 85: 259-72; 7 = Mohler and Carroll, 1984, DIS 60: 236-41; 8 = Schalet, 1986, Mutat. Res. 163: 115-44.

^β Origin of molecular coordinates a *SacI* site just proximal to the proximal breakpoint of *Df(1)C128* = *Df(1)7D1;7D5-6*, positive values to the left.

cytology: Localized to 7D1-5 based on its inclusion in *Df(1)C128* = *Df(1)7D1;7D5-6*.

molecular biology: Region cloned in an 120 bp chromosome walk from a genomic clone shown to be deleted by *Df(1)C128*. Southern blots identify lesions associated with four alleles, which map to positions 39 to 51 kb distal to the proximal breakpoint of *Df(1)C128*. Poly(A)⁺RNAs migrating as a doublet at 7.6 kb and a band at 5.9 kb are transcribed from this region in ovaries; these transcripts are present in 0 to 4 h embryos but are not detectable at later stages. During pupal stages a 2.4 kb transcript is detected. The largest RNAs are derived from a 20-kb chromosomal region encompassing the sites of all mapped *fs(1)h* alleles. The *fs(1)h* transcription unit is less than 500 base pairs distal to that of *mys*.

fs(1)K10

location: 1-0.5 (based on 191 recombinants between *y* and *w*).

origin: Induced by ethyl methanesulfonate.

references: Wieschaus, Marsh, and Gehring, 1978, Wilhelm Roux's Arch. Dev. Biol. 184: 75-82 (fig.).

Wieschaus, 1980, Development and Neurobiology of *Drosophila* (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York and London, pp. 89-94 (fig.).

phenotype: Homozygous females lay eggs with hyperplasia of the anterior chorionic appendages to form a collar around the micropile. Pole-cell-transplantation studies demonstrate chorionic phenotype to depend on germ-line genotype; pattern of overlying follicle cells apparently depends on cues from oocyte. *In situ* hybridization and antibody staining indicate that expression is confined to the primary oocyte and that the protein product is sequestered in the oocyte nucleus (Prost, Deryckere, Roos, Haenlin, Pantescio, and Mohier, 1988, Genes Dev. 2: 891-900). Eggs of mutant females seldom fertilized; those that are exhibit abnormalities of gastrulation with the anterior ends showing dorsal patterns of development both ventrally and laterally. Some larvae produced with dorsal cuticular pattern covering entire circumference; last few segments have normal ventral hypoderm pattern. Ventralizing mutants *grk* and *top* both epistatic to the dorsalizing effects of *fs(1)K10* (Schüpbach, 1987, Cell 49: 699-707). Germ-line mosaics produced by mitotic exchange indicate *fs(1)K10* activity required during oogenesis (Marsh, Wieschaus, and Gehring, 1976, Experientia 32: 803). Ovarian clones of homozygous cells useful in investigating kinetics of oogenesis (Wieschaus and Szabad, 1979, Dev. Biol. 68: 29-46).

alleles: Four ethyl-methanesulfonate-induced alleles; *fs(1)K10*¹ isolated by Wieschaus *et al.* (1978); *fs(1)K10*², *fs(1)K10*³, and *fs(1)K10*⁴ isolated as *fs(1)M9* by Mohler (Mohler, and Carroll, 1984, DIS 60: 236-41).

cytology: Placed 2E2-F1 based on its being deleted by *Df(1)278* = *Df(1)2E2-F3;3A5-B4* but not *Df(1)2F1;3A4*. (Perrimon, Engstrom, and Mahowald, 1984, Genetics 108: 559-72).

molecular biology: Region cloned in a 200-kb chromosomal walk initiated from cloned sequences from microdissected polytene region 2E2-F1 (Haenlin, Steller, Pirrotta and Mohier, 1985, Cell 40: 827-37). *fs(1)K10* mutants rescuable by transformation with a 5-kb sequence. This sequence detects mRNA molecules of four different sizes on Northern blots; all have apparently the same initiation sites; species of 3.1 and 2.8 kb have different polyadenylation sites and both have had an 854-bp intron removed; a 4.0-kb species retains the intron and has the same polyadenylation site as the 3.1 kb molecule; a low-abundance mRNA of 6.0 kb retains the intron and extends beyond the known polyadenylation sites. The 3.1 and 2.8 messages are expressed in the ovaries and early embryos; the two larger transcripts are more generally expressed. Transcription from left to right (Haenlin, Roos, Cassab, and Mohier, 1987, EMBO J. 6: 801-07). Sequencing of cDNA clones yields a conceptual polypeptide of 463 amino acid residues of 51.5 kd and an isoelectric point of pH 11.2; the molecule has three domains: the amino terminal domain up to residue

225 contains seven eight-residue repeats with consensus sequence GlnGlnGlnHisProSerPro and variants thereof; the middle domain of ca. 112 amino acids is apolar, containing 37% proline; the carboxyterminus contains a helix-turn-helix motif in residues 390-418 (Prost *et al.*).

fs(1)K79–fs(1)K1563: Female sterile of Komitopoulou

A series of ethyl-methanesulfonate-induced, sex-linked-recessive female-sterile mutants reported by Komitopoulou, Gans, Margaritis, Kafatos, and Masson (1983, Genetics 105: 897-920). The same classification scheme as used in the *fs(1)A* series was employed for *fs(1)K* mutants. Only class II mutants have been characterized. Information on mapping, allelism, and phenotypic class are presented in the accompanying table; additional phenotypic description follows the table.

<i>mutant</i>	genetic location	phenotype	allelism ^α	cytology
<i>fs(1)K79</i>		II		<i>8E-9B1</i>
<i>fs(1)K93</i>	near <i>sc</i>	II		<i>3C4-11</i>
<i>fs(1)K155</i>			<i>ovo</i>	
<i>fs(1)K163</i>	near <i>ct</i>	II		
<i>fs(1)K184</i>		II	<i>yl</i>	
<i>fs(1)K254</i>	1-17	II t.s.		<i>5D5-E1</i>
<i>fs(1)K294</i>		II	<i>yl</i>	
<i>fs(1)K313</i>		II	<i>fs(1)A59 K1</i>	
<i>fs(1)K418</i>		II	<i>sn</i>	
<i>fs(1)K451</i>	near <i>g</i>	II		<i>12A6-D3</i>
<i>fs(1)K467</i>		II	<i>dec-1</i>	
<i>fs(1)K473</i>		II	<i>sn</i>	
<i>fs(1)K499</i>		II	<i>fs(1)A1459 K1</i>	
<i>fs(1)K524</i>		I		
<i>fs(1)K575</i>		II t.s.	<i>fs(1)A1059 K1</i>	<i>4F-5A2</i>
<i>fs(1)K621</i>		II	<i>yl</i>	
<i>fs(1)K646</i>	1-15	II	<i>fs(1)M3 K1</i>	<i>5C5-D6</i>
<i>fs(1)K718</i>		II	<i>dec-1</i>	
<i>fs(1)K741</i>	<i>cv-ct</i>	I t.s.		<i>5D-6E</i>
<i>fs(1)K743</i>		II	<i>sn</i>	
*<i>fs(1)K811</i>		II		
<i>fs(1)K1075</i>		II	<i>fs(1)A273 K1</i>	
<i>fs(1)K1090</i>		II	<i>dec-1</i>	
<i>fs(1)K1124</i>		II	<i>dec-1</i>	
<i>fs(1)K1103</i>			<i>ovo</i>	
<i>fs(1)K1134</i>		I		
<i>fs(1)K1193</i>	rt. of <i>g</i>	II		
<i>fs(1)K1214</i>	1-18.17	II		<i>5D5-E1</i>
<i>fs(1)K1221</i>		I		
<i>fs(1)K1232</i>		II	<i>dec-1</i>	
<i>fs(1)K1237</i>			<i>ovo</i>	
<i>fs(1)K1274</i>		I t.s.		
<i>fs(1)K1281</i>		II	<i>fs(1)N</i>	
<i>fs(1)K1347</i>		II	<i>fs(1)N</i>	
<i>fs(1)K1421</i>		II	<i>sn</i>	
<i>fs(1)K1511</i>		II	<i>dec-1</i>	
<i>fs(1)K1540</i>		II	<i>fs(1)N</i>	
<i>fs(1)K1563</i>	1-44.7	II t.s.		<i>12D3-E1</i>

^α Mutants allelic to previously recognized or more recently renamed loci are listed as alleles of those loci.

fs(1)K79

phenotype: Actually *fs(1)K79* females, either homozygous or hemizygous, are fertile, but their eggs display short and fragile dorsal appendages.

fs(1)K93

phenotype: Homozygous females produce flaccid eggs, which readily take up neutral red.

fs(1)K163

phenotype: Survival severely reduced and development delayed; wings short and thin. Eggs of homozygous females flaccid; embryogenesis blocked at various stages; rescue effected by sperm carrying normal allele.

fs(1)K254

phenotype: Pupal lethal at 29°; female survival reduced at 23° and 25°. Female fertility reduced at 23° and abolished at 25° and 29°. Eggs of homozygous females have short thin dorsal appendages; ultrastructure of chorion abnormal; chorion defects not temperature sensitive. Mosaic analysis shows gene to function in somatic tissue (Perrimon and Gans, 1983, Dev. Biol. 100: 365-73).

fs(1)K451

phenotype: Reduced viability, especially of females; abnormalities of posterior legs, also especially in females. *fs(1)K451/Df(1)g-l* does not survive. Homozygous females produce eggs with short thin dorsal appendages and ultrastructurally defective chorions. All major chorion proteins underproduced owing to failure of chorion protein gene amplification. *Cp* genes on the third chromosome more severely affected than those on the *X* (Orr, Komitopoulou, and Kafatos, 1984, Proc. Nat. Acad. Sci. USA 81: 3773-77). In mosaic experiments *fs(1)K451* behaves as a locus required in both somatic and germinal tissues (Perrimon and Gans, 1983, Dev. Biol. 100: 365-73).

fs(1)K524

phenotype: At 29° eyes rugose and bristles short and fine; abdominal etching as well at 20°. Ovaries extremely atrophied, resembling ovaries without germ cells; however, occasional stage 14 cysts encountered.

fs(1)K741

phenotype: Viability good; ovaries atrophied at 29°. Female fertile at 23°.

****fs(1)K811***

phenotype: Low fecundity; dorsal appendages of chorion may be fused; chorion ultrastructurally normal.

fs(1)K1134

phenotype: Follicular development arrested at stage 8; transformation of larval to adult fat body appears to be blocked. May be a juvenile hormone deficiency.

fs(1)K1193

phenotype: Viability normal; homozygous females produce somewhat flaccid eggs. Development rarely proceeds to pupal stage. Mosaic studies indicate that gene functions in germ line (Perrimon and Gans, 1983, Dev. Biol. 100: 365-73).

fs(1)K1214

phenotype: Viability and fecundity of homozygous females reduced; sterility virtually complete, but a few progeny escape. Dorsal appendages of chorion short and thin. All major chorion proteins underproduced owing to a defect in amplification of *Cp* genes on both the *X* and chromosome 3 (Orr, Komitopoulou, and Kafatos, 1984, Proc. Nat. Acad. Sci. USA 81: 3773-77). Mosaic analysis indicates somatic function of gene (Perrimon and Gans, 1983, Dev. Biol. 100: 365-73).

fs(1)K1221

phenotype: Oogenesis normal but mature eggs retained by homozygous female.

fs(1)K1274

phenotype: Viability good at 29° but ovaries of homozygous females severely atrophied, probably with tumorous follicles. At 25° cysts contain 16 nurse cells and no oocyte; females fertile when raised at 23°. Mosaic studies indicate germ-line function of gene (Perrimon and Gans, 1983, Dev. Biol. 100: 365-73).

fs(1)K1563

phenotype: Viability normal; eggs produced by homozy-

gous females have thin, short dorsal chorionic appendages at 29° but not 23°; sterile at both temperatures; partially dominant at 29°. Chorion ultrastructurally abnormal. Mosaic studies indicate somatic function for gene (Perrimon and Gans, 1983, Dev. Biol. 100: 365-73).

fs(1)L186-fs(1)L211: see *yl*

fs(1)M: female sterile of Mohler

A series of ethyl-methanesulfonate-induced sex-linked-recessive female-sterile mutants reported by Mohler (1977, Genetics 85: 259-72; Mohler and Carroll, 1984, DIS 60: 236-41). The mutants were mapped and tested for complementation. The accompanying table lists the complementation groups.

THE GENOME OF *DROSOPHILA MELANOGASTER*

locus	genetic location	#M alleles α	allelism β	cytology	phenotype			
					oviposits	eggs develop	rescue γ	stage of arrest
fs(1)M1	1-10.2	1			+			
fs(1)M2	1-1	5			+	+		preblastoderm
fs(1)M3	1-15	5		5C5-D6				
fs(1)M4	1-0	6			+	+		preblastoderm
<i>fs(1)M5</i>		3	fs(1)A147 M1					
<i>fs(1)M6</i>		5	fs(1)N					
<i>fs(1)M7</i>		1	mk					
fs(1)M8	y-cv	1			+	+		leaky; rescuable
<i>fs(1)M9</i>		3	fs(1)K10					
<i>fs(1)M10</i>		8	pcx					
fs(1)M11	y-cv	1			+	-		leaky
<i>fs(1)M12</i>		3	fs(1)Ya					
fs(1)M13	1-14	5		5C7-D6	+	+		leaky, fragile chorions; some eggs collapse
fs(1)M14	near cv	2		5A8-C1	+	-		
fs(1)M15	near cv	2			+	+	+	
<i>fs(1)M16</i>			fs(1)h					
fs(1)M17	1-15	1			+	+		preblastoderm
<i>fs(1)M18</i>		6	gd					
fs(1)M19	1-37	11		11A3-B2	+			
fs(1)M20	v-f	2		12E1-13A5	+	+	+	preblastoderm
fs(1)M21	v-f	1			+	+	+	preblastoderm
<i>fs(1)M22</i>		1	fs(1)A457 M1					
<i>fs(1)M23</i>		2	fs(1)A1459 M1					
fs(1)M24	v-f	3			+	+	+	
<i>fs(1)M25</i>		14	yl					
fs(1)M26	v-f	2		10A4-B3	+	+		leaky
fs(1)M27	v-f	1			+	+	+	
fs(1)M28	v-f	1			+	+	+	
fs(1)M29	1-53	6			+	+	+	postgastrula
fs(1)M30	v-f	1			+	-		leaky; collapsed eggs
fs(1)M31	near f	1			+	?		fragile chorions
fs(1)M32	near f	2			+	+	+	leaky
fs(1)M33	near f	1			+	-		eggs collapse with fragile chorions
<i>fs(1)M34</i>		15	r					
<i>fs(1)M35</i>		1	Yp2	9A-B	+	?		
fs(1)M36	1-58	6			+	+		pregastrula; some eggs collapse
<i>fs(1)M37</i>		5	mei-41					
fs(1)M38	1-10.2	1			+	-		eggs collapse; fragile chorion
fs(1)M39	cv-v	1			+	?		
fs(1)M40	y-cv	1			+	+	+	pregastrula
fs(1)M41	y-cv	1			+	+	+	
<i>fs(1)M42</i>		2	dnc					
fs(1)M43	cv-v	1		10B1-17 δ	+	-		
<i>fs(1)M44</i>		3	swa					
<i>fs(1)M45</i>		4	sn					
fs(1)M46	near v	2		10A4-B3	+	-		
<i>fs(1)M47</i>		1	pt					
fs(1)M48	cv-v	1			+	-		eggs collapse; fragile chorions
fs(1)M49	cv-v	3		9A	+	-		some eggs collapse
<i>fs(1)M50</i>			cin					
fs(1)M51	v-f	1			+	-		small eggs; some collapse; fragile chorions
fs(1)M52	cv-v	1		8E-F	+	-		leaky
fs(1)M53	near v	1			+	+		pregastrula
fs(1)M54	v-f	1			+	+		
fs(1)M55	near f	1			+	-		leaky, some eggs collapse
fs(1)M56	v-f	1			+	+	+	
fs(1)M57	f-sfa	4			+	+		postgastrula, some eggs collapse; fragile chorion
fs(1)M58	f-sfa	1			+	+	+	
fs(1)M59	f-sfa	1			+	+	+	
<i>fs(1)M60</i>		1	fs(1)1059 M1					
fs(1)M61								
fs(1)M62	cv-v	1		10A4-B3	+			

locus	genetic location	#M alleles α	allelism β	cytology	phenotype			
					oviposits	eggs develop	rescue γ	stage of arrest
<i>fs(1)M63</i>	v-f	2			+	-		leaky; small eggs
<i>fs(1)M64</i>	y-cv	1			+			
<i>fs(1)M65</i>	y-cv	1			+			
<i>fs(1)M66</i>		1	<i>fs(1)A456 M1</i>					
<i>fs(1)M68</i>	cv-v	2			+			
<i>fs(1)M69</i>		2	<i>lz</i>					
<i>fs(1)M70</i>	cv-v	1			+			
<i>fs(1)M71</i>		1	<i>ptg</i>					
<i>fs(1)M72</i>	1-23.6	1		7E10-8A5	+	-		no sperm stored in hemizygous female; homozygotes fertile
<i>fs(1)M73</i>	cv-v	1		7C-D	+			
<i>fs(1)M74</i>	cv-v	1			+			
<i>fs(1)M76</i>	cv-v	1			+			
<i>fs(1)M77</i>	cv-v	1			+			
<i>fs(1)M78</i>	v-f	1		9E3-11B2	+			
<i>fs(1)M79</i>	v-f	1		12D	+			
<i>fs(1)M81</i>	v-f	1			+			
<i>fs(1)M82</i>	v-f	1			+			
<i>fs(1)M83</i>	near f	1			+			
<i>fs(1)M84</i>	near f	1			+			
<i>fs(1)M85</i>	v-f	1			+			
<i>fs(1)M86</i>	f-sfa	1			+			
<i>fs(1)M87</i>	f-sfa	1			+			
<i>fs(1)M88</i>	f-sfa	1			+			
<i>fs(1)M89</i>	f-sfa	1			+			
<i>fs(1)M91</i>	w-v	1			+			
<i>fs(1)M93</i>	w-v	1			+			leaky
<i>fs(1)M94</i>	w-v	1		7E10-8A5	+			leaky
<i>fs(1)M95</i>	w-v	1		7E10-8A5	+			
<i>fs(1)M101</i>		14	<i>otu</i>					
<i>fs(1)M103</i>	1-5	2			+	+		
<i>fs(1)M104</i>	1-1.8	3			-			ovaries with misshapen yolky eggs
<i>fs(1)M105</i>	1-1.9	3			-			ovaries have misshapen yolky eggs
<i>fs(1)M106</i>		2	<i>Sxl</i>					
<i>fs(1)M108</i>	1-52	1			+			
<i>fs(1)M111</i>	1-21	5		7D1-6	-			females retain normal appearing eggs
<i>fs(1)M112</i>	1-25.1	2		7E10-8A5	-			stage 8 & 9 eggs accumulate; adults with disturbed ocellar bristle pattern
<i>fs(1)M114</i>		1	<i>fs(1)A273 M1</i>					
<i>fs(1)M116</i>	1-30	2		9E3-10A1	-			stage 8 oocytes accumulate
<i>fs(1)M120</i>	1-0.5	1			+			
<i>fs(1)M121</i>	near f	1			+			
<i>fs(1)M122</i>	21.64	1		7C-D	+			

α The number of alleles for each complementation group recovered by Mohler.

β Allelism to previously named or recently renamed loci; e.g., *fs(1)M5* alleles fail to complement *fs(1)A147*; Mohler's three alleles are accordingly designated *fs(1)A147^{M1}*, *fs(1)A147^{M2}*, and *fs(1)A147^{M3}*.

γ *fs(1)*-bearing ova from homozygous females rescued by fertilization with *fs(1)⁺*-bearing sperm.

δ 1 = Zhimulev, Belyaeva, Pokholkova, Kotcheneva, Fomina, Bgatov, Khudyakov, Patzevitch, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1981, DIS 56: 192-96; 2 = Zhimulev, Pokholkova, Bgatov, Umbetova, Solovjeva, Khudyakov, and Belyaeva, 1987, Biol. Zentralbl. 106: 699-720.

fs(1)N: female sterile (1) of Nasrat

location: 1-0.0 (closely linked to *sc*).

origin: Induced by an unspecified chemical mutagen.

discoverer: Nasrat, 1952.

references: Counce and Ede, 1957, J. Embryol. Exp. Morphol. 5: 404-21 (fig.).

Degelmann, Hardy, Perrimon, and Mahowald, 1986, Dev. Biol. 115: 479-89 (fig.).

synonym: *fs(1)^{nas}*.

phenotype: Maternal lethal. Developmental study of *fs(1)N¹* by Counce and Ede. Eggs of *fs(1)N/fs(1)N*

females will not support development of normal embryos. About half the eggs contain little or no yolk; development may or may not begin in such eggs but never progresses beyond a highly abnormal cleavage. In eggs containing more yolk, major effect is on synchrony of cleavage and blastoderm mitoses. Twenty percent of these embryos cease development before blastoderm formation. The remainder have abnormal blastoderms and aberrant gastrulation. Final pattern of damage determined by degree of abnormality of earlier stages, but some embryos show larval differentiation. A few of the

least abnormal embryos may emerge but never move about or feed. Formation of polar granules abnormal. RK3.

alleles: Homozygous *fs(1)N*¹² females produce embryos with defects in anterior and posterior development (Degelmann *et al.*). Head involution fails; anterior endodermal derivatives are deficient and the absence of pharyngeal musculature causes collapse of the cephalo-pharyngeal apparatus. Embryos lack all posterior endodermal derivatives as well as structures characteristic of abdominal segments 8 to 10 usually, but have six abdominal denticle belts, and occasionally partial seventh or seventh and eighth. In addition, posterior midgut, hindgut, Malpighian tubules, filzkörper, spiracles, anus, anal pad, and anal tuft absent; lateral tracheal trunks end blindly, usually in the sixth segment; posterior disorganization of CNS seen. Pole cells form abnormally and seldom migrate to form the gonad; posterior blastoderm nuclei fail to cellularize and the centrosome position *vis à vis* the blastoderm nuclei erratic; remarkably free of microtubules. Six rather than seven stripes of *ftz* transcription seen in blastoderm, with the sixth stripe wider than normal and separated from the fifth by a wider-than-normal space. At gastrulation neither posterior midgut invagination nor proctodeal invagination occur normally.

allele	origin	discoverer	synonym	ref ^α	comments
<i>fs(1)N</i> ¹		Nasrat, 1952			
<i>fs(1)N</i> ²	EMS		<i>fs(1)A371</i>	1	
<i>fs(1)N</i> ³	EMS		<i>fs(1)A379</i>	1	
<i>fs(1)N</i> ⁴	EMS		<i>fs(1)A1038</i>	1	
<i>fs(1)N</i> ⁵	EMS		<i>fs(1)K1281</i>	2	
<i>fs(1)N</i> ⁶	EMS		<i>fs(1)K1347</i>	2	
<i>fs(1)N</i> ⁷	EMS		<i>fs(1)K1540</i>	2	
<i>fs(1)N</i> ⁸	EMS	Mohler	<i>fs(1)M6</i> ¹²⁻¹¹⁶⁶	3	
<i>fs(1)N</i> ⁹	EMS	Mohler	<i>fs(1)M6</i> ¹²⁻¹⁴²⁰	3	
<i>fs(1)N</i> ¹⁰	EMS	Mohler	<i>fs(1)M6</i> ¹⁴⁻⁵⁷⁴	3	
<i>fs(1)N</i> ¹¹	EMS	Romans	<i>fs(1)M6</i> ^{12N2X2D}	3	
<i>fs(1)N</i> ¹²	EMS	Engstrom	<i>fs(1)N</i> ²¹¹	3	
<i>fs(1)N</i> ¹³	EMS	Morales	<i>fs(1)N</i> ^{dvm}	4	

^α 1 = Gans, Audit and Masson, 1975, Genetics 81: 683-704; 2 = Komitopoulou, Gans, Margaritis, Kafatos and Masson, 1983, Genetics 105: 897-920; 3 = Mohler and Carroll, 1984, DIS 60: 236-41; 4 = Perez-Chiesa, Ramos, Morales, Caceres, Cardona and Vazquez, 1983, DIS 59: 160.

cytology: No detectable chromosomal rearrangements (Slizynska). Placed in 1E1-2A by Peréz-Chiesa.

***fs(1)ne*: female sterile (1) no eggs**

A subset of hybrid-dysgenesis-induced, X-linked female-sterile mutations that are characterized by the oviposition of few if any eggs [Orr, Galanopoulos, Romano, and Kafatos, 1989, Genetics 122: 847-58 (fig.); see also Galanopoulos, Orr, Szabad, and Kafatos, 1989, Dev. Genet. 10:87-97 (fig.)]. The loci are numbered between 1 and 13 with alleles designated by lower case letters in the superscript. The X chromosomes in these lines harbor multiple P elements thus complicating cytological localizations. No case of allelism with previously identified sex-linked female sterile mutations detected. Various defects in respiratory appendages and chorionic architecture of hand dissected eggs described in above refer-

ences.

locus	location	number of alleles	comments
<i>fs(1)ne</i> ¹	<i>sc-ec</i>	8	soma dependent
<i>fs(1)ne</i> ²	<i>sc-ec</i>	2	
<i>fs(1)ne</i> ³		2	double mutant?
<i>fs(1)ne</i> ⁴	<i>f-car</i>	2	germ-line dependent
<i>fs(1)ne</i> ⁵	near <i>car</i>		germ-line dependent
<i>fs(1)ne</i> ⁶			double mutant?
<i>fs(1)ne</i> ⁷	<i>f-car</i>		
<i>fs(1)ne</i> ⁸	<i>ct-m</i>	1	germ-line dependent
<i>fs(1)ne</i> ⁹	near <i>car</i>	1	germ-line dependent
<i>fs(1)ne</i> ¹⁰	<i>ct-m</i>		
<i>fs(1)ne</i> ¹²			double mutant?
<i>fs(1)ne</i> ¹³			double mutant?

fs(1)pcx: see *pcx*

***fs(1)Ya*: female sterile of Young**

location: 1-[between *l(1)zw7* and *l(1)zw5*; to the right of *fs(1)Yb*].

origin: Induced by ethyl methanesulfonate.

discoverer: Young.

references: Young and Judd, 1978, Genetics 88: 723-42.

Lin and Wolfner, 1989, Mol. Gen. Genet. 215: 257-65.

phenotype: Homozygous females completely sterile; lay normal numbers of eggs. Eggs remain vacuolated after deposition. No evidence of syngamy (Zalokar, Audit, and Erk, 1975, Dev. Biol. 47: 419-32).

alleles: Expression female germ-line autonomous (Perri-mon, Engstrom, and Mahowald, 1986, Genetics 113: 695-712); no expression detectable in daughters of *tud* females (Lin and Wolfner). Transcript detectable in nurse cells and oocyte as early as stage 7; evenly distributed throughout cytoplasm of 0-2 h embryos, but not detectable later (Lin and Wolfner).

allele	discoverer	synonym	ref ^α
<i>fs(1)Ya</i> ¹	Young	<i>fs(1)Y</i> ²	3
<i>fs(1)Ya</i> ²	Young	<i>fs(1)Y</i> ⁵	3
<i>fs(1)Ya</i> ³	Young	<i>fs(1)Y</i> ⁶	3
<i>fs(1)Ya</i> ⁴		<i>fs(1)A73</i>	1
<i>fs(1)Ya</i> ⁵	Mohler	<i>fs(1)M12</i> ¹³⁻¹⁹⁷⁰	2
<i>fs(1)Ya</i> ⁶	Mohler	<i>fs(1)M12</i> ^{13B-76}	2
<i>fs(1)Ya</i> ⁷	Mohler	<i>fs(1)M12</i> ^{13F-50}	2

^α 1 = Gans, Audit and Masson, 1975, Genetics 105: 897-920; 2 = Mohler and Carroll, 1984, DIS 60: 236-41; 3 = Young and Judd, 1978, Genetics 88: 723-42.

cytology: Placed in 3B4-6 by deficiency mapping.

molecular biology: Identified on the basis of homology with a 2.4-kb female-specific transcript within a 32-kb walk through region 3B4-6. Germ-line transformants with an 8.5-kb fragment of wild-type DNA with homology to only the above transcript rescue fertility of both *fs(1)Ya* and *fs(1)Yb* females. Transcription unit shown to be 2.7 kb long and to be transcribed from left to right (Lin and Wolfner).

fs(1)Yb

location: 1-[between *l(1)zw7* and *l(1)zw5*; to the left of *fs(1)Ya*].

origin: Induced by ethyl methanesulfonate.

discoverer: Young.

references: Young and Judd, 1978, Genetics 88: 723-42.

phenotype: Homozygous females semisterile; lay reduced

numbers of eggs, most of which remain vacuolated after deposition; a few develop into normal-appearing adults. Mutation without effect on males.

alleles: *fs(1)Yb*¹, *fs(1)Yb*², and *fs(1)Yb*³ recovered as *fs(1)Y*¹, *fs(1)Y*³, and *fs(1)Y*⁷, respectively.

cytology: Placed in 3B4-6 by deficiency mapping.

fs(1)yolkless: see *yl*

Fs(2)1: see *Fs(2)M*

fs(2)3A-fs(2)308A: see *fs(2)HO3-fs(2)HO308*

fs(2)A

A series of mutants that have been subjected to complementation and physiological analysis but not mapped genetically.

origin: Induced by ethyl methanesulfonate.

references: Bakken, 1973, *Dev. Biol.* 33: 100-22 (fig.).

fs(2)A1

phenotype: Homozygous females anatomically normal; lay eggs that do not hatch.

alleles: Two alleles recovered among 79 second-chromosome female-sterile mutants analyzed.

fs(2)A2

phenotype: Homozygous females anatomically normal; lay eggs that do not hatch. First cleavage division normal; subsequent divisions often contain extra chromosomes; both clumped and scattered chromosomes observed. Some eggs have large spherical masses of fragmented chromosomes. No peripheral migration of nuclei.

alleles: Seven alleles recovered among 79 mutants analyzed.

fs(2)A3

phenotype: Homozygous females anatomically normal; lay eggs that do not hatch.

fs(2)A4

phenotype: Homozygous females anatomically normal; seem to be ovulation deficient since they accumulate normal-appearing stage-14 oocytes in their ovarioles.

alleles: 39 alleles recovered among 79 mutants analyzed.

fs(2)A5

phenotype: Homozygous females anatomically normal; lay eggs that do not hatch.

fs(2)A6

phenotype: Homozygous females anatomically normal but do not oviposit; ovaries may be abnormal.

fs(2)A7

phenotype: Homozygous females anatomically normal; lay eggs that do not hatch. Abnormal cleavage figures seen after the fourth division. Peripheral migration of nuclei restricted to only a part of the egg surface. Many blastoma nuclei large and reticulate; others small and condensed. Yolk nuclei clumped and unevenly disposed.

fs(2)A8

phenotype: Homozygous females anatomically normal; lay eggs that do not hatch.

fs(2)A9

phenotype: Homozygous females anatomically normal; lay eggs with fragile chorions, which do not hatch. Some eggs reach syncytial blastema but not cellular blastoderm formation; blastema nuclei unequal in size, irregularly shaped, and sometimes pycnotic. Distribution over egg surface nonuniform.

alleles: Two alleles among 79 mutants analyzed.

fs(2)A10

phenotype: Homozygous females anatomically normal; seem to be ovulation deficient since they accumulate stage-14 oocytes in their ovarioles.

fs(2)A11

phenotype: Homozygous females anatomically normal. Distribution of nuclei in syncytial blastema irregular. Oldest embryos show abnormal gastrulation.

fs(2)A12

phenotype: Homozygous females anatomically normal; lay foreshortened eggs with truncated anterior tip and short fat chorionic filaments. Eggs similar to stage-11 oocytes, some with adhering follicle and nurse cells. A few eggs lack stainable chromatin; most have one to three large spherical masses of fragmented chromosomes or tripolar spindles and abnormally elongated mitotic figures.

alleles: Six alleles among 79 analyzed mutants.

fs(2)A13

phenotype: Homozygous females anatomically normal, lay eggs that do not hatch. Eggs have abnormal cleavage figures, multipolar spindles, nonuniformity of chromosome contraction during anaphase, mitotic asynchrony, and uneven distribution of mitotic figures through ooplasm. In some, no mitotic figures are recognizable; only large masses of fragmented chromosomes or amorphic clumps are seen.

fs(2)A14

phenotype: Homozygous females anatomically normal; lay eggs that do not hatch. Most eggs display meiotic metaphase II chromosomes, without visible spindle fibers, and a sperm head lying close by. Oocyte chromosomes may be scattered or regularly grouped.

fs(2)A15

phenotype: Homozygous females have small abnormally organized ovaries; ovaries approximately 200 μm long; no ovariole formation; no organized follicles; no recognizable oocytes. Nuclei of different sizes and shapes but not in groups expected from cystocyte divisions; some are pycnotic.

fs(2)A16

phenotype: Homozygous females have small (400-600 μm long) abnormally organized ovaries. Ultrastructural studies by King and Buckles (1980, *DIS* 55: 74-75) reveal that oogonial cysts appear to be normal; surrounded by wedge-shaped follicle cells but not separated by interfollicular stalks so that the conventional moniliform vitellarium is not observed. Posterior end of ovariole filled with fusing follicles.

fs(2)A17

phenotype: Homozygous females have smaller-than-normal ovaries with nurse cell nuclei arrested in stage 4. Follicle cells continue normal differentiation for some time before degenerating. Vitellogenesis defective; oocyte arrests in stage 7. Transplantation experiments (Postlethwait and Handler, 1978, *Dev. Biol.* 67: 202-13) show that *fs(2)A17* ovaries develop autonomously in wild-type hosts and that *fs(2)A17* hosts support vitellogenesis in implanted normal and *ap* ovaries.

alleles: Six alleles among 79 analyzed mutants.

fs(2)A18

phenotype: Most egg chambers of homozygous females arrested in stage 7; no evidence of vitellogenesis. Height of follicular columnar epithelium increased. Posterior half of ovarioles filled with degenerating stage-8-sized egg chambers. Transplantation experiments (Postlethwait and Handler, 1978, *Dev. Biol.* 67: 202-13) show that *fs(2)A18* is ovary autonomous when transplanted into a wild-type host but nonautonomous when transplanted into a *fs(2)A17* host. *fs(2)A18* hosts support some vitellogenesis of + and *ap* implants.

fs(2)adp: see *adp*^{fs}

fs(2)B: female sterile (2) of Bridges

location: 2-5.

origin: Spontaneous.

discoverer: Bridges, 29c25.

synonym: *fes*.

references: King, Sang, and Leth, 1961, *Exp. Cell. Res.* 23: 108-17 (fig.).

King, Koch, and Cassens, 1961, *Growth* 25: 45-65 (fig.).

Koch and King, 1964, *Growth* 28: 325-69 (fig.).

King, 1969, *Nat. Cancer Inst. Monogr.* 31: 323-45.

phenotype: External morphology and sexual behavior normal (Burnet, Connolly, Kearney, and Cook, 1973, *J. Insect Physiol.* 19: 2421-31). Males fertile; females sterile. After rearing on glucose diet mutant females are less receptive to copulation attempts and more vigorously courted than wild-type virgins (Cook and Connolly, 1976, *J. Insect Physiol.* 22: 1727-35). Germaria of ovaries of homozygous females larger than normal; cysts contain 3 times the normal number of cells; metaphase figures found throughout germarium. Just over half the oogonia undergo complete cleavage with the loss of ring canal connections to other cells; remaining cells in small clusters with tenuous connections (Johnson and King, 1972, *Biol. Bull.* 143: 525-47). Vitellaria subdivided into a series of sausage-shaped cell aggregates, each surrounded with an ill-defined follicular epithelium and filled with hundreds to thousands of mitotically active oogonia-like cells [King, Burnett, and Staley, 1957, *Growth* 21: 239-61 (fig.)]. These cells occasionally differentiate into cells resembling nurse cells, which may have polytene chromosomes, and rarely into oocytes. *fs(2)B* ovaries transplanted into wild-type hosts in late larval stages and reciprocal transplants develop autonomously (Clancy and Beadle, 1937, *Biol. Bull.* 72: 47-56; Klug, Bodenstein, and King, 1968, *J. Exp. Zool.* 167: 151-56). The cells of the corpus-allatum corpus-cardiacum complex of homozygous *fs(2)B* females appear to be prevented from releasing their hormone products and undergo degenerative changes; these effects

reversible by vitellogenic activity of wild-type ovary implanted into the abdomen of the *fs(2)B* female (King, Aggarwal, and Bodenstein, 1966, *J. Exp. Zool.* 161: 151-76; Aggarwal and King, 1971, *J. Morphol.* 134: 437-46). Abdominal fat cells of *fs(2)B* females 1.5 times size of those in wild type. They resemble male fat cells in having more fat and less glycogen than those of normal females; return to normal size and composition following implantation of wild-type ovary (Butterworth and Bodenstein, 1968, *J. Exp. Zool.* 167: 207-18). *fs(2)B* ovaries show reduced levels of thymidylate synthetase (Carpenter, 1973, *Genetics* 75: 113-22). RK3.

Fs(2)D: see *Fs(2)G*

Fs(2)D10

location: 2 (unmapped).

origin: Induced by ethyl methanesulfonate.

references: Tearle and Nüsslein-Volhard, 1987, *DIS* 66: 209-26.

phenotype: Dominant female sterile. Development ceases before cellularization of blastoderm; syncytial nuclear division appears normal.

Fs(2)D-M: see *Fs(2)M*

fs(2)E: female sterile (2) Edmondson

origin: A series of nine ultraviolet-induced mutations at different loci.

references: Edmondson, 1952, *DIS* 26: 61-62. 1960, *DIS* 34: 49. CP627.

mutation	genetic location	synonym	phenotype
<i>fs(2)E1</i> ^α	2-57.6	<i>fs2.1</i>	no eggs; rudimentary gonads
<i>*fs(2)E2</i>	2-22.0	<i>fs2.2</i>	eggs don't hatch
<i>*fs(2)E3</i>	2-47.5	<i>fs2.3</i>	no eggs;
			narrow curved wings
<i>*fs(2)E4</i>	2-48.5	<i>fs2.4</i>	narrow curved wings;
			few eggs, don't hatch
<i>*fs(2)E5</i>	2-50.4	<i>fs2.5</i>	embryos degenerate;
			few larvae hatch
<i>*fs(2)E6</i>	2-54.4	<i>fs2.6</i>	eggs don't hatch
<i>*fs(2)E7</i>	2-55.2	<i>fs2.7</i>	vitelline membrane defective
<i>*fs(2)E8</i>	2-62.6	<i>fs2.8</i>	no eggs
<i>*fs(2)E9</i>	2-35.6	<i>fs2.9</i>	eggs don't hatch

^α Dorsal appendages of chorion convoluted or fused (King and Koch, 1963, *Quant. J. Microscop. Sci.* 104: 297-320).

fs(2)eo: female sterile (2) early oogenesis (T. Schüpbach)

A group of female-sterile mutations on the second chromosome which affect early stages of oogenesis (before yolk uptake). They were all induced by ethyl methanesulfonate, and recovered in screens by Schüpbach and Wieschaus (1989, *Genetics* 121: 110-17).

locus	genetic location	synonym	phenotype
<i>fs(2)eo1</i>	2-30	<i>fs(2)eoPV30</i>	few if any egg chambers; early oogenic arrest
<i>fs(2)eo2</i>	2-54	<i>fs(2)eoPL3</i>	few if any egg chambers; early oogenic arrest
<i>fs(2)eo3</i> ^α	2-{54}	<i>fs(2)eoRV64</i>	many degenerating egg chambers
<i>fs(2)eo4</i>	2-64	<i>fs(2)eoPB6</i>	egg chambers with variable

<i>locus</i>	genetic location	synonym	phenotype
<i>fs(2)eo5</i>	2-85	<i>fs(2)eoWP19</i>	number of nurse cells and degenerating material in ovaries ovarian tumors with many small, undifferentiated cells in ovaries
<i>fs(2)eo6</i>	2-105	<i>fs(2)eoQS2</i>	ovarian tumors with many small, undifferentiated cells in ovaries
<i>fs(2)eo7</i>	2-105	<i>fs(2)eoPP22</i>	few if any egg chambers; early oogenic arrest

^α Placed in 36E4-38A7 on the basis of its inclusion in *Df(2L)TW50 = Df(2L)36E4-F1;38A6-7*.

Fs(2)G: Female sterile (2) of Grell

location: 2- (not located).

origin: Induced by ethyl methanesulfonate.

discoverer: E. H. Grell, 65e.

synonym: *Fs(2)D*.

references: Yarger and King, 1971, *Dev. Biol.* 24: 166-77 (fig.).

phenotype: Heterozygous females raised at 25° or 30° completely sterile; minimal development noted of zygotes resulting from eggs of females raised at 20°; heterozygous males fertile. Ten percent as many egg chambers as normal, and 10% of chambers have fewer than 16 cells, generally lacking an oocyte. Ovaries of heterozygous females developing at 30° frequently fail to attach to oviducts. At 25° and 30°, heterozygous females take one day longer to develop than controls; viability of *Fs(2)G/+* females 70-75% at 20° and 25°, 5% at 30°. Wings of heterozygotes normal at 20°, frequently incised at 30°, greatly reduced and crumpled at 30°. Bristles short; thorax broad and flattened with air bubbles under cuticle. RK3.

fs(2)HO: female sterile (2) of Hardy and Orevi

A series of female-sterile mutations isolated from a sample of lethal-free ethyl-methanesulfonate treated second chromosomes produced by Hardy and Orevi.

mutation	synonym	ref ^α
<i>fs(2)HO3</i>	<i>fs(2)3A</i>	
<i>fs(2)HO8</i>	<i>fs(2)8B</i>	1
<i>fs(2)HO20</i>	<i>fs(2)20B</i>	1
<i>fs(2)HO100</i>	<i>fs(2)100A</i>	1
<i>fs(2)HO103</i>	<i>fs(2)103B</i>	
<i>fs(2)HO144</i>	<i>fs(2)144A</i>	
<i>fs(2)HO164</i>	<i>fs(2)164A</i>	
<i>fs(2)HO192</i>	<i>fs(2)192A</i>	1
<i>fs(2)HO260</i>	<i>fs(2)260A</i>	
<i>fs(2)HO294</i>	<i>fs(2)294A</i>	
<i>fs(2)HO297</i>	<i>fs(2)297A</i>	
<i>fs(2)HO308</i>	<i>fs(2)308A</i>	

^α 1 = Postlethwait and Handler, 1976, *Dev. Biol.* 67: 202-13.

phenotype: Females homozygous for all four mutants dealt with by Postlethwait and Handler have oogenesis aborted at early previtellogenic stages, are ovary autonomous in transplants into wild-type females, and support vitellogenesis in implanted wild-type ovaries.

fs(2)K: female sterile (2) of Kikkawa

location: 2-100.

origin: Spontaneous.

discoverer: Kikkawa, 1960.

synonym: *fs(2)K*.

references: 1960, DIS 34: 51.

phenotype: Female sterile; male fully fertile. RK3.

fs(2)lto: female sterile (2) late oogenesis (T. Schüpbach)

A group of female-sterile mutations on the second chromosome which lead to defects in later stages of oogenesis (during yolk uptake, chorion synthesis, or egg laying). They were all ethyl methanesulfonate induced and isolated in screens by Schüpbach and Wieschaus (1989, *Genetics* 121: 101-17).

<i>locus</i>	genetic location	synonym	phenotype
<i>fs(2)lto1</i>	2-51	<i>fs(2)ltoPN48</i>	Homozygous females lay eggs with fragile chorions, which remain unfertilized.
<i>fs(2)lto2</i>	2-[54] ^α	<i>fs(2)ltoHD43</i>	Homozygous females lay collapsed eggs.
<i>fs(2)lto3</i>	2-[54] ^α	<i>fs(2)ltoRE57</i>	Homozygous females lay eggs which often lack dorsal appendages, and appear more pointed at the posterior end.
<i>fs(2)lto4</i>	2-62	<i>fs(2)ltoQB3</i>	Homozygous females lay tiny eggs, which remain unfertilized.
<i>fs(2)lto5</i>	2-[86] ^β	<i>fs(2)ltoDF6</i>	Homozygous females lay short eggs.

^α In *Df(2L)TW50 = Df(2)36E4-F1;38A6-7*.

^β In *Df(2R)PC4 = Df(2)55A;55F*.

Fs(2)M: Female sterile (2) of Meyer

location: 2- [between *al* (2-0.4) and *dp* (2-13.0), (Szabad, Erdélye, Hoffman, Szidonya, and Wright, 1989, *Genetics* 122: 823-35)].

origin: X ray induced.

synonym: *Fs(2)I*, *Fs(2)D-M*.

references: Meyer, 1966, DIS 41: 167.

phenotype: Sterile *Fs(2)M/+* females deposit very few, mostly flaccid eggs that never bear dorsal chorionic appendages (Szabad, Erdélye, Hoffman, Szidonya, and Wright, 1989, *Genetics* 122: 823-35). Flies slightly reduced in size with discernably reduced eyes. *Fs(2)M/Cy* females show less curled wings than *+/Cy* (Craymer).

fs(2)OW: female sterile (2) of Oshima and Watanabe

A series of up to 34 different complementation units isolated from a natural population 3 times during the year; as many as 7 more isolated from cage populations. The loci inferred are numbered from 101 to 107, 202 to 217, and 801 to 812 from the natural population and c1 to c7 from the population cage. Complementation testing was the only procedure to which these recessive female-sterile mutants were submitted. The designator *OW* did not appear in the original publication.

origin: Spontaneous.

references: Oshima and Watanabe, 1973, *Genetics* 74: 351-61.

fs(2)Q342: see *Vm26A2*

Fs(2)Sz: Female sterile (2) of Szabad

A series of ethyl-methanesulfonate-induced dominant-female-sterile mutations on the second chromosome described by Szabad, Erdélye, Hoffmann, Szidonya, and Wright (1989, *Genetics* 122: 823-35). Meiotic mapping

possible in germ-line-independent and incompletely penetrant mutants; remainder mapped mitotically. X-ray- or EMS-induced revertants usually recessive lethals; complementation analysis of revertants identify 13-15 loci. The mutants named for Hungarian families that vanished by the beginning of the 14th century, as indicated in the following table.

locus	name	location	comments ^α
Fs(2)Sz1	Barsa	2-(<i>nub-stw</i>)	little or no embryogenesis
Fs(2)Sz2	Billa	2-(<i>nub-lt</i>)	little or no embryogenesis (Reuter)
Fs(2)Sz3	Dorog	2L	dorsal appendages reduced
Fs(2)Sz4	Etre	2-71.4	thin chorion; not germ line dependent (Wright)
Fs(2)Sz5	Himca	2L	pre-pronuclear-fusion arrest
Fs(2)Sz6	Hont	2-(<i>al-dp</i>)	meiotic defects (Wright)
Fs(2)Sz7	Ketel	2-(<i>lt-sca</i>)	meiotic defects
Fs(2)Sz8	Kompolt	2-(<i>al-dp</i>)	abnormal nuclear divisions
Fs(2)Sz9	Tarhos		abnormal nuclear divisions
Fs(2)Sz10	Tekele	2-(<i>al-nub</i>)	abnormal nuclear divisions
Fs(2)Sz11	Told	2-(<i>nub-sca</i>)	15% of embryos→head defects
Fs(2)Sz12	Ugra	2-18	agametic; germ line independent
Fs(2)Sz13	Vaja	2-15.1	Incompletely penetrant; variable head defects; may be <i>BicD</i> allele

^α Phenotype of eggs laid by *Fs(2)Sz/+* females; parentheses indicate origin of alleles not found in Szeged.

fs(2)TLM: female sterile (2) of Trippa, Louverre, and Micheli

location: 2-89.7 (between *L* and *Pin*).

origin: Spontaneous.

references: Trippa, 1977, DIS 52: 3 (fig.).

Trippa, Loverre, and Michele, 1977, DIS 52: 75.

Trippa, Loverre, and Cicchetti, 1980, Genetics 95: 399-412 (fig.).

Trippa, Loverre, and Cicchetti, 1980, J. Exp. Zool. 214: 277-85.

phenotype: Homozygous females sterile, produce no eggs, have underdeveloped ovaries; fewer ovarioles than normal; vitellogenesis apparently absent. One third of homozygous males sterile; remainder show normal fecundity, become sterile during first week of life. Testes abnormally shaped and reduced in size.

fs(2)TW1: female sterile (2) Ted Wright 1

location: 2-54 (estimated from cytological location).

origin: Induced by ethyl methanesulfonate.

discoverer: T.R.F. Wright in saturation studies of the *Df(2L)130* region.

phenotype: Eggs produced by homozygous females show no visible signs of development (Schüpbach, and Wieschaus, 1989, Genetics 121: 101-17).

alleles: Eight independent occurrences recovered by Wright, four more by Schüpbach.

cytology: Placed between 37C2 and 37D1 on the basis of its inclusion in the region of overlap of *Df(2L)36F2-6;37C6-D1* and *Df(2L)VA12 = Df(2L)37C2-D1;38B2-C1*.

Fs(2)X10

location: 2 (unmapped).

origin: Induced by ethyl methanesulfonate.

references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Dominant female sterile, gives cup-like eggs.

Fs(2)Y12

location: 2 (unmapped).

origin: Induced by ethyl methanesulfonate.

references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Semidominant female sterile, gives cup-like eggs.

fs(3)6m45

location: 3- (not mapped).

discoverer: Rice.

references: Bownes and Hames, 1978, J. Embryol. Exp. Morphol. 47: 111-20.

phenotype: Oocytes of homozygous females show no signs of vitellogenesis. Yolk proteins accumulate in hemolymph. *fs(3)6m45* ovaries transplanted fail to take up yolk proteins and are nonvitellogenic. Wild-type ovaries take up yolk protein and develop normally in *fs(3)6m45* host.

fs(3)108-350

locus	location	origin	discoverer	ref ^α
fs(2)108-17	3-(<i>ss-k</i>)	EMS	Nüsslein-Volhard	2
fs(2)110-8		EMS	Nüsslein-Volhard	1
fs(2)272-9	3-(<i>st-cu</i>)	EMS	Nüsslein-Volhard	1, 2
fs(2)293-19	3-(<i>st-ss</i>)	EMS	Nüsslein-Volhard	1, 2
fs(2)350-7	3R	EMS	Nüsslein-Volhard	2

^α 1 = Snyder, Galanopoulos, and Kafatos, 1985, Genetics 110: 53; 2 = 1986, Proc. Nat. Acad. Sci. USA 83: 3341-45.

fs(3)108-17

phenotype: Eggs of homozygous females have short thin chorionic respiratory appendages. Endochorionic structures disrupted; chorionic protein levels normal.

fs(3)110-8

phenotype: Eggs of homozygous females deficient in the first-deposited inner endochorionic layers; early chorion proteins deficient, although transcript levels appear normal.

fs(3)272-9

phenotype: Egg shells produced by homozygous females extremely disrupted. Deficiency of endochorionic material. All six chorion proteins and their mRNA's present at reduced levels; deficiency in the level of chorion-gene amplification in follicle cells.

alleles: Two; *fs(3)272-9¹* recovered by Nüsslein-Volhard; *fs(3)272-9²* EMS induced by Lindsley; originally designated *fs(3)SD78*.

fs(3)293-19

phenotype: Eggs of homozygous females defective in late-deposited outer layers of endochorion; endochorionic material deficient. Late-gene expression as measured by mRNA or protein levels defective; deficiency in the level of chorion-gene amplification in follicle cells.

fs(3)350-7

phenotype: Eggs of homozygous females have short thin chorionic respiratory appendages; endochorion structure disrupted; chorionic-protein levels normal.

fs(3)A

A series of mutants that have been subject to complementation and phenotypic analysis but have not been mapped genetically.

origin: Induced by ethyl methanesulfonate.

references: Baaken, 1973, *Dev. Biol.* 33: 100-22 (fig.).

fs(3)A1

phenotype: Oocyte development normal through stage 7, then arrested. Every ovariole has the normal number of 6 follicles. No vitellogenesis. Very little degeneration seen. Effects of mutant not affected by transplantation of *fs(3)A1* ovaries into wild-type hosts; *fs(2)A1* hosts support vitellogenesis of implanted wild-type ovaries poorly (Postlethwait and Handler, 1978, *Dev. Biol.* 67: 202-13).

alleles: Two alleles recovered.

fs(3)A2

phenotype: Homozygous females morphologically normal; lay eggs that do not hatch.

fs(3)A3

phenotype: Same as *fs(3)A2*.

fs(3)A4

phenotype: Same as *fs(3)A2*.

fs(3)A5

phenotype: Homozygous females morphologically normal; oocytes contain mature eggs but ovoposition does not take place.

fs(3)A6

phenotype: Same as *fs(3)A5*.

fs(3)A7

phenotype: Same as *fs(3)A5*.

fs(3)A8

phenotype: Same as *fs(3)A5*.

alleles: Two alleles recovered.

fs(3)A9

phenotype: Same as *fs(3)A5*.

fs(3)A10

phenotype: Ovaries ~180 μ m long; appear immaturely developed rather than malformed. Ovariole formation proceeds halfway down the ovary; stage 3 or 4 the most mature follicles seen. Posterior half of ovary contains degenerating egg chambers.

fs(3)A11

phenotype: Homozygous females morphologically normal; lay eggs that lack all discernable chromatin and fail to hatch.

fs(3)A12

phenotype: Same as *fs(3)A11*.

fs(3)A13

phenotype: Homozygous females produce eggs that appear to develop normally for the first 10 hr following fertilization but fail to hatch.

fs(3)A14

phenotype: Mostly the same as *fs(3)A11*; a few eggs produced by homozygous females contain large amorphous chromatin bodies; one embryo observed to reach syncytial blastema stage.

fs(3)A15

phenotype: Fertilized eggs of homozygous females have tripolar spindles, longitudinally split metaphase plates with too many chromosomes, precocious pulling of some chromosomes to poles, and numerous anaphase bridges. Some eggs had 1-3 large spherical or fragmented chromosomes.

fs(3)A16

phenotype: Ovaries of homozygous females show retardation of oocyte growth; stage 7 oocytes smaller than nurse cells; many egg chambers begin degenerating at stage 7. Some continue to grow, and nurse cells and follicle cells differentiate normally, but follicle is dwarfed.

fs(3)A17

phenotype: A few ovaries produce one or two yolky oocytes and the rest of the posterior half is filled with degenerating egg chambers. Most ovaries show very long slender stage-10 and stage-11 chambers. The globular appearance of the ooplasm suggests that many are degenerating.

Fs(3)Apc

location: 3- 39.5 (63/81 the distance of *h* to *th*).

origin: Induced by ethyl methanesulfonate.

discoverer: Szabad and Erdélye.

references: Szabad and Hoffman, 1989, *Dev. Biol.* 131: 1-10.

phenotype: Majority of eggs produced by *Fs(3)Apc/+* females collapse; 1-5% not flaccid. Respiratory appendages of chorion rudimentary as in the anterior chorion. When fertilized, resulting embryos rarely develop cuticular derivatives. Follicle cells in ovarioles fail to migrate between the oocyte and nurse cells. Mutant does not successfully revert. Germ-line chimaeras demonstrate that *fs(3)Apc* is follicle-cell autonomous.

fs(3)G2

phenotype: Females almost sterile; produce rare surviving progeny. Oogenesis incomplete, usually stops in early phases of vitellogenesis. Most (89%) follicles contain 32 cells instead of normal 16 as a result of an extra oogonial division. The 32 cells of an incipient cyst enclosed in two chambers in 6% of the cases. Position of oocyte in follicle abnormal in 28% of cases. Males partially sterile. Viability low. RK3.

fs(3)G3

phenotype: Oogenesis incomplete; most follicles stop development during yolk deposition (after stage 9). Males fertile. RK3.

fs(3)G5

phenotype: Oogenesis incomplete; ovarioles contain excessive numbers of follicles, which usually stop developing at or before stage 9. Males fertile. RK3.

fs(3)H: female sterile (3) of Handler

location: 3- (not mapped).

origin: Two complementing mutants, *fs(3)H23* and *fs(3)H172*, induced by ethyl methanesulfonate.

discoverer: Handler.

references: Postlethwait and Handler, 1978, *Dev. Biol.* 67: 202-13.

fs(3)H23

phenotype: Follicles cease development at early previtellogenetic stage; the few vitellogenic follicles that do appear are highly abnormal. Ovary autonomous in transplants, but some vitellogenesis induced by administration of juvenile hormone analogue, ZR-515. *fs(3)H23* females support vitellogenesis in implanted wild-type ovaries.

fs(3)H172

phenotype: Oocytes occasionally begin vitellogenesis, but process halts prematurely and only rarely do mature oocytes develop. Mature follicles look normal. Ovary autonomous in transplants into wild-type host and supports vitellogenesis in implanted wild-type ovaries.

fs(3)HO: female sterile (3) of Hardy and Orevi

A series of female-sterile mutations isolated from a sample of lethal-free, ethyl-methanesulfonate-treated third chromosomes produced by Hardy and Orevi.

mutation	genetic location	synonym	ref ^α
<i>fs(3)HO5A</i> ^β	3-46.3	<i>fs(3)jfl</i> ^{5A}	1
<i>fs(3)HO5B</i> ^β	3-48.4	<i>fs(3)j2</i> ^{5A}	1
<i>fs(3)HO29</i>	3-	<i>fs(3)29A</i>	2
<i>fs(3)HO115</i>	3-	<i>fs(3)115A</i>	2
<i>fs(3)HO127</i>	3-	<i>fs(3)127A</i>	2
<i>fs(3)HO133</i>	3-	<i>fs(3)133A</i>	2
<i>fs(3)HO191</i>	3-	<i>fs(3)191A</i>	
<i>fs(3)HO231</i>	3-	<i>fs(3)231A</i>	

^α 1 = Nishida, 1980, Jpn. J. Genet. 55: 427-39 (fig.); 2 = Postlethwait and Handler, 1978, Dev. Biol. 67: 202-13.

^β Isolated from same mutagen-treated chromosome along with *ms(3)HO5A* and *ms(3)HO5B*.

fs(3)HO5A

phenotype: Homozygous females do not lay eggs; oocyte development arrested in a previtellogenetic stage. Attachment of ovaries to oviduct fragile. Germania filled with polytene cells; no vitellana.

cytology: Polytene chromosomes normal.

fs(3)HO5B

phenotype: Homozygous females lay fertilized eggs, which fail to develop. Maternal effect lethal.

cytology: Polytene chromosomes normal.

fs(3)HO29

phenotype: Oocytes exhibit abortive vitellogenesis. Mutant effect is ovary autonomous in transplants into wild-type hosts and mutant females support development of implanted wild-type ovaries.

fs(3)HO115

phenotype: Oocyte development arrested in late previtellogenesis. Mutant-effect ovary autonomous in transplants into wild-type hosts and mutant females support vitellogenesis in implanted wild-type ovaries.

fs(3)HO127

phenotype: Similar to that of *fs(3)HO115*.

fs(3)HO133

phenotype: Similar to that of *fs(3)HO115*.

fs(3)K1

location: 3- (not mapped).

discoverer: Mohler.

references: King and Mohler, 1975, Handbook of Genetics

(R.C. King, ed.). Plenum Press, New York, and London, Vol. 3, pp. 83.

phenotype: Homozygous females produce eggs with abnormal dorsal chorionic appendages.

Fs(3)Sz: Female sterile (3) of Szeged

A series of ethyl-methanesulfonate-induced dominant or incompletely dominant female-sterile mutations on the third chromosome described by Erdélye and Szabad (1989, Genetics 122: 111-27). X-ray- or EMS-induced revertants identify 27-34 loci. The mutants are named for Hungarian families that vanished by the beginning of the 14th century, as indicated in the following tables.

locus	Table I: Complete Dominants name	location	comments ^α
<i>Fs(3)Sz1</i>	<i>Apc</i>	3-39.5	incomplete anterior egg coverings; flaccid eggs with rudimentary chorionic appendages
<i>Fs(3)Sz2</i>	<i>Avar</i>	3-36.3	agametic; retains eggs
<i>Fs(3)Sz3</i>	<i>Baksa</i>	3L	eggs fertilized, but sperm pronuclei do not divide and there is no development
<i>Fs(3)Sz4</i> ^β	<i>Bercel</i>	3-(<i>ru-h</i>)	only chitin granules in embryos
<i>Fs(3)Sz5</i>	<i>Bojla</i>	3L	eight sperm nuclei along A-P axis
<i>Fs(3)Sz6</i>	<i>Botond</i>	3L	eggs fertilized, meiosis completed, but no further development
<i>Fs(3)Sz7</i> ^β	<i>Damasa</i>	3L	eggs fertilized, meiosis completed, but usually no further development
<i>Fs(3)Sz8</i>	<i>Farkas</i>	3-(<i>ru-sr</i>)	several divisions of pronuclei without fusion
<i>Fs(3)Sz9</i>	<i>Gerec</i>	3-(<i>h-th</i>)	pronuclei approach but no fusion or division
<i>Fs(3)Sz10</i>	<i>Hodos</i>	3-(<i>ru-h</i>)	several divisions of pronuclei without fusion
<i>Fs(3)Sz11</i>	<i>Horka</i>	3R	pronuclei approach but no fusion or division
<i>Fs(3)Sz12</i> ^γ	<i>Huba</i>	3L	pronuclei may or may not divide; no fusion; cleavage may or may not occur
<i>Fs(3)Sz13</i>	<i>Jutas</i>	3R	meiotic figures among cleavage nuclei
<i>Fs(3)Sz14</i> ^δ	<i>Kavar</i>	3L	head lesions in embryos; some posterior structures present
<i>Fs(3)Sz15</i>	<i>Keled</i>	3-(<i>cu-sr</i>)	poorly formed cuticle fragments in 50% of the embryos
<i>Fs(3)Sz16</i>	<i>Keve</i>	3-(<i>h-th</i>)	mid abdominal segments missing
<i>Fs(3)Sz17</i>	<i>Kun</i>	3-(<i>e-ca</i>)	sperm but not egg pronuclei divide a few times
<i>Fs(3)Sz18</i>	<i>Laborc</i>	3L	1-3 large nuclei (probably sperm pronuclei)
<i>Fs(3)Sz19</i>	<i>Palat</i>	3-(<i>ru-h</i>)	head lesions
<i>Fs(3)Sz20</i>	<i>Pilis</i>	3-(<i>sr-e</i>)	poorly formed cuticle fragments in 50% of embryos
<i>Fs(3)Sz21</i>	<i>Tevel</i>	3-(<i>h-th</i>)	head lesions
<i>Fs(3)Sz22</i> ^δ	<i>Tomaj</i>	3-(<i>h-th</i>)	sperm pronuclei may or may not divide but no fusion or further development
<i>Fs(3)Sz23</i>	<i>Tonuz</i>	3R	sperm but not egg pronuclei divide a few times
<i>Fs(3)Sz24</i>	<i>Varas</i>	3-(<i>ru-h</i>)	sperm but not egg pronuclei divide a few times
<i>Fs(3)Sz25</i>	<i>Zerind</i>	3R	sperm but not egg pronuclei divide a few times
<i>Fs(3)Sz26</i>	<i>Zombor</i>	3-(<i>h-th</i>)	several divisions of pronuclei without fusion

locus	name	location	comments α
Fs(3)Sz27 Kartal		3-(<i>cu-ca</i>)	areas without nuclei in syncytial blastoderm; cleavage abnormalities
Fs(3)Sz28 Levente		80.7	no development after syncytial blastoderm; cleavage abnormalities
Fs(3)Sz29 Purdur		3-(<i>ru-cu</i>)	eggs fertilized, meiosis completed, but no development
Fs(3)Sz30 Rosd		3-(<i>cu-sr</i>)	head lesions
Fs(3)Sz31 Vencellin		28.1	head lesions
Fs(3)Sz32 Zolta		3-(<i>cu-sr</i>)	eggs rudimentary and flaccid; follicle cell epithelium ventralized during oogenesis

α Phenotype of eggs laid by *Fs(3)Sz/+* females.

β Two mutant alleles.

γ Four mutant alleles.

δ Three mutant alleles.

fs(3)T: female sterile (3) of Tabriz

location: 3-108.6.

origin: Spontaneous.

references: Mostashfi and Koliantz, 1972, DIS 48: 104.

phenotype: Homozygous females sterile and exhibit a pinkish eye color.

fs(4)34

location: 4- (not mapped).

origin: Spontaneous.

discoverer: Hochman, 64d.

references: 1972, DIS 48: 17.

phenotype: Viability of homozygotes normal; males fertile; females sterile. As originally described by Hochman, ovaries morphologically normal; females can be inseminated and can oviposit, but eggs fail to develop. In subsequent work, King and Buckles (1980, DIS 55: 74-75) report altered phenotype with oogenesis arrested in the germarium, where there are large numbers of oogenia not organized into cysts anteriorly and a solid plug of profollicle cells posteriorly.

cytology: Induced in neither *Df(4)M = Df(4)101E-F;102B6-17* nor *Df(4)G = 102E2-10*; tip (Hochman, 1974, Cold Spring Harbor Symp. Quant. Biol. 38: 581-89); probably lies between.

fsh: see *fs(1)h*

ft: fat

location: 2-12.0.

origin: Spontaneous.

discoverer: Mohr, 20b15.

references: 1923, *Studia Mendeliana* (Brunae). pp. 266-87.

1929, *Z. Indukt. Abstamm. Vererbungsl.* 50: 113-200 (fig.).

Bryant, Huettner, Held, Ryerse, and Szidonya, 1988, *Dev. Biol.* 129: 541-54.

phenotype: Viable alleles characterized using *ft*¹. Abdomen short and fat. Thorax broad. Wings short and broad with crossveins much closer together than normal. Scutellum shortened; scutellar bristles far apart. Viability good. Second- and third-instar larvae, particularly when there is little yeast in the food, show vacuoles in cytoplasm of salivary gland cells. Two waves of vacuole formation; vacuoles may be membrane-bound lipoprotein bodies (Chandhuri, 1969, DIS 44: 118). Tip of X disfigured, possibly as a result of several small puffs intermingled with hard, nonpuffed bands. In about 1% of

larvae, salivary glands distally expanded and crooked [Slizynski, 1964, *Cytologia* (Tokyo), 29: 330-36 (fig.)]. Lethal alleles characterized in study of *ft*⁸ (formerly *fd* = floppy disc) by Bryant *et al.*. *ft*⁸ classified as an amorphic mutation based on the similarity in lethal phenotype of *ft*⁸/*ft*⁸ and *ft*⁸/*Df(2L)M25A-11*. Larvae characterized by imaginal-disc hyperplasia such that mutant discs are much larger and more convoluted than wild type; the disc remains a single epithelial layer but in the highly convoluted proximal regions, of the wing disc at least, the columnar cells give way to cuboidal epithelial cells, which are deficient in cytoskeletal elements. Pupariation is delayed 3.2 days in mutants and the discs contain 122,000 cells at the end of nine days compared to 50,000 cells in wild type discs, which attain full growth at 5 days. Disc phenotype autonomous in transplants into adult hosts. Occasional mutant pupae reach the pharate-adult stage so that adult structures can be studied. Abdomens normal; eyes often swollen and may be split into two parts; extra head bristles; distal parts of antennae and legs may be missing; wings often fail to evaginate. Legs most severely affected; joints short and thick; missing tarsal joints and claws and tarsal fusions; increased bristle densities with deviant chaetal polarities; frequent outgrowths and ingrowths of cuticle; the latter giving rise to cuticle bound vesicles within the legs.

alleles:

allele	origin	discoverer	synonym	ref α	comments
ft 1	spont	Mohr, 20b15		3, 4	viable allele
ft 2			<i>l(2)sz32</i>	5	viable allele
ft 3			<i>l(2)sz71</i>	5	viable allele
ft 4			<i>l(2)a13</i>	5	pupal lethal
ft 5			<i>l(2)a17</i>	5	pupal lethal
ft 6			<i>l(2)a27</i>	5	pupal lethal
ft 7			<i>l(2)blq</i>	5	pupal lethal
ft 8	HD		<i>l(2)ft^{fd}</i>	1	pupal lethal
ft 9	EMS		<i>l(2)gd1</i>	2	pupal lethal
ft 10			<i>l(2)h1</i>	5	pupal lethal
ft 11			<i>l(2)h27</i>	5	pupal lethal
ft 12			<i>l(2)sz12</i>	5	pupal lethal
ft 13			<i>l(2)sz29</i>	5	pupal lethal
ft 14			<i>l(2)sz37</i>	5	pupal lethal
ft 15			<i>l(2)sz44</i>	5	embryonic lethal
ft 16			<i>l(2)sz52</i>	5	pupal lethal
ft 17			<i>l(2)sz81</i>	5	pupal lethal

α 1 = Bryant, Huettner, Held, Ryerse and Szidonya, 1988, *Dev. Biol.* 129: 541-54; 2 = Gateff, 1977, DIS 52: 4-5; 3 = Mohr, 1923, *Studia Mendeliana* (Brunae). pp. 266-87; 4 = Mohr, 1929, *Z. Indukt. Abstamm. Vererbungsl.* 50: 113-200 (fig.); 5 = Szidonya and Reuter, 1988, *Genet. Res.* 51: 197-208.

cytology: Placed in 24D5-8 based on its deletion by *Df(2L)sc19-1 = Df(2L)24D5-6;25C8* but not by *Df(2L)dp-h28 = Df(2L)24D8;24F6-7*.

other information: *G* = Gull, a dominant allele of *ft*.

ftd: see *qs*

fty: see *fru*

fu: fused

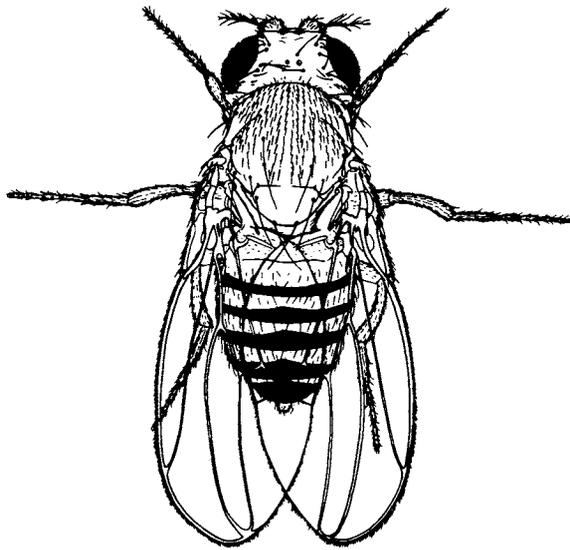
location: 1-59.5.

origin: Spontaneous.

discoverer: Bridges, 12k4.

references: Morgan and Bridges, 1916, *Carnegie Inst. Washington Publ.* No. 237: 55-58 (fig.).

Lynch, 1919, *Genetics* 4: 501-33.



fu: fused

Edith M. Wallace, unpublished.

King, 1970, Ovarian Development in *Drosophila melanogaster*, Academic Press, New York, London.

Busson, Limbourg-Bouchon, Mariol, Preat, and Lamour-Isnard, 1988, Roux's Arch. Dev. Biol. 197: 221-30.

phenotype: Veins L3 and L4 fused from base to beyond anterior crossvein with elimination of anterior crossvein and first basal cell. L3 and L4 fused at tip; this fusion may reach back to basal cell. L3 may be thickened and branched to varying degrees; L4 may be partially or completely absent. Mosaic wings in which the posterior compartment is nearly entirely *fu/fu* may develop normally; alternatively, patches of + tissue in a *fu* wing may develop a *fu* phenotype (Fausto-Sterling, 1978, Dev. Biol. 63: 358-69). Wings usually extended; flightless owing to cuticle defect (Deak). Ocelli reduced or absent; bristles of ocellar region small or absent. Eyes small and slightly rough. Phototactic response normal (Benzer, 1967, Proc. Nat. Acad. Sci. USA 58: 1112-19). Anterior scutellar bristles reduced in number and scutellum shortened. Female late to eclose and has decreased longevity. Ovaries histologically normal at eclosion but with half the normal number of ovarioles (Beatty, 1949, Proc. Roy. Soc. Edinburgh, B 63: 249-70); fecundity 4% normal. Developing egg chambers may fuse or become tumorous with age (King, Burnett, and Staley, 1957, Growth 21: 239-61 (fig.)). All aspects of phenotype respond in a coordinated fashion to experimental manipulation (Wust and Hanratty, 1979, Can. J. Genet. Cytol. 21: 335-46). Proportion of tumorous egg chambers increases by 6% per day. Females raised at 18° show only 10% the tumor development of those raised at 25°. Tumor formation also enhanced by presence of YS (King, 1969, Nat. Cancer Inst. Monogr. 31: 323-45). Ovarian effects in females carrying *fu* and a deficiency for *fu* [i.e. *In(1)Cl^{Ly}4R = In(1)A5-B1;17A6-B1^L1A8-B1;18A3-4^R*] are more extreme than those in *fu* homozygote (King, 1959, DIS 33: 142-43; 1970). Alleles vary in strength; measures of

ovarian tumor formation revealed that $fu^1 = fu^{14} > fu^7 > fu^{13} > fu^{11} = fu^{12}$ [Smith and King, 1966, J. Nat. Cancer Inst. 36: 445-63 (fig.)]. *fu/fu* ovaries transplanted into *fu+* hosts develop autonomously in regard to fertility (Clancy and Beadle, 1937, Biol. Bull. 72: 47-56; Sobels, 1950, Experientia 6: 139-40) and tumor formation (Smith, Bodenstein, and King, 1965, J. Exp. Zool. 159: 333-36). The few normal-appearing eggs that are laid by *fu/fu* females produce adults only if they have been fertilized by *fu+*-bearing sperm (Lynch, 1919, Genetics 4: 501-33). Eggs fertilized by *fu*- or Y-bearing sperm, with rare exceptions (Counce), develop into embryos that become abnormal 5-5½ hr after fertilization. Lethal embryos exhibit incompletely penetrant segment polarity defects in which the anterior denticle-belt-bearing half of each segment shows mirror-image duplication replacing the naked cuticle of the posterior half; more pronounced in thoracic segments; penetrance in right and left hemisegments at the same level is not necessarily correlated. (Nüsslein-Volhard and Wieschaus, 1980, Nature 287: 795-801, Gergen and Wieschaus, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 49-62). Elimination of regions of dorsal pattern and occasional head defects are also observed. *fu* eggs from *fu/+* mothers develop normally. Segment polarity defects autonomous in mosaics (Gergen and Wieschaus). Heterozygous daughters from homozygous mothers on the other hand, often have abnormal abdominal segmentation and, as embryos, have abnormal musculature. This is a maternal effect not found in the reciprocal cross, and it is temperature sensitive (Armstrong and Sobels). RK1.

alleles: No complementation observed in 120 heteroallelic combinations (Wurst and Hanratty).

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>fu</i> ¹	spont	Bridges, 12k4		1, 4, 9, 11, 12, 14, 17	WIWI
<i>fu</i> ²		Sturtevant		3	
<i>fu</i> ³		Stern		3	
<i>fu</i> ⁴		Moore	<i>fu</i> ^{3M}	3	
<i>*fu</i> ⁵	heat	Grossman, 1932	<i>fu</i> ⁸	3, 4, 7	
<i>fu</i> ⁶		L.V.Morgan	<i>fu</i> ^{20I}	18	
<i>fu</i> ⁷	H ₂ CO	Auerbach, 1951	<i>fu</i> ^{ff}	2, 3, 11, 12, 14	
<i>*fu</i> ⁸	UV	Edmondson, 51e	<i>fu</i> ^{51e}	4, 13	
<i>fu</i> ⁹	azm γ	Purdom, 57a	<i>fu</i> ^{57a}	4, 11, 12	
<i>*fu</i> ¹⁰	azm	Purdom, 57f	<i>fu</i> ^{57f}	4, 12	
<i>fu</i> ¹¹	spont	R.F.Grell, 1959	<i>fu</i> ⁵⁹	1, 4, 11	WIWW
<i>*fu</i> ¹²	γ ray	Fahmy, 62f1	<i>fu</i> ^{61f1}	4, 11	
<i>*fu</i> ¹³	γ ray	Fahmy, 62f2	<i>fu</i> ^{62f2}	4, 11	
<i>fu</i> ¹⁴	γ ray	Fahmy, 62f3	<i>fu</i> ^{62f3}	1, 4, 11	WSWI
<i>fu</i> ¹⁵	NNG	Kaufman, 1968	<i>fu</i> ⁶⁸	10	
<i>fu</i> ¹⁶	P	Gans	<i>fu</i> ^{mimi}	1	SWIS
<i>fu</i> ¹⁷		Deak	<i>fu</i> ^{DB203}	5	
<i>fu</i> ¹⁸	EMS	Wurst	<i>fu</i> ¹⁵	18	
<i>fu</i> ¹⁹	EMS	Wurst	<i>fu</i> ²⁰	18	
<i>fu</i> ²⁰	EMS	Wurst	<i>fu</i> ⁴⁰	18	
<i>fu</i> ²¹	EMS	Wurst	<i>fu</i> ⁴¹	18	
<i>fu</i> ²²	EMS	Wurst	<i>fu</i> ⁶⁷	18	
<i>fu</i> ²³	EMS	Wurst	<i>fu</i> ⁸³	18	
<i>fu</i> ²⁴	EMS	Wurst	<i>fu</i> ⁴⁹	18	
<i>fu</i> ²⁵	EMS	Hanratty	<i>fu</i> ⁴²⁹	18	
<i>fu</i> ²⁶	EMS	Hanratty	<i>fu</i> ²³⁴¹	18	
<i>fu</i> ²⁷	EMS	Hanratty	<i>fu</i> ²³²²	18	
<i>fu</i> ²⁸	EMS	Hanratty	<i>fu</i> ^{KD}	18	
<i>fu</i> ²⁹	EMS	Hanratty	<i>fu</i> ^{MH63}	18	
<i>fu</i> ³⁰	EMS	Hanratty	<i>fu</i> ^{0A}	18	
<i>fu</i> ³¹	EMS		<i>fu</i> ^{mH63}	1, 18	pupal lethal ^δ
<i>fu</i> ³²	EMS		<i>fu</i> ^{ecn}	1, 6, 15	WSWS
<i>fu</i> ³³			<i>fu</i> ^{ts1} ; <i>who</i>	8	8
<i>fu</i> ³⁴			<i>fu</i> ^{ts2}		
<i>fu</i> ³⁵			<i>fu</i> ^{ts3}		

allele	origin	discoverer	synonym	ref ^α	comments ^β fu ³¹
fu 36	spont	Schalet	<i>fu</i> ^S	1	SWWW
fu 37	EMS	Perrimon	<i>fu</i> ^{2P}	16	
fu 38	EMS	Perrimon	<i>fu</i> ^{9PZ}	16	
fu 39	EMS	Perrimon	<i>fu</i> ^{1PP7}	16	pupal lethal
fu 40	EMS	Perrimon	<i>fu</i> ^{9P2.3}	1, 16	pupal lethal ^δ
fu 41	X ray		<i>fu</i> ^A	1	SIWS
fu 42	DEB		<i>fu</i> ^{C10}	1	ISWI
fu 43	DEB		<i>fu</i> ^{DB3}	1	SISS
fu 44	DEB		<i>fu</i> ^{DB4}	1	ISS-
fu 45	DEB		<i>fu</i> ^{DB5}	1	WWIW
fu 46	DEB		<i>fu</i> ^{DB6}	1	IIWW
fu 47	DEB		<i>fu</i> ^{DB9}	1	III
fu 48	DEB		<i>fu</i> ^{DB10}	1	IIWI
fu 49	DEB		<i>fu</i> ^{DB11}	1	III
fu 50	DEB		<i>fu</i> ^{G3}	1	SWSS
fu 51	DEB		<i>fu</i> ^{J3}	1	IIIS
fu 52	DEB		<i>fu</i> ^{JB3}	1	WWS-
fu 53	DEB		<i>fu</i> ^{L4}	1	SWWS
fu 54	DEB		<i>fu</i> ^{M1}	1	SSIS
fu 55	DEB		<i>fu</i> ^{MC2}	1	SISS
fu 56	DEB		<i>fu</i> ^{new}	1	SSWS
fu 57	DEB		<i>fu</i> ^{W3}	1	SSWI
fu 58	DEB		<i>fu</i> ^{Y1}	1	SSS-

^α 1 = Busson, Limbourg, Bouchon-Mariol, Preat and Lamour-Isnard, 1988, Roux's Arch. Dev. Biol. 197: 221-30; 2 = Counce, 1956, Z. Indukt. Abstamm. Vererbungsl. 87: 462-81; 3 = CP552 4 = CP627 5 = Deak, 1976, J. Insect Physiol. 22: 1159-65; 6 = Gergen and Wieschhaus, 1986, Roux's Arch. Dev. Biol. 195: 49-62; 7 = Grossman, 1934, DIS 1: 30; 8 = Homyk and Grigliatti, 1983, Dev. Genet. 4: 501-33; 9 = Lynch, 1919, Genetics 4: 501-33; 10 = Kaufman, 1969, DIS 44: 44; 11 = King, 1970, Ovarian Development in *Drosophila Melanogaster*, Academic Press, New York, London; 12 = King, Burnet and Staley, 1957, Growth 21: 239-61; 13 = Meyer and Edmondson, 1951, DIS 25: 72; 14 = Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. 237: 55-58 (fig.); 15 = Nüsslein-Volhard and Wieschhaus, 1980, Nature 287: 795-801; 16 = Perrimon and Mahowald, 1987, Dev. Biol. 119: 587-600; 17 = Smith and King, 1966, J. Nat. Cancer Inst. 36: 445-61 (fig.); 18 = Wrust and Hanratty, 1979, Can. J. Genet. Cytol. 21: 335-46.

^β Phenotype as determined by Busson *et al.* with respect to wing phenotype, viability, fecundity and maternal effect respectively; W = weak, I = intermediate, S = strong. Wing phenotype, veins L3 and L4 fused only proximally = W, at several points = I, all along length = S. Viability as the ratio of the number of fused flies to wild type in *fu*⁺ X *fu*^Y: 0.75-1.0 = W, 0.5-0.75 = I, 0.25-0.5 = S. Fecundity as the ratio of eggs laid by *fu*^{fu} versus *fu*⁺ sisters: 0.5-1.0 = W, 0.25-0.5 = I, 0-0.1 = S. Maternal effect on segmentation as percent of embryos with all segments entirely duplicated: 0-25% = W, 25-70% = I, 70-100% = S.

^γ azm = azo-mustard.

^δ Phenotype described below.

cytology: Placed in 17C3-D2 based on its inclusion in *Df(1)fu-H4 = Df(1)17C3-7;17D1-2* (Busson, Limbourg-Bouchon, Mariol, Preat and Lamour-Isnard, 1988, Roux's Arch. Dev. Biol. 197: 221-30).

molecular biology: Included in a 250-kb walk from 17C to 17E; no alterations in restriction map were observed for 12/12 viable *fu* alleles nor for the three pupal-lethal alleles listed in the allele table. *Df(1)fu-Z4* determined to be deficient for 40-kb; a cosmid covering this region when injected into embryos effects partial rescue of the maternal effect of homozygosity for *fu* in some 2% of embryos; similar results obtained from a 14-kb subclone. Two adjacent *Eco*RI subclones identify four transcripts on Northern blots of mRNA from 0-4 hr embryos of 1.3 and 1.6 kb for the more distal fragment and 2.5 and 3.5 kb for the more proximal fragment; the *fu* transcript or transcripts not identified (Mariol, Preat and Limbourg-Bouchon, 1987, Mol. Cell. Biol. 7: 3244-51).

phenotype: Homozygous and hemizygous females and hemizygous males die in pupal stage; lethality suppressible by *Su(fu)*. *fu*³¹/*fu*⁴¹ and *fu*³¹/*Y* have all segments entirely duplicated; lack mouth hooks and some have no head. *fu*³¹/*+* embryos from such clones exhibit very low paternal rescue; small correction of segmental phenotype, low hatch, and preadult mortality (Busson, Limbourg-Bouchon, Mariol, Preat and Lamour-Isnard, 1988, Roux's Arch. Dev. Biol. 197: 221-30).

fu³³

phenotype: A temperature-sensitive allele. *fu* wing-vein phenotype occurs at all temperatures; ocelli absent when raised at 22° but not at 17°. TSP for absence of anterior ocellus from second instar through pupal stage, for posterior ocelli restricted to late third instar and early pupal stage. Flightless when raised at 22° or 29° but not at 17°; wings held horizontally perpendicular to body at 29°. Musculature abnormal when raised at restrictive temperatures [Homyk and Grigliatti, 1983, Dev. Genet. 4: 77-97 (fig.)].

fu⁴⁰

phenotype: *fu*⁴⁰/*Y* sons of *fu*⁴⁰/*+* mothers die as late pupae or newly emerged adults; lethality suppressible by *Su(fu)*. *fu* embryos produced by homozygous germ-line clones are mostly lethal (70%); those that hatch mostly achieve adulthood (Perrimon and Mahowald, 1987, Dev. Biol. 119: 587-600).

Fuc: alpha-Fucosidase

location: 3-35.5 [based on 45 recombinants between *h*(26.5) and *st* (44.0) (Repp)].

phenotype: Structural gene for alpha fucosidase (EC 3.2.1.51). A diffuse intermediate zone of activity in *Fuc*^F/*Fuc*^S heterozygotes suggests multimeric nature of enzyme (MacIntyre). Flies heterozygous for a null allele and a deficiency for *Fuc* are viable and fertile (Bond).

alleles: *Fuc*^F and *Fuc*^S are naturally occurring alleles; also one null allele, *Fuc*^{nl}, induced by ethyl methanesulfonate (Bond).

cytology: Placed between 67F2 and 68D6 on the basis of its inclusion in the region deleted by *Df(3L)vin2 = Df(3L)67F2-3;68D6* (Bond).

Fuh: see *Fum*

fum: fused mushroom bodies (J.C. Hall)

location: 2- (not mapped).

origin: Induced by ethyl methanesulfonate.

discoverer: Heisenberg and Fischbach.

phenotype: Beta lobes of mushroom bodies in dorsal brain are fused at the midline; penetrance incomplete.

Fum: Fumarase

location: 1-19.9.

synonym: *Fuh*: Fumarate hydratase.

references: Madhavan and Ursprung, 1973, Mol. Gen. Genet. 120: 379-80.

phenotype: The structural gene for fumarase [FUM; L-malate hydro-lyase (EC 4.2.1.2)]. Enzyme level observed depends on genetic background (Laurie-Ahlberg, Maroni, Bewley, Lucchesi, and Wier, 1980, Proc. Nat. Acad. Sci. USA 77: 1073-77). Enzyme levels higher in females than in males in midlarval, pupal, and

pharate-adult stages; female levels drop and are surpassed by male levels in older adults (Whitney and Lucchesi, 1972, *Insect Biochem.* 2: 367-70). Head and thorax have higher activities than abdomen (Pipkin, Chakrabarty, and Bremner, 1977, *J. Hered.* 68: 245-52). Activity equally distributed between mitochondrial and cytoplasmic fractions (Pipkin *et al.*).

alleles: Two electrophoretic alleles, *Fum^F* and *Fum^S*, found in natural populations. (Designated *Fum⁶* and *Fum⁴* by Research Triangle Park Group, 1978, *DIS* 53: 117).

cytology: Placed in 5C-6C11 by method of segmental aneuploidy (Pipkin *et al.*).

furled: see *fd*

furrowed: see *fw*

fused: see *fu*

fused filament: see *fft*

fused mushroom bodies: see *fum*

fuzzy: see *fy*

fw: furrowed

location: 1-36.85 (Lefevre, 1970, *DIS* 40: 45).

origin: Spontaneous.

discoverer: Duncan, 14k.

references: 1915, *Am. Naturalist* 49: 575-82.

Morgan and Bridges, 1916, *Carnegie Inst. Washington Publ. No. 237*: 80.

Nachtsheim, 1919, *Z. Indukt. Abstamm. Vererbungsl.* 20: 118-56.

phenotype: Eyes with vertical fold and furrows. Head and scutellum shortened. Bristles gnarled and shortened, especially the postscutellars. Best classification character is short, blunt notopleurals. RK2. Phenotype can become nearly wild type on inbreeding (Lefevre).

alleles: Most information on *fw* alleles tabulated below. Descriptions of special phenotypic features differing from those described above for *fu¹* are detailed separately following the table.

allele	discoverer	origin	ref ^α
<i>fw¹</i>	Duncan, 14k	spont	1, 2, 3, 9, 10
<i>*fw²βγ</i>	Muller, 31a	X ray in <i>In(1)dl-49</i>	1, 2
<i>*fw^{20c}</i>	E. Wallace		1
<i>*fw^{33d}</i>	E. Wallace		1
<i>*fw^{33e}</i>	Bridges		1
<i>fw^{34e}γ</i>	Duncan, 34e20		1, 2
<i>fw^{35k}</i>	Spencer, 35k4		1
<i>*fw^{39k}</i>	Ives		1
<i>fw^{49c}βγ</i>	R.C. King, 49c28	32p	2, 8
<i>fw⁵⁹βγ</i>	García-Bellido, 59i21	X ray	2, 4
<i>fw⁶⁰βγ</i>	García-Bellido, 60k8	X ray	2, 4
<i>fw⁶⁷</i>	Hayman, 67j9	X ray	5
<i>fw⁶⁸</i>	Kaufman	nitrosoguanidine	7
<i>*fw^c</i>	Bridges, 15l27		2
<i>*fw^wγ</i>	Ives, 43b24	spont	6
<i>fw^{wr}γ</i>	R.M. Valencia, 1959	X ray in <i>In(1)sc^{SIL}sc^{8R}+dl-49</i>	11, 12

^α 1 = CP552; 2 = CP627; 3 = Duncan, 1915, *Am. Nat.* 49: 575-82; 4 = García-Bellido, 1963, *Genet. Iber.* 15: 1-102; 5 = Hayman and Maddern, 1969, *DIS* 44: 50; 6 = Ives, 1946, *DIS* 19: 46; 7 = Kaufman, 1969, *DIS* 44: 44; 8 = King, 1949, *DIS* 23: 62; 9 = Morgan and Bridges, 1916, *Carnegie Inst. Washington Publ.* 237: 80; 10 = Nachtsheim, 1919, *Z. Indukt. Abstamm. Vererbungsl.* 20: 118-

56; 11 = Valencia, 1959, *DIS* 33: 100; 12 = Valencia, 1965, *DIS* 40: 36.

β Only differences from the basic *fu¹* phenotype indicated.
γ Phenotype described separately below.

cytology: Placed in 11A4 (Lefevre, 1981, *Genetics* 99: 461-80).

***fw²**

phenotype: Extreme *fw*. Female sterile. RK2A.

fw^{34e}

phenotype: Originally showed eye surface medium folded; bristles much gnarled. Schultz and Curry report that stock in 1940 showed gnarled bristles and eye small but no vertical fold. RK2.

fw^{49c}

phenotype: Eyes furrowed; distal portions of arisal branches hooked; wings divergent and often stringy; scutellar groove reduced. Bristles split, bent, and often erect; acrostichal hair pattern distributed with whorls and naked areas. Late hatching, poorly viable, and mostly sterile. *fw^{49c}/fw* phenotypically intermediate but more like *fw/fw* than *fw^{49c}/fw^{49c}*. RK3.

fw⁵⁹

phenotype: Eyes rough and creased; facets irregular, 15% fewer than normal. Eyes browner than normal; pterine concentration reduced in the eyes and, except for isoxanthopterin, increased in testis sheath. Riboflavin accumulates in Malpighian tubules. Large bristles of head and thorax short, thick, angled, blunt, and occasionally reduced to stumps. Arista thick with contorted and supernumerary branches. Scutellum small with groove between it and thorax reduced. Hatchability and larval development normal; larval anal plates swollen and surrounded by melanotic halo. Melanotic anal region persists in pupa; pupa also has melanotic spots elsewhere that may result in nonpigmented areas on the imaginal integument. Extrusion of anterior and posterior spiracles in prepupa incomplete. Many *fw⁵⁹* flies die either after 24-30 hr of pupal development or at the time of eclosion. Fecundity of female reduced owing to reduced number of ovarioles. RK2.

fw⁶⁰

phenotype: Like *fw⁵⁹* but with lower penetrance and expressivity. RK2.

***fw^w: furrowed-weak**

phenotype: Affects only bristles, particularly the scutellars and postalars. Eyes normal. Normal fertility and viability. RK2.

fw^{wr}: furrowed-wrinkled

synonym: *wr*.

phenotype: Eye surface in folds. Some bristles shortened, thickened, or curved; many doubled and may be fused. Viability low. RK2.

fwd: four wheel drive (M. Fuller)

location: 3- {0}.

discoverer: Wolf, 1988.

synonym: *ms(3)neol*.

origin: P-element insert marked with neomycin resistance, from the single element mobilization screen of Cooley and Sprading, 1988.

references: Wolf and Fuller, unpublished.

phenotype: Recessive male sterile. Failure of cytokinesis after meiosis I and meiosis II in males, usually resulting in onion stage early spermatids with four equal sized nuclei associated with a single, large mitochondrial derivative. Cells in meiosis II contain two spindles. Females fertile.

cytology: Placed in 61A1-C4 based on its inclusion in $Df(3L)emc-E12 = Df(3L)61A;61D3$ but not in $Df(3L)Ar14-8 = Df(3L)61C3-4;62A8$.

fx: see *fa^{fx}*

***fy: fuzzy**

location: 2-33.

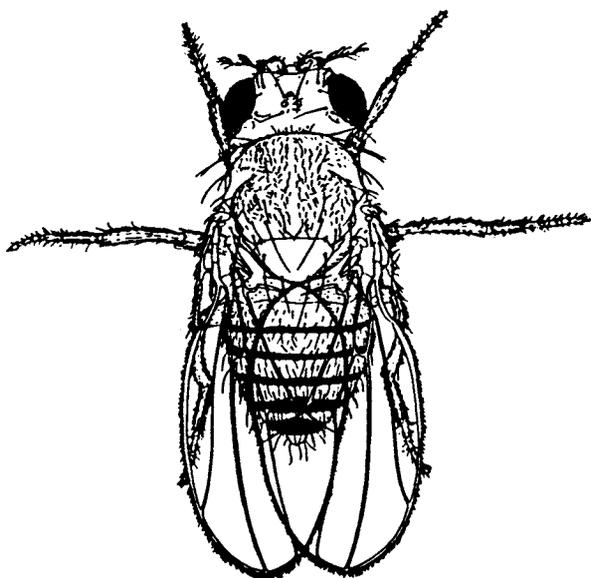
origin: Spontaneous.

discoverer: Ives, 39a.

references: 1940, DIS 13: 49.

phenotype: Hairs on abdomen and thorax irregular and directed toward midline. Hairs on wing margins erect. Hairs on legs also show abnormal polarities (Held, Duarte, and Derakhshanian, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 145-57). Resembles *fz*. Fertility and viability below normal. RK2.

alleles: A possible second allele, *fy²*, described by Grell (1969, DIS 44: 46-47) but maps to 2-24.1.



fz: frizzled

From Bridges and Brehme, 1944, Carnegie Inst. Washington Publ. No. 552: 85.

fz: frizzled

location: 3-41.7.

origin: Spontaneous.

discoverer: Bridges, 38b18.

references: Adler, Charlton, and Vinson, 1987, Dev. Genet. 8: 99-119.

phenotype: Hairs on thorax directed irregularly toward midline. Thoracic bristles also inturned and often wavy. Postverticals may turn outward. Hairs on wing edge and feet nearly erect; trichomes on wings of flies carrying weaker alleles tend to form swirls rather than lying parallel to one another and pointing distally; stronger alleles

can cause random orientation of trichomes. Polarity of chaetae deranged in characteristic ways on wings, notum, halteres, legs, tergites, and sternites; *fz M⁺* clones in *M/+* wings cause derangement of polarity in *M/+* cells surrounding clone (Gubb and García-Bellido, 1982, J. Embryol. Exp. Morphol. 68: 37-57). In a wild-type background clones of wing cells homozygous for *fz* alleles that cause eye roughening, but not of those without effect on eye texture, cause adjacent normal trichomes in regions distal, anterior, and posterior, but not proximal to the clone, to orient toward the clone rather than distally as they normally do; no effect on trichomes on opposite surface of the wing (Vinson and Adler, 1987, Nature 329: 549-51). Wing may be reduced. A low level of doubling of trichomes and splitting of chaetae observed. Sex combs may be irregular. Most alleles cause eyes to be rough. Two weak alleles, *fz²⁴* and *fz³⁴* and two neomorphic alleles, *fz¹³* and *fz²⁰*, have normal eye textures. Extra leg joints tend to form as mirror-image duplications proximal to the normal joints on tarsal segments one to four. Also polarities of bristles, hairs, and bracts on legs abnormal (Held, Duarte, and Derakhshanian, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 145-57). RK2.

alleles: Most alleles fit into a hypomorphic to amorphic series with many hemizygotes displaying a more severe phenotype than homozygotes. Four strong alleles are lethal in homozygotes; three are associated with chromosome rearrangements (*fz³*, *fz²⁶* and *fz³⁰*) and one with a normal sequence (*fz¹⁴*); they complement one another in all pairwise combinations and survive in combination with *fz* deficiencies; therefore lethality is not associated with the *fz* locus.

allele	origin	discoverer	synonym	ref ^α	phenotype ^β	cytology
<i>fz¹</i>	spont	Bridges, 38b18		1, 3, 6	MMV	
<i>fz²</i>	spont	Ives, 46f20	<i>fz^{46f}</i>	5		
<i>fz³</i>	X ray	Velissariou		2	SSL ^γ	<i>In(3L)70D6-7;</i> <i>75D8 +</i> <i>In(3L)73D3-5;</i> <i>80-81</i>
<i>fz⁴</i>	X ray	Velissariou		2		<i>In(3L)70D6-7;</i> <i>80-81 +</i> <i>In(3LR)79F;</i> <i>87D-E</i>
<i>fz⁵</i>	X ray			4		
<i>fz⁶</i>	X ray			4		
<i>fz⁷</i>	X ray			4		
<i>fz⁸</i>	X ray			4		
<i>fz⁹</i>	EMS			4		
<i>fz¹⁰</i>	EMS			4		
<i>fz¹¹</i>	EMS			4		
<i>fz¹²</i>	EMS			4		
<i>fz¹³</i>	EMS	Adler	<i>fz^{F31}</i>	1, 6	MMV	
<i>fz¹⁴</i>	EMS	Adler	<i>fz^{H41}</i>	1	SSL ^γ	+
<i>fz¹⁵</i>	EMS	Adler	<i>fz^{H51}</i>	1	SSV	+
<i>fz¹⁶</i>	EMS	Adler	<i>fz^{I12}</i>	1	MMV	
<i>fz¹⁷</i>	EMS	Adler	<i>fz^{I22}</i>	1	MMV	
<i>fz¹⁸</i>	EMS	Adler	<i>fz^{J21}</i>	1	MMV	
<i>fz¹⁹</i>	EMS	Adler	<i>fz^{J22}</i>	1	MMV	
<i>fz²⁰</i>	EMS	Adler	<i>fz^{N21}</i>	1, 6	MMV	+
<i>fz²¹</i>	EMS	Adler	<i>fz^{P21}</i>	1	MMV	
<i>fz²²</i>	EMS	Adler	<i>fz^{R51}</i>	1	MMV	
<i>fz²³</i>	EMS	Adler	<i>fz^{R52}</i>	1	MMV	
<i>fz²⁴</i>	EMS	Adler	<i>fz^{R53}</i>	1	MMV	+
<i>fz²⁵</i>	EMS	Adler	<i>fz^{R54}</i>	1, 6	SSV	+
<i>fz²⁶</i>	γ ray	Adler	<i>fz^{C21}</i>	1	SSL ^γ	<i>Tp(3;2)40;</i> <i>66B-D;</i> <i>70D4-7</i>
<i>fz²⁷</i>	γ ray	Adler	<i>fz^{EA3}</i>	1	MMV	<i>T(2;3)40;</i>

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	synonym	ref ^α	phenotype ^β	cytology
fz 28	γ ray	Adler	<i>fz EAB</i>	1	MMV	70D4-7
fz 29	γ ray	Adler	<i>fz EF2A</i>	1	MMV	+
fz 30	γ ray	Adler	<i>fz K21</i>	1	SSL ^γ	<i>In(3L)70D4-7;</i> <i>75A-B12</i>
fz 31	HD	Adler	<i>fz CAD3</i>	1	SSV	+
fz 32	HD	Adler	<i>fz CI9</i>	1	MMV	+
fz 33	HD	Adler	<i>fz CT8A</i>	1	MMV	+
fz 34	HD	Adler	<i>fz CT8C</i>	1	WWV	+
fz 35	HD	Adler	<i>fz CT9B</i>	1	MMV	+
fz 36	HD	Adler	<i>fz CT43</i>	1	MMV	+
fz 37	HD	Adler	<i>fz KD4a</i>	6		molecular deletion
fz 38	P derived	Adler	<i>fz CT8CX2</i>			<i>In(3L)61C;</i> <i>69A;70D4-7</i>

^α 1 = Adler, Charlton and Vinson, 1987, Dev. Genet. 8: 99-119; 2 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou and Walker, 1981, DIS 56: 186-90; 3 = CP627; 4 = Gubb and Garcia-Bellido, 1982, J. Embryol. Exp. Morph. 68: 37-57; 5 = Ives, 1946, DIS 20: 65; 6 = Vincent and Adler, 1987, Nature 329: 549-51.

^β Phenotypic analysis according to Adler *et al.*; the first letter scores thoracic-bristle phenotype and the second wing-hair disorientation (S = strong, M = moderate, W = weak); the third letter indicates viability (V) or lethality (L).

^γ Lethal alleles complement each other as well as *fz* deficiencies indicating three of the four lethal chromosomes are rearranged; they complement each other.

molecular biology: Genomic sequence isolated by transposon tagging. A single 4-kb mRNA identified in Northern blots of pupal RNA. A full-length cDNA contains a single long open reading frame which encodes a 581-amino-acid polypeptide. The N terminus contains a putative signal sequence. Following a long N-terminal

region, there are seven strongly hydrophobic regions typical of transmembrane domains three of which contain a proline residue. No striking homologies to other polypeptide sequences found in databases (Vinson, Conover and Adler, 1989, Nature 338: 263-64).

cytology: Placed in 70D6-7 based on commonality of breakpoints in *In(3L)fz*³ and *In(3L)fz*⁴ (Ashburner *et al.*). Adler places *fz* in 70D4-5 based on *in situ* hybridization of *P*-element probe to polytene chromosomes of *fz*³⁴.

fz 13

phenotype: A neomorphic allele causing severe disruption of trichome orientation in the costal wing cell, a region little affected by most alleles; phenotype of homozygote more severe than that of hemizygote. Also unlike other alleles, homozygous clones of *fz*¹³ cells in the wing do not affect orientation of trichomes in surrounding normal cells. A third notable feature of *fz*¹³ is that it is without effect on eye texture.

fz 20

phenotype: Like *fu*¹³.

fzy: fizzy

location: 2-51.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82 (fig.).

phenotype: Homozygous lethal; ventral outside and central nervous system degenerate.

alleles: Eight; *fzy*¹ and *fzy*² (isolated as *IB* and *IH*) retained.

g: garnet**location:** 1-44.4.**discoverer:** Bridges, 15b19.**synonym:** *salmon*.**references:** Bridges, 1916, *Genetics* 1: 151.

phenotype: Eye color brownish, darkening with age; both pteridine and ommochrome pigments variably reduced in different alleles, giving rise to such descriptions as deep purplish ruby, pinkish, brownish, yellowish ruby, orange, etc. Nolte (1959, *Heredity* 13: 233-41) measured both pigments spectroscopically for *g* alleles (g^1, g^2, g^3, g^4) and detected subtle differences. Hexter (1963, *Proc. Nat. Acad. Sci. USA* 50: 372-79) claims g^1, g^2, g^3 , and g^4 indistinguishable phenotypically but g^{53d} to be orange eyed and easily separable. Earlier authors claims g^2 lighter than g^1 . Pigmentation of Malpighian tubules also reduced (Brehme and Demerec, 1942, *Growth* 6: 351-56). Eye color (g^2) autonomous in transplants into wild type host (Beadle and Ephrussi, 1936, *Genetics* 21: 225-47).

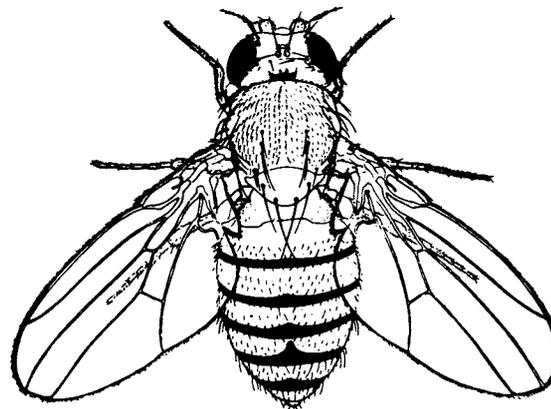
alleles:

allele	origin	discoverer	ref ^α	phenotype ^β
<i>g</i> ¹	spont	Bridges, 15b19	1-5	
<i>g</i> ²	spont	Bridges, 18c28	5, 13	partially dominant in <i>car</i> homozygotes (Schultz)
<i>g</i> ^{2rv}				
<i>g</i> ³	spont	Bridges, 22d22	5, 13	
<i>g</i> ^{4γ}	X ray	Glass, 1929	5, 6	
<i>g</i> ^{17Bδ}	X ray		5, 20	male sterile
<i>g</i> ²⁶⁻¹⁰	X ray	Sobels, 57j	5, 19	
<i>g</i> ²⁶⁻¹⁵	SMS	Sobels, 57j	5, 19	
<i>*g</i> ²⁶⁻⁴¹	SMS	Sobels, 57j	5, 19	weak allele
<i>*g</i> ²⁸⁻¹⁰	SMS	Sobels, 1957	5, 19	
<i>g</i> ²⁸⁻⁴⁰	SMS	Sobels, 1957	5, 19	
<i>*g</i> ^{29h}	spont	E. Wallace, 29h	4	
<i>*g</i> ^{30d}	spont	Bridges, 30d	4	
<i>*g</i> ^{32d}	spont	Emerson, 32d	4	
<i>*g</i> ^{33j}	spont	Bridges, 33j	4	
<i>*g</i> ³³ⁱ	spont	Ives, 33e	4	
<i>*g</i> ^{34e}	spont	Duncan 34e	4	
<i>*g</i> ^{37c}	spont	Mossige, 37c	4	
<i>g</i> ^{37f}	spont	Eeken, 37f	4	
<i>*g</i> ^{37k}	spont	Mather, 37k	4	
<i>*g</i> ^{38b}	spont	Bridges, 38b	4	
<i>g</i> ^{42a}	X ray	Green, 42a16	4	
<i>g</i> ^{49h}	32p	King, 49h	11	females have 60% normal viability
<i>g</i> ^{50e}			2, 3	
<i>g</i> ^{53d}	spont	Hexter, 53d	9, 10	orange eye
<i>*g</i> ^{55k}	spont	Williams, 55k	15	
<i>g</i> ^{64bε}	X ray	Ives, 64b11		
<i>g</i> ^{68d}	EMS	Maddern, 68el	8	
<i>g</i> ^{70k}	spont	Schwink, 70k10	18	
<i>g</i> ⁷⁹ⁱ	spont	Najera	16	
<i>*g</i> ²⁷¹⁻²	X ray	Demerec, 1933	5	male lethal, cell lethal
<i>*g</i> ²⁷¹⁻⁶	X ray	Demerec, 34a	5	male lethal, cell lethal
<i>*g</i> ²⁷¹⁻⁹	X ray	Hoover, 35h	5	male lethal
<i>*g</i> ²⁷¹⁻¹⁰	X ray	Hoover, 35h	5	male lethal
<i>*g</i> ^e	spont	Gottschewski, 34g17	7	
<i>g</i> ^{EMS}				
<i>g</i> ^F				
<i>g</i> ^{im}	spont	Waddle, 1974		
<i>g</i> ^{-l}				
<i>g</i> ^{S1}	spont	Schalet	3	
<i>g</i> ^{S2}	spont	Schalet	17	
<i>g</i> ^{S3}	spont	Schalet	17	
<i>g</i> ^{tuh1}	spont	Kuhn	12	
<i>g</i> ^{tuh2}	spont	Kuhn	12	reduced viability, fertility
<i>g</i> ^{wζ}	X ray	Muller	2, 3, 14	subliminal allele

allele	origin	discoverer	ref ^α	phenotype ^β
<i>g</i> ^{Xη}	X ray	Muller	14	female fertility reduced
^α				1 = Bridges, 1916, <i>Genetics</i> 1: 151; 2 = Chovnick, 1958, <i>Proc. Nat. Acad. Sci. USA</i> 44: 333-37; 3 = Chovnick, 1961, <i>Genetics</i> 46: 493-507; 4 = CP552; 5 = CP627; 6 = Glass, 1935, <i>DIS</i> 2: 7; 7 = Gottschewski, 1935, <i>DIS</i> 4: 8,15; 8 = Hayman and Maddern, 1969, <i>DIS</i> 44: 50; 9 = Hexter, 1958, <i>Proc. Nat. Acad. Sci. USA</i> 44: 768-71; 10 = Hexter, 1963, <i>Proc. Nat. Acad. Sci. USA</i> 50: 372-79; 11 = King, 1950, <i>DIS</i> 24: 58; 12 = Kuhn, 1972, <i>DIS</i> 49: 38; 13 = Morgan, Bridges, and Sturtevant, 1925, <i>Bibliogr. Genet.</i> 2: 226 (fig.); 14 = Muller, 1946, <i>DIS</i> 20: 67; 15 = Muller, 1956, <i>DIS</i> 30: 30; 16 = Najera, 1985, <i>DIS</i> 61: 215; 17 = Schalet, 1986, <i>Mutat. Res.</i> 163: 115-44; 18 = Schwinck and Schwinck, 1972, <i>DIS</i> 49: 39; 19 = Sobels, 1958, <i>DIS</i> 32: 84; 20 = Valencia, 1966, <i>DIS</i> 41: 58.
^β				Lists only special phenotypic attributes not mentioned for most alleles.
^γ				Induced in <i>In(1)dl-49</i> .
^δ				Associated with <i>In(1)12B14-15;19F</i> superimposed on <i>In(1)sc^{SIL}SC^{SR}+dl-49</i> .
^ε				See <i>DIS</i> 60: 230.
^ζ				Associated with <i>In(1)</i> with breakpoints between <i>dy</i> and <i>f</i> .
^η				Associated with <i>In(1)12;19-20</i> .

cytology: Placed in 12B6-7 (Lefevre, 1976, *Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, p59.

other information: Alleles separable by conversion; g^{53d} to the left of g^2 .

**G: Gull**

From Mohr, 1929, *Z. Indukt. Abstamm. Vererbungsl.* 50: 113-200.

G: Gull**location:** 2-12.0.**origin:** Spontaneous.**discoverer:** Mohr, 19k23.**references:** 1923, *Studia Mendeliana* (Brunae), pp. 266-87 (fig.).1927, *Proc. Intern. Congr. Genet.*, 5th., Vol. 2: 1136.1929, *Z. Indukt. Abstamm. Vererbungsl.* 50: 113-200 (fig.).

phenotype: Wings large, held out from sides at 45-90° angle, curved downward, and somewhat pointed. Vein L1 thickened; crossveins closer together, sometimes broken. Thoracic and vertical bristles duplicated in majority of flies. *G/ft* has exaggerated *ft* phenotype. Partially

inhibited by *ds/+* and much inhibited by *ds/ds*. Homozygous lethal. RK2.

cytology: Placed in 24D3-8 based on its inclusion in *Df(2L)M = Df(2L)24D3-4;25A2-3* but not in *Df(2L)dp-h28 = Df(2L)24D7-8;24F7-25A1* (Szydony and Reuter, 1988, Genet. Res. 51: 197-208).

other information: Causes local shortening of map by about 1.1 units. Is a deficiency for or an allele of *ft*.

***G^{rv}*: Gull-reverted**

origin: Spontaneous derivative of *G*.

discoverer: Bridges, 1930.

phenotype: Does not show *G* phenotype. Allelic to *ft* but does not exaggerate *ft*. Lethal in combination with *G*. RK2.

G* protein: see *Gα* and *Gβ

g2: see *Tkr*

g-l: see *sno*

g, Inh: see *g^X*

***Gα*: G protein α subunit**

G proteins belong to a family of membrane-associated guanine nucleotide-binding proteins that couple specific receptors for extracellular signals to specific intracellular effectors, thus regulating the activity of these effectors. When not interacting with the receptor, G proteins are usually in the form of a heterotrimer made up of α, β, and γ subunits, with the α subunit bound to GDP. Upon activation by the receptor, the α subunit exchanges GDP for GTP, dissociates from the β-γ subunits, and interacts with the effector. Afterwards GTP is hydrolyzed, and the heterotrimer of α, β, and γ subunits is formed again. In *Drosophila melanogaster*, three genes coding for different α subunits have been identified.

gene	location	α subunit encoded	ref ^α	cytology
<i>G α 47A</i> ^β	2-[60]	G _o 1, G _o 2	1, 5-7	47A
<i>G α 60A</i>	2-[106]	G _s	2, 6	60A
<i>G α 65A</i> ^γ	3-[20]	G _i	3, 4, 6	65A

^α 1 = DeSousa, Hoveland, Yarfitz, and Hurley, 1989, J. Biol. Chem. 264: 18544-51; 2 = Provost, Somers, and Hurley, 1988, J. Biol. Chem. 263: 12070-76; 3 = Quan, Wolfgang, and Forte, 1990, Nat. Acad. Sci. USA 86: 4321-25; 4 = Quan and Forte, 1990, Mol. Cell Biol. 10: 910-17; 5 = Thambi, Quan, Wolfgang, Spiegel, and Forte, 1989, J. Biol. Chem. 264: 18552-60; 6 = Wolfgang, Quan, and Forte, 1990, unpublished; 7 = Yoon, Shortridge, Bloomquist, Schneuwly, Perdew, and Pak, 1989, J. Biol. Chem. 264: 18536-43.

^β Synonym: *dgo* (Yoon *et al.*, 1989).

^γ Synonym: *DGα1* (Provost *et al.*, 1989).

***Gα47A*: G protein α subunit at 47A**

phenotype: Encodes a G protein α subunit that is expressed mainly in the nervous system (DeSousa *et al.*, 1989; Thambi *et al.*, 1989) and shows homology to mammalian *Goα*. Low levels of this G protein are found in the embryo until completion of germband shortening; then high levels begin to be expressed in the neuropil (Wolfgang, *et al.*, 1990).

molecular biology: The gene has been cloned using rat, bovine, and human cDNA as probes. Genomic, cDNA and putative amino acid sequences have been obtained (DeSousa *et al.*, 1989; Thambi *et al.*, 1989; Yoon *et al.*, 1989). Three transcripts have been identified, a 3.8-4.2 kb transcript expressed at high levels in heads and bodies

of adults, a 5.3-6 kb transcript expressed at high levels in the heads only, and a 3.4-3.5 kb transcript found in bodies (Thambi *et al.*, 1989; Yoon *et al.*, 1989). Transcripts are also found in embryos, larvae, and pupae. The mRNA occurs abundantly in the cell bodies of neurons in the cortex of the brain and the thoracic ganglia; a low level of mRNA is found in nurse cells and oocytes (De Sousa *et al.*, 1989). Two classes of cDNA that differ in DNA sequences in the 5'-noncoding region and the 5'-most part of the coding region are generated by the splicing together of seven exons. The first exon in the splice differs in length and location in the two classes; the last six exons are identical in both classes (Yoon *et al.*, 1989). This alternative splicing results in the encoding of two proteins 354 amino acids long that differ in seven amino acids in the amino terminal region (De Sousa *et al.*, 1989; Thambi *et al.*, 1989; Yoon *et al.*, 1989). Transcripts corresponding to both classes of cDNA are found in the central nervous system (De Sousa *et al.*, 1989). Membrane preparations from *Drosophila melanogaster* heads contain high levels of a pertussin toxin (PTX) substrate with 82% sequence identity to vertebrate G proteins that are modified similarly by PTX (De Sousa *et al.*, 1989; Thambi *et al.*, 1989).

***Gα60A*: G protein α subunit at 60A**

phenotype: Encodes a stimulatory G protein α subunit with high homology to mammalian *Gsα* that is responsible for the coupling of extracellular receptors to adenylate cyclase and an increase in the second messenger cAMP (Quan *et al.*, 1989). The protein is expressed at low levels until completion of germband shortening; then high levels begin to be expressed in the neuropil (Wolfgang *et al.*, 1990).

molecular biology: The gene has been cloned from head DNA using bovine cDNA as a probe. The genomic, cDNA and putative amino acid sequences determined (Quan *et al.*, 1989; Quan and Forte, 1990). The gene is 4.5 kb long. Transcripts are most abundant in the central nervous system; mRNAs were also isolated (using PCR) from whole flies and bodies (Quan and Forte, 1990). There are 9 exons separated by 8 introns, the introns showing a size variation of 56 bp to 1.4 kb. Alternate splicing at the 3' end of intron 7 produces transcripts that encode a long or short G protein α subunit. The cDNAs differ by the inclusion or deletion of nine nucleotides at the exon 7-8 junction. The long or short protein products differ by inclusion or deletion of three amino acids and the substitution of a Ser in the long protein and for a Gly in the short protein (Quan and Forte, 1990). The apparent molecular weights of these two forms are 51,000 and 48,000 daltons.

***Gα65A*: G protein α subunit at 65A**

phenotype: Encodes a G protein α subunit displaying sequence homology to mammalian *Giα* that inhibits adenylate cyclase activity. The G protein is uniformly distributed in oocytes, becomes restricted to the posterior pole of the embryo during early cleavage and is lost during the blastoderm stage (Wolfgang *et al.*, 1990).

molecular biology: The gene has been cloned using bovine transducin α subunit cDNA as a probe; the genomic, cDNA, and putative amino acid sequences were determined. There are five exons separated by four introns. A major 2.3 kb transcript and a minor 1.7 kb

transcript have been identified. These transcripts, first isolated in very young embryos, are most abundant in the embryonic and pupal stages. The gene encodes a G protein α subunit with amino acid sequences showing 77-78% identity to the sequences in bovine inhibitory G proteins; an isoleucine in the *Drosophila* protein, however, has been substituted for a cysteine in the vertebrate proteins (Provost *et al.*, 1989).

Gal: β -Galactosidase

location: 2-[20].

references: Kotarski, Pickert, and MacIntyre, 1983, *Genetics* 105: 387-407.

Knipple and MacIntyre, 1984, *Mol. Gen. Genet.* 198: 75-83.

Fargnol, Hyde, and Sofer, 1985, *Genetics* 110: s7.

phenotype: Inferred to be the gene that codes for the enzyme β galactosidase [B-GAL (EC 3.2.1.23)] because of response of the enzyme level to gene dosage. A null strain (*Gal^{ng}*) does not survive on medium containing lactose (Fargnol *et al.*, 1985), but the Bethylie wild-type strain shows more than 95% survival on lactose medium. Induction in cell culture by hormone studied by Best-Bellpomme, Courgen, and Rambach (1978, *Proc. Nat. Acad. Sci. USA* 75: 6102-06).

alleles: *Galⁿ¹* and *Galⁿ²* (Knipple and MacIntyre, 1984), *Gal^{ng}* (Fargnol *et al.*, 1985).

cytology: Placed in 26A8 since located between autosomal breakpoints of *T(Y;2)H69 = T(Y;2)26A7-8* and *T(Y;2)D211 = T(Y;2)26B3-5* and uncovered by *Df(2L)GpdhA = Df(2L)26D7-E1;26A8-9* but not by *Df(2L)c17 = Df(2L)26D7-E1;26A7-8* (Knipple and MacIntyre, 1984).

gap: see *gp*

Gapdh1: Glyceraldehyde dehydrogenase 1

location: 2-[57].

references: Tso, Sun, Wu, 1985, *J. Biol. Chem.* 260: 8820-28.

phenotype: One of two genes (*Gapdh1* and *Gapdh2*) that code for the enzyme glyceraldehyde-3-phosphate dehydrogenase [G3PD (EC.1.2.12)]. These genes are expressed in almost all cells, especially those that undergo a high rate of glycolysis.

cytology: Glyceraldehyde 1 has been located at 43E-F by *in situ* hybridization to the salivaries.

molecular biology: *Gapdh1* has been cloned and its nucleotide and deduced amino acid sequences determined. *Gapdh1* and *Gapdh2* show a striking degree of identity in their coding regions but are entirely different in their 5' and 3' flanking regions. Since, for the enzymes encoded by these genes, there are only eight amino acid residue differences in a total of 332 residues, Tso *et al.* suggest that the two genes have arisen by duplication and translocation events. The transcription initiation site and the polyadenylation site of *Gapdh1* have been determined; the gene is transcribed from left to right and its transcript is present in adult flies. In its -30 bp region, *Gapdh1* resembles many RNA polymerase II transcribed promoters in the lack of a sequence homologous to the TATA box. No introns are found in its coding region.

Gapdh2: Glyceraldehyde dehydrogenase 2

location: 1-[54].

references: Tso, Sun, Wu, 1985, *J. Biol. Chem.* 260: 8820-28.

Sun, Lis, and Wu, 1988, *Genes Dev.* 2: 743-53.

phenotype: Another gene that codes for the enzyme glyceraldehyde-3-phosphate dehydrogenase (see *Gapdh1*). The level of expression of *Gapdh2* has been found to be regulated developmentally (Sun *et al.*, 1988).

cytology: Glyceraldehyde 2 has been located at 13F by *in situ* hybridization to the salivaries.

molecular biology: *Gapdh2* has been cloned and its nucleotide and deduced amino acid sequences determined. As in *Gapdh1*, the transcription initiation site and the polyadenylation sites of *Gapdh2* have been determined. The promoter region of *Gapdh2* (unlike that of *Gapdh1*) contains a consensus TATA box sequence as well as a CAAT box sequence (Tso *et al.*, 1985). There is an intron located in the 5' noncoding region, but no other intervening sequences. There are two distinct regulatory regions, URS1 and URS2, in the first 145 bp of the 5' flanking region of *Gapdh2*. URS1 acts throughout development to activate transcription; URS2 acts along with URS1 to activate transcription in larval and adult stages, but represses transcription in Schneider cells and perhaps some embryonic stages (Sun *et al.*, 1988).

garnet: see *g*

Gart: see *ade3*

gastrulation defective: see *gd*

gat: gate (J.C. Hall)

location: 2-60.

origin: Induced by ethyl methanesulfonate.

references: Jackson, 1983, *J. Neurogenet.* 1: 3-15.

phenotype: Eclosion of flies is poorly synchronized in light-dark cycle; eclosion becomes arrhythmic in constant darkness; fluctuations of male's courtship song interpulse intervals define a sloppy rhythm or are arrhythmic (Jackson and Kyriacou).

other information: Allelic to *psi-2*, based on aberrant short-term rhythms of courtship song in *gat/psi-2* males (Kyriacou).

G β 13F: G protein β subunit at 13F

location: 2-[54].

references: Yarfitz, Provost and Hurley, 1988, *Proc. Nat. Acad. Sci. USA* 85: 7134-38.

phenotype: This gene encodes one isoform of the G protein β subunit in *Drosophila melanogaster*. After the G protein α has interacted with the effector, the β - γ subunits reassociate with the α subunit and bring the α subunit to its specific receptor.

cytology: Located at 13F by *in situ* hybridization to the salivaries.

molecular biology: Gene cloned using bovine transducin β subunit cDNA as a probe, and the genomic, cDNA and the putative amino acid sequences determined. There is an open reading frame (ORF) of 1023 bases which codes for a predicted protein of 340 amino acids showing more than 80% identity to the corresponding mammalian proteins. The *Drosophila* protein is unique at 15% of the amino acid positions. Transcripts of 5.2, 4.2, 3.3, 3.0 and 1.9 kb are expressed from mid-embryo through adult

stages, the highest level of expression being in late embryos and pupae. Expression is low in larvae and can hardly be detected in adults. Early embryos also show transcripts of 3.1, 2.5 and 2.0 kb. In adult flies there is more mRNA in the heads than in the bodies. Restriction mapping and DNA sequencing indicate the absence of introns in the presumptive coding region of the gene; however, at least two introns are present in the 5' noncoding region and there is evidence for alternative splicing in this region.

gcd: giant cell defect (J.C. Hall)

location: 1-48.

origin: Induced by ethyl methanesulfonate.

discoverer: Heisenberg.

phenotype: Cellular cortex of central brain shows one giant cell body lateral to base of each calyx (bulges located relatively dorsally and at posterior edge of cortex); penetrance of this phenotype approximately 85%.

gd: gastrulation defective

location: 1-36.78.

origin: Induced by ethyl methanesulfonate.

synonym: *fs(1)A573*, *fs(1)M18*.

references: Gans, Audit, and Masson, 1975, *Genetics* 81: 683-704.

Anderson and Nüsslein-Volhard, 1984a, *Nature (London)* 311: 223-27.

1984b, *Pattern Formation (Malacinski and Bryant, eds.)*. Macmillan, New York. pp. 269-89.

Konrad and Mahowald, 1984, *Molecular Aspects of Early Development (Malacinski and Klein, eds.)*. Plenum, New York. pp. 167-88.

Anderson, Bokla, and Nüsslein-Volhard, 1985, *Cell* 42: 791-98.

phenotype: Maternal-effect lethal; embryos produced by homozygous females exhibit hyperplasia of dorsal cuticular elements and aplasia of ventral elements; phenotype of strong alleles, e.g. *gd*⁷, indistinguishable from that of *dl*. Weaker alleles display some ventral elements. Gastrula exhibits excessive furrowing both dorsally and ventrally [Zalokar, Audit, and Erk, 1975, *Dev. Biol.* 47: 419-32 (fig.)]. Polarity of the egg shell unaffected. Temperature-sensitive period of temperature-sensitive alleles begins several hours prior to ovoposition and persists until 1.5 h after fertilization. Five of eight mutants classed as weak on the basis of filzkörper development; these five tend to be more dorsalized in posterior than in anterior parts of the embryo, as the setal bands become progressively narrowed posteriorly. *gd*⁷/*gd*⁷; *Toll*+ females produce lateralized embryos; most ventral and dorsal pattern elements missing; lateral cephalic furrow seen both dorsally and ventrally, and ventral setae formed at all dorsal-ventral positions.

alleles:

allele	synonym	discoverer	ref ^α
<i>gd</i> ¹	<i>fs(1)A573</i>	Audit	2, 7
<i>gd</i> ²	<i>fs(1)M18 11-1524</i>	Mohler	4, 5
<i>gd</i> ³	<i>fs(1)M18 12-4955</i>	Mohler	4, 5
<i>gd</i> ⁴	<i>fs(1)M18 13-935</i>	Mohler	4, 5
<i>gd</i> ⁵	<i>fs(1)M18 13-1697</i>	Mohler	5
<i>gd</i> ⁶ β	<i>fs(1)M18 13-1853</i>	Mohler	4, 5
<i>gd</i> ⁷ γ	<i>fs(1)M18 14-743</i>	Mohler	1, 5
<i>gd</i> ⁸	<i>fs(1)190.5</i>		1, 6

- α 1 = Anderson and Nüsslein-Volhard, 1984, *Pattern Formation (Malacinski and Bryant, eds.)*. Macmillan, New York, pp. 269-89; 2 = Gans, Audit, and Masson, 1975, *Genetics* 81: 683-704; 3 = Konrad and Mahowald, 1984, *Molecular Aspects of Development (Malacinski and Klein, eds.)*. Plenum, New York, pp. 167-88; 4 = Mohler, 1977, *Genetics* 85: 259-72; 5 = Mohler and Carroll, 1984, *DIS* 60: 236-41; 6 = Tearle and Nüsslein-Volhard, 1987, *DIS* 66: 209-26; 7 = Zalokar, Audit, and Erk, 1975, *Dev. Biol.* 47: 419-32 (fig.).
- β *gd*⁶ complements *gd*³ and partially complements *gd*².
- γ Pattern of *ftz* protein stripes in homozygous females identical to that of other maternal dorsalizing mutants (Carroll, Winslow, Twombly, and Scott, 1987, *Development* 99: 327-32).

cytology: Placed in 11A1-7, since uncovered by *Df(1)KA10 = Df(1)11A1;11A7-8* (Tearle and Nüsslein-Volhard, 1987, *DIS* 66: 209-26).

***Gd: Gulloid**

location: 3-78.

origin: Spontaneous in *Dp(2;3)P*.

discoverer: Bridges, 22g26.

phenotype: *Gd*+ wings shorter, blunter, slightly more spread, and have crossveins closer together than wild type. Homozygous lethal. RK3A.

cytology: Inseparable from *Dp(2;3)P = Dp(2;3)58E3-F2;60D14-E2;96B5-C1*.

Gdh: Glutamate dehydrogenase

location: 3-81.7 (between *hh* and *tx*).

references: Caggese, dePinto, and Ferrandino, 1982, *Biochem. Genet.* 20: 449-60.

Novak and Piechowska, 1986, *DIS* 63: 102-04, 104-07.

phenotype: Structural gene for NAD⁻ dependent glutamate dehydrogenase [GDH (EC1.4.1.4)], which comprises six 57,000 dalton subunits. Enzyme activity inducible by addition of glutamate to medium. Nearly all larval activity can be isolated with the mitochondria.

alleles: *Gdh*^F and *Gdh*^S identified; heterozygote produces broad band on cellulose acetate gel.

Gdh: see *Gpdh*

gdl: gonadal

location: 3-{42}.

references: Schultz and Butler, 1989, *Genes Dev.* 3: 232-42.

Schultz, Schlomchik, Cherbas, and Cherbas, 1989, *Dev. Biol.* 131: 515-23.

phenotype: Member of cluster of overlapping genes; *gdl* is proximally overlapped by *Eip28/29* and distally by *Z600*. The function of the *gdl* gene in the germ lines of both males and females is unknown at present (Schultz and Butler, 1989).

cytology: Located in 71C3-D4 (Schultz and Butler, 1989).

molecular biology: Gene cloned and partially sequenced. *gdl* is expressed in either of two modes (male or female). There are four transcripts, both due to different transcription initiation sites and multiple polyadylation sites at their 3' ends. *gdl*^M transcripts are first detectable during late larval development, appear at higher levels in pupae, and occur abundantly in the testes of adults, being 1200 and 1500 nucleotides long; *gdl*^F transcripts are found in adult ovaries, early embryos and Kc cells, are 1000 and 1300 nucleotides long. and have the same terminal exons as *gdl*^M transcripts. A common ORF occurs in all four transcripts. None of these transcripts are found in male or female animals lacking a germ line. In each mode, the

longer transcript is the result of a polyadenylation site within the 5' exon of *Eip28/29*. The two genes *Eip28/29* and *gdl* do not share coding DNA. Transformation experiments indicate that *gdl* can be expressed outside its normal overlapping gene environment.

Gdt-3: see *Gpdh^H* and *Gpdh^L*

***ge: genitalless**

location: 1-0.1.

origin: Induced by methyl methanesulfonate (CB. 1540).

discoverer: Fahmy, 1955.

references: 1958, DIS 32: 70.

phenotype: External male genitalia absent or grossly deformed. Bristles fine; wings often small and deformed. Tergites abnormal; abdomen frequently contains melanotic tumors. Males viable but sterile. RK3.

Gerec: see *Fs(3)Sz9*

gespleten: see *gv*

gfA: giant fiber A (J.C. Hall; R.A. Wyman)

location: 1- 62 (between *f* and the centromere).

origin: Induced by ethyl methanesulfonate.

references: Thomas, 1980, Neurosci. Abstr. 6: 742.

Thomas and Wyman, 1984, J. Neurosci. 4: 530-38.

phenotype: Adults have an aberrant startle response; they do not jump when presented with a lights-off stimulus. The dorso-longitudinal indirect flight muscles (DLMs) are abnormally driven by the giant fiber pathway neurons (see entry for *ben*). DLMs are driven at long and variable latencies after brain stimulation. Morphology of the giant fiber and its response to stimulation is normal. The motor neuron of the tergotrochanteral (jump) muscle is driven normally by the giant fiber. The physiological defect in *gfA* is probably located at the synapses between the peripherally synapsing interneuron (PSI) and the DLM motor neurons in the thoracic ganglion. *gfA* complements *pas*.

alleles:

allele	discoverer	synonym
<i>gfA</i> ¹	Thomas	<i>nj-54</i>
<i>gfA</i> ²	Thomas	<i>nj-75</i>
<i>gfA</i> ³	Tanouye	<i>4-22-09</i>
<i>gfA</i> ⁴	Baird	<i>nj-507</i>
<i>gfA</i> ⁵	Baird	<i>nj-520</i>
<i>gfA</i> ⁶	Baird	<i>nj-522</i>

cytology: Located between 18A5 to 18D1-2, based on uncovering by *Df(1)JA27 = Df(1)18A5;18D1-2*.

other information: *gfA*¹/*Df(1)JA27* is said to be indistinguishable from *gfA*¹/*gfA*¹ (Thomas and Wyman, 1984). Physiological effects of *gfA*¹ and *gfA*² are ostensibly the same.

gg: goggle

location: 1-23.1 (no crossovers with *oc* among 4300 flies).

phenotype: Eyes protruding and bulging, placed far back on a narrow head. Facets very large in rough areas. Wings smaller with fringed marginal hairs; dusky; pebbly appearance caused by large cells. Bristles coarse and irregular; hairs sparse and irregular, especially on abdomen. Body small in late counts. Viability reduced. Females usually sterile; males usually fertile. RK3.

alleles:

allele	origin	discoverer	ref ^α	comments ^β
<i>gg</i> ¹	spont	Nichols-Skoog, 34e14	2	20% viability
<i>gg</i> ²	spont	Waletsky, 37e	1, 2	postorbital bristles sparse; no oocytes at 19°, few at 25°, no ovoposition
<i>gg</i> ³	spont	R.F. Grell, 53d	2, 3	both sexes fertile
<i>gg</i> ⁴	spont	Mohler, 54j28	2, 5	weak allele-pebbly wings diagnostic
<i>gg</i> ^{5γ}	X ray	Lefevre	4	
<i>gg</i> ^{6δ}	X ray	Lefevre	4	
<i>gg</i> ^{IS1}	spont	Schalet		reduced viability
<i>gg</i> ^{IS2}	spont	Schalet		reduced viability

^α 1 = Beatty, 1949, Proc. R. Soc. Edinburg Sect. B: Biol. 63: 249-70; 2 = CP627; 3 = Grell, 1953, DIS 27: 59; 4 = Lefevre, 1981, Genetics 99: 461-80; 5 = Mohler, 1956, DIS 30: 78.

^β Special phenotypic features differing from description of nominate allele.

^γ Synonym: *l(1)C210*. Cytology: *Tp(3;1)7F9;76B;80B*.

^δ Synonym: *l(1)N83*. Cytology: *In(1)7F9-8A1;9A4*.

cytology: Placed in 7F10 by Lefevre (1981, Genetics 99: 461-80) on the basis of two rearranged *gg* alleles [*In(1)N83 = In(1)7F1;9A4* and *Tp(3;1)C210 = Tp(3;1)7F1;76B;80*].

gho: ghost

location: 2-68.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82 (fig.)

phenotype: Homozygous lethal; embryonic cuticle undifferentiated.

alleles: Three.

giant: see *gt*

Giant: see *Gt*

giant cell defect: see *gcd*

giant fiber A: see *gfA*

giant nuclei: see *gnu*

giant ring gland: see *grg*

giantoid: see *gtd*

gl: glass

location: 3-63.1.

references: Bridges and Morgan, 1923, Carnegie Inst. Wash. Publ. No. 327: 188 (fig.).

Morgan, Bridges and Sturtevant, 1925, Bibliog. Genet. 2: 214.

Pak, Grossfield and White, 1969, Nature (London) 222: 351-54.

Moses, Ellis and Rubin, 1989, Nature (London) 340: 531-36.

phenotype: Null mutations of *glass* remove photoreceptor cells in all of the three organs in which they occur. The mutants have compound eyes that are reduced in size and elliptical or diamond shaped; the texture is glassy from fused facets and irregular surface. Ocelli are flattened and lack pigment; there is no ocellar neuropil (Moses *et al.*, 1989). In larvae, the visual Bolwig organs are absent in *gl*², but present in *gl*³. Neither mutant expresses chaoptin (Moses *et al.*, 1989). Color of compound eyes and Malpighian-tubules variably reduced depending on

allele. In the presence of the dimorphic sex-linked modifier, *msd(gl)^d*, males have more pigment than females, whereas males have the same eye color as females when they carry the monomorphic allele *msd(gl)^m* [Birchler, 1984, Genet. Res. 44: 125-32]. *dsx* homozygotes of either *X* constitution but especially *XX* produce more pigment than *dsx⁺* (Smith and Lucchesi, 1969, Genetics 61: 607-18); however *msd(gl)* was not controlled by Smith and Lucchesi. Pigmentation increases with increased developmental temperature. At high or very low levels of pigment production the sexual dimorphism disappears. Weak alleles have small smooth eyes and normal pigment levels in both sexes. Mild alleles have eyes reduced to two-thirds normal size and approximately 20% normal eye-pigment levels and male levels approximately 2X female levels. Moderate alleles reduce eye size to half normal and pigment to about 5% normal levels with male levels approximately 3X those of females. Strong alleles have eye area less than half normal, and eyes are virtually colorless in both sexes (Smith and Lucchesi). The optic lamina is virtually nonexistent and the medulla is very small and disorganized in *gl¹*; the lamina and medulla are both small and disorganized in *gl²* and *gl³* [Meyerowitz and Kankel, 1978, Dev. Biol. 62: 112-42 (fig.)]. Mosaic studies using *gl³* show that the *gl* genotype of the eye, rather than that of the optic lobe, determines the axon array of the optic lobe (Meyerowitz and Kankel, 1978). Structures of individual neurons studied by S.H. Garen and Kankel (1983, Dev. Biol. 96: 445-56). Retinula cells of the mutant are irregular and rhabdomeres are lacking; the electroretinogram shows no response to light (Pak, Grossfield and White, 1969). *gl* flies are nonphototactic and males exhibit depressed courtship activity [Merrell and Underhill, 1956, Genetics 41: 469-85; Hall, Tomkins, Kyriacou, Siegal, Von Schilcher and Greenspan, 1980, Developmental Neurobiology of *Drosophila* (Siddiqi, Babu Hall and Hall, eds.). Plenum Press, New York, pp. 425-56]. Wing beat frequency increased (Williams and Reed, 1944, Am. Nat. 78: 214-23).

alleles:

allele	origin	discoverer	ref ^α	strength
<i>gl¹</i>	spont	Muller, 18b	2, 4, 5, 11 12, 15	moderate
<i>gl²</i>	spont	R.L. King, 1927	4, 5, 11, 13 15	mild
<i>gl³</i>	spont	Stern	4-6, 11, 13 15	weak
* <i>gl⁴</i>	spont	Villee, 40d	4, 5, 16, 17	moderate
* <i>gl^{38l}</i>	spont	Steinberg, 38l13	4	
* <i>gl^{40h}</i>	spont	Ives, 40h	4, 5, 9	weak
* <i>gl^{41e}</i>	spont	Oliver, 41e1	5, 14	weak
* <i>gl^{51k}</i>	spont	Edmondson, 51k	5, 7	moderate
<i>gl^{54g}</i>	spont	Hexter, 54g	5, 8	strong
<i>gl^{60j}</i>			15	
<i>gl^{62d}</i>	spont	Tano, 62d	3	weak
* <i>gl^{63a}</i>	spont	Ashburner, 63a14	1, 15	mild
<i>gl^{63dβ}</i>	γ ray	Ives, 63d29	10	weak
<i>gl^{63f}</i>	spont	Ashburner, 63f6	1, 15	mild
<i>gl^{B2}</i>	EMS	Moses	13	
<i>gl^{B16γ}</i>	EMS	Moses	13	
<i>gl^{BX2}</i>	X ray	Moses	13	
<i>gl^{BX5}</i>	X ray	Moses	13	
<i>gl^{BX6δ}</i>	X ray	Moses	13	
<i>gl^{BX7γ}</i>	X ray	Moses	13	
<i>gl^{BX9}</i>	X ray	Moses	13	

allele	origin	discoverer	ref ^α	strength
<i>gl^{WG1}</i>	γ ray	Moses	13	
<i>gl^{WG2}</i>	γ ray	Moses	13	
<i>gl^{WG3}</i>	γ ray	Moses	13	

- ^α 1 = Ashburner and Hudson, 1966, DIS 41: 60; 2 = Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. 327: 118 (fig.); 3 = Burdick, 1963, DIS 37: 47; 4 = CP552; 5 = CP627; 6 = Csik, 1929, Biol. Zentralbl. 49: 419-21; 7 = Edmondson, 1952, DIS 26: 60; 8 = Hexter, 1956, DIS 30: 72; 9 = Ives, 1941, DIS 14: 39; 10 = Ives, 1965, DIS 40: 35; 11 = Meyerowitz and Kankel, 1978, Dev. Biol. 62: 112-42 (fig.); 12 = Morgan, Bridges and Sturtevant, 1925, Bibliogr. Genet. 2: 214 (fig.), 226; 13 = Moses, Ellis, and Rubin, 1989, Nature (London) 340: 531-36; 14 = Oliver, 1942, DIS 16: 53; 15 = Smith and Lucchesi, 1969, Genetics 61: 607-18; 16 = Villee, 1941, DIS 14: 40; 17 = Villee, 1942, Univ. Calif. Publ. Zool. 49: 137;
- ^β Associated with *T(2;3)gl^{63d} = T(2;3)47B;91A* (Robinson and Curtis, 1972, Can. J. Cytol. Genet. 14: 129-37).
- ^γ Lethal allele.
- ^δ Associated with *In(3R)gl^{BX6}* (Moses *et al.*, 1989).

cytology: Located in 91A1-2 since all the DNA in the central restriction map of a wild-type *gl* genomic clone lies within this polytene band; also *In(3R)gl^{BX6}* and *T(2;3)gl^{63d}* appear to have breakpoints within this region (Moses *et al.*, 1989).

molecular biology: The glass gene was cloned by chromosome walking and the limits of the *gl²* gene determined by *P*-element mediated germline transformation (Moses *et al.*, 1989). The probe from one of the transforming fragments (9,954 bp) hybridized to a 3.0 kb poly(A)⁺ RNA in the heads of adult flies. All of the viable alleles tested affect only this transcript, believed to be that of glass, which is first detected in the early third larval instar, and remains to the adult stage in which it is head-specific. There are five exons and four introns. The 9,954 bp fragment has been sequenced and the predicted sequence of a protein of 604 amino acids determined. The protein encoded by the *gl* transforming fragment includes five zinc-finger repeats similar to those of the *Kr* protein but some sequences (Ser-Gln-Ser) have not been found in other zinc-finger proteins. Another fragment was obtained that extends further toward the centromere end of the chromosome and does not rescue glass gene function. The mutant *gl^{WG1}* deletes about 600 bp near the centromere end of the *gl* transforming fragment; *gl¹*, *gl²*, *T(2;3)gl^{63d}*, and *In(3R)gl^{BX6}* show breaks within the next region. The weak allele *gl³* inserts about 2.5 kb and the allele *gl^{60j}* inserts more than 30 kb into the third region, and *gl^{BX3}* removes about 4.5 kb from the distal end of the transforming fragment. All of these alleles are viable. The lethal alleles *gl^{B16}* and *gl^{BX7}* occupy the DNA distal to glass at 91A1-2 as well as the distal part of the *gl* transforming fragment, deleting about 20 and 18.5 kb respectively.

GI: Glued

location: 3-41.4 [0.9 unit from *Ly* (Mossige, 1935, DIS 4: 59; 1938, Hereditas 24: 110-16)].

references: Plough and Ives, 1934, DIS 1: 31-4.

Plough, 1934, DIS 2: 34-5.

Plough and Ives, 1935, Genetics 20: 42-69 (fig.).

Harte and Kankel, 1982, Genetics 101: 477-501 (fig.)

Swaroop, Paco-Larson and Garen, 1985, Proc. Nat. Acad. Sci. USA 82: 1751-55.

Swaroop, Sun, Paco-Larson and Garen, 1986, Mol. Cell

Biol. 6: 833-41.

Swaroop, Swaroop and Garen, 1987, Proc. Nat. Acad. Sci. USA 84: 6501-05.

phenotype: Eyes rough, smaller, and oblong; facets rounded; surface smooth and shiny like *gl*. Pattern, as indicated by staining with monoclonal antibodies, abnormal in the posterior eye disk (Renfrenz and Benzer, 1989, Dev. Biol. 136: 411-29). Architecture of the optic ganglia severely deranged; attributable to the mutant genotype of the overlying eye tissue (Meyerowitz and Kankel, 1978, Dev. Biol. 62: 112-42). Structures of individual neurons studied by S.H. Garen and Kankel (1983, Dev. Biol. 96: 445-56). Somatic-crossover studies and temperature-shift experiments with *Gl^{rv30}*, a temperature-sensitive allele, suggest that *Gl⁺* gene product is required in mid third instar; abnormal retinula fiber projections first observed in late third instar [Harte and Kankel, 1983, Dev. Biol. 99: 88-102 (fig.)]. No electro-retinogram [Grossfield, 1975, Handbook of Genetics (R.C. King, ed.). Plenum Press, New York and London, Vol. 3, p. 690]. Bristles generally shortened slightly and straighter than normal. Viability and fertility of heterozygote good. With respect to severity of expression *Df(3L)Gl2/+* = wild type; *Gl/+/+* = very weak Glued; *Gl/+* = weak Glued; *Gl/+* = Glued; *Gl/Gl/+* = few extreme Glued survivors, *Gl/Df(3L)Gl⁻* = *Gl/Gl* = lethal (Harte and Kankel). RK1.

alleles: In addition to *Gl¹*, which occurred spontaneously among the progeny of heat treated flies (Ives, 31f5), both Harte and Kankel (1982) and Garen, Miller, and Paco-Larson (1984, Genetics 107: 645-55) induced a number of new mutations and rearrangements at the locus; these fall into two general classes: (1) new lethal alleles in which the dominant phenotype is weak or absent, and (2) revertants of *Gl¹* induced by either ionizing radiation, ethyl methanesulfonate, or hybrid dysgenesis. 17 ethyl-methanesulfonate-induced lethal mutants are lethal in combination with either *Gl¹* or *Df(3L)Gl2* and appear to be point mutations, since they complement lethals in flanking loci. The time of death varies.

allele	synonym	ref ^α	<i>Gl¹/Df(3L)Gl2</i> lethal period
<i>Gl¹</i>		3	embryo/early first instar
<i>Gl¹¹</i>	<i>Gl¹⁻³</i>	1, 2	embryo/early first instar, cell lethal in eye or cuticle
<i>Gl¹²</i>	<i>Gl⁷⁻¹</i>	1, 2	embryo/early first instar
<i>Gl¹³ β δ</i>	<i>Gl⁷⁻⁶</i>	1, 2	early to mid pupa at 18°, late third instar/early pupa at 29°
<i>Gl¹⁴</i>	<i>Gl⁵⁻⁵</i>	1, 2	embryo/early first instar
<i>Gl¹⁵</i>	<i>Gl^{7E}</i>	1, 2	embryo/early first instar
<i>Gl¹⁶</i>	<i>Gl^{15C}</i>	1, 2	embryo/early first instar
<i>Gl¹⁷</i>		1	embryo/early first instar
<i>Gl¹⁸</i>		1	embryo/early first instar
<i>Gl¹⁹</i>		1	embryo
<i>Gl¹¹⁰</i>		1	embryo
<i>Gl¹¹¹</i>		1	early pupa
<i>Gl¹¹²</i>		1	late embryo/early first instar
<i>Gl¹¹³</i>		1	late embryo/early first instar
<i>Gl¹¹⁴</i>		1	early pupa
<i>Gl¹¹⁵</i>		1	late embryo/early first instar
<i>Gl¹¹⁶</i>		1	late embryo/early first instar
<i>Gl¹¹⁷</i>		1	late embryo/early first instar
<i>Gl¹¹⁸</i>	<i>Gl^{5J}</i>	2	embryo/early first instar
<i>Gl¹¹⁹</i>	<i>Gl^{5K}</i>	2	embryo/early first instar
<i>Gl¹²⁰</i>	<i>Gl²⁹⁻¹</i>	2	embryo/early first instar
<i>Gl¹²¹</i>	<i>Gl³²⁻²</i>	2	embryo/early first instar
<i>Gl¹²² γ</i>	<i>Gl³⁴⁻³</i>	2	mid to late pupa at 18°, early to mid pupa at 29°

allele	synonym	ref ^α	<i>Gl¹/Df(3L)Gl2</i> lethal period
α			1 = Garen, Miller, and Paco-Larson, 1984, Genetics 107: 645-55; 2 = Harte and Kankel, 1982, Genetics 101: 477-501; 3 = Plough and Ives, 1934, DIS 1: 31-4.
β			Surviving homozygotes produced at both 18° and 29°; homozygous stock can be established at 18° but not 29°; in crosses to wild type, homozygous males fertile at both temperatures and eggs maturing at 18° but not at 29° able to produce progeny (Harte and Kankel, 1982).
γ			<i>Gl²²</i> complements or partially complements other lethal alleles of <i>Gl</i> : <i>Gl²²/Gl¹</i> lethal at 29°; eyes extreme Glued, bristles reduced and wings small and outheld at 18°; enhances phenotype of <i>Gl¹¹⁸</i> and <i>Gl¹⁶</i> . * <i>Gl¹²¹</i> also complemented.
δ			<i>Gl¹²⁰/Gl¹³</i> rarely survive as feeble adults.

Approximately one-fourth (22/96) revertants of *Gl* induced by X or γ irradiation fail to complement one or more flanking lethals and are therefore presumed to be deficiencies; those induced by ethyl methanesulfonate and hybrid dysgenesis on the other hand complement all flanking lethals. Phenotypic revertants induced by ionizing radiation and ethyl methanesulfonate remain lethal in heterozygous combination with either *Gl* or *Df(3L)Gl2* and with each other; revertants tested in combination with *Df(3L)Gl2* die during the first larval instar. The majority of hybrid-dysgenesis-induced revertants (67/78) are viable in heterozygous combination with *Df(1)Gl2* and most *Gl¹* alleles, whereas they are lethal in combination with *Gl¹*, *Gl¹⁹*, and *Gl¹¹⁰*; 7/67 are viable in all the above combinations and 4/67 are inviable in all combinations. Available phenotypic observations on *Gl^{rv}/+* summarized in accompanying table.

allele	phenotype of <i>Gl^{rv}/+</i>
<i>Gl^{rv3}</i>	wild type
<i>Gl^{rv9}</i>	wild type
<i>Gl^{rv17}</i>	wild type
<i>Gl^{rv18}</i>	wild type
<i>Gl^{rv22}</i>	wild type
<i>Gl^{rv24}</i>	female sterile
<i>Gl^{rv26}</i>	weak Glued
<i>Gl^{rv32}</i>	very weak Glued
<i>Gl^{rv33}</i>	weak Glued
<i>Gl^{rv34}</i>	wild type
<i>Gl^{rv50}</i>	weak Glued
<i>Gl^{rv70}</i>	weak Glued
<i>Gl^{rv72}</i>	wild type
<i>Gl^{rv73}</i>	wild type
<i>Gl^{rv80}</i>	wild type
<i>Gl^{rv86}</i>	wild type
<i>Gl^{rv296}</i>	wild type

cytology: Placed in 70C2 on basis of *in situ* hybridization of cloned sequence (Garen *et al.*, 1984).

molecular biology: The Glued locus was cloned by P-element insertion in 70C2 as a chromosome marker (Swaroop *et al.*, 1986). The dominant *Gl* allele differs from *Gl⁺* in the presence of the transposon *roo* near the 3' end of the transcribed region, resulting in the formation of a truncated 5.1 kb transcript instead of the 6 kb transcript of *Gl⁺* (Swaroop *et al.*, 1985). The normal transcript has been found in about all of the tissues of *Gl⁺* homozygotes. The complete cDNA sequence of *Gl⁺* contains an open reading frame encoding 1319 amino acids (Swaroop *et al.*, 1987). There are five exons, two of them in the 5' untranslated region. One of the introns interrupting the Glued ORF encodes at least two polyadenylated transcripts. The Glued polypeptide

predicted from the sequence encoded by the ORF has extensive α -helical internal domains (Swaroop *et al.*, 1987). Similar in sequence and structure to those of filamentous proteins of other species.

gl-l: see *gl*^{41e}

Gla: Glazed

location: 2- (rearrangement).

references: Morgan, Bridges, and Schultz, 1936, Year Book - Carnegie Inst. Washington 35: 293.

phenotype: Eye reduced to one-fourth normal area and narrowed to a point ventrally. Eye color generally diluted but with some black patches. Ommatidia coalesce into gleaming, smooth sheet. Malpighian tubes of larva somewhat lighter than wild type; difficult to classify (Brehme and Demerec, 1942, Growth 6: 351-56). Abdomen of heterozygous female fails to distend with eggs; fertility impaired (Craymer, 1980, DIS 55: 200). Homozygous lethal. RK2A.

cytology: Associated with *In(2LR)Gla = In(2LR)27D;51E*, superimposed on *In(2L)t = In(2L)22D3-E1;34A8-9*.

glass: see *gl*

glass-like: see *gl*^{41e}

glassy mouth part: see *gim*

Glazed: see *Gla*

Gld: Glucose dehydrogenase

location: 3-48 (based on 30 *st-cu* recombinants).

synonym: *Go*: Glucose oxidase.

references: Cavener, 1980, Biochem. Genet. 18: 927-37. Cavener and MacIntyre, 1983, Proc. Nat. Acad. Sci. USA 80: 6268-88.

Whetten, Organ, Krasney, Cox-Foster, and Cavener, 1988, Genetics 120: 475-84.

phenotype: The structural gene for FAD glucose dehydrogenase [GLD (EC1.1.99.10)]. The formation of a hybrid band in *Gld^F/Gld^S* heterozygotes indicates a dimeric enzyme. Molecular weight given as 110,000 daltons. Gene expressed during pupal stages of both sexes; enzyme found in pupal stage 3-4 moulting fluid where it modifies puparium cuticle. *Gld* also expressed in the ejaculatory duct of males; genital disc transplantation experiments indicate that GLD expression in ejaculatory duct is autonomous (Cavener); enzyme transferred from males to females during copulation. Lethality of null alleles in the pupal stage observed; *Gldⁿ* flies can be rescued by pre-eclosion excision of pupal operculum.

alleles: Two naturally occurring electrophoretic alleles, *Gld^F* and *Gld^S* and five EMS-induced null alleles *Gldⁿ¹* - *Gldⁿ⁵*.

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>Gldⁿ¹</i>	EMS	Grigliatti	<i>l(3)4.11</i>	1	viable γ
<i>Gldⁿ²</i>	X ray	Denell	<i>l(3)d5</i>	1-3	<i>T(2;3)84C8-D1</i> ; homozygous lethal
<i>Gldⁿ³</i>	EMS	Cavener	<i>l(3)g9</i>	1	lethal
<i>Gldⁿ⁴</i>	EMS	Cavener	<i>l(3)g10</i>	1,3	lethal
<i>Gldⁿ⁵</i>	EMS	Cavener	<i>l(3)g11</i>	1	lethal
<i>Gld^F</i>	spont	Cavener	<i>Go^F</i>	2	
<i>Gld^S</i>	spont	Cavener	<i>Go^S</i>	2	

^α 1 = Cavener and MacIntyre, 1983, Proc. Nat. Acad. Sci. USA 80: 6268-88; 2 = Cavener, Otteson and Kaufman, 1986, Genetics 114: 111-13; 3 = Lewis, Kaufman, Denell, and Tallierico, 1980,

Genetics 95: 367-81.

^β Measured in *Gldⁿ/Df(3R)Antp17*.

^γ 1% wild-type enzyme activity in pupae and adult males. *Gld²* through *Gld⁵* lethal in all pairwise combinations except that *Gld²* partially complements *Gld⁴* and *Gld⁵*.

cytology: Placed in 84D on the basis of its being in the region of overlap between *Df(3R)A41 = Df(3R)84B1-2;84D1-2* and *Df(3R)dsx29 = Df(3R)84D2-3;84F8-10* (Cavener, Otteson and Kaufman, 1986, Genetics 114: 111-23).

molecular biology: Structural gene cloned from EMBL-4 Oregon-R genomic library (Cavener, Corbett, Cox, and Whetten, 1986, EMBO J. 5: 2939-48). Clones identified by locating deficiency and translocation breakpoints delimiting *Gld*. A 2.8 kb poly(A) RNA transcript identified with temporal and spatial expression similar to that of *Gld*. Expression of *Gld* induced immediately before larval and pupal moults. The three 5' exons of the gene have been sequenced and the start sites for transcription and translation identified (Whetten *et al.*, 1988). The first exon contains 335 nucleotides; the second exon contains the *Gld* translation start codon. The putative amino acid sequence at the amino terminus has three serine-alanine tandem repeats and numerous cysteine residues. The *Gld* lethal phenotype was rescued by *P*-element-mediated germline transformation (Whetten *et al.*, 1988).

gleam: see *gm*

***gli: glide**

location: 1-38.0.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).

discoverer: Fahmy, 1954.

references: 1958, DIS 32: 70.

phenotype: Wings held horizontally at right angles to body. Pigmentation of tergites frequently interrupted along mid-dorsal line; tergites occasionally show a nick in the posterior border. Males sterile; viability about 70% wild type. RK2.

glisten: see *gn*

glossy: see *lz*

glossy like: see *sno*

Glt: Glutactin

location: 2-{30}.

references: Fessler and Fessler, 1989, Annu. Rev. Cell Biol. 5: 309-39.

phenotype: Encodes an acidic sulfated glycoprotein of basement membranes.

cytology: Placed in 29D by *in situ* hybridization.

molecular biology: Gene sequenced and found to code for a 1024-residue polypeptide that has a signal peptide, a major amino domain of 600 residues with the coding region interrupted by one intron, and an acidic carboxyl domain. Amino and carboxyl domains separated by 13 threonine residues; four O-sulfated tyrosines present. The protein preferentially binds Ca⁺⁺ in the presence of excess Mg⁺⁺.

Glu: β -Glucuronidase

location: 3-{101}.

references: Langley, S.D., Wilson, Gross, and Finnerty, 1983, J. Biol. Chem. 258: 7416-24.

phenotype: May or may not be a structural gene for β -D-Glucuronidase (EC 3.2.1.31). The enzyme is a glycoprotein and exists in two chromatographically separable forms. Form I is membrane bound, has a pI of 8.0 - 8.5, and can be irreversibly inactivated by either incubation at 55° for 20 min or by incubation at 37° in the presence of 6M urea; form II exists in both membrane-bound and free states, has a pI of 4.5, and is resistant to inactivation by the above treatments. The two are kinetically similar, having similar K_m and V_{max} and are precipitated by antibody raised against form II.

alleles: Glu^H has high activity owing to the presence of both forms I and II; Glu^L has the same level of form II activity but lacks form I and exhibits low activity. Heteroallelic genotypes exhibit intermediate levels of form I activity. Observations suggests that Glu^H may be a duplication or Glu^L is an amorphic mutant in a duplicate gene.

cytology: Placed in 98F-100F by gene dosage studies.

Glucose dehydrogenase: see *Gld*

Glucose oxidase: see *Gld*

β -**Glucuronidase:** see *Glu*

Glue proteins: see *Sgs*

Glued: see *Gl*

Glutactin: see *Glt*

Glutamate dehydrogenase: see *Gdh*

Glutamate oxaloacetic transaminase: see *Got*

Glutamine pyruvate transaminase: see *Gpt*

Glutamine synthetase: see *Gs*

Glyceraldehyde dehydrogenase: see *Gapdh*

α -**Glycerol phosphate dehydrogenase:** see *Gpdh*

α -**Glycerophosphate oxidase:** see *Gpo*

Glycinamide ribotide transformylase:
see *ade3*

gly: see *lz*⁸

gm: gleam

location: 3- (not located).

origin: Spontaneous.

discoverer: Bridges, 27c1.

phenotype: Eyes small and rough; irregular hairs and facets cause glints. Body small. Viability about 10% wild type but variable. RK3.

cytology: Associated with *In(3L)P*, according to Bridges (Morgan, Bridges, and Schultz, 1937, Year Book - Carnegie Inst. Washington 36: 301).

gmp: see *fliF*

***gn: glisten**

location: 3-67.3.

origin: γ ray induced.

discoverer: Wallbrun, 61i6.

references: Eyes rough but of normal size; facets and hairs irregular. RK2.

gnd: grounded (J.C. Hall)

location: 1-58 (proximal to *f*).

origin: Induced by ethyl methanesulfonate.

synonym: Originally called 694, 623, 200 (Deak *et al.*, 1980).

references: Deak, Rahmi, Bellamy, Bienz, Blumer, Fenner, Golin, Ramp, Reinhardt, Dubendorfer, and Cotton, 1980, Development and Neurobiology of *Drosophila* (Siddiqi, Babu, Hall, and Hall, ed.). Plenum Press, New York, pp. 183-92.

Deak, Bellamy, Bienz, Dubuis, Fenner, Gollin, Rahmi, Ramp, Reinhardt, and Cotton, 1982, J. Embryol. Exp. Morphol. 69: 61-81.

Homyk and Emerson, 1988, Genetics 119: 105-21.

phenotype: Flight is defective; indirect flight muscles have relatively mild abnormalities of Z-bands and myofibrils; mosaic analysis suggests that thoracic nerves or muscles could be primary site of mutant defect. All alleles lead to reduced amount of at least two high molecular-weight proteins from indirect flight muscles.

alleles: Three mutant alleles: *gnd*¹, *gnd*², *gnd*³.

other information: Could be allelic to *fltM*, *fltN*, *fltO*, *flrdR*, or *flrdQ*.

gnu: giant nuclei

location: 3-42.5.

origin: Induced by ethyl methanesulfonate.

references: Freeman, Nüsslein-Volhard, and Glover, 1986, Cell 46: 457-68.

phenotype: Maternal-effect lethal. Embryos produced by homozygous or hemizygous females exhibit normal levels of DNA synthesis, but do not display normal nuclear division; a small number of giant nuclei formed. Cytoplasmic elements such as centrosomes, microtubules, and actin appear to attempt to follow the normal developmental program, demonstrating that control of nuclear and cytoplasmic components of syncytial nuclear divisions can be uncoupled.

cytology: Placed in 70D2 or 70E4-8 on the basis of its inclusion in *Df(3L)jz-m21 = Df(3L)70D2-3;70E4-5* but not *Df(3L)jz-d21 = Df(3L)70D3;70E3-8*.

Go: see *Gld*

Go: Gold tip

location: 2-64.3 (57.5 to 71.1; between *cn* and *cg*).

origin: Spontaneous.

discoverer: Sturtevant, 1948.

references: 1948, DIS 22: 55.

phenotype: Tips of many bristles and hairs pale and curved. Bristles often short (tips broken off?). Wild-type bristles sometimes have pale tips, thus interfering with positive classification. Lethal when homozygous. Expression best at low temperatures. RK2.

goggle: see *gg*

Gold tip: see *Go*

gonadal: see *gdl*

gooseberry: see *gsb*

Got1: Glutamate oxaloacetic transaminase

location: 2-75 (between *L* and *nw*^D).

synonym: Designated *Got2* by Band (1975) and also by Cavener and Clegg (1976).

references: Band, 1975, Genetics 80: 761-71.

Cavener and Clegg, 1976, J. Hered. 67: 313-14.

Grell, 1976, Genetics 83: 753-64.

Chase and Kankel, 1987, J. Neurobiol. 18: 15-41.

phenotype: The structural gene for the more rapidly anodally migrating of two glutamate oxaloacetic transaminases [GOT1 (EC 2.6.1.1) = L-aspartate: 2-oxoglutarate aminotransferase] found in *Drosophila*.

alleles: Two alleles described by Grell, *Got1^M*, monomorphic in all stocks examined and *Got1^{lo}* an ethyl methanesulfonate-induced allele with low activity; *Got1^{lo}* homozygotes normal in viability and fertility.

cytology: Located at 52D9-15.

Got2

location: 2-4.8 [based on 70 *al-cl* recombinants (Cavener and Clegg)].

references: Band, 1975, Genetics 80: 761-71.

Cavener and Clegg, 1976, J. Hered. 67: 313-14.

Grell, 1976, Genetics 83: 753-64.

Chase and Kankel, 1987, J. Neurobiol. 18: 15-41.

phenotype: The structural gene for the more slowly anodally migrating of the two glutamate oxalacetic transaminases. Enzyme is a dimer based on formation of a hybrid molecule.

alleles: The most common allele is designated *Got1* by Band, *Got2⁴* by Voelker and Langley (1978, DIS 53: 117), and *Got2^M* by Grell; we establish *Got2¹* as the designation.

allele	synonym	ref ^α	anodal migration
Got2¹	<i>Got1¹</i>	1, 2	< <i>Got2²</i>
	<i>Got2⁴</i>	6	
	<i>Got2^M</i>	3	
Got2²	<i>Got1²</i>	1, 2	> <i>Got2¹</i>
	<i>Got2⁶</i>	6	
Got2⁷		6	> <i>Got2²</i>
Got2^{Jβ}		3	> <i>Got2¹</i>
Got2ⁿ⁴	<i>Got2^{γ40058}</i>	5	
Got2^{nJ}	<i>Got2^{nJH385}</i>		= <i>Got2¹</i> ?
Got2^{nNC}	<i>Got2^{nNC1}</i>	4	= <i>Got2¹</i>
	<i>Got2^{nNC2}</i>	4	= <i>Got2¹</i>
	<i>Got2^{nNC3}</i>	4	

^α 1 = Band, 1975, Genetics 80: 761-71; 2 = Cavener and Clegg, 1976, J. Hered. 67: 313-14; 3 = Grell, 1976, Genetics 83: 753-64; 4 = Langley, Voelker, Leigh Brown, Ohnishi, Dickson and Montgomery, 1981, Genetics 99: 151-56; 5 = Racine, Langley, and Voelker, 1980, Environ. Mutagen. 2: 167-77; 6 = Research Triangle Park, 1978, DIS 53: 117.

^β Induced by EMS in *Got2¹*.

cytology: Placed in 22A7-B7 on the basis of its being distal to the 2L breakpoint of *T(Y;2)H56 = T(Y;2)22B5-8* and its inclusion in *Df(2L)S2 = Df(2L)21D1;22A6-B1*.

gouty legs: see *gy*

gp: gap

location: 2-74.

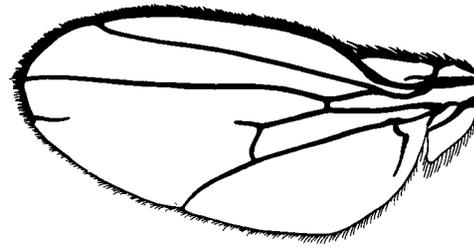
origin: Spontaneous.

discoverer: Bridges, 12a10.

references: Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 208 (fig.).

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 212 (fig.), 226.

phenotype: Vein L4 weak or has section missing beyond posterior crossvein. Overlaps wild type when homozygous; semidominant as heterozygote. RK3.



gp: gap

From Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 209.

Gpdh: α-Glycerol phosphate dehydrogenase

location: 2-17.8 [located with respect to *cl* (16.5) and *Sp* (22.0) (Grell)]; 2-20.5 [based on 422 recombinants between *dp* (13.0) and *b* (48.5)(O'Brien and MacIntyre, 1972)].

synonym: *Gdh* (Grell, 1967); *αGpdh-1* (O'Brien and MacIntyre, 1968); *Gpd* (Research Triangle Park Group, 1978, DIS 53: 117).

references: Grell, 1967, Science 158: 1319-20.

O'Brien and MacIntyre, 1968, DIS 43: 60.

Niesel, Pan, Bewley, Armstrong, and Li, 1982, J. Biol. Chem. 257: 979-83.

Skuse and Sullivan, 1985, EMBO J. 4: 2275-80.

Wright, Shaffer and Bewley, 1985, J. Biol. Chem. 260: 5863-66.

Cook, Shaffer, MacIntyre, Wright and Bewley, 1985, Genetics 110: s13.

Cook, Shaffer, Bewley, MacIntyre and Wright, 1986, J. Biol. Chem. 261: 11751-55.

Cook, Bewley and Shaffer, 1988, J. Biol. Chem. 263: 10858-64.

Kalm, Weaver, DeMarco, MacIntyre, and Sullivan, 1989, Proc. Nat. Acad. Sci. USA 86: 5020-24.

phenotype: The structural gene for α glycerol-3-phosphate dehydrogenase (NAD⁺) [α GPDH (EC 1.1.1.8)], a homodimer with subunit molecular weight 31700 (Collier, Sullivan, and MacIntyre, 1976, Biochim. Biophys. Acta 429: 316-23). Purification and structural analysis of enzyme by Niesel, Bewley, Miller, Armstrong, and Lee (1980, J. Biol. Chem. 255: 4073-80). Three isozymes designated GPDH-1, GPDH-2, and GPDH-3 in order of decreasing rate of migration toward the anode (Wright and Shaw, 1968, Biochem. Genet. 3: 343-53). The three forms respond alike to electrophoretic alleles, null alleles, and dosage of *Gpdh*⁺ (Bewley, Rawls, and Lucchesi, 1974, J. Insect. Physiol. 20: 153-75); all three are products of the same structural gene for developmental profile (see Bewley, 1981, Dev. Genet. 113-29). GPDH-1 first appears in late pupae and is present in high concentration in the adult thorax where it functions to provide energy for flight muscles; GPDH-2 also appears in late pupae and is present in low concentration throughout the fly; GPDH-3 is present throughout the life cycle; it is concentrated in the larval fat body (Rechsteiner, 1970, J. Insect. Physiol. 16: 1179-92) and the adult abdomen (Wright and Shaw); GPDH-1 and GPDH-3 present in equal amounts in adult head. Product of paternally inherited allele first appears at 22 hr, just

before hatching of larva (Wright and Shaw). GPDH-1 is stable at 50° but decays at 57°; GPDH-3 is labile at 50° (Bewley *et al.*). Studies by Bewley and Luccesi indicate the presence of a heat-labile RNase-resistant factor in crude larval extracts able to convert GPDH-1 into GPDH-2 and GPDH-3 but not *vice versa*; GPDH-3 lacks three C-terminal amino acids present on GPDH-1 (Niesel, Bewley, Miller, Armstrong, and Lee, 1980, J. Biol. Chem. 255: 4073-80). Homozygotes for null alleles are fertile, show reduced viability, and are unable to sustain flight (O'Brien and MacIntyre, 1972, Genetics 71: 127-38); ultrastructural integrity of flight muscle sarcomeres degenerates prematurely [O'Brien and Shimada, 1974, J. Cell Biol. 63: 864-82 (fig.)]. Homozygous stocks maintained for 25 generations regain the ability to fly despite continued absence of GPDH activity (O'Brien and Shimada).

alleles: Natural populations are polymorphic for electrophoretic variants as well as for regulatory elements that determine enzyme level; induced electromorphs have also been recovered. These variants are tabulated first.

allele	synonym	origin	mobility
Gpdh^A	<i>Gpdh²</i> , <i>Gpdh^F</i> , <i>Gpdh^R</i>		Fast
Gpdh^B	<i>Gpdh^A</i> , <i>Gpdh^S</i>		Slow
Gpdh^C	α <i>Gpdh-1^{CC}</i>	EMS(<i>Gpdh^B</i>)	Slower
Gpdh^D			
Gpdh^H α			
Gpdh^L α			
Gpdh^P		EMS(<i>Gpdh^B</i>)	
Gpdh^{UF}		spont	Faster

α *Gpdh^H* and *Gpdh^L* determine high versus low rates of accumulation of GPDH-3 in both larvae and adults (Bewley, Dev. Genet. 2: 113-192). Bewley attributes this polymorphism to a separate regulatory gene, *Gdt-3*, but we prefer to designate the alternatives as alleles of *Gpdh* based on Bewley's finding that their effect is *cis* acting and his inability to separate them from *Gpdh* genetically. *Gpdh^H* has been found in association with both *Gpdh^A* (*Gpdh^{HA}*) and *Gpdh^B* (*Gpdh^{HB}*), whereas *Gpdh^L* has been found only with *Gpdh^A* (*Gpdh^{LA}*). The physical properties of the enzymes produced under the control of these two regulatory elements are identical (Shaffer and Bewley, 1983, J. Biol. Chem. 258: 10027-33). Control postulated at the level of transcription (Wilkins, Shaffer, and Bewley, 1982, Dev. Genet. 3: 129-42).

cytology: Placed in 25F5 by deficiency analysis (Kotarski, Pickert and MacIntyre, 1983, Genetics 105: 371-86) and

in 26A (distal) by *in situ* hybridization to the salivaries (Cook *et al.*, 1986).

molecular biology: Genomic and cDNA clones of the gene have been isolated (Cook *et al.*, 1985, 1986, 1988), and the genomic and predicted amino acid sequences determined (Cook *et al.*, 1988; Kalm *et al.*, 1989). The transcript of *Gpdh⁺* is about 4.9 kb in length and is composed of eight exons interrupted by seven introns. There is a tandem duplication of a portion of the coding region. The duplication is truncated at the 5' end and is polymorphic in natural populations [Koga, Kusakabe, Tajima, Harada, Bewley and Mukai, 1988, Proc. Japan Acad. 64 (B): 9-12]. The DNA sequence data predict three transcripts, each differing in the 3' untranslated region (Cook *et al.*, 1988). Poly(A) site selection may occur in exon 6, exon 7, or exon 8, producing mRNAs of different size ranges from alternate splicing pathways and producing three different isozymes (GPDH-1, GPDH-2, GPDH-3) differing in the amino acid sequences at their COOH-terminal ends although encoded by the same structural gene (Skuse and Sullivan, 1985; Cook *et al.*, 1988). Each transcript and each isozyme is tissue-specific in its expression. The amino acid sequence at the COOH terminal end of GPDH-3 is Asn-His-Glu-His-Met-COOH; the sequence of GPDH-1 is extended by the three amino acid sequence Glu-Asn-Leu-COOH. *Xenopus laevis* oocytes injected with poly(A)+ RNA from *Drosophila melanogaster* direct synthesis of two immunologically-related proteins, a 32-kd protein from *Gpdh⁺* and a 34-kd protein from a CRM-null *Gpdh* mutant (Wright *et al.*, 1985).

other information: A series of *Gpdh-Gpo* double mutants were constructed by Davis and MacIntyre (1988, Genetics 120: 755-66); four of these mutants were found to be viable and flightless; two others were allele-dependent synthetic lethals. A *trans* acting regulatory element tightly linked to the *Gpdh* locus has been isolated in a natural population of *Drosophila melanogaster* in Tasmania (Gibson, Wilks, Cao and Freeth, 1986, Experientia 42: 191-92). Flies homozygous for second chromosomes carrying the element (designated *H31*) have half the GPDH activity of normal homozygotes.

There are a number of spontaneous and induced amorphic and hypomorphic alleles. These are tabulated also.

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	synonym	origin		ref α	enzymatic phenotype			Physiological Phenotype	
		allele	treatment		%CRM β	%wildtype activity γ	mobility δ	relative viability	flight ability
Gpdh n0	Gpdh B0-0	<i>Gpdh B</i>	EMS	1, 3, 4	16, 31	0	> <i>Gpdh B</i>	.20	-
Gpdh n1-4	Gpdh B0-1-4	<i>Gpdh B</i>	EMS	1, 3, 4	>5	1	> <i>Gpdh B</i>	.20	-
Gpdh n1-5	Gpdh B0-1-5	<i>Gpdh B</i>	EMS	3		26.1	< <i>Gpdh B</i>	1.1	+
Gpdh n5-4	Gpdh B0-5-4	<i>Gpdh B</i>	EMS	1, 3, 4	0	0	+ <i>Gpdh B</i>	.28	-
Gpdh n452A									
Gpdh nGB1			spont	2		+	= <i>Gpdh B</i>		
Gpdh nBG2			spont	2		+	= <i>Gpdh A</i>		
Gpdh nGL									
Gpdh nMC1	Gpdh AW338	<i>SM1, Gpdh A</i>	spont	1, 3, 5	0	0, 1.3	= <i>Gpdh A</i>		
Gpdh nMC2	Gpdh AW409	<i>Gpdh B</i>	spont	1, 5	0	0	+		
Gpdh nMC3	Gpdh JH149	<i>SM1, Gpdh A</i>	spont	5			+		
Gpdh nMC4	Gpdh JH151	<i>SM1, Gpdh A</i>	spont	1, 5	8	9	\pm		
Gpdh nMC6	Gpdh JH253 ϵ	<i>SM1, Gpdh A</i>	spont	2	9	10	+		
Gpdh nMC1	Gpdh nNC504		spont	2, 4	30	+	> <i>Gpdh A</i>		
Gpdh nNC2	Gpdh nNC718		spont	2		+			
Gpdh nNC3	Gpdh nNC738		spont	2, 4	64	-	= <i>Gpdh A</i>		
Gpdh nNC4	Gpdh nNC967		spont	2, 4	32	+	= <i>Gpdh A</i>		
Gpdh nNC5	Gpdh nNC1009		spont	2, 3, 4	0	6.0	= <i>Gpdh A</i>		
Gpdh nNC5	Gpdh nNC1023		spont	2, 3, 4	42	6.0	= <i>Gpdh A</i>		
Gpdh nNC7	Gpdh nNC1405		spont	2, 3, 4	22	7.5	= <i>Gpdh A</i>		
Gpdh nR1	Gpdh n γ 20066	<i>Gpdh A</i>	γ ray	6					
Gpdh nR2	Gpdh n γ 50066	<i>SM1, Gpdh A</i>	γ ray 1, 6	0.8	10	-			
Gpdh nR3	Gpdh n γ 50075	<i>Gpdh A</i>	γ ray	6					
Gpdh nR4	Gpdh n γ 50078	<i>Gpdh A</i>	γ ray	6	0				
Gpdh nR5	Gpdh n γ 70018	<i>SM1, Gpdh A</i>	γ ray	1, 4, 6	14	0	= <i>Gpdh A</i>		
Gpdh nGL1		<i>Gpdh B</i>	EMS	3	++	4.4	< <i>Gpdh B</i>	0.70	+
Gpdh nGL2		<i>Gpdh B</i>	EMS	3	++	9.5	= <i>Gpdh B</i>	1.1	+
Gpdh nGL3		<i>Gpdh B</i>	EMS	3	++	22.2	< <i>Gpdh B</i>	1.3	+
Gpdh nRZ1		<i>Gpdh B</i>	EMS	3	+	0.0	-	0.20	-
Gpdh nS1		<i>Gpdh B</i>	EMS	3	++	25.4	= <i>Gpdh B</i>	0.78	+
Gpdh nS2		<i>Gpdh B</i>	EMS	3	+	0.0	-	0.33	-
Gpdh nS3		<i>Gpdh B</i>	EMS	3	++	0.0	= <i>Gpdh B</i>	0.85	+
Gpdh nS4		<i>Gpdh B</i>	EMS	3	++	78.5	= <i>Gpdh B</i>	1.0	+
Gpdh nS5		<i>Gpdh B</i>	EMS	3	++	56.6	= <i>Gpdh B</i>	0.88	+
Gpdh nS6		<i>Gpdh B</i>	EMS	3	++	4.6	< <i>Gpdh B</i>	0.93	+
Gpdh nS7		<i>Gpdh B</i>	EMS	3	++	52.4	> <i>Gpdh B</i>	1.30	+
Gpdh nS8		<i>Gpdh B</i>	EMS	3	++	0.0	= <i>Gpdh B</i>	0.81	+
Gpdh nS10		<i>Gpdh B</i>	EMS	3	++	74.8	< <i>Gpdh B</i>	0.82	+
Gpdh nSP1		<i>Gpdh B</i>	EMS	3	++	6.5	< <i>Gpdh B</i>	0.82	+
Gpdh nSP2		<i>Gpdh B</i>	EMS	3	+	4.0	< <i>Gpdh B</i>	0.99	+
Gpdh nSP3		<i>Gpdh B</i>	EMS	3	+	0.0	< <i>Gpdh B</i>	0.23	-
Gpdh nSP4		<i>Gpdh B</i>	EMS	3	++	0.0	-	0.06	+
Gpdh nSP5		<i>Gpdh B</i>	EMS	3	++	71.4	< <i>Gpdh B</i>	0.68	+
Gpdh nSP6		<i>Gpdh B</i>	EMS	3	++	8.3	= <i>Gpdh B</i>	0.35	+
Gpdh nSP7		<i>Gpdh B</i>	EMS	3	++	28.0	< <i>Gpdh B</i>	1.00	+
Gpdh nSP8		<i>Gpdh B</i>	EMS	3	++	9.2	< <i>Gpdh B</i>	0.95	+
Gpdh nSP10		<i>Gpdh B</i>	EMS	3	++	9.2	< <i>Gpdh B</i>	0.95	+
Gpdh nSP11		<i>Gpdh B</i>	EMS	3	++	30.4	= <i>Gpdh B</i>	0.98	+
Gpdh nSP12		<i>Gpdh B</i>	EMS	3	-	0.0	-	0.42	-
Gpdh nSP14		<i>Gpdh B</i>	EMS	3	+	0.0	-	0.27	-
Gpdh nSP15		<i>Gpdh B</i>	EMS	3	+	11.1	= <i>Gpdh B</i>	0.54	\pm
Gpdh nSP16		<i>Gpdh B</i>	EMS	3	++	0.0	< <i>Gpdh B</i>	0.55	\pm
Gpdh nSP17		<i>Gpdh B</i>	EMS	3	++	1.8	= <i>Gpdh B</i>	0.74	+
Gpdh nSP18		<i>Gpdh B</i>	EMS	3	++	74.5	= <i>Gpdh A</i>	0.99	+
Gpdh nSP19		<i>Gpdh B</i>	EMS	3	++	18.8	> <i>Gpdh B</i>	0.76	+

α 1 = Bewley, DeZurille, and Pagelson, 1980, Mol. Gen. Genet. 178: 301-308; 2 = Burkhart, Montgomery, Langley, and Voelker, 1984, Genetics 107: 295-306; 3 = Kotarski, Pickert, Leonard, LaRosa, and MacIntyre, 1983, Genetics 105: 387-407; 4 = Lee, Niesel, and Bewley, 1980, Biochem. Genet. 18: 1003-18; 5 = Mukai and Cockerham, 1977, Proc. Nat. Acad. Sci. USA 74: 2514-17; 6 = Racine, Langley, and Voelker, 1980, Environ. Mutagen. 2: 167-77.

β Cross reacting material measured by a) rocket immunoelectrophoresis (numerical values), b) immunoprecipitation (++) or c) radioimmune assay (+).

γ Activity measured in some studies (numerical values); merely noted in others (+ or -).

δ Mobility measurable directly in mutants with residual enzyme activity and indirectly in nulls that form an active heterodimer. + = heterodimer formed but mobility not noted. - = no active heterodimer.

ϵ *Gpdh JH253* and *Gpdh JH254* probably independent recoveries of same mutant.

Gpo: α -Glycerophosphate oxidase

location: 2-75.5.

references: O'Brien and MacIntyre, 1972, Biochem Genet. 7: 141-61.

O'Brien and Gethmann, 1973, Genetics 75: 155-67.

Davis and MacIntyre, 1988, Genetics 120: 755-66.

phenotype: The structural gene for sn-glycerol-3-

phosphate oxidoreductase [α GPO (EC 1.1.99.5)], a 100,000 dalton protein localized on the inner mitochondrial membrane.

alleles: *Gpo* mutants were induced by ethyl methanesulfonate; a hypomorphic allele *Gpo n318* and an almost null mutant *Gpo n322* were recovered (Davis and MacIntyre, 1988), both viable but flightless.

cytology: Localized to 52C9-D3 by deficiency analysis (Davis and MacIntyre) since included in *Df(2R)WVG = Df(2R)52A4-B1;52D7-E1* but in neither *Df(2R)XTE18 = Df(2R)51E3-4;52C9-D1* nor *Df(2R)KL9 = Df(2R)52D3;-52D7-9*.

other information: A series of *Gpdh-Gpo* mutants were constructed (Davis and MacIntyre, 1988); four of these mutants were viable but flightless; two others were allele-dependent synthetic lethals.

Gpt: Glutamate pyruvate transaminase

location: 1-42.6.

references: Leigh Brown and Voelker, 1980, *Biochem. Genet.* 18: 303-09.

phenotype: Structural gene for glutamate-pyruvate transaminase [GPT (EC 2.6.1.2)]. Molecular weight is 87,000 daltons, and similar to the enzyme from other sources, it is most likely dimeric in structure (Leigh Brown, 1980, *DIS* 55: 82-84). Homozygotes exhibit three bands of activity on starch gels, which could represent different degrees of binding of a pyridoxal phosphate ligand to the enzyme.

alleles: Three alleles designated *Gtp*², *Gtp*⁴, and *Gtp*⁶ in order of increasing electrophoretic mobility in starch gel.

cytology: Placed in region 11F1 through 12A2 based on its inclusion in *Df(1)C246 = Df(1)11D;12A1-2* but not *Df(1)N12 = Df(1)11D1-2;11F1-2*.

***gr: gracile**

location: 1-36.4.

origin: Induced by L-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1953.

references: 1959, *DIS* 37: 86.

phenotype: Small fly with narrow abdomen. Wings frequently held atypically, either upward or downward. Very inviable, many dying less than 24 hr after eclosion; males sterile. RK3.

gr: see *grt*

gra: see *grn*

gra: gravel

location: 1-28.5.

origin: Spontaneous.

references: Thompson, 1973, *DIS* 50: 59 (fig.).

phenotype: Eye texture rough owing to irregularly arranged and roughened facets. Suppresses *ve*, making veins L2, L3, and usually L4 complete; in *ve*⁺ flies produces extra vein segments in the marginal, distal, and second and third posterior cells.

gracile: see *gr*

grain: see *grn*

grandchildless: see *gs*

grandchildless on chromosome 2 of Mariol:
see *gs(2)M*

gravel: see *gra*

***gre: green body color**

location: 1- (not located).

origin: Spontaneous.

discoverer: Bridges, 13e.

references: Morgan and Bridges, 1916, Carnegie Inst.

Washington Publ. No. 237: 73.

phenotype: Body color tinged greenish black with marked trident pattern. Overlaps wild type. RK3.

other information: Possibly an allele of *ptg*.

grg: giant ring gland

location: 1-17.0.

origin: Induced by triaziquone.

synonym: *l(1)grg*.

references: Klose, 1980, *Wilhelm Roux's Arch. Dev. Biol.* 189: 57-67.

phenotype: Recessive lethal; third larval instar protracted. Ring gland enlarged, and neurosecretory cells of brain abnormal. Ecdysteroid levels low; not correctable by exogenous hormone.

grk: gurken

location: 2-30.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach, 1987, *Cell* 49: 699-707.

phenotype: Maternal-effect lethal. Wild-type allele required for normal dorsoventral pattern in the egg. In mutants, the ventral regions of the chorion and the embryo are expanded at the expense of the dorsal regions. The pattern of the chorion is altered, a second micropyle and a small patch of operculum-like material often forming at the posterior pole in extremely mutant eggs; fewer cells contribute to the dorsal appendage, which is usually shifted posteriorly, but more follicle cells contribute to the main body of the chorion. In the embryo the major increase in cell mass occurs in the mesoderm as an invagination on the ventral side during early gastrulation. Analysis of mosaic females in which germ cells and sister nurse cells are of different genotype indicate that *grk* mutations act only in the germ line.

alleles:

allele	synonym	phenotype of eggs and embryos
<i>grk</i> ¹	<i>grk</i> ^{DC}	intermediate and strong
<i>grk</i> ²	<i>grk</i> ^{HG}	weak and intermediate
<i>grk</i> ³	<i>grk</i> ^{HK}	intermediate and strong
<i>grk</i> ⁴	<i>grk</i> ^{HL}	intermediate and strong
<i>grk</i> ⁵	<i>grk</i> ^{QI}	mostly intermediate, but can vary from weak to strong
<i>grk</i> ⁶	<i>grk</i> ^{WG}	mostly intermediate but can vary from weak to strong

cytology: Placed in 29C.

grn: grain

location: 3-47.

origin: Induced by ethyl methanesulfonate.

synonym: *gra*.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95 (fig.).

phenotype: Homozygous lethal; filzkörper or embryo not elongated; head skeleton defective.

alleles: Two.

gro: see *E(spl)*²

grooved: see *gv*

grooveless: see *gvl*

grotle: see *grt*

groucho: see *E(spl)*²

grounded: see *gnd*

grt: grotle

location: 1-64 [Recombination tests by Roberts and Hewitt place *grt* 15.3 cM to the right of *f*; however, it is to the left of *ot* at 1-65.7, since it is not covered by *y*⁺*Ymal106* which does cover *ot* (Schalet, 1972, DIS 49: 36)].

origin: Spontaneous.

synonym: *gr* (preoccupied).

references: Roberts and Hewitt, 1969, DIS 44: 49.

phenotype: Homozygotes and heterozygotes have wing abnormalities with variable expression. Extreme expression, wings with fluid-filled sac covering 75% of wing area, leaving wings crumpled when sac bursts; weak expression, small blisters and incised margins of posterior cells on wings. Penetrance complete.

cytology: Distal to region 19 based on failure of *y*⁺*Ymal*¹⁰⁶ to cover *grt* (Schalet, 1972, DIS 49: 36; Schalet and Lefevre, 1973, Chromosoma 44: 183-202).

gs: see *gv*

gs: grandchildless

location: 1-21 (less than 1cM to the right of *ct*).

origin: Induced by ethyl methanesulfonate.

references: Thierry-Mieg, Masson, and Gans, 1972, C.R. Hebd. Seances. Acad. Sci. Ser. D 175: 2751-54.

Thierry-Mieg, 1976, J. Microsc. Biol. Cell 25: 1-6.

phenotype: Homozygous females normal in appearance. When oogenesis in such females proceeds at 28.5° 80-85% of the eggs cease development before the blastoderm stage. Most embryos reaching gastrulation continue development normally except for the frequent absence of pole cells; 11-15% of surviving adult progeny have agametic gonads; if the maternal females are also homozygous for an X-linked modifier, *mod*, the incidence of agametic survivors increases to 60-65%. Oogenesis at 10° leads to 62% hatch and 6-10% agametic, which is decreased to <3% in the presence of homozygous *mod*. Outcome not influenced by genotype of father. The temperature-sensitive period for survival of zygotes produced by *mod gs* females is monophasic extending from stages 9 to 14 of oocyte development, whereas that for agametic gonads is diphasic with one sensitive period at stages 6-7 and the other more severe period from stages 10-14; stages 7-9 temperature insensitive. The histology of agametic gonads of both males and females resemble those produced by ultra-violet irradiation of embryonic pole cells (Lauge, Sauphanov, and Randrianandrianina, 1977, C.R. Hebd. Seances Aca. Sci. Ser. D 284: 1187-89).

gs(1)N26

location: 1-33.8.

origin: Induced by ethyl methanesulfonate.

references: Niki and Okada, 1981, Wilhelm Roux's Arch. Dev. Biol. 190: 1-10 (fig.).

Niki, 1984, Dev. Biol. 103: 182-89 (fig.).

phenotype: Fecundity and fertility of homozygous females low; mortality of sons higher than that of daughters. Fraction of surviving progeny agametic depends on temperature of oogenesis; 93% agametic at 25°, 56% agametic at 18°. In eggs produced at 25° migration of nuclei to posterior pole abnormal; almost no pole cells

produced in half the embryos. Polar granules present in posterior egg cytoplasm, defects in failure of nuclear migration.

gs(1)N41

location: 1-39.6.

origin: Induced by ethyl methanesulfonate.

synonym: *gs(1)N441*.

references: Nikki and Okada, 1981, Wilhelm Roux's Arch. Dev. Biol. 190: 1-10 (fig.).

phenotype: Fecundity and fertility of homozygous females normal. 71% and 19% agametic progeny when females raised at 25° and 18°, respectively. Peripheral migration of nuclei at blastoderm formation normal; pole cell formation inhibited. Temperature sensitive period extends from stages 9-13 of oocyte development.

gs(2)M: grandchildless on chromosome 2 of Mariol

location: 2- (between *S* and *Sp*).

origin: Induced by ICR-170.

references: Mariol, 1981, Mol. Gen. Genet. 181: 505-11.

phenotype: At 28.5°, homozygous females lay normal numbers of eggs but about 20% fail to hatch and about 40% die just after hatching; these mostly lack pole cells. A fraction of the surviving embryos are also devoid of pole cells and develop into adults with agametic gonads. Abnormalities most frequent in 8-12-day-old females. Paternal genotype without influence. At 16°, 75% of eggs hatch and develop into normal fertile adults. Last four days of oogenesis at 28.5° temperature insensitive. Incidence of agametic progeny increases with successive generations of homozygosity of mothers from one to four, constant thereafter; one generation of heterozygosity returns level to ground state.

gsb: gooseberry

location: 2-107.6.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard and Wieschaus, 1980, Nature (London) 287: 795-801 (fig.).

Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82 (fig.).

Bopp, Burri, Baumgartner, Frigerio and Noll, 1986, Cell 47: 1033-40.

Baumgartner, Bopp, Burri and Noll, 1987, Genes Dev. 1: 1247-67.

Côte, Preiss, Haller, Schuh, Kienlin, Seifert, and Jäckle, 1987, EMBO J. 6: 2793-801.

Perrimon and Mahowald, 1987, Dev. Biol. 119: 587-600.

phenotype: Homozygous lethal; embryos show segment-polarity defects. The posterior portion of each segment is deleted and the anterior portion duplicated in mirror image fashion; the ventral segments are almost entirely covered with denticles, the posterior fraction of which point anteriorly. Segment boundaries persist normally and segments maintain their individuality. The mutants show alterations in the identity of neurons, both underneath and outside the modified ectoderm (Patel, Schafer, Goodman, and Holmgren, 1989, Genes Dev. 3: 890-904); posterior commissures are almost totally absent.

alleles: No point mutations were obtained in embryonic lethal screens by Nüsslein-Volhard *et al.*, 1984, or Côte *et al.*, 1987.

cytology: Placed in 60E9-F1 (Nüsslein-Volhard *et al.*, 1984) since uncovered by *Df(2R)gsb = Df(2R)60E9-10;60F1-2*.

molecular biology: Wild-type *gsb* function provided by two closely-linked duplicated genes, the proximal *gsb-p* (=BSH4) and the distal *gsb-d* (=BSH9) (Bopp *et al.*, 1986; Baumgartner *et al.*, 1987; Côté *et al.*, 1987). These *gsb* genes were mapped by microdissection and microcloning of bands in the 60E9-60F1 region followed by chromosome walking from the proximal breakpoint of *Df(2R)SBI* to the distal breakpoint of *Df(2R)gsb*. The *gsb-p* transcript is made up of five exons and is transcribed in the opposite direction from the two-exon *gsb-d* transcript. The almost complete DNA sequence and corresponding amino acid sequence of the putative protein has been obtained for the two *gsb* genes (Baumgartner *et al.*, 1987). The longest open reading frame of *gsb-p* would encode a protein of 452 amino acids; the longest ORF of *gsb-d* would encode a protein of 427 amino acids.

Gsl: Glutamine synthetase I

location: 2-{0}.

references: Scalenghe and Ritossa, 1976, Atti Accad. Naz. Lincei. Cl. Sci. Fis. Nat. Rend. 13: 439-538.

Caizzi and Ritossa, 1983, Biochem. Genet. 21: 267-85.

Caggese, Caizzi, Bozetti, Barsanti, and Ritossa, 1988, Biochem. Genet. 26: 571-84.

phenotype: Structural gene for glutamine synthetase I [GS (EC 6.3.1.2)] in *Drosophila melanogaster*. The enzyme catalyzes the formation of glutamate and ammonia by cleaving ATP into ADP and phosphate (Caggese *et al.*, 1988). The GSI protein was purified from *Drosophila* larvae (Scalenghe and Ritossa, 1976; Caggese and Ritossa, 1983); it is a multimer of two subunits (MW 43,000 and 64,000) (Caggese and Ritossa, 1983); the 43,000 dalton protein is usually the more abundant. The molecular weight for the complete enzyme is apparently 380,000. A fast electrophoretic variant (*Gsl^f*), an intermediate and common electrophoretic variant (*Gsl^c*), and a slow electrophoretic variant (*Gsl^s*) have been found. Hybrids between variants show a strongly stained intermediate band (Caggese *et al.*, 1988). The GSI protein but not the GSII protein is found in the epidermis of *Drosophila* (Scalenghe and Ritossa, 1976).

alleles:

allele	origin	synonym	comments
<i>Gsl¹</i>	EMS	<i>l(2)C7</i>	null derivative of <i>Gsl^s</i>
<i>Gsl²</i>	EMS	<i>l(2)C8</i>	hypomorphic derivative of <i>Gsl^s</i>
<i>Gsl³</i>	EMS	<i>l(2)M9</i>	null derivative of <i>Gsl^c</i>
<i>Gsl⁴</i>	ENU	<i>l(2)R26</i>	null derivative of <i>Gsl^c</i>
<i>Gsl⁵</i>	ENU	<i>l(2)R34</i>	null derivative of <i>Gsl^c</i>
<i>Gsl^c</i>	nature		common allele in nature
<i>Gsl^f</i>	nature		rare fast allele
<i>Gsl^s</i>	nature		rare slow allele

cytology: Located in 21B3-6 since between proximal breakpoints of *Df(2L)PMI = Df(2L)21A1;21B3-5* and *Df(2L)PMG = Df(2L)21A1-2;21B4-6*.

molecular biology: *Gsl* and *GsII* show partial DNA identity with each other and with the DNA from the hamster (De Pinto, Caggese, Prezioso, and Ritossa, 1987, Biochem. Genet. 25: 821-36).

other information: Mutations in *Gsl* do not affect the

GSII enzyme (Caggese *et al.*, 1988).

GsII: Glutamine synthetase II

location: 1-{34}.

references: Scalenghe and Ritossa, 1976, Atti Accad. Naz. Lincei. Cl. Sci. Fis. Nat. Rend. 13: 439-538.

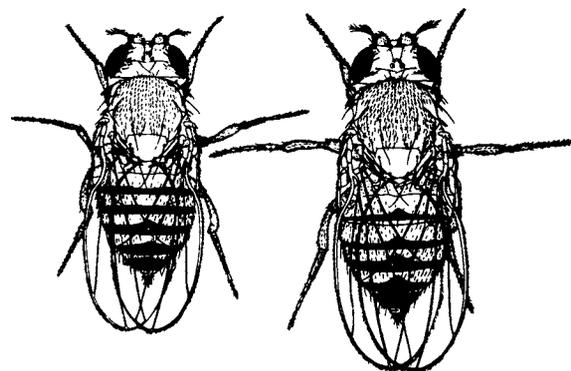
Caggese, Caizzi, Grieco, Bozetti, and Ritossa, 1986, Mol. Gen. Genet. 204: 208-13.

De Pinto, Caggese, Prezioso, and Ritossa, 1987, Biochem. Genet. 25: 821-36.

phenotype: Structural gene for glutamine synthetase II. Completely separable from glutamine synthetase I by DEAE chromatography. The GSII protein was purified from adult *Drosophila* (Scalenghe and Ritossa, 1976) and was isolated by sequence identity with the hamster gene (De Pinto *et al.*, 1987). The enzyme is a multimer of a single subunit (MW 42,000). The complete enzyme has an approximate molecular weight of 360,000 and differs from the GSI enzyme in both subunit molecular weight and in isoelectric point. 90% of glutamine synthetase activity is due to GSII, which is the most abundant adult form.

cytology: Located in 10B8-11 by *in situ* hybridization. Variation in the dose of a chromosomal segment from 9F3 to 10C1-2 results in proportional variation in the amount of the GSII enzyme without influencing the amount of GSI (Caggese *et al.*, 1986).

molecular biology: Shows partial DNA identity with *Gsl* and with the hamster gene (De Pinto *et al.*, 1987).



gt: giant

Left: wild type female. Right: *giant* female.

From Bridges and Gabritschevsky, 1928, Z. Indukt. Abstamm. Vererbungsl. 46: 231-47.

gt: giant

location: 1-0.9 (0.04 cM to the left of *tko*).

origin: Spontaneous.

discoverer: Gabritschevsky, 25i2.

references: Bridges and Gabritschevsky, 1928, Z. Indukt. Abstamm. Vererbungsl. 49: 231-47 (fig.).

Gabritschevsky and Bridges, 1928, Z. Indukt. Abstamm. Vererbungsl. 49: 248-84.

Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307.

Narachi and Boyd, 1985, Mol. Gen. Genet. 199: 500-06.

Petschek, Perrimon and Mahowald, 1987, Dev. Biol. 19: 175-89.

Mohler, Eldon and Pirrotta, 1989, EMBO J. 8: 1539-48.

phenotype: Larval development 4 days longer than normal resulting in giant larvae, pupae, and imagos. Adult weight 1.7 times normal; increased size caused by increase in cell size and not cell number [Simpson and Morata, 1980, Development and Neurobiology of *Drosophila* (Siddiqi, Babu, Hall and Hall, eds.). Plenum Press, New York and London, pp. 129-40]. Pupariation delayed owing to delayed increase in ecdysteroid titers; level reached at pupariation lower than normal; pupal interval of normal length (Schwartz, Imberski, and Kelly, 1984, Dev. Biol. 103: 85-95). Not all genetically giant flies show the giant character, the rest have normal size; distribution sharply bimodal. Percentage giant greatest in well-fed cultures, also raised by modifying action of *bb¹¹*. Penetrance of viable alleles enhanced in heterozygotes with lethal alleles and deficiencies; viability decreased (Kaufman, 1972, Genetics 71: s28-29). Abnormalities in DNA metabolism found in homo- or heteroallelic third instar *gt* females (Narachi and Boyd, 1985). Salivary gland chromosomes of double thickness in some cells (Bridges, 1935, J. Heredity 26: 60-64). Feulgen staining shows extra round of DNA synthesis; polytene chromosome can be analyzed in *gt/Df* larvae (Kaufman, 1972, Genetics 71: s28-29). Embryos carrying lethal giant mutations have defects in the anterior and the posterior domains (Petschek *et al.*, 1987; Mohler *et al.*, 1989). Posterior compartment of the labial segment deleted from blastoderm; cell death at germ-band elongation deletes anterior compartments of abdominal segments 5-7. Posterior-compartment structures of A5-7 in the peripheral nervous system fuse in mature embryos (Petschek and Mahowald). Hemizygotes for lethal alleles, *gt^{13z}* and *gt^{X11}*, fail to hatch (Kaufman, 1973, Genetics 74: s133); denticle belts of the fifth through the seventh abdominal segments partially or completely absent; internally corresponding neuromeres absent; eight abdominal neuromeres disconnected from remainder. Head does not complete involution, shows characteristic "buttonhead" phenotype with ventral skeleton extruded from the anterior end of the larva (Mohler *et al.*, 1989), pharynx and pharyngeal chitinized sclerites shorter, and brain lobes somewhat smaller than normal; extensive cell death in epidermal and neural components of embryo (Honisch and Campos-Ortega, 1982, DIS 58: 76-77). Effect cell autonomous in mosaic embryos. (Gergen and Wieschaus, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 49-62). Germline clones viable (García-Bellido and Robbins, 1983, Genetics 103: 235-47). *gt* stocks (homo- or heteroallelic) show an increase in the frequency of spontaneous mutations, including deletions of *y* and *w* loci (Green, 1982, Proc. Nat. Acad. Sci. USA 79: 5367-69); these stocks also synthesize DNA of a reduced molecular weight and show many single-strand and double-strand breaks, suggesting abnormalities in DNA metabolism (Narachi and Boyd, 1985). RK3.

alleles: In addition to those tabulated, Wieschaus, Nüsslein-Volhard, and Jürgens (1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307) report eight embryonic lethal alleles, of which four are weak; these exhibit defects in head and in fifth through seventh abdominal

segments.

alleles	origin	discoverer	acquisition number	ref ^α	viability
<i>gt^{1β}</i>	spont	Gabritschevsky, 25i2		1, 3, 9	viable-low penetrance
<i>gt^{13z}</i>	NNG ^γ	Kaufman		5, 6	lethal
<i>gt³⁰</i>	P			9	
<i>gt¹²³</i>				4	sub vital
<i>gt^{E6}</i>	EMS	Kaufman		5, 6	viable-low penetrance
<i>gt¹¹</i>			190	2	lethal
<i>gt^{12δ}</i>			1685	2, 9	lethal
<i>gt¹³</i>			1710	2	lethal
<i>gt¹⁴</i>			1859	2	lethal
<i>gt¹⁵</i>			1903	2	lethal
<i>gt¹⁶</i>			1907	2	lethal
<i>gt¹⁷</i>			2413	2	lethal
<i>gt¹⁸</i>			2684	2	lethal
<i>gt¹⁹</i>			2868	2	lethal
<i>gt¹¹⁰</i>			4798	2	lethal
<i>gt¹¹¹</i>			6749	2	lethal
<i>gt¹¹²</i>			7331	2	lethal
<i>gt¹¹³</i>			8868	2	lethal
<i>gt^{M102}</i>	MMS			7	lethal
<i>gt^{Q292}</i>	EMS	Shen			
<i>gt^{X11}</i>	X ray	Falk		6, 8, 9	lethal
<i>gt^{XH}</i>				9	lethal
<i>gt^{YA82}</i>				9	lethal

^α 1 = Bridges and Gabritschevsky, 1928, Z. Indukt. Abstamm. Vererbungsl. 49: 231-47 (fig.); 2 = Dutttagupta, Das, and Dutta, 1984, DIS 60: 90-91; 3 = Gabritschevsky and Bridges, 1928, Z. Indukt. Abstamm. Vererbungsl. 49: 248-84; 4 = García-Bellido and Robbins, 1983, Genetics 103: 235-47; 5 = Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56; 6 = Kaufman, 1972, Genetics 71: s28-29; 7 = Liu and Lim, 1975, Genetics 79: 601-11; 8 = Mohler, Eldon and Pirrotta, 1989, EMBO J. 8: 1539-48; 9 = Petschek, Perrimon and Mahowald, 1987, Dev. Biol. 19: 175-89.

^β Associated with two inserts of DNA into the *gt* locus, one near +17 and the other near +32 on the molecular map (Mohler *et al.*, 1989).

^γ N-methyl-N¹-nitro-N-nitrosoguanidine.

^δ *Tp(1;1)12 = Tp(1;1)3A2;8D;10B1*; weak lethal allele induced by irradiation (Mohler *et al.*, 1989).

cytology: Placed in 3A2 by Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56; and Lefevre, 1981, Genetics 99: 461-80; later placed in 3A1 by Mohler *et al.*, 1989.

molecular biology: Rearrangement breakpoints associated with *gt* were positioned on the genomic clone map of Mariani, Pirrotta, and Manet (1985, EMBO J. 4: 2045-52). Mohler *et al.*, 1989, report confirmation of the cloning of *gt* by partial rescue with *P*-elements. The region from +18 to +21 on the molecular map is practically identical to a 1.9 kb RNA expressed in 2- to 4-hour embryos but barely detectable in 4- to 6-hour embryos; this region is included in the DNA fragment used in the complete rescue of the abdominal segmentation defects of the giant mutant *gt^{X11}* (Mohler *et al.*, 1989). Sequencing of the cDNA of *gt* suggests that the gene encodes a novel protein that shows identity to *opa* but not to other pattern-forming genes (Eldon and Pirrotta, 1989).

other information: Used by Bridges (1935) in the construction of salivary chromosome maps. Preliminary evidence of interallelic complementation with respect to phenotype and viability presented by Dutttagupta, Das, and Dutta (1984, DIS 60: 90-91).

***Gt2: Giant in chromosome 2**

location: 2- (not located).

origin: Spontaneous.

discoverer: Bridges, 14i28.

phenotype: Heterozygote normal but, in presence of

homozygous *gt3*, gives giant, male-sterile flies. Homozygous lethal. RK3.

***gt3: giant in chromosome 3**

location: 3-64.

origin: Spontaneous.

discoverer: Bridges, 14i28.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 120 (fig.).

phenotype: Body size much larger than normal. Late hatching. Entirely sterile in male. Giant character produced only in flies homozygous for *gt-3* and heterozygous for *Gt-2*. RK3.

gt4

location: 2-24.0.

origin: Spontaneous.

discoverer: Bridges, 30b14.

phenotype: Giant flies hatch very late. Viability variable but around 15% wild type. RK3.

***gtd: giantoid**

location: 1-0.5.

origin: Spontaneous.

discoverer: Bridges, 21c12.

references: Bridges and Gabritschewsky, 1928, Z. Indukt. Abstamm. Vererbungsl. 46: 232 (fig.).

phenotype: Body size larger, especially head. Late hatching. Viability erratic, about 50% wild type. Separation difficult in females, easier in males. RK3.

gua1: guanosine requiring

location: 1-31.

origin: Induced by ethyl methanesulfonate.

synonym: *gual-1^{ts}*.

references: Falk and Nash, 1974, Genetics 76: 755-56.

phenotype: A temperature-sensitive guanosine auxotroph. Hemizygous viability on guanosine-free medium reduced at 25°; virtually lethal at 29° although completely supplementable at that temperature. Normal allele thought to function in the conversion of inosinic acid to guanic acid.

cytology: Placed in 9D1-E1 by deficiency analysis; temperature sensitive on minimal medium in combination with *Df(1)ras-v = Df(1)9D1-2;10A2-3*, but not *Df(1)^v64f = Df(1)9E7-8;10A2-3* or *Dp(1;2)^v63i = Dp(1;2)9D4-E1;10A11-B1;56A* (Johnson, Woloshyn, and Nash, 1979, Mol. Gen. Genet. 174: 287-92). This small region carries in addition *pur1*, *ras*, and lethals, which fail to complement the other three loci (Nash, Woloshyn, and Janca, 1979, Genetics 92: s87).

gua2: see *bur*

guanosine requiring: see *gua1*

Guanosine triphosphate cyclohydrolase:
see *Pu*

Gull: see **G**

Gulloid: see **Gd**

gumper: see *fliF*

gurken: see **grk**

gus: gustatory (J. C. Hall)

A series of ethyl-methanesulfonate-induced taste mutants selected by Tompkins *et al.*, 1979. All mutants were crudely mapped and complementation tests performed between those mapping to the same interval.

references: Tompkins, Cardoza, White, and Sanders, 1979, Proc. Nat. Acad. Sci. USA 76: 884-86.

mutant	location	alleles
<i>gusA</i>	1- (v - f)	<i>gusA^{Q1}</i> , <i>gusA^{Q2}</i> , <i>gusA^{Q3}</i> , <i>gusA^{Q4}</i>
<i>gusC</i>	1- (cv - v)	<i>gusC^{N10}</i>
<i>gusD</i>	1- (v - f)	<i>gusD^{N9}</i> , <i>gusD^{N12}</i>
<i>gusE</i>	1- (v - f)	<i>gusE^{N1}</i>

gusA

phenotype: Attracted to quinine (C₂₀H₂₄N₂O₂), unlike wild type, which is repelled; normal aversion to sodium chloride (NaCl). One allele, *gusA^{Q1}*, leads to cold-sensitive phenotype, in that larvae or adults, raised at 22°, show the aberrant attraction to quinine, but when raised at higher temperatures, these animals show normal behavior; the temperature-sensitive period of *gusA^{Q1}* is during embryogenesis (Tompkins, 1979, Dev. Biol. 73: 174-77).

other information: Not allelic to *gusD* or *gusE*.

gusB: see *gustC*

gusC

phenotype: Attracted to sodium chloride (NaCl), unlike wild type, which is repelled; normal aversion to quinine (C₂₀H₂₄N₂O₂).

gusD

phenotype: Attracted to quinine (C₂₀H₂₄N₂O₂) and to sodium chloride (NaCl), unlike wild type, which is repelled by both.

other information: Not allelic to *gusA* or *gusE*.

gusE

phenotype: Attracted to quinine (C₂₀H₂₄N₂O₂) and to sodium chloride (NaCl), unlike wild type, which is repelled by both; larval chemosensory behavior normal; is cold-sensitive, such that exposure of third larval instar to 22° (but not higher temperatures) results in aberrantly responding adults (Tompkins, 1979, Dev. Biol. 73: 174-77).

other information: Not allelic to *gusA* or *gusD*.

gusF: see *gustB*

gust: gustatory defective (J.C. Hall)

Another series of ethyl-methanesulfonate-induced taste mutants that affect the fly's responses to attractants and repellents. All mutants were crudely mapped and complementation tests performed between those mappings to the same interval. Complementation tests with *gus* mutants were also performed. Sodium chloride (NaCl) elicits responses from two sensory neurons (L1 and L2) in each taste sensillum; another sensory neuron (S) involved in response to sugars (Siddiqi, Joshi, Arora, and Rodrigues, 1989, Genome 31: 646-51). A fourth neuron is inhibited by sodium chloride (NaCl) and sugars (Arora *et al.*, 1987).

mutant	location	synonym	ref ^α	alleles
<i>gustA</i>	1-45	<i>gust^{x1}</i> , <i>gust^{x2}</i>	3, 4	<i>gustA^{x1}</i> , <i>gustA^{x4}</i>

mutant	location	synonym	ref ^α	alleles
<i>gustB</i>	1-38	<i>gust</i> ^{x5} , <i>gustF</i>	1, 3-5	<i>gustB</i> ^{x5} , <i>gustB</i> ^{x7} <i>gustB</i> ^{FN5} , <i>gustB</i> ^{FN32}
<i>gustC</i>	1- (v - f)	<i>gustB</i> , <i>gust</i> ^{x2}	3-5	<i>gustC</i> ^{x2} , <i>gustC</i> ^{B04} , <i>gustC</i> ^{B05}
<i>gustD</i>	1- (v - f)	<i>gust</i> ^{x3} , <i>gust</i> ^{x6}	3, 4	<i>gustD</i> ^{x3} , <i>gustD</i> ^{x6}
<i>gustE</i> ^β	1-		4	<i>gustE</i> ^{v86}
<i>gustF</i>			4	
<i>gustM</i>	3- {71}		2	
<i>GustR</i>	3- (ru - h)		4	
<i>GustS</i>			4	

^α 1 = Arora, Rodrigues, Joshi, Shanbhag, and Siddiqi, 1987, Nature (London) 330: 62-63; 2 = Morea, 1985, Experientia 41: 1381-84; 3 = Rodrigues and Siddiqi, 1978, Proc. Indian Acad. Sci. 87B: 147-60; 4 = Siddiqi, Joshi, Arora, and Rodrigues, 1989, Genome 31: 646-51; 5 = Siddiqi and Rodrigues, 1980, Development and Neurobiology of *Drosophila* (Siddiqi, Babu, Hall, and Hall.), Plenum Press, New York, pp. 347-59.

^β Cytological location between 6C11 and 6E4-5 (Siddiqi *et al.*, 1989).

gustA

phenotype: Insensitive to pyranose sugars (C₇H₁₂O₆), but shows normal response to fructose (C₆H₁₂O₆) and normal aversion to quinine (C₂₀H₂₄N₂O₂) and sodium chloride (NaCl). Receptor defective mutant (Siddiqui *et al.*, 1989). One type of sense hairs on the labellum shows reduced physiological responses to pyranoses (Rodrigues and Siddiqi, 1981, Mol. Gen. Genet. 81: 406-08); another type of sense hairs is insensitive to the normal inhibition by pyranose sugars.

gustB

phenotype: Originally said to be "indifferent" to sodium chloride (NaCl), but now known to give positive responses (greater than wild type) to this salt in the feeding preference test which consist of behavioral (proboscis extension) and physiological (labellar recording) responses (Arora). Responds normally to sucrose (C₁₂H₂₂O₁₁) and quinine (C₂₀H₂₄N₂O₂). In this mutant, the S neuron, usually involved in the sugar response, is excited by sodium chloride (NaCl) (Arora *et al.*, 1987). Two forms of alpha-glucosidase activity (enriched in tarsal leg segments relative to other tissues in wild type) are lower than normal in two of the *gustB* alleles (unspecified) (Bhavsar, Rodrigues, and Siddiqi, 1983, J. Biosci. 5: 279-87).

cytology: Placed in 10E1 since included in *Df(1)m259-4 = Df(1)10C2-3;10E1-2* and *Df(1)KA6 = Df(1)10E1;11A7-8* (Arora *et al.*, 1987).

other information: Allelic to *gusF* of Tompkins, Cardoso, White, and Sanders (1979, Proc. Nat. Acad. Sci. USA 76: 884-86).

gustC

phenotype: Originally said to be "indifferent" to sodium chloride (NaCl), but now known to give responses (greater than wild type) to this salt in the feeding preference test (Arora). Indifferent to quinine (C₂₀H₂₄N₂O₂), and sucrose (C₁₂H₂₂O₁₁). Also at least one allele (*gustC*^{x2}) leads to lower amounts of two forms of alpha-glucosidase activity that are normally found in the legs [and are enriched in the tarsi of these appendages in wild type (Bhavsar *et al.*, 1983)].

other information: Allelic to *gusB* (Tompkins *et al.*, 1979).

gustD

phenotype: Insensitive to quinine (C₂₀H₂₄N₂O₂), but shows normal responses to sucrose (C₁₂H₂₂O₁₁) or to sodium chloride (NaCl); both alleles are heat-sensitive, in that rearing at 28°C leads to aberrant responses of adults, but development at low temperature leads to normal-behaving flies; levels of leg-specific alpha-glucosidases are normal or nearly so (Bhavsar *et al.*, 1983).

other information: Not allelic to *gusA*, *gusB*, *gusD*, or *gusE*.

gustE

phenotype: No attraction to sodium chloride (NaCl) in feeding tests (*gustE*^{v86}); response to potassium chloride (KCl) is the same as in wild type.

gustF

phenotype: Electrophysical sensitivity to both sodium chloride (NaCl) and potassium chloride (KCl) reduced; behavioral characteristics not tested by Siddiqui *et al.*, (1989).

gustM

phenotype: Insensitive to sodium chloride (NaCl) and quinine sulfate (C₂₀H₂₄N₂O₂) in proboscis extension responses (i.e. these substances fail to inhibit the sugar-induced reflex); further behavioral testing, however, reveals abnormally prolonged drinking of salt solutions and attraction to quinine; sugar responses normal.

cytology: The mutant is located at 93D and associated with *In(3R)AFA (86C4-5;93D1-2)*; the behavioral abnormalities are uncovered by *Df(3R)e-N12 (93B1-2;93D6-7)*, *Df(3R)e-GC9 (93B11-13;93D9-10)*, and *Df(3R)e-D7 (93C3-6;93F5-8)*, implying that the righthand inversion breakpoint is responsible.

GustR

phenotype: Attracted to sodium chloride (NaCl) but not potassium chloride (KCl); shows reduced attraction to all sugars. L1 neurons involved in sodium chloride (NaCl) response (Siddiqi *et al.*, 1989).

GustS

phenotype: Attracted to sodium chloride (NaCl) but not potassium chloride (KCl); shows reduced attraction to all sugars.

gustatory: see *gus*

gustatory defective: see *gust*

****gv:* grooved**

location: 3-37.3 (based on its position of 0.2cM to the left of *eyg*).

origin: Spontaneous.

discoverer: Ives, 43128.

references: 1946, DIS 20: 65.

synonym: *gs: gespleten*.

phenotype: A longitudinal medial groove in thorax; in extreme individuals, thorax nearly cleft. Eyes reduced, sometimes missing. Irregular and often extra alar bristles. Viability and fertility good. RK1.

allele	origin	discoverer	synonym	ref ^α
* <i>gv</i> ¹	spont	Ives, 43128		2
<i>gv</i> ²	spont	Smelink-den-Hollander, 561	<i>gs</i> ^P	3
<i>gv</i> ^P	γ ray		<i>gs</i> ^U	1
<i>gv</i> ^U ^β	γ ray		<i>gs</i> ^U	1

allele	origin	discoverer	synonym	ref ^α
α	1	Akam, Roberts, Richards, and Ashburner, 1978, Cell 13: 215-25; 2 = Ives, 1946, DIS 20: 65; 3 = Smelink-den-Hollander, 1957, DIS 31: 85.		
β	Associated with	$ln(3LR)gv^u = ln(3LR)69C1-2;81$; homozygous lethal.		

cytology: Placed in 69C on the basis of its exclusion from $Df(3L)vin^7 = Df(3L)68C8-11;69B3-C1$ and the *gv* phenotype of $gv/ln(3LR)gv^u = ln(3LR)69B5-C4;81$ (Akam, Roberts, Richards, and Ashburner, 1978, Cell 13: 215-25).

gvl: grooveless

location: 4-0.2 [in diplo-4 triploids (Sturtevant, 1951, Proc. Nat. Acad. Sci. USA 37: 405-7)].

origin: Spontaneous.

discoverer: Bridges, 33e10.

references: 1935, Biol. Zh. (Moscow) 4: 401-20.

phenotype: Short transverse groove between scutellum and thorax is nearly eliminated; no overlap with wild type. Black scars appear on scutellar groove at sides, in pleural region, and behind sternopleurals. Viable and fertile. RK1.

gy: gouty legs

location: 4-0.2.

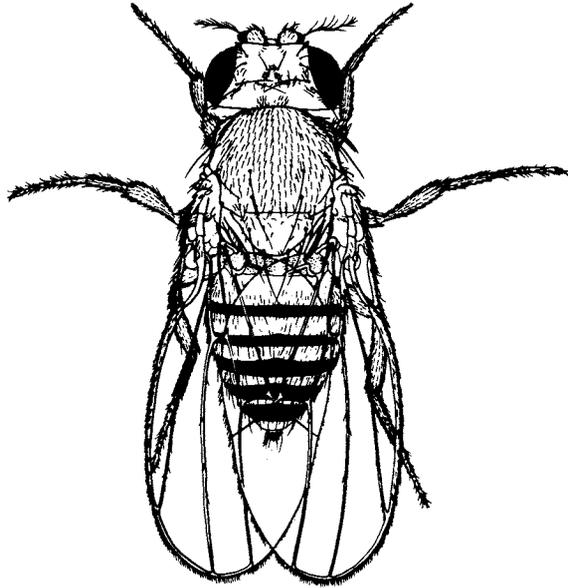
origin: Spontaneous.

discoverer: Muller.

references: 1965, DIS 40: 36.

phenotype: Legs shortened and thickened, especially the metatarsi of the hind legs, which are often swollen. Usually classifiable; viability and fertility good. gy/ey^D is *gy*. RK2.

cytology: Tentatively placed at 102B10-E9 between $Df(4)M4 = Df(4)101E-F;102E2-10$ and tip (Hochman, 1971, Genetics 67: 235-52).



h: hairy

From Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 202.

h: hairy

location: 3-26.5.

synonym: *barrel* (Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-69).

references: Mohr, 1922, Z. Indukt. Abstamm. Vererbungsl. 28: 17.

Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 202 (fig.).

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 214 (fig.).

Neel, 1944, Genetics 26: 52-68 (fig.).

Nüsslein-Volhard and Wieschaus, 1980, Nature (London) 287: 795-801.

Holmgren, 1984, EMBO J. 3: 569-73.

Ingham, Howard, and Ish-Horowicz, 1985a, Nature (London) 318: 439-45.

Ingham, Pinchin, Howard, and Ish-Horowicz, 1985b, Genetics 111: 463-86.

Ish-Horowicz, Howard, and Ingham, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 135-44.

Ish-Horowicz, Howard, Pinchin, and Ingham, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 135-44.

Ish-Horowicz and Pinchin, 1987, Cell 51: 405-15.

Carroll, Laughon, and Thalley, 1988, Genes Dev. 2: 833-90.

Howard, Ingham, and Rushlow, 1988, Genes Dev. 2: 1037-46.

Carroll and Whyte, 1989, Genes Dev. 3: 905-16;

Rushlow, Hogan, Pinchin, Howe, Lardelli, and Ish-Horowicz, 1989, EMBO J. 8: 3095-3103.

phenotype: The pair-rule gene hairy regulates the development of alternate segments in the embryo as well as the spatial expression of another pair-rule gene fushi tarazu (Holmgren, 1984; Carroll *et al.*, 1988; Rushlow *et al.*, 1989). A later phenotypic expression of hairy, the adult bristle pattern, is established during larval and pupal

stages (Nüsslein-Volhard and Wieschaus, 1980; Ingham *et al.*, 1985a, 1985b). In the embryo, *h* mutations delete the posterior part of each odd-numbered segment, weak alleles deleting less than a whole segment and strong alleles deleting regions greater than one segment. In mutant adults, extra microchaetae are found along wing veins, L2 more so than L4 or 5, and on wing membrane; also on dorsal and ventral scutellum and top of head. Extra sensilla present on longitudinal wing veins in a gradient in which sensilla are concentrated proximally and hairs distally; intermediate structures found in the middle (Spivey and Thompson, 1984, Genetics 107: s102). Extra acrostichal row on either side of midline between dorsocentral bristles (Claxton, 1971, DIS 46: 133); also occupy thin arch of cuticle connecting ventral scutellum and pleurae. Microchaetae found on mesopleurae (mean of 13 in males and 20 in females versus none in wild type) and pteropleurae (Murphy, 1972, J. Exp. Zool. 179: 51-62). Used by García-Bellido and Ferrus (1975, Wilhelm Roux's Arch. Dev. Biol. 178: 337-40) to provide cuticular markers on pleurae for fate mapping. Additional hair-forming cells present in 19-hr pupa (Lees and Waddington, 1942, DIS 16: 70). Autonomous expression in clones produced prior to the last eight hr of larval life; clones produced during the last eight hr before pupation exhibit normal phenotype; attributable to perdurance of wild-type gene product (García-Bellido and Merriam, 1971, Proc. Nat. Acad. Sci. USA 68: 2222-26). Reduced *ac*⁺ function as in *ac*³ or *ac*^{3/+} suppresses *h* phenotype; extra doses of *ac*⁺ enhance *h* expression and can render *h* partially dominant (Sturtevant, 1969, Dev. Biol. 21: 48-61; Botas, Moscoso del Prado, and García-Bellido, EMBO J. 1: 307-10). Three doses of *h*⁺ suppress *Hw* (Botas, *et al.*). *h* expression also enhanced by combination with rearrangements that place the *ac-sc* region in juxtaposition with substantial quantities of heterochromation (Green, 1960, Proc. Nat. Acad. Sci. 46: 524-28). Interactions with *sc* alleles detailed by Sturtevant (1969). *h*² less severe than and partially complements *h*¹ (Sturtevant). As with *ci*⁺, expression of *h*⁺ may be altered in the direction of *h* by rearrangements with breakpoints in the vicinity of the *h* locus (Dubinin and Sidorov, 1934, Biol. Zh. 3: 307-31; see also Jeffrey, 1979, Genetics 91: 105-25). Unlike the *ci* case, however, rearranged *h* chromosomes do not show evidence of altered gene action (Stern, 1944, DIS: 18:56).

alleles:

alleles	origin	discoverer	synonym	ref ^α	comments	cytology
<i>h</i> ^{1β}	spont	Mohr, 1811		1, 3, 10, 12	viable	
<i>h</i> ²	spont	Bridges, 28d23		3	viable	
<i>*h</i> ³	spont	Bridges	<i>*h</i> ^{26c}	2		
<i>*h</i> ⁴	spont	Bridges	<i>*h</i> ^{30a}	2		
<i>*h</i> ⁵	spont	Nichols-Skoog	<i>*h</i> ^{33k}	2		
<i>*h</i> ⁶	spont	Bridges	<i>*h</i> ^{34e}	2		
<i>*h</i> ⁷	spont	Curry	<i>*h</i> ^{38d}	2		
<i>*h</i> ⁸	spont	Neel	<i>*h</i> ^{41k}	2		
<i>*h</i> ⁹	spont	Neel	<i>*h</i> ^{42a}	2		
<i>*h</i> ¹⁰	X ray	Alexander	<i>*h</i> ^{100.12}	14	viable	<i>In(3L)61A2-3; 66D</i>
<i>*h</i> ¹¹	X ray	Alexander	<i>*h</i> ^{100.239}	14	semilethal	<i>In(3L)66D11-12; 80C</i>
<i>*h</i> ¹²	X ray	Alexander	<i>*h</i> ^{100.271}	14	lethal	<i>T(2;3)41; 66D14-E1</i>

alleles	origin	discoverer	synonym	ref ^α	comments	cytology
<i>h</i> ¹³ γ	X ray	Green	<i>h</i> ^s	4	nearly lethal	
<i>h</i> ¹⁴	X ray	Braver	<i>h</i> ^{R06}	9		<i>T(1;3)1B10-C1</i> ; <i>66D14-E1</i>
<i>h</i> ¹⁵	X ray	Braver	<i>h</i> ^{R15}	9	lethal	<i>In(3LR)66D14-E1</i> ; <i>85B5-C12</i>
<i>h</i> ¹⁶	X ray	Jeffrey	<i>h</i> ^{R38}	8, 9	15% viable	<i>In(3L)66D14-E1</i> ; <i>80F</i>
<i>h</i> ¹⁷	X ray	Jeffrey	<i>h</i> ^{R40}	8, 9	lethal	<i>Tp(3;2)30B7-C1</i> ; <i>66D14-E1</i> ; <i>73B7-C1</i>
<i>h</i> ¹⁸	X ray	Jeffrey	<i>h</i> ^{R44}	8, 9	27% viable	<i>Tp(3;4)63A3-B1</i> ; <i>66F2-67A1</i> ; <i>101F</i>
<i>h</i> ¹⁹	X ray	Jeffrey	<i>h</i> ^{R47}	8, 9	5% viable	<i>T(2;3)23A2-B1</i> ; <i>34C2-4</i> ; <i>66D6-10</i> ; <i>98F12-99A1</i>
<i>h</i> ²⁰ γ	EMS	Jürgens, Nüsslein-Volhard	<i>h</i> ^{brr}	12	lethal	
<i>h</i> ²¹	EMS	Nüsslein-Volhard	<i>h</i> ^{9N53}	5	lethal	
<i>h</i> ²²	EMS		<i>h</i> ^{C1}	5, 7	lethal	
<i>h</i> ²³	EMS		<i>h</i> ^{C2}	12	lethal	
<i>h</i> ²⁴	EMS		<i>h</i> ^{1K93}	7, 10	lethal	
<i>h</i> ²⁵	EMS in <i>h</i> ¹	Jürgens	<i>h</i> ^{5H07}	7, 13	lethal	
<i>h</i> ²⁶	EMS in <i>h</i> ¹	Jürgens	<i>h</i> ^{7H94}	7, 13	lethal	
<i>h</i> ²⁷	EMS	Jürgens	<i>h</i> ^{8K115}	7, 13	lethal	
<i>h</i> ²⁸	EMS in <i>h</i> ¹	Jürgens	<i>h</i> ^{9K78}	7, 13	lethal	
<i>h</i> ²⁹	EMS in <i>h</i> ¹	Nüsslein-Volhard	<i>h</i> ^{11D}	7, 13	lethal	
<i>h</i> ³⁰	EMS	Nüsslein-Volhard	<i>h</i> ^{12C}	7, 13	lethal	
<i>h</i> ³¹	EMS	Nüsslein-Volhard	<i>h</i> ^{14H}	7, 13	lethal	<i>In(3LR)66D14-E1</i> ; <i>85B5-C12</i>
<i>h</i> ³²	HD	Ingham	<i>h</i> ^{d1.1}	7	lethal	
<i>h</i> ³³	HD	Ingham	<i>h</i> ^{d1.2}	7	lethal	
<i>h</i> ³⁴	HD	Ingham	<i>h</i> ^{d3}	7	lethal	
<i>h</i> ³⁵	HD	Ingham	<i>h</i> ^{d4.1}	7	lethal	
<i>h</i> ³⁶	HD	Ingham	<i>h</i> ^{d6}	7	lethal	
<i>h</i> ³⁷	HD	Ingham	<i>h</i> ^{d7}	7	lethal	
<i>h</i> ³⁸	HD	Ingham	<i>h</i> ^{d8}	7	lethal	
<i>h</i> ³⁹	HD	Ingham	<i>h</i> ^{d9}	7	lethal	
<i>h</i> ⁴⁰	HD	Ingham	<i>h</i> ^{d11}	7	lethal	<i>In(3L)66D10-15</i> ; <i>72C</i>
<i>h</i> ⁴¹	HD	Jürgens	<i>h</i> ^{1L79K}	7	lethal	
<i>h</i> ⁴² δ	X ray	Ingham	<i>h</i> ^{K1}	6, 7	viable	<i>In(3L)65B1-2</i> ; <i>66D6-10</i>
<i>h</i> ⁴³	X ray	Moscoso del Prado	<i>h</i> ^{M1}		viable	
<i>h</i> ⁴⁴	X ray	Moscoso del Prado	<i>h</i> ^{M2}		viable	
<i>h</i> ⁴⁵	X ray	Moscoso del Prado	<i>h</i> ^{M3}		viable	
<i>h</i> ⁴⁶	EMS	Moscoso del Prado	<i>h</i> ^{M4}			<i>T(1;3)</i>
<i>h</i> ⁴⁷	X ray	Ingham	<i>h</i> ^{m1}	7	lethal	<i>T(2;3)41A</i> ; <i>66D9-11</i>
<i>h</i> ⁴⁸	X ray	Ingham	<i>h</i> ^{m2}	7	lethal	<i>In(3L)66D5-11</i> ; <i>80C</i>
<i>h</i> ⁴⁹ δ	X ray	Ingham	<i>h</i> ^{m3}	6, 7	lethal	<i>Tp(3;3)61E</i> ; <i>66C:66D</i>
<i>h</i> ⁵⁰ δ	X ray	Ingham	<i>h</i> ^{m7}	6	lethal	<i>In(3L)66D6</i> ; <i>67D8</i>
<i>h</i> ⁵¹ δ	X ray	Ingham	<i>h</i> ^{m8}	6	lethal	

^α 1 = Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 202 (fig.); 2 = CP552; 3 = CP627; 4 = Green, 1960, Proc. Nat. Acad. Sci. USA 46: 524-28; 5 = Holmgren, 1984, EMBO J. 3: 569-73; 6 = Howard, Ingham, and Rushlow, 1988, Genes Dev. 2: 1037-46; 7 = Ingham, Pinchin, Howard, and Ish-Horowicz, 1985, Genetics 111: 463-86; 8 = Jeffrey, 1971, DIS 47: 37; 9 = Jeffrey, 1979, Genetics 91: 105-25; 10 = Mohr, 1922, Z. Indukt. Abstamm. Vererbungsl. 28: 17.; 11 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 214 (fig.); 12 = Nüsslein-Volhard and Wieschaus,

1980, Nature 287: 795-801; 13 = Tearle, and Nüsslein-Volhard, 1987, DIS 66: 209-69; 14 = Ward and Alexander, 1957, Genetics 42: 42-54.

^β Result of *gypsy* insertion (Holmgren, 1984).

^γ Phenotype described separately; alleles also described in *h*²⁰.

^δ Regulatory mutation causing alterations in hairy expression in cellular blastoderm that result in a partial version of the wild-type pattern (Howard *et al.*, 1988).

cytology: Placed in 66D15 on the basis of the *h* phenotypes of many rearrangements with breakpoints between 66D14 and 66E1 (Jeffrey, 1979). Placed in 66D9-11 by Ingham *et al.* (1985) based on the left breakpoint of *T(2;3)h^{m1} = T(2;3)66D9-11;41A*.

molecular biology: The hairy region was tagged and cloned with the transposing element *gypsy* (Holmgren, 1984) and with *P* elements (Ish-Horowicz *et al.*, 1985). Cloned genes were able to rescue *h* segmentation mutants when introduced into flies by *P*-element transposition (Rushlow *et al.*, 1989). The gene hairy encodes a major transcript of 2.1 to 2.3 kb that is expressed at high levels in 2-4 hr embryos but at lower levels later (Ish-Horowicz *et al.*, 1985; Rushlow *et al.*, 1989) and a minor 2.0 kb transcript that is expressed at low levels in 2-4 hr embryos but at higher levels in larvae and adults. Two other transcripts, 1 kb and 3 kb, have been reported during larval life (Ish-Horowicz *et al.*, 1985); the time of expression and chromosomal origin of these RNAs indicate that they are involved in hairy bristle function. All RNAs are transcribed from distal to proximal and must differ at their 5' ends. Genomic and cDNA sequences have been obtained (Rushlow *et al.*, 1989); they show that the hairy transcription unit is interrupted by two introns. The gene encodes a 337 amino acid protein, which contains a helix-loop motif (Rushlow *et al.*, 1989), and functions both in segmentation in the embryo and bristle patterning in the adult. The protein is found in cell nuclei and is localized in eight distinct regions of the early embryo (Carroll *et al.*, 1988); the major protein stripes are located in the posterior and adjacent anterior parts of alternate segment primordia. There are striking patterns of nuclear protein distribution during larval and pupal imaginal disc development with transient hairy expression in the eye, leg, and wing discs (Carroll and Whyte, 1989).

other information: *h*¹ shown to recombine with and lie to the right of *h*² (Sturtevant; Rasmussen).

*h*¹³

synonym: *h*^s, hairy-subliminal.

phenotype: Homozygote nearly lethal but has no *h* phenotype. Heterozygote with *h* and *h*² also wild type. *h*^s/+ has extra hairs on wings, head, pleurae, halteres, and occasionally on scutellum if also heterozygous for certain *X*-chromosome inversions that variegate for *Hw*, including *In(1)sc*⁸, *In(1)sc*⁵¹, and *In(1)y*^{3P}. Presence of *y*⁺*Y* also induces extra hairs. RK3.

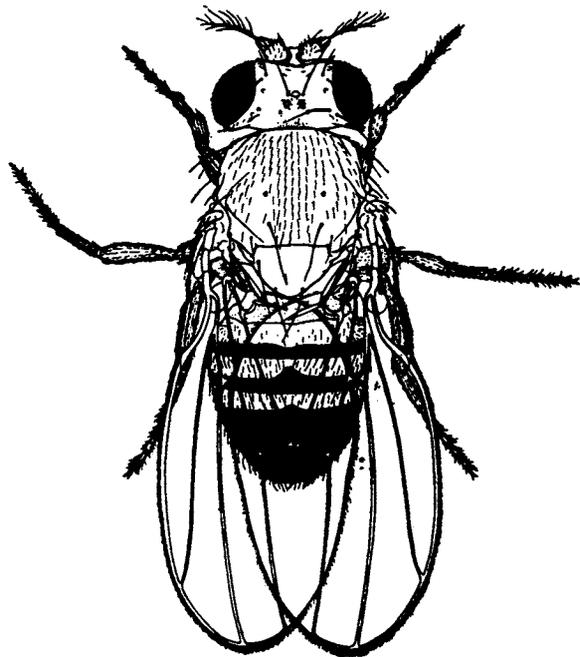
*h*²⁰

synonym: *h*^{brr}, hairy-barrel.

phenotype: Homozygous lethal. Newly hatched larvae lack denticle belts of alternate segments; *i.e.* the prothoracic, metathoracic and even numbered abdominal segments; naked cuticle missing from mesothorax and odd numbered abdominal segments (a member of the pair-rule class of Nüsslein-Volhard and Wieschaus). Pattern

of persisting segments often slightly irregular.

alleles: Eight embryonic lethal alleles, including four with weak expression, reported by Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding (1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95).



H: Hairless

From Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 161.

H: Hairless

location: 3-69.5.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 161 (fig.).

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 170 (fig.), 227.

Nash, 1965, Genet. Res. 6: 175-89.

Bang, Hartenstein, and Posakony, 1990, Development, in press.

phenotype: Bristles, especially postverticals and abdominals, missing in heterozygous *H* flies. Bristle sockets present at some sites, not at others. Expressed most distinctly on head; occipital; post vertical and ocellar bristles affected. Bristles of antennae and vibrissae show mutant phenotype much less frequently. Sockets without shafts also found on thorax, scutellum, abdominal tergites, external genitalia, wings, and legs. No shaftless sockets appear on the bracted costa of the wing. Some 40% of bristle organs located on distal part of femur differentiate neither shaft nor bract; bracts absent whenever shaft missing but present when shaft present; abnormally short shaft may be accompanied by normal-sized bract (from description of *H*², by Tobler, Rothenbühler, and Nöthiger, 1973, *Experientia* 29: 370-71). Veins L4 and L5 do not reach wing margin; occasionally true of L2 also. Eyes larger than wild type; body color somewhat paler. Lees and Waddington [1942, *Proc. Roy. Soc. (London)*, Ser. B. 131: 87-110 (fig.)] show that trichogen

cell forms a socket instead of a bristle shaft at some sites. Phenotypic expression of *H* responds linearly to dosage of *Su(H)*⁺ in region 35B6-10 on the left arm of chromosome 2. The number of microchaetae in *H*/+ flies varies from approximately 35 in the presence of a single dose of 35B6-10 to fewer than 10 in the presence of four doses (Ashburner, 1982, *Genetics* 64: 471-79). Interactions with other mutants studied by House (1953, *Genetics* 38: 199-215, 309-27; 1959, *Genetics* 44: 516; 1955, *Anat. Record* 122: 471; 1959, *Anat. Record* 134: 581-82). *H* suppresses wing notching of *N*, *fa*, *fa*^{no}, and *nd*, enhances *Ax*; also *H* enhances eye effect of *spl*, and removes more bristles in combination with *spl* (House, Von Halle). Reduction in the number of copies of the wild-type allele of *H* decreases the mutant phenotype of heterozygous *N* and *Dl* flies, but increase in the number of copies of the wild-type allele of *H* enhances the mutant phenotype of heterozygous *N*, *Dl*, and *E(spl)* flies (Vässin, Vielmetter, and Campos-Ortega, 1985, *J. Neurogenet.* 2: 291-308). *H* shows some superadditive interaction with *en*, *ci*, *ci*^W, and *ci*^D relative to degree of L4 interruption. L2 interruption augmented in combinations with *ve* and *ri*; L3 interruption augmented in combinations with *ve* and *tt*. Triploid, *H*/+/, intermediate between wild type and *H*/+. *H*/*H*/+ most extreme type with bristles absent from head, thorax, and abdomen [Gowen, 1933, *Am. Nat.* 67: 178-80 (fig.)]. Homozygous lethal. *H* null homozygotes die during larval and pupal stages (Bang *et al.*). Animals surviving to pharate adult are completely devoid of macrochaetes and microchaetes on the head and notum, with occasional "double sockets" remaining on the abdominal tergites. Bristles on the legs significantly resistant to loss of *H*⁺ function; many "double sockets" and some normal bristles remain on the legs of *H* null homozygotes. In regions of the notum exhibiting bristle "loss" in adult *H* mutants, macrochaete and microchaete primary precursor cells undetectable (Bang *et al.*). RK1.

alleles	origin	discoverer	synonym	ref ^α	comments
<i>H</i> ¹	spont	Bridges, 16c4	<i>H</i> ¹	5, 7, 10, 11, 13	
<i>H</i> ²	spont	Sturtevant	<i>H</i> ²	5, 13	
<i>H</i> ³		Sturtevant	<i>H</i> ³		
<i>H</i> ⁴	spont	Bridges, 30b20	* <i>H</i> ⁴		
<i>H</i> ⁵	X ray	Dobzansky, 1930	<i>H</i> ^{<i>Dl</i>}	6, 7	
<i>H</i> ⁶	X ray	Oliver, 28k4	<i>H</i> ^{28k}	6, 12	
<i>H</i> ⁷	X ray	Oliver, 32l28	<i>H</i> ^{32y}	6, 12	
<i>H</i> ⁸	X ray	Oliver, 32l28	<i>H</i> ^{32z}	6, 12	
<i>H</i> ⁹	X ray	Oliver, 33a2	<i>H</i> ^{33a}	6, 12	
<i>H</i> ¹⁰	X ray	Oliver, 35a5	<i>H</i> ^{35a}	6, 12	
<i>H</i> ¹¹	32 P	Bateman, 1939	<i>H</i> ^{P2}	4	
<i>H</i> ¹²	γ ray	Ives, 58b25	* <i>H</i> ^{58b}	9	T(Y;3)
<i>H</i> ^{13β}		Gloor	<i>H</i> ^{57c}	2, 14	
<i>H</i> ¹⁴	γ ray	Harrington	<i>H</i> ⁸⁰	2	
<i>H</i> ^{15δ}	spont	Gallego	<i>H</i> st	8	
<i>H</i> ^{16γ}	spont	Albornoz	<i>H</i> ^r	1	recessive
<i>H</i> ¹⁷			<i>H</i> ^{C19}	3	
<i>H</i> ¹⁸			<i>H</i> ^{C20}	3	
<i>H</i> ¹⁹			<i>H</i> ^{C25}	3	
<i>H</i> ²⁰			<i>H</i> ^{C28}	3	
<i>H</i> ²¹			<i>H</i> ^C	3	like <i>H</i> ²²
<i>H</i> ²²		Bang	<i>H</i> ^C	3	recessive
<i>H</i> ²³	EMS	Weigle	<i>H</i> ^{A4.1}		
<i>H</i> ²⁴	X ray	Schrons	<i>H</i> ^{KX1}		T(2;3)41;92F1-2
<i>H</i> ²⁵	X ray	Jiménez	<i>H</i> ^{w11}	15	
<i>H</i> ²⁶		Campos-Ortega	<i>H</i> ^{XIM}		

- α 1 = Albornoz, 1984, DIS 60: 44-45; 2 = Ashburner, 1982, Genetics 101: 447-59; 3 = Bang, Hartenstein, and Posakony, 1990, Development, in press; 4 = Bateman, 1950, DIS 24: 55; 5 = Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 161; 6 = CP552; 7 = CP627; 8 = Gallego, Garcia-Dorado, and Lopez-Fanjul, 1982, DIS 58: 64; 9 = Ives, 1959, DIS 33: 95; 10 = Morgan, Bridges, and Sturtevant, 1925, Bibliogr. Genet. 2: 170, 227; 11 = Nash, 1965, Genet. Res. 6: 175-89; 12 = Oliver, 1939, DIS 12: 48; 13 = Plunkett, 1926, J. Exp. Zool. 46: 181-244; 14 = Van Breugel, Ray, and Gloor, 1968, Genetica 39: 165-92; 15 = Väassin, Vielmetter, and Campos-Ortega, 1985, J. Neurogenet. 2: 291-308.
- β Occurred simultaneously with, but independently of $Tp(3;3)H^{57c} = Tp(3;3)86F7-11;95C1-2;97D1-2;98C5$.
- γ Fuller phenotypic description follows.
- δ Affects number of sternopleural bristles (Gallego *et al.*, 1982).

cytology: Located in 92E12 to 92F1-2 on the basis of the right breakpoint of $Df(3R)H-KX2$ (Campos-Ortega) and the third chromosome breakpoint of $T(2;3)H^{KX1}$ (Bang, Hartenstein, and Posakony, 1990, Development, in press).

H^{16}

phenotype: Recessive allele of Hairless (formerly called Hairless-recessive). Homozygotes have nearly all bristles and hairs substituted by double and triple abnormal sockets; veins L4 and L5 fail to reach margin. H^{16}/H lethal, probably in pupal stage; few escapers short lived with extreme Hairless phenotype; all bristles and hairs suppressed or substituted by abnormal sockets; wings reduced; veins L2, L4 and L5 abnormal.

H^{22}

phenotype: Less severe than H^{16} . Almost completely recessive. Heterozygotes most frequently wildtype in phenotype, but occasionally a "double socket" appears on the head in the position of a postvertical macrochaete or on the abdominal tergites. Homozygotes display a much stronger and more extensive mutant phenotype than H null heterozygotes. Many head and notum macrochaetes and approximately 50% of notum microchaetes missing; remaining 50% exhibit a spectrum of "double socket" phenotypes. Homozygotes also exhibit loss of wing vein tissue from L4 and L5. Approximately 50% of flies carrying H^{22r} *in trans* to a H null allele die as pharate adults. The remainder survive to eclosion, but are short-lived and exhibit extensive loss of both macrochaetes and microchaetes on the head, notum, and abdominal tergites; only 20% of the notum microchaetes remain, all with a completely transformed "double socket" phenotype.

H : see *Lvp*

$H2.0$: Homeobox 2.0

location: 2-.

references: Barad, Jack, Chadwick, and McGinnis, 1988, EMBO J. 7: 2151-61.

phenotype: Encodes a novel, tissue-specific homeobox. A large deletion including $H2.0$ is homozygous lethal, but the lethality may not be due to the homeobox gene. Although the structure of most organs as well as the epidermis appears to be normal in these lethal embryos, the midgut forms a balloon like yolk-filled sac.

cytology: Located in 26B1-3 by *in situ* hybridization to the salivaries.

molecular biology: Gene cloned from a library of Oregon-R genomic DNA using a *Scr* probe. The nucleotide sequence of the cDNA and the presumed amino acid

sequence were determined. A single major transcript of 1.85 kb is present throughout development, beginning at 6 hr of embryogenesis and becoming abundant between 6 and 24 hours. The cDNA includes a single methionine codon and an open reading frame of 1257 nucleotides with a highly divergent homeobox. There is a M or opa repeat near the center of the ORF with 21 CAX triplets coding for histidine and glutamine. The predicted protein is made up of 410 amino acids. This homeobox shows less than 40% identity to the *eve* and *z2* homeoboxes.

Tissue-specific expression of $H2.0$ was first detected at embryonic stage 10 at the flexure point between the posterior midgut invagination and the extended germ band. In stage 11, the homeobox is expressed in the visceral mesoderm in continual bands on either side of the midline from the posterior midgut invagination to the anterior midgut invagination. It is later detected in all cells destined to form the visceral musculature and especially in all cells of the splanchnopleura. Ectodermal expression occurs in a restricted lateral part of the posterior compartment of each segment. A line of $H2.0$ expressing cells connects each patch of ectodermal expression with the expression in the splanchnopleura. No expression was observed in the gut ectoderm or the somatic mesoderm.

****ha*: hair bristles**

location: 1-22.7.

origin: Induced by L-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1954.

references: 1958, DIS 32: 70.

phenotype: Small fly with extremely fine, short bristles. Males viable and fertile. Females less viable and highly infertile. RK3.

$H37$: see *elf*

Hab : see *BXC*

***Had*: β -Hydroxy-acid-dehydrogenase**

location: 1-54.4 (between *sl* and *r*).

references: Borack, Water, and Sofer, 1971, DIS 46: 43.

Borack and Sofer, 1971, J. Biol. Chem. 246: 5345.

Borack, 1974, Experientia 30: 31.

Tobler and Grell, 1978, Biochem. Genet. 16: 333-42.

phenotype: Thought to be the structural gene for β Hydroxy acid dehydrogenase (L- β -hydroxyacid: NAD oxidoreductase, E.C 1.1.1.45). The enzyme is a dimer, based on the formation of hybrid enzyme in *D.simulans* X *D. melanogaster* hybrids, of molecular weight 6.3 X 10⁴ daltons. Purification and biochemistry by Borak and Sofer. Activity high in Malpighian tubules, less in muscles, intestine, and fat body; absent from brain, salivary gland, and imaginal disks. (Borak, 1972, DIS 48: 73). Activity peaks at 72 hr after hatching, then declines through larval and pupal stages, and increases to stable maximum level at 6 days of adult life (Tobler and Grell). Enzyme expendible; hemizygotes and homozygotes for null allele survive normally (Tobler and Grell).

alleles: Three electrophoretic variants Had^F , Had^I and Had^S , with fast intermediate, and slow mobility described by Borack *et al.*; an ethyl-methanesulfonate-induced inactive allele, Had^{m1} , described by Tobler and Grell.

hair bristles: see *ha*

Hairless: see *H*

hairy: see *h*

Hairy wing: see *Hw* under **ASC**

half out: see *hat*

halfway: see *hfw*

Haltere mimic: see *Cbx^{Hm}* under **BXC**

Hang-glider: see *Hg*

hap: hapless

location: 1-50.2.

origin: Induced by ethyl methanesulfonate.

references: Eberle and Hilliker, 1988, *Genetics* 88: 109-120.

phenotype: The embryonic lethal phenotype is characterized by an undispersed central yolk plug, no visible Malpighian tubules, unsclerotized mouthparts, and poorly visible cuticle.

cytology: Located in 12B9-13F.

Haplo-diplo lethal: see *Hdl*

haplo-female sterile: see *Fs(1)10A*

hat: half out

location: 3-{47}.

references: Cavener, Otteson, and Kaufman, 1986, *Genetics* 114: 111-23.

phenotype: Pharate adults appear morphologically normal, but only partially eclose. Heads and thoraxes may emerge, but fail to complete emergence. Not complemented by *T(2;3)Ta¹*.

alleles:

alleles	origin	discoverer	synonym
<i>hat¹</i>	EMS	Denell	<i>l(3)d1</i>
<i>hat²</i>	EMS	Kaufman	<i>l(3)k1</i>
<i>hat³</i>	EMS	Kaufman	<i>l(3)k13</i>
<i>hat⁴</i>	EMS	R. Lewis	<i>l(3)r8</i>

cytology: Located in 84C1-2 since not complemented by *T(2;3)Ta¹ = T(2;3)51E1-2;84C1-2*. Unlike *stk*, *hat* is not included in *Df(3R)Scx2 = Df(3R)84A4-5;84C1-2*.

hau: haunted

location: 3-48.4.

origin: Induced by ethyl methanesulfonate.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95 (fig.).

phenotype: Homozygous lethal; only head skeleton visible in homozygous embryo; no differentiation of cuticle.

alleles: Four.

hay: haywire

location: 3-34.4.

origin: Induced by ethyl methanesulfonate

synonym: *ms(3)nc2*.

references: Fuller, 1986, *Proc. Soc. Dev. Biol.* 74: 19-41. Regan and Fuller, 1988, *Genes and Development* 2: 82-92.

phenotype: The original allele, *hay^{nc2}*, is homozygous male sterile; some derivative alleles are recessive lethal. Homozygous *hay^{nc2}* males display defects in meiosis, flagellar elongation, and nuclear shaping. *hay^{nc2}* also

male sterile in *trans* heterozygotes with recessive male-sterile alleles of β *Tub85D* or with *wrl^{nc4}* suggesting a role for the haywire product in microtubule function. Deficiencies of *hay* are fertile in heterozygous combination with the above interacting mutations, indicating that the extragenic failure *hay^{nc2}* to complement is based on a poison product mechanism. This feature was used to select alleles (*hay^{nc2rv1-8}*) that revert the failure of *hay^{nc2}* to complement $\beta 2t^n$ (= β *Tub85Dⁿ*).

alleles:

allele	origin	ref ^α	phenotype ^β
<i>hay^{nc2}</i>	EMS	1	a
<i>hay^{nc2rv1}</i>	EMS	2	b
<i>hay^{nc2rv2}</i>	EMS	2	b
<i>hay^{nc2rv3}</i>	EMS	2	b
<i>hay^{nc2rv4}</i>	EMS	2	b
<i>hay^{nc2rv5}</i>	EMS	2	c
<i>hay^{nc2rv6}</i>	EMS	2	b
<i>hay^{nc2rv7}</i>	EMS	2	d

^α 1 = Regan and Fuller, 1988, *Genes and Development* 2: 82-92; 2 = Regan and Fuller, 1990.

^β a = Recessive male sterile with defects in meiosis, flagellar elongation and nuclear shaping during spermatogenesis. Reduced female fertility. Homozygous viable at 25° but semi-lethal at 28°. Dominant enhancer of tubulin mutations; b = Recessive larval lethal; c = Recessive zygotic lethal, and dominant maternal effect embryonic semi-lethal; d = Recessive male sterile.

cytology: Located in 67E-F by Regan and Fuller (1988) on basis of its being uncovered by *Df(3L)lxd6 = Df(3L)67E1-2;68C1-2* and *Df(3L)lxd15 = Df(3L)67E;68C10-15* but not by *Df(3L)vin2 = Df(3L)67F2-3;68D6*. Also uncovered by *Df(3L)E(z)1R1* and *Df(3L)E(z)1R4* (Mounkes, Jones, and Fuller).

molecular biology: *hay* is the next transcription unit distal to *E(z)*, and encodes a 2.6 kb message present in embryos, larvae, pupae and adults (Mounkes, Jones, and Fuller).

hb: hunchback

location: 3-48 (distal to *p*).

synonym: *Rg-pbx*.

references: Lewis, 1968, *Proc. Int. Congr. Genet.* 12th 2: 96-7.

Nüsslein-Volhard and Wieschaus, 1980, *Nature* 287: 795-801.

Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95.

Bender, Turner, and Kaufman, 1987, *Dev. Biol.* 119: 418-32.

Lehmann and Nüsslein-Volhard, 1987, *Dev. Biol.* 119: 402-17.

Tautz, Lehmann, Schürich, Schuh, Seifert, Kienlin, Jones, and Jäckle, 1987, *Nature* 327: 383-89.

Bender, Horikami, Cribbs, and Kaufman, 1988, *Dev. Genet.* 9: 715-32.

Hülkamp Schröder Pfeifle Jäckle and Tautz 1989 *Nature* 338: 629-32.

Irish, Lehmann, and Akam, 1989, *Nature* 338: 646-48.

Struhl, 1989, *Nature* 338: 741-44.

Stanojević, Hoey, and Levine, 1989 *Nature* 41: 331-35.

Treisman and Desplan, 1989, *Nature* 41: 335-37.

phenotype: Homozygotes for null alleles of *hb* (class I alleles of Lehmann and Nüsslein-Volhard) are embryonic lethals of the gap type. Gastrulation abnormal; no cephalic fold; cell death evident at 6 hr later becoming

extensive, predominantly in the neuroectoderm; germ band extension curtailed at 50% of embryonic length. After germ band shortening embryos lack thoracic and labial segments; cephalopharyngeal skeleton present but poorly formed; head involution fails. Seventh and eighth abdominal segments fused by the deletion of parasegment 13; A1 segment 1.5 times normal width, with eight to ten deranged denticle rows compared to the normal number of four, and a widened region of naked cuticle. Filzkörper material reduced; posterior spiracles fail to evert. Three ventral ganglia absent; gap appears between suboesophageal region of ventral nerve cord and more posterior trunk ganglia. Extreme mutants display a reduced number of stripes of *ftz* expression at cellular blastoderm; the first stripe is widened and followed by a narrowed gap of nonexpression preceding the second stripe; the last pair of stripes are fused (Carroll and Scott, 1986, Cell 45: 113-26). Hypomorphic alleles display variably less severe disruption depending on allele ($hb^{Drv6} = hb^{b2} = hb^{e21} > hb^{b7} > hb^{Drv9}$), the least severe, hb^{Drv9} lacking only T2. Class II alleles (Lehmann and Nüsslein-Volhard) resemble the null alleles except that some or all of the prothorax and A7 are retained. The class III allele retains the labial segment as well. Class IV alleles lack only the mesothoracic segment. Class V mutants exhibit segment transformations as well as gaps and are described separately below. Temperature sensitive period of hb^{ts1} during first four hr of development. $hb/+$ offspring produced from homozygous oogenic clones develop normally; homozygous embryos resulting from such clones display enhanced zygotic phenotype; gnathal, thoracic, and the first three abdominal segments replaced by two or three segments of abdominal identity in mirror image relation to the more posterior abdominal segments; weak alleles without maternal effect; extra doses of hb^+ in female without effect on phenotype of hb offspring. The anterior zone of hb expression extended posteriorly by six additional cells in the absence of Kr^+ ; conversely the zone of Kr expression expanded anteriorly by six to eight cells in hb mutants; posterior zone appears insensitive to Kr constitution (Jäckle, Tautz, Schuh, Seifert, and Lehmann, 1986, Nature 324: 668-70). hb^+ appears to set the boundaries of *Ubx* expression (White and Lehmann, 1986, Cell 47: 311-21); zone of *Ubx* expression expanded in both anterior and posterior directions in hb mutant embryos at the stage of full germ band elongation; segmental disposition of expression characteristically deranged prior to the advent of cell death. Although *Ubx* expression in the ventral nerve chord at the stage of fully shortened germ band extends from parasegments 5-13, *Ubx* protein detected in parasegments 1, 7-12 and 14 in hb^{12} , 3 and 7-14 in hb^1 , and head to parasegment 1 plus parasegments 7-12 and 14 in hb^7 (White and Lehmann). Phenotypic effects of *ftz* and hb in double mutants additive in thorax and anterior abdomen, but more severe than expected in head and posterior regions.

alleles: hb^{Drv} alleles derived from hb^{Dt} .

allele	origin	discoverer	synonym	ref ^α	comments
hb 1	EMS		<i>hb^{IIU}</i>	3, 5	class III
hb 2	EMS		<i>hb^{6N}</i>	2, 3	class I
hb 3	EMS		<i>hb^{7L}</i>	2, 3	class II
hb 4	EMS		<i>hb^{7M}</i>	2, 3	class I

allele	origin	discoverer	synonym	ref ^α	comments
hb 5	EMS		<i>hb^{7O}</i>	2, 3	class II
hb 6	EMS		<i>hb^{9K49}</i>	2, 3	class V β
hb 7	EMS		<i>hb^{9K57}</i>	2, 3	class V β
hb 8	EMS		<i>hb^{9Q}</i>	2, 3	class I
hb 9	EMS		<i>hb^{9R}</i>	2, 3	class IV
hb 10	EMS		<i>hb^{11C}</i>	2, 3	class II
hb 11	EMS		<i>hb^{14C}</i>	2, 3	class V β
hb 12	EMS		<i>hb^{14F}</i>	2, 3	class I
hb 13	EMS		<i>hb³⁴⁹</i>	2, 3	class II
hb 14	X ray		<i>hb^{FFE}</i>	2, 3	class I
hb 15	X ray	Lehmann	<i>hb^{FB92}</i>	3	class I
hb b1		Bender	<i>Rg-pbx^{b1}</i>	1	amorph
hb b2		Bender	<i>Rg-pbx^{b2}</i>	1	hypomorph
hb b7		Bender	<i>Rg-pbx^{b7}</i>	1	hypomorph
hb b16		Bender	<i>Rg-pbx^{b16}</i>	1	amorph
hb b50		Bender	<i>Rg-pbx^{b50}</i>	1	amorph
hb bs23		Bender	<i>Rg-pbx^{bs23}</i>	1	amorph β
hb D1	EMS	Bacher	<i>Rg-pbx</i>	4	viable β
hb D2	γ ray	Groger			lethal β
hb Drv1	γ ray	Lewis	<i>Rg-pbx +RG1</i>	1, 3	class II
hb Drv2	γ ray	Lewis	<i>Rg-pbx +RG2</i>	1, 3	class II
hb Drv3	γ ray	Lewis	<i>Rg-pbx +RG3</i>	1	
hb Drv4	EMS	Bender	<i>Rg-pbx +RE1</i>	1	
hb Drv5	EMS	Bender	<i>Rg-pbx +RE2</i>	1	
hb Drv6	EMS	Bender	<i>Rg-pbx +RE3</i>	1	
hb Drv7	EMS	Bender	<i>Rg-pbx +RE4</i>	1	
hb Drv8	EMS	Bender	<i>Rg-pbx +RE5</i>	1	
hb Drv9	EMS	Bender	<i>Rg-pbx +RE6</i>	1	hypomorph
hb Drv10	EMS	Bender	<i>Rg-pbx +RE7</i>	1	
hb Drv11	EMS	Bender	<i>Rg-pbx +RE9</i>	1	hypomorph
hb e21			<i>Rg-pbx^{e21}</i>	1	hypomorph
hb ts1			<i>Rg-pbx^{ts1}</i>	1	

α 1 = Bender, Tumer, and Kaufman, 1987, Dev. Biol. 119: 418-32 (fig.); 2 = Jürgens, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95 (fig.); 3 = Lehmann and Nüsslein-Volhard, 1987, Dev. Biol. 119: 402-17 (fig.); 4 = Lewis, 1968, Int. Congr. Genet. 12th 2: 265-70; 5 = Nüsslein-Volhard and Wieschaus, 1980, Nature 287: 795-801; 6 = Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95 (fig.).

β Described more fully below.

cytology: Placed by cytological mapping in 85A3-B1 (Tautz *et al.*, 1987) or in 85A5-7;85B2-3 (Bender *et al.*, 1987; Lehmann, and Nüsslein-Volhard, 1987).

molecular biology: Genomic sequences that include the gene *hb* cloned by chromosome walking (Tautz *et al.*, 1987; Bender *et al.*, 1988); gene also sequenced. Two embryonic mRNAs were identified (Tautz *et al.*, 1987), one of 2.9 kb and another of 3.2 kb; the larger is present in the maternal RNA and persists during the first eight hours of embryonic development, whereas the smaller is detected only between the second and sixth hours. The two primary transcripts are transcribed from the same strand and have identical exons; the 3.2 kb mRNA has a 5' exon of 500 nucleotides separated from exon 2 by a 3.2 kb intron, and is separated from the 3' exon by a short intron; the same splice acceptor site is utilized in both messages. The experiments of Bender *et al.* (1988) identified five embryonic transcripts in two discrete classes; one class includes transcripts of 2.6 and 2.8 kb that are expressed only during embryogenesis with their highest level at 2-4 hr; and the other class includes transcripts of 3.0, 3.2, and 3.5 kb that accumulate to their highest levels over the first eight hours of embryogenesis, but are also present in adult females and males. The 5' to 3' direction of transcription of all five transcripts was found to be from distal to proximal along the chromosome. Primer extension and S1 protection experiments

indicate the presence of two distinct promoters and three polyadenylation sites; transcripts from the upstream promoter are found in adults (males and females) as well as in 0-12 hr embryos (Bender *et al.*, 1988). Maternal message is uniformly distributed at the time of egg deposition, but in stage 8 embryos, a gradient decreasing posterior is visible (Foe and Alberts, 1983, *J. Cell Sci.* 61: 31-70). Zygotic message first seen in stage 11 in the anterior 45% and the posterior 25% of the embryo; message subsequently disappears from the two poles of the embryo and becomes distributed in three and then two stripes in the anterior region; in general, the regions of *hb* message do not overlap those of *Kr* message. An overlap in protein distribution is shown by *hb* and *Kr* (Gaul and Jäckle, 1989, *Development* 107: 651-62). The proteins encoded by *hb* (and *Kr*) show sequence-specific DNA binding to sites upstream of the two *hb* promoters (Stanojević *et al.*, 1989; Treisman and Desplan, 1989). The posterior-group gene *nos* suppresses the activity of *hb* in the posterior half of the body; if both *nos* and *hb* are absent, the embryo is normal (Hülskamp *et al.*, 1989; Struhl, 1989).

***hb*⁶**

phenotype: *hb*⁶/*hb*⁶ embryos lack meso- and metathorax, but nine abdominal segments are formed, the most anterior being T2 transformed into A1 and the next the normal A1.

***hb*⁷**

phenotype: Homozygous embryos lack labium and all thoracic segments; head and gnathal segments transformed into posterior abdominal segments as is A1. Expressed only in homozygotes, not in hemizygotes; *hb*⁷/*Df(3R)hb* displays class III phenotype. Lethality of *hb*⁷ homozygotes not rescued by *Dp(3;Y)P92* which is able to cover the other alleles; attempts to implicate a linked lethal mutation negative.

***hb*¹¹**

phenotype: As in the case of *hb*⁷, resembles a class I mutant, but with transformation of gnathal and first abdominal segments into posterior abdominal segments. Expression in homozygotes more extreme than in hemizygotes.

***hb*^{D1}: hunchback-Dominant**

phenotype: A gain-of-function mutation; viable both in heterozygous and homozygous condition. Phenotype resembles that of *pbx*; insensitive to additional doses of *hb*⁺ but suppressed by extra doses [e. g., five copies of *BXC*⁺ (Lewis)]; enhanced in heterozygous combination with null alleles of *ftz*.

cytology: Associated with *In(3R)hb*^{D1} = *In(3R)85B;88C*. The inversion is lethal homozygous, but lethality covered by duplication for 88C breakpoint; *hb*^{D1} associated with proximal breakpoint.

***hb*^{D2}**

phenotype: Homozygous lethal; lethal when heterozygous to *hb* null alleles (e.g. *hb*¹²). Has two dominant phenotypes: 1) homeotic transformation of parasegment six to parasegment five, resembling that produced by *bx* and *pbx*; 2) a pair-rule segmentation defect, consisting of partial deletion of even-numbered abdominal segments, principally A2 and A4. Homozygote shows more extreme

expression of both phenotypes; penetrance and expressivity of first effect enhanced in double heterozygous combination with null alleles of *ftz* (e.g. *hb*^{D2}/*ftz*⁷¹⁴); second phenotype enhanced by *Df(2R)eve*, such that only a few adult escapers of the doubly heterozygous genotype are observed. Also has a recessive phenotype, revealed either when homozygous or heterozygous to an *hb* null allele; deletion of parasegment 13 and reduction of filzkörper; labial and thoracic segments normal. Thus, affects posterior, but not anterior, domain of *hb*⁺ function. Viable in *trans* to some hypomorphic alleles that do not affect parasegment 13 (e.g. *hb*⁶).

cytology: Associated with *In(3R)hb*^{D2} = *In(3R)84B;85A*.

Breakpoint of associated inversion approximately 1 kb upstream of transcription start for 3.2 kb transcript.

Hdl: Haplo-diplo lethal

location: 1-[43].

references: Merriam, Yamamoto, Stewart, Rahman, and Nicolau, 1986, unpublished.

phenotype: Females with one dose of the normal allele and males with two are almost completely lethal; escapers normal in phenotype.

alleles: No mutant alleles known.

cytology: Localized to 12A by segmental aneuploidy.

hdp: heldup

At least three different genetic entities appear to have been designated *hdp* on the basis of similarity of phenotype and map position to the now-lost *hdp* of Fahmy (1958, *DIS* 32: 70). Deak (1977, *J. Embryol. Exp. Morphol.* 40: 35-63) so designated *wup-A* of Hotta and Benzer [1972, *Nature* (London 240: 527-35)]. Lifschytz and Green (1979, *Mol. Gen. Genet.* 171: 153-59) selected reversions and suppressors of *Bx*, both of which had a held-up phenotype and were so designated. Engels and Preston (1981, *Cell* 26: 421-28) selected P-factor induced sex-linked mutants with held-up wings; 93% are associated with chromosome rearrangements with one breakpoint in 17C2-3; the other 7% are not associated with rearrangements and act as suppressors of *Bx*; the latter group of mutants complement both the other 93% of mutants recovered by Engels and Preston and those described by Deak. We resurrect the name *wupA*: wingsupA for the mutants studied by Deak; we designate the *Bx* suppressors *hdp-a* and the mutants associated with 17C2-3 breakpoints *hdp-b*. In addition Fahmy (1956, *DIS* 32: 74) describes *rwg*: reduced wings, a mutant with the same map position and a slightly different phenotype from other held-up-like mutants; we arbitrarily designate it an *hdp-a* allele.

hdp-a

location: 1-59.4 (0.0045 unit to the left of *Bx* based on separation of *Bx*³ and *hdp-a*).

synonym: *hld*: heldwing (Deak, Bellany, Bienz, Dubuis, Fenner, Gollin, Rähmi, Ramp, Reinhardt, and Cotton, 1982, *J. Embryol. Exp. Morphol.* 69: 61-81); *ftiH* (Homyk, 1977, *Genetics* 87: 105-28; Homyk and Shepard, 1977, *Genetics* 87: 95-104).

phenotype: Wings held up to various degrees; may overlap wild type. *hdp-a* alleles act as dominant suppressors of *Bx* in either *cis* or *trans* (Lifschytz and Green, 1979, *Mol. Gen. Genet.* 171: 153-59). Lifschytz and Green consider *hdp-a* mutants to be hypomorphic or amorphic alleles of

a gene that is under *cis* control of Bx^+ ; Bx mutants cause overproduction $hdp-a^+$ product.

alleles:

allele	synonym	origin ^α	selected as β	ref ^γ	male fertility
* <i>hdp-a</i> ¹	<i>hdp</i>	CB3007		2	+
<i>hdp-a</i> ²	<i>RB6</i>	X ray	Bx^r	4	-
<i>hdp-a</i> ³	<i>RB7</i>	X ray	Bx^r	4	-
<i>hdp-a</i> ⁴	<i>RB13</i>	X ray	Bx^r	4	+
<i>hdp-a</i> ⁵	<i>RBE1</i>	EMS	Bx^r	4	-
<i>hdp-a</i> ⁶	<i>SBM1</i>	X ray	$Su(Bx^3)$	4	-
<i>hdp-a</i> ^{102δ}	<i>flH</i>	EMS		3	-
<i>hdp-a</i> ^{ϕ1ε}	<i>hdp</i>	P factor		1	+
* <i>hdp-a</i> ^{rwg}	<i>rwg</i>	CB1506		2	-

^α CB1506 = 2-chloroethyl methanesulfonate; CB3007 = DL-*p*-N,N-di-(2-chloroethyl) aminophenylalanine

^β Bx^1 = revertant of Bx^3 ; $Su(Bx^3)$ recovered as a trans suppressor of Bx^3 .

^γ 1 = Engels and Preston, 1981, Cell 26: 421-28; 2 = Fahmy, 1958, DIS 32: 67-77; 3 = Homyk and Emerson, 1988, Genetics 119: 105-21; 4 = Lifschytz and Green, 1979, Mol. Gen. Genet. 171: 153-59.

^δ Shows partial complementation over *hdp-a*².

^ε Representative of 7% of the *hdp*-like mutants spontaneously generated by hybrid dysgenesis in an X chromosome that carries a P factor inserted at 17C2-3. These alleles associated with P-factor excision.

cytology: Placed in 17C2-3 on the basis of perfect correlation between *hdp-a* mutation in a dysgenic cross and the loss of a P element from 17C2-3 (Engels and Preston, 1981, Cell 26: 421-28).

other information: Relation between *hdp-a* and *hdp-b* unclear, they are in the same polytene bands but complement each other completely. They are separated by Bx (Mattox).

hdp-b

location: 1-59.4 (inferred from polytene position).

origin: Induced by hybrid-dysgenesis in an X chromosome with a P-factor in 17C2-3.

references: Engels and Preston, 1981, Cell 26: 421-28.

phenotype: Same as *hdp-a*, except that there is no interaction with Bx .

alleles: 93% of the hybrid-dysgenesis-induced *hdp*-like mutants in an X chromosome in which a P factor resides at 17C2-3 are *hdp-b* alleles.

cytology: All dysgenesis induced *hdp-b* alleles, are associated with rearrangements with one breakpoint in 17C2-3.

hdp: see *wupA*

Heat-shock cognate: see *Hsc*

Heat-shock protein: see *Hsp*

Heat-shock RNA at 93D: see *Hsr93D*

Heat-shock RNA α β: see *Hsr* α β

heavy-vein: see *hv*

hedgehog: see *hh*

heldout: see *dpp*^{ho}

heldup: see *hdp*

heldwing: see *hpd-a*

Henna: see *Hn*

her: hermaphrodite (G.S. Carson)

location: 2-52.9 (between *b* and *pr*).

origin: Induced by ethyl methanesulfonate.

references: Baker and Belote, 1983, Ann. Rev. Genet. 17: 345-93.

phenotype: Original allele was recovered as a recessive temperature-sensitive lethal with low homozygous viability (3-7% of the viability *her/+*) at 29° C and near-normal viability at 18° C in both sexes. Surviving 29° C homozygotes have small bodies, rough eyes, incised wings, and etched abdominal tergites. XX homozygotes surviving at 29° C are intersexual, with partially formed sexcombs, male-colored abdominal patches, and rudimentary male and female external genitalia. XY escapers have female-like sixth abdominal sternite bristles, but otherwise appear sexually normal. *her/her* flies of both sexes raised at 18° C are morphologically normal and fertile; however, XX homozygotes exhibit reversible temperature-dependent sterility when mated at 29° C. Homozygous females exert a mild maternal effect upon the sexual phenotype of *her/+* offspring which develop at 29° C; females show abnormal basitarsal bristle arrangement and patches of male abdominal pigmentation; males show female basitarsal bristles among sexcomb teeth and the presence of sixth sternite bristles.

alleles: *her*², spontaneous on *In(2LR)SM1,Cy*.

cytology: Placed between 36B4 and 36C2-4 as a result of its complementation by *Dp(2;Y)B106* and *Dp(2;Y)B108* = *Dp(2;Y)35F;36DE*, *Dp(2;Y)H1* = *Dp(2;Y)36B4;40F* + *Df(2L)37F4;39C2* and *Dp(2;Y)H3* = *Dp(2;Y)36B4;40F* + *Dp(2L)38B2;39E2* and its exclusion from *Df(2L)TW137* = *Df(2;Y)36C2-4;37B9-C1* and *Dp(2L)TW50* = *Df(2;Y)36E4-F1;38A6-7*.

molecular biology: Tritiated uridine incorporation in third instar salivary gland chromosomes suggests X chromosome transcriptional activity is reduced in *her/her* larvae compared with heterozygous siblings.

Hermaphrodite: see *dsx*^D

heterochromatic-recombination-inducer: see *hir*

Hex-3: see *Hex-C*

Hex-A: Hexokinase A

location: 1-29.2.

synonym: *Hex-A,B*.

references: Voelker, Langley, Leigh Brown, and Ohnishi, 1978, DIS 53: 200.

Moser, Johnson, and Lee, 1980, J. Biol. Chem. 255: 4673-79.

phenotype: The structural gene for hexokinases A, B1, and B2 (EC 2.7.1.1). The enzyme is monomeric and of molecular weight 47,000 ± 3000 for HEX-A and 48,000 ± 3000 for HEX-B1 and HEX-B2. The three forms of the enzyme are immunologically identical but exhibit pI values of 5.1, 5.3, and 5.5 respectively upon isoelectric focusing, and these values covary in allelic variants. All three forms are found in the adult, but HEX-A is absent from larval preparations; HEX-A mainly localized in adult flight muscle. Three forms modified products of same structural gene. For purification and biochemical characterization see Moser *et al.*

alleles: Electrophoretic variants *Hex-A*² which migrates more slowly than *Hex-A*⁴.

cytology: Localized to 8D4-E1 (Voelker).

Hex-C

location: 2-74.5 (Mukai and Voelker, 1977, Genetics 86: 175-85).

synonym: *Fk*: Fructokinase; *Hex-3*.

references: Jelnes, 1971, Hereditas 67: 291-93.

Fox and Madhavan, 1971, DIS 46: 42.

Madhavan, Fox, and Ursprung, 1972, J. Insect. Physiol. 18: 1523-30.

Moser, Johnson, and Lee, 1980, J. Biol. Chem. 255: 4673-79.

phenotype: The structural gene for hexokinase-C [HEX-C (EC 2.7.1.1)]. The enzyme is a monomer of estimated molecular weight 42,000 ± 3,000 (Moser *et al.*) or 35,000 (Leigh Brown in Racine, Langley, and Voelker, 1980, Environ. Mutagen. 2: 167-77). Activity present in all developmental stages except freshly laid eggs; restricted to fat body in larvae, but present in many organs in adult. Weakly active or inactive in cell line (Deber, 1974, Wilhelm Roux's Arch. Entwicklungsmech. Org. 174: 1-9). HEX-C activity not necessary for survival; null mutants viable (Burkhart, Montgomery, Langley and Voelker, 1984, Genetics 107: 295-306). Purification and biochemical characterization of enzyme described by Moser *et al.*.

alleles:

allele	origin	synonym	ref ^α	comments
<i>Hex-C²</i>		<i>Hex-C^S</i>	1, 3	natural polymorphism
<i>Hex-C^{2.5}</i>	spont	<i>Hex-C^{AW399}</i>	4	<i>Hex-C⁴</i> derivative on <i>SM1</i>
<i>Hex-C⁴</i>		<i>Hex-C^I</i>	1, 3	natural polymorphism
<i>Hex-C⁶</i>		<i>Hex-C^F</i>	1, 3	natural polymorphism
<i>Hex-C^{nGB1}</i>	spont		2	natural population
<i>Hex-C^{nMC1}</i>	spont	<i>Hwx-C^{nJH302 β}</i>	4	<i>Hex-C⁴</i> derivative on <i>SM1</i>
<i>Hex-C^{nNC1}</i>	spont		6	natural population
<i>Hex-C^{nNC2}</i>	spont		7	<i>Hex-C⁴</i> derivative on <i>SM1</i>
<i>Hex-C^{nR1}</i>	γ ray	<i>Hex-C^{n γ50036}</i>	5	<i>Hex-C⁴</i> derivative on <i>SM1</i>

^α 1 = Fox and Madhavan, 1971, DIS 46: 42; 2 = Langley, Voelker, Leigh Brown, Ohnishi, Dickson and Montgomery, 1981, Genetics 99: 151-56; 3 = Madhavan, Fox, and Ursprung, 1972, J. Insect. Physiol. 18: 1523-30; 4 = Mukai and Cockerham, 1977, Proc. Nat. Acad. Sci. USA 74: 2514-17; 5 = Racine, Langley, and Voelker, 1980, Environ. Mutagen. 2: 167-77; 6 = Voelker, Langley, Leigh Brown, Ohnishi, Dickson, Montgomery, and Smith, 1980, Proc. Nat. Acad. Sci. USA 77: 1091-95; 7 = Voelker, Schaffer, and Mukai, 1980, Genetics 94: 961-68.

^β *Hex-C^{JH301}*, *Hex-C^{JH302}*, *Hex-C^{JH303}*, and *Hex-C^{JH309}* probably independent recoveries of descendents of a single mutation.

cytology: Placed in 51B-52E (Burkhart, Montgomery, Langley, and Voelker, 1984, Genetics 107: 295-306).

hfs: see *Fs(1)10A*

hfw: halfway

location: 1-0.4 [0.008 map units to right of *dor* (Aizenzon and Belyaeva, 1982, DIS 58: 3-7)].

phenotype: Recessive lethal; for some alleles, development arrested in third instar, the anterior half initiating puparium formation while the posterior half remains larval. Extent of development influenced by administration of ecdysone and by temperature; pupariation occasionally completed at 18°. Some alleles yield a few survivors with characteristic wing abnormalities and occasionally swollen abdomens.

alleles: Seven alleles, superscripted 1-7.

allele	origin	synonym	ref ^α	comments
<i>hfw¹</i>			3	lethal
<i>hfw²</i>	EMS	<i>swiⁱ³²</i>	1	lethal
<i>hfw³</i>	EMS	<i>swiⁱ⁶³</i>	1	lethal
<i>hfw⁴</i>	EMS	<i>swiⁱ²⁰⁰</i>	1	lethal
<i>hfw⁵</i>	EMS	<i>swiⁱ²¹⁹</i>	1	rare survivors
<i>hfw⁶</i>	EMS	<i>swiⁱ²⁵¹</i>	1	rare survivors
<i>hfw^{7β}</i>	EMS	<i>swiⁱ⁴⁶⁷</i>	1, 2	lethal as late prepupae

^α 1 = Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelova, and Zhimulev, 1980, Chromosome 81: 281-306; 2 = Belyaeva and Zhimulev, 1986, Chromosome 86: 151-63; 3 = Rayle, 1967, Genetics 56: 583.

^β No late larval or pupal puffs in polytene chromosomes of homozygotes (Belyaeva and Zhimulev, 1986).

cytology: Inseparable from *dor*, which has been placed in 2B11-12 by Lefevre [1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 31-66].

Hg: Hang glider (M. Ashburner)

location: 2-57.5 (no recombinants with *on* among 1532 flies).

origin: Induced by ethyl methanesulfonate.

discoverer: Detwiler.

phenotype: Wings upheld and spread from body; wing blade not flat but arced.

hh: hedgehog

location: 3-81.

references: Nüsslein-Volhard and Wieschaus, 1980, Nature 287: 795-801.

Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

Mohler and Wieschaus, 1985, Genetics 110: s35.

Mohler, 1988, Genetics 120: 1061-72.

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-69.

phenotype: A segment polarity type of embryonic lethal. Homozygous embryos have the posterior naked portion of the ventral surface of each segment deleted and replaced by a mirror image of the anterior denticle belts. Embryos appear to lack segmental boundaries. In strong alleles, there is no obvious segmentation; the larvae are approximately 40% the length of the wild-type larvae, and there is a lawn of denticles arranged in a number of whorls on the ventral surface as a result of loss of naked cuticle. In intermediate alleles, naked cuticle is also lost from the ventral region, but the lawn of denticles is arranged in segmental arrays in mirror-image symmetry. The weak alleles show fusions that delete the naked cuticle usually between abdominal segments 1 and 2 and 6, 7, and 8 (Mohler, 1988). Temperature shift experiments with a temperature-sensitive allele (viable and normal at 18°, and mutant at 25°) indicate two phases of *hh* activity at 25°, the first during early embryogenesis (3-6 hr of development) and the second during the late larval and early pupal stages (4-7 days of development).

alleles:

allele	origin	synonym	ref ^α	comments
<i>hh¹</i>	spont	<i>bar-3</i>	1	viable hypomorph
<i>hh²</i>	EMS	<i>hh^{6L}</i>	4	weak
<i>hh³</i>	EMS	<i>hh^{6N}</i>	2-4	strong; ts
<i>hh⁴</i>	EMS	<i>hh^{9K}</i>	2-4	weak; ts

allele	origin	synonym	ref ^α	comments
hh 5	EMS	<i>hh</i> ^{10B}	2-4	weak
hh 6	EMS	<i>hh</i> ^{11C}	4	
hh 7	EMS	<i>hh</i> ^{11K}	3	
hh 8	EMS	<i>hh</i> ^{13C}	2-4	strong
hh 9	EMS	<i>hh</i> ^{13E}	2-4	intermediate
hh 10	γ ray	<i>hh</i> ^{GR1}	3	<i>In(3R)81F;94D10-E5;</i> strong
hh 11	γ ray	<i>hh</i> ^{GS1}	3	<i>In(3R)92B4-11;94D10-E5;</i> strong
hh 12	γ ray	<i>hh</i> ^{GS2}	3	
hh 13	γ ray	<i>hh</i> ^{GW1}	3	
hh 14	γ ray	<i>hh</i> ^{GW2}	3	
hh 15	HD	<i>hh</i> ^{HL1}	3	<i>Tp(3R)85D;94D10-E5;97E7-8;</i> strong
hh 16	HD	<i>hh</i> ^{HL2}	3	
hh 17	HD	<i>hh</i> ^{HL3}	3	
hh 18	EMS	<i>hh</i> ^{IIK}	2-4	
hh 19	EMS	<i>hh</i> ^{IIO}	2-4	
hh 20	EMS	<i>hh</i> ^{IIX}	4	
hh 21	EMS	<i>hh</i> ^{IJ}	2-4	strong
hh 22	TEM	<i>hh</i> ^{TW1}	3	strong

^α 1 = Ives, 1950, DIS 24: 58. 2 = Jürgens, Wieschaus, and Nüsslein-Volhard, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95; 3 = Mohler, 1988, Genetics 120: 1061-72; 4 = Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-69.

cytology: Placed in 94E by segmental aneuploidy. Placed in 94D10-E5 based on breakpoint common to four *hh* rearrangements (Mohler, 1988).

hh1

phenotype: A weak hypomorphic allele that is not complemented by other *hh* alleles. Eye of homozygote small and narrow with about 150 facets. Eye disc size reduced; deep cleft at anterior edge cell; clusters at cleft look mature (Renfranz and Benzer, 1989, Dev. Biol. 136: 411-29).

***hi: high**

location: 2- (not located).

origin: Found in Florida natural population.

discoverer: Ives, 1943.

references: 1943, Genetics 28: 77.

1950, Evolution 4: 236-52.

phenotype: Male homozygous for *hi* produces sperm containing 10 times normal frequency of mutations. Heterozygous *hi/+* causes a mutation rate 2-7 times normal. Ratio of sex-linked lethal to visible mutations about 8 to 1. Inversions associated with about 5% of mutations. RK3.

cytology: Salivary chromosomes normal.

other information: Homozygous *hi* constructed by crossing two balanced lethal stocks, *11 hi/Cy X 12 hi/Cy*. Since these stocks have developed a common lethal, it is now difficult to obtain *hi* homozygotes.

***Hi: Hirsute**

location: 3-{rearrangement}.

origin: X ray induced.

discoverer: Bishop, 1939.

phenotype: All bristles except postscutellars and postdorsocentrals multiplied, especially on head and anterior thorax. Eyes smaller and facets irregular. Homozygous lethal. RK2A.

cytology: Associated with *In(3LR)Hi = In(3LR)71A;91F*.

other information: Possibly the same as *Brd*.

Hia: Hiatus

location: 2- (not located).

origin: Spontaneous.

discoverer: Bridges, 29b12.

phenotype: Terminal interruption of L2. More obvious in heterozygous male than in heterozygous female. Homozygous viable. RK3.

Hid: see *term*

high: see *hi*

Himca: see *Fs(2)Sz5*

Hin-d: see *dpp*

hindsight: see *hnt*

hir: heterochromatic-recombination-inducer

location: 2- (left end of 2L).

references: Hiraizumi, 1980, Genetics 94: s45-46.

phenotype: Females homozygous for *hir* in combination with certain cytoplasmic constitutions show increased recombination between *lt* and *Rsp*. Effect decreases with maternal age. *hir* without effect in males.

His: Histone

location: 2-55 [between *l(2)crc* and *lt*].

references: Pardue, Kedes, Weinberg, and Bernstiel, 1977, Chromosoma 63: 135-51.

Lifton, Goldberg, Karp, and Hogness, 1977, Cold Spring Harbor Symp. Quant. Biol. 42: 1047-51.

phenotype: *His* refers collectively to five structural genes (*His1*, *His2a*, *His2b*, *His3*, and *His4*) for the five different histones (H1, H2A, H2B, H3, and H4). H2A, H2B, H3 and H4 participate in equimolar quantities in the formation of histone octomers, which form the protein core of nucleosomes. H1 associates with DNA between nucleosomes. The ratio of H1 to nucleosome core histones is higher in the salivary glands of larvae than in the cells of young embryos (Holmgren, Johansson, Lambertsson, and Rasmusson, 1985, Chromosoma 93: 123-31). For primary structure of H2B see Elgin, Schilling, and Hood (1979, Biochemistry 18: 5679-85). The expression of the histone genes changes in mid-embryogenesis (Ambrosio and Shedl, 1985, Dev. Biol. 111: 220-31; Ruddell and Jacobs-Lorena, 1986, Proc. Nat. Acad. Sci. USA 82: 3316-19). The egg chambers contain a variable and low level of mRNA during nurse cell polytenization; however, at the end of stage 10, all the nurse cells accumulate histone mRNA which is turned over to the growing oocytes as the nurse cells degenerate. Heterozygosity for full or partial deficiency of the histone genes suppresses variegation (*B^{SV}*, *Sb^v*, *w^{md}*); duplications without effect on level of variegation (Moore, Sinclair, and Grigliatti, 1983, Genetics 105: 327-44). Transcription not repressed by heat shock (Spradling, Pardue, and Penman, 1977, J. Mol. Bio. 109: 559-87).

cytology: Localized to polytene bands 39D3 through 39E1-2 and possibly 39D2 as well by *in situ* hybridization of sea urchin histone messenger RNA (Pardue *et al.*). Orphon sequences homologous to *His2B* and *His3* detected outside the tandem array but not localized further (Childs, Maxson, Cohn, and Kedes, 1981, Cell 23: 651-63).

molecular biology: The five histone genes are deployed in the order *His1 His3 His4 His2a His2b* in a tandemly

repeating unit that is represented in 100-200 copies per haploid complement. Units of 5.0 and 4.75 kb are found depending on whether or not there is a 250 base-pair insertion in the spacer region between *His1* and *His3* (Matsuo and Yamazaki, 1989, *Nucleic Acids Research* 17: 225-38); the 5 kb units are present from one to four times more frequently than 4.8 kb units, depending on the particular strain (Strausbaugh and Weinberg, 1982, *Chromosoma* 85: 489-505). Magnification and compensation of histone gene sequences in *Df(2L)TW161* described by Chernyshev, Bashkirov, and Khesin (1980, *Mol. Gen. Genet.* 178: 663-68) and Chernyshev (1982, *Mol. Biol. Moscow* 16: 593-603). The repeating unit has been cloned, and the relative positions and directions of transcription of the five histone genes have been determined by Lifton *et al.* A 68 base-pair segment between *His3* and *His4* specifically binds the B transcription factor (Parker and Topol, 1984, *Cell* 36: 357-69). Sequence analysis by Goldberg (1979, PhD. Thesis, Stanford University) reveals the absence of introns. Among the first genes to be transcribed, transcription beginning 90 minutes after oviposition; transcription rates and message stability are high in early embryogenesis when the rate of DNA synthesis is maximal and both decrease markedly as development proceeds (Anderson and Lengyel, 1980, *Cell* 21: 717-27). When *Drosophila* tissue culture cells are heat shocked, H2B protein synthesis is increased, but H1, H2A, H3, and H4 protein synthesis is decreased (Farrell-Towt and Sanders, 1984, *Mol. Cell Biol.* 4: 2676-85). Histone messages non polyadenylated (Burkhardt and Birmstiel, 1978, *J. Mol. Biol.* 18: 61-79). Regular development of nucleosomes with respect to repeating sequence of histone genes studied by means of nuclease sensitivity (Samal, Worcel, Louis, and Schedl, 1981, *Cell* 23: 401-10). Bands, interbands, and puffs in the polytene chromosomes are recognized by a monoclonal antibody to an epitope in the carboxy-terminal tail of *His1* (Hill, Watt, Wilson, Fifis, Underwood, Tribbick, Geysen, and Thomas, 1989, *Chromosoma* 98: 411-21).

other information: One H2A-like sequence variant has been found (Donahue, Palmer, Condie, Sabatini, and Blumenfeld, 1986, *Proc. Nat. Acad. Sci. USA* 83: 4744-48). Within the H3 gene, the average nucleotide difference within a chromosome was 52% of that within a population (Matsuo and Yamazaki, 1989, *Genetics* 122: 87-97).

Hiv: see *L(1)7C*

Hirsute: see *Hi*

hk: hook

location: 2-53.9.

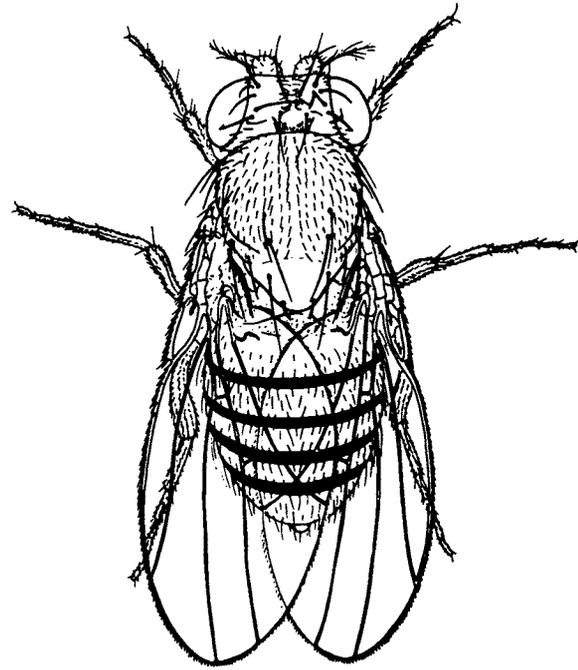
origin: Spontaneous.

discoverer: Mohr, 24a4.

references: 1927, *Hereditas* 9: 169-79 (fig.).

phenotype: Bristles nearly all hooked at tip or blunted, some bent at right angles. Scutellars and verticals especially affected. Acrostichal hairs fewer and outer rows separated. Eyes slightly roughened. Wings usually divergent and may be smaller. Body sometimes small and chunky. Less extreme expression at 19°, especially the wing character, but classification reliable. RK2.

alleles: *hk*² (Bridges, 33a31) spontaneous and less extreme than *hk*¹. *hk*¹²⁷ and *hk*¹³¹ X ray induced (Wright,



hk: hook

From Mohr, 1927, *Hereditas* 9: 169-79.

Hodgetts, and Sherald, 1976, *Genetics* 84: 267-85).

cytology: Located to 37B10-14 by deficiency analysis (Wright, Beermann, Marsh, Bishop, Seward, Black, Tomsett, and Wright, 1981, *Chromosoma* 83: 45-58). *Df(2L)130/Df(2L)203 = Df(2L)37B9-C1;37D1-2/Df(2L)36E4-F1;37B9-C1* survives and is *hk* in phenotype (Wright *et al.*).

Hk: Hyperkinetic (J.C. Hall)

location: 1-30.1.

references: Kaplan and Trout, 1969, *Genetics* 61: 399-409.

Ikeda and Kaplan, 1974, *Amer. Zool.* 14: 1055-66.

Thompson, 1977, *DIS* 52: 2.

Kaplan, 1979, *Psychological Survey* (Connolly, ed.).

Allen and Unwin, London, Vol. 2, pp. 90-109.

Stern and Ganetzky, 1989, *J. Neurogenet.* 5: 215-28.

phenotype: Isolated as a dominant allele that induces ether-induced leg shaking in flies (Kaplan and Trout, 1969); later, hyperkinetic alleles showed recessive behavior, both in regard to the adult leg shaking phenotype and to the larval electrophysiological phenotype (Stern and Ganetzky, 1989). The vigorous leg shaking of flies can be induced by nitrogen or triethylamine, as well as ether, but not by chloroform (Ganetzky and Wu, 1982, *Genetics* 100: 597-614); ether also induces rhythmic bursts of impulses in certain cells of the thoracic ganglia of adults (summarized by Ikeda and Kaplan, 1974). The "patch clamp" experiments on neurons from *Hk* larvae reveals inward currents of unusually high conductance (Sun and Wu, 1985, *Neurosci. Abstr.* 11: 787). In *Hk* larvae, the amplitude and duration of the post synaptic response to a brief high frequency nerve stimulation is increased up to the level characteristic of *Sh* mutants (Stern and Ganetzky, 1989). Shadow stimuli induce jump response, which maps to the head in mosaic experiment (Kaplan, 1979). *Hk* can overcome heat-induced paralytic

effects of *para*^{ts} (i.e. stobe light stimuli to the double mutant elicit jumps). *Hk*-induced leg shaking suppressed by *nap*^{ts} at its relatively low permissive temperature (Ganetsky and Wu, 1982). Courtship performed by *Hk* males is abnormal (Burnet, Eastwood, and Connolly, 1974, *Behav. Genet.* 4: 227-35). Lifespan is shorter than normal and rate of oxygen consumption is greater than normal in *Hk* (Trout and Kaplan, 1970, *Exper. Geront.* 5: 83-93). Mutant focus of life-shortening effect maps to ventral anterior part of thorax as does leg shaking (Trout and Kaplan, 1982). Mutant shows weak orientation to spots in Y-maze test (Bulthoff, 1982, *Biol. Cybernet* 45: 63-7).

alleles:

allele	origin	discoverer	synonym	ref ^β
<i>Hk</i> ¹	EMS	Pasternak	<i>Hk</i> ^{1P}	1, 2
<i>Hk</i> ²	EMS	Trout	<i>Hk</i> ^{2T}	1
<i>Hk</i> ³	spont	Parris, 1973	<i>Hk</i> ⁷³	3

^α In addition to the alleles listed in the table, γ ray-induced alleles have been isolated by Schlimgen on the basis of their failure to complement the leg shaking of *Hk*¹ (Stern and Ganetzky, 1989).

^β 1 = Kaplan and Trout, 1969, *Genetics* 61: 399-409. 2 = Stern and Ganetzky, 1989, *J. Neurogenet.* 5: 215-28. 3 = Thompson, 1977, *DIS* 52: 2.

cytology: Located in 9A-C since included in *Df(1)Hk* = *Df(1)9A;9C* (Stern and Ganetzky, 1989).

hl: see *bx*^{hl}

hld: see *hdp-a*

Hm: see *Cbx*^{Hm} under *BSC*

Hmg: Hmg-CoA-reductase

location: 3-{81}.

references: Gertler, Chiu, Richter-Mann, and Chin, 1988, *Mol. Cell Biol.* 8: 2713-21.

phenotype: Encodes 3-methylglutaryl coenzyme A (HMG CoA) reductase, the rate-controlling enzyme for cholesterol synthesis in mammals. In *Drosophila melanogaster*, the enzyme is not regulated by sterols (Brown, Havel, and Watson, 1983, *J. Biol. Chem.* 258: 8512-15; Silberkang, Havel, Friend, McCarthy, and Watson, 1983, *J. Biol. Chem.* 258: 8503-11); it synthesizes mevalonate for the production of nonsterol isoprenoids which are needed for growth and differentiation. The enzyme is found in cultured Kc and Schneider cells.

cytology: Located in 95A.

molecular biology: The gene was cloned and its nucleotide and putative amino acid sequences determined (Gertler *et al.*, 1988). An open reading frame of 2,748 bp encodes a polypeptide of 916 amino acids similar to hamster HMG CoA reductase; the C-terminal region showing 56% identical residues and the N-terminal region 32 to 60% identical residues. A 4 kb transcript was found in early embryos, increased in abundance in late embryos, and was expressed at low levels in larvae, pupae, and adults; a 3.2 kb transcript was found at low levels in third-instar larvae, but increased in abundance in pupae and adults. Schneider cell fed mevalonate showed a reduction both in the 4 kb transcript and in enzyme activity.

hmr: hybrid male rescue

location: 1-31.8

origin: Spontaneous in the Ukraine, USSR.

references: Hutter and Ashburner, 1987, *Nature* 327: 331-33.

phenotype: Rescues *D. melanogaster* / *simulans* hybrid males that would not survive in the absence of the gene. Hybrid males that carry both *hmr* and a duplication (thought to be *hmr*⁺) are lethal, so the rescue is considered recessive. The hybrids are sterile. Hybrid females from the reciprocal cross show low viability. *D. melanogaster* / *mauritanica* and *D. melanogaster* / *sechellia* hybrid males also rescued. The rescue of *D. melanogaster* / *simulans* and *D. melanogaster* / *mauritanica* males by *hmr* almost complete at 18°; at 25° *D. melanogaster* / *mauritanica* male rescue is good, but *D. melanogaster* / *simulans* males is poor at this temperature. At 18° only one third of the *D. melanogaster* / *sechellia* males are rescued.

cytology: Located in bands 9D1-2;9E1-2. No chromosome abnormalities are shown.

*hn*³: see *Hn*^{r2}

Hn: Henna

location: 3-23.0

phenotype: A recessive eye color mutant, amorphic alleles of which show slight dominance. The first allele described, being associated with a deficiency, was homozygous lethal, and therefore only the slight dominant phenotype of homogenously dark, dull brown eye color could be scored; thus the dominant symbol *Hn* was applied. All subsequent alleles homozygous viable, exhibiting dark brown sepia-like or clot-like eyecolor in homozygous flies. Red pteridine eye pigments, drosopterin, reduced and sepiapterin accumulates; sepiapterin reductase levels reduced (Barthelmess and Robertson, 1970, *Genet. Res.* 15: 65-86). Eye color of *Hn*^r and *Hn*^{r3} autonomous in transplants of optic disk into wild-type hosts (Beadle and Ephrussi, 1936, *Genetics* 21: 230). Larval Malpighian tubes bright yellow as in wild type (Beadle, 1937, *Genetics* 22: 587-611). RK1 or 2 as homozygote.

alleles:

alleles	origin	discoverer	ref ^α
<i>Hn</i> ^{1β}	X ray	Van Atta, 30k	1, 4, 8, 9
<i>Hn</i> ^{53k}	ultraviolet	Meyer, 53k	1, 5
<i>Hn</i> ^{Pγ}	spont	Craymer	2
<i>Hn</i> ^r	spont	Bridges, 33r20	1, 6
* <i>Hn</i> ^{r2δ}		Nordensköld, 33b9	1, 7
<i>Hn</i> ^{r3ε}	spont	Weinstein, 1927	1, 4
* <i>Hn</i> ^{r53j}	ultraviolet	Meyer, 53j	1, 5
* <i>Hn</i> ^{r1}	spont	Ives, 45j17	1, 3
* <i>Hn</i> ^{rN}	spont	Williamson, 53j	1, 10

^α 1 = CP627; 2 = Craymer, 1980, *DIS* 55: 197-200; 3 = Ives, 1946, *DIS* 20: 65; 4 = Lewis, 1956, *DIS* 30: 130; 5 = Meyer, 1954, *DIS* 28: 76; 6 = Mohr, 1937, *DIS* 8: 12; 7 = Nordensköld, 1937, *DIS* 7: 18; 8 = Van Atta, 1932, *Am. Nat.* 66: 93-95; 9 = Van Atta, 1932, *Genetics* 17: 637-59; 10 = Williamson, 1955, *DIS* 29: 75.

^β Associated with *Df(3L)66A;66B*; *T(2;3)Hn=T(2;3)53E-54A;77A;96A* induced simultaneously but separable from *Hn*.

^γ Present in most if not all *TM6* chromosomes.

^δ Formerly *bu*: *brunette*.

^ε Formerly *sed*: *sepioaid*; *Hn*^{r3} more extreme than *Hn*^{r2}. Homozygotes of *Hn*^{r3} and *ry*⁶ found to be viable (Thompson, 1983, *DIS* 54: 128-29). *Hn*^{r3} lacks tetrahydropterin and has increased levels of tetrahydrobiopterin (Guillamón and Ferré, 1988, *Biochem.*

Biophys. Res. Commun. 152: 49-55).

cytology: Placed in 66A-B on basis of its association with **Df(3L)Hn* = *Df(3L)66A;66B* (Lewis, 1956, DIS 30: 130).

hn RNA-binding protein: see *Hrb*

hnt: hindsight

location: 1-7.

origin: Induced by ethyl methanesulfonate.

references: Wieschaus, Nüsslein-Volhard, and Jürgens 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307.

phenotype: Homozygous embryonic lethal; no germ band retraction; embryo U-shaped with head facing posteriorly. Germ line clones obtained; no maternal effect (Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-52).

alleles: Three.

cytology: Placed in 4B1-C15.

ho: see *dpp*^{d-ho}

Ho: see *tbs*

Hodos: see *Fs(3)Sz10*

hold-up: see *hwp*

Homeobox 2.0: see *H2.0*

homothorax: see *hth*

Hont: see *Fs(2)Sz6*

hook: see *hk*

Hooked-veins: see *Hv*

hop: hopscotch

location: 1-34.6 (between *dsh* and *dlg*).

references: Perrimon and Mahowald, 1986a, Dev. Biol. 118: 28-41.

1986b, Symp. Soc. Dev. Biol. 44: 221-35.

phenotype: The wild-type allele of *hop* is required for the continued cell division of all diploid cells as well as the establishment of the normal array of segments. Most of the mutants are homozygous late zygotic (L-P) lethals; one mutant is a larval lethal; two other mutants have some adult survivors (hemizygous males being morphologically normal, but 40% of the homozygous females and 85% of the hemizygous females showing major defects). Most of the heteroallelic females are lethal, with the following exceptions:

genotype	percent viable
<i>hop</i> ²⁹ / <i>hop</i> ²⁵	12%
<i>hop</i> ³ / <i>hop</i> ²⁵	16%
<i>hop</i> ¹⁴ / <i>hop</i> ²⁵	40%
<i>hop</i> ¹² / <i>hop</i> ²⁵	68%
<i>hop</i> ²⁷ / <i>hop</i> ²⁵	100%
<i>hop</i> ³² / <i>hop</i> ²⁵	100%
<i>hop</i> ³³ / <i>hop</i> ²⁵	100%

All viable heteroallelic combinations are female sterile, failing to produce eggs or laying abnormal eggs that are small, with a clear chorion and with chorionic filaments absent or partially fused (Perrimon and Mahowald, 1986a). There is a maternal effect on thoracic and abdominal segments, the most extreme embryos

[produced from homozygous *l(1)hop* germline clones that have not received a paternal copy of *hop*⁺] showing defects in the posterior spiracles and in segments T2 (denticle belt deleted). T3, A4, and A5 (segment missing) and A8 (segment reduced in size); the least extreme mutant embryos from germline clones show defects in segment A5. Defects visible in early segmentation stages. The extent of the defects is dependent on the strength of the maternal alleles and the paternal contribution. Wild-type sperm can rescue all defects, except those in A5. A few of the rescued progeny hatch and develop into adults.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>hop</i> ¹	X ray	Lefevre	<i>l(1)L4</i>	1, 4, 6	L-P lethal
<i>hop</i> ²	X ray	Lefevre	<i>l(1)C111</i>	5, 6	L-P lethal
<i>hop</i> ³	X ray	Lefevre	<i>l(1)GA32</i>	5, 6	L-P lethal
<i>hop</i> ⁴	X ray	Lefevre	<i>l(1)HC257</i>	5, 6	L-P lethal
<i>hop</i> ⁵	X ray	Lefevre	<i>l(1)HC289</i>	5	
<i>hop</i> ⁶	X ray	Lefevre	<i>l(1)KC28</i>	5	
<i>hop</i> ⁷	X ray	Lefevre	<i>l(1)RA56</i>	5	
<i>hop</i> ⁸	EMS	Lefevre	<i>l(1)DA506</i>	3	
<i>hop</i> ⁹	EMS	Lefevre	<i>l(1)DC764</i>	3	
<i>hop</i> ¹⁰	EMS	Lefevre	<i>l(1)EA39</i>		
<i>hop</i> ¹¹	EMS	Lefevre	<i>l(1)EF447</i>	3	
<i>hop</i> ¹²	EMS	Lefevre	<i>l(1)VA85</i>	3, 6	L/MER
<i>hop</i> ¹³	EMS	Lefevre	<i>l(1)VA108</i>	3, 6	L-P lethal
<i>hop</i> ¹⁴	EMS	Lefevre	<i>l(1)VA275</i>	3, 6	L-P lethal
<i>hop</i> ¹⁵	EMS	Lefevre	<i>l(1)VA312</i>	3, 6	L-P lethal
<i>hop</i> ¹⁶	EMS	Lefevre	<i>l(1)VE666</i>	3, 6	L-P lethal
<i>hop</i> ¹⁷	EMS	Lefevre	<i>l(1)VA928</i>	3	
<i>hop</i> ¹⁸	EMS	Geer	<i>l(1)v12</i>	2	
<i>hop</i> ¹⁹	EMS	Geer	<i>l(1)v20</i>	2	
<i>hop</i> ²⁰	EMS	Geer	<i>l(1)v48</i>	2	larval lethal
<i>hop</i> ²¹	EMS	Geer	<i>l(1)v82</i>	2	
<i>hop</i> ²²	EMS	Geer	<i>l(1)v109</i>	2	
<i>hop</i> ²³	EMS	Geer	<i>l(1)v148</i>	2	
<i>hop</i> ²⁴	EMS	Geer	<i>l(1)v223</i>	2	
<i>hop</i> ²⁵	EMS	Geer	<i>ms(1)v1; msv1</i>	1, 2, 6	viability and fertility poor in males and females ^β
<i>hop</i> ²⁶	EMS	Geer	<i>msn1</i>	2	viability poor; male sterile
<i>hop</i> ²⁷	ENU	Voelker	<i>l(1)M4</i>	6	L-P lethal
<i>hop</i> ²⁸	ENU	Voelker	<i>l(1)M10</i>		
<i>hop</i> ²⁹	ENU	Voelker	<i>l(1)M13</i>	6	L-P lethal
<i>hop</i> ³⁰	ENU	Voelker	<i>l(1)M25</i>		
<i>hop</i> ³¹	ENU	Voelker	<i>l(1)M28</i>		
<i>hop</i> ³²	ENU	Voelker	<i>l(1)M38</i>	6	L-P lethal
<i>hop</i> ³³	ENU	Voelker	<i>l(1)M75</i>	6	L-P lethal
<i>hop</i> ³⁴	EMS	Perrimon	<i>l(1)9P5</i>	6	L-P lethal
<i>hop</i> ³⁵	EMS	Perrimon	<i>l(1)1PP7</i>	6	L-P lethal

^α 1 = Dybas, Harden, Machnicki, and Geer, 1983, J. Exp. Zool. 226: 293-302; 2 = Geer, Lischwe, and Murphy, 1983, J. Exp. Zool. 225: 107-18; 3 = Lefevre. 4 = Lefevre, 1971, Genetics 67: 497-513; 5 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 6 = Perrimon and Mahowald, 1986, Dev. Biol. 118: 28-41.

^β Testes of hemizygous males are usually rudimentary and lack sperm; ovaries of homozygous and hemizygous females usually abnormal, lack egg chambers [believed to be somataic defect since eggs appear normal when allele is analyzed in germline clones (Perrimon and Mahowald, 1986a)].

cytology: Placed in 10B6-8 (Perrimon and Mahowald, 1986a); included in *Df(1)N71* = *Df(1)10B5;10D4* but not in *Df(1)DA622* = *Df(1)10B8;10D2*.

Horka: see *Fs(3)Sz11*

hp: humped**location:** 3- (rearrangement).**origin:** Spontaneous.**discoverer:** Bridges, 31a22.**phenotype:** Thorax shortened and strongly humped with thoracoscutellar groove almost absent. Eyes sharply reduced, may be absent at 29°. Bristles Minute-like and occasionally missing. Viability 10% wild type. RK3A.**cytology:** Associated with *In(3R)P* (Craymer).***hpa: hyperantenna****location:** 1-50.1.**origin:** Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).**discoverer:** Fahmy, 1954.**references:** 1959, DIS 33: 86.**phenotype:** Antennae enlarged or have duplicated parts, sometimes an extra antennal base near the eye. Grossly deformed head and eyes. Wings have rounded tips and incised inner margins. An occasional bristle absent or shorter. Phenotype variable; minimal expression shown by slightly altered eye shape and blunt wing tips. Males viable and infertile; females sterile. RK3.**Hpb: Hydroxyphenyl-buten-1****location:** 2- (chromosomal location tentative).**origin:** Induced by ethyl methanesulfonate.**references:** Kikuchi, 1973, Nature 243: 36-38.**phenotype:** Flies carrying *Hpb-1* are attracted to compounds that repel wild type. The strongest of these is 4-(0-Hydroxyphenyl)3-buten-2-one, where 71% of mutant bearing flies respond positively and 67% of wild type flies negatively to the stimulus, an odds ratio of approximately 5. Slightly lower differentials are seen with eleven among 63 other compounds tested.*Hr*: see *dsx*^D**Hrb1: hnRNA-binding protein 1 (S. Haynes)****location:** 3-{98}.**origin:** Isolated from a cDNA library using *pen* repeat probe.**references:** Haynes, Raychaudhuri and Beyer, 1990, Mol. Cell. Biol. 10: 316-23.

Haynes, Rebbert, Mozer, Forquignon, and Dawid, 1987, Proc. Natl. Acad. Sci. USA 84: 1819-23.

phenotype: Homology to the mammalian A and B hnRNP proteins based on amino acid sequence inferred from nucleotide sequence.**cytology:** Placed in 98D-E by *in situ* hybridization. Located 15 kb distal of *fkh*.**molecular biology:** Genomic clone isolated from Maniatis library (partial sequence); cDNA clones from ovarian, embryonic and pupal libraries (complete sequences). Eight transcripts are produced by use of the alternative promoters, exons, and splice acceptor sites; these transcripts can encode four protein isoforms. Transcripts are expressed throughout development with highest levels in ovaries, early embryos, and pupae.**Hrb2: hnRNA-binding protein 2 (S. Haynes)****origin:** Isolated from a cDNA library by homology with *Drosophila Hrb1* gene.**references:** Haynes, Raychaudhuri, Johnson, Amero, and Beyer, 1990, Mol. Biol. Rep. 14: 93-4.**phenotype:** Homology to the mammalian A and B hnRNP

proteins based on amino acid sequence inferred from nucleotide sequence.

cytology: Placed in 87F by *in situ* hybridization.**molecular biology:** Genomic clone isolated from Maniatis library (partial sequence); cDNA clones from ovarian, and embryonic libraries (complete sequences). Two transcripts (2.2 and 1.7 kb) are produced by use of alternative polyadenylation sites. Transcripts are expressed throughout development with highest levels in ovaries, early embryos, late larvae and early pupae.**Hsc70: Heat-shock-cognate 70**Three gene sequences that share homology with *Hsp70* as demonstrated by *in situ* hybridization to polytene chromosomes and subsequent sequence determination. Although their transcription is developmentally regulated, it is temperature independent. Sequence analysis of the amino terminus reveals 70-80% identity between *Hsp70* and its three cognates. Unlike *Hsp70*, the *Hsc70* genes carry sequences in the 5' non-coding region that encode three additional amino acids. In *Hsc70-1*, the codon specifying amino acid 66 is interrupted by an 1.7 kb insertion; in *Hsc70-2*, the codon specifying amino acid 55 is also interrupted by an insertion (Craig *et al.*, 1983). *Hsc70-4* and *Hsp70*, however, have no insertions in the region coding for amino acids 1-101.

locus	cytology	length	ref ^α	transcript abundance		
				embryo	larva	adult
<i>Hsc70-1</i>	70C	1.7 kb	1, 2	+	-	++
<i>Hsc70-2</i> ^β	87D	0.65 kb	1	-	-	+
<i>Hsc70-4</i>	88E		1	++++	++++	++++

^α 1 = Craig, Ingolia, and Manseau, 1983, Dev. Biol. 99: 418-26;

2 = Ingolia and Craig, 1982, Proc. Nat. Acad. Sci. USA 79: 525-29.

^β Between *ry* and *pic*.**Hsp: Heat-shock protein**Exposure of cells to pulses of elevated temperature initiates the heat-shock response. A restricted subset of genes, the *Hsp* genes, is activated and the majority of transcription and translation is shut-down. However, mitochondrial- and histone-gene activities persist (Spradling, Pardue, and Penman, 1977, J. Mol. Biol. 109: 559-87). This response follows a pulse of 36° to 40°; treatments above 40° inhibit all activity and lead to death; treatments of 30°-35° induce heat-shock-protein synthesis without repressing normal protein synthesis (Tissières, Mitchell, and Tracy, 1974, J. Mol. Biol. 84: 389-98). Similar response inducible by other stressful treatments. The response may be elicited at all stages of the life cycle and in cultured cells. Stage specific phenocopies result from heat shocking early stages of *Drosophila* development [Mitchell and Petersen, 1982, Heat Shock from Bacteria to Man (Schlesinger, Ashburner, and Tissières, eds.). Cold Spring Harbor Laboratory, New York, pp. 345-52]. In polytene cells existing puffs regress and a novel group quickly appears [33B, 63C, 64F, 67B, 70A, 87A, 87C, 93D, 95D (Ashburner, 1970, Chromosoma 31: 356-76; Tissières *et al.*, 1974)]. Activation of transcription of *Hsp* genes apparently involves the sequential binding of two or more protein factors in vicinity of TATA box (Wu, 1984, Nature (London) 309: 229-34). Binding sites for these proteins are multiple short upstream sequence elements called HSEs or heat shock consensus elements (Pelham, 1982, Cell

30: 517-28; Xiao and Lis, 1988, *Science* 239: 1139-42). Polymerase II dissociates from most chromosome regions and accumulates at the new puff sites (Bonner and Kerby, 1982, *Chromosoma* 85: 93-108). 3H-uridine incorporation ceases at its usual positions and commences at new puff sites. Preexisting polysomes disaggregate and within a few minutes a new population of polysomes appears containing newly transcribed mRNA; this RNA hybridizes to some of the heat-shock puffs. The effects of heat shock may be abrogated to some degree by pre-treatment with a pulse of a slightly lower temperature (Mitchell, Moller, Petersen, and Lipps-Sarmiento, 1979, *Dev. Genet.* 1: 181-92 Peterson and Mitchell, 1981, *Proc. Nat. Acad. Sci. USA* 78: 1708-11). For reviews of the heat-shock response see Ashburner and Bonner (1978, *Cell* 17: 241-54) and Heat Shock from Bacteria to Man (cited above). The different heat-shock genes are designated by the molecular weights in kilodaltons of the polypeptides they produce: *Hsp22*, *Hsp23*, *Hsp26*, *Hsp27*, *Hsp68*, *Hsp70* and *Hsp83*.

gene	location	ref ^α	cytology
<i>Hsp22</i> ^β	3-[28]	1-3, 5, 10, 11	67B
<i>Hsp23</i> ^β	3-[28]	1-3, 5, 10, 11	67B
<i>Hsp26</i> ^β	3-[28]	1-3, 5, 10, 11	67B
<i>Hsp27</i> ^β	3-[28]	1-3, 5, 10, 11	67B
<i>Hsp-G1</i> ^γ	3-[28]	1, 10	67B
<i>Hsp-G2</i> ^γ	3-[28]	1, 10	67B
<i>Hsp-G3</i> ^γ	3-[28]	1, 10	67B
<i>Hsp68</i>	3-[81]	4	95D
<i>Hsp70</i> ^δ	3-[51]	4, 6-9	87A7;87C1
<i>Hsp83</i>	3-[5]	4	63B-C

^α 1 = Ayme and Tissières, 1985, *EMBO J.* 29: 49-54; 2 = Corces, Holmgren, Freund, Morimoto, and Meselson, 1980, *Proc. Nat. Acad. Sci. USA* 77: 5390-93; 3 = Craig and McCarthy, 1980, *Nucleic Acids Res.* 8: 4441-57; 4 = Holmgren, Livak, Morimoto, Freund, and Meselson, 1979, *Cell* 18: 1359-70; 5 = Ingolia and Craig, 1982, *Proc. Nat. Acad. Sci. USA* 79: 2360-64; 6 = Ish-Horowitz and Pinchon, 1980, *J. Mol. Biol.* 156: 21-35; 7 = Ish-Horowitz, Pinchon, Gausz, Gyurkovics, Benceze, Goldschmidt-Clermont, and Holden, 1979a, *Cell* 17: 565-717; 8 = Ish-Horowitz, Pinchon, Schedl, Artavanis-Tsakonas, and Mirault, 1979b, *Cell* 18: 1351-58; 9 = Mason, Torek, Kiss, Karch, and Udardy, 1982, *J. Mol. Biol.* 156: 21-35; 10 = Pauli, Arrigo, Vasquez, Tonka, and Tissières, 1989, *Genome* 31: 671-76; 11 = Voellmy, Goldschmidt-Clermont, Southgate, Tissières, Levis, and Gehring, 1981, *Cell* 23: 261-70.

^β Located genetically 2.4 units to the right of *h* (Peterson, Moller, and Mitchell, 1979, *Genetics* 92: 891-902).

^γ Synonym: *gene1*, *gene2*, *gene3* [Southgate, Mirault, Ayme, and Tissières, 1985, *Changes in Gene Expression in Response to Environmental Stress* (Atkinson and Walden, eds.). Academic Press, New York, pp. 1-30]; *gene4* (=2), *gene5* (=3) (Sirotkin and Davidson, 1982, *Dev. Biol.* 89: 196-210).

^δ Cytological location by deficiency mapping and *in situ* hybridization (Ish-Horowitz *et al.*, 1979).

Hsp22 - Hsp-G3

phenotype: There are seven closely related heat-shock genes at 67B (Ayme and Tissières, 1985; Pauli, Arrigo, Vasquez, Tonka, and Tissières, 1989, *Genome* 31: 671-76). In addition to the four small heat-shock genes previously identified (*Hsp22*, *Hsp23*, *Hsp26*, and *Hsp27*), three more genes (*Hsp-G1*, *Hsp-G2*, and *Hsp-G3*, formerly called *Gene1*, *Gene2*, and *Gene3*) have been found clustered within 15 kb of DNA at the same 67B cytological location. All seven genes are heat-shock inducible in almost all cells at the stages tested (Ayme and Tissières, 1985). The genes are also transcribed during certain developmental stages in the absence of heat shock (Sirotkin and Davidson, 1982, *Dev. Biol.* 89: 196-210). Pauli *et al.* (1989) report that the maximum accumulation of developmental rRNA in a majority of these small heat-shock genes occurs in the white pupae stage; in *Hsp-G2*, however, a small transcript is found in embryos, first and second instar larvae, and young pupae; and a larger transcript in the pupal and adult stages of males (Pauli and Tonka, 1987, *J. Mol. Biol.* 198: 235-40; Pauli, Tonka, and Ayme-Southgate, 1988, *J. Mol. Biol.* 200: 47-53). In absence of stress, the expression of *Hsp26* has been observed in spermatocytes, nurse cells, epithelium, imaginal discs, proventriculus, and neurocytes (Glaser, Wolfner, and Lis, 1986, *EMBO* 5: 747-54). Transcripts of *Hsp26* and *Hsp27* accumulate in adult ovaries, apparently originating in nurse cells (Zimmerman, Petri, and Meselson, 1983, *Cell* 32: 1161-70).

alleles: A mutant allele of *Hsp27* that produces small amounts of a large, heat-induced transcript has been cloned and found to contain a defective *P*-element (Eissenberg and Elgin, 1987, *Genetics* 115: 333-40).

molecular biology: All of the small heat-shock genes are located on the same 15 kb fragment. The order from distal to proximal is *Hsp-G3*, *Hsp-G2*, *Hsp22*, *Hsp26*, *Hsp-G1*, *Hsp23*, *Hsp27*, with *Hsp22*, *Hsp-G1*, and *Hsp-G3* transcribed from one strand and the remainder transcribed from the other (Corces, Holmgren, Freund, Morimoto, and Meselson, 1980, *Proc. Nat. Acad. Sci. USA* 77: 5390-93; Voellmy, Goldschmidt-Clermont, Southgate, Tissières, Levis, and Gehring, 1981, *Cell* 23: 261-70; Szauter and Pardue, 1982, *Genetics* 100: s67 68; Ayme and Tissières, 1985; Pauli *et al.*, 1989).

Genomic and putative amino acid sequences obtained from all seven genes (Ingolia and Craig, 1982, *Proc. Nat. Acad. Sci. USA* 79: 2360-64; Southgate, Ayme, and Voellmy, 1983, *J. Mol. Biol.* 165: 35-57; Ayme and Tissières, 1985; Pauli and Tonka, 1987; Pauli *et al.*, 1988). Activation of the small heat-shock genes by stress depends on consensus sequences upstream of the transcription start sites. A third and larger transcript is induced by heat shock in *Hsp-G2*; this transcript extends through *Hsp22* to the 3' end of the latter gene (Pauli *et al.*, 1988). Heat-shock proteins 22, 23, 26, and 27 are encoded by the corresponding genes; protein products of the other three heat-shock genes have not been identified yet, although the amino acid sequences have been estimated from genomic sequences. All seven genes show homologies, both to each other, and to a lesser extent, to mammalian lens protein, the α -crystallins. There is considerable upstream sequence identity between heat-shock genes expressed strongly in ovaries [i.e., *Hsp26*, *Hsp27*, and the larger heat-shock gene *Hsp83* (Xiao and Lis, 1988, *Science* 239: 1139-42)]. Peaks in *Hsp26* and *Hsp28* mRNA are correlated with peaks in ecdysteroid titres in mid-embryogenesis pupariation, and mid pupation (Thomas and Lengyel, 1986, *Dev. Biol.* 115: 434-38).

Hsp68

phenotype: The structural gene for the 68,000 dalton heat-shock protein (HSP68).

molecular biology: The gene has been cloned and restriction mapped; it is represented but once in the genome (Holmgren, Livak, Morimoto, Freund, and Meselson, 1979, *Cell* 18: 1359-70). Thermal stability tests indi-

cates that there is about 15% sequence divergence between *Hsp68* and *Hsp70*. The mRNA of *Hsp68* is 2.1 kb in length; there are no introns in the genomic sequence.

Hsp70

phenotype: The structural genes that code for the 70,000 dalton heat-shock protein (HSP70), the most abundant of the heat-shock proteins. HSP70 returns to preshock levels more rapidly than other heat-shock proteins following return to 25° (DiDomenico, Bugaisky, and Lindquist, 1982, Proc. Nat. Acad. Sci. USA 79: 6181-85). The protein becomes concentrated in nuclei during heat shock; disperses to cytoplasm during recovery; returns to nucleus upon further heat shock (Velazquez and Lindquist, 1984, Cell 36: 655-62). Appears not to be expressed in the testis in response to heat-shock stimulation (Bonner, Parks, Parker-Thornberg, Mortin, and Pelham, 1984, Cell 37: 979-91). Deletion of either the 87A7 or the 87C1 sequences does not eliminate the HSP70 heat-shock response; simultaneous deletion of both sequences does eliminate the HSP70 heat-shock response (Ish-Horowitz *et al.*, 1979).

molecular biology: mRNA for a 70 kilodalton heat-shock protein hybridizes to both 87A and 87C on the salivaries (Spradling, Pardue, and Penman, 1977, J. Mol. Biol. 109: 559-87). Several clones isolated from each region and sequences determined. There are two copies of *Hsp87A*, and three copies of *Hsp87C* (Holmgren, Livak, Morimoto, Freund, and Meselson, 1979, Cell 18: 1359-70). Restriction maps of clones from the two regions differ slightly (Artavanis-Tsakonis, Steward, Gehring, Mirault, Goldschmidt-Clermont, Moran, and Tissières, 1978, Cell 14: 921-29). Two copies of *Hsp70* coding sequence at 87A arranged in divergent order with ATG codons separated by about 1700 nucleotide pairs (Goldschmidt-Clermont, 1980, Nucleic Acids Res. 8: 235-52). Sequence between the two determined (Mason, Torok, Kiss, Karch, and Udvardy, 1982, J. Mol. Biol. 156: 21-35). At 87° two copies separated by about 1000 nucleotides pairs are transcribed in the proximal to distal direction; the third sequence, about 38,000 nucleotide pairs proximal to the other two, is transcribed in the opposite direction (Ish-Horowitz and Pinchon, 1980, J. Mol. Biol. 142: 231-45; Mason *et al.*, 1982). The proximal copy of *Hsp70* at 87C sequenced by Ingolia, Craig, and McCarthy (1980, Cell 21: 669-79). Sequence 5' to *Hsp70* coding sequences compared (Mason *et al.*, 1982). Between the single proximal and two distal *Hsp70* genes at 87C lie several repeated sequences designated α , β , and γ . *Hsp70* exhibits 48% homology with *dnaK*, a heat-shock gene in *E. coli* (Bardwell and Craig, 1984, Proc. Nat. Acad. Sci. USA 81: 848-52). 74% homology with the yeast *Hsp70* genes, and 85% homology with mouse *Hsp70* genes (Moran, Chauvin, Kennedy, Korri, Lowe, Nicholson, and Perry, 1983, Can. J. Biochem. Cell Biol. 61: 488-99). Activation of transcription on exposure to heat shock is carried out by upstream consensus elements or HSEs (see introduction) as indicated by deletion analyses of *Hsp70* (Pelham, 1982, Cell 30: 517-28; McGarry and Lindquist, 1985, Cell 42: 903-11). Determination of sequence requirements by *P*-element germline transformation (Cohen and Meselson, 1984, Proc. Nat. Acad. Sci. USA 81: 5509-13). The HSE for *Hsp70*

was thought to be a 14-bp element, (Pelham, 1982); however, a very low level of heat-shock expression was obtained with two copies of the 14-bp HSE in flies carrying the *Hsp70-lacZ* hybrid gene. Recently, a HSE made up of contiguous arrays of inverted repeats of a 5-bp unit, -GAA-, has been proposed (Amin, Ananthan, and Voellmy, 1988, Mol. Cell Biol. 8: 3761-69; Xiao and Lis, 1988, Science 239: 1139-42; Perisic, Xiao, and Lis, 1989, Cell 59: 797-806); the *Drosophila melanogaster* *Hsp70* gene has four arrays of three or four 5-bp units each (Perisic, Xiao, and Lis, 1989).

Hsp83

phenotype: The structural gene for the 83,000 dalton heat-shock protein (HSP83). During development, the gene is expressed at high levels in the absence of heat shock in many tissues, especially ovaries where it apparently originates in nurse cells (Zimmerman, Petri, and Meselson, 1983, Cell 32: 1161-70). During heat shock, however, the expression level is only raised several fold (Xiao and Lis, 1989, Mol. Cell Biol. 9: 1746-53). Deletion of sequences upstream from the coding region eliminates normal developmental expression and results in regulation of *Hsp83* in a manner similar to that of *Hsp70* which is activated only in response to heat shock.

molecular biology: The sequence, which is unique, has been cloned, restriction mapped, and sequenced (Hackett and Lis, 1983, Nucleic Acids Res. 11: 7011-30; Blackman and Meselson, 1986, J. Mol. Biol. 188: 499-515). The mRNA is 2.6 kb in length; no introns found in genomic sequence. The expression of the gene during normal development (in the absence of heat shock) was thought to be due to the affinity of three overlapping, 14-bp heat shock consensus elements for a *trans*-acting heat shock factor (Xiao and Lis, 1986, Mol. Cell Biol. 6: 3200-06). Deletion mutants carrying the multiple HSEs, however, lost most of their developmental expression, but responded to heat shock like *Hsp70*. Recently Xiao and Lis (1989, Mol. Cell Biol. 9: 1746-53) have proposed that regulation of *Hsp83* is the result of a single array of eight 5-bp units (-GAA). Upstream regions necessary for expression of ovarian development carry a 7-bp sequence CGTTTTG and multiple copies of the shorter sequence GTTTT (Xiao and Lis, 1989). A peak in *Hsp83* mRNA is correlated with a peak in ecdysteroid titre in mid-embryogenesis, pupariation, and mid-pupation (Thomas and Lengyel, 1986, Dev. Biol. 115: 434-38).

Hsr93D: Heat-shock RNA at 93D

location: 3-71 (based on cytological position with respect to *e*).

synonym: *hsr ω* .

references: Bonner and Pardue, 1976, Cell 8: 43-50.

Mohler and Pardue, 1984, Genetics 106: 249-65.

Walldorf, Richter, Ryseck, Steller, Edstrom, Bautz, and Hovemann, 1984, EMBO J. 3: 2499-2505

Garbe and Pardue, 1986, Proc. Nat. Acad. Sci. USA 83: 1812-16.

Garbe, Bandena, Alfano, and Pardue, 1986, J. Biol. Chem. 261: 16889-94.

Bandena, Garbe, Traverse, Lakhotia, and Pardue, 1989, J. Cell Biol. 108: 2017-28.

Garbe, Bandena, and Pardue, 1989, Genetics 122: 403-

15.

Lakhotia, 1989, *Genome* 31: 677-803.

phenotype: A sequence that responds to heat shock by generation of a large puff in polytene chromosomes. The 93D region transcribes mRNA that apparently is not translated. *Hsr93D* is active in almost all cells in *Drosophila melanogaster*; the activity is greatly increased by heat shock (Bonner and Pardue, 1976) and is induced independently by benzamide (Lakhotia and Mukherjee, 1970, *DIS* 45: 108). The inducibility of the locus is selectively repressed by a combination of heat shock with another inducer, by rearing larvae at 10°, by heterozygous deficiency for 93D or by treating wild-type salivaries with beta-alanine (Lakhotia, 1989). The 93D heat shock mRNA is predominately polyA⁻ in the cytoplasm, whereas nuclear transcripts are both polyA⁺ and polyA⁻ (Lengyel, Randson, Graham, and Pardue, 1980, *Chromosoma* 80: 237-52). Heat-shock response in homozygous deficiencies for *Hsr93D* is indistinguishable from normal except for the absence of the 93D puff and transcripts.

cytology: Placed between 93D4 and 93D9 by deficiency mapping.

molecular biology: Transcribed region of the genome cloned and sequenced (Walldorf *et al.*, 1984; Garbe and Pardue, 1986; Garbe *et al.*, 1986 1989). Shown by *Tacql* digestion to comprise a 10-12 kilobase tandem array of 280 nucleotide repeat sequences. Transcripts produced following heat shock complementary to *Tacql* repeat sequence but not to flanking sequences. There are three overlapping transcripts, all from the same start site. The largest transcript, $\omega 1$, is limited to the nucleus and is composed of about 3 kb of unique regions followed by 7-17 kb of short tandem repeats (Bendena *et al.*, 1989). The second transcript, $\omega 2$, also nuclear, contains the first 2-3 kb of the unique region. The cytoplasmic transcript, $\omega 3$, has the same sequence as $\omega 2$ minus an intron of about 700 bp (Garbe *et al.*, 1986). All three transcripts are produced in nonstressed cells, but the transcript level is increased significantly when the cells are heat shocked. Drugs such as benzamide that increase puffing, lead to large increase in the $\omega 1$ transcript. There are no long open reading frames (Garbe *et al.*, 1989). Although *Hsr93D* does not encode any known heat-shock protein, there is some evidence that the cytoplasmic mRNA $\omega 3$ contains a very small open reading frame (ORF ω) that would encode 23 to 27 amino acids and is conserved in *D. melanogaster*, *D. pseudoobscura*, and *D. hydei* (Fini, Bendena, and Pardue, 1989, *J. Cell Biol.* 108: 2045-57; Garbe *et al.*, 1989).

Hsr $\alpha \beta$

location: 3-51 (based on cytological location).

references: Lis, Prestidge, and Hogness, 1978, *Cell* 14: 901-19.

Livak, Freund, Schweber, Wensink, and Meselson, 1978, *Proc. Nat. Acad. Sci. USA* 75: 5613-17.

Hackett and Lis, 1981, *Proc. Nat. Acad. Sci. USA* 78: 6196-6200.

phenotype: Repeated sequences that are transcribed in response to heat shock. Not translated.

cytology: Localized to 87C1.

molecular biology: A series of tandemly repeating units, each comprising an α (0.49 kb) and a β (1.10 kb), located

between the proximal two, divergently oriented, *Hsp70* gene sequences in 87C. There are several tandem arrays separated by nontranscribed spacer regions. In some of the elements β is replaced by γ (0.87 kb), which is homologous to sequences 5' to all five *Hsp70* genes.

HsrW: see *Hsr93D*

hth: homothorax

location: 3-48.

origin: Induced with ethyl methanesulfonate.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95 (fig.).

phenotype: Homozygous lethal. Thoracic segments of embryo similar to one another; morphology of denticle bands intermediate between that of normal first and second thoracic segments.

cytology: Placed in 85E-86B by segmental aneuploids.

Hu: see *Antp^{Hu}* under *ANTC*

Huba: see *Fs(3)Sz12*

Humeral patch: see *Hup*

humped: see *hp*

humpy: see *hy*

hunchback: see *hb*

hup: hold-up

location: 2-44 (probably to the left of *abo* based on failure of 8 recombinants between *da* and *hup* to separate *hup* from *abo*).

origin: Induced by ethyl methanesulfonate.

references: Sandler, 1977, *Genetics* 86: 567-82.

Lindsley, D.E., Goldstein, and Sandler, 1980, *DIS* 55: 84-85.

phenotype: Nearly completely penetrant upheld wing. Homozygotes viable and semisterile in both sexes. Lethal in combination with deficiency. Mortality of zygotes produced by *hup* mothers increased by the presence of a paternally inherited Y chromosome but not by the quantity of paternally derived X heterochromatin. Maternal effect at 25° more severe than at 19°. Fertility also temperature sensitive; spermatids of sterile males show micronuclei and occasional double basal bodies and axonemes.

cytology: Placed in 31F-32E based on inclusion in *Df(2L)J39 = Df(2L)31A-B;32E* but not *Df(2L)J27 = Df(2L)31B-D;31F* of *Df(2L)Mdh-2J = Df(2L)30D-F;31F*.

other information: Complements *abo*, *dal*, and *wd*².

Hup: Humeral patch

location: 1-{20}.

references: Merriam, Yamamoto, Stewart, Rahman, and Nicolau, unpublished.

phenotype: A haplo-sensitive site on the X chromosome. Patch on either side of the thorax including the humeral bristles appears extended as a knobby outgrowth.

cytology: Placed in 7C by segmental aneuploidy.

hv: heavy-vein

location: 2-104.0.

discoverer: Curry, 36115.

phenotype: Veins thick and knobby, especially at ends of

crossveins; posterior crossvein oblique and may show break in middle; extra crossveins sometimes present. Wings broad, thick, dark, warped, divergent, and droopy. Eyes small and bulging. Posterior scutellars blunt, short, and crossed. Overlaps wild type at 25° but useful at 19°. RK2.

***Hv: Hooked-veins**

location: 1-66.

discoverer: Tanaka, 35a4.

references: 1935, DIS 4: 16.

1936, DIS 5: 8.

1937, DIS 8: 11.

phenotype: Heterozygous female shows small branches from posterior crossvein and L5. Eyes small and rough.

Homozygous female lethal. RK3A.

cytology: Associated with *In(1)Hv*.

Hw: see **ASC**

Hx: Hexaptera

location: 2- (not located).

origin: Spontaneous.

discoverer: Herskowitz, 47j.

references: 1949, Genetics 34: 10-25 (fig.).

phenotype: Expression same in *Hx/+* and *Hx/Hx*, varies from absence of a detectable difference from normal through various intermediate types to presence of large appendage on prothorax. Entire abnormal structure may remain beneath exoskeleton. Appendage varies from small amorphous mass to highly differentiated wing. May also produce haltere- and leg-like appendages. Penetrance same in homozygote and heterozygote; prothorax to mesothorax transformation, enhanced by crowding and by high temperature (at 20°, male 1.5 and female 3.3%; at 25°, male 6.5 and female 24.2%); and affected by genotype, *e.g.*, suppressed by *In(2L+2R)Cy* and by *In(2LR)bw^{VI}*. RK3.

hy: humpy

location: 2-93.3.

origin: Spontaneous.

discoverer: Bridges, 18j22.

references: 1937, Cytologia (Tokyo), Fujii Jub., Vol. 2: 745-55.

phenotype: Thorax strongly ridged with commas anteriorly and two pairs of vortices. Wings obliquely truncated to one-half normal length. An irregular contraction of larval muscles at time of pupation (Waddington, 1941, Proc. Zool. Soc. London Ser. A 111: 181-88). Viability low and erratic. Both sexes highly infertile. RK2.

cytology: Placed in region 57 on basis of its being to the right of *In(2R)NS = In(2R)52A2-B1;56F9-13* and to the left of *Df(2R)M-1 = Df(2R)57F11-58A1;58F8-59A1* (Bridges, 1937).

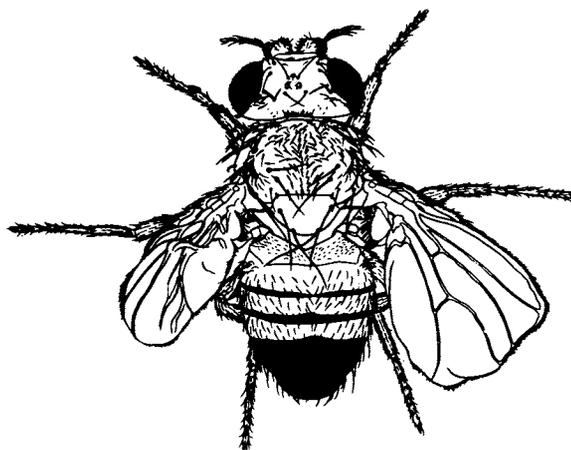
hybrid male rescue: see *hmr*

Hydroxy acid dehydrogenase: see *Had*

Hydroxphenyl buten-1: see *Hpb*

hyperantenna: see *hpa*

Hyperkinetic: see *Hk*



hy: humpy

Edith M. Wallace, unpublished.

hypoA: hypoactive-A (J.C. Hall)

location: 1-1.0.

origin: Induced by ethyl methanesulfonate.

references: Homyk and Sheppard, 1977, Genetics 87: 95-104.

phenotype: Inactive; difficult to arouse for flight; runs and climbs slowly; jumps and flies abnormally short distances; slow optomotor response; debilitated after mechanical stress.

hypoB: see *iav* (in addendum)

hypoC (J.C. Hall)

location: 1-44.3.

origin: Induced by ethyl methanesulfonate.

references: Homyk and Sheppard, 1977, Genetics 87: 95-104.

Homyk, 1977, Genetics 87: 105-28.

O'Dell and Burnet, 1988, Heredity 61: 199-207.

O'Dell, Burnet and Jallon, 1989, Heredity 62: 373-81.

phenotype: Relatively inactive (Homyk and Sheppard, 1977); difficult to arouse for flight; flies abnormally slow (hovers and fixates on object while flying); runs and climbs slowly; slow optomotor response. Jumping ability very poor (O'Dell and Burnet, 1988). Mosaic analysis (Homyk, 1977) suggests primary defect could be in central nervous system or in muscles. In open field activity chamber tests (O'Dell and Burnet, 1988), moves as much as wild-type initially (0-10 minutes) but becomes less active later (10-20 minutes); both speed and amount of movement affected (*e.g.* in aging tests, both metrics increase in parallel from day 1 to 16 of adulthood, whereas wild types show near-maximal levels of activity as young adults, with slow decline during subsequent one month). When homozygous in females, causes reduced mating propensity and extended courtship durations (O'Dell *et al.*, 1989).

alleles: *hypoC¹*, *hypoC²*.

hypoD: see *slrp*

hypoE (J.C. Hall)

location: 1-39.2.

origin: Induced by ethyl methanesulfonate.

references: Homyk, Szidonya, and Suzuki, 1980, *Molec. Gen. Genet.* 177: 553-65.

O'Dell and Burnet, 1988, *Heredity* 61: 199-207.

O'Dell, Burnet and Jallon, 1989, *Heredity* 62: 373-81.

phenotype: Subnormal activity (Homyk *et al.*, 1980); requires extensive agitation to induce movements and becomes inactive more quickly than normal when agitation ceases; males inactive in courtship. Jumping ability very poor (O'Dell and Burnet, 1988). In open field chamber tests (O'Dell and Burnet, 1988), speed of locomotion (number of squares entered/unit time) reduced but not the amount (proportion of time spend moving); amount of speed changes in wild-type-like

manner with advancing adult age (*cf. hypoC*) When *hypoE* homozygous in females, their receptivity to male courtship and mating attempts is essentially the same as for wild-type females (O'Dell *et al.*, 1989).

other information: Complements the closely linked *comt* mutations.

hypoF: see *rdgB*

hypoG (J.C. Hall)

location: 1-50.8.

references: Homyk, Szidonya, and Suzuki, 1980, *Molec. Gen. Genet.* 177: 553-65.

phenotype: Subnormal activity, similar to that caused by *hypoE*, except *hypoG* more abnormal, including decreasing and relatively quick cessation of wing beats in flight plus weak optomotor response.

i: see *E(B)*

I: see *E(B)*

I-f: see *E(f)*

iab: see *BXC*

***iav*: inactive (J.C. Hall)**

location: 1-18.8.

references: Kaplan, 1977, DIS 52: 1.

O'Dell and Burnet, 1986, DIS 63: 107-08.

phenotype: Morphology normal; adults extremely inactive; population of mutants remains quiet and spread out evenly in a container; will walk or fly when container disturbed, but settles into inactivity soon after a disturbance; gene could be allelic to *hypoB* since *hypoB/iav* flies showed a marked reduction in activity for twenty generations (O'Dell and Burnet, 1986). Fertile. RK1.

cytology: Located in 7A5-7C1.

Ic D: see *bw*^{VD}

***Idh*: Isocitrate dehydrogenase**

location: 3-25.4 [based on Ohnishi and Voelkers mapping of *Idh*^F vs *Idh*^S to a position between *ju* and *se* (1982, DIS 58: 121); Fox (1970, DIS 45: 35) mapped same alternatives to 27.1].

3-27.2 [based on Bentley, Meidinger, and Williamson's (1983, Biochem. Genet. 21: 725-33) mapping of *Idh*^{nGB1} vs *Idh*⁺ to a position between *h* and *th*].

synonym: *Idh-NADP*.

references: Fox, D.J., 1971, Biochem. Genet. 5: 69-80.

Williamson, Krochko, and Bentley, 1980, Comp. Biochem. Physiol. 65B: 339-43.

Kuhn and Cunningham, 1986, Dev. Genet. 7: 21-34.

phenotype: Term for the structural gene or genes for NADP⁺-dependent isocitrate dehydrogenase [threo-D-isocitrate: NADP⁺ oxydoreductase (decarboxylating)]; [IDH(E.C 1.1.1.42)]. Studies of purified enzyme (Williamson) indicate it to have a molecular weight of 110,000 and to be a dimer of subunits with molecular weights 60,000 and 50,000. Whether these are products of one or of two closely linked genes is uncertain; formation of a hybrid dimer in *Idh*^F/*Idh*^S heterozygotes suggests a single locus, but the mapping results outlined above are suggestive of two. Only maternally derived enzyme present during most of embryogenesis with vigorous zygotic production beginning just prior to hatching (Wright and Shaw, 1970, Biochem. Genet. 4: 385-94); peak activity reached in third larval instar after which the level falls until just prior to eclosion when it rises once more (Fox, 1971, Biochem. Genet. 5: 69-80). Activity found in all tissues, but especially high in larval fatbody and the sperm pump of adult males and to a lesser extent in the larval and adult midgut and the seminal receptacle and spermathecae (Fox, Conscience-Egli, and Abächerli, 1972, Biochem. Genet. 7: 163-75). Staining activity distributed nonuniformly in the following imaginal discs: eye-antenna, all thoracic discs, and genital, plus in larval gut, adult ovaries, and internal male genitalia. Staining patterns formed gradually in eye-antennal and wing disks. IDH staining pattern and intensity changed in third instar larvae as ommatidia are differentiated. When the tissue type is transformed by homeotic genes, the IDH pattern is altered in a specific

way (Kuhn and Cunningham, 1986). Labial discs, histoblasts, and salivary glands stain uniformly; clypeus-labrum disc, imaginal foregut cells and imaginal ring of hind gut show no activity (Cunningham and Kuhn, 1981, Insect Biochem. 11: 277-85).

allele	synonym	ref ^α	phenotype	
			residual activity	heterodimer formation
<i>Idh</i> ^F	<i>Idh</i> ⁶	3, 4		
<i>Idh</i> ^{nGB1}		2, 6, 7	0	+
<i>Idh</i> ^{nGB2}	<i>Idh</i> ^{GB2}	1, 2, 6, 7	5% β	-
<i>Idh</i> ^{nNC1}		2, 6, 7	0	+
<i>Idh</i> ^S	<i>Idh</i> ⁴	3, 4		
<i>Idh</i> ^{SS}	<i>Idh</i> ²	5		

^α 1 = Bentley, Meidinger, and Williamson, 1983, Biochem. Genet. 21: 725-33; 2 = Burkhart, Montgomery, Langley, and Voelker, 1984, Genetics 107: 295-306; 3 = Fox, 1970, DIS 45: 35; 4 = Fox, 1971, Biochem. Genet. 5: 69-80; 5 = Fox, 1972, DIS 48: 20; 6 = Langley, Voelker, Leigh Brown, Ohnishi, Dickson, and Montgomery, 1981, Genetics 99: 151-56; 7 = Voelker, Langley, Leigh Brown, Ohnishi, Dickson, and Montgomery, and Smith, 1980 Proc. Nat. Acad. USA 77: 1091-95.

^β 24% wild-type levels of CRM; kinetic properties of residual activity indistinguishable from those of wild type.

cytology: Placed in region 66B-D by Voelker and Ohnishi (Burkhardt, Montgomery, Langley and Voelker, 1984, Genetics 107: 295-306).

***if*: inflated**

location: 1-55.

references: Brower, Wilcox, Piovant, Smith, and Reger, 1984, Proc. Nat. Acad. Sci. USA 81: 7485-89.

Wilcox, Brown, Piovant, Smith, and White, 1984, EMBO J. 3: 2307-13.

Bogaert, Brown, and Wilcox, 1987, Cell 511: 929-40.

Leptin, Aebersold, and Wilcox, 1987, EMBO J. 6: 1037-43.

Brower and Jaffe, 1989, Nature (London) 342: 285-87

Brown, King, Wilcox, and Kalfatos, 1989, Cell 59: 185-95.

phenotype: Structural gene for the α-subunit of position specific integrin 2 (PS2), a large transmembrane protein (Bogaert *et al.*, 1987). The β-subunit can associate with either of the two α-subunits, PS1 or PS2 (Brower *et al.*, 1984; Wilcox, Brown, Piovant, Smith, and White, 1984, EMBO J. 3: 2307-13). Both α and β integrin are expressed in embryonic and larval tissues. In early development, PS2 is found in the mesoderm, localized to muscle attachments (Bogaert *et al.*, 1987). Later, PS1 is expressed in the presumptive dorsal epithelium of the third instar imaginal wing discs; also, PS2 is found in the ventral epithelium, both integrins being important for the joining of the dorsal and ventral surfaces of the wing blade (Brower and Jaffe, 1989). Null mutations cause embryonic lethality (Wilcox, DiAntonio, and Leptin). In the mutant *if*¹, the adult wing is inflated with lymph and smaller than normal; venation is defective. Wings later become dry and blistered.

alleles:

alleles	origin	discoverer	ref ^α	comments
<i>if</i> ¹	spont	Weinstein, 1916	4	poorly viable and fertile
<i>if</i> ^{3β}	spont	Curry, 38b	1, 2	resembles <i>if</i> ¹
<i>if</i> ^{k27e}			1	
<i>if</i> ^{Nβ}	neutrons	Schalet	3	viable and fertile; wing phenotype resembles <i>if</i> ³

alleles	origin	discoverer	ref ^α	comments
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^α 1 = Brower and Jaffe, 1989, Nature (London) 342: 285-87; 2 = Curry, 1939, DIS 12: 45; 3 = Schalet, Leigh, and Paradi, 1983, Proc. Int. Congr. Rad. Res. 7th, pp. c4-12; 4 = Weinstein, 1918, Genetics 3: 157.

^β Further descriptions below.

cytology: Located in 15A1-5 by *in situ* hybridization of cloned DNA (Bogaert *et al.*, 1987).

molecular biology: The gene encoding the PS2 α -subunit has been cloned, genomic and cDNA sequences obtained (Bogaert *et al.*, 1987). The predicted N terminus of the PS2 α -subunit is part of a 4182-bp open reading frame that encodes a 1394 amino acid protein of 154 kd; the *Drosophila* N-terminal sequences show identity to the sequences of both the heavy and light chains of α -subunits of the vertebrate fibronectin receptor family (Bogaert *et al.*, 1987; Leptin *et al.*, 1987). The C-terminal region of the *Drosophila* α -subunit that shows no identity to the vertebrate α -subunit is characterized by the presence of multiple serine repeats.

***if*³**

phenotype: Longitudinal veins thickened, especially at wing base. Anterior crossvein thickened. *if*³/*if*³ flies show reduced levels of PS2 integrin on the surfaces of some imaginal disc cells (especially in the ventral region), but levels of this integrin in muscle, salivary glands, and most other tissues seem to be normal (Brower and Jaffe, 1989). Adult wings typically show large round wing blisters, but the penetrance of this phenotype in homo- and hemizygotes is variable. *if*³/*if*³*k27e* flies show an increase in penetrance (from 15-20% in homozygotes to 60-70% in the heteroalleles); penetrance is reduced in *if*³/*if*³*k27e* flies by low temperature and crowding (Brower and Jaffe, 1989).

***if*^N**

other information: Allele induced simultaneously with a *f* mutation (Schalet *et al.*, 1983).

***If*: Irregular facets**

location: 2-107.6 [0.6 unit to the right of *sp*, according to Ives; inseparable from *Kr* by recombination in 2589 opportunities. (Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Dev. Biol. 104: 172-86)].

origin: Spontaneous.

discoverer: Casey, 65116.

references: Ives, 1967, DIS 42: 39.

Renfranz and Benzer, 1989, Dev. Biol. 136: 411-29.

phenotype: In heterozygote, eye area about one-half normal; narrow and pointed ventrally; facets irregular and often missing across middle of eyes, sometimes fused or absent in ventral portion. In homozygote, eyes are narrow slits with smooth glossy surface. In the eye disk of late third instar larvae, fairly large number of cell clusters in irregular arrangement, especially in ventral half of disk (Renfranz and Benzer, 1989). Viability and fertility good. RK1.

cytology: Placed in 60F3 on the basis of the concordant reversion of *If* and induction of *Kr*^{API} associated with a molecularly, but not cytologically, discernable deletion of *Kr*⁺. (Preiss, Rosenberg, Kienlin, Seifert, and Jäekle, 1985, Nature 313: 27-32).

***Ifm*: Indirect flight muscle (J.C. Hall)**

A collection of ethyl-methanesulfonate-induced autosomal dominant flight-defective mutants characterized by Mogami and Hotta (1981, Mol. Gen. Genet. 183: 409-17). Heterozygotes frequently hold wings up. Fine structure examination of myofibrils reveals abnormalities in every case, and in some cases particular proteins missing from three-dimensional gels of homozygotes. Specific features detailed in the following entries.

Ifm(2)3 : see *Mhc*

Ifm(2)11

location: 2-56 (just to right of *pr*).

phenotype: 10% of homozygotes hold wings erect. Myofibrils have thin or ruptured A bands; may appear faint.

other information: Fails to complement *Ifm(2)3* even though it maps to a separate second-chromosome region.

Ifm(3)1 : see *Act88F*

Ifm(3)2 : see *Act88F*

Ifm(3)3 : see *Tm2*

Ifm(3)4 : see *Act88F*

Ifm(3)5, Ifm(3)6

location: 3-55 (between *red* and *ss*).

phenotype: Homozygotes for *Ifm(3)5* and *Ifm(3)6* display characteristic departures from wild-type protein patterns in two-dimensional gels (Mogami and Hotta, 1981). *Ifm(3)5* homozygotes lack myofibrils; *Ifm(3)6* homozygotes have opaque strings of myofibrils. Both mutants are flightless as heterozygotes. The myofibrils of *Ifm(3)6* heterozygotes are frayed.

other information: *Ifm(3)5* and *Ifm(3)6* are recombinationally inseparable.

Ifm(3)7 : see *Act88F*

****im*: interrupted margin**

location: 1-3.1.

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 86-87.

phenotype: Wing margin nicked to various degrees with costal vein frequently interrupted. Extra wing venation often present and occasionally anastomoses, giving a plexus, particularly at the wing apex. Eyes smaller and sometimes slightly rough. Bristles thin. Males small, late eclosing; viability reduced. Female sterile. RK3.

***Imp-E*: Inducible membrane-bound polysomes-Early**

origin: From imaginal disc cultures treated with 20-hydroxyecdysone.

references: Natzle, Hammonds, and Fristrom, 1986, J. Biol. Chem. 261: 5575-83.

Natzle, Fristrom, and Fristrom, 1988, Dev. Biol. 129: 428-38.

phenotype: Name given to three genes encoding transcripts associated with membrane-bound polysomes in imaginal discs and expressed only in response to 20-hydroxyecdysone (20 HOE). Expression studied both *in vitro* and *in vivo*. *Imp-E* denotes three "early" genes

involved in elongation of appendage-forming regions of the imaginal discs by means of epithelial-cell rearrangements (Natzle *et al.*, 1988).

cytology: Three *Imp-E* genes have been isolated; their cytological location as determined by *in situ* hybridization is shown in the following table.

gene	location	cytology
<i>Imp-E1</i> ^α	3-[26]	66C
<i>Imp-E2</i>	3-[6]	63E
<i>Imp-E3</i>	3-[48]	84E

^α Present in single copy.

molecular biology: Genes cloned by use of a hybridization screen for ecdysone inducible expression in imaginal discs (Natzle *et al.*, 1986). *Imp-E1* probe detects a 7.5 kb transcript in cultured imaginal discs treated with 20-HOE as well as in imaginal discs from third instar larvae and white pupae, where transcript occurs in leg, wing, haltere, and genital discs, but not in the ommatidial region of the eye-antennal discs (Natzle *et al.*, 1988). The transcript accumulates rapidly after 20-HOE treatment, but no transcript is found without ecdysone. *In vivo*, the 7.5 kb transcript can be detected, not only in larvae in the imaginal discs, but also (after pupariation) in glial cells around the brain. The gene product is believed to be involved in cell rearrangements in the epithelium of imaginal discs and in the glial layers (Natzle *et al.*, 1988).

Imp-L: Imp-Late

origin: From imaginal disc cultures treated with 20-hydroxyecdysone.

references: Natzle, Hammonds, and Fristrom, 1986, J. Biol. Chem. 261: 5575-83.

Osterbur, Fristrom, Natzle, Tojo, and Fristrom, 1988, Dev. Biol. 129: 439-44.

phenotype: *Imp-L* encoding transcripts associated with membrane-bound polysomes in imaginal discs and expressed only in response to 20-hydroxyecdysone (20 HOE). Expression studied both *in vitro* and *in vivo*. *Imp-L* denotes three "late" genes involved in the eversion to the exterior of the elongated regions of discs by means of local changes in cell shape (Osterbur *et al.*, 1988).

cytology: Three *Imp-L* genes have been isolated; their cytological location as determined by *in situ* hybridization is shown in the following table.

gene	location	cytology
<i>Imp-L1</i>	3-[40]	70A
<i>Imp-L2</i> ^α	3-[12]	64B
<i>Imp-L3</i>	3-[21]	65B

^α Present in single copy.

molecular biology: Genes cloned by use of a hybridization screen for ecdysone-inducible expression is found in imaginal discs (Natzle *et al.*, 1986). *Imp-L3* probe detects a 3.0 kb transcript in cultured imaginal discs treated with 20-HOE as well as in third instar larvae and pupae (maximum transcript accumulation occurring just after pupation). *In vivo*, the 3.0 kb transcript can be detected in cells of the peripodial epithelium (precursors of head and thorax epithelium) and later in the imaginal histoblasts (precursors of the adult abdominal epithelium) (Osterbur *et al.*, 1988). The gene product may be involved in the flattening of the epithelial cells in preparation for fusion into a

contiguous sheet.

in: inturned

location: 3-46.75 (Arajärvi and Hannah-Alava, 1969, DIS 44: 73-4).

origin: Spontaneous.

discoverer: Bridges, 26k20.

references: Gubb and García-Bellido, 1982, J. Embryol. Exp. Morphol. 68: 37-57.

phenotype: Hairs and bristles on thorax and abdominal tergites, directed irregularly toward midline; trichomes and chaetae on mesonotum coordinately misdirected (Toney and Thompson, 1980, *Experientia* 36: 344-45). Marginal hairs of wing stand out from wing margin; trichomes on wing blade no longer directed in parallel with longitudinal veins; frequently divergent therefrom. 50-60% of trichome-bearing cells express duplicated or rarely triplicated trichomes. Wings slightly spread and tend to be long and narrow. Somatic clones of homozygous cells in wing described by Gubb and García-Bellido. Incomplete extra leg joints tend to form as mirror-image duplications proximal to the normal joints on tarsal segments three and four (Held, Duarte, and Derakhshanian, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 145-57). RK1.

alleles:

allele	origin	discoverer	ref ^α	cytology
<i>in</i> ¹	spont	Bridges, 26b20	3, 4	+
<i>in</i> ⁶⁰			1	T(2;3)
<i>in</i> ^{61j2}			1, 5	Dp(1;3)
<i>in</i> ^{SS306}	X ray	S. Smith	2	
<i>in</i> ^{wvco}	X ray	Clausen	3, 4	T(1;3) _w ^{vco}

^α 1 = Arajärvi and Hannah-Alava, 1969, DIS 44: 73; 2 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95; 3 = CP552; 4 = CP627; 5 = Hannah-Alava, 1971, Mol. Gen. Genet. 113: 191-203.

cytology: Placed in 77C on the basis of *Dp(1;3)in*^{61j2} = *Dp(1;3)20;77C*, an insertion of the nucleolus organizer into 3L (Hannah-Alava, 1971, Mol. Gen. Genet. 113: 191-203).

In-f: see *E(f)*

In-ho: see *ho*⁴⁰

inaA: inactivation-no-afterpotential-A (J. Hall)

location: 2-44 (between *Sp* and *B1*).

origin: Induced by ethyl methanesulfonate.

references: Pak, 1979, Neurogenetics: Genetic Approaches to the Nervous System (Breakefield, ed.). Elsevier, New York, pp. 67-99.

phenotype: Mutant eyes lack the prolonged depolarizing afterpotential (PDA) that is induced in wild-type R1-6 photoreceptors by a bright or prolonged blue light stimulus, which photoconverts a large fraction of rhodopsin in these photoreceptors to metarhodopsin; *inaA*-induced phenotype differs from that observed in *nina* mutants because the R1-6 photoreceptors of *ina* mutants are inactivated after the blue stimulus, *i.e.*, are insensitive to subsequent blue light stimuli; an orange light stimulus, which cancels the PDA of wild type by photoconverting metarhodopsin back to rhodopsin, restores the R1-6 cell sensitivity of *ina* mutants.

alleles: One allele isolated as *P226*.

***inaB* (J.C. Hall)**

location: 2-14 (between *S* and *Sp*).
origin: Induced by ethyl methanesulfonate.
references: Pak, 1979, Neurogenetics: Genetic Approaches to the Nervous System (Breakefield, ed.). Elsevier, New York, pp. 67-99.
phenotype: Same basic phenotype as *inaA*; also, *inaB* lacks on and off transient spikes of electroretinogram (ERG).
alleles: Two mutant alleles *inaB*¹ and *inaB*², formerly *P222* and *P223*.
cytology: Located in 25CD-28B. Normal allele carried by *Y*^{P2D} element of *T(1;Y)H52 = T(1;Y)28B*, but not by that of *T(1;Y)D6 = T(1;Y)25C-D* (Pye).

***inaC* (J.C. Hall)**

location: 2-82 (distal to *L*).
origin: Induced by ethyl methanesulfonate.
references: Pak, 1979, Neurogenetics: Genetic Approaches to the Nervous System (Breakefield, ed.). Elsevier, New York, pp. 67-99.
phenotype: Same basic phenotype as *inaA*; also, *inaC* lacks off transient of ERG, and (in such physiological recordings) there is a slow return to baseline potential at stimulus offset.
alleles: Eight; *InaC*¹ - *InaC*³ isolated by Pak as *P207*, *P209*, and *P234*; *InaC*⁴ - *InaC*⁸ isolated in Merriam's laboratory as *US2167*, *US3741*, *US3841*, *US4173*, and *US4186*.

***inaD* (J.C. Hall)**

location: 2-101.
origin: Induced by ethyl methanesulfonate.
references: Pak, 1979, Neurogenetics: Genetic Approaches to the Nervous System (Breakefield, ed.). Elsevier, New York, pp. 67-99.
phenotype: Semi-dominant mutant of visual physiology, such that the phenotype (re PDA) of heterozygote similar to that produced by other *ina* homozygotes; *inaD* homozygotes exhibit ERG response that decays to baseline during bright light stimulus, and off transient is absent.
alleles: One mutant allele, isolated as *P215*.
cytology: Located between 58F1 and 60F1, based on coverage by *bw*⁺ duplication in *bw*⁺*Yy*⁺.

***inaE* (J.C. Hall)**

location: 1 (unlocalized).
origin: Induced by ethyl methanesulfonate.
references: Pak, 1979, Neurogenetics: Genetic Approaches to the Nervous System (Breakefield, ed.). Elsevier, New York, pp. 67-99.
phenotype: Same basic eye-physiology phenotype as in *inaA*, *inaB*, and *inaC*; one allele (at least, *i.e.* *inaE*¹) causes age-dependent photoreceptor degeneration.
alleles: Two mutant alleles; *inaE*¹ and *inaE*² isolated as *N125* and *P19*.

inactive: see *iaV*

****inb*: incised balloon**

location: 2-55.
origin: Spontaneous.
discoverer: Neel, 41d9.
references: 1942, DIS 16: 50.
phenotype: Wings held at 45° angle to body. Wing margins incised, varying from slight nicks to extreme reduc-

tion to small fluid-filled sacs. RK2.

cytology: Salivary chromosomes normal.

****Ind*: Indented**

location: 2-63.
origin: Spontaneous.
discoverer: Cole, 40e.
references: Whittinghill and Parker, 1945, Genetics 30: 27-28.
 Whittinghill, 1947, DIS 21: 72.
phenotype: Eye usually kidney shaped with indentation anteriorly; shape sometimes normal but facets irregular. Often indented posteriorly as well as anteriorly, sometimes dividing eye into two spots or with only upper lobe persisting. Rarely eyeless. More extreme at 28° than at 25°. RK2.

indented thorax: see *int*

Indirect flight muscle: see *lfm*

Inducible membrane-bound polysomes:
 see *Imp-E* and *Imp-L*

Inflated: see *if*

***infrabar*:** see *B*ⁱ

***infrabar Bar*:** see *BB*ⁱ

Inosine dehydrogenase: see *bur*

***Inr*: Insulin receptor**

location: 3-{72}.
references: Petruzzelli, Herrera, Garcia, and Rosen, 1985a, Cancer Cells: 115-21.
 1985b, J. Biol. Chem. 260: 16072-75.
 Nishida, Hata, Nishizuka, Rutter, and Ebina, 1986, Biochem. Biophys. Res. Commun. 141: 474-81.
 Petruzzelli, Herrera, Arenas-Garcia, Fernandez, Birbaum, and Rosen, 1986, Proc. Nat. Acad. Sci. USA 83: 4710-14.
 Fernandez-Almonacid and Rosen, 1987, Mol. Cell Biol. 7: 2718-27.
phenotype: Encodes the insulin-binding (α) and insulin-dependent protein tyrosine kinase (β) subunits of the insulin receptor of *Drosophila melanogaster*. In *Drosophila* cell lines the insulin receptor contains insulin-binding α subunits of 110 or 120 kd, a 95-kd β subunit that is phosphorylated on tyrosine in response to insulin, and a 170-kd protein that may be an incompletely processed receptor. All of the components are processed from a proreceptor, joined by disulfide bonds, and exposed on the cell surface (Petruzzelli *et al.*, 1985a, 1985b, 1986; Fernandez-Almonacid and Rosen, 1987). Subunits in man and in *Drosophila* are similar both in molecular structure and in insulin-binding and protein tyrosine kinase activities; the latter activity is detected only during certain embryonic periods in *Drosophila*.
cytology: Located in 93E by *in situ* hybridization of cloned DNA (Rosen and Young; confirmed by Chovnick); single copy gene.
molecular biology: *Inr* has been cloned using human α- and β-subunit-specific probes (Nishida *et al.*, 1986; Petruzzelli *et al.*, 1986); nucleotide and deduced amino acid sequences of the kinase domain of the β subunits have been obtained. Fourteen clones hybridized to the human α subunit; two clones hybridized to the human β

subunit. A cDNA probe detects mRNA's of 8.6 and 11 kb. The 86-kb mRNA is relatively frequent in oocytes and early embryos whereas the 11-kb message is more prevalent in later embryos, in the developing nervous system and in imaginal discs (Garofalo and Rosen, 1988, Mol. Cell Biol. 8: 1638-47).

int: indented thorax

location: 1-43.5.

origin: Induced by ethyl methanesulfonate.

discoverer: Ghysen.

references: Deak, 1977, J. Embryol. Exp. Morphol. 40: 35-63.

Deak, Bellamy, Bienz, Dubuis, Fenner, Gollin, Rähmi, Ramp, Reinhardt, and Cotton, 1982, J. Embryol. Exp. Morphol. 69: 61-81 (fig.).

Homyk and Emerson, 1988, Genetics 119: 105-21.

phenotype: 80-85% of *int* flies hold wings ventrolaterally, 10% hold them vertically and the remainder maintain normal wing position. In many flies mesonotum indented to variable degree and at various positions; 85% in flies raised at 29° and 45% when raised at 18°. More extreme expression in *y* flies; partially dominant in *y* genotypes. Fate mapping indicates focus in presumptive thoracic musculature. Indirect flight muscle structure badly deranged, Z-bands disappear as muscles degenerate; also lose a 54k protein. *int/int* adults flightless and unable to jump; *int/+* adults normal.

alleles: *int*¹, *int*², *int*³ and *int*⁴ mentioned in Deak *et al.*

other information: Claimed to be allelic to *up* (1-43.5) by Deak *et al.*, 1982 but earlier mapping data of Deak not reconciled; therefore tentatively considered to be a separate locus.

α *Integrin*: see *if*

β *Integrin*: see *mys*

intensifier: see *e*()

Intensifier: see *E*()

interrupted margin: see *im*

Interruptus: see *ci*^W

intersex: see *ix*

intersex on chromosome 3: see *dsx*^{60l}

intersex-62c: see *dsx*

intro: introvert

location: 1-66.

references: Schalet and Lefevre, 1973, Chromosoma 44: 183-202.

Schalet and Lefevre, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902.

Lefevre and Watkins, 1986, Genetics 113: 869-95.

Schalet, 1986, Mutat. Res. 163: 115-44.

Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31.

Schlösser, Boschert, and Fischbach, unpublished data.

phenotype: Many mutants exhibit a pupal lethal phase, although some are larval lethals. [The name "introvert" is derived from abnormality of some hemizygous pupae in which the head remains inverted within the thorax

(Schlösser *et al.*)]. Pupal-phase mutants do not contract at the beginning of pupation, remaining slender like larvae. Eye development very rudimentary. Germline clone analysis shows variable lethal phenotypes, including collapsed eggs (Perrimon *et al.*).

alleles:

allele	origin	discoverer	synonym	ref ^{α}	comments
<i>intro</i> ¹	EMS	Lifschytz	<i>l(1)M28</i>	4	on <i>y</i> ⁺ <i>Ymal</i> ⁺
<i>intro</i> ²	EMS	Lifschytz	<i>l(1)Q2</i>	3	
<i>intro</i> ³	EMS	Lifschytz	<i>l(1)Q56</i>	2, 3, 5, 6	
<i>intro</i> ⁴	EMS	Lifschytz	<i>l(1)R-9-3</i>	3	
<i>intro</i> ⁵	EMS	Lifschytz	<i>l(1)R-9-13</i>	3	
<i>intro</i> ⁶	X ray	Lefevre	<i>l(1)A125</i>	1	<i>T(1;3)18F-19A;20;83C</i>
<i>intro</i> ⁷	X ray	Lefevre	<i>l(1)GE214</i>	1	<i>T(1;2)20A-B:27-28</i>
<i>intro</i> ⁸	X ray	Lefevre	<i>l(1)HF381</i>	1	
<i>intro</i> ⁹	EMS	Lefevre	<i>l(1)DC789</i>	2	
<i>intro</i> ¹⁰	EMS	Lefevre	<i>l(1)VA42</i>	2	
<i>intro</i> ¹¹	EMS	Lefevre	<i>l(1)VE793</i>	2	
<i>intro</i> ¹²	spont	Schalet	<i>l(1)11-55</i>	7	
<i>intro</i> ¹³	spont	Schalet	<i>l(1)19-98</i>	7	
<i>intro</i> ¹⁴	<i>mei-9</i>	Schalet	<i>l(1)18-1</i>		
<i>intro</i> ^{15β}	<i>mei-9</i>	Schalet	<i>l(1)M1-5</i>		
<i>intro</i> ¹⁶	EMS	Lifschytz	<i>l(1)YT1</i>	4	on <i>mal</i> ⁺ <i>Y</i>

^{α} 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 3 = Lifschytz and Falk, 1969, Mutat. Res. 8: 147-55; 4 = Lifschytz and Yakobovitz, 1978, Mol. Gen. Genet. 161: 275-84; 5 = Schalet, 1986, Mutat. Res. 163: 115-44; 6 = Schalet and Lefevre, 1973, Chromosoma 44: 183-202; 7 = Schalet and Lefevre, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902.

^{β} Most flies exhibit a pupal lethal phase like *intro*¹² and *intro*¹³. However, those males and females that eclose and mate appear normal and are fertile (Schalet, 1986). Viability of heterozygotes with *intro*¹² or *intro*¹³ is low (5% or less).

cytology: Located in 20A (Lefevre and Watkins, 1986).

inv: invected

location: 2-{62} (located immediately to the left of *en*).

synonym: *engrailed-related*.

references: Poole, Kauvar, Drees, and Kornberg, 1985, Cell 40: 37-43.

Coleman, Poole, Wier, Soeller, and Kornberg, 1987, Genes Dev. 1: 19-28.

Drees, Ale, Soeller, Coleman, Poole, and Kornberg, 1987, EMBO J. 6: 2803-09.

phenotype: Although a developmental role for *inv* is indicated by hybridization, experiments with embryonic and larval cells using cDNA probes, the function in development of the protein encoded by *inv* is not known (Coleman *et al.*, 1987). No mutant alleles were reported.

cytology: Located in 48A.

molecular biology: DNA spanning the *en* locus was cloned by DNA walking using a collection of *en* mutants of known cytology (Poole *et al.*, 1985). Two clones obtained from this region indicated the presence of another gene located 17 kb proximal to *en*. Unlike *en*, this gene (*inv*) is transcribed from left to right (Poole *et al.*, 1985; Coleman *et al.*, 1987). Nucleotide sequences and presumed amino acid sequences were obtained. *inv* shows striking sequence identity to *en* (52 out of 60 amino acids identical) at the carboxy-terminal end in a 117-amino-acid region containing homeobox sequences; however, no sequence similarities were observed elsewhere in the presumed protein-coding region, which is similar in size in both genes (1740 bp in *inv* and 1656 bp in *en*). *inv* has four exons of 1588, 6, 161, and 694 bp

and three introns; the first intron, the second exon, and the second intron span about 26 kb, the third intron (417 bp) splits the homeobox. Genomic DNA probes from *inv* hybridized to transcripts of 3.4, 2.7, and 1.2 kb (Poole *et al.*, 1985; Drees *et al.*, 1987); the 2.7-kb transcript is believed to represent the mature invected mRNA (Coleman *et al.*, 1987). This mRNA is thought to encode a 576-amino acid protein of about 60 kd. cDNA probes specific to *inv* and *en* were used for *in situ* hybridization to sections from embryos, larvae, and imaginal discs. Both genes were expressed in the posterior developmental compartment of embryonic and larval cells, as well as in the hindgut, clypeolabrum and neural ganglia, but *inv* expression appeared at a later period than *en* expression (Coleman *et al.*, 1987).

inturned: see *in*

invected: see *inv*

Irregular facets: see *If*

It: see *ci*^W

***ix*: intersex**

location: 2-60.5.

origin: Spontaneous.

discoverer: L. V. Morgan, 1943.

references: Morgan, Redfield, and Morgan, 1943, Year Book - Carnegie Inst. Washington 42: 171-74.
Kroeger, 1959, Arch. Entwicklungsmech. Organ. 151: 301-22 (fig.).

Baker and Belote, 1983, Annu. Rev. Genet. 17.

McRobert and Tompkins, 1985, Genetics 111: 89-96.

phenotype: Females changed into sterile intersexes with a set of reduced male and a set of irregular female external genitalia. Gonads also mixed. They have no sex combs; pigmentation of abdomen intermediate between male and female. A large mass of chitinized tissue protrudes from

vaginal opening. Homozygous males look normal but behave like intersexes (McRobert and Tompkins, 1985). They court young males as much as normal males do, and occasionally court females; however, they are attractive to normal mature males. Heterozygous *ix/+* males court females and young males, but seldom court mature males. The *ix* locus acts with the *dsx* female function to allow somatic differentiation in females (Nagoshi, McKeown, Burtis, Belote, and Baker, 1988, Cell 53: 229-36).

***ix*²**

origin: Ultraviolet induced.

discoverer: Meyer, 50k.

synonym: *tom*: *tomboy*.

references: Meyer and Edmondson, 1951, DIS 25: 73.

Meyer, 1958, DIS 32: 83.

phenotype: Females homozygous for *ix*² have male-like pigmentation of posterior tergites, rudimentary ovaries, and are sterile. Expression extreme and viability reduced at 27°; at 17°, expression less extreme and viability greater. Homozygous males appear normal but have nonmotile sperm. RK2.

other information: The possibility that the male sterility is at another locus has not been excluded.

***ix*³**

origin: Induced by ethyl methanesulfonate.

discoverer: Duncan.

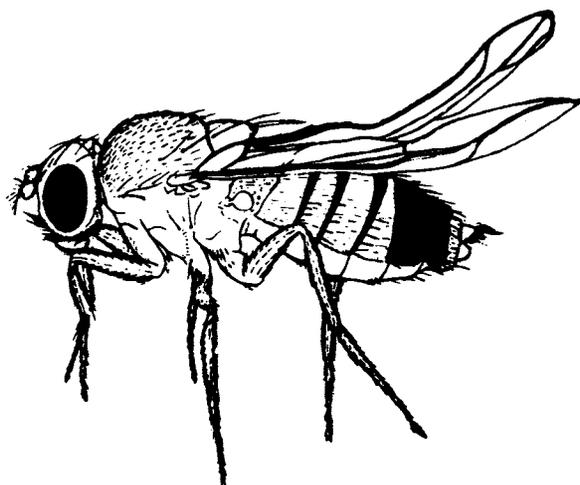
synonym: *ix*^{D100.36}.

references: Nagoshi, McKeown, Burtis, Belote, and Baker, 1988, Cell 53: 229-36.

Ix: see *BX-C*

ix^{62C}: see *dsx*

ix-3: see *dsx*^{60l}

**j: jaunty**

From Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 148.

j: jaunty

location: 2-48.7.

origin: Spontaneous.

discoverer: Bridges, 11111.

references: Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 160 (fig.).

Clausen, 1924, J. Exp. Zool. 38: 423-36.

Stern, 1927, Biol. Zentralbl. 47: 361-69.

phenotype: Distal half of wing upturned but not twisted as in *Cy*. Curling is strong if wing unfolds at 25°-30° but is weak or overlaps wild type if wing unfolds below 25°.

RK2.

alleles:

allele	origin	discoverer	ref ^α	phenotype
<i>j</i> ¹	spont	Bridges, 11111	3, 4, 7	
<i>j</i> ²	spont	Stern, 25d31	7, 8	<j
<i>j</i> ^{49j}	spont	Mossige	6	<j
<i>j</i> ^{50e}	spont	Mossige	6	<j
<i>j</i> ⁵⁸ⁱ	spont	Andrew	1	>j ^β
<i>j</i> ⁶³				
<i>j</i> ^{67b}	EMS	Grell	5	>j
<i>j</i> ^{SF7}	EMS	Littlewood	2	=j

^α 1 = Andrew, 1959, DIS 33: 82; 2 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95; 3 = Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 160 (fig.); 4 = Clausen, 1924, J. Exp. Zool. 38: 423-36; 5 = Grell, 1969, DIS 44: 47; 6 = Mossige, 1951, DIS 25: 69; 7 = Stern, 1927, Biol. Zentralbl. 47: 361-69; 8 = Stern, 1934, DIS 1: 35.

^β Wing bends sharply upward in vicinity of anterior crossvein in extreme cases, with a small dark blot near vein L3 at level of deflection. Anterior crossveins may be reduced or absent.

cytology: Placed in 34E2. Within *Df(2L)b82a2* = *Df(2L)34D2-4;34E1-2* and *Df(2L)fn7* = *Df(2L)34E2-4;35B3-5*, but not in *Df(2L)b82a1* or *Df(2L)el80fl* = *Df(2L)34E2-4;35C3-5* (Ashburner).

J: Jammed

location: 2-41.0.

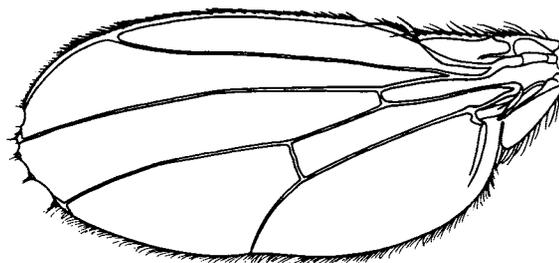
origin: Spontaneous.

discoverer: Bridges, 23d3.

phenotype: Wings often compressed into narrow strips, sometimes filled with fluid. Alula larger and square tipped with clumped bristles and bare regions. Alula modification is characteristically least likely to overlap wild type. Completely overlaps wild type at 19°, almost never at 28° or 30°. Not lethal when homozygous; viability, as in heterozygote, about 70% wild type. Classifiable in single dose triploids (Schultz, 1934, DIS 1: 55). RK1 at 28°-30°; RK2 at 25°.

alleles: *J*¹ and *J*^{34e} (Duncan); like *J*¹ but produces more vigorous stock; RK1 at 28°. Two X-ray-induced revertants *J*^{rv99} and *J*^{rv272} are not deficient for flanking markers; *J*^{rv99} is associated with *T(Y;2)J*^{rv99} (Salas and Lengyel, 1984, DIS 60: 243-44).

cytology: Salivary chromosomes apparently normal (Bridges in Morgan, Bridges, and Schultz, 1937, Year Book - Carnegie Inst. Washington 36: 301). Placed in 31B-F on basis of region common to deficiencies recovered as revertants of *J* (Sandler, 1977, Genetics 86: 567-82).

**Jag: Jagged**

Edith M. Wallace, unpublished.

Jag: Jagged

location: 2-54.9 (0.1 unit from *Bl*).

discoverer: L. V. Morgan, 34b20.

phenotype: *Jag*⁺ has end of wing cut off; better in early counts and above 25°. *Jag/Jag* has reduced and roughened eyes and extremely jagged wings. RK2 as heterozygote; RK3 as homozygote.

Jammed: see *J***jan: janus**

location: 3-99D (close to *Sryβ*).

synonym: *Y*.

references: Yanicostas, Vincent, and Lepesant, 1989, Mol. Cell Biol. 9: 2526-35.

phenotype: Two partially overlapping genes, *janA* and *janB*, regulate the expression of somatic sex differentiation. *janA* is expressed in a complex way in both males and females; *janB* is only expressed in males (Yanicostas *et al.*, 1989).

molecular biology: Genomic and cDNA clones were isolated and nucleotide and deduced amino acid sequences obtained. The genomic and cDNA sequences showed that both *janA* and *janB* contain a single long open reading frame. Both genes are transcribed in the same direction; the 5' end of the *janB* mRNA maps within the 3' untranslated region of the *janA* transcribed sequence, the

overlapping region being 118 bases long. Some other attributes are listed in the following table:

gene	transcripts		predicted protein	
	size (kb)	sex	amino acids	mol. wt.
<i>janA</i>	1.1	males	115	12 kd
	0.95	males		
	0.8	males and females		
<i>janB</i>	0.84	males	140	15 kd

The cDNA sequences of *janA* suggests the existence of alternative transcript structures which have not been definitely determined yet. The size difference between the 0.8 kb and the 0.95 kb transcripts is the result of a difference in length of their poly(A) tails. The male-specific 0.95 kb *janA* transcript and the male-specific *janB* transcript are only expressed in germ line tissues, but the non-sex-specific 0.8 kb *janA* transcript is known to be expressed in both germ line and somatic tissues since detected in germ-line-less as well as normal adults. 37% of the amino acid residues of the two janus genes are identical at homologous positions and the last two 3' introns of both genes are located at identical positions with respect to their protein-coding sequences, suggesting that *janA* and *janB* originated from a duplication of an ancestral transcription unit.

jaunty: see *j*

jaunty x: see *jyx*

javelin: see *ju*

javelinlike: see *jvl*

***je:** *jelly*

location: 3-46.

origin: Spontaneous; arose simultaneously with *mussed* (3-50).

discoverer: Mohr, 37121.

references: Mossige, 1939, DIS 12: 47.

phenotype: Dark pinkish eye color. RK1.

Jon: Jonah

A complex family of about 20 genes distributed at widely dispersed sites on the chromosomes of *Drosophila melanogaster* and expressed only in the midgut of larvae and adults. The function of the gene products are not known. The genes are found in small clusters at different chromosomal sites, as indicated in the following table.

genes (single or clustered)	location	cytology ^α	number of genes at site
<i>Jon25B</i> ^β	2-[13]	25B	3
<i>Jon44E</i>	2-[58]	44E	1
<i>Jon65A</i> ^β	3-[20]	65A	4
<i>Jon66C</i> ^β	3-[26]	66C	2
<i>Jon99C1</i> ^γ	3-[101]	99C	2
<i>Jon99C2</i> ^{βγ}	3-[101]	99C	3
<i>Jon99F</i>	3-[101]	99C	2

^α As indicated by *in situ* hybridization to the salivaries (Carlson and Hogness, 1985, Dev. Biol. 108: 341-54). A weak site at 67B was also identified by long exposure to the label but no genomic clones were identified (Wolfner, 1980, Ph.D thesis, Stanford University). Six gene clusters listed have repeats (direct, inverted, or both).

^β Described in detail at the end of text.

^γ Allelic variants at 99C. Synonym: *JonC* α and *JonC* β.

origin: From cDNA clones from the poly(A)⁺ RNA of third instar larvae before the beginning of larvae metamorphosis. Sites identified by *in situ* hybridization with the reference cDNA probe.

references: Akam and Carlson, 1985, EMBO J. 4: 155-61.

Carlson and Hogness, 1985a, Dev. Biol. 108: 341-54.

Carlson and Hogness, 1985b, Dev. Biol. 108: 355-68.

phenotype: The *Jon* genes are expressed in various regions of the larval midgut (AMG, MMG, PMG) during all the larval stages (Akam and Carlson, 1985); expression disappears at pupation, but reappears in the adult midgut shortly after eclosion and remains throughout adult life.

molecular biology: Genomic clones and subclones carrying *Jonah* genes from different chromosomal sites were constructed and isolated (Carlson and Hogness, 1985a, 1985b), revealing a family of imperfectly repeated genes. *Jonah* mRNA is abundant during all three larval instars and in the adult life, but different *Jonah* RNA's show spatial and temporal differences (Akam and Carlson, 1985; Carlson and Hogness, 1985a). The mature mRNA is about 910 bp in length and encodes a 28 kd translation product whose function has not been determined (Carlson and Hogness, 1985b).

Jon25B

molecular biology: mRNA hybridizes strongly to larvae, weakly to adults (Carlson and Hogness, 1985b).

Jon65A

molecular biology: Weak identity to other *Jonah* genes. mRNA hybridizes to both anterior and middle midgut of larvae (Akam and Carlson, 1985; Carlson and Hogness, 1985b).

Jon66C

molecular biology: mRNA hybridizes to larvae only (Carlson and Hogness, 1985b).

Jon99C2

molecular biology: Shows considerable identity to many other *Jonah* genes. mRNA hybridizes strongly to anterior part of the posterior midgut of larvae (Akam and Carlson, 1985; Carlson and Hogness, 1985b).

jpl: see *gfa*

ju: jumper

location: 3-91.09 (between *se* and *e*).

origin: Wild population from Yugoslavia.

references: Lenicek and Sesta, 1986, DIS 63: 163.

phenotype: Flies have opened wings, oriented at an angle of 65° with respect to the longitudinal axis, and 10-30° with respect to the horizontal axis. Homozygotes show decreased flying ability and move from place to place by jumping.

Jutas: see *Fs(3)Sz13*

ju: javelin

location: 3-19.2 (0.9 unit to left of *dv*).

discoverer: Mohr, 31j29.

references: 1937, DIS 8: 12.

Mohr and Mossige, 1943, Skrifter Norske Videnskaps-Akad. Oslo, I: Mat.-Naturv. Kl., No. 7. 51 pp. (fig.).

phenotype: All bristles and hairs cylindrical, instead of tapered, with small enlargement before tip. Cell autonomous and useful as a marker for epidermal clones (Morata and Ripoll, 1975, Dev. Biol. 42: 211-21). RK2.

alleles: ju^1 and ju^{70b} .

cytology: Placed between 64C12 and 65E1 on the basis of its inclusion in $Df(3L)Vn = Df(3L)64C12-D1;65D2-E1$.

ju1: javelinlike

location: 3-56.7.

origin: Spontaneous.

discoverer: Ives, 4012.

references: 1942, DIS 16: 48.

phenotype: Resembles *ju* but bristles sometimes more crooked. Viability and productivity somewhat lower than normal. RK2.

****jyx: jaunty-x***

location: 1-24.

origin: Spontaneous.

discoverer: Bridges, 14112.

phenotype: Wings curved up at tips. Viability about 60% wild type. RK3.



k: kidney

From Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 77.

k: kidney

location: 3-64.
origin: Spontaneous.
discoverer: Bridges, 12f26.
references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 72 (fig).
 Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 214 (fig.), 227.
phenotype: Eye size reduced by indentation of front margin. Tuft of vibrissae and hairs below eye. Penetrance varies with allele as does expression; moderate alleles overlap wild type significantly. RK3.

allele	origin	discoverer	ref ^α	phenotype
<i>k</i> ¹	spont	Bridges, 12f26	1, 4	moderate
<i>*k</i> ²	spont	Goldschmidt, 1927	2	moderate
<i>k</i> ³	spont	Gottschewski, 1937	2	weak
<i>k</i> ^{6b}	spont	G.L. Lee	3	strong
<i>k</i> ^D	spont	Puro, 60c11	5	β

^α 1 = Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 72 (fig.); 2 = Gottschewski and Ma, 1937, Z. Indukt. Abstamm. Vererbungsl. 73: 584-97; 3 = Lee, G.L., 1972, DIS 48: 18; 4 = Morgan, Bridges, and Sturtevant, 1925, Bibliogr. Genet. 2: 214 (fig.), 227; 5 = Puro, 1964, DIS 39: 65.
^β Derivative of *k*¹; phenotype described separately below.

***k*^D: kidney-Dominant**

phenotype: Eyes of heterozygote reduced at anterior edges. Expression variable; in extreme cases, eye size about one-third normal. *k*^D/*k* more extreme. Eyes of homozygote reduced about as much as *k*^D/*k* but occasionally one or both eyes missing; antennae usually slightly deformed with thickened aristae. RK1.

***kar*: karmoisin**

location: 3-51.7.
phenotype: Eye color like *st* but less bright. Ocelli white. Eyes contain 29% wild-type brown pigment (Nolte, 1954, J. Genet. 52: 111-26). Larval Malpighian tubes of *kar*¹ but not *kar*² considerably lighter than wild type; difficult to classify in living larvae (Brehme and Demerec, 1942, Growth 6: 351-56). Phenoxazinone synthetase level subnormal in mutants; maybe a structural gene for this enzyme; 3-hydroxykynurenine accumulates (Sullivan, Kitos, and Sullivan, 1974, J. Exp. Zool. 188: 225-34). RK1.
cytology: Placed in 87C8 by Gausz, Bencze, Gyurkovics, Ashburner, and Ish-Horowicz (1979, Genetics 93: 917-34).
alleles: Many deficiencies have been given allelic designa-

tions in the past; they are tabulated under *Df(3R)kar*. Alleles believed unaccompanied by deficiencies are summarized in the accompanying table.

allele	origin	discoverer	ref ^α	cytology
<i>kar</i> ¹	spont	Pariser	2	
<i>kar</i> ²	spont	Bridges		
<i>kar</i> ^{5l}		Gelbart	3	<i>Tp(3;1)</i>
<i>kar</i> ^{6l}				<i>ln(3R)</i>
<i>kar</i> ^{Dβ}	X ray	Henikoff	4	<i>ln(3R)</i>
<i>kar</i> ^{H4}	X ray	Henikoff	4	<i>ln(3R)</i>
<i>kar</i> ^{H6}	X ray	Henikoff	4	<i>ln(3R)</i>
<i>kar</i> ^S		Schalet		<i>TM3</i>
<i>kar</i> ^{Sz1}	EMS		1	
<i>kar</i> ^{Sz2}	EMS		1	
<i>kar</i> ^{Sz3}	EMS		1	
<i>kar</i> ^{Sz4}	EMS		1	
<i>kar</i> ^{Sz5γ}	EMS		1	
<i>kar</i> ^{Sz6}	EMS		1	
<i>kar</i> ^{Sz7}	EMS		1	
<i>kar</i> ^{Sz8γ}	EMS		1	
<i>kar</i> ^{Sz9}	EMS		1	
<i>kar</i> ^{Sz10}	EMS		1	
<i>kar</i> ^{Sz11γ}	EMS		1	
<i>kar</i> ^{Sz12γ}	EMS		1	
<i>kar</i> ^{Sz13γ}	EMS		1	

^α 1 = Gausz, Bencze, Gyurkovics, Ashburner, Ish-Horowicz, and Holden, 1979, Genetics 93: 917-34; 2 = Gottschewski, 1935, DIS 4: 15; 3 = Hall and Kankel, 1976, Genetics 83: 517-35; 4 = Henikoff, 1979, Genetics 93: 105-15.
^β Described more fully below.
^γ Independently induced *Df(3R)kar* with the same identifying *Sz* number tabulated under deficiencies.

***kar*^D**

phenotype: *kar*^D/*+* flies exhibit bright red eyes characteristic of *kar* homozygotes; ocelli not completely colorless; seem to variegate. Addition of an extra Y chromosome to *kar*^D/*+* shifts the phenotype toward wild type. Homozygotes show reduced viability.
cytology: Associated with *In(3R)kar*^D=*In(3R)81F;87C8* which variegates for a recessive lethal as well as for *kar*.

***karmoisin ghost*:** see *sad*

***Kartal*:** see *Fs(3)Sz27*

***Kavar*:** see *Fs(3)Sz14*

***kay*: kayak**

location: 3-99.
origin: Induced by ethyl methanesulfonate.
references: Jürgens, Wieschhaus, Nüsslein-Volhard and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95 (fig.).
 Roark, Mahoney, Graham, and Lengyel, 1985, Dev. Biol. 109: 476-88.
phenotype: Homozygous lethal; embryo open dorsally.
alleles: Two.
cytology: Placed in 99A-100A.

***kdn*: knockdown (J.C. Hall)**

location: 1- (between *cv* and *v*).
origin: Induced by ethyl methanesulfonate.
discoverer: Christensen.
references: Ganetzky and Wu, 1982, Genetics 100: 587-614.
phenotype: Temporary paralysis following mechanical shock; sensitive only to initial shock, refractory thereafter. The bang-sensitive phenotype is suppressed by *nap*^{ts} at its permissive temperature; the electrophy-

siology of the larval neuromuscular junction appears normal.

alleles: One mutant allele, called *kdn*^{PC64}.

other information: Could be allelic to *rex*.

kdu: kudu

location: 2-{22}.

origin: Hybrid dysgenesis.

references: Berg and Spradling, 1989.

phenotype: Recessive female-sterile mutation; eggs have fused or thin membranes.

cytology: Placed in 28A.

***ke: kidney eye**

location: 1-28.6.

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 87.

phenotype: Eyes small and extremely rough; anterior border indented, giving a kidney shape. Wings small, abnormal, outspread, or upheld. Veins thick and often interrupted or fail to reach wing margin, which is usually incised. Deformed antennae. Bristles straggly; occasionally, one is missing. Flies short lived; 50% die less than 24 hr after eclosion. Sterile, probably because they are too weak to mate. RK3.

kel: kelch

location: 2-{53}.

origin: Induced by ethyl methanesulfonate.

discoverer: Schüpbach.

references: Steward and Nüsslein-Volhard, 1986, Genetics 113: 665-78.

phenotype: Recessive female sterile. Nurse cell cytoplasm not incorporated in oocyte. Small eggs "open" at anterior pole since egg membranes are not deposited around whole oocyte.

cytology: Located in 36E4; included in *Df(2L)H20* = *Df(2L)36A8-9;36E3-4* and *Df(2L)TW330* = *Df(2L)36E4-F1;37D1-2*.

Keled: see *Fs(3)Sz15*

Ketel: see *Fs(2)Sz7*

Keve: see *Fs(3)Sz16*

kf: see *cut*

Kf1: Kynurenine formamidase 1

location: 3-{62-70}.

references: Moore and Sullivan, 1975, Biochem. Biophys. Acta 397: 486-77.

1978, Biochem. Genet. 16: 619-34.

phenotype: The structural gene for one of the two forms of kynurenine formamidase found in *Drosophila melanogaster* [arylformylamine amidohydrolase (EC 3.5.1.9)]. Formamidase I is a dimer of 34,000 subunit molecular weight, which catalyzes the conversion of N-formylkynurenine to kynurenine. Biochemical characterization by Moore and Sullivan.

cytology: Localized to 91B-93F by dosage response to segmental aneuploidy.

Kf2

location: 2-{13-21}.

references: Moore and Sullivan, 1975, Biochem. Biophys. Acta 397: 468-77.

1978, Biochem. Genet. 16: 619-34.

phenotype: Structural gene for the second form of kynurenine formamidase in *Drosophila melanogaster*. Formamidase II is monomeric with a molecular weight of 31,000. Amino acid compositions of formamidase I and II are virtually indistinguishable, suggesting that they are duplicate genes. They do not interconvert in vitro. The supposed duplicate nature of the coding sequence postulated as the explanation of the inability to recover mutants defective in the conversion of N-formylkynurenine to kynurenine.

cytology: Dosage studies in segmental aneuploids place the structural gene in 25A-27E.

***Kg: Kugel**

location: 3-47.5 [changed from 48.2 in order to lie 0.1 unit to left of *Ki*; as determined using *Kg*^V (Craymer, 1980, DIS 55: 199)].

origin: Spontaneous.

discoverer: Benz, 1953.

references: 1956, Rev. Suisse Zool. 63: 208-16.

phenotype: Larva, pupa, and adult shorter and thicker than normal. Most striking in pupa. Homozygote more extreme than heterozygote. Homozygote viability 68% of wild type and fertility somewhat reduced. RK2.

Kg^V: Kugel of Valencia

origin: X ray induced.

discoverer: Valencia, 1964.

synonym: *Ch*^V: Chubby of Valencia.

references: 1968, DIS 43: 60.

Craymer, 1980, DIS 55: 198.

phenotype: Larvae, pupae, and adults short and thickset; especially easy to score preadult stages. Homozygous males viable and fertile.

Ki: Kinked

location: 3-47.6 [Location in 3R demonstrated by mitotic recombination. (Merriam and García-Bellido, 1972, DIS 44:51) and by recovery in newly induced *C(3R)RM* but not *C(3L)RM* chromosomes (Lütolf, 1971, Experientia 27: 1357)].

origin: Spontaneous.

discoverer: R. F. Grell, 571.

references: 1958, DIS 32: 80.

phenotype: All bristles and hairs of heterozygote shortened and twisted. Resembles *sn*. Viability and fertility excellent; classification easy. Homozygote has more extreme bristle and hair effects. Expression much reduced in flies raised at 18° (Hardy). *Ki*⁺ an excellent marker for cuticular clones in *Ki*/+ heterozygotes. Viability somewhat reduced but fertility near normal. RK1 as heterozygote.

alleles: *Ki*⁷² induced by ethyl methanesulfonate (García-Bellido and Dapena, 1974, Mol. Gen. Genet. 128: 117-30).

cytology: Placed in 83DE by deficiency analysis (Roehrdanz and Lucchesi, 1980, Genetics 95: 355-66).

other information: Very closely linked to the triplo-lethal region of 83DE; attempts to revert *Ki* by deletion unsuccessful (Kaufman, 1978, Genetics 90: 579-96), and *Ki*

frequently deleted simultaneously with *Tpl* (Roehrdanz and Lucchesi).

kidney: see *k*

kidney eye: see *ke*

killer of males: see *km*

Killer of prune: see *awd^K*

Kin: Kinesin

location: 2-[78].

discoverer: Christensen.

references: Saxton, Porter, Cohn, Scholey, Raff, and McIntosh, 1988, Proc. Nat. Acad. Sci. USA 85: 1109-13.

Yang, Saxton, and Goldstein, 1988, Proc. Nat. Acad. Sci. USA 85: 1864-86.

Scholey, Heuser, Yang, and Goldstein, 1989, Nature 338: 355-57.

Yang, Laymon, and Goldstein, 1989, Cell 56: 879-89.

phenotype: Encodes a protein from *Drosophila* that behaves similarly in its effect on movement of microtubules and is antigenically similar to the heavy chain kinesin of squid and sea urchin (Saxton *et al.*, 1988). The *Drosophila* kinesin is found in embryos, larvae, adults, and tissue culture cells. Flies deficient for *Kin* survive through early embryogenesis (Saxton).

molecular biology: Antiserum recognizing the heavy chain of *Drosophila melanogaster* kinesin was employed in the isolation of cDNA clones. The complete nucleotide and predicted protein sequence of a 3.8 kb cDNA clone were obtained (Yang *et al.*, 1988, 1989); this clone produced a protein synthesized *in vitro* that, like heavy chain kinesin in other organisms, binds to microtubules in the presence of the non-hydrolyzable analogue of ATP, AMP-PNP, but not in the presence of ATP or 0.1 M KCl. The cDNA is 3547 nucleotides long and has one long open reading frame; the predicted protein has a molecular weight of 110,428 daltons and is made up of 975 amino acids. Analysis of the protein indicates the presence of a 50 kd globular amino-terminal domain with sites for microtubule and nucleotide binding (the motor domain), a 50-60 kd α -helical coiled-coil stalk region, and a short carboxy-terminal region that is also globular and interacts with light chains (Yang *et al.*, 1989). These structural elements show good correlation with the corresponding structure of mammalian, squid, and sea urchin kinesin heavy chains observed under the electron microscope (Amos, 1987, J. Cell Science 87: 105-11; Hirakawa, Pfister, Yorufuji, Wagner, Brady, and Bloom, 1989, Cell 56: 867-78; Scholey *et al.*, 1989).

Kinked: see *Ki*

kinked femur: see *kf* under *ct*

kis: kismet (J.A. Kennison)

location: 2-0 (between *net* and *art*).

origin: Induced by ethyl methanesulfonate.

discoverer: Kennison, 1984.

synonym: *Su(Pc)21AB*.

references: Kennison and Tamkun, 1988, Proc. Nat. Acad. Sci. USA 85: 8136-40.

phenotype: Isolated as a dominant suppressor of *Pc* alleles. Homozygous lethal. Duplications for *kismet*

strongly enhance the dominant *Pc* phenotypes. *kismet* mutations suppress the antennal transformations of *Antp^B*, *Antp^{73b}*, and *Antp^{Ns}*, as well as the transformations caused by ectopic expression of *Antp* protein under the control of a heat-inducible promoter. *kismet* mutations in mitotic clones induced during larval stages transform the fifth abdominal segment into a more anterior segmental identity.

alleles: Eight alleles induced by ethyl methanesulfonate [including three on *Dp(2;Y)L124*], two by gamma irradiation, and one associated with an insertion of a *P*-element derived *ry* vector.

cytology: Located in 21B6-7 based on its failure to complement *Df(2L)net - PMF = Df(2L)21A1;21B7-8* and *Df(2L)net - PMC = Df(2L)21A1;21B6-7* but not *Df(2L)net - PM47C = Df(2L)21A1;21B6-7*.

kis^S: kismet-Spradling

origin: *P*-element-induced insertion.

discoverer: Spradling.

other information: *ry⁺* transformant that fails to complement *kismet* mutations for viability. Failure to complement *kismet* mutations is revertable at high frequency in the presence of *P*-element transposase.

***kk: kinky**

location: 1-42.

origin: Spontaneous.

discoverer: Philip.

references: 1937, DIS 8: 10.

phenotype: Bristles slightly bent or forked. RK3.

other information: May be an allele of *fw*.

kkv: krotzkopf verkehrt

location: 3-49.

origin: Induced by ethyl methanesulfonate.

references: Jürgens, Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95 (fig.).

phenotype: Homozygous lethal; head skeleton crumbled; denticle bands narrower; embryo rarely inverted in egg case.

alleles: Twenty including one weak and one temperature sensitive.

KL: Male fertility complex in the long arm of the Y chromosome

synonym: Mutants in the Y-linked male-fertility genes have in the past been symbolized *ms(Y)* followed by an identifying symbol; this convention fails to indicate allelic relations among independently recovered Y-linked sterile mutants. Accordingly, mutant terminology now uses the symbol of the fertility gene involved with the particular allele designated by the previously used identifying symbol as a superscript. *e.g.*, *ms(Y)G9* now is *kl-3^{G9}*

references: Brosseau, 1960, Genetics 45: 257-74.

Kennison, 1981, Genetics 98: 529-48.

1983, Genetics 103: 219-34.

Hazelrigg, Fornili, and Kaufman, 1982, Chromosoma 87: 535-59.

Gatti and Pimpinelli, 1983, Chromosoma 88: 349-73.

Bonaccorsi, Pisano, Puoti, and Gatti, 1988, Genetics 120: 1015-34.

The male fertility complex of the long arm of the Y chromosome, originally called *K1* by Stern (1929, Z.

Indukt. Abstamm. Vererbungsl. 51: 253-353) and subsequently called *KL* by Brosseau comprises four male-fertility genes designated *kl-1*, *kl-2*, *kl-3*, and *kl-5* (*kl-4* described by Brosseau, has not been confirmed). These loci have been defined by complementation analysis (Brosseau), by segmental aneuploidy (Kennison) and by cytological localization of male-sterile, *Y*-chromosome breakpoints (Gatti and Pimpinelli).

kl-1

phenotype: Males deficient or mutant at *kl-1* are sterile. One allele of Brosseau's, probably *kl-1*^{B13}, studied by Kiefer, produces motile sperm which are transferred to females but do not reach the female sperm-storage organs; substantial amounts of sperm degeneration observed [Kiefer, 1969, *Genetics* 61: 157-66 (fig.)]. The spermatogenic lesion resembles the late phenotypes of other sterile mutants suggesting that the primary departure from normality has been overlooked. Cross sections of sperm tails nonuniform within a cyst and the number of tails at various levels in a bundle varies. The minitubules, which fill the interstices among the individualized sperm tails of mature bundles, not formed. [Hardy, Tokuyasu, and Lindsley, 1981, *Chromosoma* 83: 593-617 (fig.)].

alleles: In addition to the alleles tabulated, Williamson (1972, *Mol. Gen. Genet.* 119: 43-47) reported fifteen non-complementing and five complementing alleles, all induced by ethyl methanesulfonate.

allele	origin		discoverer	synonym	ref ^α
	treatment	treated			
<i>kl-1</i> ^{B13}	X ray	y ⁺ Y	Brosseau	<i>ms(Y)L13</i>	2, 3
<i>kl-1</i> ^{B25}	X ray	y ⁺ Y	Brosseau	25	2
<i>kl-1</i> ^{B29}	X ray	y ⁺ Y	Brosseau	29	2
<i>kl-1</i> ^{B30}	X ray	y ⁺ Y	Brosseau	30	2
<i>kl-1</i> ^{B33}	X ray	y ⁺ Y	Brosseau	33	2
<i>kl-1</i> ^{E3}	EMS	B ^S Yy ⁺	Kennison	<i>ms(Y)E3</i>	6
<i>kl-1</i> ^{E4}	EMS	B ^S Yy ⁺	Kennison	<i>ms(Y)E4</i>	6
<i>kl-1</i> ^{G6}	γ ray	B ^S Yy ⁺	Kennison	<i>ms(Y)G6</i>	6
<i>kl-1</i> ^{G16} β	γ ray	B ^S Yy ⁺	Kennison	<i>ms(Y)G16</i>	5
<i>kl-1</i> ^{G17} γ	γ ray	B ^S Yy ⁺	Kennison	<i>ms(Y)G17</i>	5
<i>kl-1</i> ^{ts1}	EMS	Y	E91		1
<i>kl-1</i> ^{ts2}	EMS	Y	A141		1
<i>kl-1</i> ^{X1}	X ray	B ^S Yy ⁺	Lms1	A12	4
<i>kl-1</i> ^{X2}	X ray	B ^S Yy ⁺	Lms1	A18	4
<i>kl-1</i> ^{X3}	X ray	B ^S Yy ⁺	Lms1	A22	4
<i>kl-1</i> ^{X4}	X ray	B ^S Yy ⁺	Lms1	B8	4

^α 1 = Ayles, Sanders, Kiefer, and Suzuki, 1973, *Dev. Biol.* 32: 239-57; 2 = Brosseau, 1960, *Genetics* 45: 257-74; 3 = CP627; 4 = Hazelrigg, Fomili, and Kaufman, 1982, *Chromosoma* 87: 535-59; 5 = Kennison, unpublished information; 6 = Kennison, 1983, *Genetics* 103: 219-34.

β Induced on the same chromosome with *kl-5*^{G16}.

γ Induced on the same chromosome with *kl-3*^{G17}.

cytology: *Y-h14*.

kl-2

phenotype: Males deficient or mutant for *kl-2* are sterile; primary ultrastructural lesion not identified. Such sterile males lack a 300-325 kilodalton sperm polypeptide thought to be a structural component of the axoneme (Goldstein, Hardy, and Lindsley, 1982, *Proc. Nat. Acad. Sci. USA* 79: 7404-09; Hardy, Lindsley, Livak, Lewis, Sivertsen, Joslyn, Edwards, and Bonaccorsi, 1984, *Genetics* 107: 591-610).

alleles: In addition to the alleles tabulated, Williamson (1972, *Mol. Gen. Genet.* 119: 43-47) reported five

ethyl-methanesulfonate induced complementing alleles.

allele	origin		discoverer	synonym	ref ^α
	treatment	treated			
<i>kl-2</i> ^{B37}	X ray	y ⁺ Y	Brosseau	<i>ms(Y)L37</i>	2, 3, 4
<i>kl-2</i> ^{B38}	X ray	y ⁺ Y	Brosseau	<i>ms(Y)L38</i>	2, 3, 4
<i>kl-2</i> ^{E5}	EMS	B ^S Yy ⁺	Kennison	<i>ms(Y)E5</i>	5
<i>kl-2</i> ^{E6}	EMS	B ^S Yy ⁺	Kennison	<i>ms(Y)E6</i>	5
<i>kl-2</i> ^{E7}	EMS	B ^S Yy ⁺	Kennison	<i>ms(Y)E7</i>	5
<i>kl-2</i> ^{E8}	EMS	B ^S Yy ⁺	Kennison	<i>ms(Y)E8</i>	5
<i>kl-2</i> ^{G7}	EMS	B ^S Yy ⁺	Kennison	<i>ms(Y)G7</i>	5
<i>kl-2</i> ^{G8}	EMS	B ^S Yy ⁺	Kennison	<i>ms(Y)G8</i>	5
<i>kl-2</i> ^{ts1} β	EMS	Y	A66		1
<i>kl-2</i> ^{ts2} β	EMS	Y	A82		1
<i>kl-2</i> ^{ts3} β	EMS	Y	A145		1
<i>kl-2</i> ^{X1}	X ray	B ^S Yy ⁺	Lms2	A48	4

^α 1 = Ayles, Sanders, Kiefer, and Suzuki, 1973, *Dev. Biol.* 32: 239-57; 2 = Brosseau, 1960, *Genetics* 45: 257-74; 3 = CP627; 4 = Hazelrigg, Fomili, and Kaufman, 1982, *Chromosoma* 87: 535-59; 5 = Kennison, 1983, *Genetics* 103: 219-34.

β Listed as involving either *kl-2* or *kl-4*.

cytology: *Y-h10*.

kl-3

phenotype: Males mutant for *kl-3* fail to assemble the outer dynein arms associated with the peripheral nine microtubule doublets of the sperm-tail axoneme [Hardy, Tokuyasu, and Lindsley, 1981, *Chromosoma* 83: 593-617 (fig.)]. Such males also fail to produce a *kl-3*-specific 300-325 kilodalton sperm polypeptide presumed to be a component of the outer dynein arms (Goldstein, Hardy, and Lindsley, 1982, *Proc. Nat. Acad. Sci. USA* 79: 7404-09). Such mutants display an exceedingly low level of fertility at 25° but not at 18° immediately upon eclosion, but not thereafter (Kennison, 1983, *Genetics* 103: 219-34). Deficiencies for *kl-3* are completely sterile, and in addition to the above mutant phenotype, they fail to elaborate the loops (ribbon-like structure) observed in the primary spermatocyte nuclei of normal males by light microscopy and the reticular material ordinarily observed by electron microscopy in spermatocyte nuclei (Hardy *et al.*, 1981). The *kl-3* loops are visible in living spermatocytes and in fixed cells stained with the protein-specific dye CBB; these loops are also demonstrated by the polyclonal antibody Sph-155 but do not react with monoclonal antibody S5 (Bonaccorsi *et al.*, 1988).

alleles: In addition to the alleles tabulated, Williamson (1972, *Biol. Gen. Genet.* 119: 43-47) reported 23 non-complementing and nine complementing alleles induced by ethyl methanesulfonate.

allele	origin		discoverer	synonym	ref ^α
	treatment	treated			
<i>kl-3</i> ^{B11}	X ray	y ⁺ Y	Brosseau	<i>ms(Y)L11</i>	1, 2, 3
<i>kl-3</i> ^{B20}	X ray	y ⁺ Y	Brosseau	<i>ms(Y)L20</i>	2
<i>kl-3</i> ^{B27}	X ray	y ⁺ Y	Brosseau	<i>ms(Y)L27</i>	2
<i>kl-3</i> ^{B41}	X ray	y ⁺ Y	Brosseau	<i>ms(Y)L41</i>	2
<i>kl-3</i> ^{E9}	EMS	B ^S Yy ⁺	Kennison	<i>ms(Y)E9</i>	5
<i>kl-3</i> ^{E10}	EMS	B ^S Yy ⁺	Kennison	<i>ms(Y)E10</i>	5
<i>kl-3</i> ^{E11}	EMS	B ^S Yy ⁺	Kennison	<i>ms(Y)E11</i>	5
<i>kl-3</i> ^{G9}	γ ray	B ^S Yy ⁺	Kennison	<i>ms(Y)G9</i>	4
<i>kl-3</i> ^{G10}	γ ray	B ^S Yy ⁺	Kennison	<i>ms(Y)G10</i>	5
<i>kl-3</i> ^{G11}	γ ray	B ^S Yy ⁺	Kennison	<i>ms(Y)G11</i>	5
<i>kl-3</i> ^{G17} β	γ ray	B ^S Yy ⁺	Kennison	<i>ms(Y)G17</i>	4
<i>kl-3</i> ^{X1}	X ray	B ^S Yy ⁺	Lms3	A1	3
<i>kl-3</i> ^{X2}	X ray	B ^S Yy ⁺	Lms3	A40	3
<i>kl-3</i> ^{X3}	X ray	B ^S Yy ⁺	Lms3	A51	3
<i>kl-3</i> ^{X4}	X ray	B ^S Yy ⁺	Lms3	B10	3

allele	origin		discoverer	synonym	ref ^α
	treatment	treated			
^α	1 = Brosseau, 1960, Genetics 45: 257-74; 2 = CP627; 3 = Hazelrigg, Fornili and Kaufman, 1982, Chromosoma 87: 535-59; 4 = Kennison, unpublished information; 5 = Kennison, 1983, Genetics 103: 219-34.				
^β	Induced on the same chromosome with <i>kl-1</i> ^{G17} .				

cytology: *Y-h7*.

kl-4

Existence inferred by Brosseau (1960, Genetics 45: 257-74) on the basis of the failure of a *kl-3*⁻ *Y* to complement a *kl-5*⁻ *Y* for male fertility. No single site mutant or breakpoint interrupting *kl-4* alone reported by Brosseau, Kennison (1983, Genetics 103: 219-34), Hazelrigg, Fornili and Kaufman (1982, Chromosoma 87: 535-59), or Gatti and Pimpinelli (1983, Chromosoma 88: 349-73). Williamson (1972, Mol. Gen. Genet. 119: 43-47), on the other hand reported seven noncomplementing and seven complementing alleles induced by ethyl methanesulfonate.

kl-5

phenotype: Males mutant for *kl-5* fail to assemble the outer dynein arms associated with the A tubules of the peripheral doublets of the sperm tail axoneme [Hardy, Tokuyasu, and Lindsley, 1981, Chromosoma 83: 593-617 (fig.)]. Such males also fail to produce *kl-5*-specific 300-325 kilodalton sperm polypeptide considered to be a component of the outer arms (Goldstein, Hardy, and Lindsley, 1982, Proc. Nat. Acad. Sci. USA 79: 7404-09). *kl-5* mutants exhibit very low level of fertility at 25° but not at 18° and in young but not older males (Kennison, 1983, Genetics 103: 219-34). Deficiencies for *kl-5* completely sterile, and in addition to the mutant phenotype they fail to produce aggregates of tubuli normally seen in thin sections of primary spermatocyte nuclei (Hardy *et al.*). Within the *kl-5* fertility region of the spermatocyte nucleus, a loop-forming site has been demonstrated by its reaction with the monoclonal antibody S5 (Bonaccorsi *et al.*, 1988). These loops are stained with the protein-specific dye CBB, but are not visible in living preparations.

alleles: In addition to the alleles tabulated, Williamson (1972, Mol. Gen. Genet. 119: 43-47) reported 26 non-complementing and 9 complementing alleles induced by ethyl-methanesulfonate.

allele	origin		discoverer	synonym	ref ^α
	treatment	treated			
<i>kl-5 B3</i>	X ray	y ⁺ Y	Brosseau	<i>ms(Y)L3</i>	2, 3
<i>kl-5 B28</i>	X ray	y ⁺ Y	Brosseau	<i>ms(Y)L28</i>	2
<i>kl-5 B34</i>	X ray	y ⁺ Y	Brosseau	<i>ms(Y)L34</i>	2
<i>kl-5 E12</i>	EMS	B ^S Yy ⁺	Kennison	<i>ms(Y)E12</i>	6
<i>kl-5 E13</i>	EMS	B ^S Yy ⁺	Kennison	<i>ms(Y)E13</i>	6
<i>kl-5 E14</i>	EMS	B ^S Yy ⁺	Kennison	<i>ms(Y)E14</i>	6
<i>kl-5 E15</i>	EMS	B ^S Yy ⁺	Kennison	<i>ms(Y)E15</i>	6
<i>kl-5 E16</i>	EMS	B ^S Yy ⁺	Kennison	<i>ms(Y)E16</i>	6
<i>kl-5 E17</i>	EMS	B ^S Yy ⁺	Kennison	<i>ms(Y)E17</i>	6
<i>kl-5 G12</i>	γ ray	B ^S Yy ⁺	Kennison	<i>ms(Y)G12</i>	6
<i>kl-5 G13</i>	γ ray	B ^S Yy ⁺	Kennison	<i>ms(Y)G13</i>	6
<i>kl-5 G14</i>	γ ray	B ^S Yy ⁺	Kennison	<i>ms(Y)G14</i>	6
<i>kl-5 G15</i>	γ ray	B ^S Yy ⁺	Kennison	<i>ms(Y)G15</i>	6
<i>kl-5 G16</i> ^β	γ ray	B ^S Yy ⁺	Kennison	<i>ms(Y)G16</i>	6
<i>kl-5 ts1</i>	EMS	Y		A12	1
<i>kl-5 ts2</i> ^γ	EMS	Y		B119	1, 4
<i>kl-5 ts3</i>	EMS	Y		H39	1

allele	origin		discoverer	synonym	ref ^α
	treatment	treated			
<i>kl-5 X1</i>	X ray	B ^S Yy ⁺		<i>Lms4 A3</i>	5
<i>kl-5 X2</i>	X ray	B ^S Yy ⁺		<i>Lms4 A36</i>	5
<i>kl-5 X3</i>	X ray	B ^S Yy ⁺		<i>Lms4 B5</i>	5

^α 1 = Ayles, Sanders, Kiefer, and Suzuki, 1973, Dev. Biol. 32: 239-57; 2 = Brosseau, 1960, Genetics 45: 257-74; 3 = CP627; 4 = Goldstein, Hardy, and Lindsley, 1982, Proc. Nat. Acad. Sci. USA 79: 7405-09; 5 = Hazelrigg, Fornili, and Kaufman, 1982, Chromosoma 87: 535-59; 6 = Kennison, unpublished information.

^β Induced on same chromosome with *kl-1*^{G16}.

^γ Produces normal quantities of its high-molecular-weight polypeptide at restrictive temperature; however, outer-arm assembly defective.

cytology: *Y-h1-2* and *h3*.

klis: klarsicht

location: 3-0.0.

discoverer: Nüsslein-Volhard.

phenotype: Maternal effect mutant; without effect on viability or fertility of homozygotes. Homozygous females produce eggs with clear distinction between yolk and germ layers. Facilitates *in vivo* observation of developmental processes.

kmA: killer of males

location: 2-45.

origin: Induced by ethyl methanesulfonate.

references: Pierre, 1972, DIS 48: 16.

phenotype: Homozygous males die during embryogenesis.

kmB

location: 2-20.

origin: Induced by ethyl methanesulfonate.

references: Pierre, 1972, DIS 48: 16.

phenotype: Homozygous males die during embryogenesis.

kn: knot

location: 2-72.3.

discoverer: Nichols-Skoog, 31h1.

references: Díaz-Benjumea, Gaitán, and García-Bellido, 1989, Genome 31: 612-19.

phenotype: Veins L3 and L4 shifted closer together in region of anterior crossvein, which is either extremely thick or eliminated by regional fusion of L3 and L4. Frequently, extra crossvein between L3 and L4 near end of wing. Shift in positions of sensilla and extra chaetae accompanies shift in vein positions. Wing narrowed. Head narrowed and flattened, so the long axis of eye is at oblique angle. May overlap wild type at high temperatures and in late counts. Best at 19°. RK2.

cytology: Located in 51C-E (MacIntyre).

kni: knirps

location: 3-{46}.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard and Wieschaus, 1980, Nature (London) 287: 795-801 (fig.).

Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95 (fig.).

Carroll and Scott, 1986, Cell 45: 113-26.

Jäckle, Tautz, Schuh, Seifert, and Lehmann, 1986, Nature (London) 324: 668-70.

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

Nauber, Pankratz, Kienlin, Seifert, Klemm and Jäckle, 1988, Nature (London) 336: 489-92.

Pankratz, Hoch, Seifert, and Jäckle, 1989, Nature (London) 341: 337-40.

Rothe, Nauber, and Jäckle, 1989, EMBO J. 8: 3087-94.

phenotype: Zygotic gap gene whose mutants are homozygous lethal; their denticle belts in segments one through seven are fused into a single field (Nüsslein-Volhard and Wieschaus, 1980), but the head, thorax, eighth abdominal segment, and tail region appear normal. Embryos homozygous for a strong *kni* mutant show a wide band of *ftz* staining instead of the normal number of *ftz* stripes (Carroll and Scott, 1986).

alleles: The following alleles are listed by Tearle and Nüsslein-Volhard, 1987:

allele	origin	synonym	comments
<i>kni</i> ¹	EMS	<i>kni</i> ^{5F}	
<i>kni</i> ²	EMS	<i>kni</i> ^{7G}	strong
<i>kni</i> ³	EMS	<i>kni</i> ^{14F}	weak
<i>kni</i> ⁴	EMS	<i>kni</i> ¹⁷	
<i>kni</i> ⁵	EMS	<i>kni</i> ¹⁹	strong
<i>kni</i> ⁶	EMS	<i>kni</i> ^{30I}	strong
<i>kni</i> ⁷	EMS	<i>kni</i> ³⁵⁷	strong
<i>kni</i> ⁸	X ray	<i>kni</i> ^{FC13} ^α	strong
<i>kni</i> ⁹	EMS	<i>kni</i> ^{1L}	strong
<i>kni</i> ¹⁰	EMS	<i>kni</i> ^{11D}	strong
<i>kni</i> ¹¹	EMS	<i>kni</i> ^{11E}	strong
<i>kni</i> ¹²	EMS	<i>kni</i> ^{11V}	strong

^α Also see Nauber *et al.*, 1988.

cytology: Placed in 77E1-2 by *in situ* hybridization of cloned DNA to salivaries (Nauber *et al.*, 1988). Included in *Df(3L)ri79c = Df(3L)77B-C;77F-78A* (Jürgens *et al.*, 1984).

molecular biology: The 77E1 region was microcloned and used to start a chromosome walk (Nauber *et al.*, 1988). Breakpoints of chromosomal aberrations involving *kni* were located on the molecular map. Two transcripts of 2.2 and 2.5 kb were identified; the smaller transcript is expressed transiently at the blastoderm stage, but the larger one continues to be expressed after gastrulation. These transcripts are not present in embryos homozygous for *kni*^{FC13}, a small molecular deletion of 2 kb. Genomic and cDNA sequences and predicted amino acid sequences were determined (Nauber *et al.*, 1988). Analysis of the DNA sequences showed three exons interrupted by two introns of 733 bp and 214 bp. A protein of 429 amino acids would be encoded by the single open reading frame of 1,287 bp. This protein shows similarity in the N terminus to proteins of the vertebrate nuclear hormone receptor family (Nauber *et al.*, 1988). The zinc-finger domain of *knirps* and its amino-acid motif, the *kni* box, show 80-88% identity with the corresponding domains of *egon* and *knrl*, two genes that also map in the 77-79 region [Oro, Ong, Margolis, Posakony, McKeown, and Evans, 1988, Nature (London) 336: 493-96; Rothe *et al.*, 1989].

knickkopf: see *knk*

knirps: see *kni*

knirps-related: see *knrl*

knk: *knickkopf*

location: 3-49.

origin: Induced by ethyl methanesulfonate.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol.

193: 283-95 (fig.).

phenotype: Homozygous lethal; head skeleton defective; denticle bands narrowed; embryo rarely inverted in egg case.

alleles: Four, including one weak and one temperature-sensitive allele.

cytology: Placed in 85E1-10 based on its inclusion in *Df(3R)GB104 = Df(3R)85D11-13;85E10* and *Df(3R)by62 = Df(3R)85D11-14;85E16* but not *Df(3R)by416 = Df(3R)85D10-12;85E1-2*.

***kno:** *knobbyhead*

location: 1-63.9.

origin: Induced by triethylenemelamine (CB. 1246).

discoverer: Fahmy, 1951.

references: 1958, DIS 32: 70.

phenotype: Abnormal head with one or both eyes irregularly shaped, often drastically reduced in size. Occipital region frequently has hairy tufts, often carried on protuberances. Males highly infertile; viability about 10% wild type. RK2.

other information: One allele induced by D-1:6-dimethanesulfonyl mannitol (CB. 2511).

knockdown: see *kdn*

knot: see *kn*

knrl: *knirps-related*

location: 3-{46}.

origin: Isolated from a genomic library using a human retinoic acid receptor cDNA probe (Oro *et al.*, 1988) or a *Drosophila kni* zinc-finger probe (Rothe *et al.*, 1989).

references: Oro, Ong, Margolis, Posakony, McKeown, and Evans, 1988, Nature (London) 336: 493-96.

Rothe, Nauber, and Jäckle, 1989, EMBO J. 8: 3087-94.

phenotype: Expressed maternally at low levels in very early stages; expression increases during early embryogenesis and continues in larvae and adults (in contrast to *kni*, which is first expressed zygotically and does not continue beyond embryonic stages) (Oro *et al.*, 1988; Rothe *et al.*, 1989).

alleles: No loss-of-function alleles isolated.

cytology: Located in 77E1-2 by *in situ* hybridization; location not distinguishable by this technique from that of *kni*.

molecular biology: Gene cloned and sequenced (Oro *et al.*, 1988). One clone of 3,505 bp contains an open reading frame encoding 647 amino acids showing 85% identity with the predicted *knirps* gene product and 47% identity with the human retinoic acid receptor. The genes *knrl*, *kni*, and *egon* show more than 80% sequence identity in the first part of the zinc-finger domain and share a *kni* box downstream from this domain (Rothe *et al.*, 1989). In the carboxyl-terminal region, however, there is little sequence similarity between *knrl* and the other receptors.

kohtalo: see *kto*

Kompolt: see *Fs(2)Sz8*

Kpn: see *awd*^K

Kr: Krüppel

location: 2-107.6 (mapped with respect to *sp* and *If*).

discoverer: Graber.

references: Gloor, 1950, Arch. Julius Klaus-Stift. Vererbungsforsch. Sozialanthropol. Rassenhyg. 25: 38-44 (fig.).

1954, Arch. Julius Klaus-Stift. Vererbungsforsch. Sozialanthropol. Rassenhyg. 29: 277-87.

Nüsslein-Volhard and Wieschaus, 1980, Nature (London) 287: 795-801 (fig.).

Jürgens, Wieschaus, Nüsslein-Volhard and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Dev. Biol. 104: 172-86. (fig.).

Knipple, Seifert, Rosenberg, Preiss, and Jäckle, 1985, Nature (London) 317: 40-44.

Preiss, Rosenberg, Kienlin, Seifert, and Jäckle, 1985, Nature (London) 313: 27-32.

Rosenberg, Preiss, Seifert, Jäckle, and Knipple, 1985, Nature (London) 313: 703-06.

Jäckle, Tautz, Schuh, Seifert, and Lehmann, 1986, Nature (London) 324: 18-25.

Rosenberg, Schröder, Preiss, Kienlin, Côté, Riede, and Jäckle, 1986, Nature (London) 319: 336-39.

Schuh, Aicher, Gaul, Côté, Preiss, Maier, Seifert, Nauber, Schröder, Kemler, and Jäckle, 1986, Cell 47: 1025-32.

Gaul and Jäckle, 1987, Cell 51: 549-55.

Gaul, Seifert, Schuh, and Jäckle, 1987, Cell 50: 639-47.

Gaul, Redemann, and Jäckle, 1989, Proc. Nat. Acad. Sci. USA 86: 4599-4603.

Gaul and Jäckle, 1989, Development 107: 651-62.

Stanojevic, Hoey, and Levine, 1989, Nature (London) 341: 331-35.

Treisman and Desplan, 1989, Nature (London) 341: 335-37.

phenotype: The wild-type allele of Krüppel controls the development of the thoracic and abdominal segments of *Drosophila*; homozygous mutants show a gap in the larval pattern in these regions (Nüsslein-Volhard and Wieschaus, 1980; Knipple, *et al.*, 1987). *Kr*^{+/+} adult sometimes has thoracic malformation; a leg or a wing may be absent; penetrance low. Heterozygous larvae show small defects in denticle bands of thorax and abdomen as do heterozygous deficiencies for *Kr*; penetrance about 80%. Homozygotes are embryonic lethal. *Kr*¹ homozygotes exhibit shortened germ band of but three to four segments with three to four tracheal pits; visible beginning at 7h of embryogenesis; head and gnathal segments apparently normal. At later stages only three to four abdominal and thoracic segments clearly visible; normal telson and segments 8 and 7 followed by enlarged sixth, a rudimentary fifth and apparently mirror image sixth segment. Weak and intermediate mutant alleles lack the mirror-image duplications (Gaul and Jäckle, 1987, Trends Genet. 3: 127-30). Ventral chain of ganglia disconnected; tracheal system defective; Malpighian tubules missing; salivary glands normal. Homozygotes for hypomorphic alleles display more nearly complete segmentation. Homozygous *M*⁺ *Kr* clones develop normally in all parts of adult cuticle of *M*⁺ flies. Metamorphic potential of *Kr*/*Kr* embryos cultured in female abdomens restricted in that wing-disc-derived structures not observed. Germline clones of homozygous

Kr cells capable of normal oogenesis; no maternal effect of *Kr*⁺ observed. Requirement for *Kr*⁺ function apparently restricted to early embryogenesis. *Kr* affects *ftz* producing abnormal intensity and spacing of *ftz* stripes in thorax and anterior abdomen (Carroll and Scott, 1986, Cell 45: 113-26).

alleles: Twentyfour alleles are listed in the following table. Deficiencies are listed in the rearrangement section.

allele	origin	synonym	ref ^α	phenotype ^β	cytology	molecular biology ^γ
<i>Kr</i> ²	EMS		3, 5	S		
<i>Kr</i> ³	EMS		3, 5	S		
<i>Kr</i> ⁵	EMS	6A69	5	S		
<i>Kr</i> ⁷	EMS		3, 5	S		
<i>Kr</i> ⁸	EMS		3, 5	S		
<i>Kr</i> ⁹	EMS		3-5	S; no MT		cys → ser
<i>Kr</i> ^{11δ}	X ray	<i>UR1</i>	3	S	<i>In(2R)58A;60F3</i>	-0 to +9
<i>Kr</i> ^{12δ}	X ray	<i>API</i>	3	S		+1 to >+34
<i>Kr</i> ^{13δ}	X ray	<i>AK1</i>	3	S	<i>T(Y;2)60F3-5</i>	
<i>Kr</i> ¹⁵	spont	<i>Kr</i> ¹¹	3, 5	S		+5 to >+34
<i>Kr</i> ¹⁶	EMS	<i>Kr</i> ¹¹ <i>A102</i>	3, 5	I		
<i>Kr</i> ¹⁷	EMS	<i>Kr</i> ^{IV}	1, 3, 5	I		phe → ile
<i>Kr</i> ¹⁸	EMS	<i>Kr</i> ^{VI}	3, 5	I		
<i>Kr</i> ¹⁹	EMS	<i>Kr</i> ¹ ⁰⁶⁶	1-3, 5	W; MT short		gly → glu
<i>Kr</i> ²⁰	EMS	<i>Kr</i> ¹¹ <i>ES7</i>	3, 5	W		
<i>Kr</i> ²¹	EMS	<i>Kr</i> ^V	1-3, 5	W; MT variable		cys → stop
<i>Kr</i> ²²	EMS	<i>Kr</i> ^{VII}	3, 5	W		
<i>Kr</i> ²³	EMS	<i>Kr</i> ^{VIII}	3, 5	W		
<i>Kr</i> ²⁴	EMS	<i>Kr</i> ^{IX}	3, 5	W		
<i>Kr</i> ²⁵	EMS	<i>Kr</i> ^X	1-3, 5	W; MT variable		tyr → asn
<i>Kr</i> ²⁶	EMS	<i>Kr</i> ^{XI}	3, 5	W		
<i>Kr</i> ²⁷	EMS	<i>Kr</i> ^{XII}	3, 5	W		
<i>Kr</i> ²⁸	P	<i>Kr</i> ²¹⁻¹¹	2	S		
<i>Kr</i> ²⁹	P	<i>Kr</i> ⁶²⁻¹⁴	2	W		

^α 1 = Gaul, Redemann, and Jäckle, 1989, Proc. Nat. Acad. Sci. USA 86: 4599-4603; 2 = Harbecke and Janning, 1989, Genes Dev. 3: 114-22; 3 = Preiss, Rosenberg, Kienlin, Seifert, and Jäckle, 1985, Nature (London) 313: 27-32; 4 = Redemann, Gaul, and Jäckle, 1988, Nature (London) 332: 90-92; 5 = Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

^β S = Strong alleles: segments T1-3 and A1-5 deleted; A6 and sometimes A7 and 8 duplicated with reverse polarity. I = Intermediate alleles: segments T1-3 and A1-4 deleted; duplicated segments not observed. W = Weak alleles: segments T2 and 3 and A1 deleted; A2-4 variably deleted depending on allele. MT = Malpighian tubules.

^γ Approximate extents of molecular deletions on coordinates of Preiss *et al.*, 1985; amino acid substitutions in zinc-finger region (Gaul *et al.*, 1989) as indicated by sequence analysis.

^δ Recovered as revertants of *If*.

cytology: Placed in 60F3 based on proximal breakpoint of *In(2R)Kr*^{UR1} and the 2R breakpoint of *T(Y;2)Kr*^{AK1}; mutant phenotype associated with five *Kr* deficiencies (Preiss *et al.*, 1985).

molecular biology: *Kr* was cloned by microdissection from the salivary band 60F3 followed by chromosome walking. A physical map was constructed from 50 kb of the *Kr* region in a series of overlapping clones (Preiss *et al.*, 1985). *Kr*⁺ activity contained in a 4 kb subsegment (approximate coordinates +5 to +9) was identified by the region of overlap between molecular deletions *Kr*¹¹ and *Kr*¹⁵. DNA sequencing indicates a single open reading frame of 1,398 bp split by a 372 kb intron and encoding a putative protein of about 466 amino acids (Rosenberg *et al.*, 1986). The protein shares structural elements with the DNA-binding zinc-finger domain of TFIIIA, a *Xenopus* transcription factor [Schuh *et al.*, 1986; Gaul *et al.*, 1987; Stanojevic *et al.*, 1989, Nature (London)

341: 331-35]. This gene product binds to the sequence AAGGGGTAA, the binding sites being located upstream of the *hb* promoter (Stanojevic *et al.*, 1989; Treisman and Desplan, 1989). One cloned segment rescues *Kr* embryos when injected and identifies a 2.5 kb blastoderm-specific polyadenylated RNA (Preiss *et al.*, 1985). Injection of anti-sense RNA into wild-type embryos produces *Kr/Kr* phenocopies (Rosenberg *et al.*, 1985). Transcripts are first detected at the syncytial blastoderm stage, and can be demonstrated at the cellular blastoderm in the central part of the embryo (Knipple *et al.*, 1985). In the absence of *hb* expression, the *Kr*⁺ transcript domain extends anteriorly up to segment A3 (Jäckle *et al.*, 1986); in the absence of *kni* expression, the extent of this domain is posterior to its normal boundary. At the start of gastrulation, an anterior transcript domain appears between head and thorax. Later, the transcripts are restricted to certain cells of the the brain and nervous system, to the mesoderm, and to the Malpighian tubules (Knipple *et al.*, 1985). At the beginning of germ-band shortening, there is a very low level of transcript accumulation throughout the embryo. The protein domain of *Kr* involves the activity of maternal effect genes and two adjacent gap genes, *hb* and *kni* (Gaul *et al.*, 1987). The *Kr* protein can be detected in the middle of the embryo (by antibody staining) at syncytial blastoderm and is demonstrated in a wide band of increasing density during cellular blastoderm (Gaul and Jäckle, 1987, 1989). Antibody staining is strongest in the center of the domain, growing weaker toward the anterior and posterior margins. The band in the middle of the embryo decreases in width at the end of cellularization and two more protein domains have been established as a stripe and a posterior cap. Domains of protein expression in *hb* and *Kr* overlap in wild-type embryos (Gaul and Jäckle, 1989) as do the protein domains of *Kr* and *kni* [Hülskamp, Pfeifle, and Tautz, 1990, *Nature* (London) 346: 577-80].

krotzkopf verkehrt: see *kkv*

KS: Male fertility complex on the short arm of the Y chromosome

synonym: see *KL*.

references: Brosseau, 1960, *Genetics* 45: 257-74.

Kennison, 1981, *Genetics* 98: 529-48.

1983, *Genetics* 103: 219-34.

Hazelrigg, Formili, and Kaufman, 1982, *Chromosoma* 87: 535-59.

Gatti and Pimpinelli, 1983, *Chromosoma* 88: 349-73.

The male fertility complex of the short arm of the Y chromosome, originally called *K2* by Stern (1929, *Z. Indukt. Abstamm. Vererbungsl.* 51: 253-353) and subsequently called *KS* by Brosseau (1960, *Genetics* 45: 257-74), contains two complementing units designated *ks-1* and *ks-2*.

ks-1

phenotype: Males deficient for *ks-1* sterile. Cross sectional profiles of sperm tails in coiled bundles abnormal in outline; minute tubules ordinarily present among individualized spermtails in coiled bundles missing; replaced by membrane bound vesicles [Hardy, Tokuyasu, and Lindsley, 1981, *Chromosoma* 83: 593-617 (fig.)]. Within the *ks-1* fertility region of the spermatocyte nucleus, a loop-forming site has been demonstrated by

antibody staining using the monoclonal S5 (Bonaccorsi *et al.*, 1988). The loops are not visible in living preparations, but like the *kl-5* loops, can be stained with the dye CBB.

alleles: In addition to the alleles tabulated, Williamson (1972, *Mol. Gen. Genet.* 119: 43-47) reported 26 non-complementing and nine complementing alleles induced by ethyl methanesulfonate.

allele	origin		discoverer	synonym	ref ^α
	treatment	treated			
<i>ks-1 B2</i>	X ray	y ⁺ Y	Brosseau	<i>ms(Y)S2</i>	1, 2
<i>ks-1 B4</i>	X ray	y ⁺ Y	Brosseau	<i>ms(Y)S4</i>	1
<i>ks-1 B6</i>	X ray	y ⁺ Y	Brosseau	<i>ms(Y)S6</i>	1
<i>ks-1 B7</i>	X ray	y ⁺ Y	Brosseau	<i>ms(Y)S7</i>	1
<i>ks-1 B8</i>	X ray	y ⁺ Y	Brosseau	<i>ms(Y)S8</i>	1
<i>ks-1 B10</i>	X ray	y ⁺ Y	Brosseau	<i>ms(Y)S10</i>	1
<i>ks-1 B11</i>	X ray	y ⁺ Y	Brosseau	<i>ms(Y)S11</i>	1
<i>ks-1 B12</i>	X ray	y ⁺ Y	Brosseau	<i>ms(Y)S12</i>	1
<i>ks-1 B13</i>	X ray	y ⁺ Y	Brosseau	<i>ms(Y)S13</i>	1
<i>ks-1 E1</i>	EMS	B ^S Yy ⁺	Kennison		4
<i>ks-1 G1</i>	γ ray	B ^S Yy ⁺	Kennison		4
<i>ks-1 X1</i>	X ray	B ^S Yy ⁺		<i>Sms1 A34</i>	3
<i>ks-1 X2</i>	X ray	B ^S Yy ⁺		<i>Sms1 A35</i>	3

^α 1 = Brosseau, 1960, *Genetics* 45: 257-74; 2 = CP627; 3 = Hazelrigg, Formili, and Kaufman, 1982, *Chromosoma* 87: 535-59; 4 = Kennison, 1983, *Genetics* 103: 219-34.

cytology: *Y-h21* and *h23*.

ks-2

phenotype: Males deficient for *ks-2* are sterile. The earliest departure from normal spermiogenesis is the abnormal alignment of the axoneme with the furrow demarcating the two halves of the nebenkern. Subsequent development of the major and minor mitochondrial derivatives and their interactions with endoplasmic reticulum grossly abnormal [Hardy, Tokuyasu, and Lindsley, 1981, *Chromosoma* 83: 593-617 (fig.)].

alleles: In addition to the tabulated alleles, Williamson (1972, *Mol. Gen. Genet.* 119: 43-47) reported eleven noncomplementing and eleven complementing alleles induced by ethyl methanesulfonate.

allele	origin		discoverer	synonym	ref ^α
	treatment	treated			
<i>ks-2 B5</i>	X ray	y ⁺ Y	Brosseau	<i>ms(Y)S5</i>	1, 2
<i>ks-2 E2</i>	EMS	B ^S Yy ⁺	Kennison		5
<i>ks-2 G2</i>	γ ray	B ^S Yy ⁺	Kennison		5
<i>ks-2 G3</i>	γ ray	B ^S Yy ⁺	Kennison	<i>ms(Y)G3</i>	4
<i>ks-2 X1</i>	X ray	B ^S Yy ⁺		<i>Sms2 A45</i>	3

^α 1 = Brosseau, 1960, *Genetics* 45: 257-74; 2 = CP627; 3 = Hazelrigg, Formili, and Kaufman, 1982, *Chromosoma* 87: 535-59; 4 = Kennison, unpublished information; 5 = Kennison, 1983, *Genetics* 103: 219-34.

cytology: *Y-h25*.

KS63: see *mud*

kto: kohtalo (J.A. Kennison)

location: 3-46.

origin: Induced by ethyl methanesulfonate.

discoverer: Kennison, 1983.

references: Kennison and Tamkun, 1988, *Proc. Nat. Acad. Sci. USA* 85: 8136-40.

phenotype: Isolated as a dominant suppressor of *Pc* mutations. Also suppresses *Pcl* alleles as well as *Msc*, *Mrt*, and *Antp*^{Scx}. Associated with recessive larval lethality.

THE GENOME OF *DROSOPHILA MELANOGASTER*

alleles: One mutant allele, called *kto*¹.

cytology: Placed in 76B1-76D5 based on *Df(3L)kto2 = Df(3L)76B1-2;76D5* (Ashburner).

kudu: see *kdu*

kug: *kugelei*

location: 3-47.

origin: Induced by ethyl methanesulfonate.

discoverer: Nüsslein-Volhard.

references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Female sterile. Mature eggs not laid and almost round.

alleles: Nineteen alleles.

Kugel: see *Kg*

Kun: see *Fs(3)Sz17*

kurz: see *kz*

Kynurenine formamidase: see *Kf*

kz: *kurz*

location: 1-0.9.

discoverer: Stern, 26a23.

references: Stern, 1930, Z. Indukt. Abstamm. Vererbungsl. 53: 279-86.

1934, DIS 1: 35.

Gvozdev, Gostimsky, Gerasimova, Dubrovskaya, and Braslavskaya, 1975, Mol. Gen. Genet. 141: 269-75.

Gvozdev, Gerasimova, Kovalev, and Ananiev, 1977, DIS 52: 67-8.

Perrimon, Engstrom, and Mahowald, 1984, Genetics 108: 559-72.

Haenlin, Stellar, Pirrotta, and Mohier, 1985, Cell 40: 827-37.

phenotype: In the viable allele *kz*¹, bristle are shorter and finer, like a slight Minute (Stern, 1930, 1934). Postscutellars often absent. Mutant hatches somewhat late. Viability fair; both sexes fertile. Recessive lethal alleles are slow to develop. First or second instar larvae stop developing; larvae may survive up to one week in arrested state (Perrimon *et al.*, 1984). All lethal alleles are cell lethal in germ-line clones. *kz*¹ females, heterozygous for a deficiency for the locus or for a lethal allele, emerge occasionally, but only after a delay of several days (Perrimon *et al.*, 1984).

allele:

allele	origin	synonym	ref ^α	comments
<i>kz</i> ¹	spont		7, 8	viable; fertile
<i>kz</i> ²	EMS	<i>kz</i> ⁴	1	lethal
<i>kz</i> ³	EMS	<i>kz</i> ⁵	1	lethal

allele	origin	synonym	ref ^α	comments
<i>kz</i> ⁴	EMS	<i>kz</i> ¹⁷	1	lethal
<i>kz</i> ⁵	EMS	<i>kz</i> ²¹	1	lethal
<i>kz</i> ⁶	EMS	<i>kz</i> ³²	1	lethal
<i>kz</i> ⁷	EMS	<i>kz</i> ³⁴	1	lethal
<i>kz</i> ⁸	EMS	<i>kz</i> ³⁶	1	lethal
<i>kz</i> ⁹	EMS	<i>kz</i> ⁴¹	1	lethal
<i>kz</i> ¹⁰	EMS	<i>kz</i> ⁴⁷	1	lethal
<i>kz</i> ¹¹	EMS	<i>kz</i> ⁵⁵	1	lethal
<i>kz</i> ¹²	EMS	<i>kz</i> ⁶¹	1	lethal
<i>kz</i> ¹³	EMS	<i>kz</i> ⁶³	1	lethal
<i>kz</i> ¹⁴	EMS	<i>kz</i> ⁶⁵	1	lethal
<i>kz</i> ¹⁵	EMS	<i>kz</i> ⁶⁷	1	lethal
<i>kz</i> ¹⁶	EMS	<i>kz</i> ⁷⁷	1	lethal
<i>kz</i> ¹⁷	EMS	<i>kz</i> ⁸⁰	1	lethal
<i>kz</i> ¹⁸	EMS	<i>kz</i> ⁸⁸	1	lethal
<i>kz</i> ¹⁹	EMS	<i>kz</i> ⁹⁷	1	lethal
<i>kz</i> ²⁰	X ray	<i>l(1)C33</i>	4	lethal;
<i>kz</i> ²¹	X ray	<i>l(1)C115</i>	4	lethal
<i>kz</i> ²²	X ray	<i>l(1)GA26</i>	4	lethal;
<i>kz</i> ²³	X ray	<i>l(1)HA90</i>	2, 4, 6	lethal
<i>kz</i> ²⁴	X ray	<i>l(1)RA34</i>	4	lethal
<i>kz</i> ²⁵	EMS	<i>l(1)DC776</i>	5, 6	lethal
<i>kz</i> ²⁶	EMS	<i>l(1)DF942</i>	5, 6	lethal
<i>kz</i> ²⁷	EMS	<i>l(1)EF469</i>	5	lethal
<i>kz</i> ²⁸	EMS	<i>l(1)EF514</i>	5	lethal
<i>kz</i> ²⁹	EMS	<i>l(1)VA217</i>	5	lethal
<i>kz</i> ³⁰	EMS	<i>l(1)VA252</i>	5	lethal
<i>kz</i> ³¹	EMS	<i>l(1)VA296</i>	5, 6	lethal
<i>kz</i> ³²	EMS	<i>l(1)VA337</i>	5	lethal
<i>kz</i> ³³	EMS	<i>l(1)VE748</i>	5	lethal
<i>kz</i> ³⁴	HMS ^β	<i>l(1)HM13</i>	3	lethal
<i>kz</i> ³⁵	HMS ^β	<i>l(1)HM51</i>	3	lethal
<i>kz</i> ³⁶	HMS ^β	<i>l(1)HM405</i>	3	lethal
<i>kz</i> ³⁷ ^γ	HD	<i>l(1)L271</i>	5	lethal

^α 1 = Gvozdev, Gerasimova, Kovalev, and Ananiev, 1977, DIS 52: 67-8; 2 = Haenlin, Stellar, Pirrotta, and Mohier, 1985, Cell 40: 827-37; 3 = Kramers, Schalet, Paradi, and Huiser-Hoogteyting, 1983, Mutat. Res. 107: 187-201; 4 = Lefevre, 1981, Genetics 99: 461-80; 5 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 6 = Perrimon, Engstrom, and Mahowald, 1984, Genetics 108: 559-72; 7 = Stern, 1930, Z. Indukt. Abstamm. Vererbungsl. 53: 279-86; 8 = Stern, 1934, DIS 1: 35.

^β HMS = hycanthon methanesulfonate.

^γ Discoverer: Sobels.

cytology: Placed in polytene band 2E3 by Lefevre (1981, Genetics 99: 461-80) and in 2E1-3 by Gvozdev (*et al.*, 1977).

molecular biology: The gene *kurz* was located in a clone microdissected from the 2E2-2F3 region of the salivary chromosomes; this clone (*cos9*), containing a 43 kb insert, was able to rescue the lethal mutant *kz*²³; viable non-*FM7* males were produced from a cross between heterozygous *kz*²³/*FM7* females and males carrying the cosmid on an autosome (Haenlin *et al.*, 1985).

L: Lobe

location: 2-72.0.

phenotype: Variable reduction in eye size of heterozygotes and homozygotes as well as in homozygous viability, depending on allele. Most extreme allele, L^2 , exhibits reduced eyes in heterozygote and tiny eyes and poor viability in homozygote; classifiable in single dose in triploids (Schultz, 1934, DIS 1: 55). $L^1/+$ have slightly reduced eyes with nick in anterior edge and the lower half of eye reduced more than upper; overlaps wild type; eyes of homozygote much smaller and less variable; bipartite eyes occasionally formed. L^r shows weakest expression, with the heterozygote indistinguishable from wild type and the homozygote with small kidney-shaped eyes at 25°, but overlapping wild type at 19°. Tested alleles (L^4 , L^B , L^d , L^r) exhibit reduced expression at 19° compared to 25°. Expression enhanced (L^1 , L^2 , L^4) in combination with Minutes [$M(2)58F$, $M(3)69E$, $M(3)95A$] (Dunn and Coyne, 1935, Biol. Zentr. 55: 385-89). Reduced numbers of cells enter into formation of eye disks (L^2 , L^4 , L^5 , Steinberg, 1944, Proc. Nat. Acad. Sci. USA 30: 5-13); reduced size of cephalic complex detectable in 24-hr larva, but subsequent growth rate is similar to wild type (L^4 , Medvedev, 1935, Z. Indukt. Abstamm. Vererbungsl. 70: 55-72; Tr. Inst. Genet. Akad. Nauk. SSSR 10: 119-51).

alleles:

allele	origin	discoverer	synonym	ref ^α	phenotype ^β
L^1	spont	Bridges, 18i24		2, 10	
L^2	spont	Mohr, 20b2		2, 9, 10	
L^3	spont	Bridges, 24d10		2, 10	$L^1 > L^3 > L^r$
L^4	spont	Sturtevant, 23f	L^Y	2	$L^2 > L^4 > L^1$
L^5	spont	Mohr, 31k26		2, 3	$L^1 > L^5 > L^r$
L^{34}	spont	Glass, 1934		2, 4	$L^2 > L^{34} > L^4$
L^{52}	spont	Nakayama, 52c		2, 11	$= L^1$
L^B	spont	Becker		1a, 2	$L^2 > L^B > L^1$
L^d	spont	Kodani		2, 12	$= L^1$
L^{dq}	spont	Kadel & Jenkins, 55g	dq	2, 5, 6	$= L^r$
L^K	spont	Krivshenko, 1957		2, 7	$L^2 > L^K > L^1$
L^r	spont	L. V. Morgan, 29h23		2	
L^{rm}	spont			2a	strong allele, outgrowths at anterior edge of eye
L^{ro}	Ultraviolet	Edmondson, 49k		2, 8	$L^2 > L^{ro} > L^4$
L^{rv1}	X ray			1	cytology normal
L^{rv3}	X ray			1	heterochromatic rearrangement
L^{rv5}	X ray			1	$In(2LR)26F;51B5C2$
L^{si}	spont	Morgan, 1932			$L^1 > L^{si} > L^r$

^α 1 = Baker and Ridge, 1980, Genetics 94: 383-423; 1a = Becker, Z. Indukt. Abstamm. Vererbungsl. 88: 333-73 (fig.); 2 = CP627; 2a = Craymer, 1980, DIS 55: 197-206; 3 = Dunn, 1935, DIS 4: 14; 4 = Glass, 1939, DIS 12: 47; 5 = Kadel, 1956, DIS 30: 73-74; 6 = Kadel, 1957, DIS 31: 83; 7 = Krivshenko, 1958, DIS 32: 81; 8 = Meyer, Edmondson, Byers, and Erickson, 1950, DIS 24: 60; 9 = Mohr, 1924, Z. Indukt. Abstamm. Vererbungsl. 32: 216; 10 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 230 (fig.); 11 = Nakayama, 1953, DIS 37: 59; 12 = Zimm, 1951, J. Exp. Zool. 116: 289-319 (fig.).

^β Strength of phenotypic expression, graded with respect to standard phenotypes L^1 , L^2 , and L^r .

^γ In $In(2L+2R)Cy$, therefore only heterozygote testable. Sectors of ommatidia replaced by chitin and bristles; lower half of head also reduced at 25°. Lower half of eye apparently produced from fewer than the normal nine or ten presumptive ommatidium-producing cells. Temperature-sensitive period for ommatidium formation first and second instars; third instar as well for head reduction.

cytology: Placed in 51A2-B1 based on its inclusion in

$Df(2L)L4 = Df(2L)51A2;51A12-B1$ (Baker and Ridge, 1980, Genetics 94: 383-423).

l: lethal

Some gene functions are dispensable to survival of flies in culture, whereas others are vital to survival such that amorphic alleles are lethal. Intermediate degrees of dispensability and hypomorphic alleles lead to intermediate situations. Whether a vital locus is named according to the phenotype of a hypomorphic allele or an escaper from a usually lethal genotype, or as a lethal is largely an accident of circumstance. Add to this uncertainty the recent practice of naming lethal mutants according to their lethal phenotype rather than as lethals, and the inconsistencies of nomenclatural usage become severe. In this revision, when a specific phenotype, either in the adult or an immature stage, can be associated with a mutation, that phenotype is used in its designation; otherwise, more general terms such as "lethal" are applied. In this regard, stage of death is not necessarily considered a specific phenotype.

In this revision an attempt is made to generate some order in the nomenclature for lethal alleles, which have heretofore been designated in a most capricious manner. Inasmuch as the cytological map has developed into such an exquisite instrument for gene localization, the method of indicating lethal loci currently used by Wright for the lethals in regions 37B and C has been adopted insofar as possible to the entire genome. Lethals are named according to the lettered subdivision of the polytene map that they occupy; separate loci are differentiated by lower case letters, e.g., $l(1)IAa$, and $l(1)IAb$, etc. for lethals in region 1A; in case a lettered subdivision has more than 26 lethal complementation groups, $l(1)Az$ will be followed by $l(1)Aaa$, $l(1)Abb$, etc. Since cytogenetic mapping is imperfect, this system of nomenclature cannot be expected to be perfect; there are bound to be instances where the lethal name misidentifies the position of the locus, being slightly off in most cases, but seriously so in a few. The order in which loci within a subdivision are named is arbitrary, although when the linear order is known, it may be reflected in the locus designations; however, **alphabetical order is not a reliable indication of map order!** Where the cytological information is available, lethal loci in a lettered subdivision are tabulated in bold face according to lettered subdivision and their alternative designation(s) indicated in body type. Lethal mutations named according to their more specific phenotypes are tabulated according to lettered subdivision in body type and their current designation in bold face in the column headed "synonym", e.g., **$l(1)IAa = l(1)EC1$** and **$l(1)IAe = ewg$** . Tabulations of loci in a lettered subdivision are generally followed by tabulations of the known alleles at each locus. Lethals named according to their more specific phenotypes are described and their alleles listed under the more specific name and not with the lethals. Where the cytological locations are unavailable, lethals are listed as in "Genetic Variations of *Drosophila melanogaster*", but where possible, groups of lethals with some aspect in common are designated alike and tabulated. As cytological determinations and complementation tests are made, additional lethals can be renamed according to cytological conventions and non-complementing lethals listed under the same locus sym-

bol. The column designated "comments" contains a variety of information. The notations represented by upper case letters indicate the lethal phase: E = embryonic; L1, L2, L3 = larval instars; EP = early pupa; LP = late pupa; A = adult; PP = polyphasic. In the case of X-chromosome lethals, notations with a slash bar are from Perrimon (e.g. Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14; 1989, Genetics 121: 333-52). The letters preceding the slash bar indicate the lethal phase; the letters following the slash bar indicate the behavior of homozygous oogenic clones in otherwise heterozygous females: NME = no maternal effect, i.e. the lethal phenotype of males produced by homozygous germ-line clones is indistinguishable from that of males produced by heterozygous mother; ME = maternal effect; MER = maternal effect partially rescued by paternal +; VME = variable maternal expression; AO = abnormal oogenesis; L = lethal. Rearrangements listed as associated with lethals are described under the appropriate rearrangement type, usually with breakpoints indicated.

For those numbered divisions in which saturation mutagenesis has been carried out, the lethal tabulations are followed by tables of the deficiency mapping results for the region. Cytological localizations of deficiency breakpoints are presented even though some are at variance with the genetic data. The orders indicated cannot be completely accurate, since all lethals have not been tested with all indicated deficiencies.

l(1)

Lethal mutations on the X chromosome are among the easiest to recover and the least convenient to characterize genetically. Accordingly they comprise the most represented and least characterized subset of lethal mutations. There are doubtlessly numerous instances of undetected allelism and therefore, in the following treatment, the number of named loci must substantially exceed the number actually represented.

***l(1)1: lethal (1) 1**

location: 1-1.1.

origin: Spontaneous.

discoverer: Rawls, 12b.

references: 1913, Biol. Bull. 24: 115-24.

Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. No. 237: 31.

other information: First recessive lethal found in *D. melanogaster*.

l(1)1A

Eight lethally mutable loci tentatively identified in region 1A; these include the former *l(1)J1* of Jacobs-Muller, *pch*, *cin*, *ewg*, and *arth*. Order established by deficiency mapping by Fleming, Schalet, Lim, and White; Complementation tests between mutants described by Maddern and the remainder not carried out; allelic relations arbitrarily assigned.

locus	genetic		cytological		included in	excluded from	synonym
	location	location	location	location			
<i>l(1)1Aa</i>	1-0.0		<i>Df(1)SJ1b</i>	<i>Df(1)y75e</i>	<i>dmd</i> , <i>l(1)EC1</i> , <i>pch</i>		
<i>l(1)1Ac</i>	1-0.0	1A6	<i>Df(1)y75e</i>	<i>Df(1)cin-arth</i>	<i>l(1)J1</i>		
<i>l(1)1Ad</i>	1-0.0	1A5	<i>Df(1)SJ1d</i>	<i>Df(1)SJ1b</i>	<i>l(1)EC2</i>		
<i>l(1)1Ae</i>	1-0.0	1A7	<i>Df(1)SJ1a</i>	<i>Df(1)SJ1d</i>	<i>cln</i>		
<i>l(1)1Af</i>	1-0.0	1A7-8					

locus	genetic		cytological		included in	excluded from	synonym
	location	location	location	location			
<i>l(1)1Ag</i>	1-0.0	1A8	<i>Df(1)SJ1a</i>	<i>Df(1)SJ1d</i>	<i>Df(1)sc19</i>		<i>ewg</i> , <i>l(1)EC3</i>
<i>l(1)1Ah</i>	1-0.0		<i>Df(1)sc19</i>	<i>Df(1)SJ1a</i>			<i>arth</i>

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)1Ac</i>¹	X ray	Jacobs-Muller	<i>l(1)J1</i>	1	<i>In(1)sc</i> ^{J4}
<i>l(1)1Ac</i>²	X ray	Meyer	<i>l(1)HM1</i>		
<i>l(1)1Ac</i>³	X ray	Meyer	<i>l(1)HM2</i>		
<i>l(1)1Ac</i>⁴	EMS	Lim	<i>l(1)J1</i> ¹		
<i>l(1)1Ac</i>⁵	EMS	Lim	<i>l(1)J1</i> ⁶⁹		
<i>l(1)1Ac</i>⁶	EMS	Lim	<i>l(1)J1</i> ⁷¹		
<i>l(1)1Ac</i>⁷	EMS	Lim	<i>l(1)J1</i> ⁸⁹		
<i>l(1)1Ac</i>⁸	EMS	Lim	<i>l(1)J1</i> ⁹⁶		
<i>l(1)1Ac</i>⁹	EMS	Lim	<i>l(1)J1</i> ¹¹⁵		
<i>l(1)1Ac</i>¹⁰	EMS	Lim	<i>l(1)J1</i> ¹²⁹		
<i>l(1)1Ac</i>¹¹	MMS	Lim	<i>l(1)J1</i> ^{M7}		
<i>l(1)1Ac</i>¹²	X ray	Lefevre	<i>l(1)RF17</i>	2	
*<i>l(1)1Ac</i>¹³	X ray	Lefevre	<i>l(1)G11</i> <i>l(1)G1</i> <i>l(1)L61</i>	2	<i>T(1;2)1A5;32F1-2</i>
*<i>l(1)1Ac</i>¹⁴	X ray	Lefevre	<i>l(1)HA35</i>	2	complex aberration
*<i>l(1)1Ac</i>¹⁵	X ray	Lefevre	<i>l(1)JC56</i>	2	<i>T(1;3)1A5;66C1</i>
*<i>l(1)1Ac</i>¹⁶	X ray	Lefevre	<i>l(1)HF341</i>	2	complex aberration
<i>l(1)1Ac</i>¹⁷	EMS	Lefevre	<i>l(1)EA106</i>	3	L/NME
<i>l(1)1Ac</i>¹⁸	spont	Schalet	<i>l(1)19-30DP</i>	5	
<i>l(1)1Ac</i>¹⁹	spont	Schalet	<i>l(1)20-97</i>	5	
<i>l(1)1Ac</i>²⁰	EMS	White			
<i>l(1)1Ac</i>²¹	EMS	White			
<i>l(1)1Ac</i>²²	EMS	White			
<i>l(1)1Ac</i>²³	EMS	White			
<i>l(1)1Ac</i>²⁴	EMS	White			
<i>l(1)1Ac</i>²⁵	EMS	White			
<i>l(1)1Ac</i>²⁶	X ray	White			
<i>l(1)1Ad</i>¹	spont	Maddern	<i>l(1)2804</i>	4	
<i>l(1)1Ad</i>²	EMS	Lim	<i>l(1)EC2</i> ⁵⁷	2	
<i>l(1)1Ad</i>³	EMS	Lefevre	<i>l(1)VE619</i>	3	
<i>l(1)1Ad</i>⁴	EMS	Lefevre	<i>l(1)VE899</i>	3	L3/L
<i>l(1)1Ad</i>⁵	EMS	White			
*<i>l(1)1A1</i>¹	X ray	Lefevre	<i>l(1)HC107</i>	2	complex aberration

^α 1 = CP627; 2 = Lefevre, 1981, Genetics 99: 461-80; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 4 = Maddern, 1977, DIS 52: 82; 5 = Schalet, 1986, Mutat. Res. 163: 115-44.
^β From DNA-injected egg.

l(1)1B

Eleven lethally mutable loci identified in region 1B including named loci *sc*, *svr*, *elav*, *vnd*, and *M(1)Bld*.

locus	genetic		cytological		included in	excluded from	synonym
	location	location	location	location			
<i>l(1)1Ba</i>	1-0.0	1B4	<i>In(1)sc</i> ⁴	<i>In(1)sc</i> ⁹			<i>l(1)sc</i>
<i>l(1)1Bb</i>	1-0.0		<i>Df(1)260.1</i>	<i>Df(1)sc19</i>			<i>l(1)EC4</i>
<i>l(1)1Bc</i>	1-0.0		<i>Df(1)yT6-151</i>	<i>Df(1)260.1</i>			<i>l(1)EC5</i>
<i>l(1)1Bd</i>	1-0.0	1B5-6	<i>Df(1)yT6-151</i>	<i>Df(1)260.1</i>			<i>svr</i>
<i>l(1)1Be</i>	1-0.0	1B4-9	<i>Df(1)yT7-155</i>	<i>Df(1)yT6-151</i>			<i>elav</i> , <i>l(1)EC7</i>
<i>l(1)1Bf</i>	1-0.0	1B9	<i>Df(1)yT8-184</i>	<i>Df(1)yT7-155</i>			<i>vnd</i> , <i>l(1)EC6</i>
<i>l(1)1Bg</i>	1-0.0	1B10-11	<i>Df(1)su(s)E2</i>	<i>Df(1)svr</i> <i>Df(1)su(s)83</i>			
<i>l(1)1Bh</i>	1-0.0		<i>Df(1)y74k10.1</i>	<i>Df(1)yT9-7</i>			
<i>l(1)1Bi</i>	1-0.0		<i>Df(1)y74k24.1</i>	<i>Df(1)y74k10.1</i>			
<i>l(1)1Bj</i>	1-0.0	1B11-12	<i>Df(1)y74k24.1</i>	<i>Df(1)y74k10.1</i>			<i>M(1)Bld</i>
<i>l(1)1Bk</i>	1-0.0		<i>Df(1)yT14-546</i>	<i>Df(1)yT13-464</i>			

l(1)1Bb

phenotype: Homozygous clones in eyes display swelling and vacuolization of pigment cells (alleles designated MM2 and TE10). Deletion for *l(1)1Bb* enhances central-nervous-system and eye effects of ASC deficiencies (Jiménez and Campos-Ortega, 1987, J. Neurogenet. 4: 179-200; González, Romani, Cubas, Modolell, and Campuzano, 1989, EMBO J. 8: 3553-62).

allele	origin α	discoverer	synonym	ref β	comments
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<i>l(1)1Bb</i> ¹	EMS	Lim	<i>l(1)EC4</i> ⁴	3	
<i>l(1)1Bb</i> ²	EMS	Lim	<i>l(1)EC4</i> ⁶		
<i>l(1)1Bb</i> ³	EMS	Lim	<i>l(1)EC4</i> ⁸		
<i>l(1)1Bb</i> ⁴	EMS	Lim	<i>l(1)EC4</i> ⁰¹¹		
<i>l(1)1Bb</i> ⁵	EMS	Lim	<i>l(1)EC4</i> ⁰¹³		
<i>l(1)1Bb</i> ⁶	EMS	Lim	<i>l(1)EC4</i> ⁰²¹		
<i>l(1)1Bb</i> ⁷	EMS	Lim	<i>l(1)EC4</i> ⁵⁴		
<i>l(1)1Bb</i> ⁸	EMS	Lim	<i>l(1)EC4</i> ⁶⁵		
<i>l(1)1Bb</i> ⁹	EMS	Lim	<i>l(1)EC4</i> ⁸¹		
<i>l(1)1Bb</i> ¹⁰	EMS	Lim	<i>l(1)EC4</i> ⁸⁶		
<i>l(1)1Bb</i> ¹¹	EMS	Lim	<i>l(1)EC4</i> ¹²⁰		
<i>l(1)1Bb</i> ¹²	MMS	Lim	<i>l(1)EC4</i> ^{M20}		
<i>*l(1)1Bb</i> ¹³	X ray	Lefevre	<i>l(1)A7</i>	3	
<i>*l(1)1Bb</i> ¹⁴	X ray	Lefevre	<i>l(1)C84</i>	3	
<i>l(1)1Bb</i> ¹⁵	X ray	Lefevre	<i>l(1)C35</i>	3	
<i>l(1)1Bb</i> ¹⁶	EMS	Lefevre	<i>l(1)DC724</i>	4	
<i>l(1)1Bb</i> ¹⁷	EMS	Lefevre	<i>l(1)VA265</i>	4	
<i>l(1)1Bb</i> ¹⁸	EMS	Lefevre	<i>l(1)VE637</i>	4	
<i>l(1)1Bb</i> ¹⁹	EMS	Lefevre	<i>l(1)VE859</i>	4	P/NME
<i>l(1)1Bb</i> ²⁰	spont	Schalet	<i>l(1)11-53</i>		
<i>l(1)1Bb</i> ²¹	EMS	White	<i>l(1)EC4</i> ^{mmz}		
<i>l(1)1Bb</i> ²²	EMS	White	<i>l(1)EC4</i> ^{TE10}		
<i>l(1)1Bb</i> ²³	EMS	White			
<i>l(1)1Bb</i> ²⁴	EMS	White			
<i>l(1)1Bb</i> ²⁵	EMS	White			
<i>l(1)1Bb</i> ²⁶	EMS	White			
<i>l(1)1Bb</i> ²⁷	EMS	White			
<i>l(1)1Bb</i> ²⁸	ENU	Voelker	<i>l(1)A74V</i>		
<i>l(1)1Bb</i> ²⁹	ENU	Voelker	<i>l(1)A84</i>		
<i>l(1)1Bb</i> ³⁰	ENU	Voelker	<i>l(1)A91</i>		
<i>l(1)1Bb</i> ³¹	ENU	Voelker	<i>l(1)A112</i>		
<i>l(1)1Bb</i> ³²	ENU	Voelker	<i>l(1)A133</i>		
<i>l(1)1Bb</i> ³³	HMS		<i>l(1)HM439</i>	2	
<i>l(1)1Bc</i> ¹	EMS	Lim	<i>l(1)EC5</i> ⁰¹⁷		
<i>l(1)1Bc</i> ²	EMS	Lim	<i>l(1)EC5</i> ⁴²		
<i>l(1)1Bc</i> ³	EMS	Lim	<i>l(1)EC5</i> ⁵⁸		
<i>l(1)1Bc</i> ⁴	EMS	Lim	<i>l(1)EC5</i> ¹¹⁹		
<i>l(1)1Bc</i> ⁵	EMS	Lim	<i>l(1)EC5</i> ¹²⁶		
<i>l(1)1Bc</i> ⁶	EMS	Lim	<i>l(1)EC5</i> ¹⁴³		
<i>l(1)1Bc</i> ⁷	TEM	Lim	<i>l(1)EC5</i> ^{T001}		
<i>l(1)1Bc</i> ⁸	TEM	Lim	<i>l(1)EC5</i> ^{T006}		
<i>l(1)1Bc</i> ⁹	TEM	Lim	<i>l(1)EC5</i> ^{T007}		
<i>*l(1)1Bc</i> ¹⁰	X ray	Lefevre	<i>l(1)RC10</i>	3	
<i>l(1)1Bc</i> ¹¹	X ray	Lefevre	<i>l(1)HA81</i>	3	
<i>l(1)1Bc</i> ¹²	EMS	Lefevre	<i>l(1)EC209</i>	4	
<i>l(1)1Bc</i> ¹³	EMS	Lefevre	<i>l(1)EC288</i>	4	
<i>l(1)1Bc</i> ¹⁴	EMS	Lefevre	<i>l(1)VA76</i>	4	L3/NME
<i>l(1)1Bc</i> ¹⁵	EMS	Lefevre	<i>l(1)VE648</i>	4	
<i>l(1)1Bc</i> ¹⁶	spont	Schalet	<i>l(1)6-4</i>		
<i>l(1)1Bc</i> ¹⁷	EMS	White			
<i>l(1)1Bc</i> ¹⁸	EMS	White			
<i>l(1)1Bc</i> ¹⁹	EMS	White			
<i>l(1)1Bc</i> ²⁰	ENU	Voelker	<i>l(1)A8</i>		
<i>l(1)1Bc</i> ²¹	ENU	Voelker	<i>l(1)A72</i>		
<i>l(1)1Bc</i> ²²	ENU	Voelker	<i>l(1)A113</i>		
<i>l(1)1Bc</i> ²³	ENU	Voelker	<i>l(1)A137</i>		
<i>l(1)1Bg</i> ¹	X ray	Lefevre	<i>l(1)C244</i>	3	complex aberration
<i>l(1)1Bg</i> ²	X ray	Lefevre	<i>l(1)HC147</i>	3	
<i>l(1)1Bg</i> ³	X ray	Lefevre	<i>l(1)HF379</i>	3	<i>T(1;3)1B11;67B + 69A</i> ; heterochromatin
<i>*l(1)1Bh</i> ¹	X ray	Lefevre	<i>l(1)C22</i>	3	complex aberration
<i>l(1)1Bh</i> ²	X ray	Lefevre	<i>l(1)C73</i>	3	

allele	origin α	discoverer	synonym	ref β	comments
<i>*l(1)1Bh</i> ³	X ray	Lefevre	<i>l(1)RF16</i>	3	
<i>l(1)1Bh</i> ⁴	EMS	Lefevre	<i>l(1)EF406</i>	4	PP/L
<i>l(1)1Bh</i> ⁵	EMS	White	<i>l(1)PB</i> ³		
<i>l(1)1Bh</i> ⁶	EMS	White	<i>l(1)PB</i> ^{TE6}		
<i>l(1)1Bh</i> ⁷	EMS	White	<i>l(1)PB</i> ^{LP9}		
<i>l(1)1Bh</i> ⁸	ENU	Voelker	<i>l(1)A90</i>	1	P
<i>l(1)1Bh</i> ⁹	ENU	Voelker	<i>l(1)B21</i>		semilethal; adults with short thin bristles

<i>l(1)1Bh</i> ¹⁰	ENU	Voelker	<i>l(1)B24</i>		
<i>l(1)1Bh</i> ¹¹	ENU	Voelker	<i>l(1)B28</i>		
<i>l(1)1Bh</i> ¹²	ENU	Voelker	<i>l(1)B31</i>		
<i>l(1)1Bh</i> ¹³	ENU	Voelker	<i>l(1)B33</i>		
<i>l(1)1Bh</i> ¹⁴	ENU	Voelker	<i>l(1)B34</i>		
<i>l(1)1Bh</i> ¹⁵	ENU	Voelker	<i>l(1)B35</i>		
<i>l(1)1Bh</i> ¹⁶	ENU	Voelker	<i>l(1)B40</i>		
<i>l(1)1Bh</i> ¹⁷	EMS	Voelker	<i>l(1)C3</i>		
<i>l(1)1Bh</i> ¹⁸	DEB	Voelker	<i>l(1)DB1</i>		

<i>l(1)1Bi</i> ¹	EMS	Lefevre	<i>l(1)DC703</i>	4	
<i>l(1)1Bi</i> ²	EMS	White	<i>l(1)D65</i>		
<i>l(1)1Bi</i> ³	HMS	Kramers	<i>l(1)HM52</i>	2	
<i>l(1)1Bi</i> ⁴	HMS	Kramers	<i>l(1)HM419</i>	2	
<i>l(1)1Bi</i> ⁶	ENU	Voelker	<i>l(1)A7</i>	1	L1-2
<i>l(1)1Bi</i> ⁷	ENU	Voelker	<i>l(1)A43</i>		
<i>l(1)1Bi</i> ⁸	ENU	Voelker	<i>l(1)A99</i>		
<i>l(1)1Bi</i> ⁹	ENU	Voelker	<i>l(1)A105</i>		
<i>l(1)1Bi</i> ¹⁰	ENU	Voelker	<i>l(1)B4</i>		
<i>l(1)1Bi</i> ¹¹	ENU	Voelker	<i>l(1)B10</i>		
<i>l(1)1Bi</i> ¹²	ENU	Voelker	<i>l(1)B30</i>		
<i>l(1)1Bi</i> ¹³	ENU	Voelker	<i>l(1)B36</i>		
<i>l(1)1Bi</i> ¹⁴	ENU	Voelker	<i>l(1)B38</i>		
<i>l(1)1Bi</i> ¹⁵	ENU	Voelker	<i>l(1)B48</i>		semilethal

<i>l(1)1Bk</i> ¹	EMS	Lefevre	<i>l(1)DF979</i>	4	
<i>l(1)1Bk</i> ²	HMS	Kramers	<i>l(1)HM441</i>	1	E-L1
<i>l(1)1Bk</i> ³	ENU	Voelker	<i>l(1)A24</i>		
<i>l(1)1Bk</i> ⁴	ENU	Voelker	<i>l(1)B3</i>		
<i>l(1)1Bk</i> ⁵	DEB	Voelker	<i>l(1)DB2</i>		

α Lethals in Voelker's B series were induced in *Dp(1;3)E1*.
 β 1 = Eberl, Hilliker, and Voelker, 1988, DIS 67: 36; 2 = Kramers, Schalet, Paradi, and Huizer-Hoogteyting, 1983, Mutat. Res. 107: 187-201; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Lefevre and Watkins, 1986, Genetics 113: 869-95.

l(1)1C

Two lethally mutable loci recognized, one of which is *mul*.

locus	included in	excluded from	synonym
<i>l(1)1Ca</i>	<i>Df(1)yT15-5</i>	<i>Df(1)yT14-546</i>	
<i>l(1)1Cb</i>	<i>Df(1)yT16-14</i>	<i>Df(1)yT15-5</i>	<i>mul</i>

allele β	origin	discoverer	synonym	ref α	comments
<i>*l(1)1Ca</i> ¹	X ray	Lefevre	<i>l(1)HA34</i>	2	
<i>*l(1)1Ca</i> ²	X ray	Lefevre	<i>l(1)HC111</i>	2	
<i>*l(1)1Ca</i> ³	X ray	Lefevre	<i>l(1)RC7</i>	2	
<i>*l(1)1Ca</i> ⁴	EMS	Lefevre	<i>l(1)EA148</i>	3	
<i>l(1)1Ca</i> ⁵	ENU	Voelker	<i>l(1)A19</i>	1	P
<i>l(1)1Ca</i> ⁶	EMS	Voelker	<i>l(1)C4</i>		
<i>l(1)1Ca</i> ⁷	EMS	Voelker	<i>l(1)C5</i>		

α 1 = Eberl, Hilliker, and Voelker, 1988, DIS 67: 36; 2 = Lefevre, 1981, Genetics 99: 461-80; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95.

β Lefevre's and Voelker's mutants have arbitrarily been declared alleles, since all of Lefevre's are lost and the alternative is to postulate two loci, one of which Lefevre sampled four times and the other of which Voelker mutated three times.

I(1)1D

Region 1D contains three lethally mutable loci, one of which is *brc*.

locus	included in	excluded from	synonym
I(1)1Da	<i>Df(1)yT18-319</i>	<i>Df(1)yT17-600</i>	
I(1)1Db	<i>Df(1)yT19-16</i>	<i>Df(1)yT18-319</i>	brc
I(1)1Dc		<i>Df(1)yT19-16</i> <i>Df(1)pn7b</i>	

allele	origin	discoverer	synonym	ref ^α	comments
I(1)1Da¹	HMS	Kramers	<i>I(1)HM454</i>	1, 2	L1-2
I(1)1Da²	ENU	Voelker	<i>I(1)A29</i>	1	L1-2
I(1)1Da³	ENU	Voelker	<i>I(1)A32</i>		
I(1)1Da⁴	ENU	Voelker	<i>I(1)A139</i>		
I(1)1Dc¹	X ray	Lefevre	<i>I(1)A118</i>	3	
I(1)1Dc²	X ray	Lefevre	<i>I(1)A146</i>	3	
I(1)1Dc³	X ray	Lefevre	<i>I(1)GA39</i>	3	
I(1)1Dc⁴	X ray	Lefevre	<i>I(1)GF326</i>	3	<i>T(1;2)1D4;36C</i> ; fails to complement <i>Df(1)sus)83</i>
I(1)1Dc⁵	X ray	Lefevre	<i>I(1)JC45</i>	3	
I(1)1Dc⁶	EMS	Lefevre	<i>I(1)DF917</i>	4	complex <i>T(1;?)1D4;het</i>
I(1)1Dc⁷	EMS	Lefevre	<i>I(1)VE846</i>	4	L/L

^α 1 = Eberl, Hilliker, and Voelker, 1988, DIS 67: 36; 2 = Kramers, Schalet, Paradi, and Huizer-Hoogteyling, 1983, Mutat. Res. 107: 187-201; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Lefevre and Watkins, 1986, Genetics 113: 869-95.

I(1)1E

Six lethally mutable loci, mostly identified by Lefevre.

locus	cytological		included in	excluded from
	location	location		
I(1)1Ea	1E2		<i>Df(1)S39</i>	<i>Df(1)A94</i>
I(1)1Eb	1E3		<i>Df(1)S39</i>	<i>Df(1)A94</i>
I(1)1Ec	1E4		<i>Df(1)A94</i>	
I(1)1Ed	1E5		<i>Df(1)A94</i>	
I(1)1Ee				
I(1)1Ef				

allele	origin	discoverer	synonym	ref ^α	comments
I(1)1Ea¹	EMS	Lefevre	<i>I(1)EA48</i>	4	L3-P/L
I(1)1Ea²	EMS	Lefevre	<i>I(1)VE737</i>	4	
I(1)1Eb¹	X ray	Lefevre	<i>I(1)N3</i>	3	<i>T(1;3)1E1;71C1</i> + complex other
I(1)1Eb²	EMS	Lefevre	<i>I(1)DA565</i>	4	L2-3/L
I(1)1Eb³	EMS	Lefevre	<i>I(1)DA681</i>	4	
I(1)1Eb⁴	EMS	Lefevre	<i>I(1)VA209</i>	4	L1-2/L
I(1)1Eb⁵	HMS		<i>I(1)HM14</i>	2	
I(1)1Ec¹	X ray	Lefevre	<i>I(1)A102</i>	3	L/NME
I(1)1Ec²	X ray	Lefevre	<i>I(1)RC66</i>	3	<i>T(1;2)1E4;58E</i>
I(1)1Ec³	X ray	Lefevre	<i>I(1)GA77</i>	3	L/
I(1)1Ec⁴	X ray	Lefevre	<i>I(1)GA119</i>	3	<i>T(1;3)1E1;93A</i>
I(1)1Ec⁵	EMS	Lefevre	<i>I(1)DA553</i>	3	
I(1)1Ec⁶	EMS	Lefevre	<i>I(1)DC755</i>	4	
I(1)1Ec⁷	EMS	Lefevre	<i>I(1)EA1</i>	4	
I(1)1Ec⁸	EMS	Lefevre	<i>I(1)VE606</i>	4	
I(1)1Ec⁹	HMS		<i>I(1)HM20</i>	2	
I(1)1Ec¹⁰	HMS		<i>I(1)HM53</i>	2	
I(1)1Ec¹¹	MR		<i>I(1)28/6A</i>	1	
I(1)1Ed¹	X ray	Lefevre	<i>I(1)HF358</i>	3	
I(1)1Ed²	X ray	Lefevre	<i>I(1)RC47</i>	3	
I(1)1Ed²	X ray	Lefevre	<i>I(1)RC66</i>	3	<i>T(1;2)1E4;58E</i>
I(1)1Ee¹	X ray	Lefevre	<i>I(1)HF308</i>	3	<i>Tp(1;3)1E4;12A;81</i>
I(1)1Ef¹	X ray	Lefevre	<i>I(1)C24</i>	3	E/NME

allele	origin	discoverer	synonym	ref ^α	comments
I(1)1Ef²	X ray	Lefevre	<i>I(1)GA91</i>	3	<i>T(1;3)1F4;99A</i>
I(1)1Ef³	X ray	Lefevre	<i>I(1)GE241</i>	3	E/NME
I(1)1Ef⁴	X ray	Lefevre	<i>I(1)HA29</i>	3	
I(1)1Ef⁵	EMS	Lefevre	<i>I(1)DC836</i>	4	
I(1)1Ef⁶	EMS	Lefevre	<i>I(1)VE625</i>	4	
I(1)1Ef⁷	EMS	Lefevre	<i>I(1)VE811</i>	4	

^α 1 = Eeken, Sobels, Hyland, and Schalet, 1985, Mutation Res. 150: 261-75; 2 = Kramers, Schalet, Paradi, and Huizer-Hoogteyling, 1983, Mutat. Res. 107: 187-201; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Lefevre and Watkins, 1986, Genetics 113: 869-95.

I(1)EF

Ten loci placed in 1E3-2A3 by Belyaeva and Aizenzon on the basis of their mapping to the region common to *Df(1)AT127* and *Df(1)RA19*. Not tested for allelism with *I(1)IE* or *I(1)IF* mutations of Lefevre.

locus	synonym
I(1)1EFa	<i>I(1)BA1</i>
I(1)1EFb	<i>I(1)BA2</i>
I(1)1EFc	<i>I(1)BA3</i>
I(1)1EFd	<i>I(1)BA4</i>
I(1)1EFe	<i>I(1)BA5</i>
I(1)1EFf	<i>I(1)BA6</i>
I(1)1EFg	<i>I(1)BA7</i>
I(1)1EFh	<i>I(1)BA8</i>
I(1)1EFi	<i>I(1)BA9</i>
I(1)1EFj	<i>I(1)BA10</i>

allele	origin	synonym	comments
I(1)1EFa¹	EMS	<i>I(1)t44</i>	
I(1)1EFa²	EMS	<i>I(1)t70</i>	
I(1)1EFa³	EMS	<i>I(1)t156</i>	
I(1)1EFa⁴	EMS	<i>I(1)t348</i>	
I(1)1EFa⁵	EMS	<i>I(1)t373</i>	
I(1)1EFb¹	EMS	<i>I(1)t11</i>	
I(1)1EFb²	EMS	<i>I(1)t34</i>	
I(1)1EFb³	EMS	<i>I(1)t130</i>	
I(1)1EFb⁴	EMS	<i>I(1)t155</i>	
I(1)1EFb⁵	EMS	<i>I(1)t170</i>	
I(1)1EFb⁶	EMS	<i>I(1)t192</i>	
I(1)1EFc¹	EMS	<i>I(1)t6</i>	
I(1)1EFc²	EMS	<i>I(1)t7</i>	
I(1)1EFc³	EMS	<i>I(1)t37</i>	
I(1)1EFc⁴	EMS	<i>I(1)t381</i>	
I(1)1EFc⁵	EMS	<i>I(1)t402</i>	
I(1)1EFd¹	EMS	<i>I(1)t9</i>	
I(1)1EFd²	EMS	<i>I(1)t49</i>	
I(1)1EFd³	EMS	<i>I(1)t153</i>	
I(1)1EFd⁴	EMS	<i>I(1)t163</i>	
I(1)1EFd⁵	EMS	<i>I(1)t242</i>	
I(1)1EFe¹	EMS	<i>I(1)t16</i>	
I(1)1EFe²	EMS	<i>I(1)t40</i>	
I(1)1EFe³	EMS	<i>I(1)t46</i>	
I(1)1EFe⁴	EMS	<i>I(1)t214</i>	
I(1)1EFe⁵	EMS	<i>I(1)t233</i>	
I(1)1EFe⁶	EMS	<i>I(1)t341</i>	
I(1)1EFf¹	EMS	<i>I(1)t42</i>	
I(1)1EFf²	EMS	<i>I(1)t101</i>	
I(1)1EFf³	EMS	<i>I(1)t186</i>	complements <i>I(1)EFf⁴</i>
I(1)1EFf⁴	EMS	<i>I(1)t55</i>	complements <i>I(1)EFf³</i>
I(1)1EFg¹	EMS	<i>I(1)t29</i>	
I(1)1EFg²	EMS	<i>I(1)t485</i>	
I(1)1EFh¹	X ray	<i>I(1)R1</i>	

allele	origin	synonym	comments	side	breakpoint	variant	DNA coordinates ^α
I(1)1EF1¹	EMS	<i>I(1)169</i>		left		<i>IJ1⁺Y</i>	100-103
I(1)1EF1¹	EMS	<i>I(1)147</i>		right		<i>Df(1)1-96</i>	136-140
				right		<i>Df(1)B428</i>	121-133
						<i>T(1;4)ewg^P</i>	109-118
						arth	
						y²	
				left	1B1-2	<i>In(1)y^{3P}</i>	73.7 to 72.3
						<i>Df(1)yT1a-394</i>	72.3 to 71.6
						<i>Df(1)yT1a-473</i>	72.3 to 71.6
						<i>Df(1)yT1a-653</i>	72.3 to 71.6
						T6	
				-5'			
				left	1A8-B1	<i>In(1)y⁴</i>	71.6 to 70.6
					1B1	y	
						<i>Df(1)yT1a-276</i>	71.5 to 69.5
						<i>Df(1)yT1a-741</i>	71.5 to 69.5
						<i>Df(1)yT1b-627</i>	69.5 to 68.7
						<i>Df(1)yT1b-733</i>	68.6 to 67.8
						<i>Df(1)yT1b-81</i>	67.8 to 67.7
						T6	
				3'		<i>Df(1)yT1b-94</i>	67.7 to 67.2
						<i>Df(1)yT1b-303</i>	67.2 to 66.3
						<i>Df(1)yT1b-628</i>	67.2 to 66.3
						<i>Df(1)yT1b-689</i>	67.2 to 66.3
						<i>Df(1)yT1b-497</i>	65.6 to 65.3
						<i>Df(1)yT1b-625</i>	65.3 to 64.8
						<i>Df(1)yT1b-488</i>	64.2 to 63.2
						<i>Df(1)yT1b-589</i>	62.9 to 62.4
						<i>Df(1)yT1b-195</i>	62.4 to 61.8
						<i>Df(1)yT1b-633</i>	62.4 to 61.8
						<i>Df(1)yT1b-659</i>	62.4 to 61.8
						<i>Df(1)yT1b-242</i>	61.2 to 60.3
						<i>Df(1)yT1b-331</i>	61.2 to 60.3
						<i>Df(1)yT1b-739</i>	60.3 to 59.9
						<i>Df(1)yT1b-691</i>	59.9 to 59.7
						<i>Df(1)yT1b-626</i>	59.7 to 58.9
				left	1B2-3	<i>In(1)ac³</i>	59.6 to 58.8
						<i>Df(1)yT1b-688</i>	
						<i>Df(1)yT1b-96</i>	
				3'	1B2-3	ac	
						<i>Df(1)yT2-398</i>	58.9 to 58.2
						<i>Df(1)yT2-558</i>	58.9 to 58.2
				5'	1B2-3	ac	
						<i>Df(1)yT2-293</i>	57.6 to 56.6
						<i>Df(1)yT2-212</i>	56.8 to 56.3
						<i>Df(1)yT2-655</i>	55.8 to 54.3
						<i>Df(1)yT2-97</i>	55.8 to 54.3
				left	1B2-3	<i>In(1)sc^{V2}</i>	55.8 to 54.3
						<i>Df(1)yT2-503</i>	54.3 to 53.2
						<i>Df(1)yT2-624</i>	54.3 to 53.2
						<i>Df(1)yT2-95</i>	54.3 to 53.2
						<i>Df(1)yT2-701</i>	53.2 to 52.4
						<i>Df(1)yT2-84</i>	53.2 to 52.4
						<i>Df(1)yT2-86</i>	53.2 to 52.4
						<i>Df(1)yT2-216</i>	52.4 to 50.6
						<i>Df(1)yT2-356</i>	52.4 to 50.6
						<i>Df(1)yT2-520</i>	52.4 to 50.6
						<i>Df(1)yT2-590</i>	52.4 to 50.6
						<i>Df(1)yT2-90</i>	52.4 to 50.2
						<i>Df(1)yT2-438</i>	50.6 to 49.4
						<i>Df(1)yT2-92</i>	50.6 to 49.4
						<i>Df(1)yT2-83</i>	50.2 to 49.4
						<i>Df(1)yT2-637</i>	49.4 to 47.9
						<i>Df(1)yT2-730</i>	49.4 to 47.9
						<i>Df(1)yT2-93</i>	49.4 to 47.9
						<i>Df(1)yT2-336</i>	47.9 to 46.8
						<i>Df(1)yT2-351</i>	47.9 to 46.8
						<i>Df(1)yT2-742</i>	47.9 to 46.8
						<i>In(1)sc⁸</i>	47.9 to 46.8
				left	1B2-3	<i>Df(1)yT2-288</i>	46.0 to 45.4
						<i>Df(1)yT2-643</i>	46.0 to 45.4
						<i>Df(1)yT2-85</i>	46.0 to 45.4
						<i>Df(1)yT2-89</i>	46.0 to 45.4
						<i>Df(1)yT1b-685</i>	45.4 to 43.9
						<i>Df(1)yT2-165</i>	45.4 to 43.9
				left		<i>Df(1)arth</i>	119 to 121
				left		<i>Df(1)sc19</i>	100-103
						<i>Df(1)yT2-623</i>	45.4 to 44.4; 36.7 to 34.4
						<i>Df(1)yT2-630</i>	45.4 to 43.9

^α *I* = Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90.

I(1)1F

Six lethally mutable loci identified by Lefevre.

locus	cytological location	included in	excluded from
I(1)1Fa		<i>Df(1)A94</i>	<i>Df(1)1F2-2B7</i>
I(1)1Fb		<i>Df(1)1F2-2B7</i>	
I(1)1Fc			
I(1)1Fd	1F4	<i>y²Y59k9.4</i>	
I(1)1Fe			<i>Df(1)GA120</i>
I(1)1Ff			

^α *1* = Lefevre, 1981, Genetics 99: 461-80; *2* = Lefevre and Watkins, 1986, Genetics 113: 869-95.

DEFICIENCY MAP OF REGION 1

side	breakpoint	variant	DNA coordinates ^α
left	1A1-3	<i>Df(1)SJ1a</i>	
left	1A1-3	<i>Df(1)SJ1b</i>	
left	1A1-3	<i>Df(1)SJ1c</i>	
left	1A1-3	<i>Df(1)SJ1d</i>	
	1A4	I(1)1Aa	
	1A5	dmd	
left		<i>Df(1)y75e</i>	
	1A6	I(1)1Ac	
left		<i>Df(1)cin-arth</i>	
right		<i>Df(1)5-13</i>	
right		<i>Df(1)17-8</i>	
right		<i>Df(1)259</i>	
right		<i>Df(1)403</i>	
right	1A6-7	<i>Df(1)SJ1b</i>	
right	1A6-7	<i>Df(1)SJ1c</i>	
	1A6-B1	I(1)1Ad	
right		<i>Df(1)05-22-1</i>	
right	1A6-B1	<i>Df(1)SJ1d</i>	
	1A7	cin	
		<i>T(1;4)cin</i>	140-151
	1A7-8	I(1)1Af	
	1A8	ewg	
right	1A8-B1	<i>Df(1)SJ1a</i>	
left		<i>Df(1)arth</i>	119 to 121
left		<i>Df(1)sc19</i>	100-103
		<i>Df(1)yT2-288</i>	46.0 to 45.4
		<i>Df(1)yT2-643</i>	46.0 to 45.4
		<i>Df(1)yT2-85</i>	46.0 to 45.4
		<i>Df(1)yT2-89</i>	46.0 to 45.4
		<i>Df(1)yT1b-685</i>	45.4 to 43.9
		<i>Df(1)yT2-165</i>	45.4 to 43.9
		<i>Df(1)yT2-623</i>	45.4 to 44.4; 36.7 to 34.4
		<i>Df(1)yT2-630</i>	45.4 to 43.9

THE GENOME OF *DROSOPHILA MELANOGASTER*

side	breakpoint	variant	DNA coordinates α	side	breakpoint	variant	DNA coordinates α
		<i>Df(1)yT2-304</i>	44.4 to 43.9			<i>Df(1)yT7-329</i>	
		<i>Df(1)yT2-29</i>	42.3 to 39.5			<i>Df(1)yT7-341</i>	
		<i>Df(1)yT2-524</i>	42.3 to 42.1			<i>Df(1)yT7-348</i>	
		<i>Df(1)yT2-551</i>	42.3 to 39.5			<i>Df(1)yT7-358</i>	
		<i>Df(1)yT2-629</i>	42.3 to 39.5			<i>Df(1)yT7-366</i>	
		<i>Df(1)yT2-103</i>	42.1 to 41.5			<i>Df(1)yT7-371</i>	
		<i>Df(1)yT2-252</i>	42.1 to 41.5			<i>Df(1)yT7-376</i>	
		<i>Df(1)yT2-618</i>	39.1 to 37.1			<i>Df(1)yT7-377</i>	
		<i>Df(1)yT2-233</i>	38.4 to 36.3			<i>Df(1)yT7-400</i>	
		<i>Df(1)yT2-748</i>	38.4 to 36.3			<i>Df(1)yT7-401</i>	
		<i>Df(1)yT2-631</i>	37.1 to 35.5			<i>Df(1)yT7-404</i>	
		<i>Df(1)yT2-650</i>	37.1 to 35.5			<i>Df(1)yT7-406</i>	
		<i>Df(1)yT2-343</i>	36.3 to 33.8			<i>Df(1)yT7-435</i>	
		<i>Df(1)yT2-26</i>				<i>Df(1)yT7-446</i>	
		<i>Df(1)yT2-634</i>				<i>Df(1)yT7-448</i>	
		<i>Df(1)yT2-695</i>				<i>Df(1)yT7-458</i>	
		<i>Df(1)yT2-696</i>	complex			<i>Df(1)yT7-518</i>	
		<i>Df(1)yT2-734</i>				<i>Df(1)yT7-525</i>	
		<i>Df(1)yT2-735</i>				<i>Df(1)yT7-532</i>	
		<i>Df(1)yT2-738</i>				<i>Df(1)yT7-547</i>	
		<i>Df(1)yT2-744</i>				vnd	
		<i>Df(1)yT2-749</i>		left	1B9	<i>Df(1)su(s)E2</i>	
		<i>Df(1)yT2-99</i>				<i>Df(1)yT8-9</i>	
	1B3-4	sc				<i>Df(1)yT8-12</i>	
		sis-b				<i>Df(1)yT8-18</i>	
		<i>Df(1)yT3-150</i>	33.8 to 33.4			<i>Df(1)yT8-101</i>	
		<i>Df(1)yT3-214</i>	33.4 to 32.7			<i>Df(1)yT8-124</i>	
left	1B3-4	<i>ln(1)sc^{L8}</i>	30.9 to 28.8			<i>Df(1)yT8-128</i>	
left	1B3-4	<i>ln(1)sc^{S1}</i>	28.2 to 26.9			<i>Df(1)yT8-143</i>	
left		<i>ln(1)sc⁴</i>	25.8 to 24.0			<i>Df(1)yT8-148</i>	
		<i>Df(1)yT3-15</i>				<i>Df(1)yT8-158</i>	
		<i>Df(1)yT3-22</i>				<i>Df(1)yT8-184</i>	
		<i>Df(1)yT3-24</i>				<i>Df(1)yT8-185</i>	
		<i>Df(1)yT3-55</i>				<i>Df(1)yT8-197</i>	
		I(1)Bb				<i>Df(1)yT8-206</i>	
	1B4-8	su(b)				<i>Df(1)yT8-241</i>	
		<i>Df(1)yT4-8</i>				<i>Df(1)yT8-271</i>	
		<i>Df(1)yT4-19</i>				<i>Df(1)yT8-361</i>	
		<i>Df(1)yT4-20</i>				<i>Df(1)yT8-362</i>	
		<i>Df(1)yT4-25</i>				<i>Df(1)yT8-368</i>	
		I(1)1Bc				<i>Df(1)yT8-369</i>	
	1B5-6	svr				<i>Df(1)yT8-385</i>	
		<i>Df(1)yT6-151</i>				<i>Df(1)yT8-409</i>	
		<i>Df(1)yT6-188</i>				<i>Df(1)yT8-420</i>	
		<i>Df(1)yT6-244</i>				<i>Df(1)yT8-500</i>	
		<i>Df(1)yT6-291</i>				<i>Df(1)yT8-530</i>	
		<i>Df(1)yT6-522</i>				I(1)1Bg	
		<i>Df(1)yT6-544</i>		left	1B10-11	<i>Df(1)su(s)83</i>	
		<i>Df(1)yT6-506</i>	-17 to -14		1B10	<i>Df(1)yT9-7</i>	
		<i>Df(1)yT6-450</i>	-10 to -8			<i>Df(1)yT9-21</i>	
	1B4-9	elav			1B10-12	<i>Df(1)yT9-220</i>	
		<i>Df(1)yT7-263</i>	-1 to +4			<i>Df(1)yT9-275</i>	
		<i>Df(1)yT7-437</i>	-1 to +4			I(1)1Bh	
		<i>Df(1)yT7-191</i>	+4 to +14			<i>Df(1)y74k10.1</i>	
		<i>Df(1)yT7-107</i>	+15 to +23		1B5-6	<i>Df(1)svr</i>	
		<i>Df(1)yT7-104</i>			1B9-10	<i>Df(1)yT10-102</i>	
		<i>Df(1)yT7-108</i>				<i>Df(1)yT10-106</i>	
		<i>Df(1)yT7-109</i>				<i>Df(1)yT10-581</i>	
		<i>Df(1)yT7-114</i>				<i>Df(1)yT10-665</i>	
		<i>Df(1)yT7-115</i>				<i>Df(1)yT10-673</i>	
		<i>Df(1)yT7-129</i>				<i>Df(1)yT10-677</i>	
		<i>Df(1)yT7-130</i>				I(1)1Bi	
		<i>Df(1)yT7-155</i>				M(1)Bid	
		<i>Df(1)yT7-157</i>		right	1B11-12	<i>Df(1)y74k24.1</i>	
		<i>Df(1)yT7-178</i>			1B9-10	<i>Df(1)yT12-173</i>	
		<i>Df(1)yT7-194</i>				<i>Df(1)yT12-187</i>	
		<i>Df(1)yT7-208</i>				<i>Df(1)yT12-200</i>	
		<i>Df(1)yT7-218</i>				<i>Df(1)yT12-206</i>	
		<i>Df(1)yT7-229</i>				<i>Df(1)yT12-246</i>	
		<i>Df(1)yT7-234</i>				<i>Df(1)yT12-667</i>	
		<i>Df(1)yT7-250</i>		right	1B14	<i>y²Y611</i>	
		<i>Df(1)yT7-284</i>				su(s)	
		<i>Df(1)yT7-292</i>				<i>Df(1)yT13-423</i>	
		<i>Df(1)yT7-296</i>				<i>Df(1)yT13-464</i>	
		<i>Df(1)yT7-301</i>				I(1)1Bk	
		<i>Df(1)yT7-311</i>		right		<i>Df(1)su(s)E2</i>	

side	breakpoint	variant	DNA coordinates α
		<i>Df(1)yT14-546</i>	
		<i>Df(1)yT14-576</i>	
		<i>l(1)1Ca</i>	
		<i>Df(1)yT15-5</i>	
		<i>Df(1)yT15-6</i>	
		<i>Df(1)yT15-28</i>	
		<i>Df(1)yT15-56</i>	
		<i>Df(1)yT15-105</i>	
		<i>Df(1)yT15-158b</i>	
		<i>Df(1)yT15-161</i>	
		<i>Df(1)yT15-177</i>	
		<i>Df(1)yT15-183</i>	
		<i>Df(1)yT15-189</i>	
		<i>Df(1)yT15-219</i>	
		<i>Df(1)yT15-222</i>	
		<i>Df(1)yT15-270</i>	
		<i>Df(1)yT15-274</i>	
		<i>Df(1)yT15-286</i>	
		<i>Df(1)yT15-300</i>	
		<i>Df(1)yT15-318</i>	
		<i>Df(1)yT15-325</i>	
		<i>Df(1)yT15-327</i>	
		<i>Df(1)yT15-334</i>	
		<i>Df(1)yT15-364</i>	
		<i>Df(1)yT15-365</i>	
		<i>Df(1)yT15-383</i>	
		<i>Df(1)yT15-390</i>	
		<i>Df(1)yT15-392</i>	
		<i>Df(1)yT15-405</i>	
		<i>Df(1)yT15-408</i>	
		<i>Df(1)yT15-410</i>	
		<i>Df(1)yT15-412</i>	
		<i>Df(1)yT15-433</i>	
		<i>Df(1)yT15-449</i>	
		<i>Df(1)yT15-460</i>	
		<i>Df(1)yT15-468</i>	
		<i>Df(1)yT15-502</i>	
		<i>Df(1)yT15-509</i>	
		<i>Df(1)yT15-511</i>	
		<i>Df(1)yT15-517</i>	
		<i>Df(1)yT15-533</i>	
		<i>Df(1)yT15-543</i>	
		<i>Df(1)yT15-548</i>	
		<i>Df(1)yT15-552</i>	
		<i>Df(1)yT15-564</i>	
		<i>Df(1)yT15-568</i>	
		<i>Df(1)yT15-575</i>	
		<i>Df(1)yT15-586</i>	
		<i>Df(1)yT15-597</i>	
		<i>Df(1)yT15-599</i>	
		<i>mul</i>	
		<i>Df(1)yT16-14</i>	
		<i>Df(1)yT16-171</i>	
		<i>Df(1)yT16-411</i>	
		<i>Df(1)yT16-499</i>	
		<i>Df(1)yT16-534</i>	
		<i>Df(1)yT16-554</i>	
		<i>tw</i>	
		<i>Df(1)yT17-570</i>	
		<i>Df(1)yT17-600</i>	
		<i>l(1)1Da</i>	
		<i>Df(1)yT18-319</i>	
		<i>brc</i>	
		<i>Df(1)yT19-16</i>	
		<i>Df(1)yT19-253</i>	
right	1D6-E1	<i>Df(1)su(s)83</i>	
		<i>l(1)1Dc</i>	
left		<i>Df(1)f3</i>	
left	1E1-2	<i>Df(1)pn7b</i>	
left	1E1-2	<i>Df(1)s39</i>	
left	1E1-2	<i>Df(1)sta</i>	
		1E2	
		1E3	
left	1E3-4	<i>Df(1)A94</i>	
left	1E3-4	<i>Df(1)AT127</i>	
left	1E3-4	<i>Df(1)dor1T</i>	

side	breakpoint	variant	DNA coordinates α
left	1E3-4	<i>Df(1)RA19</i>	
right	1E3-4	<i>Dp(1;Y)y⁺sc</i>	
		1E4	<i>l(1)1Ec</i>
right	1E4-F1	<i>Dp(1:f)18</i>	
right	1E4-F1	<i>Dp(1:f)112</i>	
		1E5	<i>l(1)1Ed</i>
			<i>l(1)EFa</i>
			<i>l(1)EFb</i>
			<i>l(1)EFc</i>
			<i>l(1)EFd</i>
			<i>l(1)EFe</i>
			<i>l(1)EFf</i>
			<i>l(1)EFg</i>
			<i>l(1)EFh</i>
			<i>l(1)EFI</i>
			<i>l(1)EFJ</i>
			<i>l(1)1Fa</i>
			<i>l(1)1Fb</i>
left	1F2	<i>Df(1)1F2-2B7</i>	
		<i>l(1)1Fc</i>	
		<i>l(1)1Fd</i>	
right		<i>y⁻Y59k9.4</i>	
right	2A2-3	<i>Df(1)A127</i>	

α For coordinates in the *y* to *sc* region, (1) see Fleming, DeSimone, and White (1989, Mol. Cell Biol. 9: 719-25) when values are > 100, (2) see Ruiz-Gómez and Modolell (1987, Genes Dev. 1: 1238-46), when values are < 100. 0 is defined as the more distal *EcoR1* site within the *sc*³² molecular deficiency; positive values extend to the left. For coordinates in the *elav* region, see Campos, Rosen, Robinson and White (1987, EMBO J. 6: 425-31). 0 is defined as the *BamHI* site closest to the proximal breakpoint of *In(1)elav*⁴; positive values extend to the right. The *Df(1)yT* series comprises terminal deficiencies that were induced in a *mu2* background; such deficiencies are unstable and lose about 75 terminal nucleotides per generation (Biessman and Mason, 1988, EMBO J. 7: 1081-86); hence the above determinations represent the situation prior to 1987.

l(1)2A

At least six lethally mutable loci, including *sta*. Two loci identified by Belyaeva and Aizenzon; their *l(1)BA11* arbitrarily assigned to *l(1)2Ac*, but complementation tests not performed; when these are done the locus designation is likely to change. Their *l(1)BA12* assigned to the *l(1)2Ad* locus on the basis of its localization to the right of *Dp(1:f)101* and to the left of *sta*. These lethals placed between 2A2 and 2B4 by Aizenzon and Belyaeva. Schalet records spontaneous lethals at two loci within *Df(1)HA18*, *l(1)13-80* and *l(1)4-134*, and Kramers *et al.* record an HMS-induced allele of the latter *l(1)HM31*; allelism tests of these mutants to known alleles of *l(1)2Ab*, *l(1)2Ac*, and *l(1)2Ad* have not been carried out.

locus	cytological		excluded from	synonym
	location	included in		
<i>l(1)2Aa</i>	2A1		<i>Df(1)A127</i>	
			<i>Df(1)HA18</i>	
<i>l(1)2Ab</i>	2A1		<i>Df(1)A127</i>	
			<i>Df(1)HA18</i>	
<i>l(1)2Ac</i>	2A2	<i>Df(1)HA18</i>		
		<i>Dp(1:f)101</i>		
<i>l(1)2Ad</i>	2A2-3	<i>Df(1)HA18</i>		
		<i>Dp(1:f)101</i>		<i>l(1)BA11</i>
<i>l(1)2Ae</i>		<i>Df(1)HA18</i>	<i>Dp(1:f)101</i>	<i>l(1)BA12</i>
<i>l(1)2Af</i>	2A3-5	<i>Df(1)sta</i>		<i>sta</i>

allele	origin	discoverer	synonym	ref α	comments
<i>l(1)2Aa</i>¹	EMS	Lefevre	<i>l(1)VA185</i>	4	L/L
<i>l(1)2Ab</i>¹	EMS	Lefevre	<i>l(1)EA97</i>		E/NME

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allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)2Ab</i>²	EMS	Lefevre	<i>l(1)VE896</i>	4	E/NME
<i>l(1)2Ac</i>¹	X ray	Lefevre	<i>l(1)A70</i>	3	
*<i>l(1)2Ac</i>²	X ray	Lefevre	<i>l(1)HC278</i>	3	
*<i>l(1)2Ac</i>³	X ray	Lefevre	<i>l(1)RF51</i>	3	
<i>l(1)2Ac</i>⁵	EMS	Lefevre	<i>l(1)VE721</i>	4	
<i>l(1)2Ac</i>⁶	EMS	Lefevre	<i>l(1)VA305</i>	4	L3/L
<i>l(1)2Ad</i>¹	X ray	Lefevre	<i>l(1)A60</i>	3	L1/L
*<i>l(1)2Ad</i>²	X ray	Lefevre	<i>l(1)RC32</i>	3	
<i>l(1)2Ad</i>³	EMS	Lefevre	<i>l(1)VE795</i>	4	
<i>l(1)2Ad</i>⁴	EMS		<i>l(1)t5</i>	1, 2	
<i>l(1)2Ad</i>⁵	EMS		<i>l(1)t23</i>	1, 2	
<i>l(1)2Ad</i>⁶	EMS		<i>l(1)t62</i>	1, 2	
<i>l(1)2Ad</i>⁷	EMS		<i>l(1)t185</i>	1, 2	
<i>l(1)2Ad</i>⁸	EMS		<i>l(1)t416</i>	1, 2	
<i>l(1)2Ae</i>¹	X ray	Lefevre	<i>l(1)C220</i>	3	<i>sta</i> mutant
<i>l(1)2Ae</i>²	X ray	Lefevre	<i>l(1)C229</i>	3	
*<i>l(1)2Ae</i>³	X ray	Lefevre	<i>l(1)HA93</i>	3	
<i>l(1)2Ae</i>⁴	X ray	Lefevre	<i>l(1)HC206</i>	3	<i>ln(1)1E1-2;2A1-2</i>
<i>l(1)2Ae</i>⁵	X ray	Lefevre	<i>l(1)JC53</i>	3	
<i>l(1)2Ae</i>⁶	EMS	Lefevre	<i>l(1)EC223</i>	4	
<i>l(1)2Ae</i>⁷	EMS	Lefevre	<i>l(1)VA326</i>	4	
<i>l(1)2Ae</i>⁸	X ray		<i>l(1)R4</i>	1, 2	
<i>l(1)2Ae</i>⁹	EMS		<i>l(1)t12</i>	1, 2	
<i>l(1)2Ae</i>¹⁰	EMS		<i>l(1)t27</i>	1, 2	
<i>l(1)2Ae</i>¹¹	EMS		<i>l(1)t224</i>	1, 2	

^α 1 = Aizenzon and Belyaeva, 1982, DIS 58: 3-7; 2 = Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Lefevre and Watkins, 1986, Genetics 113: 869-95.

l(1)2B

Two collections of lethals in region 2B (Lefevre; Belyaeva *et al.*) have been complementation tested *inter se*, but have not been tested against each other. Lefevre identifies eleven complementing groups, whereas Belyaeva *et al.* identify nine plus two or three others that are identified here as alleles of *l(1)2A* loci but could lie in 2B. Where a single lethal complementation group from each series occupies the same region as defined by deficiency mapping, they are treated as a single locus. Lethal alleles of *br* and *dor* are included in both collections. Belyaeva *et al.* identify a complex of complementing lethals (designated *o.c.c.* = overlapping complementation complex) that includes the lethal alleles of *br*, and which is not recognized by Lefevre. These are tabulated below as *l(1)2Ba* to *l(1)2Bd*; *l(1)2Bad* alleles fail to complement four mutually complementing lethals and *l(1)2Bab* fails to complement *l(1)2Ba* and *l(1)2Bb*. The loci identified by Lefevre in the *br-dor* region and which he places in 2B4-12 have been equated arbitrarily to the complementing complex, which Belyaeva *et al.* place in 2B3-8; future crosses will determine the rectitude of this assignment. These lethals are described with the broad complex (*BRC*). In addition, Schalet (1983, DIS 59: 107) describes three complementation groups between the breakpoints of *y²Y67g19.1* and *Dp(1;3)w^{vco}*; not tested against *l(1)2Bj* through *l(1)2Bt*. *arm*, selected by Wieschaus, Nüsslein-Volhard and Jürgens, has not been tested in combination with any of the others.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(1)2Ba</i>	1-0.3	2B4-5 2B7	<i>Df(1)S39</i>	<i>Df(1)sta</i> <i>Df(1)Sz280</i>	<i>br</i>

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(1)2Bab</i>		2B4-5	<i>Df(1)S39</i>	<i>Df(1)sta</i> <i>Df(1)Sz280</i>	
<i>l(1)2Bad</i>		2B4-5	<i>Df(1)S39</i>	<i>Df(1)sta</i> <i>Df(1)Sz280</i>	<i>npr</i>
<i>l(1)2Bb</i>		2B4-5 2B8	<i>Df(1)S39</i>	<i>Df(1)sta</i> <i>Df(1)Sz280</i>	<i>rbp</i>
<i>l(1)2Bc</i>		2B4-5 2B9	<i>Df(1)S39</i>	<i>Df(1)sta</i> <i>Df(1)Sz280</i>	<i>l(1)pp1</i>
<i>l(1)2Bd</i>		2B4-5 2B10	<i>Df(1)S39</i>	<i>Df(1)sta</i> <i>Df(1)Sz280</i>	<i>l(1)pp2</i>
<i>l(1)2Be</i>	1-0.4	2B6-7 2B11-12	<i>y²Y67g24.2</i>	<i>Df(1)S39</i>	<i>dor</i>
<i>l(1)2Bf</i>		2B6-7 2B11-1	<i>y²Y67g24.2</i>	<i>Df(1)S39</i>	<i>htw</i> , <i>swi</i>
<i>l(1)2Bg</i>		2B12-14	<i>Dp(1;Y)2E</i>	<i>Df(1)RA19</i>	
<i>l(1)2Bh</i>		2B12-14	<i>Dp(1;Y)2E</i>	<i>Df(1)RA19</i>	
<i>l(1)2Bi</i>	1-(0.5)	2B15	<i>y²Y67g19.1</i>	<i>Dp(1;Y)2E</i>	<i>tlc</i>
<i>l(1)2Bj</i>			<i>y²Y67g19.1</i>	<i>Df(1)A94</i>	
<i>l(1)2Bk</i>			<i>y²Y67g19.1</i>	<i>Df(1)A94</i>	
<i>l(1)2Bl</i>			<i>y²Y67g19.1</i>	<i>Df(1)A94</i>	
<i>l(1)2Bm</i>			<i>y²Y67g19.1</i>	<i>Df(1)A94</i>	
<i>l(1)2Bn</i>			<i>y²Y67g19.1</i>	<i>Df(1)A94</i>	
<i>l(1)2Bo</i>			<i>y²Y67g19.1</i>	<i>Df(1)A94</i>	
<i>l(1)2Bp</i>			<i>y²Y67g19.1</i>	<i>Df(1)A94</i>	
<i>l(1)2Bq</i>			<i>y²Y67g19.1</i>	<i>Df(1)A94</i>	
<i>l(1)2Br</i>			<i>y²Y67g19.1</i>	<i>Df(1)A94</i>	
<i>l(1)2Bs</i>			<i>y²Y67g19.1</i>	<i>Df(1)A94</i>	
<i>l(1)2Bt</i>			<i>y²Y67g19.1</i>	<i>Df(1)A94</i>	
<i>l(1)2Bu</i>			<i>y²Y67g19.1</i>		
<i>l(1)2Bv</i>	1-1.2	2B15	<i>y²Y67g19.1</i>	<i>Df(1)svr</i>	<i>arm</i>

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)2Bg</i>¹	HMS	Kramers	<i>l(1)HM38</i>	1, 2	
<i>l(1)2Bg</i>²	EMS	Lefevre	<i>l(1)DA681</i>	4	
<i>l(1)2Bh</i>¹	HMS	Kramers	<i>l(1)HM40</i>	1, 2	
<i>l(1)2Bj</i>¹	X ray	Lefevre	<i>l(1)A68</i>	3	
<i>l(1)2Bj</i>²	X ray	Lefevre	<i>l(1)GA29</i>	3	
<i>l(1)2Bj</i>³	X ray	Lefevre	<i>l(1)HC192</i>	3	
<i>l(1)2Bj</i>⁴	X ray	Lefevre	<i>l(1)HF347</i>	3	<i>T(1;3)2B;91</i> (complex)
<i>l(1)2Bj</i>⁵	EMS	Lefevre	<i>l(1)DA641</i>	4	
<i>l(1)2Bj</i>⁶	EMS	Lefevre	<i>l(1)DF902</i>	4	
<i>l(1)2Bj</i>⁷	EMS	Lefevre	<i>l(1)VE792</i>	4	PP
<i>l(1)2Bj</i>⁸	EMS	Geer	<i>l(1)y23</i>		
<i>l(1)2Bk</i>¹	EMS	Lefevre	<i>l(1)VE810</i>	4	L2/L
<i>l(1)2Bk</i>²	EMS	Lefevre	<i>l(1)VE831</i>	4	
<i>l(1)2Bl</i>¹	EMS	Lefevre	<i>l(1)EC293</i>	4	
<i>l(1)2Bl</i>²	EMS	Lefevre	<i>l(1)VA177</i>	4	P/NME
<i>l(1)2Bm</i>¹	X ray	Lefevre	<i>l(1)HC225</i>	4	
<i>l(1)2Bm</i>²	EMS	Lefevre	<i>l(1)VA341</i>	4	
<i>l(1)2Bm</i>³	EMS	Lefevre	<i>l(1)VA355</i>	4	E/L
<i>l(1)2Bn</i>¹	X ray	Lefevre	<i>l(1)C143</i>	3	
<i>l(1)2Bn</i>²	X ray	Lefevre	<i>l(1)HC126</i>	3	
<i>l(1)2Bn</i>³	EMS	Lefevre	<i>l(1)DC831</i>	4	
<i>l(1)2Bn</i>⁴	EMS	Lefevre	<i>l(1)EF490</i>	4	
<i>l(1)2Bn</i>⁵	EMS	Lefevre	<i>l(1)VE672</i>	4	L2/L
<i>l(1)2Bn</i>⁶	spont	Schalet	<i>l(1)17-114</i>		
<i>l(1)2Bo</i>¹	EMS	Lefevre	<i>l(1)VA143</i>	4	
<i>l(1)2Bp</i>¹	EMS	Lefevre	<i>l(1)DC836</i>	4	
<i>l(1)2Bq</i>¹	X ray	Lefevre	<i>l(1)HC180</i>	3	
<i>l(1)2Br</i>¹	EMS	Lefevre	<i>l(1)VA130</i>	4	L/L
<i>l(1)2Bs</i>¹	X ray	Lefevre	<i>l(1)A106</i>	3	

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)2Bt</i>¹	EMS	Lefevre	<i>l(1)DF949</i>	4	
<i>l(1)2Bu</i>¹	spont	Schalet	<i>l(1)10-153-2</i>		

^α 1 = Aizenzon and Belyaeva, 1982, DIS 58: 3-7; 2 = Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Lefevre and Watkins, 1986, Genetics 113: 869-95.

^β Three mutants in the same region; the two HMS-induced alleles shown not to complement; *l(1)2Bg*² arbitrarily assigned to *l(1)2Bg*, but could equally be allelic to *l(1)2Bh*.

l(1)2C

Six lethally mutable loci including *Actn* and *usp*.

locus	genetic location		included in	excluded from	synonym
	location	location			
<i>l(1)2Ca</i>	2C1			<i>y</i> ² <i>Y67g19.1</i>	
<i>l(1)2Cb</i>	1-1.0	2C3	<i>Dp(1;3)w^{vco}</i>	<i>Dp(1;Y)w⁺303</i>	<i>ftiA Actn</i>
<i>l(1)2Cc</i>		2B17-D2	<i>Dp(1;3)w^{vco}</i>	<i>Dp(1;Y)w⁺303</i>	
<i>l(1)2Cd</i>		2C9			
<i>l(1)2Ce</i>		2B17-D2	<i>Dp(1;3)w^{vco}</i>	<i>Dp(1;Y)w⁺303</i>	
<i>l(1)2Cf</i>		2C9	<i>Dp(1;3)w^{vco}</i>	<i>Dp(1;Y)w⁺303</i>	<i>usp</i>

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)2Ca</i>¹	X ray	Lefevre	<i>l(1)HC282</i>	2	<i>T(1;2)2C1-2;32B</i>
<i>l(1)2Ca</i>²	spont	Schalet	<i>l(1)6-62</i>		

l(1)2Cc

phenotype: *l(1)2Cc*¹² larval lethal; *l(1)2Cc*³ polyphasic; *l(1)2Cc*¹ carries independent lethal and cannot be tested in *lDp* males. Germ line clones in females exhibit nurse-cell degeneration; therefore the normal allele required for normal oogenesis (Perrimon, Engstrom, and Mahowald, 1985, Genetics 111: 23-41).

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)2Cc</i>¹	EMS	Lefevre	<i>l(1)VA56</i>	3, 4	L
<i>l(1)2Cc</i>²	EMS	Lefevre	<i>l(1)VE651</i>	3, 4	L2-3/AO
<i>l(1)2Cc</i>³	EMS	Lefevre	<i>l(1)VE782</i>	3, 4	PP
<i>l(1)2Cd</i>¹	X ray	Lefevre	<i>l(1)C134</i>		
<i>l(1)2Ce</i>¹	X ray	Lefevre	<i>l(1)GA55</i>	2, 4	L/NME
<i>l(1)2Ce</i>²	X ray	Lefevre	<i>l(1)GF316</i>	2, 4	E
<i>l(1)2Ce</i>³	EMS	Lefevre	<i>l(1)DF943</i>	3, 4	PP

^α 1 = Kramers, Schalet, Paradi, and Huizer-Hoogteyting, 1983, Mutation Res. 107: 187-201; 2 = Lefevre, 1981, Genetics 99: 461-80; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 4 = Perrimon, Engstrom, and Mahowald, 1985, Genetics 111: 23-41.

l(1)2D

Four lethally mutable loci, three of which have specific names: *csw*, *Pgd*, and *wapl*.

locus	genetic location		included in	excluded from	synonym
	location	location			
<i>l(1)2Da</i>		2D1-2	<i>Dp(1;Y)w⁺303</i>	<i>Df(1)pn38</i>	
<i>l(1)2Db</i>		2D1-2	<i>Dp(1)JA52</i>	<i>Df(1)pn38</i>	<i>l(1)107</i>
<i>l(1)2Dc</i>		2D1-2	<i>Dp(1)JA52</i>	<i>Df(1)pn38</i>	<i>l(1)405</i>
<i>l(1)2Dd</i>		2D3-4	<i>Df(1)pn38</i>	<i>Df(1)Pgd-kz</i>	<i>csw</i>
<i>l(1)2De</i>	1-0.6	2D4-6	<i>Df(1)Pgd-kz</i>	<i>Df(1)64c18</i>	<i>Pgd</i>
<i>l(1)2Df</i>	1-0.65	2D4-6	<i>Df(1)Pgd-kz</i>	<i>Df(1)64c18</i>	<i>wapl</i>
<i>l(1)2Dg</i>					
<i>l(1)2Dh</i>					
<i>l(1)2Di</i>					

l(1)2Da

phenotype: Larval lethal; also cell lethal in female germ line.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)2Da</i>¹	X ray	Lefevre	<i>l(1)RC38</i>	3	
<i>l(1)2Da</i>²	EMS	Lefevre	<i>l(1)DF967</i>	4, 5	
<i>l(1)2Da</i>³	EMS			2	
<i>l(1)2Da</i>⁴	EMS			2	
<i>l(1)2Da</i>⁵	EMS			2	
<i>l(1)2Da</i>⁶	EMS			2	
<i>l(1)2Da</i>⁷	EMS			2	
<i>l(1)2Da</i>⁸	EMS			2	
<i>l(1)2Da</i>⁹	EMS			2	semilethal
<i>l(1)2Db</i>¹	EMS			2	
<i>l(1)2Db</i>²	EMS			2	
<i>l(1)2Db</i>³	X ray			2	
<i>l(1)2Dc</i>¹	X ray			2	
<i>l(1)2Dg</i>¹	EMS	Gvozdev	<i>l(1)N3</i> ⁹⁰	1	
<i>l(1)2Dg</i>²	X ray	Lefevre	<i>l(1)A7</i>	3	<i>T(1;A)2E-F;?</i>
<i>l(1)2Dh</i>¹	EMS	Gvozdev	<i>l(1)N5</i> ⁷²	1	
<i>l(1)2Di</i>¹	EMS	Gvozdev	<i>l(1)N6</i> ²⁷	1	

^α 1 = Alatorsev and Tolchikov, 1985, DIS 61: 24; 1 = Dura, Randsholt, Deatrick, Erk, Santamaria, Freeman, Weddell, and Brock, 1987, Cell 51: 829-39; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 5 = Perrimon, Engstrom, and Mahowald, 1985, Genetics 111: 23-41.

Gvozdev and colleagues recovered three complementing lethals between *wapl* and *l(1)2Ea*, two of which are lost; Lefevre reports one. Allelism of Lefevre's mutant arbitrarily assigned.

l(1)2E

Two lethally mutable loci, one of which is *kz*.

locus	genetic location		included in	excluded from	synonym
	location	location			
<i>l(1)2Ea</i>		2E2	<i>Df(1)64c18</i>	<i>Df(1)278B-4-1a</i>	
<i>l(1)2Eb</i>	1-0.9	2E3	<i>Df(1)278B-4-1a</i>	<i>Df(1)2F1-3A4</i>	<i>kz</i>

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)2Ea</i>¹	X ray	Lefevre	<i>l(1)C99</i>	2	<i>In(1)2E3;20</i> ; fails to complement <i>pn</i>
<i>l(1)2Ea</i>²	X ray	Lefevre	<i>l(1)JA127</i>	2, 4	
<i>l(1)2Ea</i>³	X ray	Lefevre	<i>l(1)JC105</i>	2, 4	Fails to complement <i>pn</i> and <i>l(1)2Cd</i> ² ; L1-2/L
<i>l(1)2Ea</i>⁴	X ray	Lefevre	<i>l(1)RF4</i>	2	
<i>l(1)2Ea</i>⁵	EMS	Lefevre	<i>l(1)EC205</i>	3, 4	
<i>l(1)2Ea</i>⁶	EMS	Lefevre	<i>l(1)VA70</i>	3	
<i>l(1)2Ea</i>⁷	EMS	Lefevre	<i>l(1)VA283</i>	3	
<i>l(1)2Ea</i>⁸	EMS	Lefevre	<i>l(1)VE712</i>	3	
<i>l(1)2Ea</i>⁹	NMU	Gvozdev	<i>l(1)N7</i> ⁶⁹	1	
<i>l(1)2Ea</i>¹⁰	NMU	Gvozdev	<i>l(1)N7</i> ⁷⁰	1	
<i>l(1)2Ea</i>¹¹	EMS	Gvozdev	<i>l(1)N7</i> ⁸¹	1	
<i>l(1)2Ea</i>¹²	gamma	Gvozdev	<i>l(1)N7</i> ¹⁰⁷	1	

^α 1 = Gvozdev, Gerasimova, Kovalev, and Ananiev, 1977, DIS 52: 67-68; 2 = Lefevre, 1981, Genetics 99: 461-80; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 4 = Perrimon, Engstrom, and Mahowald, 1984, Genetics 108: 559-72.

Kramers, Schalet, Paradi, and Huizer-Hoogteyting (1983, Mutation Res. 107: 187-201) place *pn* and

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*l(1)2Ea*¹⁰ between the left breakpoints of *Df(1)TEM304* on the left and *Df(1)TEM1* and *Df(1)TEM501* on the right; Lefevre places his alleles adjacent to *pn*, either to the left or right; no complementation tests between Lefevre's and Gvozdev's series.

l(1)2Fd

Five lethally mutable loci detected by both Gvozdev *et al.* and Lefevre, complementation relations among the two collections of mutants not fully worked out.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(1)2Fa</i>	1-(0.95)	2F1	<i>Df(1)278.4B.1</i>	<i>Df(1)2F1-3A4</i>	crn
<i>l(1)2Fb</i>		2F1-3	<i>Df(1)2F1-3A4</i>	<i>Df(1)JC19</i>	
<i>l(1)2Fc</i>		2F1-3	<i>Df(1)2F1-3A4</i>	<i>Df(1)JC19</i>	
<i>l(1)2Fd</i>		2F1-3	<i>Df(1)2F1-3A4</i>	<i>Df(1)JC19</i>	
<i>l(1)2Fe</i>		2F6	<i>Df(1)JC19</i>	<i>Df(1)62g18</i>	phi

l(1)2Fb

phenotype: *l(1)2Fb*¹, *l(1)2Fb*³, and *l(1)2Fb*⁵ die as second- or third-instar larvae; germ-line clones of *l(1)2Fb*¹ and *l(1)2Fb*³, but not *l(1)2Fb*⁵ are cell lethal.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)2Fb</i> ¹	X ray	Lefevre	<i>l(1)JC155</i>	3, 5	
<i>l(1)2Fb</i> ²	X ray	Lefevre	<i>l(1)GA1</i>	3	<i>T(1;2)E-F;41</i> ; also <i>rx</i> ?
<i>l(1)2Fb</i> ³	EMS	Lefevre	<i>l(1)DC798</i>	4, 5	
<i>l(1)2Fb</i> ⁴	EMS	Lefevre	<i>l(1)DC807</i>	4	
<i>l(1)2Fb</i> ⁵	EMS	Lefevre	<i>l(1)VA172</i>	4, 5	L3/NME
<i>l(1)2Fb</i> ⁶	EMS	Lefevre	<i>l(1)VA353</i>	4	
<i>l(1)2Fb</i> ⁷	EMS	Gvozdev	<i>l(1)N9</i> ¹⁶	1	
<i>l(1)2Fb</i> ⁸	EMS	Gvozdev	<i>l(1)N9</i> ¹⁸	1	
<i>l(1)2Fb</i> ⁹	EMS	Gvozdev	<i>l(1)N9</i> ²²	1	
<i>l(1)2Fb</i> ¹⁰	EMS	Gvozdev	<i>l(1)N9</i> ²⁴	1	
<i>l(1)2Fb</i> ¹¹	EMS	Gvozdev	<i>l(1)N9</i> ²⁸	1	
<i>l(1)2Fb</i> ¹²	EMS	Gvozdev	<i>l(1)N9</i> ⁵⁷	1	
<i>l(1)2Fb</i> ¹³	EMS	Gvozdev	<i>l(1)N9</i> ⁵⁸	1	
<i>l(1)2Fb</i> ¹⁴	EMS	Gvozdev	<i>l(1)N9</i> ⁶⁸	1	
<i>l(1)2Fb</i> ¹⁵	EMS	Gvozdev	<i>l(1)N9</i> ⁷⁸	1	
<i>l(1)2Fb</i> ¹⁶	EMS	Gvozdev	<i>l(1)N9</i> ⁸²	1	
<i>l(1)2Fb</i> ¹⁷	EMS	Gvozdev	<i>l(1)N9</i> ⁹⁶	1	
<i>l(1)2Fb</i> ¹⁸	EMS	Gvozdev	<i>l(1)N9</i> ⁹⁹	1	
<i>l(1)2Fb</i> ¹⁹	EMS	Gvozdev	<i>l(1)N9</i> ¹⁰²	1	
<i>l(1)2Fb</i> ²⁰	EMS	Gvozdev	<i>l(1)N9</i> ¹⁰⁸	1	
<i>l(1)2Fb</i> ²¹	HMS		<i>l(1)HM401</i>	2	

*l(1)2Fb*¹⁴ fails to complement *l(1)2Fb*²¹; Gvozdev's alleles arbitrarily assigned to this locus although complementation with Lefevre's alleles not tested; could equally well be allelic to *l(1)2Fd*.

l(1)2Fc

phenotype: Lethal phase second or third larval instars; few eggs produced by homozygous germ-line clones in young *l(1)2Fc*¹/+ but not *l(1)2Fc*²/+ females; clones in older females of both genotypes exhibit previtellogenic arrest.

allele	origin	discoverer	synonym	ref ^α
<i>l(1)2Fc</i> ¹	X ray	Lefevre	<i>l(1)HF311</i>	3, 5
<i>l(1)2Fc</i> ²	X ray	Lefevre	<i>l(1)HF330</i>	3, 5
<i>l(1)2Fc</i> ³	EMS	Lefevre	<i>l(1)EF420</i>	4
<i>l(1)2Fc</i> ⁴	NMU	Gvozdev	<i>l(1)N12</i> ¹	1
<i>l(1)2Fc</i> ⁵	EMS	Gvozdev	<i>l(1)N12</i> ⁴⁶	1
<i>l(1)2Fc</i> ⁶	EMS	Gvozdev	<i>l(1)N12</i> ⁵⁴	1
<i>l(1)2Fc</i> ⁷	EMS	Gvozdev	<i>l(1)N12</i> ⁶²	1
<i>l(1)2Fc</i> ⁸	EMS	Gvozdev	<i>l(1)N12</i> ⁷⁵	1
<i>l(1)2Fc</i> ⁹	EMS	Gvozdev	<i>l(1)N12</i> ⁸⁹	1
<i>l(1)2Fc</i> ¹⁰	EMS	Gvozdev	<i>l(1)N12</i> ⁹⁸	1
<i>l(1)2Fc</i> ¹¹	HMS		<i>l(1)HM438</i>	2

allele origin discoverer synonym ref^α

^α 1 = Gvozdev, Gerasimova, Kovalev, and Ananiev, 1977, DIS 52: 67-68; 2 = Kramers, Schalet, Paradi, and Huizer-Hoogteyling, 1983, Mutation Res. 107: 187-201; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 5 = Perrimon, Engstrom, and Mahowald, 1985, Genetics 111: 23-41.

Failure of *l(1)2Fc*¹¹ to complement either *l(1)2Fc*² or *l(1)2Fc*⁵ establishes allelism between the two series.

l(1)2Fd

phenotype: Hemizygotes die during second or third instar; homozygous germ-line clones for all alleles tested are cell lethal.

allele	origin	discoverer	synonym	ref ^α
<i>l(1)2Fd</i> ¹	EMS	Lefevre	<i>l(1)EC226</i>	4, 5
<i>l(1)2Fd</i> ²	EMS	Lefevre	<i>l(1)EF462</i>	4, 5
<i>l(1)2Fd</i> ³	EMS	Lefevre	<i>l(1)VA99</i>	4
<i>l(1)2Fd</i> ⁴	EMS	Lefevre	<i>l(1)VA196</i>	4
<i>l(1)2Fd</i> ⁵	EMS	Gvozdev	<i>l(1)N11</i> ¹⁴	2
<i>l(1)2Fd</i> ⁶	EMS	Gvozdev	<i>l(1)N11</i> ¹⁵	2
<i>l(1)2Fd</i> ⁷	EMS	Gvozdev	<i>l(1)N11</i> ¹⁹	2
<i>l(1)2Fd</i> ⁸	EMS	Gvozdev	<i>l(1)N11</i> ²⁰	2
<i>l(1)2Fd</i> ⁹	EMS	Gvozdev	<i>l(1)N11</i> ²³	2
<i>l(1)2Fd</i> ¹⁰	EMS	Gvozdev	<i>l(1)N11</i> ²⁶	2
<i>l(1)2Fd</i> ¹¹	EMS	Gvozdev	<i>l(1)N11</i> ³¹	2
<i>l(1)2Fd</i> ¹²	EMS	Gvozdev	<i>l(1)N11</i> ⁴²	2
<i>l(1)2Fd</i> ¹³	EMS	Gvozdev	<i>l(1)N11</i> ⁴³	2
<i>l(1)2Fd</i> ¹⁴	EMS	Gvozdev	<i>l(1)N11</i> ⁵³	2
<i>l(1)2Fd</i> ¹⁵	EMS	Gvozdev	<i>l(1)N11</i> ⁶⁰	2
<i>l(1)2Fd</i> ¹⁶	EMS	Gvozdev	<i>l(1)N11</i> ⁸⁴	2
<i>l(1)2Fd</i> ¹⁷	EMS	Gvozdev	<i>l(1)N11</i> ¹⁰³	2
<i>l(1)2Fd</i> ¹⁸	EMS	Gvozdev	<i>l(1)N11</i> ¹⁰⁵	2
<i>l(1)2Fd</i> ¹⁹	MR	Sobels	<i>l(1)D62</i>	1, 5
<i>l(1)2Fd</i> ¹⁹	MR	Sobels	<i>l(1)D72</i>	1, 5
<i>l(1)2Fd</i> ²⁰	MR	Sobels	<i>l(1)D82</i>	1, 5
<i>l(1)2Fd</i> ²¹	HMS		<i>l(1)HM28</i>	3
<i>l(1)2Fd</i> ²²	HMS		<i>l(1)HM450</i>	3

^α 1 = Eekens, Sobels, Hyland, and Schalet, 1985, Mut. Res. 150: 261-75; 2 = Gvozdev, Gerasimova, Kovalev, and Ananiev, 1977, DIS 52: 67-68; 3 = Kramers, Schalet, Paradi, and Huizer-Hoogteyling, 1983, Mutation Res. 107: 187-201; 4 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 5 = Perrimon, Engstrom, and Mahowald, 1985, Genetics 111: 23-41.

Allelism established between HMS-induced mutants and *l(1)2Fd*¹⁰. Arbitrarily assigned to *l(1)2Fd*, but could equally well be *l(1)2Fb* alleles.

DEFICIENCY MAP OF REGION 2

side	breakpoint	variant	coordinates ^α
right	2A1-4	<i>Df(1)At127</i>	
		<i>l(1)2Aa</i>	
left	2B1-2	<i>Df(1)HA18</i>	
		<i>l(1)2Ab</i>	
		<i>l(1)2Ac</i>	
left		<i>Dp(1.f)101</i>	
		<i>l(1)2Ad</i>	
		<i>sta</i>	
right	2B1-2	<i>Df(1)HA18</i>	
right	2B1-2	<i>Dp(1)dory21T</i>	
right	2B3-4	<i>Df(1)sta</i>	94 to 98.5 kb
left		<i>BRC</i>	
		<i>br</i>	
		<i>rdp</i>	
		<i>l(1)2Bc</i>	
		<i>l(1)2Bd</i>	
right		<i>BRC</i>	
		<i>Cp70</i>	
right	2B3-6		
right	2B3-4	<i>Df(1)br-R1</i>	125.5 to 129 kb

side	breakpoint	variant	coordinates α
right	2B5-6	<i>Df(1)pn7b</i>	144.5 to 147 kb
left	2B4-5	<i>Df(1)P154</i>	174 to 176 kb
right	2B5-6	<i>Df(1)ST469</i>	181 to 183 kb
right	2B5-6	<i>Df(1)S39</i>	208 to 213 kb
left	2B6-8	<i>Df(1)ST472</i>	210.1 to 214.5 kb
right	2B5-6	<i>Df(1)P154</i>	214 to 216 kb
left	2B6-8	<i>Df(1)dor2T</i>	
		dor	
		<i>Df(1)HF333</i>	
		hfw	
right	2B6-8	<i>y²Y67g24.2</i>	
right	2B6-8	<i>y²Y21T</i>	
right	2B6-8	<i>y²Y53T</i>	
		fmf	
right	2B9-10	<i>y²Y22T</i>	
right	2B9-10	<i>Dp(1;Y)dor13T</i>	
right	2B9-11	<i>y²Y40T</i>	
right	2B11-12	<i>Df(1)ST472</i>	
right	2B11-12	<i>Df(1)RA19</i>	
right	2B11-12	<i>Df(1)A94</i>	
		l(1)2Bg	
		l(1)2Bh	
right		<i>Dp(1;f)A1</i>	
right	2B12-13	<i>Dp(1;Y)2E</i>	
right	2B17-18	<i>Df(1)dor1T</i>	
		tlc	
right	2B17-18	<i>y²Y67g19.1</i>	
right	2B17-18	<i>Dp(1;Y)Sz78/4</i>	
right	2B17-18	<i>Dp(1;Y)Sz170</i>	
right	2B17-18	<i>Dp(1;Y)Sz303</i>	
right	2C1-2	<i>Dp(1;Y)dor1T</i>	
right	2C1-2	<i>Dp(1;Y)dor10T</i>	
right	2C1-2	<i>Dp(1;Y)dor17T</i>	
right	2C1-2	<i>Dp(1;Y)Sz71/4</i>	
left		<i>Dp(1;3)w^{vco}</i>	
		Actn	
		l(1)2Cc	
		l(1)2Cd	
		l(1)2Ce	
		usp	
right	2C10-D1	<i>Df(1)T34</i>	
	2C12	<i>Dp(1;Y)Sz280</i>	
right	2D1-2	<i>Dp(1;Y)dorY18T</i>	
left	2D1-2	<i>w^Y</i>	
left	2D1-2	<i>Dp(1;Y)w⁺303</i>	
left	2D3	<i>Df(1)JA52</i>	
		l(1)2Da	
		l(1)2Db	
		l(1)2Dc	
left	2D3-4	<i>Df(1)pn38</i>	
	2D2	csw	
		ph	
left	2D3-4	<i>Df(1)Pgd-kz</i> inferred lethal	
right		<i>Df(1)JA52</i>	
left	2D4	<i>Dp(1;Y)y²sc</i>	
		l(1)2Dg	
		Pgd	
left	2D3-6	<i>Df(1)JC105</i>	
	2D3-6	wapl	
left		<i>Df(1)J24</i>	
right	2E1-2	<i>Df(1)dor2T</i>	
right	2E1-2	<i>Dp(1)dqrY3T</i>	
right	2E1-2	<i>Dp(1)y²Y43T</i>	
left	2E1	<i>Df(1)pn7a</i>	
left	2E1-2	<i>Df(1)64c18</i>	
left	2E1-2	<i>Df(1)c99-w^{m4}</i>	
left	2E2-F1	<i>Df(1)TEM304</i> (Perrimon)	
right	2E1-2	<i>Dp(1;Y)dorY3T</i>	
	2E1	pn	
		l(1)2Ea	
		<i>Df(1)TEM501</i> (Schalet)	
		<i>Df(1)TEM1</i> (Schalet)	
right	2D6	<i>Df(1)JC105</i>	
right		<i>Df(1)J24</i>	

side	breakpoint	variant	coordinates α
left	2E3-F3	<i>Df(1)278.4B.1a</i>	5.5 to 9.0 kb
right	2E2-4	<i>y²Y43T</i>	
right	2E2-F1	<i>Df(1)Pgd35</i>	
right	2E4-F1	<i>Dp(1;f)18</i>	
right	2E1-2	<i>Df(1)dor2T</i>	
	2E	pcx	
right	2E3	<i>Df(1)pn38</i>	
left		<i>Df(1)L271</i>	55 kb
	2E3	kz	
		fs(1)k10	
right		<i>Df(1)L271</i>	61 kb
	2F1	crn	65 to 70 kb
left		<i>Df(1)2F1-3A4</i>	73.5 to 75.0 kb
		l(1)2Fb	
		l(1)2Fc	
		l(1)2Fd	
right	2F5	<i>Df(1)Pgd-kz</i>	
left		<i>Df(1)JC19</i>	
left		<i>Df(1)X12</i>	
		phl	
left		<i>Df(1)62g18</i>	
left		<i>Df(1)TEM75</i>	
		gt	
right	3A4	<i>Dp(1;Y)y²sc</i>	

α Coordinates in 2B from Chao and Guild (1986, EMBO J. 5: 143-50); coordinates in 2E-F from Haenlin, Steller, Pirrotta, and Mohier (1985, Cell 40: 827-37).

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(1)3Aa</i>	1-0.95	3A2	<i>Df(1)62g18</i>	<i>Df(1)w-rJ1</i>	gt
<i>l(1)3Ab</i>	1-0.99	3A2-3	<i>Df(1)w-rJ1</i>	<i>Df(1)64c4</i>	tko
l(1)3Ac	1-1.04	3A4	<i>Df(1)K95</i>	<i>Dp(1;3)w^{67k27}</i>	<i>l(1)zw1</i>
l(1)3Ad	1-1.15	3A6	<i>Dp(1;2)w^{67k27}</i>	<i>Dp(1;3)w⁺70h31</i>	<i>l(1)zw8</i>
l(1)3Ae	1-1.16	3A7	<i>Dp(1;2)w^{67k27}</i>	<i>Dp(1;3)w⁺70h31</i>	<i>l(1)zw4</i>
<i>l(1)3Af</i>	1-1.17	3A8	<i>Dp(1;3)w⁺70h31</i>	<i>Df(1)w-rJ2</i>	mlt(1)15 , <i>l(1)zw10</i>
l(1)3Ag	1-1.26	3A9			<i>l(1)zw13</i>
l(1)3Ah	1-1.29	3A9	<i>Df(1)w-rJ2</i>	<i>Df(1)64f1</i>	<i>l(1)zw2</i>

l(1)3Ac

phenotype: Tight alleles [*l(1)3Ac*⁹, *l(1)3Ac*¹², *l(1)3Ac*²³, *l(1)3Ac*³³] are polyphasic, dying during L2, L3, and post puparial stages; cell viable. Semilethal alleles [*l(1)3Ac*² and *l(1)3Ac*⁶] die as pupae; escapers have rough eyes, shriveled or blistered wings, and crippled metathoracic legs; phenotype of *X0* more extreme than that of *XY* males. *XY* escapers fertile but females sterile [*l(1)3Ac*²/*l(1)3Ac*² and *l(1)3Ac*²/*l(1)3Ac*⁶] or nearly so [*l(1)3Ac*⁶/*l(1)3Ac*⁶]. The latter females sterile in crosses to *l(1)3Ac*⁶ males, but produce heterozygous daughters when outcrossed (Shannon, Kaufman, Shen, and Judd, 1975, Genetics 72: 615-38). In line freed of *l(1)3Ba*⁴ (Robbins), *l(1)3Ac*¹⁴ survives as epidermal clones and as oogenic clones, which produce lethal zygotes (García-Bellido and Robbins, 1983, Genetics 103: 235-47).

molecular biology: Likely included in the chromosome walk of Mariani, Pirrotta, and Manet (1985, EMBO J. 4: 2045-52).

allele	origin	discoverer	synonym	ref α	comments β
l(1)3Ac ¹	NNG	Kaufman	<i>l(1)zw1</i> ^{5x}	1	
l(1)3Ac ²	NNG	Kaufman	<i>l(1)zw1</i> ^{7w}	1	
l(1)3Ac ³	NNG	Kaufman	<i>l(1)zw1</i> ^{15j}	1	
l(1)3Ac ⁴	NNG	Kaufman	<i>l(1)zw1</i> ^{16x}	1	
l(1)3Ac ⁵	NNG	Kaufman	<i>l(1)zw1</i> ^{28e}	1	
l(1)3Ac ⁶	NNG	Kaufman	<i>l(1)zw1</i> ³²ⁱ	1	
l(1)3Ac ⁷	NNG	Kaufman	<i>l(1)zw1</i> ^{43v}	1	

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	synonym	ref ^α	comments ^β	allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>l(1)3Ac</i>⁸	NNG	Kaufman	<i>l(1)zw1</i> ^{45a}	1		<i>l(1)3Ac</i>⁸⁴	X ray	Lefevre	<i>l(1)HA18</i>	3	also lethal in 2A
<i>l(1)3Ac</i>⁹	X ray	Abrahamson	<i>l(1)zw1</i> ^{a1}	1		<i>l(1)3Ac</i>⁸⁵	X ray	Lefevre	<i>l(1)HA37</i>	3	<i>T(1;2)3A4;32A1-2</i>
<i>l(1)3Ac</i>¹⁰	X ray	Abrahamson	<i>l(1)zw1</i> ^{a2}	1		<i>l(1)3Ac</i>⁸⁶	X ray	Lefevre	<i>l(1)HF318</i>	3	<i>Tp(3;1)3A3-4;66D-E;80</i>
<i>l(1)3Ac</i>¹¹	X ray	Judd	<i>l(1)zw1</i> ^{a17}	1		<i>l(1)3Ac</i>⁸⁷	X ray	Lefevre	<i>l(1)HF407</i>	3	
<i>l(1)3Ac</i>¹²	X ray	Elequin	<i>l(1)zw1</i> ^{b16}	1		<i>l(1)3Ac</i>⁸⁸	X ray	Lefevre	<i>l(1)JC31</i>	3	<i>ln(1)1E5;3A1-2</i>
<i>l(1)3Ac</i>¹³	X ray	Judd	<i>l(1)zw1</i> ^{b22}	1	no MZI	<i>l(1)3Ac</i>⁸⁹	X ray	Lefevre	<i>l(1)JC46</i>	3	<i>ln(1)3A4;4E1</i>
<i>l(1)3Ac</i>¹⁴	X ray	Judd	<i>l(1)zw1</i> ^{d8}	1		<i>l(1)3Ac</i>⁹⁰	X ray	Lefevre	<i>l(1)N4</i>	3	<i>T(1;2)3A5;55A</i>
<i>l(1)3Ac</i>¹⁵	X ray	Judd	<i>l(1)zw1</i> ^{d13}	1	with <i>l(1)3Ba</i> ⁴ , MZI	<i>l(1)3Ac</i>⁹¹	X ray	Lefevre	<i>l(1)RF43</i>	3	<i>T(1;4)3A2-3 (complex)</i>
<i>l(1)3Ac</i>¹⁶	EMS	Judd	<i>l(1)zw1</i> ^{E1}	1		<i>l(1)3Ac</i>⁹²	X ray	Lefevre	<i>l(1)RC45</i>	3	<i>T(1;2)3A;3C1;59D</i>
<i>l(1)3Ac</i>¹⁷	EMS	Judd	<i>l(1)zw1</i> ^{E2}	1		<i>l(1)3Ac</i>⁹³	X ray	Lefevre	<i>l(1)RC59</i>	3	
<i>l(1)3Ac</i>¹⁸	EMS	Judd	<i>l(1)zw1</i> ^{E3}	1		<i>l(1)3Ac</i>⁹⁴	X ray	Lefevre	<i>l(1)S76</i>	3	<i>T(1;2)3A2-3;42A</i>
<i>l(1)3Ac</i>¹⁹	X ray	Judd	<i>l(1)zw1</i> ^{e6}	1		<i>l(1)3Ac</i>⁹⁵	EMS	Lefevre	<i>l(1)DC827</i>	4	
<i>l(1)3Ac</i>²⁰	X ray	Abrahamson	<i>l(1)zw1</i> ^{f2}	1		<i>l(1)3Ac</i>⁹⁶	EMS	Lefevre	<i>l(1)DF941</i>	4	
<i>l(1)3Ac</i>²¹	X ray	Abrahamson	<i>l(1)zw1</i> ^{f5}	1		<i>l(1)3Ac</i>⁹⁷	EMS	Lefevre	<i>l(1)EA31</i>	4	
<i>l(1)3Ac</i>²²	X ray	Judd	<i>l(1)zw1</i> ^{g9}	1		<i>l(1)3Ac</i>⁹⁸	EMS	Lefevre	<i>l(1)EA94</i>	4	
<i>l(1)3Ac</i>²³	X ray	Judd	<i>l(1)zw1</i> ^{g17}	1		<i>l(1)3Ac</i>⁹⁹	EMS	Lefevre	<i>l(1)EC276</i>	4	
<i>l(1)3Ac</i>²⁴	X ray	Judd	<i>l(1)zw1</i> ^{g19}	1		<i>l(1)3Ac</i>¹⁰⁰	EMS	Lefevre	<i>l(1)EF418</i>	4	
<i>l(1)3Ac</i>²⁵	X ray	Judd	<i>l(1)zw1</i> ^{g26}	1		<i>l(1)3Ac</i>¹⁰¹	EMS	Lefevre	<i>l(1)VA31</i>	4	
<i>l(1)3Ac</i>²⁶	X ray	Alexander	<i>l(1)zw1</i> ^{g30}	1		<i>l(1)3Ac</i>¹⁰²	EMS	Lefevre	<i>l(1)VA259</i>	4	
<i>l(1)3Ac</i>²⁷	X ray	Judd	<i>l(1)zw1</i> ^{g31}	1	<i>ln(1)3A3-4;6B2-3, MZI</i>	<i>l(1)3Ac</i>¹⁰³	EMS	Lefevre	<i>l(1)9-96</i>	4	
<i>l(1)3Ac</i>²⁸	X ray	Alexander	<i>l(1)zw1</i> ⁱ¹³	1		<i>l(1)3Ac</i>¹⁰⁴	<i>mei-9</i> ^γ	Schalet	<i>l(1)zw1</i> ^{S1}		
		+ EI				<i>l(1)3Ac</i>¹⁰⁵	<i>mei-9</i>	Schalet	<i>l(1)zw1</i> ^{S2M}		
<i>l(1)3Ac</i>²⁹	X ray	Alexander	<i>l(1)zw1</i> ^{j20}	1		<i>l(1)3Ac</i>¹⁰⁶	<i>mei-9</i>	Schalet	<i>l(1)zw1</i> ^{S3M}		
<i>l(1)3Ac</i>³⁰	X ray	Alexander	<i>l(1)zw1</i> ^{j28}	1		<i>l(1)3Ac</i>¹⁰⁷	<i>mei-9</i>	Schalet	<i>l(1)zw1</i> ^{S4M}		
<i>l(1)3Ac</i>³¹	X ray	Alexander	<i>l(1)zw1</i> ^{k4}	1		<i>l(1)3Ac</i>¹⁰⁸	HMS		<i>l(1)HM34</i>	2	
<i>l(1)3Ac</i>³²	X ray	Judd	<i>l(1)zw1</i> ^{k5}	1		<i>l(1)3Ac</i>¹⁰⁹	HMS		<i>l(1)HM413</i>	2	
<i>l(1)3Ac</i>³³	X ray	Judd	<i>l(1)zw1</i> ^{k6}	1		<i>l(1)3Ac</i>¹¹⁰	HMS		<i>l(1)HM429</i>	2	
<i>l(1)3Ac</i>³⁴	X ray	Judd	<i>l(1)zw1</i> ^{k26}	1		<i>l(1)3Ac</i>¹¹¹	HMS		<i>l(1)HM432</i>	2	
<i>l(1)3Ac</i>³⁵	EMS		<i>l(1)zw1</i> ^{e2}	5		^α 1 = Judd, Shen, and Kaufman, 1972, <i>Genetics</i> 71: 139-56; 2 = Kraemers, Schalet, Paradi, and Huizer-Hoogteyling, 1983, <i>Mutat. Res.</i> 107: 187-201; 3 = Lefevre, 1981, <i>Genetics</i> 99: 461-80; 4 = Lefevre and Watkins, 1986, <i>Genetics</i> 113: 869-95; 5 = Lim and Snyder, 1974, <i>Genet. Res.</i> 24: 1-10; 6 = Liu and Lim, 1975, <i>Genetics</i> 79: 601-11.					
<i>l(1)3Ac</i>³⁶	EMS		<i>l(1)zw1</i> ^{e4}	5		^β MZI = maternal-zygotic interaction; demonstrated in males carrying mutant allele from heterozygous mother and variegating allele in a paternally derived duplication (Robbins, 1983, <i>Genetics</i> 103: 633-48).					
<i>l(1)3Ac</i>³⁷	EMS		<i>l(1)zw1</i> ^{e9}	5		^γ Spontaneous in the paternal X chromosome of a cross between wild-type males and <i>mei-9</i> females, such that the F ₁ females were <i>l(1)3Ac/mei-9</i> .					
<i>l(1)3Ac</i>³⁸	EMS		<i>l(1)zw1</i> ^{e10}	5		<i>l(1)3Ad</i> <i>l(1)3Ad</i> ⁶ lethal in epidermal and female germ-line clones (García-Bellido and Robbins, 1983, <i>Genetics</i> 103: 235-47).					
<i>l(1)3Ac</i>³⁹	EMS		<i>l(1)zw1</i> ^{e15}	5		allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>l(1)3Ac</i>⁴⁰	EMS		<i>l(1)zw1</i> ^{e16}	5		<i>l(1)3Ad</i>¹	NNG	Kaufman	<i>l(1)zw8</i> ^{6b}	1	t.s. semilethal, MZI
<i>l(1)3Ac</i>⁴¹	EMS		<i>l(1)zw1</i> ^{e18}	5		<i>l(1)3Ad</i>²	NNG	Kaufman	<i>l(1)zw8</i> ^{20m}	1	
<i>l(1)3Ac</i>⁴²	EMS		<i>l(1)zw1</i> ^{e24}	5		<i>l(1)3Ad</i>³	NNG	Kaufman	<i>l(1)zw8</i> ^{28y}	1	
<i>l(1)3Ac</i>⁴³	EMS		<i>l(1)zw1</i> ^{e25}	5		<i>l(1)3Ad</i>⁴	NNG	Kaufman	<i>l(1)zw8</i> ^{33o}	1	
<i>l(1)3Ac</i>⁴⁴	EMS		<i>l(1)zw1</i> ^{e33}	5		<i>l(1)3Ad</i>⁵	NNG	Kaufman	<i>l(1)zw8</i> ^{44j}	1	t.s. semilethal, MZI
<i>l(1)3Ac</i>⁴⁵	EMS		<i>l(1)zw1</i> ^{e34}	5		<i>l(1)3Ad</i>⁶	X ray	Judd	<i>l(1)zw8</i> ⁸¹⁰	1	MZI
<i>l(1)3Ac</i>⁴⁶	EMS		<i>l(1)zw1</i> ^{e40}	5		<i>l(1)3Ad</i>⁷	X ray	Judd	<i>l(1)zw8</i> ⁸²⁸	1	MZI
<i>l(1)3Ac</i>⁴⁷	EMS		<i>l(1)zw1</i> ^{e49}	5		<i>l(1)3Ad</i>⁸	EMS		<i>l(1)zw8</i> ^{e5}	5	
<i>l(1)3Ac</i>⁴⁸	EMS		<i>l(1)zw1</i> ^{e52}	5		<i>l(1)3Ad</i>⁹	EMS		<i>l(1)zw8</i> ^{e17}	5	
<i>l(1)3Ac</i>⁴⁹	EMS		<i>l(1)zw1</i> ^{e54}	5		<i>l(1)3Ad</i>¹⁰	EMS		<i>l(1)zw8</i> ^{e35}	5	
<i>l(1)3Ac</i>⁵⁰	EMS		<i>l(1)zw1</i> ^{e67}	5		<i>l(1)3Ad</i>¹¹	EMS		<i>l(1)zw8</i> ^{e45}	5	
<i>l(1)3Ac</i>⁵¹	EMS		<i>l(1)zw1</i> ^{e70}	5		<i>l(1)3Ad</i>¹²	EMS		<i>l(1)zw8</i> ^{e65}	5	
<i>l(1)3Ac</i>⁵²	EMS		<i>l(1)zw1</i> ^{e76}	5		<i>l(1)3Ad</i>¹³	EMS		<i>l(1)zw8</i> ^{e78}	5	
<i>l(1)3Ac</i>⁵³	EMS		<i>l(1)zw1</i> ^{e83}	5		<i>l(1)3Ad</i>¹⁴	EMS		<i>l(1)zw8</i> ^{e88}	5	
<i>l(1)3Ac</i>⁵⁴	EMS		<i>l(1)zw1</i> ^{e84}	5		<i>l(1)3Ad</i>¹⁵	MMS		<i>l(1)zw8</i> ^{m1}	6	
<i>l(1)3Ac</i>⁵⁵	EMS		<i>l(1)zw1</i> ^{e89}	5		<i>l(1)3Ad</i>¹⁶	MMS		<i>l(1)zw8</i> ^{m42}	6	
<i>l(1)3Ac</i>⁵⁶	EMS		<i>l(1)zw1</i> ^{e94}	5		<i>l(1)3Ad</i>¹⁷	MMS		<i>l(1)zw8</i> ^{m44}	6	
<i>l(1)3Ac</i>⁵⁷	EMS		<i>l(1)zw1</i> ^{e95}	5		<i>l(1)3Ad</i>¹⁸	MMS		<i>l(1)zw8</i> ^{m48}	6	
<i>l(1)3Ac</i>⁵⁸	TEM		<i>l(1)zw1</i> ²	5		<i>l(1)3Ad</i>¹⁹	MMS		<i>l(1)zw8</i> ^{m61}	6	
<i>l(1)3Ac</i>⁵⁹	TEM		<i>l(1)zw1</i> ²¹⁴	5	<i>T(1;3)3Ac</i> ⁵⁹	<i>l(1)3Ad</i>²⁰	MMS		<i>l(1)zw8</i> ^{m64}	6	
<i>l(1)3Ac</i>⁶⁰	TEM		<i>l(1)zw1</i> ³⁰¹	5		<i>l(1)3Ad</i>²¹	MMS		<i>l(1)zw8</i> ^{m79}	6	
<i>l(1)3Ac</i>⁶¹	TEM		<i>l(1)zw1</i> ⁴⁰¹	5		<i>l(1)3Ad</i>²²	MMS		<i>l(1)zw8</i> ^{m89}	6	
<i>l(1)3Ac</i>⁶²	TEM		<i>l(1)zw1</i> ^{e411}	5		<i>l(1)3Ad</i>²³	MMS		<i>l(1)zw8</i> ^{m92}	6	
<i>l(1)3Ac</i>⁶³	MMS		<i>l(1)zw1</i> ^{m7}	6		<i>l(1)3Ad</i>²⁴	MMS		<i>l(1)zw8</i> ^{m107}	6	
<i>l(1)3Ac</i>⁶⁴	MMS		<i>l(1)zw1</i> ^{m13}	6		<i>l(1)3Ad</i>²⁵	MMS		<i>l(1)zw8</i> ^{m114}	6	
<i>l(1)3Ac</i>⁶⁵	MMS		<i>l(1)zw1</i> ^{m14}	6		<i>l(1)3Ad</i>²⁶	X ray	Lefevre	<i>l(1)RC2</i>	3	
<i>l(1)3Ac</i>⁶⁶	MMS		<i>l(1)zw1</i> ^{m18}	6							
<i>l(1)3Ac</i>⁶⁷	MMS		<i>l(1)zw1</i> ^{m22}	6							
<i>l(1)3Ac</i>⁶⁸	MMS		<i>l(1)zw1</i> ^{m23}	6							
<i>l(1)3Ac</i>⁶⁹	MMS		<i>l(1)zw1</i> ^{m34}	6							
<i>l(1)3Ac</i>⁷⁰	MMS		<i>l(1)zw1</i> ^{m50}	6							
<i>l(1)3Ac</i>⁷¹	MMS		<i>l(1)zw1</i> ^{m51}	6							
<i>l(1)3Ac</i>⁷²	MMS		<i>l(1)zw1</i> ^{m53}	6							
<i>l(1)3Ac</i>⁷³	MMS		<i>l(1)zw1</i> ^{m71}	6							
<i>l(1)3Ac</i>⁷⁴	MMS		<i>l(1)zw1</i> ^{m76}	6							
<i>l(1)3Ac</i>⁷⁵	MMS		<i>l(1)zw1</i> ^{m82}	6							
<i>l(1)3Ac</i>⁷⁶	MMS		<i>l(1)zw1</i> ^{m83}	6							
<i>l(1)3Ac</i>⁷⁷	MMS		<i>l(1)zw1</i> ^{m86}	6							
<i>l(1)3Ac</i>⁷⁸	MMS		<i>l(1)zw1</i> ^{m105}	6							
<i>l(1)3Ac</i>⁷⁹	MMS		<i>l(1)zw1</i> ^{m106}	6							
<i>l(1)3Ac</i>⁸⁰	MMS		<i>l(1)zw1</i> ^{m106}	6							
<i>l(1)3Ac</i>⁸¹	X ray	Lefevre	<i>l(1)C44</i>	3							
<i>l(1)3Ac</i>⁸²	X ray	Lefevre	<i>l(1)GA3</i>	3							
<i>l(1)3Ac</i>⁸³	X ray	Lefevre	<i>l(1)GE224</i>	3	<i>ln(1)3A;20F</i>						

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>l(1)3Ad²⁷</i>	X ray	Lefevre	<i>l(1)RF15</i>	3	
<i>l(1)3Ad²⁸</i>	EMS	Lefevre	<i>l(1)EC204</i>	4	
<i>l(1)3Ad²⁹</i>	EMS	Lefevre	<i>l(1)VA4</i>	4	
<i>l(1)3Ad³⁰</i>	EMS	Lefevre	<i>l(1)VA19</i>	4	
<i>l(1)3Ad³¹</i>	EMS	Lefevre	<i>l(1)VA50</i>	4	
<i>l(1)3Ad³²</i>	EMS	Lefevre	<i>l(1)VE664</i>	4	
<i>l(1)3Ad³³</i>	EMS	Lefevre	<i>l(1)VE667</i>	4	
<i>l(1)3Ad³⁴</i>	EMS	Lefevre	<i>l(1)VE815</i>	4	
<i>l(1)3Ad³⁵</i>	spont	Schalet	<i>l(1)I6-94</i> <i>l(1)zw8^{S1}</i>		
<i>l(1)3Ad³⁶</i>	HMS		<i>l(1)HM6</i>	2	
<i>l(1)3Ad³⁷</i>	HMS		<i>l(1)HM458</i>	2	

^α 1 = Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56; 2 = Kraemers, Schalet, Paradi, and Huiser-Hoogteyting, 1983, Mutat. Res. 107: 187-201; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 5 = Lim and Snyder, 1974, Genet. Res. 24: 1-10; 6 = Liu and Lim, 1975, Genetics 79: 601-11.

^β MZI = maternal-zygotic interaction; demonstrated in males carrying mutant allele from heterozygous mother and variegating allele in a paternally derived duplication (Robbins, 1983, Genetics 103: 633-48).

l(1)3Ae

phenotype: Alleles tested [*l(1)Ae²*, *l(1)Ae³*, *l(1)Ae⁶*, *l(1)Ae⁸*, *l(1)Ae¹⁰*, *l(1)Ae¹¹*] die in post puparial stage, but growth slowing starts at various earlier stages; tested alleles survive in hemizygous condition in gynandromorphs; hemizygous eyes rough. Pharate adults dissected from *l(1)3Ae¹* and *l(1)Ae⁵* pupae show rough eyes, sparse microchaetae, and incomplete dorsal midline; XY but not XO males of both rescued by *Dp(1;4)w^{m65g}*, exhibiting phenotype of dissected pharate adults described above (Shannon, Kaufman, Shen, and Judd, 1972, Genetics 72: 615-38).

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>l(1)3Ae¹</i>	NNG	Kaufman	<i>l(1)zw4²⁹¹</i>	1	
<i>l(1)3Ae²</i>	X ray	Judd	<i>l(1)zw4^{d28}</i>	1	no MZI
<i>l(1)3Ae³</i>	X ray	Judd	<i>l(1)zw4^{e4}</i>	1	no MZI
<i>l(1)3Ae⁴</i>	X ray + EI	Alexander	<i>l(1)zw4^{g8}</i>	1	
<i>l(1)3Ae⁵</i>	spont	Lefevre	<i>l(1)zw4^{g11}</i>	1	
<i>l(1)3Ae⁶</i>	X ray	Judd	<i>l(1)zw4^{g24}</i>	1	semilethal, no MZI
<i>l(1)3Ae⁷</i>	X ray + EI	Alexander	<i>l(1)zw4^{h6}</i>	1	
<i>l(1)3Ae⁸</i>	X ray + EI	Alexander	<i>l(1)zw4ⁱ²⁴</i>	1	no MZI
<i>l(1)3Ae⁹</i>	EI	Alexander	<i>l(1)zw4^{j6}</i>	1	
<i>l(1)3Ae¹⁰</i>	X ray	Alexander	<i>l(1)zw4^{j27}</i>	1	
<i>l(1)3Ae¹¹</i>	EI	Alexander	<i>l(1)zw4^{k16}</i>	1	MZI
<i>l(1)3Ae¹²</i>	EI	Alexander	<i>l(1)zw4^{k27}</i>	1	
<i>l(1)3Ae¹³</i>	EMS		<i>l(1)zw4^{e11}</i>	4	
<i>l(1)3Ae¹⁴</i>	EMS		<i>l(1)zw4^{e48}</i>	4	
<i>l(1)3Ae¹⁵</i>	EMS		<i>l(1)zw4^{e57}</i>	4	
<i>l(1)3Ae¹⁶</i>	EMS		<i>l(1)zw4^{e82}</i>	4	
<i>l(1)3Ae¹⁷</i>	EMS		<i>l(1)zw4^{e85}</i>	4	
<i>l(1)3Ae¹⁸</i>	MMS		<i>l(1)zw4^{m5}</i>	5	
<i>l(1)3Ae¹⁹</i>	MMS		<i>l(1)zw4^{m11}</i>	5	
<i>l(1)3Ae²¹</i>	MMS		<i>l(1)zw4^{m15}</i>	5	
<i>l(1)3Ae²²</i>	MMS		<i>l(1)zw4^{m20}</i>	5	
<i>l(1)3Ae²³</i>	MMS		<i>l(1)zw4^{m68}</i>	5	
<i>l(1)3Ae²⁴</i>	MMS		<i>l(1)zw4^{m101}</i>	5	
<i>l(1)3Ae²⁵</i>	X ray	Lefevre	<i>l(1)GA107</i>	2	
<i>l(1)3Ae²⁶</i>	X ray	Lefevre	<i>l(1)GE240</i>	2	
<i>l(1)3Ae²⁷</i>	EMS	Lefevre	<i>l(1)DF944</i>	3	
<i>l(1)3Ae²⁸</i>	EMS	Lefevre	<i>l(1)EC241</i>	3	
<i>l(1)3Ae²⁹</i>	EMS	Lefevre	<i>l(1)EC270</i>	3	
<i>l(1)3Ae³⁰</i>	EMS	Lefevre	<i>l(1)EF442</i>	3	

^α 1 = Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56; 2 = Lefevre, 1981, Genetics 99: 461-80; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 4 = Lim and Snyder, 1974, Genet. Res. 24: 1-10; 5 = Liu and Lim, 1975, Genetics 79: 601-11.

^β MZI = maternal-zygotic interaction; demonstrated in males carrying mutant allele from heterozygous mother and variegating allele in a paternally derived duplication (Robbins, 1983, Genetics 103: 633-48).

l(1)3Ag

allele	origin	synonym	ref ^α	comments ^β
<i>l(1)3Ag¹</i>	EMS	<i>l(1)zw13^{e50}</i>	1	MZI
<i>l(1)3Ag²</i>	EMS	<i>l(1)zw13^{e77}</i>	1	
<i>l(1)3Ag³</i>	TEM	<i>l(1)zw13^{l3}</i>	1	MZI
<i>l(1)3Ag⁴</i>	MMS	<i>l(1)zw13^{m2}</i>	2	
<i>l(1)3Ag⁵</i>	MMS	<i>l(1)zw13^{m6}</i>	2	
<i>l(1)3Ag⁶</i>	MMS	<i>l(1)zw13^{m115}</i>	2	

^α 1 = Lim and Snyder, 1974, Genet. Res. 24: 1-10; 2 = Liu and Lim, 1975, Genetics 79: 601-11.

^β MZI = maternal-zygotic interaction; demonstrated in males carrying mutant allele from heterozygous mother and variegating allele in a paternally derived duplication (Robbins, 1983, Genetics 103: 633-48). Survival of duplication-bearing males cold sensitive; *l(1)3Ag/w⁺Y* males yield frequent meiotic nondisjunction.

l(1)3Ah

phenotype: Lethal alleles [*l(1)3Ah⁷*, *l(1)3Ah⁹*, *l(1)3Ah¹¹*] die at the egg-larval boundary; *l(1)3Ah¹⁶* and *l(1)3Ah¹²* die shortly after hatching. Cell viable, but gynandromorphs show reduced survival. *l(1)3Ah⁶* semilethal with biphasic lethal expression in early larval and imago stages; escapers have rough eyes and are male fertile; inviable in combination with deficiency or lethal alleles (Shannon, Kaufman, Shen, and Judd, 1972, Genetics 72: 615-38). *l(1)3Ah¹³* viable in epidermal but not female germ-line clones (García-Bellido and Robbins, 1983, Genetics 103: 235-47).

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>l(1)3Ah¹</i>	NNG	Reichert	<i>l(1)zw2^{lf}</i>	1	
<i>l(1)3Ah²</i>	NNG	Kaufman	<i>l(1)zw2^{lu}</i>	1	
<i>l(1)3Ah³</i>	NNG	Kaufman	<i>l(1)zw2^{lx}</i>	1	
<i>l(1)3Ah⁵</i>	NNG	Kaufman	<i>l(1)zw2^{3g}</i>	1	
<i>l(1)3Ah⁶</i>	NNG	Kaufman	<i>l(1)zw2^{6p}</i>	1	
<i>l(1)3Ah⁷</i>	NNG	Kaufman	<i>l(1)zw2^{20f}</i>	1	
<i>l(1)3Ah⁸</i>	NNG	Kaufman	<i>l(1)zw2^{28c}</i>	1	
<i>l(1)3Ah⁹</i>	NNG	Kaufman	<i>l(1)zw2^{39p}</i>	1	
<i>l(1)3Ah¹⁰</i>	X ray	Abrahamson	<i>l(1)zw2^{a3}</i>	1	
<i>l(1)3Ah¹¹</i>	X ray	Le Fever	<i>l(1)zw2^{b11}</i>	1	MZI
<i>l(1)3Ah¹²</i>	X ray	Judd	<i>l(1)zw2^{b26}</i>	1	
<i>l(1)3Ah¹³</i>	X ray	Judd	<i>l(1)zw2^{c21}</i>	1	MZI
<i>l(1)3Ah¹⁴</i>	X ray	Judd	<i>l(1)zw2^{c28}</i>	1	
<i>l(1)3Ah¹⁵</i>	X ray	Abrahamson	<i>l(1)zw2^{f3}</i>	1	
<i>l(1)3Ah¹⁶</i>	X ray	Lefevre	<i>l(1)zw2^{g4}</i>	1	
<i>l(1)3Ah¹⁷</i>	X ray	Lefevre	<i>l(1)zw2^{g6}</i>	1	
<i>l(1)3Ah¹⁸</i>	EMS	Kaufman	<i>l(1)zw2^{g12}</i>	1	
<i>l(1)3Ah¹⁹</i>	X ray + EI	Alexander	<i>l(1)zw2^{h5}</i>	1	
<i>l(1)3Ah²⁰</i>	X ray + EI	Alexander	<i>l(1)zw2ⁱ²⁰</i>	1	
<i>l(1)3Ah²¹</i>	X ray	Alexander	<i>l(1)zw2ⁱ²³</i>	1	MZI
<i>l(1)3Ah²²</i>	EMS		<i>l(1)zw2^{e1}</i>	5	
<i>l(1)3Ah²³</i>	EMS		<i>l(1)zw2^{e3}</i>	5	
<i>l(1)3Ah²⁴</i>	EMS		<i>l(1)zw2^{e7}</i>	5	
<i>l(1)3Ah²⁵</i>	EMS		<i>l(1)zw2^{e8}</i>	5	
<i>l(1)3Ah²⁶</i>	EMS		<i>l(1)zw2^{e14}</i>	5	
<i>l(1)3Ah²⁷</i>	EMS		<i>l(1)zw2^{e20}</i>	5	
<i>l(1)3Ah²⁸</i>	EMS		<i>l(1)zw2^{e27}</i>	5	
<i>l(1)3Ah²⁹</i>	EMS		<i>l(1)zw2^{e31}</i>	5	
<i>l(1)3Ah³⁰</i>	EMS		<i>l(1)zw2^{e37}</i>	5	
<i>l(1)3Ah³¹</i>	EMS		<i>l(1)zw2^{e41}</i>	5	
<i>l(1)3Ah³²</i>	EMS		<i>l(1)zw2^{e43}</i>	5	
<i>l(1)3Ah³³</i>	EMS		<i>l(1)zw2^{e44}</i>	5	
<i>l(1)3Ah³⁴</i>	EMS		<i>l(1)zw2^{e46}</i>	5	
<i>l(1)3Ah³⁵</i>	EMS		<i>l(1)zw2^{e47}</i>	5	
<i>l(1)3Ah³⁶</i>	EMS		<i>l(1)zw2^{e53}</i>	5	
<i>l(1)3Ah³⁷</i>	EMS		<i>l(1)zw2^{e59}</i>	5	

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>l(1)3Ah³⁸</i>	EMS		<i>l(1)zw2^{e61}</i>	5	
<i>l(1)3Ah³⁹</i>	EMS		<i>l(1)zw2^{e66}</i>	5	
<i>l(1)3Ah⁴⁰</i>	EMS		<i>l(1)zw2^{e68}</i>	5	
<i>l(1)3Ah⁴¹</i>	TEM		<i>l(1)zw2⁸</i>	5	
<i>l(1)3Ah⁴²</i>	TEM		<i>l(1)zw2⁹</i>	5	
<i>l(1)3Ah⁴³</i>	TEM		<i>l(1)zw2¹⁴</i>	5	
<i>l(1)3Ah⁴⁴</i>	TEM		<i>l(1)zw2¹⁰⁹</i>	5	
<i>l(1)3Ah⁴⁵</i>	TEM		<i>l(1)zw2²⁰¹</i>	5	
<i>l(1)3Ah⁴⁶</i>	TEM		<i>l(1)zw2³⁰³</i>	5	
<i>l(1)3Ah⁴⁷</i>	TEM		<i>l(1)zw2³⁰⁵</i>	5	
<i>l(1)3Ah⁴⁸</i>	TEM		<i>l(1)zw2⁴⁰²</i>	5	
<i>l(1)3Ah⁴⁹</i>	TEM		<i>l(1)zw2⁴⁰⁵</i>	5	
<i>l(1)3Ah⁵¹</i>	TEM		<i>l(1)zw2⁴⁰⁶</i>	5	
<i>l(1)3Ah⁵²</i>	TEM		<i>l(1)zw2⁴¹²</i>	5	
<i>l(1)3Ah⁵³</i>	TEM		<i>l(1)zw2⁴¹³</i>	5	
<i>l(1)3Ah⁵⁴</i>	MMS		<i>l(1)zw2^{m3}</i>	6	
<i>l(1)3Ah⁵⁵</i>	MMS		<i>l(1)zw2^{m8}</i>	6	
<i>l(1)3Ah⁵⁶</i>	MMS		<i>l(1)zw2^{m26}</i>	6	
<i>l(1)3Ah⁵⁷</i>	MMS		<i>l(1)zw2^{m30}</i>	6	
<i>l(1)3Ah⁵⁸</i>	MMS		<i>l(1)zw2^{m37}</i>	6	
<i>l(1)3Ah⁵⁹</i>	MMS		<i>l(1)zw2^{m38}</i>	6	
<i>l(1)3Ah⁶⁰</i>	MMS		<i>l(1)zw2^{m43}</i>	6	
<i>l(1)3Ah⁶¹</i>	MMS		<i>l(1)zw2^{m46}</i>	6	
<i>l(1)3Ah⁶²</i>	MMS		<i>l(1)zw2^{m59}</i>	6	
<i>l(1)3Ah⁶³</i>	MMS		<i>l(1)zw2^{m60}</i>	6	
<i>l(1)3Ah⁶⁴</i>	MMS		<i>l(1)zw2^{m67}</i>	6	
<i>l(1)3Ah⁶⁵</i>	MMS		<i>l(1)zw2^{m69}</i>	6	with <i>l(1)3Ba²⁰</i>
<i>l(1)3Ah⁶⁶</i>	MMS		<i>l(1)zw2^{m70}</i>	6	
<i>l(1)3Ah⁶⁷</i>	MMS		<i>l(1)zw2^{m74}</i>	6	
<i>l(1)3Ah⁶⁸</i>	MMS		<i>l(1)zw2^{m75}</i>	6	
<i>l(1)3Ah⁶⁹</i>	MMS		<i>l(1)zw2^{m95}</i>	6	
<i>l(1)3Ah⁷⁰</i>	MMS		<i>l(1)zw2^{m99}</i>	6	
<i>l(1)3Ah⁷¹</i>	MMS		<i>l(1)zw2^{m104}</i>	6	
<i>l(1)3Ah⁷²</i>	MMS		<i>l(1)zw2^{m108}</i>	6	
<i>l(1)3Ah⁷³</i>	MMS		<i>l(1)zw2^{m109}</i>	6	
<i>l(1)3Ah⁷⁴</i>	MMS		<i>l(1)zw2^{m110}</i>	6	
<i>l(1)3Ah⁷⁵</i>	MMS		<i>l(1)zw2^{m113}</i>	6	
<i>l(1)3Ah⁷⁶</i>	X ray	Lefevre	<i>l(1)C208</i>	3	
<i>l(1)3Ah⁷⁷</i>	X ray	Lefevre	<i>l(1)HC114</i>	3	
<i>l(1)3Ah⁷⁸</i>	X ray	Lefevre	<i>l(1)JC109</i>	3	
<i>l(1)3Ah⁷⁹</i>	X ray	Lefevre	<i>l(1)RA43</i>	3	
<i>l(1)3Ah⁸⁰</i>	X ray	Lefevre	<i>l(1)RF33</i>	3	
<i>l(1)3Ah⁸¹</i>	EMS	Lefevre	<i>l(1)DC716</i>	4	
<i>l(1)3Ah⁸²</i>	EMS	Lefevre	<i>l(1)DF921</i>	4	
<i>l(1)3Ah⁸³</i>	EMS	Lefevre	<i>l(1)EA12</i>	4	
<i>l(1)3Ah⁸⁴</i>	EMS	Lefevre	<i>l(1)EA96</i>	4	
<i>l(1)3Ah⁸⁵</i>	EMS	Lefevre	<i>l(1)EC216</i>	4	
<i>l(1)3Ah⁸⁶</i>	EMS	Lefevre	<i>l(1)EF440</i>	4	
<i>l(1)3Ah⁸⁷</i>	EMS	Lefevre	<i>l(1)VA329</i>	4	
<i>l(1)3Ah⁸⁸</i>	EMS	Lefevre	<i>l(1)VA359</i>	4	
<i>l(1)3Ah⁸⁹</i>	EMS	Lefevre	<i>l(1)VE616</i>	4	
<i>l(1)3Ah⁹⁰</i>	EMS	Lefevre	<i>l(1)VE722</i>	4	
<i>l(1)3Ah⁹¹</i>	spont	Schalet	<i>l(1)17-59</i>		
			<i>l(1)zw2^{S1}</i>		
<i>l(1)3Ah⁹²</i>	mei-9 ^γ	Schalet	<i>l(1)zw2^{S2M}</i>		
<i>l(1)3Ah⁹³</i>	HMS		<i>l(1)HM434</i>	2	

^α 1 = Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56; 2 = Kramers, Schalet, Paradi, and Huiser-Hoogteyling, 1983, Mutat. Res. 107: 187-201; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 5 = Lim and Snyder, 1974, Genet. Res. 24: 1-10; 6 = Liu and Lim, 1975, Genetics 79: 601-11.

^β MZI = maternal-zygotic interaction; demonstrated in males carrying mutant allele from heterozygous mother and variegating allele in a paternally derived duplication (Robbins, 1983, Genetics 103: 633-48).

^γ Spontaneous in the paternal X chromosome of a cross between wild-type males and *mei-9* females, such that the F₁ females were *l(1)3Ah/mei-9*.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(1)3Ba</i>	1-1.32	3B1	<i>Df(1)64f1</i>	<i>Df(1)62d18</i>	sgg ; <i>l(1)zw3</i>
<i>l(1)3Bb</i>	1-1.41	3B2	<i>Df(1)62d18</i>	<i>Df(1)w258-45</i>	<i>l(1)zw6</i>

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(1)3Bc</i>	1-1.43	3B3	<i>Df(1)w258-45</i>	<i>Dp(1;3)N^{264-58a}</i>	<i>l(1)zw12</i>
<i>l(1)3Bd</i>	1-1.43	3B4	<i>Dp(1;3)N^{264-58a}</i>		<i>l(1)zw7</i>
			<i>Df(1)X12</i>		
<i>l(1)3Be</i>	1-1.47	3B5	<i>Dp(1;3)N^{264-58a}</i>		dwg
			<i>Df(1)X12</i>		<i>l(1)zw5</i>
<i>l(1)3Bf</i>	1-1.48	3B6	<i>Dp(1;3)N^{264-58a}</i>		<i>l(1)zw11</i>
			<i>Df(1)X12</i>		

l(1)3Bb

phenotype: Tested alleles die without growth in first larval instar [*l(1)3Bb⁴*, *l(1)3Bb⁶*, *l(1)3Bb⁷*] or grow slightly [*l(1)3Bb¹*, *l(1)3Bb⁵*]. Few reach second instar but fail to grow further. All survive as XY but not as XO males in presence of either *Dp(1;4)w^{m65g}* or *Dp(1;3)w^{m49a}*. Survive as gynandromorphs when only tergites and occasionally wing tissue mutant; mutant tissue etched, lacks bristles, and deformed in the case of wing tissue (Shannon, Kaufman, Shen, and Judd, 1972, Genetics 72: 615-38). *l(1)3Bb¹* and *l(1)3Bb⁴* viable in epidermal clones; only *l(1)3Bb¹* survives in oogenic clones, and they produced viable zygotes (García-Bellido and Robbins, 1983, Genetics 103: 235-47).

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>l(1)3Bb¹</i>	ICR170	Hochman	<i>l(1)zw6^{a25}</i>	1	
<i>l(1)3Bb²</i>	X ray	Elequin	<i>l(1)zw6^{b17}</i>	1	
<i>l(1)3Bb³</i>	X ray	Judd	<i>l(1)zw6^{b23}</i>	1	MZI
<i>l(1)3Bb⁴</i>	X ray	Judd	<i>l(1)zw6^{e5}</i>	1	
<i>l(1)3Bb⁵</i>	X ray	Judd	<i>l(1)zw6^{e13}</i>	1	
<i>l(1)3Bb⁶</i>	X ray	Alexander	<i>l(1)zw6^{g7}</i>	1	
<i>l(1)3Bb⁷</i>	X ray	Judd	<i>l(1)zw6^{h12}</i>	1	
<i>l(1)3Bb⁸</i>	EMS		<i>l(1)zw6^{e12}</i>	4	
<i>l(1)3Bb⁹</i>	EMS		<i>l(1)zw6^{e72}</i>	4	
<i>l(1)3Bb¹⁰</i>	EMS		<i>l(1)zw6^{e80}</i>	4	
<i>l(1)3Bb¹¹</i>	TEM		<i>l(1)zw6⁴⁰³</i>	4	
<i>l(1)3Bb¹²</i>	MMS		<i>l(1)zw6^{m31}</i>	5	
<i>l(1)3Bb¹³</i>	MMS		<i>l(1)zw6^{m47}</i>	5	
<i>l(1)3Bb¹⁴</i>	MMS		<i>l(1)zw6^{m57}</i>	5	
<i>l(1)3Bb¹⁵</i>	MMS		<i>l(1)zw6^{m58}</i>	5	
<i>l(1)3Bb¹⁶</i>	MMS		<i>l(1)zw6^{m66}</i>	5	
<i>l(1)3Bb¹⁷</i>	X ray	Lefevre	<i>l(1)A14</i>	2	<i>In(1)3B1-2;4E1</i>
<i>l(1)3Bb¹⁸</i>	X ray	Lefevre	<i>l(1)HC222</i>	2	
<i>l(1)3Bb¹⁹</i>	EMS	Lefevre	<i>l(1)EC287</i>	3	
<i>l(1)3Bb²⁰</i>	EMS	Lefevre	<i>l(1)VE734</i>	3	
<i>l(1)3Bb²¹</i>	P ^γ		<i>l(1)zw6^{P1}</i>	6	
<i>l(1)3Bb²²</i>	P ^γ		<i>l(1)zw6^{P2}</i>	6	

^α 1 = Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56; 2 = Lefevre, 1981, Genetics 99: 461-80; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 4 = Lim and Snyder, 1974, Genet. Res. 24: 1-10; 5 = Liu and Lim, 1975, Genetics 79: 601-11; 6 = Reddy, Zehring, Wheeler, Pirrotta, Hadfield, Hall, and Rosbashi, 1984, Cell 38: 701-10.

^β MZI = maternal-zygotic interaction; demonstrated in males carrying mutant allele from heterozygous mother and variegating allele in a paternally derived duplication (Robbins, 1983, Genetics 103: 633-48).

^γ No P-derived sequences detectable; possibly deletion generated by P excision.

l(1)3Bc

phenotype: Die as first instar larvae. XY but not XO males survive in combination with *Dp(1;4)w^{m65g}* and *Dp(1;3)w^{m49a}*. Mutant tissue survives and is phenotypically normal in gynandromorphs (Shannon, Kaufman, Shen, and Judd, 1972, Genetics 72: 615-38). Germ-line clones in females don't survive (García-Bellido and Robbins, 1983, Genetics 103: 235-47).

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>l(1)3Bc</i>¹	NNG	Kaufman	<i>l(1)zw12</i> ³⁵ⁿ	1	
<i>l(1)3Bc</i>²	X ray	Alexander	<i>l(1)zw12</i> ^{k1}	1	MZI
<i>l(1)3Bc</i>³	X ray	Alexander	<i>l(1)zw12</i> ^{k3}	1	
<i>l(1)3Bc</i>⁴	X ray + DMSO	Alexander	<i>l(1)zw12</i> ^{k9}	1	
<i>l(1)3Bc</i>⁵			<i>l(1)zw12</i> ^{e34}	5	
<i>l(1)3Bc</i>⁶	MMS		<i>l(1)zw12</i> ^{m19}	6	
<i>l(1)3Bc</i>⁷	MMS		<i>l(1)zw12</i> ^{m28}	6	
<i>l(1)3Bc</i>⁸	MMS		<i>l(1)zw12</i> ^{m32}	6	
<i>l(1)3Bc</i>⁹	MMS		<i>l(1)zw12</i> ^{m40}	6	
<i>l(1)3Bc</i>¹⁰	MMS		<i>l(1)zw12</i> ^{m56}	6	
<i>l(1)3Bc</i>¹¹	MMS		<i>l(1)zw12</i> ^{m65}	6	
<i>l(1)3Bc</i>¹²	X ray	Lefevre	<i>l(1)A117</i>	3	
<i>l(1)3Bc</i>¹³	X ray	Lefevre	<i>l(1)C130</i>	3	
<i>l(1)3Bc</i>¹⁴	X ray	Lefevre	<i>l(1)HC102</i>	3	
<i>l(1)3Bc</i>¹⁵	X ray	Lefevre	<i>l(1)RC53</i>	3	
<i>l(1)3Bc</i>¹⁶	EMS	Lefevre	<i>l(1)VE658</i>	4	
<i>l(1)3Bc</i>¹⁷	HMS		<i>l(1)HM16</i>	2	
<i>l(1)3Bc</i>¹⁸	HMS		<i>l(1)HM402</i>	2	

^α 1 = Judd, Shen, and Kaufman, 1972, *Genetics* 71: 139-56; 2 = Kraemers, Schalet, Paradi, and Huizer-Hoogteyling, 1983, *Mutat. Res.* 107: 187-201; 3 = Lefevre, 1981, *Genetics* 99: 461-80; 4 = Lefevre and Watkins, 1986, *Genetics* 113: 869-95; 5 = Lim and Snyder, 1974, *Genet. Res.* 24: 1-10; 6 = Liu and Lim, 1975, *Genetics* 79: 601-11.

^β MZI = maternal-zygotic interaction; demonstrated in males carrying mutant allele from heterozygous mother and variegating allele in a paternally derived duplication (Robbins, 1983, *Genetics* 103: 633-48).

l(1)3Bd

phenotype: *l(1)3Bd*¹ males die either without growth in L1 or following protracted growth to full sized L2 stage. *l(1)3Bd*³ males die as full-size L1 or variable-size L2 stage. XY but not XO males survive with slightly rough eyes and are fertile in presence of *Dp(1;4)w*^{m65g}, *Dp(1;3)w*^{m49a}, and *Dp(1;3)N*^{264-58a}. Mutant tissue does not survive in gynandromorphs (Shannon, Kaufman, Shen, and Judd, 1972, *Genetics* 72: 615-38), nor in epidermal or female germ-line clones (García-Bellido and Robbins, 1983, *Genetics* 103: 235-47).

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>l(1)3Bd</i>¹	NNG	Kaufman	<i>l(1)zw7</i> ^{31x}	1	MZI
<i>l(1)3Bd</i>²	X ray	Judd	<i>l(1)zw7</i> ^{e3}	1	MZI
<i>l(1)3Bd</i>³	X ray	Judd	<i>l(1)zw7</i> ^{g20}	1	MZI
<i>l(1)3Bd</i>⁴	EMS		<i>l(1)zw7</i> ^{e13}	4	
<i>l(1)3Bd</i>⁵	EMS		<i>l(1)zw7</i> ^{e30}	4	
<i>l(1)3Bd</i>⁶	EMS		<i>l(1)zw7</i> ^{e42}	4	
<i>l(1)3Bd</i>⁷	EMS		<i>l(1)zw7</i> ^{e92}	4	
<i>l(1)3Bd</i>⁸	TEM		<i>l(1)zw7</i> ¹⁰	4	
<i>l(1)3Bd</i>⁹	TEM		<i>l(1)zw7</i> ³⁰²	4	
<i>l(1)3Bd</i>¹⁰	TEM		<i>l(1)zw7</i> ⁴⁰⁷	4	
<i>l(1)3Bd</i>¹¹	MMS		<i>l(1)zw7</i> ^{m12}	5	
<i>l(1)3Bd</i>¹²	X ray	Lefevre	<i>l(1)C26</i>	2	
<i>l(1)3Bd</i>¹³	X ray	Lefevre	<i>l(1)GE203</i>	2	
<i>l(1)3Bd</i>¹⁴	EMS	Lefevre	<i>l(1)VE698</i>	3	
<i>l(1)3Bd</i>¹⁵	mei-9 ^γ	Schalet	<i>l(1)zw7</i> ^{SIM}	3	

^α 1 = Judd, Shen, and Kaufman, 1972, *Genetics* 71: 139-56; 2 = Lefevre, 1981, *Genetics* 99: 461-80; 3 = Lefevre and Watkins, 1986, *Genetics* 113: 869-95; 4 = Lim and Snyder, 1974, *Genet. Res.* 24: 1-10; 5 = Liu and Lim, 1975, *Genetics* 79: 601-11.

^β MZI = maternal-zygotic interaction; demonstrated in males carrying mutant allele from heterozygous mother and variegating allele in a paternally derived duplication (Robbins, 1983, *Genetics* 103: 633-48).

^γ Spontaneous in the paternal X chromosome of a cross between wild-type males and *mei-9* females, such that the F₁ females were *l(1)3Bd/mei-9*.

l(1)3Bf

phenotype: Most mutant larvae survive to L2 stage; L2 survivors grow slowly and reach half normal size before dying. XY but not XO males survive in combination with *Dp(1;4)w*^{m65g}, *Dp(1;3)w*^{m49a}, and *Dp(1;3)N*^{264-58a}; lack varying numbers of orbital, ocellar, and vertical bristles; occasionally wing veins thickened; fertile. Mutant tissue [*l(1)3Bf*⁵, *l(1)3Bf*⁶, *l(1)3Bf*⁷] does not survive in gynandromorphs (Shannon, Kaufman, Shen, and Judd, 1972, *Genetics* 72: 615-38). *l(1)Bf*⁵ lethal in both epidermal and oogenic clones (García-Bellido and Robbins, 1983, *Genetics* 103: 235-47).

allele	origin	discoverer	synonym	ref ^α
<i>l(1)3Bf</i>¹	NNG	Kaufman	<i>l(1)zw11</i> ^{3w}	1
<i>l(1)3Bf</i>²	NNG	Kaufman	<i>l(1)zw11</i> ^{14e}	1
<i>l(1)3Bf</i>³	NNG	Kaufman	<i>l(1)zw11</i> ^{20t}	1
<i>l(1)3Bf</i>⁴	NNG	Kaufman	<i>l(1)zw11</i> ^{25f}	1
<i>l(1)3Bf</i>⁵	ICR170	Hochman	<i>l(1)zw11</i> ^{a5}	1
<i>l(1)3Bf</i>⁶	X ray	Elequin	<i>l(1)zw11</i> ^{b18}	1
<i>l(1)3Bf</i>⁷	X ray	Judd	<i>l(1)zw11</i> ^{g3}	1
<i>l(1)3Bf</i>⁸	EMS		<i>l(1)zw11</i> ^{e22}	3
<i>l(1)3Bf</i>⁹	TEM		<i>l(1)zw11</i> ¹¹	3
<i>l(1)3Bf</i>¹⁰	TEM		<i>l(1)zw11</i> ⁴⁰⁴	3
<i>l(1)3Bf</i>¹¹	MMS		<i>l(1)zw11</i> ^{m4}	4
<i>l(1)3Bf</i>¹²	MMS		<i>l(1)zw11</i> ^{m39}	4
<i>l(1)3Bf</i>¹³	MMS		<i>l(1)zw11</i> ^{m85}	4
<i>l(1)3Bf</i>¹⁴	MMS		<i>l(1)zw11</i> ^{m97}	4
<i>l(1)3Bf</i>¹⁵	MMS		<i>l(1)zw11</i> ^{m111}	4
<i>l(1)3Bf</i>¹⁶	EMS	Lefevre	<i>l(1)EF525</i>	2
<i>l(1)3Bf</i>¹⁷	EMS	Lefevre	<i>l(1)VE725</i>	2

^α 1 = Judd, Shen, and Kaufman, 1972, *Genetics* 71: 139-56; 2 = Lefevre, 1981, *Genetics* 99: 461-80; 3 = Lim and Snyder, 1974, *Genet. Res.* 24: 1-10; 4 = Liu and Lim, 1975, *Genetics* 79: 601-11.

l(1)3C3

location: 1-1.6 (between *w* and *rst*).

origin: Synthetic.

discoverer: Lefevre and Wilkins.

references: 1964, *Genetics* 50: 264.

phenotype: Male lethal. *l(1)3C3/w* is normal. RK2.

cytology: The consequence of deleting 3C3 through 3C6, a region postulated to contain duplicated loci (Lefevre and Green, 1972, *Chromosoma* 36: 391-412). Originally associated with the deficiency for band 3C3 alone obtained as a single recombinant carrying the left end of *T(1;4)w*^{mJ} = *T(1;4)3C2-3;20;102C* and the right end of *In(1)rst*³ = *In(1)3C3-5;20B*. That result attributed to the combined effects of the deficiency for 3C3 and a position effect on 3C5-6, neither of which is lethal by itself (Lefevre and Green).

l(1)3C-D

3C contains two named lethally mutable loci, *crm* and *N*. 3D contains one lethally mutable locus.

locus	genetic location		cytological location		included in	excluded from	synonym
	location	location	location	location			
<i>l(1)3Ca</i>	1-1.49	3C1	<i>Df(1)w-rJ1</i>	<i>Df(1)X12</i>			<i>crm</i> , <i>l(1)zw9</i>
<i>l(1)3Cb</i>	1-3.0	3C7	<i>Df(1)T30</i>	<i>Df(1)dm75e19</i>			N
<i>l(1)3Da</i>		3D6	<i>Df(1)A53</i>	<i>Df(1)GA113</i>			

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>l(1)3Da</i>¹	X ray	Lefevre	<i>l(1)GE204</i>	1	<i>T(1;2)3D3-4;41</i> , also <i>dm</i>
<i>l(1)3Da</i>²	X ray	Lefevre	<i>l(1)GE246</i>	1	also <i>dm</i> ; <i>In(1)3B3;20F</i>

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allele	origin	discoverer	synonym	ref ^α	comments ^β	side	breakpoint	variant	DNA coordinates ^α
I(1)3Da³	X ray	Lefevre	<i>l(1)JA53</i>	1	<i>T(1;A)3D5;4B5;?</i> ; complex, also <i>dm</i>	right	3A4	<i>Df(1)pn7a</i>	
I(1)3Da⁴	X ray	Lefevre	<i>l(1)JC9</i>	1	not <i>dm</i>	right	3A4-6	<i>Df(1)62g18</i>	
I(1)3Da⁵	X ray	Lefevre	<i>l(1)RA55</i>	1	also <i>dm</i> ; EL1-2/L	right	3A4-6	<i>Df(1)62j26</i>	
I(1)3Da⁶	X ray	Lefevre	<i>l(1)RF29</i>	1	also <i>dm</i> ; <i>T(1;2;3)3D3-6;7D;41;</i> <i>93F-94A1</i>	right	3A4-6	<i>Df(1)TEM304</i>	
I(1)3Da⁷	EMS	Lefevre	<i>l(1)EC238</i>	2	also <i>dm</i> ; rearranged	left	3A4-6	<i>Df(1)w258-42</i>	
						left	3A4-6	<i>Df(1)w-N71a</i>	
						left	3A4-6	<i>Dp(1;3)w^{67k27}</i>	
						right	3A5-B4	<i>Df(1)278.4B.1a</i>	
							3A6	I(1)3Ad	
							3A7	I(1)3Ae	
						left	3A6-8	<i>Dp(1;2)w^{70h31}</i>	
							3A8	I(1)3Af	
						left	3A8-9	<i>Df(1)64j4</i>	
						left	3A8-9	<i>Df(1)w-rJ2</i>	
							3A9	I(1)3Ag	
						left	3A9	<i>Df(1)mm69</i>	
							3A9	I(1)3Ah	
						left	3A9-B1	<i>Df(1)64f1</i>	
						left	3A9-B2	<i>Dp(1;3)w^{49a}</i>	
						right	3B	<i>Df(1)TEM1</i>	
							3B1	I(1)3Ba	
						right	3B1	<i>l(1)mm69</i>	
						left	3B1-2	<i>Df(1)62d18</i>	0 to 1.0 kb
						left	3B2	<i>Df(1)TEM202</i>	5.5 to 6.6 kb
						left	3B1-2	per	
								<i>T(1;4)JC43</i>	13.3 to 13.7 kb
						right		per	
						right	3B1-2	<i>Df(1)64j4</i>	16.9 to 19.2 kb --97 kb
						right	3B1-2	<i>Df(1)K95</i>	
						left	3B1-2	<i>Df(1)w64d</i>	22.9 to 24.5 kb
						left	3B1-2	<i>Dp(1;4)w^{m65g}</i>	
							3B2	I(1)3Bb	
						left	3B2-3	<i>Df(1)w258-45</i>	--87 kb
							3B3	I(1)3Bc	
						left	3B2-4	<i>Dp(1;3)N-264-58a</i>	
						right	3B3	<i>Df(1)TEM7</i>	
						right	3B3-4	<i>Df(1)64f1</i>	--83 kb
							3B4	I(1)3Bd	
							3B5	dwg	
							3B6	<i>l(1)3Bf</i>	
						right	3B5-C1	<i>Df(1)X12</i>	
							3C1	crm	
						left	3C1-2	<i>Df(1)N-8</i>	
						left	3C1-2	<i>Dp(1;3)w^{51b}</i>	0 kb
							3C2	w	
						left	3C1-2	<i>Df(1)w67c23.2</i>	1.5 to 3.5 kb
						right	3C2-3	<i>Dp(1;2)w^{70h}</i>	--29 kb
						right	3C1-3	<i>Df(1)TEM501</i>	
						right	3C2	<i>Df(1)64c18</i>	
						right	3C2-3	<i>Df(1)64c4</i>	
						left	3C2-3	<i>Df(1)N-64i16</i>	
						left	3C2-3	<i>Df(1)N-64j15</i>	
						right	3C2-3	<i>Df(1)w64d</i>	
						right	3C2-3	<i>Df(1)w258-45</i>	--65 kb
						right	3C2-3	<i>Df(1)w-rJ1</i>	
						right	3C2-3	<i>Df(1)w-rJ2</i>	
						right	3C2-3	<i>Dp(1;2)w^{70h31}</i>	
						right	3C2-4	<i>Df(1)TEM75</i>	
						right	3C3	<i>Df(1)TEM75</i>	
						right	3C3-5	<i>Df(1)TEM202</i>	
						right	3C3-5	<i>Df(1)w258-11</i>	
						right	3C3-5	<i>Df(1)w258-42?</i>	
						right	3C3-5	<i>Dp(1;4)w^{m65g}</i>	
						right	3C3-5	<i>Dp(1;3)w^{vco}</i>	
						right	3C5	<i>Df(1)JC19</i>	
						right	3C5-6	<i>Df(1)w258-42?</i>	
						right	3C6-7	<i>Df(1)62d18</i>	--65 kb
						left	3C6-7	<i>Df(1)T30</i>	
						left	3C7	N	
						right	3C6-7	<i>Df(1)N-68f19</i>	-26.2 to -24.7 kb
						left	3C7	<i>Df(1)N-62b1</i>	-19.3 to -18.6 kb
						left		<i>Df(1)N-76b8</i>	-2.2 to -1.3 kb
						right	3C6-7	<i>Df(1)N-66i25</i>	0.8 to 2.7 kb
						right	3C7	N	
						right	3C8-9	<i>Df(1)T30</i>	

I(1)3E
Five lethally mutable loci recorded by Lefevre.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(1)3Ea</i>	1-3.6	3E1	<i>Df(1)N8</i>	<i>Df(1)GBM207</i>	slc
I(1)3Eb		3E2	<i>Dp(1;3)w^{49a}</i>	<i>Df(1)N-8</i>	
I(1)3Ec		3E3	<i>Df(1)dm75e19</i>	<i>Dp(1;3)w^{49a}</i>	
I(1)3Ed		3E4-6	<i>Df(1)WC159</i>	<i>Df(1)dm75e19</i>	
I(1)3Ee		3E7	<i>Df(1)WC159</i>	<i>Df(1)HF366</i>	

allele	origin	discoverer	synonym	ref ^α	comments
I(1)3Eb¹	X ray	Lefevre	<i>l(1)GF332</i>	1	<i>T(1;A)3E5; het</i>
I(1)3Eb²	X ray	Lefevre	<i>l(1)RF52</i>	1	<i>T(1;A)3E1-2;?</i>
I(1)3Eb³	EMS	Lefevre	<i>l(1)DA564</i>	2	
I(1)3Eb⁴	EMS	Lefevre	<i>l(1)DA618</i>	2	
I(1)3Eb⁵	EMS	Lefevre	<i>l(1)DC805</i>	2	
I(1)3Eb⁶	EMS	Lefevre	<i>l(1)DF957</i>	2	
I(1)3Eb⁷	EMS	Lefevre	<i>l(1)VA44</i>	2	
I(1)3Eb⁸	EMS	Lefevre	<i>l(1)VA148</i>	2	EL/AO
I(1)3Eb⁹	EMS	Lefevre	<i>l(1)VE619</i>	2	
I(1)3Ec¹	X ray	Lefevre	<i>l(1)HF345</i>	1	<i>T(1;2)3C7;27E</i>
I(1)3Ec²	X ray	Lefevre	<i>l(1)RF53</i>	1	<i>T(1;3)3E1;80</i>
I(1)3Ec³	X ray	Lefevre	<i>l(1)VE880</i>	1	P/NME
I(1)3Ed¹	X ray	Lefevre	<i>l(1)GA2 = l(1)GA58</i>	1	<i>In(1)3C5;3E-F;</i> <i>18A1-2</i>
I(1)3Ed²	X ray	Lefevre	<i>l(1)GE210</i>	1	
I(1)3Ed³	X ray	Lefevre	<i>l(1)JC43</i>	1	<i>T(1;4)3A10;3E5;</i> <i>102C</i>
I(1)3Ee¹	X ray	Lefevre	<i>l(1)LS6</i>	1	
I(1)3Ee²	EMS	Lefevre	<i>l(1)EC245</i>	2	
I(1)3Ee³	EMS	Lefevre	<i>l(1)EF423</i>	2	

^α 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95.

DEFICIENCY MAP OF REGION 3

side	breakpoint	variant	DNA coordinates ^α
left	2F5-3A1	<i>Df(1)TEM75</i>	
left	2F6-3A1	<i>Df(1)X12</i>	
left	3A1	<i>Df(1)TEM6</i>	
left	3A1-2	<i>Df(1)62g18</i>	
left	3A1-2	<i>Df(1)62j26</i>	
	3A3	gt	
left	3A1-2	<i>Df(1)w-rJ1</i>	
	3A2-3	tko	
right	3A3	<i>Df(1)TEM6</i>	
left	3A3	<i>Df(1)TEM7</i>	
right	3A3	<i>Df(1)TEM304</i>	
left	3A3-4	<i>Df(1)64c4</i>	
left	3A3-4	<i>Df(1)K95</i>	
left	3A3-4	<i>Df(1)w258-11</i>	
right	3A4	<i>Df(1)2F1-3A4</i>	
	3A4	I(1)3Ac	
right	3A4	<i>Df(1)pn7a</i>	
right	3A4-6	<i>Df(1)62g18</i>	
right	3A4-6	<i>Df(1)62j26</i>	
right	3A4-6	<i>Df(1)TEM304</i>	
left	3A4-6	<i>Df(1)w258-42</i>	
left	3A4-6	<i>Df(1)w-N71a</i>	
left	3A4-6	<i>Dp(1;3)w^{67k27}</i>	
right	3A5-B4	<i>Df(1)278.4B.1a</i>	
	3A6	I(1)3Ad	
	3A7	I(1)3Ae	
left	3A6-8	<i>Dp(1;2)w^{70h31}</i>	
	3A8	I(1)3Af	
left	3A8-9	<i>Df(1)64j4</i>	
left	3A8-9	<i>Df(1)w-rJ2</i>	
	3A9	I(1)3Ag	
left	3A9	<i>Df(1)mm69</i>	
	3A9	I(1)3Ah	
left	3A9-B1	<i>Df(1)64f1</i>	
left	3A9-B2	<i>Dp(1;3)w^{49a}</i>	
right	3B	<i>Df(1)TEM1</i>	
	3B1	I(1)3Ba	
right	3B1	<i>l(1)mm69</i>	
left	3B1-2	<i>Df(1)62d18</i>	0 to 1.0 kb
left	3B2	<i>Df(1)TEM202</i>	5.5 to 6.6 kb
left	3B1-2	per	
		<i>T(1;4)JC43</i>	13.3 to 13.7 kb
		per	
right	3B1-2	<i>Df(1)64j4</i>	16.9 to 19.2 kb --97 kb
right	3B1-2	<i>Df(1)K95</i>	
left	3B1-2	<i>Df(1)w64d</i>	22.9 to 24.5 kb
left	3B1-2	<i>Dp(1;4)w^{m65g}</i>	
	3B2	I(1)3Bb	
left	3B2-3	<i>Df(1)w258-45</i>	--87 kb
	3B3	I(1)3Bc	
left	3B2-4	<i>Dp(1;3)N-264-58a</i>	
right	3B3	<i>Df(1)TEM7</i>	
right	3B3-4	<i>Df(1)64f1</i>	--83 kb
	3B4	I(1)3Bd	
	3B5	dwg	
	3B6	<i>l(1)3Bf</i>	
right	3B5-C1	<i>Df(1)X12</i>	
	3C1	crm	
left	3C1-2	<i>Df(1)N-8</i>	
left	3C1-2	<i>Dp(1;3)w^{51b}</i>	0 kb
	3C2	w	
left	3C1-2	<i>Df(1)w67c23.2</i>	1.5 to 3.5 kb
right	3C2-3	<i>Dp(1;2)w^{70h}</i>	--29 kb
right	3C1-3	<i>Df(1)TEM501</i>	
right	3C2	<i>Df(1)64c18</i>	
right	3C2-3	<i>Df(1)64c4</i>	
left	3C2-3	<i>Df(1)N-64i16</i>	
left	3C2-3	<i>Df(1)N-64j15</i>	
right	3C2-3	<i>Df(1)w64d</i>	
right	3C2-3	<i>Df(1)w258-45</i>	--65 kb
right	3C2-3	<i>Df(1)w-rJ1</i>	
right	3C2-3	<i>Df(1)w-rJ2</i>	
right	3C2-3	<i>Dp(1;2)w^{70h31}</i>	
right	3C2-4	<i>Df(1)TEM75</i>	
right</			

side	breakpoint	variant	DNA coordinates ^α
right	3C10-11	<i>Df(1)w-N71a</i>	
left	3C11-12	<i>Df(1)dm75e19</i>	
right	3D2-3	<i>w⁺y</i>	
right	3D3-4	<i>Df(1)N-64j15</i>	-2 to 0 kb
right	3D3-4	<i>Dp(1;3)w⁺303</i>	
	3D4	<i>dnc</i>	
left	3D4-5	<i>Df(1)GA102</i>	
left	3D4-5	<i>Df(1)JA53</i>	
right		<i>Df(1)N-74h</i>	34 to 42 kb
right	3D4-5	<i>Df(1)N-64i16</i>	50 to 58 kb
	3D5	<i>dm</i>	
	3D6	<i>I(1)3Da</i>	
left	3D6-E1	<i>Df(1)GA113</i>	
right	3D6-E1	<i>Df(1)GBM207</i>	
right	3D6-E1	<i>Dp(1;3)w⁺51b</i>	
right	3D5-7	<i>Dp(1;3)N-264-58a</i>	
	3E1	<i>slc</i>	
right	3E1-2	<i>Df(1)N-8</i>	
	3E2	<i>I(1)3Eb</i>	
right	3E2-3	<i>Dp(1;3)w⁺49a</i>	
	3E3	<i>I(1)3Ec</i>	
right	3E3-4	<i>Df(1)dm75e19</i>	
right	3E3-4	<i>Df(1)W-N14A1-b4-1</i>	
	3E4-6	<i>I(1)3Ed</i>	
left	3E6-7	<i>Df(1)WC159</i>	
	3E7	<i>I(1)3Ee</i>	
left	3E7-8	<i>Df(1)HF366</i>	
right	3E8-F2	<i>Dp(1;3)w⁺67k27</i>	

^α Coordinates in 3B1-2 from Bargiello and Young (1984, Proc. Nat. Acad. Sci. USA 81: 2142-46); coordinates in 3B1-2 to 3C2-3 from Pirrotta, Hadfield, and Pretorius (1983, EMBO J. 2: 927-34); coordinates in 3C2-7 from Kidd, Lockett, and Young (1983, Cell 34: 421-33); and coordinates in 3D from Davis and Davidson (1986, Mol. Cell Biol. 6: 1464-70).

I(1)5CDa

location: 1-15.1 (Voelker and Wisely, 1982, DIS 58: 150-51).

origin: Induced by ethyl methanesulfonate.

synonym: *l(1)E12* (or *l(1)E7* by label mixup?).

references: Suzuki, Piternick, Hayashi, Tarasoff, Baillie, and Erasmus, 1967, Proc. Nat. Acad. Sci. USA 57: 907-12.

Tarasoff and Suzuki, 1970, Dev. Biol. 23: 492-509.

phenotype: Development protracted and adults sterile when reared at 22°; protracted even more but adults fertile when reared at 17°. Sensitive to 29° at all stages of development. Lethality in midpupal stage. Shift down prior to lethal phase improves survival.

cytology: Placed in 5C5-D6 on the basis of its inclusion in *Df(1)N73 = Df(1)5C2;5D56* but not in *Df(1)C149 = Df(1)5A8-9;5C5-6*.

other information: *l(1)E12* mapped to 35.4 by Tarasoff and Suzuki and to 15.1 by Voelker and Wisely. Could Voelker and Wisely have received a mislabeled stock of *l(1)E7* (1-15.2) from Vancouver?

**I(1)6*

location: 1-0.4.

origin: Spontaneous.

discoverer: Bridges, 14d9.

references: 1916, Genetics 1: 149.

phenotype: Rare survivors interpreted as recombinants led Bridges to place *l(1)6* 0.4 unit to the left of *y*.

I(1)6D

Eight lethally mutable loci identified, but not ordered

by Lefevre.

locus	included in	excluded from	synonym
<i>I(1)6Da</i>	<i>Dp(1;3)sn 13a1</i>	<i>Df(1)ct-J6</i>	
<i>I(1)6Db</i>	<i>Dp(1;3)sn 13a1</i>	<i>Df(1)ct-J6</i>	<i>l(1)EM24</i>
<i>I(1)6Dc</i>	<i>Dp(1;3)sn 13a1</i>	<i>Df(1)ct-J6</i>	
<i>I(1)6Dd</i>	<i>Dp(1;3)sn 13a1</i>	<i>Df(1)ct-J6</i>	
<i>I(1)6De</i>	<i>Dp(1;3)sn 13a1</i>	<i>Df(1)ct-J6</i>	<i>l(1)EM25</i>
<i>I(1)6Df</i>	<i>Dp(1;3)sn 13a1</i>	<i>Df(1)ct-J6</i>	
<i>I(1)6Dg</i>	<i>Dp(1;3)sn 13a1</i>	<i>Df(1)ct-J6</i>	
<i>I(1)6Dh</i>	<i>Dp(1;3)sn 13a1</i>	<i>Df(1)ct-J6</i>	

allele	origin	discoverer	synonym	ref ^α	comments
<i>I(1)6Da</i>¹	X ray	Lefevre	<i>l(1)C214</i>	1, 3	L-P/VME
<i>I(1)6Db</i>¹	EMS	Lefevre	<i>l(1)DC785</i>	2	
<i>I(1)6Db</i>²	EMS	Lefevre	<i>l(1)EC224</i>	2	
			<i>l(1)EM24</i>		
<i>I(1)6Db</i>³	EMS	Lefevre	<i>l(1)VA251</i>	2	
<i>I(1)6Dc</i>¹	X ray	Lefevre	<i>l(1)HC245</i>	1	
<i>I(1)6Dc</i>²	X ray	Lefevre	<i>l(1)GA31</i>	1	
<i>I(1)6Dc</i>³	EMS	Lefevre	<i>l(1)EA42</i>	2, 3	E-L/AO
			<i>l(1)EM25</i>		
<i>I(1)6Dd</i>¹	X ray	Lefevre	<i>l(1)HC233</i>	1	
<i>I(1)6Dd</i>²	EMS	Lefevre	<i>l(1)DF919</i>	2	
<i>I(1)6Dd</i>³	EMS	Lefevre	<i>l(1)EF455</i>	2	
<i>I(1)6Dd</i>⁴	EMS	Lefevre	<i>l(1)EF461</i>	2	
<i>I(1)6Dd</i>⁵	EMS	Lefevre	<i>l(1)VE636</i>	2	
<i>I(1)6Dd</i>⁶	EMS	Lefevre	<i>l(1)VE735</i>	2	
<i>I(1)6Dd</i>⁷	EMS	Lefevre	<i>l(1)VE921</i>	2, 3	L-P/NME
<i>I(1)6De</i>¹	EMS	Lefevre	<i>l(1)DF962</i>	2, 3	L-P/VME
*<i>I(1)6Df</i>¹	X ray	Lefevre	<i>l(1)S41</i>	1	<i>ln(1)5D7-8;6D7-8</i>
<i>I(1)6Df</i>²	EMS	Lefevre	<i>l(1)VA179</i>	2, 3	P/VME
<i>I(1)6Dg</i>¹	EMS	Lefevre	<i>l(1)VA234</i>	2, 3	P/AO
<i>I(1)6Dh</i>¹	X ray	Lefevre	<i>l(1)RA52</i>	1	

^α 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 3 = Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14.

I(1)6E

This region plus 6F and 7A subjected to saturation mutagenesis by Nicklas and Cline (1983, Genetics 103: 617-31) and sampled extensively by Lefevre and Watkins (1986, Genetics 99: 869-95). Where alleles still existed, Nicklas and Cline tested their mutants against those of Lefevre.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>I(1)6Ea</i>	1-18.3	6E1-2	<i>Df(1)ct-J6</i>	<i>Df(1)Sxl-bt</i>	<i>l(1)jnL4</i>
<i>I(1)6Eb</i>	1-18.3-.8	6E4	<i>Df(1)Sxl-bt</i>	<i>Df(1)HA32</i>	<i>l(1)jnLX</i>
<i>l(1)6Ec</i>	1-18.8	6E1-5	<i>Df(1)Sxl-bt</i>	<i>Df(1)HA32</i>	ogre, <i>l(1)jnL3</i>
<i>I(1)6Ed</i>	1-18.8	6E1-5	<i>Df(1)Sxl-bt</i>	<i>Df(1)HA32</i>	<i>l(1)jnL2</i>
<i>I(1)6Ee</i>	1-18.9	6E5-6	<i>Df(1)HA32</i>	<i>Df(1)Sxl-ra</i>	<i>l(1)jnL1</i>

allele	origin	discoverer	synonym	ref ^α	comments
*<i>I(1)6Ea</i>¹	X ray	Lefevre	<i>l(1)N84</i>	1	<i>ln(1)6E1-2;8B4-7</i>
<i>I(1)6Ea</i>²	EMS	Lefevre	<i>l(1)DC737</i>	2, 4	E/NME
<i>I(1)6Ea</i>³					
to					
<i>I(1)6Ea</i>⁹	EMS	Nicklas		3	E-L
<i>I(1)6Eb</i>¹	X ray	Lefevre	<i>l(1)HC217</i>	1	
<i>I(1)6Eb</i>²	EMS	Nicklas	<i>l(1)jnLX</i>	3, 4	P-A/VME

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)6Ed</i>¹ to <i>l(1)6Ed</i>⁹	EMS	Nicklas	<i>l(1)jnL2</i>	3, 4	E/NME
<i>l(1)6Ee</i>¹	X ray	Lefevre	<i>l(1)KC26</i>	1	
<i>l(1)6Ee</i>²	EMS	Lefevre	<i>l(1)EA22</i>	2	
<i>l(1)6Ee</i>³	EMS	Lefevre	<i>l(1)EC251</i>	2	
<i>l(1)6Ee</i>⁴ to <i>l(1)6Ee</i>¹⁸	EMS	Nicklas	<i>l(1)jnL1</i>	3, 4	E/NME

^α 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 3 = Nicklas and Cline, 1983, Genetics 103: 617-31; 4 = Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14.

l(1)6F

Three lethally mutable loci, one of which is *Sxl*; the single alleles of *l(1)6Fb* and *l(1)6Fc* lost before they could be tested against Niklas' and Cline's lethals.

locus	cytological location	included in	excluded from	synonym
<i>l(1)6Fa</i>	6F5	<i>Df(1)Sxl-ra</i>	<i>Df(1)cm</i>	<i>Sxl</i>
<i>l(1)6Fb</i>	6F5	<i>Df(1)HA32</i>	<i>Df(1)cm</i>	
<i>l(1)6Fc</i>	6F7			

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)6Fb</i>¹	X ray	Lefevre	<i>l(1)HC290</i>	1	<i>T(1;2)6F;40-41</i>
<i>l(1)6Fc</i>¹	X ray	Lefevre	<i>l(1)HA68</i>	1	<i>ln(1)6F;10B2; semilethal</i>

^α 1 = Lefevre, 1981, Genetics 99: 461-80;

l(1)7: see *dor*¹

l(1)7A

Five mutable loci, including one named locus, *adl-1*.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(1)7Aa</i>¹	1-19.4	7A3	<i>Df(1)ct-j4</i>	<i>Df(1)RF19</i>	<i>l(1)jnR1</i>
<i>l(1)7Ab</i>¹	1-19.6	7A6-8	<i>Df(1)RF19</i>	<i>Df(1)HA32</i>	<i>l(1)jnR2</i>
			<i>Df(1)GA34</i>	<i>Dp(1;2)sn⁺72d</i>	
<i>l(1)7Ac</i>¹	1-19.8	7A6	<i>Df(1)RF19</i>	<i>Df(1)HA32</i>	<i>l(1)jnR3</i>
			<i>Df(1)GA34</i>	<i>Dp(1;2)sn⁺72d</i>	
<i>l(1)7Ad</i>¹	1-19.9	7A8	<i>Dp(1;2)sn⁺72d</i>		<i>l(1)jnR4</i>
			<i>Df(1)Sxl-ra</i>		

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)7Aa</i>¹	X ray	Lefevre	<i>l(1)C62</i>	1	<i>ln(1)6F9;12F</i>
<i>l(1)7Aa</i>²	X ray	Lefevre	<i>l(1)JA9</i>	1	<i>ln(1)7A3;20F; L/NME</i>
<i>l(1)7Aa</i>³	X ray	Lefevre	<i>l(1)GA75</i>	1	<i>T(1;3)7A3;83E</i>
<i>l(1)7Aa</i>⁴	X ray	Lefevre	<i>l(1)GA83</i>	1	<i>ln(1)7A1-7B3</i>
<i>l(1)7Aa</i>⁵	X ray	Lefevre	<i>l(1)KC13</i>	1	
<i>l(1)7Aa</i>⁶	X ray	Lefevre	<i>l(1)RA49</i>	1	<i>T(1;3)7A1-2;80</i>
<i>l(1)7Aa</i>⁷	X ray	Lefevre	<i>l(1)RA62</i>	1	
<i>l(1)7Aa</i>⁸	X ray	Lefevre	<i>l(1)RC35</i>	1	<i>T(1;2)7A3;49A1-5</i>
<i>l(1)7Aa</i>⁹	X ray	Lefevre	<i>l(1)RF27</i>	1	
<i>l(1)7Aa</i>¹⁰	X ray	Lefevre	<i>l(1)RF42</i>	1	complex aberration
<i>l(1)7Aa</i>¹¹	X ray	Lefevre	<i>l(1)RF46</i>	1	<i>T(1;3)7A1;80-81</i>
<i>l(1)7Aa</i>¹²	EMS	Lefevre	<i>l(1)EA54</i>	2	
<i>l(1)7Aa</i>¹³	EMS	Lefevre	<i>l(1)VE758</i>	2	
<i>l(1)7Aa</i>¹⁴ to <i>l(1)7Aa</i>⁵¹	EMS	Nicklas		2	E-L
<i>l(1)7Ab</i>¹					

allele	origin	discoverer	synonym	ref ^α	comments
to <i>l(1)7Ab</i>²² <i>l(1)7Ab</i>²³	EMS	Nicklas		3	
	spont	Schalet	<i>l(1)13-95</i>		
<i>l(1)7Ac</i>¹	X ray	Lefevre	<i>l(1)HC141</i>	1	
<i>l(1)7Ac</i>²	X ray	Lefevre	<i>l(1)HF302</i>	1	<i>ln(1)7A6-8;12E; noncomplementing</i>
<i>l(1)7Ac</i>³	X ray	Lefevre	<i>l(1)JA59</i>	1	<i>noncomplementing</i>
<i>l(1)7Ac</i>⁴	X ray	Lefevre	<i>l(1)JE53</i>	1	<i>T(1;3)5A7;7A6;86D</i>
<i>l(1)7Ac</i>⁵	X ray	Lefevre	<i>l(1)RC57</i>	1	
<i>l(1)7Ac</i>⁶	X ray	Lefevre	<i>l(1)RC61</i>	1	complements <i>l(1)7Ac</i> ⁸

and *l(1)7Ac*⁹; EL/L complements *l(1)7Ac*⁸ and *l(1)7Ac*⁹; EL/L complements *l(1)7Ac*⁸ and *l(1)7Ac*⁹; EL/L complements *l(1)7Ac*⁶ and *l(1)7Ac*⁷; EL/L

<i>l(1)7Ac</i>⁷	EMS	Lefevre	<i>l(1)EF465</i>	2	
<i>l(1)7Ac</i>⁸	EMS	Lefevre	<i>l(1)VE852</i>	2	complements <i>l(1)7Ac</i> ⁶
<i>l(1)7Ac</i>⁹ to <i>l(1)7Ac</i>²⁷ <i>l(1)7Ac</i>²⁸	EMS	Nicklas		3	E
	spont	Schalet	<i>l(1)14-4</i>		

<i>l(1)7Ad</i>¹ to <i>l(1)7Ad</i>¹⁰	EMS	Nicklas		3	P-A
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^α 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95. 3 = Nicklas and Cline, 1983, Genetics 103: 617-31. 4 = Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14.

l(1)7B

Six lethally mutable loci including *kf* and *ct*, both part of the cut complex.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(1)7Ba</i>	20.0	7B1	<i>Df(1)RF19</i>	<i>Df(1)HA32</i>	<i>kf</i>
<i>l(1)7Bb</i>	20.0	7B3	<i>Df(1)GA34</i>	<i>Df(1)RF19</i>	<i>ct</i>
<i>l(1)7Bc</i>		7B5		<i>Df(1)GA34</i>	
<i>l(1)7Bd</i>					
<i>l(1)7Be</i>					
<i>l(1)7Bf</i>					

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)7Bc</i>¹	EMS	Lefevre	<i>l(1)EA68</i>	2, 3	LP/NME
<i>l(1)7Bc</i>²	EMS	Lefevre	<i>l(1)VE890</i>	2	
<i>l(1)7Bd</i>¹	X ray	Lefevre	<i>l(1)N63</i>	1	
<i>l(1)7Bd</i>²	X ray	Lefevre	<i>l(1)RC21</i>	1	
<i>l(1)7Bd</i>³	EMS	Lefevre	<i>l(1)VA156</i>	2, 3	LP/NME
<i>l(1)7Be</i>¹	EMS	Lefevre	<i>l(1)DC829</i>	2	
<i>l(1)7Bf</i>¹	X ray	Lefevre	<i>l(1)GF320</i>	1	

^α 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95. 3 = Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14.

l(1)7C: Dominant Lethal in 7C

location: 1-{22}.

origin: Inferred from the haploinviability of deficiencies for 7C.

synonym: *Hiv*.

references: Lefevre and Johnson, 1973, Genetics 74: 633-45.

cytology: Placed in 7C5-9 on the basis of the failure to recover *ct sn* deficiencies and on the failure of deficiencies for *ct* to extend to the right beyond 7C4 and of a *sn* deficiency to extend to the left beyond 7D1.

l(1)7C

Eight lethally mutable loci, only one of which has more than a single allele, identified by Lefevre.

locus	cytological		
	location	included in	excluded from
<i>l(1)7Ca</i>	7C1	<i>ct⁺Yy⁺</i>	<i>Df(1)ct4b1</i>
<i>l(1)7Cb</i>	7C2	<i>ct⁺Yy⁺</i>	<i>Df(1)ct4b1</i>
<i>l(1)7Cc</i>	7C4	<i>Dp(1;2)sn⁺72d</i>	<i>ct⁺Yy⁺</i>
<i>l(1)7Cd</i>	7C5		
<i>l(1)7Ce</i>			
<i>l(1)7Cf</i>			
<i>l(1)7Cg</i>			
<i>l(1)7Ch</i>	7C8		

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)7Ca¹</i>	EMS	Lefevre	<i>l(1)VA175</i>	2, 3	L/VME
<i>l(1)7Cb¹</i>	EMS	Lefevre	<i>l(1)VA276</i>	2, 3	L3/L
<i>l(1)7Cc¹</i>	X ray	Lefevre	<i>l(1)HC187</i>	1	
<i>l(1)7Cc²</i>	X ray	Lefevre	<i>l(1)JC107</i>	1	<i>ln(1)6A1-2;7C6-7</i>
<i>l(1)7Cc³</i>	EMS	Lefevre	<i>l(1)EC271</i>	2	
<i>l(1)7Cc⁴</i>	EMS	Lefevre	<i>l(1)VA354</i>	2	
<i>l(1)7Cd¹</i>	X ray	Lefevre	<i>l(1)C85</i>	1	<i>ln(1)4C13-14;7C6-7</i>
<i>l(1)7Ce¹</i>	X ray	Lefevre	<i>l(1)GE219</i>	1	<i>ln(1)7C6;18A</i>
<i>l(1)7Cf¹</i>	EMS	Lefevre	<i>l(1)EA24</i>	2, 3	L/MER
<i>l(1)7Cg¹</i>	X ray	Lefevre	<i>l(1)GA41</i>	1	<i>T(1;3)7C;95A;98E-F; E/L</i>
<i>l(1)7Ch¹</i>	EMS	Lefevre	<i>l(1)DF948</i>	2, 3	LP/VME

^α 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 3 = Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14.

l(1)7D

Thirteen lethally mutable loci recognized by Lefevre; order poorly understood. *l(1)adl* also in 7D; maybe allelic to mutants at one of the other loci.

locus	genetic location	cytological location	synonym		
			included in	excluded from	
<i>l(1)7Da</i>	7D		<i>Df(1)C128</i>	<i>Dp(1;3)sn13a1</i>	<i>fs(1)h</i>
<i>l(1)7Db</i>	1-23	7D1-6	<i>Df(1)C128</i>		<i>mys; l(1)EM28</i>
<i>l(1)7Dc</i>					
<i>l(1)7Dd</i>					
<i>l(1)7De</i>					
<i>l(1)7Df</i>				<i>Df(1)RA2</i>	
<i>l(1)7Dg</i>			<i>Df(1)RA2</i>	<i>Df(1)GE202</i>	<i>l(1)EM29</i>
<i>l(1)7Dh</i>		7D1			
<i>l(1)7Di</i>					
<i>l(1)7Dj</i>		7D12	<i>Df(1)GE202</i>	<i>Df(1)HA11</i>	
<i>l(1)7Dk</i>			<i>Df(1)HA11</i>		
<i>l(1)7Dl</i>					
<i>l(1)7Dm</i>		7D22			
<i>l(1)7Dn</i>	1-21.3	7D11-22	<i>Df(1)HA11</i>		<i>l(1)adl1</i>

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)7Dd¹</i>	X ray	Lefevre	<i>l(1)HC120</i>	1	
<i>l(1)7Dd²</i>	X ray	Lefevre	<i>l(1)HC261</i>	1	
<i>l(1)7Dd³</i>	X ray	Lefevre	<i>l(1)RC65</i>	1	
<i>l(1)7Dd⁴</i>	EMS	Lefevre	<i>l(1)DC704</i>	2	L/L
<i>l(1)7Dd⁵</i>	EMS	Lefevre	<i>l(1)DF914</i>	2	
<i>l(1)7Dd⁶</i>	EMS	Lefevre	<i>l(1)VA107</i>	2	

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)7De¹</i>	X ray	Lefevre	<i>l(1)HF375</i>	1	
<i>l(1)7De²</i>	EMS	Lefevre	<i>l(1)DC768</i>	2	
<i>l(1)7De³</i>	EMS	Lefevre	<i>l(1)EA30</i>	2	
<i>l(1)7Df¹</i>	X ray	Lefevre	<i>l(1)KC29</i>	1	
<i>l(1)7Df²</i>	X ray	Lefevre	<i>l(1)KC30</i>	1	
<i>l(1)7Df³</i>	EMS	Lefevre	<i>l(1)VA313</i>	2	
<i>l(1)7Df⁴</i>	EMS	Lefevre	<i>l(1)VE607</i>	2	L3/L
<i>l(1)7Df⁵</i>	EMS	Lefevre	<i>l(1)VE753</i>	2	
<i>l(1)7Dg¹</i>	EMS	Lefevre	<i>l(1)VA334</i>	2	E-L/L
<i>l(1)7Dh¹</i>	EMS	Lefevre	<i>l(1)EF430</i>	2	
<i>l(1)7Dh²</i>	EMS	Lefevre	<i>l(1)VA113</i>	2, 3	E-L/NME
<i>l(1)7Di¹</i>	X ray	Lefevre	<i>l(1)JA67</i>	1	
<i>l(1)7Di²</i>	EMS	Lefevre	<i>l(1)DA602</i>	2	
<i>l(1)7Di³</i>	EMS	Lefevre	<i>l(1)DA679</i>	2	
<i>l(1)7Di⁴</i>	EMS	Lefevre	<i>l(1)DC710</i>	2	
<i>l(1)7Di⁵</i>	EMS	Lefevre	<i>l(1)DF956</i>	2	
<i>l(1)7Di⁶</i>	EMS	Lefevre	<i>l(1)EF421</i>	2	E-L/NME
<i>l(1)7Di⁷</i>	EMS	Lefevre	<i>l(1)VA174</i>	2	
<i>l(1)7Dj¹</i>	X ray	Lefevre	<i>l(1)C113</i>	1	<i>T(1;2)7D18-19;41</i>
<i>l(1)7Dj²</i>	EMS	Lefevre	<i>l(1)DA563</i>	2	
<i>l(1)7Dj³</i>	EMS	Lefevre	<i>l(1)DA572</i>	2, 3	E/NME
<i>l(1)7Dj⁴</i>	EMS	Lefevre	<i>l(1)VA302</i>	2	
<i>l(1)7Dk¹</i>	X ray	Lefevre	<i>l(1)HC241</i>	1	
<i>l(1)7Dk²</i>	X ray	Lefevre	<i>l(1)RA63</i>	1	
<i>l(1)7Dk³</i>	EMS	Lefevre	<i>l(1)DA511</i>	2	
<i>l(1)7Dk⁴</i>	EMS	Lefevre	<i>l(1)DA512</i>	2	
<i>l(1)7Dk⁵</i>	EMS	Lefevre	<i>l(1)DC720</i>	2	
<i>l(1)7Dk⁶</i>	EMS	Lefevre	<i>l(1)EC204</i>	2	
<i>l(1)7Dk⁷</i>	EMS	Lefevre	<i>l(1)EF494</i>	2	
<i>l(1)7Dk⁸</i>	EMS	Lefevre	<i>l(1)VA86</i>	2, 3	E/L
<i>l(1)7Dk⁹</i>	EMS	Lefevre	<i>l(1)VA184</i>	2	
<i>l(1)7Di¹</i>	EMS	Lefevre	<i>l(1)VA40</i>	2, 3	L3/AO
<i>l(1)7Di²</i>	EMS	Lefevre	<i>l(1)VE662</i>	2	
<i>l(1)7Dm¹</i>	X ray	Lefevre	<i>l(1)A28</i>	1	<i>T(1;4)1F3;7D12;102</i>
<i>l(1)7Dm²</i>	X ray	Lefevre	<i>l(1)GA50</i>	1	<i>Trp(1;2)3C1;7D22;26A; complex</i>
<i>l(1)7Dm³</i>	X ray	Lefevre	<i>l(1)HA8</i>	1	<i>T(1;2)7E;57A</i>
<i>l(1)7Dm⁴</i>	X ray	Lefevre	<i>l(1)HA25</i>	1	
<i>l(1)7Dm⁵</i>	X ray	Lefevre	<i>l(1)HC110</i>	1	
<i>l(1)7Dm⁶</i>	X ray	Lefevre	<i>l(1)JC27</i>	1	<i>ln(1)7D18-21;20</i>
<i>l(1)7Dm⁷</i>	X ray	Lefevre	<i>l(1)RA5</i>	1	<i>ln(1)7D14-15;11E13</i>
<i>l(1)7Dm⁸</i>	EMS	Lefevre	<i>l(1)DA583</i>	2	
<i>l(1)7Dm⁹</i>	EMS	Lefevre	<i>l(1)DC770</i>	2	
<i>l(1)7Dm¹⁰</i>	EMS	Lefevre	<i>l(1)VA114</i>	2	
<i>l(1)7Dm¹¹</i>	EMS	Lefevre	<i>l(1)VA154</i>	2	
<i>l(1)7Dm¹²</i>	EMS	Lefevre	<i>l(1)VA162</i>	2	
<i>l(1)7Dm¹³</i>	EMS	Lefevre	<i>l(1)VA221</i>	2	
<i>l(1)7Dm¹⁴</i>	EMS	Lefevre	<i>l(1)VE616</i>	2	

^α 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 3 = Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14.

l(1)7e: see *l(1)IAa*

l(1)7E

Five lethally mutable loci including *std*.

locus	origin	discoverer	cytological		synonym
			location	included in	
<i>l(1)7Ea</i>				<i>Df(1)VE624</i>	<i>Df(1)GE202</i>
<i>l(1)7Eb</i>	7E4			<i>Df(1)VE624</i>	<i>Df(1)KA14</i>
<i>l(1)7Ec</i>	7E6			<i>Df(1)VE624</i>	<i>Df(1)KA14</i>
<i>l(1)7Ed</i>				<i>Df(1)VE624</i>	<i>Df(1)KA14</i>
<i>l(1)7Ee</i>				<i>Df(1)VE624</i>	<i>Df(1)KA14</i>
<i>l(1)7Ef</i>	7D10-F2			<i>Df(1)VE624</i>	<i>Df(1)KA14</i>

std

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)7Ea</i>¹	X ray	Lefevre	<i>l(1)G262</i>	1	
<i>l(1)7Ea</i>²	X ray	Lefevre	<i>l(1)HF382</i>	1	<i>ln(1)7E-F1;20</i>
<i>l(1)7Ea</i>³	EMS	Lefevre	<i>l(1)DC771</i>	2	
<i>l(1)7Ea</i>⁴	EMS	Lefevre	<i>l(1)EC240</i>	2	
<i>l(1)7Ea</i>⁵	EMS	Lefevre	<i>l(1)EF520</i>	2,3	EL/L
<i>l(1)7Eb</i>¹	X ray	Lefevre	<i>l(1)GF329</i>	1	
<i>l(1)7Eb</i>²	X ray	Lefevre	<i>l(1)HA57</i>	1	
<i>l(1)7Eb</i>³	X ray	Lefevre	<i>l(1)JA4</i>	1	
<i>l(1)7Eb</i>⁴	X ray	Lefevre	<i>l(1)KC40</i>	1	
<i>l(1)7Eb</i>⁵	EMS	Lefevre	<i>l(1)VA293</i>	2,3	L3-P/L
<i>l(1)7Eb</i>⁶	EMS	Lefevre	<i>l(1)VE911</i>	2	
<i>l(1)7Eb</i>⁷	spont	Schalet	<i>l(1)I4-160</i>		
<i>l(1)7Ec</i>¹	X ray	Lefevre	<i>l(1)JA11</i>	1	<i>ln(1)7B;7E5</i> lethal at both breakpoints
<i>l(1)7Ed</i>¹	EMS	Lefevre	<i>l(1)DF980</i>	1,3	L/L
<i>l(1)7Ee</i>¹	X ray	Lefevre	<i>l(1)HF348</i>	1	
<i>l(1)7Ee</i>²	X ray	Lefevre	<i>l(1)KA4</i>	1	
<i>l(1)7Ee</i>³	X ray	Lefevre	<i>l(1)RF2</i>	1	
<i>l(1)7Ee</i>⁴	EMS	Lefevre	<i>l(1)EC222</i>	2	

^α 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 3 = Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14.

l(1)7F

Six lethally mutable loci including *pt*, *gg*, and *otd*.

locus	genetic location	cytological location	included in	synonym
<i>l(1)7Fa</i>	1-23.6	7F1	<i>Df(1)KA14</i>	<i>Nrg</i>
<i>l(1)7Fb</i>	1-23.1		<i>Df(1)KA14</i>	<i>pt</i>
<i>l(1)7Fc</i>		7F3-4	<i>Df(1)KA14</i>	
<i>l(1)7Fd</i>			<i>Df(1)KA14</i>	
<i>l(1)7Fe</i>	1-23.1	7F10	<i>Df(1)KA14</i>	<i>gg</i>
<i>l(1)7Ff</i>		7F1-8A5	<i>Df(1)KA14</i>	<i>otd</i>

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)7Fc</i>¹	X ray	Lefevre	<i>l(1)GE208</i>	1	<i>T(1;2)7F;41</i>
<i>l(1)7Fc</i>²	X ray	Lefevre	<i>l(1)HC230</i>	1	
<i>l(1)7Fc</i>³	X ray	Lefevre	<i>l(1)JC18</i>	1	
<i>l(1)7Fc</i>⁴	EMS	Lefevre	<i>l(1)EC242</i>	2	
<i>l(1)7Fc</i>⁵	EMS	Lefevre	<i>l(1)DA610</i>	2	
<i>l(1)7Fc</i>⁶	EMS	Lefevre	<i>l(1)VE727</i>	2	LP/VME
<i>l(1)7Fd</i>¹	X ray	Lefevre	<i>l(1)KA26</i>	1	
<i>l(1)7Fd</i>²	EMS	Lefevre	<i>l(1)VA67</i>	2	
<i>l(1)7Fd</i>³	EMS	Lefevre	<i>l(1)VA195</i>	2	L3-P/L
<i>l(1)7Fd</i>⁴	EMS	Lefevre	<i>l(1)VA205</i>	2	

^α 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95.

****l(1)8***

location: 1-21.3 (19.0 to 23.6).

discoverer: Sobels.

references: Gloor, 1962, Rev. Suisse Zool. 69: 409-63 (fig.).

phenotype: Larvae lethal in third instar, survive up to 10 days. Testes and lymph glands degenerate. Imaginal disks develop normally after transplantation. Protein metabolism disturbed; free amino acids and peptides abnormally high. RK2.

l(1)8A

Four lethally mutable loci including *oc*. *l(1)8Aab* of Schalet fails to complement both *l(1)8Aa* and *l(1)8Ab*.

locus	genetic location	cytological location	included in	synonym
<i>l(1)8Aa</i>		8A1	<i>Df(1)KA14</i>	
<i>l(1)8Ab</i>		8A2	<i>Df(1)KA14</i>	
<i>l(1)8Ac</i>	1-23.1	8A1-2	<i>Df(1)KA14</i>	<i>oc</i>
<i>l(1)8Ad</i>		8A4	<i>Df(1)KA14</i>	

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)8Aa</i>¹	X ray	Lefevre	<i>l(1)GA19</i>	1	
<i>l(1)8Aa</i>²	X ray	Lefevre	<i>l(1)GA28</i>	1	
<i>l(1)8Aa</i>³	X ray	Lefevre	<i>l(1)GF319</i>	1	<i>Tp(1;2)8A1;9A3;10A;2L</i>
<i>l(1)8Aa</i>⁴	X ray	Lefevre	<i>l(1)HC179</i>	1	
<i>l(1)8Aa</i>⁵	X ray	Lefevre	<i>l(1)JC68</i>	1	<i>T(1;2)8A4-5;41</i>
<i>l(1)8Aa</i>⁶	EMS	Lefevre	<i>l(1)DF901</i>	2	
<i>l(1)8Aa</i>⁷	EMS	Lefevre	<i>l(1)VE661</i>	2,3	L2-3/AO
<i>l(1)8Aa</i>⁸	EMS	Lefevre	<i>l(1)VE709</i>	2	
<i>l(1)8Aab</i>¹	spont	Schalet	<i>l(1)7-07</i>		
<i>l(1)8Ab</i>¹	X ray	Lefevre	<i>l(1)HF383</i>	1	
<i>l(1)8Ab</i>²	X ray	Lefevre	<i>l(1)JA18</i>	1,3	L/L
<i>l(1)8Ab</i>³	X ray	Lefevre	<i>l(1)JA62</i>	1	<i>T(1;2)8A3;41E</i>
<i>l(1)8Ad</i>¹	X ray	Lefevre	<i>l(1)A86</i>	1	<i>T(1;2)8A3-5;34B</i>

^α 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 3 = Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14.

DEFICIENCY MAP OF REGION 6D TO 8A

side	breakpoint	variant
		<i>l(1)6Da</i>
		<i>l(1)6Db</i>
		<i>l(1)6Dc</i>
		<i>l(1)6Dd</i>
		<i>l(1)6De</i>
		<i>l(1)6Df</i>
		<i>l(1)6Dg</i>
		<i>l(1)6Dh</i>
		<i>l(1)6Di</i>
left	6D1	<i>Dp(1;3)ct⁺</i>
	6D8	<i>l(1)6Dg</i>
left	6D8-E1	<i>Df(1)ct-j6</i>
		<i>l(1)6Ea</i>
		<i>y⁺Yct⁺</i>
left	6E2	<i>Df(1)Sxl-bt</i>
		<i>l(1)6Eb</i>
		<i>ogre</i>
		<i>l(1)6Ed</i>
left	6E4-5	<i>Df(1)HA32</i>
	6E5	<i>cm</i>
	6E6	<i>l(1)6Ee</i>
		<i>l(1)6Fa</i>
		<i>Df(1)cm?</i>
left	6F5	<i>Df(1)Sxl-ra</i>
	6F5	<i>Sxl</i>
		<i>l(1)6Fc</i>
left	7A2-3	<i>Df(1)ct-j4</i>
		<i>l(1)7Aa</i>
left	7A4-5	<i>Df(1)RF19</i>
right	7A6	<i>Df(1)Sxl-bt</i>
right	7A6	<i>Df(1)HA32</i>
		<i>l(1)7Ab</i>
		<i>l(1)7Ac</i>
left		<i>Dp(1;2)sn⁺72d</i>
		<i>l(1)7Ad</i>
right	7B1-2	<i>Df(1)Sxl-ra</i>
		<i>kt</i>
right	7B1-2	<i>Df(1)RF19</i>

side	breakpoint	variant
left	7B2-4	<i>Df(1)ct4b1</i>
right	7B7-8	<i>Df(1)ct-j6</i>
right		ct <i>Df(1)GA34</i> I(1)7Bc I(1)7Bd I(1)7Be
right	7B8C1	<i>Df(1)ct-j4</i>
right	7C3	<i>Df(1)ct4b1</i> I(1)7Ca I(1)7Cb <i>ct⁺Yy⁺</i>
left		<i>Dp(1;?)FN107</i> I(1)7Cc
right	7D1-2	<i>Dp(1;3)sn13a1</i> I(1)7Cd I(1)7Ce I(1)7Cf I(1)7Cg I(1)7Ch
left	7D1	<i>Df(1)C128</i> sn I(1)7Da fs(1)M122 fs(1)h mys
right	7D5-6	<i>Df(1)C128</i> I(1)7Dc I(1)7Dd I(1)7De I(1)7Df
left	7D10	<i>Df(1)RA2</i> I(1)7Dg
left		<i>Df(1)GE202</i> I(1)7Dh I(1)7Di I(1)7Dj
left		<i>Df(1)HA11</i> I(1)7Dk
right		<i>Df(1)GE202</i> I(1)7Dl I(1)7Dm mys I(1)7Ea I(1)7Eb I(1)7Ec I(1)7Ed I(1)7Ee I(1)7Ef I(1)7Eg
left	7F1-2	<i>Df(1)KA14</i> Nrg pt I(1)7Fd I(1)7Fe gg otd I(1)8Aa I(1)8Ab oc I(1)8Ad
right	8C6	<i>Df(1)KA14</i>

I(1)9A, I(1)9E

Four complementation groups between 9E1 and 9E8 identified by Janca, Woloshyn, and Nash, (1986, Genetics 112: 43-64); three identified by Lefevre (Lefevre and Watkins, 1986, Genetics 113: 869-95); complementation studies by Janca *et al.* show that only *ras* and *I(1)9Ed*

identified by both studies.

locus	genetic		cytological		synonym
	location	location	included in	excluded from	
<i>I(1)9Aa</i>	9A2-B2		<i>Df(1)sbr9</i>	<i>Df(1)sbr8</i>	mus109
I(1)9Ea	9E1-2				<i>I(1)EM32</i>
<i>I(1)9Eb</i>	1-32.35	9E3-4	<i>Df(1)v-P14</i>	<i>Df(1)v64f29</i>	ras
I(1)9Ec	9E3-4		<i>Df(1)203</i>	<i>Df(1)v64f</i>	
I(1)9Ed	9E7-8		<i>Df(1)v64f</i>		<i>I(1)EM31</i>
			<i>Df(1)217</i>		
I(1)9Ee	9E7-8		<i>Df(1)v64f</i>	<i>Df(1)217</i>	
allele	origin	discoverer	synonym	ref ^α	comments
I(1)9Ea ¹			<i>I(1)EM32</i>	2	
			<i>I(1)DA665</i>		
I(1)9Ec ¹	EMS		<i>I(1)B13</i>	1	
I(1)9Ec ²	EMS		<i>I(1)E20</i>	1	
I(1)9Ec ³	EMS		<i>I(1)G4</i>	1	
I(1)9Ed ¹	EMS		<i>I(1)A17</i>	1	
I(1)9Ed ²	EMS		<i>I(1)G16</i>	1	haplospecific ^β
I(1)9Ed ³	EMS		<i>I(1)H22</i>	1	
I(1)9Ed ⁴	EMS		<i>I(1)M27</i>	1	haplospecific
I(1)9Ed ⁵	EMS		<i>I(1)Q10</i>	1	haplospecific
I(1)9Ed ⁶	EMS	Lefevre	<i>I(1)EM31</i>	2	L1-2/AO
			<i>I(1)DC701</i>		
I(1)9Ed ⁷	EMS	Lefevre	<i>I(1)DF935</i>	2	
I(1)9Ed ⁸	EMS	Lefevre	<i>I(1)DF981</i>	2	
I(1)9Ee ¹	EMS		<i>I(1)Q21</i>	1	

^α 1 = Janca, Woloshyn, and Nash, 1986, Genetics 112: 43-64; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95.

^β Haplospecific alleles survive as males or homozygous females, but die in heterozygous combination with a deficiency for the locus.

I(1)9F

Alleles recovered and characterized by three groups: allelism between mutants from the different collections partially carried out by Zhimulev, Pokholkova, Bgatov, Umbetova, Solovjeva, Khudyakov, and Belyaeva (1987, Biol. Zentralbl. 106: 699-720). As many as seven lethally mutable loci, including *sesB*, *fliK*, and *sbr*, identified. Deficiency map based largely on the work of Zhimulev *et al.* are labeled *I(1)BP* after Belyaeva and Pokholkova; those identified by Geer *et al.* are labeled *I(1)G*; the remainder were identified by Lefevre. Janca, Woloshyn, and Nash isolated three mutants in different complementation groups in 9E7-9F11 and four in two complementation groups in 9F10-10A2, but no complementation tests carried out with other lethal mutations in 9F and therefore not included in tabulations.

locus	genetic		cytological		synonym
	location	location	included in	excluded from	
<i>I(1)9Fa</i>	1-32.53	9F1	<i>Df(1)ras-P14</i>		sesB
			<i>Df(1)v64f</i>		
<i>I(1)9Fc</i>		9F4-8		<i>Df(1)ras-P14</i>	fliK
				<i>Df(1)v-LA</i>	
<i>I(1)9Fd</i>	1-32.6	9F5-11	<i>Df(1)v-L4</i>	<i>Df(1)v-L3</i>	sbr
I(1)9Fe	1-32.78	9F9-12	<i>Df(1)v-L3</i>	<i>Df(1)M6</i>	<i>I(1)BP1</i>
I(1)9Ff		9F9-12	<i>Df(1)v-L3</i>	<i>Dp(1;2)v⁺65</i>	<i>I(1)G3</i>
I(1)9Fg	1-32.96	9F13-10A1	<i>Df(1)v-L2</i>	<i>Df(1)v-L1</i>	<i>I(1)BP3</i>
I(1)9Fh	1-32.96	9F13-10A1	<i>Df(1)v-L2</i>	<i>Df(1)v-L1</i>	<i>I(1)EF433</i>

I(1)9Fe

alleles: All alleles lethal except *I(1)9Fe*¹², which is viable and of normal phenotype in both homozygous and heterozygous condition. However, flies heterozygous for *I(1)9Fe*¹² and any lethal allele except *I(1)9Fe*¹ or for a

deficiency for *l(1)9Fe* show irregularly missing bristles, especially humerals; *l(1)9Fe*¹ complements *l(1)9Fe*¹².

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)9Fe</i> ¹	ICR170	Carlson	<i>l(1)Q54</i>	2	
<i>l(1)9Fe</i> ²	X ray	Lefevre	<i>l(1)A62</i>	2	
<i>l(1)9Fe</i> ³	X ray	Lefevre	<i>l(1)A89</i>	2	
<i>l(1)9Fe</i> ⁴	X ray	Lefevre	<i>l(1)HA13</i>	2	euchab
<i>l(1)9Fe</i> ⁵	X ray	Lefevre	<i>l(1)JE2</i>	2	hetab
<i>l(1)9Fe</i> ⁶	EMS	Lefevre	<i>l(1)EA86</i>	3	
<i>l(1)9Fe</i> ⁷	EMS	Lefevre	<i>l(1)VE881</i>	3	
<i>l(1)9Fe</i> ⁸	EMS	Belyaeva	<i>l(1)I91</i>	4, 5	
<i>l(1)9Fe</i> ⁹	EMS	Pokholkova	<i>l(1)I71</i>	4, 5	
<i>l(1)9Fe</i> ¹⁰	EMS	Pokholkova	<i>l(1)G101</i>	4, 5	
<i>l(1)9Fe</i> ¹¹	EMS	Pokholkova	<i>l(1)G98</i>	4, 5	
<i>l(1)9Fe</i> ¹²	EMS	Fomina	<i>bir336</i>	4, 5	
<i>l(1)9Fe</i> ^{13Y}	EMS	Bgatov	<i>l(1)dpS42</i>	4, 5	on v ⁺ Yy ⁺
<i>l(1)9Fe</i> ¹⁴	EMS		<i>l(1)v126</i>	1	pupal
<i>l(1)9Fe</i> ^{15Y}	EMS		<i>l(1)v348</i>	1	on v ⁺ B ^{S-} Yy ⁺
<i>l(1)9F1</i> ¹	EMS		<i>l(1)v350</i>	1	larval-pupal
<i>l(1)9Fg</i> ¹	EMS	Pokholkova	<i>l(1)I63</i>	4, 5	
<i>l(1)9Fg</i> ²	EMS	Belyaeva	<i>l(1)I67</i>	4, 5	
<i>l(1)9Fg</i> ³	EMS	Pokholkova	<i>l(1)I83</i>	4, 5	
<i>l(1)9Fg</i> ^{4Y}	EMS	Bgatov	<i>l(1)dpS22</i>	4, 5	on v ⁺ Yy ⁺
<i>l(1)9Fg</i> ⁵		Pokholkova	<i>l(1)F6</i>	6	
<i>l(1)9Fg</i> ⁶	EMS		<i>l(2)v5</i>	1	
<i>l(1)9Fg</i> ⁷	EMS		<i>l(2)v40</i>	1	
<i>l(1)9Fg</i> ⁸	EMS		<i>l(2)v103</i>	1	
<i>l(1)9Fg</i> ⁹	EMS		<i>l(2)v172</i>	1	
<i>l(1)9Fg</i> ¹⁰	EMS		<i>l(2)v205</i>	1	
<i>l(1)9Fg</i> ¹¹	EMS		<i>l(2)v207</i>	1	
<i>l(1)9Fg</i> ^{12Y}	EMS		<i>l(2)v363</i>	1	on v ⁺ B ^{S-} Yy ⁺
<i>l(1)9Fh</i>	EMS	Lefevre	<i>l(1)EF433</i>	3	possible allele of <i>l(1)9Ff</i>

^α 1 = Geer, Lischwe, and Murphy, 1983, J. Exp. Zool. 225: 107-18; 2 = Lefevre, 1981, Genetics 99: 461-80; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 4 = Zhimulev, Belyaeva, Pokholkova, Kotchneva, Fomina, Bgatov, Khudiyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1981, DIS 56: 192-96; 5 = Zhimulev, Pokholkova, Bgatov, Semeshin, and Belyaeva, 1981, Chromosoma 82: 25-40; 6 = Zhimulev and Pokholkova, 1981, 8th European Drosophila Research Conference.

DEFICIENCY MAP OF REGION 9

side	breakpoint	variant
left	9A	<i>Df(1)ras59</i>
left	9A	<i>Df(1)ras217</i>
left	9A2	<i>Dp(1;2)v⁺75d</i>
left	9A2-4	<i>Df(1)skr10</i>
left	9A2-4	<i>Df(1)skr9</i>
	9A2-5	mus109
left	9B1-2	<i>Df(1)skr8</i>
left	9B9-10	<i>Df(1)skr1</i>
left	9D1-2	<i>Df(1)ras-v17Cc8</i>
left	9D1-2	<i>Df(1)v-P5</i>
left	9D3	<i>Df(1)v-M1</i>
left	9D3	<i>Df(1)v-M7</i>
left	9E1	<i>Dp(1;2)v⁺63i</i>
left	9E1-2	<i>Df(1)ras203</i>
left	9E1-2	<i>Df(1)ras-P14</i>
	9E2-8	ras
	9E1-8	l(1)19Ec
left	9E3-4	<i>Dp(1;3)v⁺74c</i>
left	9E7-8	<i>Df(1)v64f</i>
	9E7-8	l(1)9Ed
	9E7-8	l(1)9Ee
		l(1)9Ea
		sesB
right	9F3-4	<i>Df(1)ras-P14</i>
right	9E7-8	<i>Df(1)ras217</i>
left	9F4	v ⁺ B ^{S-} Yy ⁺
left	9F4	v ⁺ Yy ⁺
		flIK
right		<i>Df(1)HC133</i>

side	breakpoint	variant
left	9F5-6	<i>Df(1)v-L4</i>
	9F5-11	sbr
left	9F10-12	<i>Df(1)v-L3</i>
		flIG
		ts(1)BP
		l(1)9Ff
		l(1)9Ff
	9F9-12	
	9F9-12	
left	9F11-12	<i>Df(1)v-M6</i>
right	9F12-13	<i>Df(1)ras59</i>
left	9F12-13	<i>Dp(1;2)v⁺65b</i>
left	9F12-13	<i>Df(1)v65b</i>
left	9F13	<i>Df(1)v-L2</i>
right	9F13	<i>Df(1)ras203</i>
	9F13-10A1	l(1)9Fh
	9F13-10A1	l(1)9Fg
left	9F13	<i>Df(1)v-L1</i>
left	9F13-10A1	<i>Df(1)v-M5</i>
left	10A1	<i>Df(1)P22</i>
right	9F13-10A1	<i>Df(1)skr1</i>
right	9F13-10A1	<i>Df(1)skr10</i>

l(1)10A

Four independent mutation searches have been carried out in this region. Most mutants have been tested against the first group of three loci identified by Lefevre (1971, Genetics 67: 497-513), but there are indications of as many as five additional loci as inferred from the results of within-search complementation tests. In general mutants recovered in different searches have not been tested against one another. *rtv* has not been tested for complementation by other mutants in region 10A. Presumptive loci originally identified by Zhimulev *et al.* are labeled *l(1)BP* after Belyaeva and Pokholkova; those identified by Geer *et al.* are labeled *l(1)G*; the remainder were identified by Lefevre.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(1)10Aa</i>	1-33.05	10A1	<i>Df(1)v-L2</i> <i>Df(1)v-L1</i>		csk , <i>l(1)BP4</i> , <i>l(1)G6</i>
<i>l(1)10Ac</i>	1-33.51	10A3-5	<i>Df(1)v-L1</i>	<i>Df(1)v-L2</i>	<i>l(1)L12</i> , <i>l(1)BP5</i> , <i>l(1)G8</i>
<i>l(1)10Ad</i>	1-33.55	10A6-7	<i>Df(1)RA37</i> <i>Df(1)v-L3</i>		<i>l(1)BP8</i>
<i>l(1)10Ae</i>	1-33.56	10A6-7	<i>Df(1)RA37</i> <i>Df(1)v-L3</i>		<i>l(1)L8</i> , <i>l(1)BP7</i> , <i>l(1)G10</i>
<i>l(1)10Af</i>	1-33.56	10A8		<i>Df(1)v-L3</i> <i>Df(1)K7</i>	<i>l(1)G11</i> , <i>l(1)EM16</i>
<i>l(1)10Ag</i>	1-33.68	10A9-12	<i>Df(1)KA7</i>	<i>Df(1)GA112</i>	rtv , <i>l(1)L1</i> , <i>l(1)G13</i>
<i>l(1)10Ah</i>	1-{33}	10A9-12	<i>Df(1)KA7</i>	<i>Df(1)GA112</i>	<i>l(1)G12</i>
<i>l(1)10Ai</i>	1-{33}	10A9-12	<i>Df(1)KA7</i>	<i>Df(1)GA112</i>	
<i>l(1)10Aj</i>	1-{33}	10A11-?	<i>Df(1)GA112</i>		

l(1)10Ac

alleles: Complementation relations among complementing alleles complex and vary according to temperature of rearing, some combinations being heat sensitive and others cold sensitive. Allelism to *l(1)10Ac*¹ established for the Russian alleles by Zhimulev *et al.* and for the G8 alleles by Geer *et al.*

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)10Ac</i> ¹	X ray	Lefevre	<i>l(1)L12</i>	3, 6	larval lethal complementing allele
<i>l(1)10Ac</i> ²	EMS	Belyaeva	<i>l(1)E54</i>	8, 9, 11	
<i>l(1)10Ac</i> ³	EMS	Belyaeva	<i>l(1)E112</i>	8, 9, 11	
<i>l(1)10Ac</i> ⁴	EMS	Belyaeva	<i>l(1)E114</i>	8, 9, 11	
<i>l(1)10Ac</i> ⁵	EMS	Belyaeva	<i>l(1)E120</i>	8, 9, 11	
<i>l(1)10Ac</i> ⁶	EMS	Belyaeva	<i>l(1)I64</i>	8, 9, 11	complementing allele
<i>l(1)10Ac</i> ⁷	EMS	Belyaeva	<i>l(1)I69</i>	8, 9, 11	

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)10Ac⁸</i>	EMS	Belyaeva	<i>l(1)187</i>	8, 9, 11	
<i>l(1)10Ac⁹</i>	EMS	Belyaeva	<i>l(1)193</i>	8, 9, 11	
<i>l(1)10Ac¹⁰</i>		Pokholkova	<i>l(1)F230</i>	10, 11	haplospecific heat-sensitive allele
<i>l(1)10Ac¹¹</i>	EMS		<i>l(1)F409</i>	11	
<i>l(1)10Ac¹²</i>	EMS		<i>l(1)F437</i>	11	
<i>l(1)10Ac¹³</i>	EMS		<i>l(1)F439</i>	11	haplospecific heat-sensitive allele
<i>l(1)10Ac¹⁴</i>	EMS	Pokholkova	<i>l(1)G62</i>	2, 8, 9, 11	
<i>l(1)10Ac¹⁵</i>	EMS	Pokholkova	<i>l(1)G67</i>	8, 9, 11	
<i>l(1)10Ac¹⁶</i>	EMS	Pokholkova	<i>l(1)G76</i>	8, 9, 11	complementing allele
<i>l(1)10Ac¹⁷</i>	EMS	Pokholkova	<i>l(1)G93</i>	8, 9, 11	
<i>l(1)10Ac¹⁸</i>	EMS	Pokholkova	<i>l(1)G95</i>	8, 9, 11	
<i>l(1)10Ac¹⁹</i>	EMS	Pokholkova	<i>l(1)G96</i>	8, 9, 11	complementing allele
<i>l(1)10Ac²⁰</i>	EMS	Pokholkova	<i>l(1)G105</i>	8, 9, 11	
<i>l(1)10Ac²¹</i>	EMS	Pokholkova	<i>l(1)G139</i>	8, 9, 11	
<i>l(1)10Ac²²</i>	EMS	Khudyakov	<i>l(1)J21</i>	8, 9, 11	complementing allele
<i>l(1)10Ac^{23Y}</i>	EMS	Fomina	<i>l(1)dpO25</i>	8, 9, 11	on v ⁺ Yy ⁺
<i>l(1)10Ac^{24Y}</i>	EMS	Biyasheva	<i>l(1)dpZ4</i>	8, 9, 11	on v ⁺ Yy ⁺
<i>l(1)10Ac^{25Y}</i>	EMS	Bgatov	<i>l(1)dpS145</i>	8, 9, 11	on v ⁺ Yy ⁺
<i>l(1)10Ac²⁶</i>	X ray	Lefevre	<i>l(1)A114</i>	4	
<i>l(1)10Ac²⁷</i>	X ray	Lefevre	<i>l(1)C202</i>	4	<i>In(1)7A2;10A3-4</i>
<i>l(1)10Ac²⁸</i>	X ray	Lefevre	<i>l(1)GF319</i>	4	<i>T(1;2)7F-8A;9A3;10A;32B^β</i>
<i>l(1)10Ac²⁹</i>	X ray	Lefevre	<i>l(1)RA75</i>	4	<i>T(1;2;3)10A;41;100^γ</i>
<i>l(1)10Ac³⁰</i>	EMS	Lefevre	<i>l(1)DA514</i>	5	PP/L
<i>l(1)10Ac³¹</i>	EMS	Lefevre	<i>l(1)VA160</i>	5	
<i>l(1)10Ac³²</i>	EMS	Lefevre	<i>l(1)VA244</i>	5	
<i>l(1)10Ac³³</i>	EMS	Lefevre	<i>l(1)VA275</i>	5	
<i>l(1)10Ac³⁴</i>	EMS	Lefevre	<i>l(1)VA312</i>	5	
<i>l(1)10Ac³⁵</i>	EMS	Lefevre	<i>l(1)VE666</i>	5	
<i>l(1)10Ac³⁶</i>	EMS	Lefevre	<i>l(1)VE727</i>	5	
<i>l(1)10Ac³⁷</i>	EMS	Lefevre	<i>l(1)VE772</i>	5	L2/NME
<i>l(1)10Ac³⁸</i>	EMS	Lefevre	<i>l(1)VE815</i>	5	
<i>l(1)10Ac³⁹</i>	EMS		<i>l(1)v201</i>	1	
<i>l(1)10Ac⁴⁰</i>	EMS		<i>l(1)v202</i>	1	
<i>l(1)10Ac^{41Y}</i>	EMS		<i>l(1)v361</i>	1	on v ⁺ B ^{S-} Yy ⁺
<i>l(1)10Ac^{42Y}</i>	EMS		<i>l(1)v372</i>	1	on v ⁺ B ^{S-} Yy ⁺
<i>l(1)10Ac⁴³</i>	EMS	Mayoh	<i>l(1)HM21</i>	7	cold-sensitive allele; females surviving 17° sterile; 25° fertile

^α 1 = Geer, Lischwe, and Murphy, 1983, J. Exp. Zool. 225: 107-18; 2 = Kramers, Schalet, Paradi, and Huiser-Hoosteyling, 1983, Mutat. Res. 107: 187-201; 3 = Lefevre, 1971, Genetics 67: 497-513; 4 = Lefevre, 1981, Genetics 99: 461-80; 5 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 6 = Lefevre and Wiedenheft, 1974, DIS 51: 83; 7 = Mayoh and Suzuki, 1973, Can. J. Genet. Cytol. 15: 237-54; 8 = Zhimulev, Belyaeva, Pokholkova, Kotchneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1981, DIS 56: 192-96; 9 = Zhimulev, Pokholkova, Bgatov, Semeshin, and Belyaeva, 1981, Chromosoma 82: 25-40; 10 = Zhimulev and Pokholkova, 1981, 8th European Drosophila Research Conference; 11 = Zhimulev, Pokholkova, Bgatov, Umbetova, Solovjeva, Khudyakov, and Belyaeva, 1987, Biol. Zentralbl. 106: 699-720.

^β Tentative new order: 1A—7F|9A3—10A|8A—9A3|32B—60F;21A—32B|10A—20.

^γ Tentative new order: 1A—10A|41—21;60—41|100—61;20—10A|100.

l(1)10Ad

One of two loci in the region of overlap between *Df(1)RA37* and *Df(1)v-13*. Allelism of *l(1)10Ad¹* with Russian alleles and with HMS-induced alleles established by Zhimulev *et al.* *l(1)10Ad²³* and *l(1)10Ad²⁴* designated alleles on basis of deficiency mapping and complementation of *l(1)10Ae¹*. *l(1)10Ae²⁰* fails to complement the Russian alleles, but according to Geer *et al.* does complement *l(1)10Ad²¹* and *l(1)10Ad^{22Y}*, which are not mentioned in the Russian papers.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)10Ad¹</i>	ICR170	Carlson	<i>l(1)Q66</i>		

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)10Ad²</i>	EMS	Belyaeva	<i>l(1)E62</i>	4, 5, 6	
<i>l(1)10Ad³</i>	EMS	Belyaeva	<i>l(1)E72</i>	4, 5, 6	
<i>l(1)10Ad⁴</i>	EMS	Belyaeva	<i>l(1)I53</i>	4, 5, 6	
<i>l(1)10Ad⁵</i>	EMS	Belyaeva	<i>l(1)I73</i>	4, 5, 6	
<i>l(1)10Ad⁶</i>	EMS	Belyaeva	<i>l(1)I74</i>	4, 5, 6	
<i>l(1)10Ad⁷</i>	EMS	Belyaeva	<i>l(1)I75</i>	4, 5, 6	
<i>l(1)10Ad⁸</i>	EMS	Belyaeva	<i>l(1)I194</i>	4, 5, 6	
<i>l(1)10Ad⁹</i>		Pokholkova	<i>l(1)F59</i>	6	haplospecific cold-sensitive allele
<i>l(1)10Ad¹⁰</i>		Pokholkova	<i>l(1)F79</i>	6	
<i>l(1)10Ad¹¹</i>			<i>l(1)F99</i>	6	
<i>l(1)10Ad¹²</i>			<i>l(1)F431</i>	6	haplospecific cold-sensitive allele
<i>l(1)10Ad¹³</i>	EMS	Pokholkova	<i>l(1)G97</i>	4, 5, 6	
<i>l(1)10Ad^{14Y}</i>	EMS	Fomina	<i>l(1)dpO5</i>	4, 5, 6	on v ⁺ Yy ⁺
<i>l(1)10Ad^{15Y}</i>	EMS	Fomina	<i>l(1)dpO24</i>	4, 5, 6	on v ⁺ Yy ⁺
<i>l(1)10Ad^{16Y}</i>	EMS	Biyasheva	<i>l(1)dpZ3</i>	4, 5, 6	on v ⁺ Yy ⁺
<i>l(1)10Ad¹⁷</i>	HMS	Kramers	<i>l(1)HM4</i>	2, 4, 5, 6	
<i>l(1)10Ad¹⁸</i>	HMS	Kramers	<i>l(1)HM26</i>	2, 4, 5, 6	
<i>l(1)10Ad¹⁹</i>	HMS	Kramers	<i>l(1)HM445</i>	2, 4, 5, 6	
<i>l(1)10Ad²⁰</i>	EMS		<i>l(1)v145</i>	1	
<i>l(1)10Ad²¹</i>	EMS		<i>l(1)v200</i>	1	
<i>l(1)10Ad^{22Y}</i>	EMS		<i>l(1)v353</i>	1	on v ⁺ B ^{S-} Yy ⁺ larval lethal
<i>l(1)10Ad²³</i>	EMS	Lefevre	<i>l(1)VE704</i>	3	
<i>l(1)10Ad²⁴</i>	EMS	Lefevre	<i>l(1)VE774</i>	3	E-L2/MER

^α 1 = Geer, Lischwe, and Murphy, 1983, J. Exp. Zool. 225: 107-18; 2 = Kramers, Schalet, Paradi, and Huiser-Hoosteyling, 1983, Mutat. Res. 107: 187-201; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 4 = Zhimulev, Belyaeva, Pokholkova, Kotchneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1981, DIS 56: 192-96; 5 = Zhimulev, Pokholkova, Bgatov, Semeshin, and Belyaeva, 1981, Chromosoma 82: 25-40; 6 = Zhimulev, Pokholkova, Bgatov, Umbetova, Solovjeva, Khudyakov, and Belyaeva, 1987, Biol. Zentralbl. 106: 699-720.

l(1)10Ae

Allelism of *l(1)10Ae¹* with Russian alleles and *l(1)10Ae¹⁷* determined by Zhimulev *et al.*; allelism with *l(1)10Ae¹⁶* determined by Geer *et al.*

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)10Ae¹</i>	X ray	Lefevre	<i>l(1)L8</i>	3	
<i>l(1)10Ae²</i>	X ray	Lefevre	<i>l(1)HC166</i>	4	
<i>l(1)10Ae³</i>	X ray	Lefevre	<i>l(1)L57</i>	4	
<i>l(1)10Ae⁴</i>	EMS	Lefevre	<i>l(1)DC812</i>	5	L1-2/NME
<i>l(1)10Ae⁵</i>	EMS	Belyaeva	<i>l(1)E66</i>	6, 7, 8	
<i>l(1)10Ae⁶</i>	EMS	Belyaeva	<i>l(1)E67</i>	6, 7, 8	
<i>l(1)10Ae⁷</i>	EMS	Belyaeva	<i>l(1)E142</i>	6, 7, 8	
<i>l(1)10Ae⁸</i>	EMS	Belyaeva	<i>l(1)TE108</i>	6, 7, 8	
<i>l(1)10Ae⁹</i>	EMS	Belyaeva	<i>l(1)I70</i>	6, 7, 8	
<i>l(1)10Ae¹⁰</i>		Pokholkova	<i>l(1)F60</i>	8	
<i>l(1)10Ae¹¹</i>		Pokholkova	<i>l(1)F100</i>	8	
<i>l(1)10Ae¹²</i>			<i>l(1)F313</i>	8	
<i>l(1)10Ae¹³</i>			<i>l(1)F342</i>	8	
<i>l(1)10Ae¹⁴</i>	EMS	Pokholkova	<i>l(1)G52</i>	6, 7, 8	
<i>l(1)10Ae¹⁵</i>	EMS	Pokholkova	<i>l(1)G65f</i>	6, 7, 8	
<i>l(1)10Ae¹⁶</i>	EMS		<i>l(1)v153</i>	2, 8	larval lethal
<i>l(1)10Ae¹⁷</i>	MR		<i>l(1)D41</i>	1, 8	

^α 1 = Eeken, Sobels, Hyland, and Schalet, 1985, Mutat. Res. 150: 261-75; 2 = Geer, Lischwe, and Murphy, 1983, J. Exp. Zool. 225: 107-18; 3 = Lefevre, 1971, Genetics 67: 497-513; 4 = Lefevre, 1981, Genetics 99: 461-80; 5 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 6 = Zhimulev, Belyaeva, Pokholkova, Kotchneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1981, DIS 56: 192-96; 7 = Zhimulev, Pokholkova, Bgatov, Semeshin, and Belyaeva, 1981, Chromosoma 82: 25-40; 8 = Zhimulev, Pokholkova, Bgatov, Umbetova, Solovjeva, Khudyakov, and Belyaeva, 1987, Biol. Zentralbl.

THE GENOME OF *DROSOPHILA MELANOGASTER*

106: 699-720.

l(1)10Af

Allelism of *l(1)10Af*⁵ with other alleles not tested; allelism inferred from similarity of deficiency mapping positions at 10A8.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)10Af</i> ¹	EMS	Lefevre	<i>l(1)EF444</i>	2	L/VME
			<i>l(1)EM16</i>		
<i>l(1)10Af</i> ²	EMS	Lefevre	<i>l(1)EF501</i>	2	
<i>l(1)10Af</i> ³	EMS	Lefevre	<i>l(1)VE651</i>	2	
<i>l(1)10Af</i> ⁴	EMS	Lefevre	<i>l(1)VE937</i>	2	
<i>l(1)10Af</i> ⁵	EMS		<i>l(1)v147</i>	1	embryonic-larval lethal

^α 1 = Geer, Lischwe, and Murphy, 1983, J. Exp. Zool. 225: 107-18; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)10Ah</i> ¹	X ray	Lefevre	<i>l(1)JES7</i>	1	complex <i>T(1;2;3)</i> ^β
<i>l(1)10Ai</i> ¹	EMS	Lefevre	<i>l(1)VE821</i>	2	E/L
<i>l(1)10Aj</i> ¹	X ray	Lefevre	<i>l(1)JF6</i>	1	E/L; complex aberration ^γ

^α 1 = Lefevre, 1981, Genetics 99: 161-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95.

^β Tentative order: 1—5C|73F—77A|22A—60;20—14B|13B—14B|10A—13B|10A—5D|77A—61;21—22A|73F—100.

^γ Segment from 3C12 to 20A4 highly rearranged and inserted into 3L at 64B; additional breaks recorded in 9D-E, 10B7-12, 11A7, and 15B-C.

l(1)10B

Complementation and deficiency mapping of the lethals in 10B have been carried out by Sponaugle, who determined relations among mutant alleles produced by Lefevre, Geer *et al.*, and Voelker.

locus	genetic location		included in	excluded from	synonym
	location	location			
<i>l(1)10Ba</i>	1-[34]	10B4-9	<i>Df(1)GA112</i>	<i>Df(1)N71</i>	<i>l(1)G16</i>
<i>l(1)10Bb</i>	1-34.28	10B4-9	<i>Df(1)N71</i>	<i>Df(1)DA622</i>	<i>l(1)G14</i>
<i>l(1)10Bc</i>	1-34.39	10B4-9	<i>Df(1)N71</i>	<i>Df(1)DA622</i>	<i>l(1)G15</i>
<i>l(1)10Bd</i>	1-34.50	10B4-9	<i>Df(1)N71</i>	<i>Df(1)DA622</i>	dsh , <i>l(1)G17</i>
<i>l(1)10Be</i>	1-34.61	10B4-9	<i>Df(1)N71</i>	<i>Df(1)DA622</i>	hop , <i>l(1)LA</i> , <i>l(1)G18</i>
<i>l(1)10Bf</i>	1-34.82	10B8-9	<i>Df(1)DA622</i>	<i>Df(1)M13</i>	dlg , <i>l(1)L11</i> , <i>l(1)G19</i> , <i>l(1)bwn</i> , <i>l(1)d.lg-1</i> , <i>l(1).pr-2</i>
<i>l(1)10Bg</i>	1-35.08	10B8-15	<i>Df(1)M13</i>		<i>l(1)G20</i>
<i>l(1)10Bh</i>	1-35.31	10B8-15	<i>Df(1)M13</i>		<i>l(1)G21</i>
<i>l(1)10Bi</i>	1-[35]	10B8-15	<i>Df(1)RA37</i>		
<i>l(1)10Bj</i>	1-[35]	10B8-15	<i>Df(1)M13</i>		<i>l(1)G22</i>
<i>l(1)10Bk</i>	1-[35]	10B8-15	<i>Df(1)RA37</i>		
<i>l(1)10Bl</i>	1-[35]	10B8-15	<i>Df(1)GA112</i>	<i>Df(1)RA37</i>	
<i>l(1)10Bm</i>	1-[35]	10B8-15	<i>Df(1)M13</i>		<i>l(1)G23</i>
<i>l(1)10Bn</i>	1-[35]	10B8-15	<i>Df(1)RA37</i>		
<i>l(1)10Bo</i>	1-[35]	10B8-15	<i>Df(1)RA37</i>		
<i>l(1)10Bp</i>	1-[35]	10B17-C2	<i>Df(1)GA112</i>	<i>Df(1)RA37</i>	

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)10Ba</i> ¹	EMS		<i>l(1)v4</i>	1	larval-pupal lethal
<i>l(1)10Ba</i> ²	EMS	Lefevre	<i>l(1)VA273</i>	4	
<i>l(1)10Ba</i> ³	EMS	Lefevre	<i>l(1)VE663</i>	4	
<i>l(1)10Ba</i> ⁴	EMS	Lefevre	<i>l(1)VE731</i>	4	
<i>l(1)10Ba</i> ⁵	EMS	Lefevre	<i>l(1)VE874</i>	4, 6	PP/NME
<i>l(1)10Ba</i> ⁶	ENU	Voelker	<i>l(1)M26</i>		

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)10Ba</i> ⁷	ENU	Voelker	<i>l(1)M64</i>		
<i>l(1)10Bb</i> ¹	EMS		<i>l(1)v7</i>	1	embryonic-larval lethal
<i>l(1)10Bb</i> ²	X ray	Lefevre	<i>l(1)GE255</i>	3	<i>In(1) + T(1;2)</i> ^β
<i>l(1)10Bb</i> ³	EMS	Lefevre	<i>l(1)DF961</i>	4	
<i>l(1)10Bb</i> ⁴	EMS	Lefevre	<i>l(1)EC230</i>	4, 6	L1-2/L
<i>l(1)10Bc</i> ¹	EMS		<i>l(1)v16</i>	1	
<i>l(1)10Bc</i> ²	EMS		<i>l(1)v17</i>	1	
<i>l(1)10Bc</i> ³	EMS		<i>l(1)v18</i>	1	
<i>l(1)10Bc</i> ⁴	EMS		<i>l(1)v22</i>	1	
<i>l(1)10Bc</i> ⁵	EMS		<i>l(1)v64</i>	1	
<i>l(1)10Bc</i> ⁶	EMS		<i>l(1)v149</i>	1	
<i>l(1)10Bc</i> ⁷	EMS		<i>l(1)v212</i>	1	
<i>l(1)10Bc</i> ⁸	X ray	Lefevre	<i>l(1)C9</i>	3	
<i>l(1)10Bc</i> ⁹	X ray	Lefevre	<i>l(1)GA118</i>	3	<i>T(1;2)10B4;11A;37A</i> ^γ
<i>l(1)10Bc</i> ¹⁰	EMS	Lefevre	<i>l(1)EA126</i>	4	
<i>l(1)10Bc</i> ¹¹	EMS	Lefevre	<i>l(1)EF416</i>	4	
<i>l(1)10Bc</i> ¹²	EMS	Lefevre	<i>l(1)VA178</i>	4, 6	L3/AO
<i>l(1)10Bc</i> ¹³	EMS	Lefevre	<i>l(1)VE626</i>	4	
<i>l(1)10Bc</i> ¹⁴	EMS	Lefevre	<i>l(1)VE643</i>	4	
<i>l(1)10Bc</i> ¹⁵	EMS	Lefevre	<i>l(1)VE658</i>	4	
<i>l(1)10Bc</i> ¹⁶	ENU	Voelker	<i>l(1)M5</i>		
<i>l(1)10Bc</i> ¹⁷	ENU	Voelker	<i>l(1)M6</i>		
<i>l(1)10Bc</i> ¹⁸	ENU	Voelker	<i>l(1)M22</i>		
<i>l(1)10Bc</i> ¹⁹	ENU	Voelker	<i>l(1)M33</i>		
<i>l(1)10Bc</i> ²⁰	ENU	Voelker	<i>l(1)M34</i>		
<i>l(1)10Bg</i> ¹	EMS		<i>l(1)v21</i>	1	larval lethal
<i>l(1)10Bg</i> ²	X ray	Lefevre	<i>l(1)GA110</i>	3	complex rearrangement
<i>l(1)10Bg</i> ³	EMS	Lefevre	<i>l(1)EF476</i>	4	escapers → thin bristles
<i>l(1)10Bg</i> ⁴	EMS	Lefevre	<i>l(1)DC705</i>	4, 6	L/AO
<i>l(1)10Bg</i> ⁵	ENU	Voelker	<i>l(1)M2</i>		
<i>l(1)10Bg</i> ⁶	ENU	Voelker	<i>l(1)M30</i>		
<i>l(1)10Bg</i> ⁷	ENU	Voelker	<i>l(1)M40</i>		
<i>l(1)10Bg</i> ⁸	ENU	Voelker	<i>l(1)M53</i>		
<i>l(1)10Bh</i> ¹	EMS		<i>l(1)v73</i>	1	pupal lethal
<i>l(1)10Bh</i> ^{2ms}	EMS		<i>MSV12</i>	1	viable, male sterile
<i>l(1)10Bh</i> ^{3ms}	EMS		<i>MSV22</i>	1	viable, male sterile
<i>l(1)10Bi</i> ¹	EMS		<i>l(1)v28</i>	1	
<i>l(1)10Bj</i> ¹	EMS		<i>l(1)v127</i>	1	larval lethal
<i>l(1)10Bj</i> ²	ENU		<i>l(1)M44</i>	7	leaky; phenotype normal
<i>l(1)10Bk</i> ¹	X ray	Lefevre	<i>l(1)L3</i>	2, 6	PP/L
<i>l(1)10Bk</i> ²	X ray	Lefevre	<i>l(1)HC157</i>	3	
<i>l(1)10Bk</i> ³	EMS	Lefevre	<i>l(1)EC236</i>	4	
<i>l(1)10Bk</i> ⁴	EMS	Voelker	<i>l(1)K10</i>	7	
<i>l(1)10Bk</i> ⁵	EMS	Voelker	<i>l(1)K19</i>	7	
<i>l(1)10Bk</i> ⁶	EMS	Voelker	<i>l(1)K22</i>	7	
<i>l(1)10Bk</i> ⁷	ENU	Voelker	<i>l(1)M19</i>	7	
<i>l(1)10Bk</i> ⁸	ENU	Voelker	<i>l(1)M46</i>	7	
<i>l(1)10Bk</i> ⁹	EMS		<i>l(1)v217</i>	7	
<i>l(1)10Bl</i> ¹	ENU	Voelker	<i>l(1)M23</i>	7	leaky
<i>l(1)10Bl</i> ²	ENU	Voelker	<i>l(1)M27</i>	7	leaky
<i>l(1)10Bl</i> ³	ENU	Voelker	<i>l(1)M31</i>	7	leaky
<i>l(1)10Bm</i> ¹	X ray	Lefevre	<i>l(1)L9</i>	2, 6	L3-P/AO
<i>l(1)10Bn</i> ¹	X ray	Lefevre	<i>l(1)L19</i>	5, 6	E-L/VME
			<i>l(1)C248</i>		
<i>l(1)10Bo</i> ¹	EMS	Lefevre	<i>l(1)VA188</i>	4, 6	L-P/AO
<i>l(1)10Bo</i> ²	EMS	Lefevre	<i>l(1)VA328</i>	4	
<i>l(1)10Bo</i> ³	EMS	Lefevre	<i>l(1)VE916</i>	4	

^α 1 = Geer, Lischwe, and Murphy, 1981, J. Exp. Zool. 225: 107-18; 2 = Lefevre, 1971, Genetics 67: 497-513; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 5 = Mortin and Lefevre, 1981, Chromosoma 82: 237-47; 6 = Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14; 7 = Voelker, Wisely, Huang, and Gyurkovics, 1985, Mol. Gen. Genet. 201: 437-45.

β Tentative order: 1-7F|20-10A4|50B-21;20|8A-10A|50B-60.
 γ Tentative order: 1-10B4|37A-60;20-11A|10B4-11A|37-21.

l(1)10C

Searches of Lefevre, of Geer *et al.*, and of Voelker *et al.* extend into this region. 10C is subdivided into two regions by the *Df(1)v-N48* breakpoint. Of four lethally mutable loci in the more distal region, 10C1-5, *RpII215* is 0.02 map units to the left of *l(1)10Cc* (5X2/63,658) other pairs of loci have not been separated by recombination: *RpII215-tyl* (0/8,606), *tyl-l(1)10Cc* (0/81,402), and *l(1)10Cc-l(1)10Cd* (0/144,897); the fifth locus, *nod*, was tested for complementation, but not by recombination (Voelker, Wisely, Huang, and Gyurkovics, 1985, *Mol. Gen. Genet.* 201: 437-45).

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(1)10Ca</i>	1-35.66	10C1-5	<i>Df(1)v-N48</i>	<i>Df(1)GA112</i>	RpII215 , <i>l(1)L5</i>
<i>l(1)10Cb</i>	1-[36]	10C1-5	<i>Df(1)v-N48</i>	<i>Df(1)GA112</i>	tyl
<i>l(1)10Cc</i>	1-35.68	10C1-5	<i>Df(1)v-N48</i>	<i>Df(1)GA112</i>	<i>l(1)L20</i>
<i>l(1)10Cd</i>	1-[36]	10C1-5	<i>Df(1)v-N48</i>	<i>Df(1)GA112</i>	
<i>l(1)10Ce</i>	1-35.9	10C3-D5	<i>Df(1)N71</i>	<i>Df(1)v-N48</i>	<i>l(1)L16</i>
<i>l(1)10Cf</i>	1-[36]	10C3-D5	<i>Df(1)N71</i>	<i>Df(1)v-N48</i>	dsi
<i>l(1)10Cg</i>	1-[35]	10B17-C2	<i>Df(1)GA112</i>		
<i>l(1)10Ch</i>	1-[36]				
<i>l(1)10Ci</i>	1-[36]	10C1-5	<i>Df(1)v-N48</i>	<i>Df(1)GA112</i>	

allele	origin	discoverer	synonym	ref ^{α}	comments
<i>l(1)10Cc</i> ¹	X ray	Lefevre	<i>l(1)L20</i> <i>l(1)GA47</i>	2, 4	
<i>l(1)10Cc</i> ²	X ray	Lefevre	<i>l(1)HA10</i>	4, 5	L1-2/L
<i>l(1)10Cc</i> ³	EMS	Voelker	<i>l(1)K5</i>	6	
<i>l(1)10Cc</i> ⁴	HD	Voelker	<i>l(1)32/65A</i>	6	
<i>l(1)10Cd</i> ¹	EMS	Voelker	<i>l(1)M39</i>	6	
<i>l(1)10Ce</i> ¹	X ray	Lefevre	<i>l(1)L16</i>	1	
<i>l(1)10Ce</i> ²	X ray	Lefevre	<i>l(1)RA6</i>	2	
<i>l(1)10Ce</i> ³	X ray	Lefevre	<i>l(1)RA60</i>	2	
<i>l(1)10Ce</i> ⁴	X ray	Lefevre	<i>l(1)S51</i>	2	
<i>l(1)10Ce</i> ⁵	EMS	Lefevre	<i>l(1)DC833</i>	3, 5	L1-2/VME
<i>l(1)10Ce</i> ⁶	EMS	Lefevre	<i>l(1)EF464</i>	3	
<i>l(1)10Ce</i> ⁷	EMS	Lefevre	<i>l(1)VA270</i>	3	
<i>l(1)10Ce</i> ⁸	EMS	Lefevre	<i>l(1)VE710</i>	3	
<i>l(1)10Ce</i> ⁹	EMS	Lefevre	<i>l(1)VE912</i>	3	
<i>l(1)10Ce</i> ¹⁰	EMS	Lefevre	<i>l(1)VE914</i>	3	
<i>l(1)10Ce</i> ¹¹	EMS	Voelker	<i>l(1)K29</i>	6	
<i>l(1)10Ce</i> ¹²	ENU	Voelker	<i>l(1)M9</i>	6	
<i>l(1)10Ce</i> ¹³	ENU	Voelker	<i>l(1)M7</i>	6	
<i>l(1)10Ce</i> ¹⁴	ENU	Voelker	<i>l(1)M16</i>	6	
<i>l(1)10Ce</i> ¹⁵	ENU	Voelker	<i>l(1)M29</i>	6	
<i>l(1)10Ce</i> ¹⁶	ENU	Voelker	<i>l(1)M41</i>	6	
<i>l(1)10Cg</i> ¹	X ray	Lefevre	<i>l(1)JC13</i>	2	
<i>l(1)10Ch</i> ¹	X ray	Lefevre	<i>l(1)JC36</i>	2	
<i>l(1)10Ci</i> ¹	EMS	Lefevre	<i>l(1)VE623</i>	3, 5	EL/L

α 1 = Lefevre, 1971, *Genetics* 67: 497-513; 2 = Lefevre, 1981, *Genetics* 99: 461-80; 3 = Lefevre and Watkins, 1986, *Genetics* 113: 869-95; 4 = Mortin and Lefevre, 1981, *Chromosoma* 82: 237-47; 5 = Perrimon, Engstrom, and Mahowald, 1984, *Dev. Biol.* 105: 404-14; 6 = Voelker, Wisely, Huang, and Gyurkovics, 1985, *Mol. Gen. Genet.* 201: 437-45.

l(1)10D and E

locus	genetic location	cytological location	included in	excluded from
<i>l(1)10Da</i>	1-[36]	10D4-E1	<i>Df(1)m259-4</i>	<i>Df(1)N71</i>
<i>l(1)10Db</i>	1-[36]	10D4-E1	<i>Df(1)m259-4</i>	<i>Df(1)N71</i>

locus	genetic location	cytological location	included in	excluded from
<i>l(1)10Dc</i>	1-[36]	10D4-E1	<i>Df(1)m259-4</i>	<i>Df(1)N71</i>
<i>l(1)10Dd</i>	1-[36]	10D2-4	<i>Df(1)N71</i>	<i>Df(1)DA622</i>
<i>l(1)10Ea</i>	1-[36]	10E1-F1	<i>Df(1)KA6</i>	<i>Df(1)RA47</i>
<i>l(1)10Eb</i>	1-[36]	10E1-F1	<i>Df(1)KA6</i>	<i>Df(1)RA47</i>

allele	origin	discoverer	synonym	ref ^{α}	comments
<i>l(1)10Da</i> ¹	X ray	Lefevre	<i>l(1)GF303</i>	1	semilethal allele
<i>l(1)10Da</i> ²	EMS	Lefevre	<i>l(1)VE742</i>	2, 3	L-P/ME
<i>l(1)10Db</i> ¹	EMS	Szidonya	<i>l(1)878</i> ¹⁵	4	
<i>l(1)10Dc</i> ¹	EMS	Voelker	<i>l(1)A1</i>	4	
<i>l(1)10Dd</i> ¹	EMS	Lefevre	<i>l(1)EA18</i>	2	
<i>l(1)10Ea</i> ¹	X ray	Lefevre	<i>l(1)GA82</i>	1	
<i>l(1)10Ea</i> ²	EMS	Lefevre	<i>l(1)DC727</i>	2	
<i>l(1)10Ea</i> ³	EMS	Lefevre	<i>l(1)DF916</i>	2	
<i>l(1)10Ea</i> ⁴	EMS	Lefevre	<i>l(1)VE817</i>	2	
<i>l(1)10Ea</i> ⁵	EMS	Lefevre	<i>l(1)VE897</i>	2	
<i>l(1)10Eb</i> ¹	EMS	Lefevre	<i>l(1)DF939</i>	2, 3	L1-2/L
<i>l(1)10Eb</i> ²	EMS	Lefevre	<i>l(1)DC751</i>	2	
<i>l(1)10Eb</i> ³	EMS	Lefevre	<i>l(1)DC757</i>	2	

α 1 = Lefevre, 1981, *Genetics* 99: 461-80; 2 = Lefevre and Watkins, 1986, *Genetics* 113: 869-95; 3 = Perrimon, Engstrom, and Mahowald, 1984, *Dev. Biol.* 105: 404-14; 4 = Voelker, Wisely, Huang, and Gyurkovics, 1985, *Mol. Gen. Genet.* 201: 437-45.

β *l(1)10Ea*⁴ fails to complement both *l(1)10Ea* and *l(1)10Eb* alleles; possibly a small deficiency.

l(1)10F

With the exception of *qs*, mutants in 10F adequately tested for complementation. *qs* not tested against any other mutants in region.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(1)10Fa</i>	1-[37]	10F1-6	<i>Df(1)RA47</i> <i>Df(1)KA7</i>		<i>l(1)L6</i>
<i>l(1)10Fb</i>	1-[37]	10F1-6	<i>Df(1)RA47</i> <i>Df(1)KA7</i>		
<i>l(1)10Fc</i>	1-[37]	10F1-6	<i>Df(1)RA47</i> <i>Df(1)KA7</i>		
<i>l(1)10Fd</i>	1-[37]	10F1-6	<i>Df(1)RA47</i> <i>Df(1)KA7</i>		<i>l(1)L10</i>
<i>l(1)10Fe</i>	1-[37]	10F1-6	<i>Df(1)RA47</i> <i>Df(1)KA7</i>		<i>l(1)L17</i>
<i>l(1)10Ff</i>	1-[37]	10F6-8	<i>Df(1)HA85</i>	<i>Df(1)KA7</i>	
<i>l(1)10Fg</i>	1-[37]	10F6-8	<i>Df(1)HA85</i>	<i>Df(1)KA7</i>	<i>l(1)L18</i>
<i>l(1)10Fh</i>	1-[37]	10F9	<i>Df(1)RA47</i>	<i>Df(1)HA85</i>	
<i>l(1)10Fi</i>	1-[37]	10F9	<i>Df(1)RA47</i>	<i>Df(1)HA85</i>	
<i>l(1)10Fj</i>	1-[37]	10F10-11	<i>Df(1)KA6</i>	<i>Df(1)RA47</i>	
<i>l(1)10Fk</i>	1-[37]	10F10-11	<i>Df(1)KA6</i>	<i>Df(1)RA47</i>	
<i>l(1)10Fi</i>	1-39.5	10F1-10	<i>Df(1)RA47</i>		qs

allele	origin	discoverer	synonym	ref ^{α}	comments
<i>l(1)10Fa</i> ¹	X ray	Lefevre	<i>l(1)L6</i>	2	
<i>l(1)10Fa</i> ²	X ray	Lefevre	<i>l(1)A119</i>	3	
<i>l(1)10Fa</i> ³	X ray	Lefevre	<i>l(1)A20</i>	3	
<i>l(1)10Fa</i> ⁴	X ray	Lefevre	<i>l(1)GA44</i>	3	
<i>l(1)10Fa</i> ⁵	EMS	Lefevre	<i>l(1)EA10</i>	4	
<i>l(1)10Fa</i> ⁶	EMS	Lefevre	<i>l(1)DF978</i>	4, 5	L1-2/NME
<i>l(1)10Fa</i> ⁷	spont	Schalet	<i>l(1)1-38</i>	6	
<i>l(1)10Fa</i> ⁸	spont	Schalet	<i>l(1)L6</i> ^{S1}		
<i>l(1)10Fa</i> ⁹	spont	Schalet	<i>l(1)L6</i> ^{S2}		
<i>l(1)10Fb</i> ¹	EMS	Lefevre	<i>l(1)VE603</i>	3, 5	L1-2/AO
<i>l(1)10Fc</i> ¹	EMS	Lefevre	<i>l(1)VE754</i>	3	
<i>l(1)10Fd</i> ¹	X ray	Lefevre	<i>l(1)L10</i>	2	
<i>l(1)10Fd</i> ²	X ray	Lefevre	<i>l(1)C228</i>	3	
<i>l(1)10Fd</i> ³	X ray	Lefevre	<i>l(1)GA5</i>	3	
<i>l(1)10Fd</i> ⁴	X ray	Lefevre	<i>l(1)GF305</i>	3	
<i>l(1)10Fd</i> ⁵	EMS	Lefevre	<i>l(1)DA513</i>	4	
<i>l(1)10Fd</i> ⁶	EMS	Lefevre	<i>l(1)EA17</i>	4, 5	P-A/NME

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)10Fd⁷</i>	EMS	Lefevre	<i>l(1)EA21</i>	4	
<i>l(1)10Fd⁸</i>	EMS	Lefevre	<i>l(1)EC210</i>	4	
<i>l(1)10Fd⁹</i>	EMS	Lefevre	<i>l(1)EC237</i>	4	
<i>l(1)10Fd¹⁰</i>	EMS	Lefevre	<i>l(1)VA132</i>	4	
<i>l(1)10Fe¹</i>	X ray	Lefevre	<i>l(1)L17</i>	1, 2	
<i>l(1)10Fe²</i>	X ray	Lefevre	<i>l(1)RA11</i>	3	
<i>l(1)10Fe³</i>	X ray	Lefevre	<i>l(1)HC132</i>	3	
<i>l(1)10Fe⁴</i>	X ray	Lefevre	<i>l(1)HC106</i>	3	
<i>l(1)10Fe⁵</i>	EMS	Lefevre	<i>l(1)VA154</i>	4	
<i>l(1)10Fe⁶</i>	EMS	Lefevre	<i>l(1)VA222</i>	4	
<i>l(1)10Fe⁷</i>	EMS	Lefevre	<i>l(1)VE620</i>	4	
<i>l(1)10Fe⁸</i>	EMS	Lefevre	<i>l(1)VE755</i>	4, 5	L3-P/MER
<i>l(1)10Fi¹</i>	EMS	Lefevre	<i>l(1)EF421</i>	4	
<i>l(1)10Fi²</i>	EMS	Lefevre	<i>l(1)VA171</i>	1, 4	
<i>l(1)10Fi³</i>	EMS	Lefevre	<i>l(1)VE615</i>	4	
<i>l(1)10Fg¹</i>	X ray	Lefevre	<i>l(1)L18</i>	1, 2	
<i>l(1)10Fg²</i>	X ray	Lefevre	<i>l(1)HF364</i>	3	
<i>l(1)10Fg³</i>	X ray	Lefevre	<i>l(1)RA15</i>	3	
<i>l(1)10Fg⁴</i>	EMS	Lefevre	<i>l(1)EA35</i>	4	
<i>l(1)10Fg⁵</i>	EMS	Lefevre	<i>l(1)VA331</i>	4	
<i>l(1)10Fh¹</i>	EMS	Lefevre	<i>l(1)VE694</i>	1, 4	
<i>l(1)10Fi¹</i>	EMS	Lefevre	<i>l(1)VA147</i>	1, 4, 5	PP/L
<i>l(1)10Fj¹</i>	X ray	Lefevre	<i>l(1)A29</i>	1, 3, 5	PP/L; <i>T(1;2)10F9;21E3</i>
<i>l(1)10Fk¹</i>	X ray	Lefevre	<i>l(1)RF6</i>	1, 3	

^α 1 = Kulkarni and Hall, 1987, Genetics 115: 461-75; 2 = Lefevre, 1971, Genetics 67: 497-513; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 5 = Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14; 6 = Schalet, 1986, Mutat. Res. 163: 115-44.

DEFICIENCY MAP OF REGION 10

side	breakpoint	variant	DNA coordinates ^α
right	9F13-10A2	<i>Df(1)sbr1</i>	
right	10A1	<i>Df(1)sbr10</i>	
	10A1	v	
right	10A1	csk	
right	10A1	<i>Df(1)ras-v-P26</i>	
right	10A1	<i>Df(1)v-L2</i>	
right	10A1-2	<i>Df(1)sbr-K8</i>	
right	10A1-2	<i>Df(1)sbr-K9</i>	
right	10A1-2	<i>Df(1)v-B151</i>	
right	10A1-2	<i>Df(1)v-L4</i>	
right	10A1-2	<i>Df(1)v-L7</i>	
right	10A1-2	<i>Df(1)v-L11</i>	
right	10A1-2	<i>Df(1)v-L15</i>	
right	10A1-2	<i>Df(1)v-M1</i>	
right	10A1-2	<i>Df(1)v-M5</i>	
right	10A1-2	<i>Df(1)v-M6</i>	
right	10A1-2	<i>Df(1)v-M7</i>	
right	10A1-2	<i>Df(1)v-P5</i>	
right	10A2	<i>Df(1)v64f</i>	
		sev	
right	10A2	<i>Df(1)ras-v17</i>	
		ms(1)10A	
		slm	
		<i>l(1)10Ac</i>	
right	10A4-5	<i>Df(1)v-L1</i>	
left	10A8	<i>Df(1)RA37</i>	
	10A6-7	<i>l(1)10Ad</i>	
	10A6-7	<i>l(1)10Ae</i>	
right	10A7-8	<i>Df(1)v-L3</i>	
	10A8	<i>l(1)10Af</i>	
left	10A9	<i>Df(1)KA7</i>	
	10A7-11	rtv	
	10A8-11	<i>l(1)10Ah</i>	
		<i>l(1)10Ai</i>	
right	10A11	<i>Dp(1;2)v⁺63i</i>	
		tu-Sz	
left	10A11-B1	<i>Df(1)GA112</i>	
		<i>l(1)10Aj</i>	
	10B4-8	<i>l(1)10Ba</i>	
left	10B4-5	<i>Df(1)N71</i>	

side	breakpoint	variant	DNA coordinates ^α
	10B4-8	<i>sisA</i>	
	10B4-8	<i>l(1)10Bb</i>	
	10B4-8	<i>dsh</i>	
	10B4-8	<i>hop</i>	
	10B4-8	<i>l(1)10Bc</i>	
	10B4-8	<i>ny</i>	
left	10B8	<i>Df(1)DA622</i>	48 to 47 kb
	10B8-9	<i>dlg</i>	36 to 11 kb
left		<i>Df(1)M-13</i>	0 kb
	10B8-15	<i>l(1)10Bg</i>	
	10B8-15	<i>l(1)10Bh</i>	
	10B17-C2	<i>l(1)10Bi</i>	
	10B17-C2	<i>l(1)10Bj</i>	
	10B17-C2	<i>l(1)10Bl</i>	
	10B17-C2	<i>l(1)10Bm</i>	
	10B17-C2	<i>l(1)10Bn</i>	
right	10B17	<i>Df(1)RA37</i>	
	10B17-C2	<i>l(1)10Bk</i>	
	10B17-C2	<i>l(1)10Bo</i>	
		<i>l(1)10Cg</i>	
right	10C1-2	<i>v⁺Yy⁺</i>	
right	10C1-2	<i>Df(1)GA112</i>	
left	10C1-2	<i>Df(1)HA85</i>	
right	10C2	<i>Dp(1;2)v⁺75d</i>	
left	10C2-3	<i>Df(1)m259</i>	
		<i>Rpil215</i>	-7.2 to 0.2 kb
		<i>tyl</i>	0.5 to 1.3 kb (tentative)
		<i>l(1)10Cc</i>	1.5 to 2.6 kb (tentative)
		<i>l(1)10Cd</i>	
		<i>l(1)10Ch</i>	
		<i>l(1)10Ci</i>	
		<i>nod</i>	
right	10C3-5	<i>Df(1)v-N48</i>	
		<i>l(1)10Ce</i>	
		<i>dsi</i>	
right	10D2	<i>Df(1)DA622</i>	
		<i>l(1)10Dd</i>	
right	10D4	<i>Df(1)N71</i>	
right	10D4	<i>Df(1)HM456</i>	
		<i>l(1)10Da</i>	
left	10E1	<i>Df(1)KA6</i>	
	10E	<i>l(1)10Ea</i>	
	10E	<i>l(1)10Eb</i>	
right	10E1-2	<i>Df(1)m259-4</i>	
	10E1-2	<i>m</i>	
right	10E3-4	<i>v⁺B^{S-}Yy⁺</i>	
		<i>dy</i>	
left	10F1	<i>Df(1)RA47</i>	
	10F	<i>l(1)10Fa</i>	
	10F	<i>l(1)10Fb</i>	
	10F	<i>l(1)10Fc</i>	
	10F	<i>l(1)10Fd</i>	
	10F	<i>l(1)10Fe</i>	
right	10F10	<i>Df(1)KA7</i>	
	10F	<i>l(1)10Fg</i>	
	10F	<i>l(1)10Ff</i>	
right	11A1-2	<i>Df(1)HA85</i>	
	10F	<i>l(1)10Fh</i>	
	10F1-10	<i>l(1)10Fi</i>	
right	10F11	<i>Df(1)RA47</i>	
		<i>l(1)10Fj</i>	
		<i>l(1)10Fk</i>	
right	11A7	<i>Df(1)KA6</i>	

^α Region 10B coordinates by Woods and Bryant (1989, Dev. Biol. 134: 222-35); region 10C by Biggs, Searles, and Greenleaf (1985, Cell 42: 611-21).

l(1)11A

Nine lethally mutable loci including *tsg* and *agn*.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(1)11Aa</i>	1-36.66	11A2	<i>Df(1)RC29</i>	<i>Df(1)RA47</i>	<i>l(1)L13</i>
<i>l(1)11Ab</i>	1-36.79	11A2	<i>Df(1)RC29</i>		<i>tsg</i>

locus	genetic location		cytological location		included in	excluded from	synonym
	location	location	location	location			
<i>l(1)11Ac</i>							
<i>l(1)11Ad</i>			<i>Df(1)KA10</i>	<i>Df(1)RC29</i>			
			<i>Df(1)HF368</i>				
<i>l(1)11Ae</i>	1-37.0	11A6	<i>Df(1)ml3</i>	<i>Df(1)RC29</i>		<i>l(1)L2</i>	
<i>l(1)11Af</i>	1-38.9	11A7-9	<i>Df(1)v65b</i>	<i>Df(1)KA10</i>		agn	
			<i>Df(1)HF368</i>				
<i>l(1)11Ag</i>			<i>Df(1)HF368</i>	<i>Df(1)KA10</i>			
<i>l(1)11Ah</i>			<i>Df(1)HF368</i>	<i>Df(1)KA10</i>			
<i>l(1)11Ai</i>			<i>Df(1)HF368</i>	<i>Df(1)KA10</i>			

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)11Aa</i> ¹	X ray	Lefevre	<i>l(1)L13</i>	1	
<i>l(1)11Aa</i> ²	X ray	Lefevre	<i>l(1)A78</i>	2	<i>In(1)10F-11A;20</i>
<i>l(1)11Aa</i> ³	X ray	Lefevre	<i>l(1)A97</i>	2	<i>In(1)1E1-2;11A2</i>
<i>l(1)11Aa</i> ⁴	X ray	Lefevre	<i>l(1)A101</i>	2	<i>In(1)11A1-2;12E1-2;18B11</i>
<i>l(1)11Aa</i> ⁵	X ray	Lefevre	<i>l(1)C155</i>	2	
<i>l(1)11Aa</i> ⁶	X ray	Lefevre	<i>l(1)HC129</i>	2	
<i>l(1)11Aa</i> ⁷	X ray	Lefevre	<i>l(1)HC146</i>	2	<i>In(1)11A7;12A1</i>
<i>l(1)11Aa</i> ⁸	X ray	Lefevre	<i>l(1)HC268</i>	2	
<i>l(1)11Aa</i> ⁹	X ray	Lefevre	<i>l(1)N66</i>	2	<i>In(1)7A7-8;11A4</i>
<i>l(1)11Aa</i> ¹⁰	X ray	Lefevre	<i>l(1)RF23</i>	2	
<i>l(1)11Aa</i> ¹¹	X ray	Lefevre	<i>l(1)RF56</i>	2	complex breaks in 11A6, 15B, 34A, 38C, 69C-D, 77E-F, 81, 88E-F
<i>l(1)11Aa</i> ¹²	EMS	Lefevre	<i>l(1)L13</i> ⁵⁻⁸	3	
<i>l(1)11Aa</i> ¹³	EMS	Lefevre	<i>l(1)L13</i> ²⁰⁻³	3	
<i>l(1)11Aa</i> ¹⁴	EMS	Lefevre	<i>l(1)L13</i> ³²⁵	3	
<i>l(1)11Aa</i> ¹⁵	EMS	Lefevre	<i>l(1)EA80</i>	3	
<i>l(1)11Aa</i> ¹⁶	EMS	Lefevre	<i>l(1)EA121</i>	3	
<i>l(1)11Aa</i> ¹⁷	EMS	Lefevre	<i>l(1)EC252</i>	3	
<i>l(1)11Aa</i> ¹⁸	EMS	Lefevre	<i>l(1)EC276</i>	3	
<i>l(1)11Aa</i> ¹⁹	EMS	Lefevre	<i>l(1)DA600</i>	3	
<i>l(1)11Aa</i> ²⁰	EMS	Lefevre	<i>l(1)DC752</i>	3	
<i>l(1)11Aa</i> ²¹	EMS	Lefevre	<i>l(1)VA47</i>	3	
<i>l(1)11Aa</i> ²²	EMS	Lefevre	<i>l(1)VA226</i>	3	
<i>l(1)11Aa</i> ²³	EMS	Lefevre	<i>l(1)VE870</i>	3	
<i>l(1)11Aa</i> ²⁴	spont	Schalet	<i>l(1)4-92</i>	4	
<i>l(1)11Aa</i> ²⁵	spont	Schalet	<i>l(1)9-78</i>	4	
<i>l(1)11Aa</i> ²⁶	spont	Schalet	<i>l(1)20-195</i>	4	
<i>l(1)11Ac</i> ¹	X ray	Lefevre	<i>l(1)HA77</i>	2	
<i>l(1)11Ac</i> ²	X ray	Lefevre	<i>l(1)KC23</i>	2	
<i>l(1)11Ac</i> ³	X ray	Lefevre	<i>l(1)RF32</i>	2	
<i>l(1)11Ad</i> ¹	X ray	Lefevre	<i>l(1)A4</i>	2	
<i>l(1)11Ad</i> ²	X ray	Lefevre	<i>l(1)HC281</i>	2	
<i>l(1)11Ad</i> ³	EMS	Lefevre	<i>l(1)DC816</i>	3	
<i>l(1)11Ad</i> ⁴	EMS	Lefevre	<i>l(1)VA213</i>	3	
<i>l(1)11Ae</i> ¹	X ray	Lefevre	<i>l(1)L2</i>	1	
<i>l(1)11Ae</i> ²	X ray	Lefevre	<i>l(1)A92</i>	2	<i>T(1;2)11A5-6;37B + T(1;3)5A7;100</i>
<i>l(1)11Ae</i> ³	X ray	Lefevre	<i>l(1)C221</i>	2	
<i>l(1)11Ae</i> ⁴	X ray	Lefevre	<i>l(1)C227</i>	2	
<i>l(1)11Ae</i> ⁵	X ray	Lefevre	<i>l(1)HA2</i>	2	
<i>l(1)11Ae</i> ⁶	X ray	Lefevre	<i>l(1)JC79</i>	2	complex; <i>T(1;2)11A5-6;83C-D</i>
<i>l(1)11Ae</i> ⁷	X ray	Lefevre	<i>l(1)JC101</i>	2	
<i>l(1)11Ae</i> ⁸	X ray	Lefevre	<i>l(1)KC15</i>	2	
<i>l(1)11Ae</i> ⁹	EMS	Lefevre	<i>l(1)EA15</i>	3	
<i>l(1)11Ae</i> ¹⁰	EMS	Lefevre	<i>l(1)EA55</i>	3	
<i>l(1)11Ae</i> ¹¹	EMS	Lefevre	<i>l(1)EC232</i>	3	
<i>l(1)11Ae</i> ¹²	EMS	Lefevre	<i>l(1)VA342</i>	3	
<i>l(1)11Ae</i> ¹³	EMS	Lefevre	<i>l(1)VE634</i>	3	
<i>l(1)11Ae</i> ¹⁴	EMS	Lefevre	<i>l(1)VE886</i>	3	
<i>l(1)11Ae</i> ¹⁵	EMS	Geer	<i>l(1)v44</i>	3	
<i>l(1)11Ae</i> ¹⁶	EMS	Voelker	<i>l(1)A100</i>	3	
<i>l(1)11Ag</i> ¹	X ray	Lefevre	<i>l(1)HF303</i>	2	also <i>l(1)11Ah</i>
<i>l(1)11Ag</i> ²	X ray	Lefevre	<i>l(1)HF388</i>	2	
<i>l(1)11Ag</i> ³	EMS	Lefevre	<i>l(1)EC260</i>	3	
<i>l(1)11Ag</i> ⁴	EMS	Lefevre	<i>l(1)EC267</i>	3	
<i>l(1)11Ag</i> ⁵	EMS	Lefevre	<i>l(1)EF429</i>	3	
<i>l(1)11Ag</i> ⁶	EMS	Lefevre	<i>l(1)DC799</i>	3	
<i>l(1)11Ag</i> ⁷	EMS	Lefevre	<i>l(1)VA266</i>	3	
<i>l(1)11Ag</i> ⁸	EMS	Lefevre	<i>l(1)VA262</i>	3	

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)11Ah</i> ¹	X ray	Lefevre	<i>l(1)HF303</i>	2	also <i>l(1)11Ag</i>
<i>l(1)11Ah</i> ²	X ray	Lefevre	<i>l(1)MGM147</i>	2	
<i>l(1)11Ah</i> ³	X ray	Lefevre	<i>l(1)MGM194</i>	2	
<i>l(1)11Ah</i> ⁴	EMS	Lefevre	<i>l(1)DC837</i>	3	
<i>l(1)11Ah</i> ⁵	EMS	Lefevre	<i>l(1)VE759</i>	3	
<i>l(1)11Ah</i> ⁶	neutrons	Muñoz	<i>l(1)17-58</i>	3	
<i>l(1)11Ai</i> ¹	EMS	Lefevre	<i>l(1)DC799</i>	3	
<i>l(1)11Ai</i> ²	EMS	Lefevre	<i>l(1)DC837</i>	3	
<i>l(1)11Ai</i> ³	EMS	Lefevre	<i>l(1)EC262</i>	3	
<i>l(1)11Ai</i> ⁴	EMS	Lefevre	<i>l(1)EF429</i>	3	

^α 1 = Lefevre, 1971, Genetics 67: 497-513; 2 = Lefevre, 1981, Genetics 99: 461-80; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 4 = Schalet, 1986, Mutat. Res. 163: 115-44.

l(1)11D-F

The product of saturation mutagenesis and deficiency mapping in the region encompassed by *Df(1)C246* carried out by N. Scott (1987, Ph.D. thesis, University of California, San Diego).

locus	genetic location		cytological location		included in	excluded from	synonym
	location	location	location	location			
<i>l(1)11Da</i>	1-[41]	<i>11D1-E</i>	<i>Df(1)C246</i>	<i>Df(1)JA26</i>			
<i>l(1)11Db</i>	1-[41]	<i>11D1-E</i>	<i>Df(1)C246</i>	<i>Df(1)JA26</i>			
<i>l(1)11Dc</i>	1-[41]	<i>11D1-E</i>	<i>Df(1)C246</i>	<i>Df(1)JA26</i>			

allele	origin	discoverer	ref ^α	comments
<i>l(1)11Da</i> ¹	ENU	N. Scott	1, 2	L-P/L
<i>l(1)11Db</i> ¹	ENU	N. Scott	1, 2	L-P/AO
<i>l(1)11Dc</i> ¹	ENU	N. Scott	1, 2	P-A/L

^α 1 = Perrimon, Engstrom, and Mahowald, 1989, Genetics 212: 333-52; 2 = Scott, 1987, Ph.D. thesis, University of California, San Diego.

allele	origin	discoverer	ref ^α	comments
<i>l(1)11Eb</i> ¹	ENU	N. Scott	2	
<i>l(1)11Eb</i> ²	ENU	N. Scott	2	
<i>l(1)11Eb</i> ³	ENU	N. Scott	2	
<i>l(1)11Eb</i> ⁴	ENU	N. Scott	2	
<i>l(1)11Eb</i> ⁵	ENU	N. Scott	2	
<i>l(1)11Eb</i> ⁶	ENU	N. Scott	2	
<i>l(1)11Eb</i> ⁷	ENU	N. Scott	1, 2	L/L; partially complementing allele
<i>l(1)11Ec</i> ¹	ENU	N. Scott	1, 2	P/NME
<i>l(1)11Ed</i> ¹	ENU	N. Scott	1, 2	L-P/L
<i>l(1)11Ed</i> ²	ENU	N. Scott	2	
<i>l(1)11Ee</i> ¹	ENU	N. Scott	2	
<i>l(1)11Ef</i> ¹	ENU	N. Scott	2	

^α 1 = Perrimon, Engstrom, and Mahowald, 1989, Genetics 212: 333-52; 2 = Scott, 1987, Ph.D. thesis, University of California, San Diego.

locus	genetic location		cytological location	
	location	location	included in	excluded from
<i>l(1)11Fa</i>	1-(42)	11F2-12A2	<i>Df(1)C246</i>	<i>Df(1)wy2</i>
<i>l(1)11Fb</i>	1-(42)	11F2-12A2	<i>Df(1)C246</i>	<i>Df(1)wy2</i>

allele	origin	discoverer	ref ^α	comments
<i>l(1)11Fa</i>¹	ENU	N. Scott	1, 2	E-L/L
<i>l(1)11Fb</i>¹	ENU	N. Scott	2	
<i>l(1)11Fb</i>²	ENU	N. Scott	1, 2	E-L/L

^α 1 = Perrimon, Engstrom, and Mahowald, 1989, Genetics 212: 333-52; 2 = Scott, 1987, Ph.D. thesis, University of California, San Diego.

DEFICIENCY MAP OF REGION 11

side	breakpoint	variant
left	11A1	<i>Df(1)JA26</i>
left	11A1	<i>Df(1)KA10</i>
left	11A1	<i>Df(1)N105</i>
left	11A1	<i>Df(1)RC29</i>
left	11A2	<i>Df(1)HF368</i>
	11A2	<i>cac</i>
	11A2	<i>l(1)11Aa</i>
	11A2	<i>nbA</i>
	11A2	<i>gd</i>
	11A2	<i>tsg</i>
	11A2	<i>fw</i>
right	11A2	<i>Df(1)RC29</i>
right	11A1-2	<i>Df(1)HA85</i>
		<i>l(1)11Ae</i>
right	11A4-5	<i>Df(1)m13</i>
right	11A7	<i>Df(1)KA10</i>
right	11A7	<i>Df(1)KA6</i>
right	11A7	<i>Dp(1;2)v⁺65b</i>
	11A7-9	<i>agn</i>
right	11A8-9	<i>Df(1)v65b</i>
right	11B9	<i>Df(1)HF368</i>
right	11B12	<i>Dp(1;3)v⁺74c</i>
right	11C4-D1	<i>Df(1)N105</i>
left	11D1-2	<i>Df(1)C246</i>
left	11D1-2	<i>Df(1)N12</i>
		<i>l(1)11Da</i>
		<i>l(1)11Db</i>
		<i>l(1)11Dc</i>
right	11D-E	<i>Df(1)JA26</i>
		<i>sno</i>
		<i>l(1)11Eb</i>
		<i>l(1)11Ec</i>
		<i>l(1)11Ed</i>
		<i>l(1)11Ee</i>
		<i>l(1)11Ef</i>
		<i>wy</i>
right	11E9-10	<i>Df(1)wy26</i>
right	11F1-2	<i>Df(1)N12</i>
right	11F2-4	<i>Df(1)wy2</i>
		<i>l(1)11Fa</i>
		<i>l(1)11Fb</i>
		<i>crt</i>
		<i>s</i>
right	12A1-2	<i>Df(1)C246</i>

l(1)14-l(1)15

Two studies using lethal mutations in this region have been reported; neither was interested in the lethals *per se*, and the mutants reported in one study were never tested against those in the other, and no case of allelism was reported in either study. *para* is the only genetic landmark common to the two studies. Recent studies by Steller (unpublished) provide more information on rela-

tive positions of loci in 14B-C. Embryonic lethal mutations *baz* and *exd* map to this region, but allelism with the lethals tabulated not tested.

references: Falk, Roselli, Curtiss, Halladay, and Klufas, 1984, Mutat. Res. 126: 25-34.
Ganetzky, 1984, Genetics 108: 897-911.

locus	genetic location		cytological location		synonym
	location	location	included in	excluded from	
<i>l(1)14Aa</i>			<i>Dp(1;4)r⁺</i>	<i>Df(1)80e19a</i>	<i>l(1)h26a</i>
<i>l(1)14Ab</i>			<i>Df(1)80e19a</i>	<i>Df(1)80e27a</i>	<i>l(1)h5c</i>
<i>l(1)14Ca</i>	14C1-2		<i>Dp(1;2)r⁺75c</i>	<i>Df(1)81k21e</i>	<i>l(1)i19e</i>
<i>l(1)14Cb</i>			<i>Df(1)81112h</i>	<i>Df(1)80f18c</i>	<i>l(1)k17a</i>
<i>l(1)14Cc</i>	14C1-2		<i>Dp(1;2)r⁺75c</i>	<i>Df(1)81k21e</i>	<i>l(1)9-21</i>
<i>l(1)14Da</i>	1-53.9		<i>Df(1)r19</i>	<i>Df(1)82c19i</i>	<i>para</i>
<i>l(1)14DEa</i>	1-53.9	14C7-F1	<i>Df(1)D7</i>		<i>l(1)D17</i>
<i>l(1)14DEb</i>	1-53.9	14C7-F1	<i>Df(1)D7</i>		<i>l(1)D23</i>
<i>l(1)14DEc</i>	1-53.9	14C7-F1	<i>Df(1)D7</i>		<i>l(1)D30</i>
<i>l(1)14Ea</i>			<i>Df(1)80f3c</i>	<i>Df(1)82c3k</i>	<i>l(1)k22a</i>
<i>l(1)14Fa</i>			<i>Df(1)81j29i</i>	<i>Df(1)80fb8</i>	<i>l(1)l6f</i>
<i>l(1)15Aa</i>			<i>Df(1)80f18c</i>	<i>Df(1)81i8g</i>	<i>l(1)i15b</i>
<i>l(1)15Ab</i>			<i>Df(1)81k21e</i>	<i>Df(1)81f12a</i>	<i>l(1)k27e</i>
<i>l(1)15Ba</i>			<i>Dp(1;4)r⁺</i>	<i>Df(1)81k9b</i>	<i>l(1)g27a</i>

l(1)15Aa

location: 1-53.0 (1.5 units to the left of *r*).

origin: Induced by ICR170.

synonym: 17.

references: Naguib and Jarry, 1981, Genet. Res. 37: 199-207.

cytology: Placed in 14D1-15A1 based on its inclusion in *Df(1)r9 = Df(1)14D1;15D1* and *Df(1)r-D = Df(1)14B6-15A2* but not *Df(1)r7 = Df(1)15A1;15A5*.

DEFICIENCY MAP OF REGION 14-15

side	breakpoint	study 1 ^α	study 2 ^β	DNA coordinates ^γ
left	14A1	<i>Df(1)80f3c</i>		
left	14A1	<i>Df(1)80fb8</i>		
left	14A1	<i>Df(1)81j16f</i>		
left	14A1	<i>Df(1)81117h</i>		
left	14B5-18	<i>Df(1)81119i</i>		
left	14A1	<i>Df(1)82c25g</i>		
		<i>l(1)14Aa</i>		
left	14A1	<i>Df(1)80e19a</i>		
		<i>l(1)14Ab</i>		
left	14A1	<i>Df(1)80e27a</i>		
left	14A1	<i>Df(1)80f25a</i>		
left	14A1	<i>Df(1)82c19i</i>		
left	14B3-4	<i>Df(1)80g12a</i>	<i>Df(1)80g12a</i>	
left	14B3-4	<i>Df(1)81i21c</i>		
left	14B3-4	<i>Df(1)82c3k</i>	<i>Df(1)82c3k</i>	
		<i>disco</i>		
left	14B3-4	<i>Df(1)80g7d</i>	<i>Df(1)80g7d</i>	
left	14B5-18	<i>Df(1)80e3d</i>		
left	14B5-18	<i>Df(1)80f15f</i>		
left	14B5-18	<i>Df(1)81f20a</i>		
left	14B5-18	<i>Df(1)81f20e</i>		
left	14B5-18	<i>Df(1)81g1i</i>		
left	14B5-18	<i>Df(1)81i25b</i>		
left	14B5-18	<i>Df(1)81j6c</i>		
left	14B5-18	<i>Df(1)81j6e</i>		
left	14B5-18	<i>Df(1)81j29i</i>		
left	14B5-18	<i>Df(1)81j23a</i>		
left	14B5-18	<i>Df(1)81k19b</i>		
left	14B5-18	<i>Df(1)81k23b</i>		
left	14B5-18	<i>Df(1)81l1b</i>		
left	14B5-18	<i>Df(1)81l28f</i>		
left	14B5-18	<i>Df(1)82a2z</i>		
left	14B5-18	<i>Df(1)82b6w</i>		
left	14B5-18	<i>Df(1)82c5b</i>		

side	breakpoint	study 1 α	study 2 β	DNA coordinates γ	side	breakpoint	study 1 α	study 2 β	DNA coordinates γ
left	14B5-18	<i>Df(1)82d7e</i>			right	15A6-11	<i>Df(1)81128f</i>		
	14B5-18		eas		right	15A9	<i>Dp(1;2)r⁺75c</i>		
left	14B5-18	<i>Df(1)81h24b</i>	<i>Df(1)81h24b</i>	ca 36	right	15B	<i>Df(1)81k19b</i>		
	14B5-18		<i>Df(1)E150</i>	ca 43		15B	I(1)15Ba		
left	14B5-18	<i>Df(1)80f29d</i>	<i>Df(1)80f29d</i>	ca 60	right	15D1		<i>Df(1)r19</i>	
left	14B13		<i>Dp(1;2)r⁺75c</i>	ca 64					
	14C1-2		I(1)14Cc	67.1 to 76.2	α		Falk, Roselli, Curtiss, Halladay, and Klufas, 1984, <i>Mutat. Res.</i>		
	14C1-2	I(1)14Ca	I(1)14Ca	72.0 to 76.2			126: 25-34;		
	14C1-2		nonA	72.0 to 80.9	β		Data from 14B-C provided by Steller and updated from Jones and		
left	14C1-2	<i>Df(1)81k21e</i>	<i>Df(1)81k21e</i>	ca 84			Rubin (1990, <i>Neuron</i> 4: 711-23); data from 14D-15 taken from		
left	14B5-18	<i>Df(1)81112h</i>	<i>Df(1)81112h</i>				Ganetzky (1984, <i>Genetics</i> 108: 897-911).		
			bss		γ		Molecular coordinates in 14C from Jones and Rubin; in 14F-15A		
			I(1)14Cb				from D. Falk using probes from a 90-kb walk in the <i>r</i> region		
			mel-41				(Segraves, Christos, Schedl, and Jarry, 1983, <i>Mol. Gen. Genet.</i>		
	14C3-6						189: 34-40); origin not defined.		
left	14C4-5	<i>Df(1)80f18c</i>							
left	14C5-6		<i>Df(1)r-D</i>						
left	14B5-18	<i>Df(1)82a2y</i>							
left	14B5-18	<i>Df(1)81f12a</i>							
left	14C6-8	<i>Df(1)8118g</i>							
left	14C6-8	<i>Df(1)82b10w</i>							
left	14C6-8	<i>Df(1)82b26c</i>							
left	14C7-D1		<i>Df(1)D7</i>						
left	14D1		<i>Df(1)r19</i>						
	14C6-8	para	para						
			I(1)14DEb						
			I(1)14DEc						
			I(1)14DEd						
right	14C6-8	<i>Df(1)81i21c</i>							
right	14D3-4	<i>Df(1)82c19i</i>							
right	14D3-4	<i>Df(1)82d7e</i>							
right	14E	<i>Df(1)81h24b</i>							
right	14E	<i>Df(1)82b6w</i>							
right	14E	<i>Df(1)82c3k</i>							
			I(1)14Ea						
right	14E	<i>Df(1)80f3c</i>		-1 to +20					
right	14E3-F1		<i>Df(1)D7</i>						
right	14F	<i>Df(1)80f8b</i>		-1 to +20					
left	14F1-2		<i>Df(1)D34</i>						
right	14F6		<i>Df(1)D34</i>						
left	14F6		<i>Df(1)4-D17</i>						
			r						
			I(1)14Fa						
right	15A1-2	<i>Df(1)81119i</i>		-1 to +20					
right	15A1-2	<i>Df(1)81j29i</i>		+20 to +37					
right	15A1-2	<i>Df(1)81117h</i>		+37 to +43					
right	15A3-4	<i>Df(1)8118g</i>		+37 to +43					
			I(1)a5Aa						
right	15A3-4	<i>Df(1)82c5b</i>		+37 to +43					
right	15A3-4	<i>Df(1)80f18c</i>							
right	15A3-4	<i>Df(1)81f20a</i>							
right	15A3-4	<i>Df(1)81k23b</i>		+63 to +74					
right	15A3-4	<i>Df(1)8111b</i>		+63 to +74					
right	15A3-4	<i>Df(1)82c25g</i>		+63 to +74					
right	15A3-4	<i>Df(1)82a2y</i>							
right	15A3-4	<i>Df(1)82a2z</i>							
right	15A3-4	<i>Df(1)82b10w</i>							
right	15A3-4	<i>Df(1)82b26c</i>							
right	15A6-11	<i>Df(1)81f12a</i>							
right	15A5		I(1)15Ab						
right	15A5	<i>Df(1)81k21e</i>							
right	15A6		<i>Df(1)r-D17</i>						
right	15A6-11	<i>Df(1)80e3d</i>							
right	15A6-11	<i>Df(1)80e19a</i>							
right	15A6-11	<i>Df(1)80e27a</i>							
right	15A6-11	<i>Df(1)80f15f</i>							
right	15A6-11	<i>Df(1)80f25a</i>							
right	15A6-11	<i>Df(1)80f29d</i>							
right	15A6-11	<i>Df(1)80g12a</i>							
right	15A6-11	<i>Df(1)81f20e</i>							
right	15A6-11	<i>Df(1)81g1i</i>							
right	15A6-11	<i>Df(1)81i25b</i>							
right	15A6-11	<i>Df(1)81j6c</i>							
right	15A6-11	<i>Df(1)81j6e</i>							
right	15A6-11	<i>Df(1)81j16f</i>							
right	15A6-11	<i>Df(1)81j23a</i>							
right	15A6-11	<i>Df(1)81112h</i>							

I(1)16F

A series of lethal mutations in the vicinity of *Sh* isolated by Ferrus. The order of loci in 16F1-4 is as given; that of loci in 16F4-8 not determined?

locus	genetic location		cytological location	
	included in	excluded from	included in	excluded from
I(1)16Fa	1-[59]	16F1-4	$X^{P_3D} JC153$	$X^{PYD} W32$
I(1)16Fb	1-[59]	16F1-4	$X^{P_3D} JC153$	$X^{PYD} W32$
I(1)16Fc	1-[59]	16F1-4	$X^{P_3D} JC153$	$X^{PYD} W32$
I(1)16Fd	1-[59]	16F4-8	$X^{PYD} W32$	$X^{P_3D} V7$
I(1)16Fe	1-[59]	16F4-8	$X^{PYD} W32$	$X^{P_3D} V7$

allele origin synonym

I(1)16Fa ¹	EMS	I(1)305
I(1)16Fa ²	EMS	I(1)579
I(1)16Fb ¹	EMS	I(1)359
I(1)16Fb ²	EMS	I(1)62
I(1)16Fc ¹	EMS	I(1)387
I(1)16Fc ²	EMS	I(1)581
I(1)16Fc ³	EMS	I(1)583
I(1)16Fc ⁴	EMS	I(1)598
I(1)16Fd ¹	EMS	I(1)174
I(1)16Fe ¹	EMS	I(1)1614

I(1)16Fa

phenotype: Major lethal phase in early pupae. Cell lethal in somatic mosaics in the hypoderm of the adult; also lethal in female germ-line mosaics.

I(1)16Fb

phenotype: Major lethal phase in late pupae. Somatic clones of homozygous tissue in adult hypoderm show small bristles, slightly disoriented, unpigmented cuticle, and plexate veins. Gynandromorphs show vibrating appendages in the mutant territory. Germ-line mosaics show a maternal effect since the eggs produced by mutant ovarioles never develop beyond the gastrula stage, irrespective of the zygotic genotype.

I(1)16Fc

phenotype: Major lethal phase in the first larval instar. Somatic mosaics in the adult hypoderm show normal differentiation. Gynandromorphs show vibrating appendages in the mutant territory; also uncoordinated movement on the mutant side. Germ-line mosaics show a maternal effect since the eggs produced by mutant ovarioles never develop beyond the gastrula stage, irrespective of the zygotic genotype.

l(1)16Fd

phenotype: Major lethal phase in early pupae. Somatic mosaics in the adult hypoderm show fewer and smaller bristles and smooth ommatidia. Lethal in germ-line mosaics.

l(1)16Fe

phenotype: Major lethal phase in second larval instar. Somatic mosaics in the adult hypoderm show normal cuticle. Gynandromorphs exhibit vibrating appendages and abnormal wing position in the mutant side. Germ-line clones normal.

l(1)18F-19A

Five lethally mutable loci including *ot*.

locus	cytological		included in	excluded from	synonym
	location	location			
l(1)18Fa					
l(1)19Aa		<i>Df(1)mal11</i>	<i>Df(1)mal10</i>		<i>l(1)EM18</i>
l(1)19Ab	19A3-4	<i>Df(1)mal10</i>	<i>Df(1)T2-4a</i>		ot
l(1)19Ac					<i>l(1)EM19</i>
l(1)19Ad					

allele	origin	discoverer	synonym	ref ^α	comments
l(1)18Fa¹	X ray	Lefevre	<i>l(1)HC223</i>	1	
l(1)18Fa²	EMS	Lefevre	<i>l(1)VA197</i>	2, 3	L1-2/L
l(1)19Aa¹	X ray	Lefevre	<i>l(1)GA84</i>	1	
l(1)19Aa²	EMS	Lefevre	<i>l(1)DA587</i>	2	
l(1)19Ac¹	X ray	Lefevre	<i>l(1)C68</i>	1	
l(1)19Ac²	X ray	Lefevre	<i>l(1)HA64</i>	2	
l(1)19Ad¹	X ray	Lefevre	<i>l(1)C5</i>	1	
l(1)19Ad²	X ray	Lefevre	<i>l(1)HA48</i>	1	

^α 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 3 = Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14;

l(1)19B

Two lethally mutable loci, one of which is *sw*.

locus	cytological		included in	excluded from	synonym
	location	location			

l(1)19Ba	19B1	<i>Df(1)mal10</i>	<i>Df(1)T2-4a</i>		
l(1)19Bb	19B3	<i>Df(1)T2-4a</i>	<i>Df(1)mal22</i>		sw

allele	origin	discoverer	synonym	ref ^α	comments
l(1)19Ba¹	X ray	Lefevre	<i>l(1)A96</i>	1	<i>In(1)18F-19A;19B1-2</i>
l(1)19Ba²	X ray	Lefevre	<i>l(1)A149</i>	1	<i>T(1;2;3)19B1-2;20F;48F;81</i>

^α 1 = Lefevre, 1981, Genetics 99: 461-80;

l(1)19C-D

Four lethally mutable loci identified by Schalet and Lefevre [1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, vol. 1b, pp. 847-902]. Three lethally mutable loci identified in an independently derived sample (Lefevre and Watkins, 1986, Genetics 99: 869-95). Complementation tests between the two sets of mutants not performed; allelism between lethals in the

two groups arbitrarily assigned.

locus	cytological		included in	excluded from
	location	location		
l(1)19Ca	19C1	<i>Df(1)T2-4a</i>	<i>Df(1)mal²²</i>	
l(1)19Cb	19C4	<i>Df(1)mal²²</i>	<i>Df(1)T2-4a</i>	
			<i>Df(1)16-3-22</i>	

l(1)19Cc 19C6

l(1)19Da

allele	origin	discoverer	synonym	ref ^α	comments
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l(1)19Ca¹	neutron	Muñoz	<i>l(1)16-127</i>	3	
l(1)19Ca²	neutron	Muñoz	<i>l(1)17-238</i>	3	
l(1)19Ca³	neutron	Muñoz	<i>l(1)17-457</i>	3	
l(1)19Ca⁴	spont	Schalet	<i>l(1)12-3</i>		
l(1)19Cb¹	neutron	Muñoz	<i>l(1)16-398</i>	3	
l(1)19Cb²	X ray	Lefevre	<i>l(1)C157</i>	1	
l(1)19Cb³	EMS	Lefevre	<i>l(1)VA264</i>	2	E-L1/NME
l(1)19Cc¹	X ray	Singer	<i>l(1)25C4</i>	3	semilethal
l(1)19Cc²	EMS	Singer	<i>l(1)E1C1</i>	3, 4	
l(1)19Cc³	X ray	Lefevre	<i>l(1)A99</i>	1	<i>In(1)6A1-2;7C;9A3;19C-D</i>
l(1)19Cc⁴	X ray	Lefevre	<i>l(1)GA74</i>	1	
l(1)19Cc⁵	X ray	Lefevre	<i>l(1)L20</i>	1	
l(1)19Cc⁶	X ray	Lefevre	<i>l(1)N14</i>	1	
l(1)19Cc⁷	X ray	Lefevre	<i>l(1)N60</i>	1	
l(1)19Cc⁸	EMS	Lefevre	<i>l(1)DA588</i>	2	
l(1)19Cc⁹	EMS	Lefevre	<i>l(1)EF489</i>	2	
l(1)19Cc¹⁰	mei-9 ^β	Schalet	<i>l(1)017-1</i>		
l(1)19Cc¹¹	mei-9	Schalet	<i>l(1)18-2</i>		

l(1)19Da¹ X ray Lefevre *l(1)C238* 1 *T(1;2)19E1-2;35A-B*

^α 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 3 = Schalet and Lefevre, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902; 4 = Schalet and Singer, 1971, DIS 46: 131-32.

^β Spontaneous in the paternal X chromosome of a cross between wild-type males and *mei-9* females, such that the F₁ females were *l(1)3Ac/mei-9*.

l(1)19E

Six lethally mutable loci including four named loci: *run*, *shakB*, *lf*, and *unc*.

locus	cytological		included in	excluded from	synonym
	location	location			

l(1)19Ea	19E1-2	<i>Df(1)B57</i>	<i>Df(1)LB6</i>		run , <i>leg</i>
l(1)19Eb	19E4	<i>Df(1)LB6</i>	<i>Df(1)A118</i>		shakB , <i>Pas</i>
l(1)19Ec	19E6	<i>Df(1)A118</i>	<i>Df(1)A53</i>		
l(1)19Ed	19E4-5	<i>Df(1)A53</i>	<i>Df(1)Q539</i>		
l(1)19Ee	19E5-6	<i>Df(1)A53</i>	<i>Df(1)Q539</i>		lf
l(1)19Ef	19E8	<i>Df(1)Q539</i>	<i>Df(1)DCB1-35b</i>		unc

l(1)19Ec

phenotype: Mutant embryos from heterozygous females exhibit no defects in central or peripheral nervous systems; *l(1)19Ec¹* and *l(1)19Ec¹⁸* are lethal in germ-line clones, whereas *l(1)19Ec⁸* and *l(1)19Ec²⁵* germ-line clones exhibit a paternally rescuable maternal effect. Embryonic phenotype includes head and dorsal-closure defects, and most embryos are twisted (Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
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l(1)19Ec¹ Novitski *l(1)151* 2, 4, 7, 8 L/L

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)19Ec</i>²			<i>l(1)J2</i>	5	
<i>l(1)19Ec</i>³			<i>l(1)J9</i>	5	
<i>l(1)19Ec</i>⁴	spont	Himoe	<i>l(1)N5</i>		
<i>l(1)19Ec</i>⁵	EMS	Lifschytz	<i>l(1)P464</i>	4	
<i>l(1)19Ec</i>⁶	EMS	Lifschytz	<i>l(1)Q256</i>	4	
<i>l(1)19Ec</i>⁷	EMS	Lifschytz	<i>l(1)R-9-2</i>	4	
<i>l(1)19Ec</i>⁸	EMS	Lifschytz	<i>l(1)R-9-28</i>	4, 8	L/MER
<i>l(1)19Ec</i>⁹	EMS	Lifschytz	<i>l(1)YT6</i>	5	
<i>l(1)19Ec</i>¹⁰	X ray	Lefevre	<i>l(1)GA71</i>	2	
<i>l(1)19Ec</i>¹¹	X ray	Lefevre	<i>l(1)HC279</i>	2	mutant or deficient for <i>l(1)19Ed</i>
<i>l(1)19Ec</i>¹²	X ray	Lefevre	<i>l(1)HF417</i>	2	
<i>l(1)19Ec</i>¹³	X ray	Lefevre	<i>l(1)JC8</i>	2	
<i>l(1)19Ec</i>¹⁴	X ray	Lefevre	<i>l(1)KA12</i>	2	
<i>l(1)19Ec</i>¹⁵	X ray	Lefevre	<i>l(1)L39</i>	2	
<i>l(1)19Ec</i>¹⁶	EMS	Lefevre	<i>l(1)DA507</i>	3	
<i>l(1)19Ec</i>¹⁷	EMS	Lefevre	<i>l(1)DA536</i>	3	
<i>l(1)19Ec</i>¹⁸	EMS	Lefevre	<i>l(1)EC242</i>	3, 6	L1-2/L
<i>l(1)19Ec</i>¹⁹	EMS	Lefevre	<i>l(1)VE863</i>	3	
<i>l(1)19Ec</i>²⁰	EMS	Lefevre	<i>l(1)VE866</i>	3	
<i>l(1)19Ec</i>²¹	EMS	Lefevre	<i>l(1)VE926</i>	3	
<i>l(1)19Ec</i>²²	<i>mei-9</i> ^β	Schalet	<i>l(1)A-2</i>		
<i>l(1)19Ec</i>²³			<i>l(1)I7-457</i>	8	
<i>l(1)19Ec</i>²⁴			<i>l(1)AF2/19</i>	6	
<i>l(1)19Ec</i>²⁵			<i>l(1)LB2</i>	6	L/MER

^α 1 = Eeken, Sobels, Hyland, and Schalet, 1985, Mutat. Res. 150: 261-75; 2 = Lefevre, 1981, Genetics 99: 461-80; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 4 = Lifschytz and Falk, 1969, Mut. Res. 8: 147-55 10; 5 = Lifschytz and Jakobovitz, 1978, Mol. Gen. Genet. 161: 275-84; 6 = Schalet and Lefevre, 1973, Chromosoma 44: 183-202; 7 = Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31; 8 = Schalet and Lefevre, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902; 9 = Zusman, Coulter, and Gergen, 1985, DIS 61: 217-18.

^β Spontaneous in the paternal X chromosome of a cross between wild-type males and *mei-9* females, such that the *F₁* females were *l(1)3Aclmei-9*.

l(1)19Ed

phenotype: Mutant embryos have normal central nervous systems (CNS) and peripheral nervous systems (PNS); germ-line clones are lethal (Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)19Ed</i>¹	X ray	Lefevre	<i>l(1)HC279</i>	2	mutant or deficient for <i>l(1)19Ec</i>
<i>l(1)19Ed</i>²	EMS	Lefevre	<i>l(1)EC235</i>	3	L1/L
<i>l(1)19Ed</i>³	EMS	Lefevre	<i>l(1)VE909</i>	3	
<i>l(1)19Ed</i>⁴	P	Gergen	<i>l(1)5-7</i>	9	
<i>l(1)19Ed</i>⁵	P	Gergen	<i>l(1)7-2</i>	9	
<i>l(1)19Ed</i>⁶	P	Gergen	<i>l(1)24A</i>	9	
<i>l(1)19Ed</i>⁷	P	Gergen	<i>l(1)26A</i>	9	
<i>l(1)19Ed</i>⁸	P	Gergen	<i>l(1)31B(C)</i>	9	
<i>l(1)19Ed</i>⁹	P	Gergen	<i>l(1)40A</i>	9	
<i>l(1)19Ed</i>¹⁰	P	Gergen	<i>l(1)48-1</i>	9	
<i>l(1)19Ed</i>¹¹	P	Gergen	<i>l(1)BH9</i>	9	
<i>l(1)19Ed</i>¹²	P	Gergen	<i>l(1)HT1</i>	9	
<i>l(1)19Ed</i>¹³	P	Gergen	<i>l(1)PC7</i>	9	
<i>l(1)19Ed</i>¹⁴	P	Gergen	<i>l(1)PG7</i>	9	
<i>l(1)19Ed</i>¹⁵	P	Gergen	<i>l(1)PU1</i>	9	
<i>l(1)19Ed</i>¹⁶			<i>l(1)1127</i>	6	
<i>l(1)19Ed</i>¹⁷	MR		<i>l(1)D76</i>	1, 6	
<i>l(1)19Ed</i>¹⁸	MR		<i>l(1)D83</i>	1, 6	
<i>l(1)19Ed</i>¹⁹	HMS	Kramers	<i>l(1)HM435</i>	6	

^α 1 = Eeken, Sobels, Hyland, and Schalet, 1985, Mutat. Res. 150: 261-75; 2 = Lefevre, 1981, Genetics 99: 461-80; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 4 = Lifschytz and Falk,

1969, Mutat. Res. 8: 147-55 10; 5 = Lifschytz and Jakobovitz, 1978, Mol. Gen. Genet. 161: 275-84; 6 = Schalet and Lefevre, 1973, Chromosoma 44: 183-202; 7 = Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31; 8 = Schalet and Lefevre, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902; 9 = Zusman, Coulter, and Gergen, 1985, DIS 61: 217-18.

Three mutants designated D83, D76, and HM46 placed between right breakpoints of *Df(1)16-3-35* and *Df(1)A118*; complementation of *l(1)19Ec*, *l(1)19Ed*, or *lf* not tested (Kramers, Schalet, Paradi, Huizer-Hoogteyling, 1983, Mutat. Res. 107: 187-201).

l(1)19F

Seven lethally mutable loci identified, including *lfl* and *fil*; *l(1)19Fc* found only once, on *mal⁺Y* but not on the X in several large screens.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(1)19Fa</i>			<i>Df(1)DCB1-35b</i>	<i>Df(1)17-257</i>	<i>fl</i>
<i>l(1)19Fb</i>	19F2		<i>Df(1)DCB1-35b</i>	<i>Df(1)17-257</i>	
<i>l(1)19Fc</i>					
<i>l(1)19Fd</i>	19F3-4		<i>Df(1)17-257</i>		<i>fil</i>
			<i>Df(1)16-129</i>		
<i>l(1)19Fe</i>	19F4		<i>Df(1)18-80</i>	<i>Df(1)16-129</i>	
<i>l(1)19Ff</i>	19F5		<i>Df(1)HM44</i>	<i>Df(1)18-80</i>	
<i>l(1)19Fg</i>	19F6		<i>Df(1)17-351</i>	<i>Df(1)HM44</i>	

l(1)19Fb

phenotype: Pupal lethal; embryonic central nervous system (CNS) and peripheral nervous system (PNS) normal. Eggs derived from homozygous germ-line clones have variably defective chorions. The few embryos that develop are U-shaped owing to defective germ-band retraction; have severe head defects (Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)19Fb</i>¹	X ray	Lifschytz	<i>l(1)B214</i>	1, 3, 4, 5, 9	
<i>l(1)19Fb</i>²			<i>l(1)B214-1</i>	9	
<i>l(1)19Fb</i>³	EMS	Lifschytz	<i>l(1)YT16</i>	5	on <i>mal⁺Y</i>
<i>l(1)19Fb</i>⁴	EMS	Lefevre	<i>l(1)DA689</i>	2	P/AO
<i>l(1)19Fcut1</i>	EMS	Lifschytz	<i>l(1)YT57</i>	5	on <i>mal⁺Y</i>
<i>l(1)19Fe</i>¹			<i>l(1)M136</i>	5	on <i>y⁺Ymal⁺</i>
<i>l(1)19Fe</i>²			<i>l(1)NY20</i>	5	
<i>l(1)19Fe</i>³			<i>l(1)P329</i>	4, 5	
<i>l(1)19Fe</i>⁴	EMS	Lifschytz	<i>l(1)W4</i>	1, 3, 4, 5, 9	
<i>l(1)19Fe</i>⁵			<i>l(1)YT28</i>	5	on <i>mal⁺Y</i>
<i>l(1)19Fe</i>⁶			<i>l(1)YT31</i>	5	on <i>mal⁺Y</i>

^α 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 3 = Lifschytz and Falk, 1968, Mut. Res. 6: 235-44; 4 = Lifschytz and Falk, 1969, Mut. Res. 8: 147-55; 5 = Lifschytz and Jakobovitz, 1978, Mol. Gen. Genet. 161: 275-84; 6 = Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31; 7 = Schalet, 1986, Mutat. Res. 163: 115-44; 8 = Schalet and Lefevre, 1971, Chromosoma 44: 183-202; 9 = Schalet and Lefevre, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902; 10 = Schalet and Singer, 1972, DIS 46: 131-32.

l(1)19Ff

phenotype: Mutant embryos have no obvious defects of central nervous system (CNS), peripheral nervous system (PNS), or cuticle. Germ line clones lethal (Perrimon,

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Smouse, and Miklos, 1989, Genetics 121: 313-31).
alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)19F1¹</i>	X ray	Singer	<i>l(1)11P1</i>	7, 9, 10	
<i>l(1)19F1²</i>	X ray	Lifschytz	<i>l(1)A112</i>	1, 5, 6, 9, 10	L/L
<i>l(1)19F1³</i>	EMS	Lifschytz	<i>l(1)P252</i>		
<i>l(1)19F1⁴</i>	EMS	Lifschytz	<i>l(1)R-9-26</i>	4	
<i>l(1)19F1⁵</i>	EMS	Lifschytz	<i>l(1)M173</i>	5	on <i>mal⁺Y</i>
<i>l(1)19F1⁶</i>	X ray	Lefevre	<i>l(1)GF314</i>	1, 6	E-L/L
<i>l(1)19F1⁷</i>	spont	Schalet	<i>l(1)8-1</i>	6, 7	L/L
			<i>l(1)S-19F6</i>		
<i>l(1)19F1⁸</i>			<i>l(1)17-62</i>	6	E-L/L
<i>l(1)19Fg¹</i>	EMS	Baldwin	<i>l(1)LB14</i>	5, 8, 9	
<i>l(1)19Fg²</i>	EMS	Baldwin	<i>l(1)LB20</i>	1, 6, 8, 9	L-P/L
<i>l(1)19Fg³</i>	X ray	Lefevre	<i>l(1)C27</i>	1, 6	P/L
<i>l(1)19Fg⁴</i>	X ray	Lefevre	<i>l(1)KC14</i>	1	
<i>l(1)19Fg⁵</i>	X ray	Lefevre	<i>l(1)RF42</i>	1	<i>T(1;2)7A1-2;</i> <i>19F5-6,2L</i> (complex)
<i>l(1)19Fg⁶</i>	EMS	Lefevre	<i>l(1)DA618</i>	2, 6	L-P/L

^α 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 3 = Lifschytz and Falk, 1968, Mutat. Res. 6: 235-44; 4 = Lifschytz and Falk, 1969, Mutat. Res. 8: 147-55; 5 = Lifschytz and Yakobovitz, 1978, Mol. Gen. Genet. 161: 275-84; 6 = Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31; 7 = Schalet, 1986, Mutat. Res. 163: 115-44; 8 = Schalet and Lefevre, 1971, Chromosoma 44: 183-202; 9 = Schalet and Lefevre, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902; 10 = Schalet and Singer, 1972, DIS 46: 131-32.

DEFICIENCY MAP OF REGION 19

side	breakpoint	variant
left	18F4-5	<i>Df(1)mal8</i>
left	19A1	<i>Df(1)mal12</i>
		<i>amn</i>
left	19A2-3	<i>Df(1)mal11</i>
		<i>l(1)19Aa</i>
left	19A2-3	<i>Df(1)mal3</i>
left	19A2-3	<i>Df(1)mal17</i>
left	19A5	<i>Df(1)16-2-19</i>
left	19A5-6	<i>Df(1)mal10</i>
	19A3-5	<i>ot</i>
	19B1	<i>l(1)19Ba</i>
left	19B	<i>Df(1)HM44</i>
left	19B3	<i>Df(1)T2-4A</i>
	19B3	<i>sw</i>
		<i>l(1)19Ca</i>
	19C2-3	<i>mel</i>
left	19C3	<i>Df(1)mal22</i>
left	19C3	<i>Df(1)mal6</i>
right	19C4	<i>Df(1)T2-4A</i>
		<i>l(1)19Cb</i>
		<i>l(1)19Cc</i>
		<i>l(1)19Cd</i>
left	19D1	<i>Df(1)16-3-22</i>
left	19D2-3	<i>Df(1)16-3-35</i>
	19D3-E1	<i>mal</i>
left	19E2-3	<i>Df(1)N77</i>
right	19E1	<i>Df(1)mal17</i>
		<i>mell</i>
left	19E1	<i>Df(1)B12</i>
left	19E1-2	<i>Df(1)B57</i>
left	19E	<i>Df(1)GA37</i>
left	19E	<i>Df(1)GA40</i>
right	19D3	<i>Df(1)16-2-19</i>
right	19E1	<i>Df(1)mal8</i>
right	19E1	<i>Df(1)mal10</i>
right	19E1	<i>Df(1)mal11</i>
right	19E1	<i>Df(1)mal22</i>
left		<i>y+Ymal102</i>

side	breakpoint	variant
left	19E4	<i>run</i>
left		<i>Df(1)LB6</i>
		<i>y+Ymal171</i>
		<i>l(1)19Eb</i>
		<i>shakB</i>
left	19E	<i>Df(1)17-351</i>
left	19F	<i>Df(1)26B</i>
left	19E4-5	<i>Df(1)A118</i>
left		<i>Df(1)HC279</i>
left	19E4	<i>Df(1)LB7</i>
right	19E3-4	<i>Df(1)16-3-35</i>
	19E4-5	<i>l(1)19Ec</i>
left		<i>Df(1)17-489</i>
left	19E5	<i>Df(1)A53</i>
left	19E5-6	<i>Df(1)T2-14A</i>
		<i>l(1)19Ed</i>
		<i>if</i>
left	19E6-7	<i>Df(1)LB23</i>
left	19E6	<i>Df(1)Q539</i>
right		<i>Df(1)HM435</i>
	19E7-8	<i>vao</i>
left	19E8	<i>Df(1)D43L1</i>
left	19F	<i>Df(1)GA33</i>
left	19E8	<i>Df(1)S54</i>
right	19E8	<i>Df(1)A118</i>
right		<i>Df(1)HC279</i>
right		<i>Df(1)run1112</i>
right	19E7-8	<i>Df(1)T2-14A</i>
	19E8-F1	<i>unc</i>
left	19F1	<i>Df(1)16-129</i>
left	19E8-F1	<i>Df(1)54</i>
left	19F1	<i>Df(1)C74</i>
left	19F1-2	<i>Df(1)DCB1-35b</i>
left	19F1	<i>Df(1)VE969</i>
right	19F1	<i>Df(1)B57</i>
		<i>ifi</i>
		<i>l(1)19Fb</i>
left	19F	<i>Df(1)2/19B</i>
left	19F3	<i>Df(1)17-257</i>
left		<i>Df(1)18-80</i>
left	19F	<i>Df(1)GA104</i>
left	19F1-2	<i>Df(1)GE263</i>
		<i>flil</i>
right	19F3	<i>Df(1)16-129</i>
		<i>l(1)19Fe</i>
		<i>sol</i>
		<i>sig</i>
left	19F5	<i>Df(1)17-59</i>
left	19F	<i>Df(1)JC77</i>
left	19F	<i>Df(1)JA117</i>
right		<i>Df(1)18-80</i>
		<i>l(1)19Ff</i>
right	19F5-6	<i>Df(1)HM44</i>
		<i>l(1)19Fg</i>
right	19F	<i>Df(1)2/19B</i>
right	20A2	<i>Df(1)16.3.22</i>
right	19F	<i>Df(1)17-351</i>
right	19F	<i>Df(1)GA37</i>
right	19F	<i>Df(1)GA104</i>
right	19F	<i>Df(1)JA117</i>
right	19F6	<i>Df(1)Q539</i>
		<i>tuh</i>
left	20A	<i>Df(1)17-137</i>
left	20A1	<i>Df(1)JC4</i>
left		<i>Df(1)R19</i>
left		<i>Df(1)R29</i>
left		<i>Df(1)R37</i>
left		<i>Df(1)R45</i>
left		<i>Df(1)R48</i>
left	19F6-20A1	<i>y+Ymal126</i>

l(1)20A

Five lethally mutable loci identified, including named loci *eo*, *wap*, *intro*, and *uncl*.

locus	cytological		included in	excluded from	synonym
	location	location			
<i>l(1)20Aa</i>	20A1-2	<i>Df(1)B12</i>		<i>Df(1)Q539</i>	eo
<i>l(1)20Ab</i>	20A3	<i>Df(1)DCB1.35c</i>		<i>Df(1)B12</i>	wap
<i>l(1)20Ac</i>	20A	<i>Df(1)DCB1.35c</i>		<i>Df(1)B12</i>	intro
<i>l(1)20Ad</i>	20A				
<i>l(1)20Ae</i>	20A	<i>Df(1)16.2.13</i> <i>Df(1)EA113</i>		<i>Df(1)DCB1.35c</i>	uncl

allele origin discoverer synonym ref^α comments

***l(1)20Ad*¹** EMS Lifschytz *l(1)YT1* 1 on *mal*⁺*Y*

^α 1 = Lifschytz and Jakobovitz, 1978, Mol. Gen. Genet. 161: 275-84.

l(1)20B – l(1)20F

Nine lethally mutable loci identified, including *fog* in 20A, *stn* in 20B, *sph* and *su(f)* in 20E, and *bb* arbitrarily assigned to 20F.

locus	genetic		cytological		included in	excluded from	synonym
	location	location	location	location			
<i>l(1)20Ba</i>	1-65	20A5-B	<i>Df(1)JA27</i>	<i>Df(1)HF359</i>		<i>Df(1)HF359</i>	fog
<i>l(1)20Bb</i>	1-68.5	20A-B	<i>Df(1)17-439</i>	<i>Df(1)17-466</i>		<i>Df(1)17-466</i>	
<i>l(1)20Bc</i>			<i>Df(1)HF359</i>				stn
			<i>Df(1)17-439</i>				
<i>l(1)20Ca</i>		20B	<i>Df(1)17-252</i>	<i>Df(1)17-439</i>		<i>Df(1)17-439</i>	
			<i>Df(1)17-148</i>				
<i>l(1)20Cb</i>		20C-D	<i>Df(1)17-48</i>	<i>Df(1)17-252</i>		<i>Df(1)17-252</i>	
				<i>Df(1)GA131</i>		<i>Df(1)GA131</i>	
<i>l(1)20Ea</i>		20D-E	<i>Df(1)GA131</i>	<i>Df(1)16-185</i>		<i>Df(1)16-185</i>	sph
<i>l(1)20Eb</i>	1-65.9	20E	<i>Df(1)16-185</i>				su(f)
			<i>Df(1)13C3</i>				
<i>l(1)20Fa</i>	1-66.0	20F	<i>Df(1)yX15</i>	<i>Df(1)16-185</i>		<i>Df(1)16-185</i> <i>Df(1)13C3</i>	bb

allele origin discoverer synonym ref^α comments

***l(1)20Bb*¹** X ray Lefevre *l(1)A72* 2 L/L

***l(1)20Bb*²** EMS Lefevre *l(1)EA41* 3 L/L

***l(1)20Bb*³** HMS Kramers *l(1)HM1* 1

***l(1)20Bb*⁴** HMS Kramers *l(1)HM410* 1

***l(1)20Bb*⁵** HMS Kramers *l(1)HM425* 1

^α 1 = Kramers, Schalet, Paradi, and Huizer-Hoogteyting, 1983, Mut. Res. 107: 187-201; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 3 = Lefevre, 1981, Genetics 99: 461-80.

l(1)20Ca

phenotype: Embryonic-larval lethal with no cuticular, central nervous system (CNS), or peripheral nervous system (PNS) defects discernable in mutant embryos. *l(1)20Ca*¹ exhibits no maternal effect, whereas homozygous germ-line clones of *l(1)20Ca*⁸ produce lethal embryos with slight rescue by paternally supplied normal X; head involution and segmentation defective in such embryos (Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)20Ca</i>¹	X ray	Singer	<i>l(1)13E3</i>	2, 4, 5, 7	E/NME
<i>l(1)20Ca</i>²	EMS	Lifschytz	<i>l(1)P19</i>	4, 5, 7, 8	
<i>l(1)20Ca</i>³	EMS	Lifschytz	<i>l(1)P431</i>	4	
<i>l(1)20Ca</i>⁴	EMS	Lifschytz	<i>l(1)YT65</i>	5	on <i>mal</i> ⁺ <i>Y</i>
<i>l(1)20Ca</i>⁵	X ray	Lefevre	<i>l(1)GE201</i>	2	
<i>l(1)20Ca</i>⁶	X ray	Lefevre	<i>l(1)HC137</i>	2	

allele origin discoverer synonym ref^α comments

***l(1)20Ca*⁷** X ray Lefevre *l(1)N138* 2

***l(1)20Ca*⁸** X ray Lefevre *l(1)S60* 2 E-L1/MER

^α 1 = Kramers, Schalet, Paradi, and Huizer-Hoogteyting, 1983, Mut. Res. 107: 187-201; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Lifschytz and Falk, 1969, Mut. Res. 8: 147-55; 5 = Lifschytz and Jakobovitz, 1978, Mol. Gen. Genet. 161: 275-84; 6 = Schalet and Finnerty, 1968, DIS 3: 128; 7 = Schalet and Lefevre, 1973, Chromosoma 44: 183-202; 8 = Schalet and Lefevre, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902; 9 = Schalet and Singer, 1971, DIS 46: 131-32.

l(1)20Cb

phenotype: *l(1)20Cb*² is larval and the weaker *l(1)20Cb*¹³ is a polyphasic lethal; central nervous system (CNS) and peripheral nervous system (PNS) development appears normal. The stronger allele is lethal in homozygous germ-line clones; the weaker allele is not (Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(1)20Cb</i>¹	neutron	Mūnoz	<i>l(1)17-347</i>	8	
<i>l(1)20Cb</i>²	X ray	Schalet	<i>l(1)20</i>	2, 7, 8, 9	L/L
<i>l(1)20Cb</i>³		Singer	<i>l(1)26E1</i>	8	
<i>l(1)20Cb</i>⁴		Novitski	<i>l(1)97</i>	6	
<i>l(1)20Cb</i>⁵		Novitski	<i>l(1)137</i>	6	
<i>l(1)20Cb</i>⁶	EMS	Lifschytz	<i>l(1)Q463</i>	4, 5, 7, 8	
<i>l(1)20Cb</i>⁷	EMS	Lifschytz	<i>l(1)R-9-14</i>	5	
<i>l(1)20Cb</i>⁸	EMS	Lifschytz	<i>l(1)YT47</i>	5	on <i>mal</i> ⁺ <i>Y</i>
<i>l(1)20Cb</i>⁹	X ray	Lefevre	<i>l(1)JA57</i>	2	
<i>l(1)20Cb</i>¹⁰	EMS	Lefevre	<i>l(1)DA565</i>	3	
<i>l(1)20Cb</i>¹¹	EMS	Lefevre	<i>l(1)DC791</i>	3	
<i>l(1)20Cb</i>¹²	EMS	Lefevre	<i>l(1)EA9</i>	3	
<i>l(1)20Cb</i>¹³	EMS	Lefevre	<i>l(1)VA97</i>	3	L3-A/NME
<i>l(1)20Cb</i>¹⁴	EMS	Lefevre	<i>l(1)VA151</i>	3	
<i>l(1)20Cb</i>¹⁵	EMS	Lefevre	<i>l(1)VE641</i>	3	
<i>l(1)20Cb</i>¹⁶	HMS	Kramers	<i>l(1)HM24</i>	1	

^α 1 = Kramers, Schalet, Paradi, and Huizer-Hoogteyting, 1983, Mut. Res. 107: 187-201; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Lifschytz and Falk, 1969, Mut. Res. 8: 147-55; 5 = Lifschytz and Jakobovitz, 1978, Mol. Gen. Genet. 161: 275-84; 6 = Schalet and Finnerty, 1968, DIS 3: 128; 7 = Schalet and Lefevre, 1973, Chromosoma 44: 183-202; 8 = Schalet and Lefevre, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902; 9 = Schalet and Singer, 1971, DIS 46: 131-32.

DEFICIENCY MAP OF REGION 20

side	breakpoint	variant
right	19F	<i>Df(1)2/19B</i>
right	19F	<i>Df(1)17-351</i>
right	19F	<i>Df(1)GA37</i>
right	19F	<i>Df(1)GA104</i>
right	19F	<i>Df(1)JA117</i>
right	19F6	<i>Df(1)Q539</i>
		tuh
left	20A	<i>Df(1)17-137</i>
left	20A1	<i>Df(1)JC4</i>
left		<i>Df(1)JC12</i>
left		<i>Df(1)JR19</i>
left		<i>Df(1)R29</i>
left		<i>Df(1)R37</i>
left		<i>Df(1)R45</i>
left		<i>Df(1)R48</i>
left	19F6-20A1	<i>y+Ymal126</i>
	20A1-2	eo
left	20A3	<i>Df(1)13C3</i>

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side	breakpoint	variant	side	breakpoint	variant
left	20A1-2	<i>Df(1)16-2-13</i>	left		<i>Df(1)K5</i>
left	20A	<i>Df(1)17-439</i>	left		<i>Df(1)R6</i>
left	20A	<i>Df(1)17-466</i>	left		<i>Df(1)R8A</i>
left		<i>Df(1)A122</i>	right	20B-C	<i>Df(1)17-148</i>
left		<i>Df(1)DCB1-35c</i>	right	20C	<i>Df(1)17-252</i>
left		<i>Df(1)GA22</i>			I(1)20Cb
left		<i>Df(1)HM455</i>	left		<i>Df(1)GA131</i>
left		<i>Df(1)R3</i>	left		<i>Df(1)R17</i>
left		<i>Df(1)R13</i>	left	20D1-2	<i>Df(1)yx15</i>
left		<i>Df(1)R14</i>	left	20D-F	<i>Df(1)X1</i>
left		<i>Df(1)R15</i>	right	20C-D	<i>Df(1)HM430</i>
left		<i>Df(1)R20</i>			sph
left		<i>Df(1)R21</i>	left	20D-E	<i>Df(1)17-87</i>
left		<i>Df(1)R22</i>	left	20E	<i>Df(1)16-185</i>
left		<i>Df(1)R24</i>	left		<i>Df(1)R8</i>
left		<i>Df(1)R27</i>	left		<i>Df(1)R16</i>
left		<i>Df(1)R28</i>	left		<i>Df(1)R18</i>
left		<i>Df(1)R31</i>			suf(f)
left		<i>Df(1)R32</i>			B^SY
left		<i>Df(1)R33</i>	right		<i>Df(1)13C3</i>
left		<i>Df(1)R35</i>	right	20E-F	<i>Df(1)16-185</i>
left		<i>Df(1)R38</i>	right	20F	<i>Df(1)16-185</i>
left		<i>Df(1)R40</i>	right	20E-F	<i>Df(1)17-59</i>
left		<i>Df(1)R41</i>	right	20E-F	<i>Df(1)17-137</i>
left		<i>Df(1)R44</i>	right	20E-F	<i>Df(1)17-148</i>
left		<i>Df(1)R47</i>	right	20E-F	<i>Df(1)17-408</i>
left		<i>Dp(1;f)3</i>	right		<i>Df(1)17-489</i>
right	20A2	<i>Df(1)16-3-22</i>	right	20E-F	<i>Df(1)D43L1</i>
right	20A1-2	<i>Df(1)17-257</i>	right	20E-F	<i>Df(1)DCB1-35b</i>
right		<i>Df(1)26B</i>	right	20E-F	<i>Df(1)EA113</i>
right	20A1-2	<i>Df(1)B12</i>	right	20E-F	<i>Df(1)GA22</i>
right	20A1-2	<i>Df(1)GE263</i>	right	20E-F	<i>Df(1)GA40</i>
right		<i>Df(1)JC77</i>	right		<i>Df(1)GA131</i>
right	20A2-3	<i>Df(1)LB6</i>	right	20E-F	<i>Df(1)HF359</i>
right	20A2	<i>Df(1)S54</i>	right		<i>Df(1)JA27</i>
right	20A2	<i>Df(1)mal6</i>	right	20E-F	<i>Df(1)JC4</i>
right	20A1-2	<i>Df(1)A53</i>	right		<i>Df(1)JC12</i>
		wap	right		<i>Df(1)LB7</i>
		intro	right	20E-F	<i>Df(1)LB23</i>
left	20A	<i>Df(1)17-408</i>	right	20E-F	<i>Df(1)mal3</i>
left	20A	<i>Df(1)A209</i>	right	20E-F	<i>Df(1)mal12</i>
left	20A	<i>Df(1)EA113</i>	right		<i>Df(1)N77</i>
left		<i>Df(1)R12</i>	right	20E-F	<i>Df(1)suf(f)5A</i>
left		<i>Df(1)R25</i>	right		<i>Df(1)R2</i>
left		<i>Df(1)R26</i>	right		<i>Df(1)R3</i>
left		<i>Df(1)R46</i>	right		<i>Df(1)R6</i>
right		<i>Df(1)A122</i>	right		<i>Df(1)R7</i>
right	20A4	<i>Df(1)C74</i>	right		<i>Df(1)R8</i>
right		<i>Df(1)DCB1-35c</i>	right		<i>Df(1)R12</i>
		uncl	right		<i>Df(1)R13</i>
left	20B	<i>Df(1)17-252</i>	right		<i>Df(1)R14</i>
left	20B1	<i>Df(1)GA42</i>	right		<i>Df(1)R15</i>
left	20A-B	<i>Df(1)HM430</i>	right		<i>Df(1)R16</i>
left		<i>Df(1)JA27</i>	right		<i>Df(1)R17</i>
left		<i>Df(1)R2</i>	right		<i>Df(1)R18</i>
left		<i>Df(1)R7</i>	right		<i>Df(1)R19</i>
left		<i>Df(1)R10</i>	right		<i>Df(1)R23</i>
left		<i>Df(1)R23</i>	right		<i>Df(1)R26</i>
left		<i>Df(1)R30</i>	right		<i>Df(1)R28</i>
left		<i>Df(1)R36</i>	right		<i>Df(1)R29</i>
left		<i>Df(1)R42</i>	right		<i>Df(1)R31</i>
left		<i>Df(1)R43</i>	right		<i>Df(1)R32</i>
left	20A4-5	<i>Df(1)yx5</i>	right		<i>Df(1)R33</i>
right	20A4	<i>Df(1)16-2-13</i>	right		<i>Df(1)R37</i>
right		<i>Df(1)GA33</i>	right		<i>Df(1)R38</i>
		fog	right		<i>Df(1)R41</i>
		B^SY	right		<i>Df(1)R44</i>
left	20A-B	<i>Df(1)HF359</i>	right		<i>Df(1)R45</i>
left	20A3	<i>Df(1)suf(f)5A</i>			bb
right	20B	<i>Df(1)17-466</i>	right		<i>Df(1)R46</i>
		I(1)20Bb	right		<i>Df(1)R22</i>
		stn	right	20F	<i>Df(1)17-87</i>
right	20B	<i>Df(1)17-439</i>	right	20F	<i>Df(1)A209</i>
right	20B-D	<i>Df(1)54</i>	right	20F	<i>Df(1)GA42</i>
		I(1)20Ca	right	20F	<i>Df(1)GA90</i>
left	20C	<i>Df(1)GA90</i>	right		<i>Df(1)K5</i>
			right		<i>Df(1)R1</i>

side	breakpoint	variant
right		<i>Df(1)R8A</i>
right		<i>Df(1)R10</i>
right		<i>Df(1)R20</i>
right		<i>Df(1)R21</i>
right		<i>Df(1)R24</i>
right		<i>Df(1)R25</i>
right		<i>Df(1)R27</i>
right		<i>Df(1)R30</i>
right		<i>Df(1)R35</i>
right		<i>Df(1)R36</i>
right		<i>Df(1)R40</i>
right		<i>Df(1)R42</i>
right		<i>Df(1)R43</i>
right		<i>Df(1)R47</i>
right		<i>Df(1)R48</i>
right	20F	<i>Df(1)VE696</i>
right	20F	<i>Df(1)X1</i>
right	20F	<i>Df(1)yX15</i>
right	20F	<i>Df(1)yX5</i>

l(1)48j: see *mys*

**l(1)52*

location: 1- (to the right of *B*).

discoverer: Sobels.

references: Gloor, 1962, Rev. Suisse Zool. 69: 409-63 (fig.).

phenotype: Larvae die in second instar. Growth retarded. Histology of nervous system, testes, and imaginal disks abnormal. Number of nuclei in salivary glands increased. Amino acids and peptides increased. Transplanted testes and imaginal disks autonomously lethal. RK2.

l(1)55

location: 1-0.

discoverer: Burdick, 55a.

references: 1956, DIS 30: 69. 1957, DIS 31: 86.

phenotype: Rare surviving males misinterpreted by Burdick as crossovers to the left of *y* leading him to place *l(1)55* at -0.6. Heterozygote claimed to have viability about 1.5 times normal. Not allelic to *l(1)IAc¹*. RK2.

l(1)55a

location: 1-5.5.

origin: Induced by ethyl methanesulfonate.

references: Lifschytz, 1978, Dev. Biol. 66: 571-78 (fig.).

phenotype: Temperature-sensitive lethal; few *l(1)55*-bearing males develop at 27°; lethal-bearing males surviving either temperature regime display arrest of spermatogenesis. At 18-22° a variable number of cysts continues to support gonial proliferation producing giant cysts with hundreds of spermatogonia, most of which degenerate.

l(1)63

location: 1-0.3.

origin: Induced by ethyl methanesulfonate.

references: Lifschytz, 1978, Dev. Biol. 66: 571-78 (fig.).

phenotype: Temperature-sensitive lethal. Surviving *l(1)63*-bearing males raised under permissive conditions exhibit precocious spermatocyte maturation in some cysts of four and eight cells; other cysts produce sixteen primary spermatocytes. Shifting such males to 27° causes mitotic arrest leading to disappearance of gonial cells within two or three days and of primary spermatocytes after four or five days. Homozygous females raised at

18° phenotypically normal but sterile. Mutant tergites found in gynandromorphs raised at 27°; half-and-half gynandromorphs formed from embryos shifted from permissive to restrictive conditions 48 hr after oviposition; facet number in mutant eyes and bristles in mutant tissue reduced, indicating mitotic arrest.

l(1)76: see *dor^{l2}*

**l(1)184*

location: 1- (rearrangement).

origin: X ray induced.

discoverer: Lindsley, Edington, and Von Halle.

references: 1960, Genetics 45: 1649-70.

phenotype: Almost completely lethal. The few survivors have dark, rough eyes. RK2A.

cytology: Associated with *T(1;3)l-184 = T(1;3)18A;81*.

**l(1)272-13*

location: 1- (rearrangement).

origin: X ray induced.

discoverer: Demerec, 1940.

references: Sutton, 1943, Genetics 28: 210-217.

phenotype: Lethal. *l(1)272-13/sc* is scute. RK2A.

cytology: Associated with *In(1)l-272-13 = In(1)1A6-B1;11A7-8;11F2-12A1;18A4-B1*.

l(1)1074^{ts}

location: 1-16.3.

origin: Induced by ethyl methanesulfonate.

references: Datson and Brink, 1978, Aust. J. Biol. Sci. 31: 73-91.

Brink, 1979, Aust. J. Biol. Sci. 32: 597-606.

phenotype: Temperature-sensitive mutant of a gene whose product is required several times during development. The temperature-sensitive periods are towards the end of oogenesis in homozygous females, from the sixth to twelfth hour of embryogenesis and again during larval and early pupal development in mutant offspring. Embryological abnormalities first evident during gastrulation and eventually result in the breakdown of organogenesis and the absence of normal muscular contractions. Fragments of mutant gastrulae transplanted into larvae fail to show evidence of the cuticular differentiation seen in transplanted wild-type fragments. X-ray-induced mutant clones formed normally in abdominal histoblasts, but didn't survive in imaginal discs.

l(1)2269

location: 1-unmapped.

origin: Induced by ethyl methanesulfonate.

references: Gateff, 1978, Biol. Rev. 53: 123-68.

1978, Science 200: 1448-59.

phenotype: Causes malignant neuroblastoma of the adult optic neuroblasts and ganglion mother cells; also displays intermediate imaginal disc neoplasm with compact mode of growth.

l(1)3063

location: 1-0.8 (0/569 recombinants with *pn*).

origin: X ray induced.

references: Imaizumi, 1967, DIS 42: 78.

phenotype: Embryonic lethal; dies in second half of embryogenesis.

l(1)Ab: see *r*

***l(1)Ac*¹**

origin: X ray induced simultaneously with *sc*^{J1}.
discoverer: Jacobs-Muller.
references: Muller, 1932, Proc. Intern. Congr. Genet., 6th., Vol. 1: 225.
 Muller, 1935, Genetica 17: 237-52.
phenotype: Lethal. Not cell lethal (Ephrussi, 1934, Proc. Nat. Acad. Sci. USA 20: 420-22). One recorded surviving male had rough eyes and was sterile. RK2A.
cytology: Probably in 1A6. Associated with *In(1)sc*^{J1} = *In(1)1A4-5;1B4-5* (Muller, Prokofyeva, and Raffel, 1935, Nature 135: 253-55).

l(1)adi: lethal (1) adult

A series of ethyl-methanesulfonate-induced mutants identified by virtue of their reduced life span upon being shifted from their developmental temperature of 22° to 29° shortly after eclosion. Most are temperature-sensitive lethals with preimaginal as well as adult lethal phases; as such they identify a subset of lethally mutable genes whose functions are required both during development and for the maintenance of adult viability; in general, they have not been tested for allelism with unconditional lethals nearby. Others are unconditionally lethal in the adult stage and are either completely viable during development or are semilethal during development at one or both temperatures.

locus	genetic location	synonym	ref ^α	preimaginal phenotype	adult phenotype	time of death at 29° ^β
<i>l(1)adi1</i>	1-21.3	<i>addA</i> <i>J1649</i>	2, 3, 4, 6	ts E	ts paralysis	.25-.75
<i>l(1)adi2</i>	1-0.0	<i>addB</i>	2, 3, 4	+	hypoactivity	3-7
<i>l(1)adi3</i>	<i>y-cv</i>		3, 5	ts L2-3	ts female sterile	6-8
<i>l(1)adi4</i>	<i>cv-v</i>		3, 5	ts semilethal		4-7
<i>l(1)adi5</i>	<i>cv-v</i>		3	ts P	stress sensitive	7-21
<i>l(1)adi6</i>	<i>cv-v</i>		3	ts semilethal	<i>sc</i> -like, rough eyes	3-28
<i>l(1)adi7</i>	<i>v-f</i>		3	+	female sterile	2-12
<i>l(1)adi8</i>	<i>v-f</i>		3	ts semilethal		3-6
<i>l(1)adi9</i>	<i>v-f</i>		3	ts semilethal		2-10
<i>l(1)adi10</i>	<i>v-f</i>		3	ts semilethal	ts stress sensitive female sterile	6-11
<i>l(1)adi11</i>	<i>v-f</i>		3	ts L1	stress sensitive	1-4
<i>l(1)adi12</i>	<i>f-car</i>		3	ts semilethal		2-10
<i>l(1)adi13</i>	<i>car-sfa</i>		3, 5	ts L2-P	stress sensitive	4-8
<i>l(1)adi14</i>	<i>car-sfa</i>		3, 5	ts L1-2	ts	3-4
<i>l(1)adi15</i>	<i>car-sfa</i>		3	ts semilethal	ts	16-29
<i>l(1)adi16</i>	1-50.8	<i>firdl</i>	3, 5	ts E-L3	ts swollen abdomen proboscis extended flight reduced	2-20

^α 1 = Homyk and Sheppard, 1977, Genetics 87: 95-104; 2 = Homyk, Sinclair, Wong, and Suzuki, 1979, Genetics 91: s49-50; 3 = Homyk, Sinclair, Wong, and Grigliatti, 1986, Genetics 113: 367-89; 4 = Homyk, Szidonia, and Suzuki, 1980, Mol. Gen. Genet. 177: 553-65; 5 = Laffelaar and Grigliatti, 1984, Dev. Genet. 4: 199-210; 6 = Shellenbarger and Cross, 1979, Dev. Biol. 71: 308-22.

^β The period during which adults die in days after eclosion at 29°; control flies survive well beyond 25 days at that temperature.

l(1)adi1

phenotype: Temperature shift and pulse experiments delineate two major temperature sensitive periods, one during the second half of embryogenesis and the other at the time of pupation. Fate mapping revealed separate mesodermal foci for leg paralysis, and examination of embryos developing at 29° indicated abnormalities in muscle development (Homyk, Sinclair, Wong, and Grigliatti, 1986, Genetics 113: 367-89).
cytology: Located in 7D11-22 based on its inclusion in

Df(1)HA11 (Lefevre).

l(1)adi2

phenotype: Fate mapping suggests presence of bilateral domineering foci located in or near the subesophageal ganglion responsible for both hypoactivity and lethality at 29° (Homyk, Sinclair, Wong, and Grigliatti, 1986, Genetics 113: 367-89).

l(1)adi3

phenotype: Severely debilitated in phototaxis and geotaxis shortly after shift of adults raised at 22° to 29° (Laffelaar and Grigliatti, 1984, Dev. Genet. 4: 199-210).

l(1)adi4

phenotype: Gradual loss of phototaxis and geotactic abilities during three days following shift of adults raised at 22° to 29° (Laffelaar and Grigliatti, 1984, Dev. Genet. 4: 199-210).

l(1)adi13

phenotype: Motor, geotactic, and phototactic behavior severely debilitated at 22°, but lifespan shortened only when shifted to 29° (Laffelaar and Grigliatti, 1984, Dev. Genet. 4: 199-210).

l(1)adi16

phenotype: The normal pattern of behavioral loss associated with aging in *Drosophila* was contracted exactly in parallel with the contraction of the life span in *l(1)adi16*¹ flies, suggesting that mutations at this locus accelerate aging (Laffelaar and Grigliatti, 1984, Dev. Genet. 4: 199-210). *l(1)adi16*³ (formerly *firdl*) adults show weak flight, general activity and reduced life span at 25°.

alleles:

allele	origin	synonym	ref ^α	preimaginal phenotype	adult phenotype	time of death ^β
<i>l(1)adi16</i> ¹	EMS	<i>DC836</i>	2, 3	ts E-L2	ts swollen abdomen proboscis extended	5-8
<i>l(1)adi16</i> ²	EMS	<i>DC359</i>	2, 3	ts L2-3		2-3
<i>l(1)adi16</i> ³	EMS	<i>firdl</i>	1, 2	ts L2-3	ts swollen abdomen proboscis extended; flight reduced	10-20

^α 1 = Homyk and Sheppard, 1977, Genetics 87: 95-104; 2 = Homyk, Sinclair, Wong, and Grigliatti, 1986, Genetics 113: 367-89; 3 = Shellenbarger and Cross, 1979, Dev. Biol. 71: 308-22.

^β The period during which adults die in days after eclosion at 29°; control flies survive well beyond 25 days at that temperature.

l(1)AL: lethal (1) Adult Lifespan

X-ray-induced mutations at three different loci (by complementation testing), which have not been mapped (Gould and Clark, 1977, J. Exp. Gerontol. 12: 107-12).

locus	mean lifespan (days)	
	males	females
<i>l(1)AL1</i>	34.9 ± 1.2	30.1 ± 1.4
<i>l(1)AL2</i>	12.7 ± 0.2	14.5 ± 0.3
<i>l(1)AL3</i>	28.7 ± 0.4	24.4 ± 0.4
+	68.5 ± 2.2	64.2 ± 2.1

l(1)br: see *br*

l(1)C

location: 1-6 (between *ec* and *bi*).
origin: Spontaneous in *sc t*² *vs sl B* chromosome.
discoverer: Muller, 20j.
references: 1928, Genetics 13: 279-357.

phenotype: Dies as late embryo or, more commonly, as first-instar larva (Brehme, 1937, Am. Naturalist 71: 567). RK2A.

cytology: Associated with the left breakpoint of *In(1)Cl* = *In(1)4A5-B1;17A6-B1*.

l(1)carot: lethal(1) carnation to outheld

location: A series of lethally mutable loci between *car* and *ot*. Some are likely to be alleles of *l(1)18Fa* or *l(1)19Aa*.

references: Schalet, 1968, DIS 42: 64.

Schalet and Finnerty, 1968, DIS 42: 128-29.

locus	genetic location	origin	discoverer	synonym
<i>l(1)carot1</i>				<i>l(1)1</i>
<i>l(1)carot2</i>	<i>car-l(1)carot3</i>		Schalet, 1961	<i>l(1)5</i>
<i>l(1)carot3</i>	<i>M(1)n-ot</i>		Schalet, 1961	<i>l(1)34</i>
<i>l(1)carot4</i>				<i>l(1)77</i>
<i>l(1)carot5</i>				<i>l(1)106</i>
<i>l(1)carot6</i>				<i>l(1)412</i>
<i>l(1)carot7</i>			Kaplan	<i>l(1)A11</i>
<i>l(1)carot8</i>			Kaplan	<i>l(1)A12</i>
<i>l(1)carot9</i>			Kaplan	<i>l(1)B4</i>
<i>l(1)carot10</i>			Himoe	<i>l(1)D4313</i>
<i>l(1)carot11</i>			Kaplan	<i>l(1)DCB2-35b</i>
<i>l(1)carot12</i>			Novitski	<i>l(1)EN10</i>
<i>l(1)carot13</i>	1-62.7	spont	Bridges, 1923	<i>M(1)18C</i>
<i>l(1)carot14</i>	1-63.8	EMS	Wright	<i>l(1)TW5</i>

allele	origin	discoverer	synonym	ref ^α
<i>l(1)carot14 cs1</i>	EMS	Wright	<i>l(1)TW5^{cs}</i>	1, 3
<i>l(1)carot14 cs2</i>	EMS	Mayoh	<i>l(1)HM5</i>	2
<i>l(1)carot14 cs3</i>	EMS	Mayoh	<i>l(1)HM6</i>	2
<i>l(1)carot14 cs4</i>	EMS	Mayoh	<i>l(1)HM15</i>	2

^α 1 = Falke and Wright, 1975, Genetics 81: 655-82; 2 = Mayoh and Suzuki, 1973, Can. J. Genet. Cytol. 151: 237-54; 3 = Wright, 1973, Mol. Gen. Genet. 122: 101-18.

l(1)crn: see *crn*

l(1)DC: lethal (1) of David Cross

Twelve complementing heat-sensitive lethals which develop at 22° but not at 29°; phenotypic information available on three.

discoverer: Cross.

references: Shellenbarger and Cross, 1979, Dev. Biol. 71: 308-22.

l(1)DC371

phenotype: Males raised at 22° fertile, but become sterile after eight days at 28°. Sterile males have motile sperm, which are not transmitted to females.

l(1)DC836

phenotype: Males raised at 22° survive, but die when held at 28° following eclosion.

l(1)DC1112

phenotype: Males raised at 22° eclose but die prematurely at either permissive or restrictive temperature.

l(1)dd: lethal (1) discs degenerate

A group of late larval-early pupal lethals that exhibit degeneration of imaginal discs at the normal time of pupation. None of those tested is able to survive in male tissue in gynandromorphs (all but *dd10* and *dd11* tested). All have been found by Baker, Smith, and Gatti to be mitotic mutants; they argue that the maternal genotype supports cell division in the embryo, and that disc proli-

feration represents the first instance of zygotically controlled cell division; thus the defects in these mutants do not affect phenotype until late in development (Baker, Smith, and Gatti, 1982, Proc. Nat. Acad. Sci. USA 79: 1205-09; Gatti, Pimpinelli, Bove, Baker, Smith, Carpenter, and Ripoll, Proc. Int. Cong. Genet. 15th, 1983, Vol. 3, pp. 193-204; Gatti and Baker, 1989, Genes Dev. 3: 438-53).

locus	location	synonym	ref ^α	lethal phase	mitotic phenotype
<i>l(1)ddl</i>	1-13.7	<i>mus105</i> <i>l(1)d.deg.-1</i>	1, 2, 3, 6	LP	chromosome aberrations, euchromatic breaks and exchanges
<i>l(1)dd2</i>	1-25.7	<i>l(1)d.deg.-2</i>	5	LP	
<i>l(1)dd3</i> ^β	1-27.5	<i>l(1)d.deg.-3</i>	1, 2, 3, 6	P	hypercontracted chromosomes, no anaphase, polyploid cells
<i>l(1)dd4</i> ^β	1-44.4	<i>l(1)d.deg.-4</i>	1, 2, 3, 6	P	metaphase block, colchicine-like metaphases, polyploid cells
<i>l(1)dd9</i> ^β	1-29.7	<i>l(1)d.deg.-9</i>	1, 2, 3, 4	LP	condensation abnormal, chromosomes swollen
<i>l(1)dd10</i> ^β	1-46.7	<i>l(1)d.deg.-10</i>	1, 2, 3, 4	LP	hypercontracted chromosomes, no anaphase, polyploid cells
<i>l(1)dd11</i> ^β	1-27.2	<i>l(1)d.deg.-11</i>	1, 2, 3, 4	LP	highly polyploid cells, many anaphases, some multipolar
<i>l(1)dd12</i> ^β	1-65.8	<i>l(1)d.deg.-12</i>	1, 2, 3, 4, 5	L	irregular condensation of chromosomes, polyploids

^α 1 = Baker, Smith, and Gatti, 1982, Proc. Nat. Acad. Sci. USA 79: 1205-09; 2 = Gatti and Baker, 1989, Genes Dev. 3: 438-53; 3 = Gatti, Pimpinelli, Bove, Baker, Smith, Carpenter, and Ripoll, Proc. Int. Cong. Genet. 15th, 1983, Vol. 3, pp. 193-204; 4 = Kiss, Bencze, Fekete, Fodor, Gausz, Maróy, Szabad, and Szidonya, 1976, Theor. Appl. Genet. 48: 217-226; 5 = Kiss, Szabad, and Major, 1978, Mol. Gen. Genet. 164: 77-83; 6 = Stewart, Murphy, and Fristrom, 1972, Dev. Biol. 27: 71-83.

^β Fuller discussion follows.

l(1)dd3

phenotype: Discs missing or degenerate; mitotic index of larval ganglion cells three times that of controls; chromosomes highly condensed as seen in colchicine-treated cells; 20% of metaphases polyploid; chromosome fragmentation common; no anaphase figures seen (Gatti and Baker, 1989, Genes Dev. 3: 438-53). Homozygous cells in ovary lethal (Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14).

l(1)dd4

phenotype: Discs missing or degenerate; mitotic index of larval ganglion cells three times that of controls; 20% of metaphases polyploid; very few anaphase figures seen. No maternal effect of homozygosis in ovarian clones on phenotype of offspring (Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14). *l(1)dd4^S* recovered as a mutation on the paternal X in a cross of homozygous *mei-9* females by normal males [isolated as *l(1)08/20*]. Ordinarily dies just prior to eclosion, but in dry cultures, both males and females eclose and have good viability, but are almost completely sterile. Wings are held up, some bristles on head and thorax (especially scutellars) are absent; abdomen mutant effects similar to those of *bb*. Females heterozygous for *l(1)dd4^S* and a deficiency are almost completely lethal.

cytology: Placed in 12A-C based on the failure of *Y^{PX}X^{DB166}X^{PY}DB136* to complement the lethality of *l(1)dd4^S*. *Dp(1;Y)g⁺* covers lethal, sterile, and visible effects in males; *Dp(1 f)LJ9* covers lethal effect, but only

partially covers visible effects.

l(1)dd9

phenotype: Discs missing or degenerate; mitotic index of larval ganglion reduced; chromosomes unevenly condensed, appear fuzzy; sister chromatids often closely apposed; heterochromatic regions elongated; tetraploid cells and some chromosome breakage seen (Gatti and Baker, 1989, Genes Dev. 3: 438-53). No maternal effect of homozygosis in ovarian clones on phenotype of offspring (Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14).

l(1)dd10

phenotype: Discs missing or degenerate; mitotic index of larval ganglion cells three times that of controls; mitotic chromosomes more highly condensed than normal; 30% of metaphases polyploid; chromosome fragmentation common; no anaphase figures seen (Gatti and Baker, 1989, Genes Dev. 3: 438-53). Homozygous cells in ovary lethal (Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14).

l(1)dd11

phenotype: Discs missing or degenerate; mitotic index of larval ganglion cells and chromosome condensation normal; 43.5% of metaphases polyploid; cells containing 500 to 1000 chromosomes encountered; some chromosome fragmentation seen (Gatti and Baker, 1989, Genes Dev. 3: 438-53). Homozygous cells in ovary lethal (Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14).

l(1)dd12

phenotype: Discs missing or degenerate; mitotic index of larval ganglion cells reduced; chromosomes swollen and unevenly condensed; few metaphases polyploid; no chromosome fragmentation seen (Gatti and Baker, 1989, Genes Dev. 3: 438-53). Homozygous cells in ovary lethal (Perrimon, Engstrom, and Mahowald, 1984, Dev. Biol. 105: 404-14).

l(1)dh1: lethal (1) discs heterogeneous

location: 1-27.5.

origin: Induced by ethyl methanesulfonate.

synonym: *l(1)d.het.-1*.

references: Kiss, Bencze, Fekete, Fodor, Gausz, Maróy, Szabad, and Szidonya, 1976, Theor. Appl. Genet. 48: 217-26.

phenotype: Pupal lethal; imaginal leg discs small, remaining imaginal discs normal in size and structure.

l(1)dh2

location: 1-57.

origin: Induced by ethyl methanesulfonate.

synonym: *l(1)d.het.-2*.

references: Kiss, Bencze, Fekete, Fodor, Gausz, Maróy, Szabad, and Szidonya, 1976, Theor. Appl. Genet. 48: 217-26.

phenotype: Pupal lethal; leg discs degenerated; remaining imaginal discs of normal size and structure. Chromosomes in larval ganglion cells irregularly condensed, often displaying coiled and banded appearance; in addition, chromatid breaks and extremely hyperploid or tetraploid cells are frequently observed (Gatti, Pimpinelli, Bove, Baker, Smith, Carpenter, and Ripoll, Proc. Int.

Cong. Genet. 15th, 1983, Vol. 3, pp. 193-204; Gatti and Baker, 1989, Genes Dev. 3: 438-53).

l(1)dn: lethal (1) discs normal

A group of late larval and early pupal lethals in which the imaginal discs appear normal at the time that pupation normally occurs. The ability of the discs of these mutants to differentiate in a normal hormonal milieu has been examined in both gynandromorphs and when transplanted into larvae that are then allowed to undergo metamorphosis.

Mutant	genetic location	synonym	ref ^α	PHENOTYPE ^β		
				A	B	C
<i>l(1)dn1</i>	1-0.1	<i>npr1</i> <i>l(1)d.norm.-1</i>	1	±	.05	L
<i>l(1)dn2</i>	1-1.4-11.9	<i>l(1)d.norm.-2</i>	1	±	0	L
<i>l(1)dn3</i>	1-4.1-10.6	<i>l(1)d.norm.-3</i>	1	-	0	L/P
<i>l(1)dn4</i>	1-14.5	<i>l(1)d.norm.-4</i>	1	+	.23	P
<i>l(1)dn5</i>		<i>l(1)d.norm.-5</i>	1	+	.11	L/P
<i>l(1)dn6</i>	1-56	<i>l(1)d.norm.-6</i>	1	-	0	P
<i>l(1)dn7</i>	1-57	<i>l(1)d.norm.-7</i>	1	-	.60	P
<i>l(1)dn8</i>	1-60	<i>l(1)d.norm.-8</i>	1	-	.12	P
<i>l(1)dn11</i>	1-26	<i>l(1)d.norm.-11</i>	2	±	0	L
<i>l(1)dn12^γ</i>	1-0.2	<i>npr1</i> <i>l(1)d.norm.-12</i>	2	+	0	L
<i>l(1)dn13</i>	1-18.5	<i>l(1)d.norm.-13</i>	2	+	0	L/P
<i>l(1)dn14</i>		<i>l(1)d.norm.-14</i>	2	+	.08	P
<i>l(1)dn15^γ</i>	1-9.5	<i>l(1)d.norm.-15</i>	2	+		P
<i>l(1)dn16</i>		<i>l(1)d.norm.-16</i>	2	+		P
<i>l(1)dn17</i>		<i>l(1)d.norm.-17</i>	2	+		P
<i>l(1)dn18</i>		<i>l(1)d.norm.-18</i>	2	+		P
<i>l(1)dn19</i>	1-0.8	<i>l(1)d.norm.-19</i>	2	+		P
<i>l(1)dn20</i>		<i>l(1)d.norm.-20</i>	2	+		P
<i>l(1)dn21</i>	1-35.8	<i>l(1)d.norm.-21</i>	2	+		P
<i>l(1)dn22</i>		<i>l(1)d.norm.-22</i>	2	+		P
<i>l(1)dn23</i>		<i>l(1)d.norm.-23</i>	2	+		P
<i>l(1)dn24</i>	1-0.3	<i>l(1)d.norm.-24</i>	2	+		P
<i>l(1)dn25^γ</i>	1-22	<i>l(1)d.norm.-25</i>	2	+		P
<i>l(1)dn26</i>	1-56-58	<i>l(1)d.norm.-26</i>	2	+		P
<i>l(1)dn27</i>	1-41.9	<i>l(1)d.norm.-27</i>	2	+		P
<i>l(1)dn28</i>	1-61.9	<i>l(1)d.norm.-28</i>	2	±	.10	P

^α 1 = Stewart, Murphy, and Fristrom, 1972, Dev. Biol. 27: 71-83; 2 = Kiss, Bencze, Fekete, Fodor, Gausz, Maróy, Szabad, and Szidonya, 1976, Theor. Appl. Genet. 48: 217-226.

^β A = differentiation in transplants; B = survival of gynandromorphs; C = lethal phase (L = larval, P = pupal).

^γ Cell division normal (Gatti and Baker, 1989, Genes Dev. 3: 438-53).

l(1)ds: lethal (1) discs small

A group of late larval and early pupal lethals in which the imaginal discs appear normal in morphology, but smaller than their wild-type counterparts at approximately the same stage of development. The ability of the discs of these mutants to differentiate in a normal hormonal milieu has been examined both in gynandromorphs and when transplanted into larvae that are then allowed to undergo metamorphosis.

Mutant	genetic location	synonym	ref ^α	PHENOTYPE ^β		
				A	B	C
<i>l(1)ds1</i>	1-0.7	<i>l(1)d.sml.-1</i>	3	+	0.31	P
<i>l(1)ds2¹</i>	1-32.2	<i>l(1)d.sml.-2^a</i>	3	-	0.06	P
<i>l(1)ds2²</i>	1-22.8-32.5	<i>l(1)d.sml.-2^b</i>	3	+	0.22	P
<i>l(1)ds3</i>	1-42	<i>l(1)d.sml.-3</i>	3	+	1.30	P
<i>l(1)ds4</i>	1-54	<i>l(1)d.sml.-4</i>	3	-	0	P
<i>l(1)ds5</i>	1-63	<i>l(1)d.sml.-5</i>	3	-	0.14	P
<i>l(1)ds8^γ</i>	1-19.5	<i>l(1)d.sml.-8</i>	1		0	L
<i>l(1)ds9^γ</i>	1-56.9-57.1	<i>l(1)d.sml.-9</i>	1	±		L

Mutant	genetic location	synonym	ref ^α	PHENOTYPE ^β		
				A	B	C
<i>l(1)ds10</i>	1-18.5	<i>l(1)l-74</i>	2	0	P	
<i>l(1)ds11</i>	1-26.3	<i>l(1)l-43</i>	2	0	P	
<i>l(1)ds12</i>	1-58.7	<i>l(1)l-45</i>	2	0	P	
<i>l(1)ds13</i>	1-66.4	<i>l(1)l-48</i>	2	0	P	

^α 1 = Kiss, Bencze, Fekete, Fodor, Gausz, Maróy, Szabad, and Szidonya, 1976, Theor. Appl. Genet. 48: 217-226; 2 = Kiss and Szabad, 1980, DIS 55: 75-76; 3 = Stewart, Murphy, and Fristrom, 1972, Dev. Biol. 27: 71-83.

^β A = differentiation in transplants; B = survival of gynandromorphs; C = lethal phase (L = larval, P = pupal).

^γ Cell division normal (Gatti and Baker, 1989, Genes Dev. 3: 438-53).

l(1)ds10

phenotype: Discs small, folding rudimentary; ring gland normal size.

l(1)ds11

phenotype: Discs very small, no folding; ring gland small; pupariation accelerated by implantation of wild-type ring glands.

l(1)ds12

phenotype: Discs very small, no folding; ring gland small.

l(1)ds13

phenotype: Discs small, folding rudimentary; ring gland small.

l(1)E7^{ts}

location: 1-15.2 (1.5 units to the right of *cv*).

origin: Induced by ethyl methanesulfonate.

references: Suzuki, Piternick, Hayashi, Tarasoff, Baillie, and Erasmus, 1967, Proc. Nat. Acad. Sci. USA 57: 907-12.

Tarasoff and Suzuki, 1970, Dev. Biol. 23: 492-509.

phenotype: Males, but not homozygous females, able to develop at 29°; females die as embryos or young larvae; males die as late pupae or young imagos. TSP of males lasts from midpupal into early adulthood. Females sensitive during first 40 hr as well as from 60-75 hr and again during pupation. Mutant females homozygous for *tra* respond to high temperature as do *tra*⁺ females.

l(1)E12: see *l(1)5CDA*

l(1)E25^{ts}

location: 1-15.5 (1.8 units to the right of *cv*).

origin: Induced by ethyl methanesulfonate.

references: Suzuki, Piternick, Hayashi, Tarasoff, Baillie, and Erasmus, 1967, Proc. Nat. Acad. Sci. USA 57: 907-12.

Tarasoff and Suzuki, 1970, Dev. Biol. 23: 492-509.

phenotype: Hatchability of eggs deposited by *l(2)E25^{ts}* females inversely related to length of maternal exposure to 29°; no hatch after 12-15 hr at 29°. Heat effect reversible by cooling for the first few hours after ovoposition. Exposure of paternal male to restrictive temperatures without effect. Newly fertilized eggs placed at 29° produce larvae that arrest in second instar; later shift up causes later arrest. Lethal polyphasic.

l(1)E34^{ts}

location: 1-20.7 (7.0 units to the right of *cv*).

origin: Induced by ethyl methanesulfonate.

references: Suzuki, Piternick, Hayashi, Tarasoff, Baillie, and Erasmus, 1967, Proc. Nat. Acad. Sci. USA 57: 907-12.

Tarasoff and Suzuki, 1970, Dev. Biol. 23: 492-509.

phenotype: Late pupal lethal; flies raised at 29° form eye and bristle pigments and wings but fail to eclose. Rates of development heterogeneous. TSP begins at time of initiation of pupation.

l(1)EN: lethal (1) from Eugene Nonautonomous

origin: X ray induced.

references: Novitski, 1963, DIS 37: 53.

phenotype: A series of sex-linked recessive lethals selected by virtue of the survival of hemizygous lethal-bearing cells in gynandromorphs. Free amino acid concentrations in lethal-bearing immature stages estimated by means of paper chromatography; differences from wild type reported (see also CP627).

locus	genetic location	lethal phase	amino acid phenotype ^α	comments
<i>l(1)EN1</i>	1-46	L1-P	ALA,tyr	salivary glands and gastric ceca small; fat body missing in L3
<i>l(1)EN2</i>	1-0.3	L3-P	GLN,glu,asp	= <i>l(1)19Fd</i> ; red-black pigmented areas on larval cuticle
<i>l(1)EN3</i>	near <i>car</i>	P	GLN	
<i>l(1)EN4</i>	1-52	P-A	GLN	larval fat bodies and Malpighian tubules reduced
<i>l(1)EN5</i>	1-47	L1-L2	GLN	
<i>l(1)EN6</i>	1-63	E-P	GLN	
<i>l(1)EN7</i>	rearranged	P	GLN,tyr	fat bodies beaded
<i>l(1)EN8</i>	left of <i>cv</i>	L2-L3	GLN,tyr	
<i>l(1)EN9</i>	1-10	L3	GLN,tyr	fat bodies, Malpighian tubules, and salivary glands reduced; rare survivors have soft exoskeleton
<i>l(1)EN10</i>	1-59	P-A	GLN,tyr,pro	fat bodies, Malpighian tubules, and salivary glands reduced; larvae translucent; fluorescence accumulates in larval cuticle
<i>l(1)EN10a</i>	1-50	P	GLY	
<i>l(1)EN11</i>	1-43	L2-P	PHE,tyr	melanotic spots on some larvae and in pupae; odor of dying larvae urinous
<i>l(1)EN12</i>	1-3	L3-P	tyr	surviving adults have soft exoskeleton with little pigmentation; almost translucent
<i>l(1)EN13</i>	1-13.4		tyr	fluorescence accumulates in larval cuticle.
<i>l(1)EN14</i>	rearranged	L2-L3	tyr,pro	
<i>l(1)EN15</i>	near <i>car</i>	L3-P	tyr,pro	
<i>l(1)EN16</i>	1-24	L1-P	tyr,pro	

^α Upper-case symbols represent amino acids found in higher-than-normal concentrations; lower-case symbols represent amino acids found in lower-than-normal concentrations.

l(1)ER^{ts}: lethal (1) endoplasmic reticulum

location: 1-18.

origin: Induced by ethyl methanesulfonate.

references: Fullilove and Woodruff, 1974, Dev. Biol. 38: 291-307 (fig.).

phenotype: Embryonic development arrested prior to gastrulation at 29°; hatching delayed approximately two hr at 25° but development normal. Embryos produced by *l(1)ER^{ts}* females held at 29° arrested prior to gastrulation; this TSP lasts until 8 hr after fertilization. Hatching of eggs produced by such females delayed 2 hr at 21°

compared with eggs from wild-type females. Second TSP occurs during the first three days of larval life; larvae developing at 29° arrest as fully-developed pupae which fail to eclose. Electron-microscope observations made on arrested embryos; show abnormal distributions of nuclei, cytoplasm, and yolk; rough endoplasmic reticulum abnormal.

l(1)ESHS: lethal (1) of Eeken, Sobels Hyland, and Schalet

origin: MR induced. Recovered among the progeny of males whose fathers carried *MR-h12* and who inherited from their mothers *In(1)dl49*, *y w f* (experiment 1/), *y mei-9^a mei-41^{D5}* (experiments 2/ and D), or Berlin-K wild type (experiments 20/, 24/, 28/, and 32/) X chromosomes. P-element sequence inserts detected by *in situ* hybridization to polytene X chromosomes in those mutants whose acquisition numbers are followed by an asterisk, but not in those followed by double asterisks.

references: Eeken, Sobels, Hyland, and Schalet, 1985, *Mutat. Res.* 150: 261-75.

cytology: Lethals in regions covered by X duplications; subjected to more or less complete deficiency mapping, but complementation mapping with respect to previously mapped lethal mutations not generally done. Those few shown not to complement known lethal mutations are listed with the appropriate loci; the remainder are arranged below in relation to the duplications and deficiencies against which they were tested and in their order along the chromosome. Allelism tests among *l(1)ESHS* mutants mapping to the same cytological interval not invariably performed; such allelism as discerned indicated by more than a single isolation designation for a locus. As allelism of *l(1)ESHS* mutants to known loci is determined, they will be removed from this list and appended to the allelic lists of said loci. Polytene analysis revealed no case of gross chromosome rearrangement.

side	breakpoint	variant	original designation
right	1B9-10	<i>Df(1)y74k24.1</i>	
	1B9-E2	<i>l(1)ESHS1</i>	D2
	1B9-E2	<i>l(1)ESHS2</i>	D1*, D52*
left	1E1-2	<i>Df(2)sta</i>	
left	2A2	<i>Df(1)HA18</i>	
	2A2-4	<i>l(1)ESHS3</i>	2/6B2, 32/35B2*, D3*
	2A2-4	<i>l(1)ESHS4</i>	D42
right	2A4	<i>Df(1)HA18</i>	
right	2B3-4	<i>Df(1)sta</i>	
	2B3-12	<i>l(1)ESHS5</i>	32/10B*
right	2B12	<i>Df(1)A94</i>	
right	2B17	<i>y²Y67g19.1</i>	
	2B17-C2	<i>l(1)ESHS6</i>	2/11A1
	2B17-C2	<i>l(1)ESHS7</i>	2/1D
	2B17-C2	<i>l(1)ESHS8</i>	2/3B1
	2B17-C2	<i>l(1)ESHS9</i>	28/80A
	2B17-C2	<i>l(1)ESHS10</i>	32/18B4
	2B17-C2	<i>l(1)ESHS11</i>	D17
left	2C2	<i>Dp(1;3)w^{vco}</i>	
	2C2-D1	<i>l(1)ESHS12</i>	28/26A1
left	2D1	<i>w^{+Y}</i>	
	2D1-2	<i>l(1)ESHS13</i>	1/2A4
	2D1-3	<i>l(1)ESHS14</i>	2/16B1, 2/16B2
left	2D3	<i>Df(1)Pgd-kz</i>	
	2D3-F1	<i>l(1)ESHS15</i>	24/17B
left	2E2-F1	<i>Df(1)TEM304</i>	
right	3A3	<i>Df(1)TEM304</i>	
	2F5-3C5	<i>l(1)ESHS16</i>	1/12A ^{**}
	2F5-3C5	<i>l(1)ESHS17</i>	D14 ^{**}

side	breakpoint	variant	original designation
right	3A1	<i>Dp(1;f)R</i>	
right	3C5	<i>Dp(1;3)w^{vco}</i>	
	3C5-D4	<i>l(1)ESHS18</i>	1/8D
	3C5-D4	<i>l(1)ESHS19</i>	D22
right	3D3-4	<i>w^{+Y}</i>	
right	3E2-3	<i>Df(1)N⁸</i>	
	3E2-8	<i>l(1)ESHS20</i>	1/8B1
	3E2-8	<i>l(1)ESHS21</i>	1/8B2
	3E2-8	<i>l(1)ESHS22</i>	D12
	3E2-8	<i>l(1)ESHS23</i>	D81 ^{**}
right	3E8	<i>Dp(1;2)w^{+ec+}</i>	
left	6C	<i>Dp(1;3)sn^{13a}</i>	
	6C-7A3	*<i>l(1)ESHS24</i>	1/3B
	6C-7A3	*<i>l(1)ESHS25</i>	2/3B2
	6C-7A3	*<i>l(1)ESHS26</i>	D20
	6C-7A3	<i>l(1)ESHS27</i>	D7, D8
	6C-7A3	<i>l(1)ESHS28</i>	1/9C
	6C-7A3	<i>l(1)ESHS29</i>	D77
	6C-7A3	<i>l(1)ESHS30</i>	32/2B
left	7A2-3	<i>Df(1)ct-J4</i>	
	7A2-8	<i>l(1)ESHS31</i>	D47
right	7A8	<i>Dp(1;2)sn^{+72d}</i>	
left	7B2-4	<i>Df(1)ct4b1</i>	
	7B2-C1	<i>l(1)ESHS32</i>	24/52A1, 28/60A, 28/85A1
	7B2-C1	<i>l(1)ESHS33</i>	28/99A1
right	7C1	<i>Df(1)ct-J4</i>	
right	7C3	<i>Df(1)ct4b1</i>	
	7C3-D1	<i>l(1)ESHS34</i>	24/16B
	7C3-D1	<i>l(1)ESHS35</i>	D6, D69
right	7C9-D1	<i>Dp(1;3)sn^{13a}</i>	
left	7D1	<i>Df(1)sn-cl28</i>	
right	7D5-6	<i>Df(1)sn-cl28</i>	
	7D5-10	<i>l(1)ESHS36</i>	D63
	7D5-10	<i>l(1)ESHS37</i>	D67
left		<i>Df(1)D46</i>	
left	7D10	<i>Df(1)RA2</i>	
left		<i>Df(1)D31</i>	
right		<i>Df(1)D46</i>	
	7D10-8A5	<i>l(1)ESHS38</i>	D68
	7D10-8A5	<i>l(1)ESHS39</i>	D75
right		<i>Df(1)D31</i>	
right	8A5	<i>Dp(1;2)sn^{+72d}</i>	
left	9A2	<i>Dp(1;2)v^{+75d}</i>	
	9A2-B2	<i>l(1)ESHS40</i>	28/7A4
left	9B1-2	<i>Df(1)v-L15</i>	
left	9E1	<i>Dp(1;2)v⁺⁶³ⁱ</i>	
	9E1-4	<i>l(1)ESHS41</i>	D45, D79
left	9E3-4	<i>Dp(1;2)v^{+74c}</i>	
left	9F3	<i>v^{+B}S^{-Y}</i>	
	9F3-6	<i>l(1)ESHS42</i>	1/15B2
	9F3-6	<i>l(1)ESHS43</i>	D70
left	9F5-6	<i>Df(1)v-L4</i>	
left	10B3	<i>Df(1)N71</i>	
	10B3-C3	<i>l(1)ESHS44</i>	32/56A
left	10C2-3	<i>Df(1)m259-4</i>	
right	10E3-4	<i>v^{+B}S^{-Y}</i>	
	10E3-11A2	<i>l(1)ESHS45</i>	32/53B
left	11A2	<i>Df(1)HF368</i>	
	11A2-7	<i>l(1)ESHS46</i>	24/39B, D25, D29
right	11A7	<i>Dp(1;2)v^{+65b}</i>	
left	13F10	<i>Dp(1;4)r^{+75c}</i>	
	13F10-14B3	<i>l(1)ESHS47</i>	28/74A
left	14B13	<i>Dp(1;2)r^{+75c}</i>	
left	14B6	<i>Df(1)r-D</i>	
	14B13-15A4	<i>l(1)ESHS48</i>	D23
	14B13-15A4	<i>l(1)ESHS49</i>	20/9A
left	15A4	<i>Dp(1;3)j^{+71b}</i>	
right	15A9	<i>Dp(1;2)r^{+75c}</i>	
	15A9-16A2	<i>l(1)ESHS50</i>	24/47B1,2,3, 32/43B
right	15A2	<i>Df(1)r-D</i>	
	15A9-16A2	<i>l(1)ESHS51</i>	32/16A
right	16A2	<i>Dp(1;4)r^{+75c}</i>	
right	19E6-7	<i>Df(1)16-3-35</i>	
	19E6-8	<i>l(1)ESHS52</i>	D76, D83
right	19E7-8	<i>Df(1)A118</i>	
left	19F1	<i>Df(1)16-129</i>	

side	breakpoint	variant	original designation
right	19F6-20A1	I(1)ESHSS3 <i>Df(1)16-129</i>	D44

l(1)fdg: see *fdg*

***I(1)ff: lethal (1) formalin food**

location: 1- (not located).

origin: Induced by formaldehyde.

discoverer: Auerbach.

synonym: *Lff11*.

references: Ede, 1956, Arch. Entwicklungsmech. Organ. 148: 416-36 (fig.).

phenotype: Develops to late embryonic stage; at 22 hr (normal hatching time), shows vigorous muscular movements but is unable to break through vitelline membrane. Muscular activity persists several hours, but hatching does not occur; cell degeneration begins at about 25 hr. Differentiation abnormal in several ways: pharyngeal apparatus reduced and distorted; brain forms irregular mass; constriction forms behind head; segmentation distorted; body wall usually incomplete dorsally. RK2.

I(1)FM

Several lethal mutations have been recorded in *FM6* or *FM7*; they have not been further characterized.

locus	Bal	synonym ref
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I(1)FMa	FM6	<i>l(1)69a</i> (Liu and Lim, 1975, Genetics 79: 601-11)
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I(1)FMb	FM6	
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I(1)FMc	FM7	<i>l(1)TW-9</i> (Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Roux's Arch. Dev. Biol. 193: 296-307)
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I(1)HM: lethal (1) of Helen Mayo

A series of cold-sensitive sex-linked recessive lethal mutations induced by ethyl methanesulfonate. Cold-sensitive lethals are defined as exhibiting >20% survival at 22° and none at 17°, and cold-sensitive semilethals as exhibiting >30% survival at 22° and <13% at 17° (Mayoh and Suzuki, 1973, Can. J. Genet. Cytol. 15: 237-54; Falke and Wright, 1975, Genetics 81: 655-82).

mutant locus	genetic location synonym		female fertility ^α	
	17°	25°	phenotype	
I(1)HM1	1-0.6		S	S
I(1)HM2	1-57			survival of females > males
I(1)HM3	1-34		F	sS
I(1)HM4	1-55		F	F
<i>l(1)HM5</i>	1-63.8	I(1)carot14	S	S
<i>l(1)HM6</i>	1-63.8	I(1)carot14	S	S
I(1)HM7	1-0.4		F	F
I(1)HM8	1-23		F	F wings upheld at 17° and 25°
I(1)HM9	1-0		F	F
I(1)HM10			sS	sS abdominal tumors; rarely blistered wings at 17°
I(1)HM11	1-0		F	F
I(1)HM12	1-0		F	F
I(1)HM13	1-1.4		S	S
I(1)HM14	1-51		F	F
<i>l(1)HM15</i>	1-63.8	I(1)carot14		
<i>l(1)HM16</i>	1-41.8	sno	S	F short fine bristles; abdominal etching
I(1)HM17			F	F
I(1)HM18	1-61		F	F
I(1)HM19				eclosion of survivors delayed nine days
I(1)HM20	1-57		S	F short fine bristles; abdominal etching
<i>l(1)HM21</i>	1-33	<i>l(1)10Ac</i>	S	F

mutant locus	genetic location synonym		female fertility ^α	
	17°	25°	phenotype	
I(1)HM22	1-22		F	F
<i>l(1)HM23</i>	1-41.8	sno	S	F maternal effect lethal
I(1)HM24			F	F
I(1)HM25	1-48		F	F

^α F = fertile; S = sterile; sS = semisterile, producing very few progeny.

I(1)J: lethal (1) of János Szidonia

Thirty-nine complementing heat-sensitive lethals which develop at 22° but not at 29°.

discoverer: Szidonia.

references: Shellenbarger and Cross, 1979, Dev. Biol. 71: 308-22 (fig.).

I(1)J413

location: 1-between *g* and *f*.

phenotype: Males raised at 22° become sterile and lack motile sperm after six days at 28°. No postmeiotic cysts found in sterilized males. Basal third of testis contains debris; distal two-thirds accumulate primary spermatocytes.

I(1)J770

phenotype: Males raised at 22° survive but die when held at 28° following eclosion.

I(1)J1024

location: 1-19.3.

phenotype: Males raised at 22° become sterile and lack motile sperm after six days at 28°. Testicular contents disorganized in sterile males; dispersed but well-condensed sperm heads present. Thought to be blocked in spermatid elongation.

I(1)J1649

phenotype: Males raised at 22° survive but die when held at 28° following eclosion.

I(1)J1660

phenotype: Males raised at 22° fertile, but become sterile after eight days at 28°; sterile males have motile sperm, which are not transmitted to females.

I(1)J1674

phenotype: Males raised at 22° fertile, but become sterile after eight days at 28°; sterile males have motile sperm, which are not transmitted to females.

I(1)J1743

phenotype: Males raised at 22° survive but die when held at 28° following eclosion.

*l(1)J1*²⁵⁹: see *Df(1)259*

***I(1)jl: lethal (1) jawless**

location: 1-14.

origin: Ultraviolet induced.

discoverer: McQuate, 1951.

references: Oster, 1952, Heredity 6: 403-7.

phenotype: Dies during first larval instar. Mouth parts poorly formed and sometimes absent. RK2.

cytology: Salivary chromosomes normal (Valencia and McQuate, 1951, Genetics 36: 580).

l(1)L5: see *RpII215*

***I(1)m: lethal (1) malignant**

location: 1- (not located).

origin: Induced by mustard gas.

synonym: *l-mal*.

references: El Shatoury, 1955, Arch. Entwicklunsmech. Organ. 147: 496-522 (fig.).

El Shatoury and Waddington, 1957, J. Embryol. Exp. Morphol. 5: 143-52 (fig.).

phenotype: Cells originating from lymph glands in late third instar first spread to and cause destruction of imaginal buds and later may move along ventral nerve cord to attack posterior fat bodies and testes. The tumor cells eventually become melanotic after destruction of various healthy tissues. Death occurs in late larval or early pupal stages. RK2.

I(1)M

A series of sex-linked recessive lethals induced by ethyl methanesulfonate by Bryant and Zornetzer (1973, Genetics 75: 623-37). They were characterized with respect to lethal stage and survival in gynandromorphs.

mutant	genetic location	lethal stage	Gynandromorphs		
			relative survival (%)	amount of male tissue (%)	phenotype of male tissue
I(1)M1		E	0		
I(1)M3		L1	11	10	abnormal bristles
I(1)M4	1-44	L1	28	42	+
I(1)M5		L2	9	23	+
I(1)M8	1-57	L1	9	15	cuticle incompletely tanned
I(1)M9	1-37	L1	0		
I(1)M11	1-56	EP	0		
I(1)M16	1-57	L1	0		
I(1)M19		E	0		
I(1)M22		L2	0		
I(1)M24	1-52	E-L1	22	16	+
I(1)M25	1-46	LP	48	36	+
I(1)M26	1-41	L1-3	31	11	+
I(1)M36	1-41	L2	3	6	abnormal bristles
I(1)M38		L3	82	44	+
I(1)M39	1-39	L3	1	9	abnormal bristles
I(1)M42	1-57	E	0		
I(1)M46	1-12	EP	0		
I(1)M47	1-11	EP	0		
I(1)M52	1-44	LP	19	40	+
I(1)M53	1-34	L1	24	21	+
I(1)M54	1-15	L3-EP	0		
I(1)M55	1-3	E	9	15	+
I(1)M58	1-28	L1-2	12	24	abnormal bristles curled wings
I(1)M60		L1	32	21	+
I(1)M64	1-13	L2	41	42	+
I(1)M66	1-35	E	0		
I(1)M70		L3	24	26	abnormal bristles
I(1)M75		L1	0		
I(1)M77			13	14	+
I(1)M82	1-27	EP	6	17	abnormal bristles
I(1)M83		EP	0		
I(1)M84		E	0		
I(1)M96			0		

I(1)M41

location: 1-36.0.

origin: Induced by methyl methanesulfonate.

references: Lim, 1970, DIS 45: 73-74.

other information: Putative spontaneous revertants reported by Lim.

I(1)Mb and I(1)Mc: lethal (1) Madrid

A series of 89 ethyl-methanesulfonate-induced sex-linked recessive lethal mutations studied in genetic mosaics, both in gynandromorphs and epidermal clones (Ripoll, 1977, Genetics 86: 357-76). Epidermal phenotypes of subset in which male tissue had distinctive phenotype or in which gynandromorphs failed to survive described following table.

mutant	genetic location	lethal stage	relative viability of gynandromorphs	epidermal clones
I(1)Mb7	1-7	E	0.04	
I(1)Mb8	1-60	E-L	0	lethal
I(1)Mb15	1-30.1	L	0	
I(1)Mb16	1-0.5	L	0	
I(1)Mb22	1-39.7	L	0	
I(1)Mb24	1-22.4	L	0	
I(1)Mb26	1-26.8	E	0	lethal
I(1)Mb28	1-15.5	P	0	
I(1)Mb38	1-48.5	P	0.22	
I(1)Mb46	1-56.4	L	0	lethal
I(1)Mc1		P	1.63	
I(1)Mc19^α	1-44.4	E	0	lethal
I(1)Mc23	1-43.5	L	0	
I(1)Mc24	1-55.9	L	0	lethal
I(1)Mc28	1-0	E-L	0	
I(1)Mc32	1-32.4	E	0	
I(1)Mc35	1-62	E-L	0	lethal
I(1)Mc39	1-23.5	L	0.04	
I(1)Mc51	1-27	P	0.18	
I(1)Mc52	1->56	L	0	
I(1)Mc56	1-25	L	0.19	

^α Fails to complement g.

I(1)Mb7

phenotype: All bristles transparent, smaller than normal; glassy eyes; arista almost branchless.

I(1)Mb16

phenotype: Bristles and trichomes normal. 30% of clones split into several spots of similar size, which together add up in area to the area of control spots. Split clones found only in spots induced more than 48 hr before puparium formation.

I(1)Mb24

phenotype: Bristles small and unpigmented; wing trichomes about half normal size. As a consequence of reduced cell size, the area occupied by a clone does not correspond to that expected for its number of cells.

I(1)Mb28

phenotype: Bristles of the wing margin small and unpigmented. Clones in the wing blade extremely elongated. Whereas control clones are 5-10 times longer than wide, with irregular borders, clones of cells monozygous for the lethal are 30-50 times longer than wide, with smooth borders. This phenotype found only in clones induced more than 48 hr before puparium formation.

l(1)Mb38

phenotype: External genitalia do not evaginate when male or mosaic; normal structures remain within the abdomen. Other structures, when male, evaginate normally.

l(1)Mc1

phenotype: Rounded wings, about one-third shorter than normal. Rough and bulgy eyes.

l(1)Mc28

phenotype: Bristles smaller than normal. Wing trichomes small; occasionally clones of cells without epidermal processes appear. Behaves as expected for a mutation decreasing mitotic rate.

l(1)Mc39

phenotype: Very thin bristles, shorter than normal; abdomen almost completely devoid of bristles.

l(1)Mc51

phenotype: Several (2-6) trichomes per cell in wing disc derivatives. Only 75% of the individual cells show this phenotype.

l(1)Mc56

phenotype: Wings coiled dorsally.

l(1)ml: lethal (1) melanomalike

location: 1-10.

origin: Ultraviolet induced.

discoverer: McQuate, 1951.

references: Oster, 1952, *Heredity* 6: 403-7.

Oster and Sobels, 1956, *Am. Nat.* 90: 55-60.

phenotype: Larvae die in third instar. At death, they have internal melanotic masses (usually one or two, sometimes as many as ten). RK2.

cytology: Salivary chromosomes normal (Valencia and McQuate, 1951, *Genetics* 36: 580).

****l(1)mt: lethal (1) midget***

location: 1-2.5.

origin: Ultraviolet induced.

discoverer: McQuate, 1951.

references: Oster, 1952, *Heredity* 6: 403-7.

phenotype: Dies as undersized third-instar larva. RK2.

cytology: Salivary chromosomes normal (Valencia and McQuate, 1951, *Genetics* 36: 580).

l(1)nc: lethal (1) nutritionally conditional

Three mutants that are lethal on Burnett and Sang's defined casein medium (1963, *J. Insect. Physiol.* 9: 553-62) but can be rescued by addition of yeast extract to the medium.

origin: Induced by ethyl methanesulfonate.

references: Vyse and Nash, 1969, *Genet. Res.* 13: 281-87. Vyse and Sang, 1971, *Genet. Res.* 18: 117-21.

locus	genetic location	synonym	partial rescue by RNA
<i>l(1)nc1</i>	1-21.7	<i>l(1)I308</i>	+
<i>l(1)nc2</i>^α		<i>l(1)I625</i>	+
<i>*l(1)nc3</i>^β	1-52.9	<i>l(1)I1523</i>	-

^α Associated with *In(1)I625 = In(1)3F;20C*.

^β Female sterile---probably a rudimentary allele.

****l(1)nd: lethal (1) no differentiation***

location: 1- (not located).

origin: Induced by mustard gas.

references: El Shatoury, 1955, *Arch. Entwicklungsmech. Organ.* 147: 523-38 (fig.).

phenotype: Some or all imaginal buds fail to differentiate during larval third instar, apparently as a result of abnormal proliferation of imaginal disk mesoderm. Death in pupal or prepupal stage. RK2.

****l(1)ne: lethal (1) nonevaginates***

location: 1-0.1.

origin: Induced by urethane.

discoverer: Vogt, 1949.

references: 1951, *DIS* 25: 76.

Florschütz-de Waard and Faber, 1952, *DIS* 26: 99.

Faber, Sobels, Florschütz-de Waard, and Oppenoorth, 1954, *Z. Indukt. Abstamm. Vererbungslehre* 86: 293-321 (fig.).

phenotype: Lacks imaginal thoracic hypoderm. Cephalic complex and thoracic imaginal disks fail to evaginate. The unaffected abdominal hypoderm develops but ends anteriorly in a free edge that folds back on itself and forms a darkly-pigmented ring around the pupa. Genital disk capable of normal evagination but vasa deferentia do not connect to testes, which do not spiralize. Death occurs 3-5.5 days after prepuparium formation. Pupae darker than normal with sticky, irregular surface and distinctly meandering tracheal trunks. RK2.

****l(1)nib: lethal (1) no imaginal buds***

location: 1- (not located).

references: El Shatoury and Waddington, 1957, *J. Embryol. Exp. Morphol.* 5: 143-52 (fig.).

phenotype: Dies in third larval instar. Imaginal buds small or absent. Excessive proliferation of stomach epithelium leads to occlusion of gut. Proliferations degenerate into melanotic masses. RK2.

l(1)P1: lethal (1) of Parkash

location: 1-52.

origin: Recovered from thymidine-fed flies.

references: Parkash, 1969, *DIS* 44: 51.

phenotype: *l(1)P1* males completely lethal when reared at 16° and viable when reared at 26°; lethal period at larval-pupal transition. Homozygous females survive either temperature.

l(1)P2

location: 1-42.3.

origin: Recovered from thymidine-fed flies.

references: Parkash, 1971, *DIS* 46: 67.

phenotype: Mutant males survive rearing at 16° but not 26°.

l(1)P3

origin: Recovered from thymidine-fed flies.

references: Parkash, 1971, *DIS* 46: 67.

phenotype: Mutant males die when reared at 16° but survive rearing at 26° and are sterile.

****l(1)rr: lethal (1) ring gland rudimentary***

location: 1-0.3.

origin: Ultraviolet induced.

discoverer: McQuate, 1951.

references: Oster, 1952, *Heredity* 6: 403-7.

phenotype: Dies during third larval instar. Larvae live 15-30 days but do not become giant. Ring gland abnormally small, probably causing failure to undergo third molt. RK2.

cytology: Salivary chromosomes normal (Valencia and McQuate, 1951, Genetics 36: 580).

***l(1)S9**

location: 1- (to the right of *car*).

origin: Spontaneous.

discoverer: Auerbach.

references: Ede, 1956, Arch. Entwicklungsmech. Organ. 149: 256-66 (fig.).

phenotype: Almost all embryos deformed at anterior end, where there is usually some undigested yolk. Death occurs in embryonic, larval, and pupal stages. Primary abnormality is distribution of cleavage nuclei, which causes blastoderm to be fragile at its anterior end. RK2.

l(1)sc: see **ASC**

***l(1)sd: lethal (1) scheiben defekt**

location: 1-17.9.

origin: Induced by triethylenemelamine (CB. 1246).

discoverer: M. J. Fahmy.

references: Schnitter, 1961, Rev. Suisse Zool. 68: 345-418 (fig.).

phenotype: Dies during transition from larva to prepupa. Some larvae form puparia but do not differentiate further. Pattern of damage complex; most severe defects found in certain imaginal disks. Several larval organs abnormal, especially the salivary glands. RK2.

***l(1)te: lethal (1) tracheae enlarged.**

location: 1-0.3.

origin: Ultraviolet induced.

discoverer: McQuate, 1951.

references: Oster, 1952, Heredity 6: 403-7.

phenotype: Dies during third larval instar. Main tracheal tubes greatly enlarged, sometimes lack functional posterior spiracles. RK2.

cytology: Salivary chromosomes normal (Valencia and McQuate, 1951, Genetics 36: 580).

***l(1)tl: lethal (1) tracheae lacking**

location: 1-59.

origin: Ultraviolet induced.

discoverer: McQuate, 1951.

references: Oster, 1952, Heredity 6: 403-7.

phenotype: Dies during first larval instar. Main tracheal tubes absent, although small side branches present. RK2.

cytology: Salivary chromosomes normal (Valencia and McQuate, 1951, Genetics 36: 580).

***l(1)tr: lethal (1) tracheae ramified**

location: 1-56.

origin: Ultraviolet induced.

discoverer: McQuate, 1951.

references: Oster, 1952, Heredity 6: 403-7.

phenotype: Dies during first larval instar. Main tracheal tubes thick and have numerous side branches. RK2.

cytology: Salivary chromosomes normal (Valencia and McQuate, 1951, Genetics 36: 580).

***l(1)trs: lethal (1) tracheae stretched**

location: 1-8.0.

origin: Ultraviolet induced.

discoverer: McQuate, 1951.

synonym: *l(1)ts* (preoccupied).

references: Oster, 1952, Heredity 6: 403-7.

phenotype: Dies during first larval instar. Larvae very large for this stage and all tracheal tubes very thin, suggesting that they grow more slowly than larvae and thus become stretched. RK2.

cytology: Salivary gland chromosomes normal (Valencia and McQuate, 1951, Genetics 36: 580).

***l(1)ts: lethal (1) temperature sensitive**

location: 1-8.

discoverer: Falbo and Ré.

references: 1945, DIS 19: 45, 57.

phenotype: Inviabile in cultures grown at 23° but shows more than 50% survival in cultures grown at 26.5°. RK3.

l(1)ts1

location: 1-1.9.

origin: Induced with ethyl methanesulfonate.

references: Fausto-Sterling, Wiener, and Digan, 1979, J. Exp. Zool. 200: 199-210.

phenotype: Maternal-effect lethal. Homozygous females raised or aged at 28° produce embryos that fail to develop (incomplete dorsal closure, abnormal midgut formation) regardless of zygotic genotype. Females raised at 18° produce viable embryos. Viable embryos placed at 28° die as late pupae and are insensitive to maternal genotype. TSP for maternal effect after stage 7 of oogenesis; pupal TSP lasts 2.5 days.

l(1)ts

Lethals recovered as temperature-sensitive alleles by workers in several different laboratories. Some are mapped, and most have been scored for survival in patches of homozygous cuticular tissue. In general complementation tests among these lethals or between these lethals and other X-linked lethal mutations were not carried out. These names are transitional and likely to be changed if any of the mutants is further characterized.

locus	genetic location	origin	synonym	ref α	epidermal clones β
<i>l(1)ts13</i> ^γ		EMS		3	
<i>l(1)ts19</i> ^γ		EMS		3	
<i>l(1)ts66</i> ^δ	rt. of <i>f</i>	EMS		5, 6	
<i>*l(1)ts79</i>				7	
<i>l(1)ts88</i> ^γ		EMS		3	
<i>l(1)ts108</i> ^γ		EMS		3	
<i>l(1)ts120</i> ^E	1-51.5	EMS		4	t.s.
<i>*l(1)ts136</i>		EMS		7	
<i>l(1)ts155</i>	<i>cv-ct</i>	EMS	<i>l(1)5CD</i>	5, 6	
<i>l(1)ts178</i>		EMS		4	non t.s.
<i>l(1)ts340</i>	1-28.3	EMS		7	
<i>l(1)ts398</i>	1-38.9	EMS	<i>agn</i>	5, 6	
<i>l(1)ts403</i>	1-32.6	EMS	<i>sbr</i>	1	t.s.
<i>l(1)ts445a</i>	1-25.0	EMS		7	
<i>l(1)ts445b</i>		EMS		4	non t.s.
<i>l(1)ts480</i>	1-54	EMS		1	t.s.
<i>l(1)ts504</i> ^δ	1-6.0	EMS		7	t.s.
<i>l(1)ts538</i>	1-0.38	EMS		4	t.s.
<i>l(1)ts612</i>	1-17.0	EMS		7	
<i>l(1)ts613</i> ^ζ	1-62.8	EMS		4	t.s.
<i>l(1)ts622</i>	1-38.9	EMS	<i>agn</i>	5, 6	
<i>l(1)ts639</i>		EMS		4	non t.s.
<i>l(1)ts726</i> ^η	1-66.0	EMS	<i>su(f)</i>	4	t.s.

locus	genetic location	origin	synonym	ref α	epidermal clones β
<i>l(1)ts875</i>		EMS		4	non t.s.
<i>l(1)ts958</i>	1-7.25	EMS		7	
<i>l(1)ts967</i> ^{θ}		EMS		7	
<i>l(1)ts980</i>	1-38.9	EMS	<i>agn</i>	5, 6	
<i>l(1)ts982</i>	1-26.0	EMS		7	
<i>l(1)ts996</i>		EMS		1	non t.s.
<i>l(1)ts1006</i> ^{γ}		EMS		1, 3	t.s.
<i>l(1)ts1006</i> 1251		EMS		1, 3	
<i>l(1)ts1006</i> 1704		EMS		1, 3	
<i>l(1)ts1006</i> 1843		EMS		1, 3	
<i>l(1)ts1064</i>		EMS		4	non t.s.
<i>l(1)ts1075</i>		EMS		4	non t.s.
<i>l(1)ts1081</i>		EMS		4	t.s.
<i>l(1)ts1082</i> ^{ζ}	1-61.0	EMS		4	t.s.
<i>l(1)ts1107</i>		EMS		4	t.s.
<i>l(1)ts1126</i> ^{$\delta\theta$}	1-16.6	EMS		7, 8	
<i>l(1)ts1137</i>		EMS		4	non t.s.
<i>l(1)ts1233</i>		EMS		4	t.s.
<i>l(1)ts1251</i> ^{θ}	1-43	EMS		1, 2	t.s.
<i>l(1)ts1299</i>		EMS		4	non t.s.
<i>l(1)ts1343</i>		EMS		4	non t.s.
<i>l(1)ts1415</i>		EMS		4	t.s.
<i>l(1)ts1480</i>		EMS		4	non t.s.
<i>l(1)ts1681</i>		EMS		4	t.s.
<i>l(1)ts1704</i>	1-46	EMS		1	non t.s.
<i>l(1)ts1808</i>		EMS		4	t.s.
<i>l(1)ts1843</i>	1-46	EMS		1	non t.s.
<i>l(1)ts1891</i> ^{δ}	1-52.9	EMS		4	t.s.
<i>l(1)ts1901</i>		EMS		4	non t.s.
<i>l(1)ts2009</i>		EMS		4	t.s.
<i>l(1)ts2320</i> ^{γ}		EMS		3	
<i>l(1)ts2641</i> ^{γ}		EMS		3	
<i>l(1)ts2664</i> ^{γ}	1-35	EMS		1, 3	t.s.
<i>l(1)ts3733</i>	1-38	EMS		1	t.s.
<i>l(1)ts3803</i> ^{γ}	1-53	EMS		1, 3	t.s.
<i>l(1)ts3803</i> UC259		EMS		1, 3	t.s.
<i>l(1)ts4931B</i> ^{γ}		EMS		3	
<i>l(1)ts4975</i>	1-49	EMS		1	t.s.
<i>l(1)ts5141</i>	1-34	EMS		1	t.s.
<i>l(1)ts5569</i> ^{γ}		EMS		3	
<i>l(1)ts5697</i> ^{γ}	1-55	EMS		1, 3	t.s.
<i>l(1)ts5697</i> 2366		EMS		1, 3	t.s.
<i>l(1)ts5697</i> 2588		EMS		1, 3	t.s.
<i>l(1)ts6225</i>	1-20	EMS		1	non t.s.
<i>l(1)tsG1</i> ¹		ICR170		9	
<i>l(1)tsG2</i> ¹		ICR170		9	
<i>l(1)tsSD19</i>		EMS		1	t.s.
<i>l(1)tsW1</i>		ICR170		9	
<i>l(1)tsW2</i>		ICR170		9	
<i>l(1)tsW3</i>		ICR170		9	
<i>l(1)tsW4</i>		ICR170		9	
<i>l(1)tsW5</i>		ICR170		9	
<i>l(1)tsUC13</i>	1-29	EMS		1	t.s.
<i>l(1)tsUC19</i>	1-55	EMS		1	t.s.
<i>l(1)tsUC32</i>	1-45	EMS		1	t.s.
<i>l(1)tsUC34</i>	1-18	EMS		1	t.s.
<i>l(1)tsUC88</i>	1-45	EMS		1	t.s.

α 1 = Arking, 1975, Genetics 80: 519-37; 2 = Arking, 1978, Genetics 88: s4-5; 3 = King and Mohler, 1975, Handbook of Genetics (R.C. King, ed.), Plenum Press, New York and London, Vol. 3, pp. 757-91; 4 = Russell, 1974, Dev. Biol. 40: 24-39; 5 = Savvateeva and Kamyshev, 1981, Pharmacol. Biochem. Behav. 14: 603-11; 6 = Savvateeva, Pereslenny, Ivanushina, and Korochkin, 1985, Dev. Genet. 5: 157-72; 7 = Simpson and Schneiderman, 1975, Wilhelm Roux's Arch. Dev. Biol. 178: 247-75; 8 = Simpson and Schneiderman, 1976, Wilhelm Roux's Arch. Dev. Biol. 179: 215-36; 9 = Woodruff and Gander, 1974, Mut. Res. 25: 337-45.

β t.s. indicates that the incidence of X-ray-induced *sn* clones in *y/l(1)ts sn* females compared to the recovery of *y* clones (Arking) or of *y* clones in *y/l(1)ts/sn* females compared to the recovery of *sn* clones (Russell) is significantly lower in flies raised at 29° than in those raised at 22°; non t.s. indicates no significant difference.

γ Survivors at permissive temperature are female sterile.
 δ Phenotypes described more fully below.
 ϵ *l(1)ts120* and *l(1)1891* not allelic.
 ζ *l(1)ts613* and *l(1)1082* not allelic.
 η See *su(f)*.
 θ Survivors male sterile.
 ι Cold sensitive.

l(1)ts66

phenotype: Selected as a temperature-sensitive lethal in the presence of theophylline; hypersensitive to theophylline at 29°, but not at 25°. Mutant males contain lower levels of cyclic nucleotide phosphodiesterase than wild type and normal levels of adenylyl cyclase. Exhibit increased motor activity at 29°; movements uncoordinated; flight and both phototaxis and geotaxis impaired. Unable to learn (Savvateeva and Kamyshev, 1981, Pharmacol. Biochem. Behav. 14: 603-11; Savvateeva, Pereslenny, Ivanushina, and Korochkin, 1985, Dev. Genet. 5: 157-72).

l(1)ts504

phenotype: Temperature-sensitive pupal lethal. Heat pulses of one-to-three days duration applied during larval stages delay pupariation and cause duplications and deficiencies of imaginal structures in surviving adults. Similarly, rarely surviving gynandromorphs exhibit duplicated and missing structures; heterozygous females from the same cross that are malformed interpreted as cryptic gynandromorphs in which the male tissue has died. Epidermal clones of homozygous tissue very reduced in number and size in the head and thorax, but do not differ from controls in the abdomen (Simpson and Schneiderman, 1975, Wilhelm Roux's Arch. Dev. Biol. 178: 247-75).

l(1)ts1126

phenotype: Temperature-sensitive, cell-autonomous lethal mutant. Exposure of first- and second-instar larvae to 29° retards growth and delays pupariation, but adults eventually formed are normal. Exposure of third-instar larvae to 29° does not delay pupariation, but results in the production of smaller-than-normal flies. Late third-instar pulses cause delayed eclosion, and absence of the black pigment band as well as reduced bristle number and size in the tergites of survivors. Homozygous clones substantially reduced in size in the wing by heat pulses during early instars and in tergites by pulses at the end of the larval stage. In gynandromorphs that pupate at normal times *l(1)ts1126/0* compartments are smaller than normal; in gynandromorphs exhibiting delayed development, compartment sizes are normal [Simpson and Morata, 1980, Developmental Neurobiology of Drosophila (Siddiqi, Babu, Hall, and Hall, eds.), Plenum Press, New York and London, pp. 129-39]. All effects explained by a reduced rate of cell division in mutant cells (Simpson and Schneiderman, 1976, Wilhelm Roux's Arch. Dev. Biol. 179: 215-36).

l(1)ts1251

phenotype: A temperature-sensitive cell lethal; also a temperature-sensitive male sterile mutation. Larvae subjected to 29° during the latter part of the third larval instar produce pharate adults that display duplicated or missing structures or both typically resulting from cell death. Some 5% also exhibit transformation of the humeral disc into a heterotypic structure consisting proxi-

mally of foreleg tissue (coxa through first tarsal segments on basis of chaetotaxy) and distally of antennal tissue, *i.e.*, arista (Arking, 1978, Genetics 88: s4-5).

****l(1)TS-45: lethal (1) no. 45 of T. Shiom***

location: 1-5.8.

origin: X ray induced.

discoverer: Shiom, 52f.

references: 1954, DIS 28: 78.

Imaizumi and Shiom, 1955, Arch. Biol. (Liège) 66: 483-87.

phenotype: Dies before hatching. No visible morphological abnormality. Heterozygote of *l(1)TS-45/Basc* has average of 612 eye facets compared to only 402 in *+Basc*. Accumulation of urea or carbamides in larvae of heterozygote; these compounds presumably tend to normalize the Bar phenotype. RK2.

****l(1)TS-56***

location: 1-1.5.

origin: X ray induced.

discoverer: Shiom, 52f.

references: 1954, DIS 28: 78.

phenotype: Lethal in late embryonic stage. Development of tracheae, other chitinized parts, and body segments abnormal. RK2.

l(1)TW: lethal (1) of Ted Wright

A series of six ethyl-methanesulfonate-induced mutants that have reduced survival when raised at 17° compared with that when raised at 25°; the differences in survival at the two temperatures vary substantially among replicate crosses.

locus	genetic location	synonym	lethal stage	comments
<i>l(1)TW1</i>	1-26.3		L	female sterile at 16°, few eggs
<i>l(1)TW2</i>	1-41.8	sno		
<i>l(1)TW3</i>	1-2.5		P	females fertile; ether sensitive
<i>l(1)TW4</i>	1-48.4		LP	females fertile; metathoracic legs abnormal
<i>l(1)TW5</i>	1-63.8	l(1)carot14	LP	females sterile; abnormal abdomen, indented wings at 16°
<i>l(1)TW6</i>	1-37.1	nod⁸	E	females sterile at 16°, meiotic nondisjunction

allele	origin	discoverer	ref ^α
<i>l(1)TW1^{CS}</i>	EMS	Wright	1, 2
<i>l(1)TW3^{CS}</i>	EMS	Wright	1, 2
<i>l(1)TW4^{CS}</i>	EMS	Wright	1, 2
<i>l(1)TW6^{CS}</i>	EMS	Wright	1, 2

^α 1 = Falke and Wright, 1975, Genetics 81: 655-82; 2 = Wright, 1973, Mol. Gen. Genet. 122: 101-18.

l(1)v: lethal (1) variegated

origin: X ray induced.

references: Lindsley, Edington, and Von Halle, 1960, Genetics 45: 1649-70.

phenotype: A series of sex-linked recessive lethals that die as *XO* males but survive as *XY* males and homozygous females. All are associated with chromosome rearrangements with one breakpoint in the *X* chromosome and one in pericentric heterochromatin, of either the *X* or an autosome; interpreted as position-effect variegation of vital loci. In some cases the viability of *XY* males is further reduced by the known enhancers of variegation,

Df(2R)M41A10 and *Evar7*. The lethals associated with reciprocal translocations are male sterile, owing to a failure of sperm head elongation.

lethal	viability		XY male	enhanced by		cytology
	XY	XO	fertility	<i>Df(2R)M41A10</i>	<i>Evar7</i>	
<i>l(1)v3</i>	.74	0	sterile	yes	no	<i>T(1;3)4A;81</i>
<i>l(1)v11^α</i>	.78	.04	fertile	yes	no	<i>T(1;4)15;101</i>
<i>l(1)v25</i>	1.0	0	sterile			<i>T(1;2)19-20;40-41</i>
<i>*l(1)v47^β</i>	.41	0				<i>Tp(2;1)8F-9B</i>
<i>l(1)v59^γ</i>	.63	0	fertile			<i>In(1)3-4;20F</i>
<i>l(1)v75</i>	.26	<.01	sterile	no	yes	<i>T(1;2)19-20;41</i>
<i>l(1)v129</i>	.91	.26	sterile			<i>T(1;2)18B;41</i>
<i>l(1)v132</i>	.83	<.01	fertile			<i>In(1)3-4;20F</i>
<i>l(1)v135</i>	.40	<.01				<i>T(1;2)18-19;41</i>
<i>l(1)v139^δ</i>	0	0				<i>In(1LR)3C6-7</i>
<i>*l(1)v146^ε</i>	.41	0	fertile	yes	yes	<i>In(1)5-6;20F</i>
<i>l(1)v150</i>	.15	0	sterile			<i>T(1;2)16-17;40</i>
<i>l(1)v163</i>	.17	<.01	sterile			<i>T(1;3)17A-B;80-81</i>
<i>l(1)v216</i>	.15	0	sterile	yes	no	
<i>l(1)v219</i>	1.0	0	sterile	yes	no	<i>T(1;2)10A;40</i>
<i>*l(1)v223^ζ</i>	.41	0	sterile		yes	<i>T(1;2)14F;41;50E</i>
<i>l(1)v227</i>	.48	0	fertile	yes	yes	<i>1-2;20F</i>
<i>l(1)v231^η</i>	1.0	<.01	fertile			<i>In(1)1C-D;20F</i>
<i>*l(1)v252</i>	1.0	.02	sterile			
<i>*l(1)v306</i>	.78	0	fertile			<i>Tp(?)1B-E</i>
<i>l(1)v361</i>	1.0	0	sterile			<i>T(1;3)19-20;80-81</i>
<i>l(1)v451</i>	.63	.04	sterile			+
<i>l(1)v453</i>	1.0	0	sterile	yes	no	<i>T(1;3)12D;80-81</i>
<i>l(1)v454</i>	.50	0	sterile	yes	yes	<i>T(1;2;3)12B;22-23;81 + T(2;4)44F;101F</i>
<i>l(1)v455^θ</i>	low	0	sterile	yes	yes	<i>T(1;3)3C;81</i>
<i>l(1)v459^ι</i>	.78	0	fertile			<i>T(1;2;3)13D-F;XR;50;80-81</i>
<i>l(1)v463</i>	.50	.18	sterile	no	yes	<i>T(1;3)19-20;81-82</i>

- ^α Homozygous females have blistered wings and duplicated anterior scutellar and postalar bristles.
- ^β *XY* males have *gg*-like phenotype but with peripheral darkening of eye color. Associated with insertion of an unspecified segment of heterochromatin into 8F-9B; linkage tests suggest chromosome-2 origin.
- ^γ Homozygous females survive and have fewer and smaller bristles.
- ^δ *XYX* males viable and fertile; show strong variegation for *w* and *rst*. Recombinant carrying the left end of the *X^{P4D}* element to *T(1;4)w^{m5}* and the right end of *In(1LR)-v139* is variegated for *w* but not for *rst* and is male viable.
- ^ε Viability of *XXY>XX*; homozygous females frequently lacking dorso-central bristles.
- ^ζ Variegates for absence of external genitalia.
- ^η Surviving *XO* males have rough reduced eyes.
- ^θ Variegates for *w*.
- ^ι *XY* males have rough eyes and deformed wings and wing veins.

****l(1)w***

location: 1-66.

discoverer: Schubel, 1934.

references: 1934, Am. Nat. 68: 278-82.

phenotype: Males survive; homozygous females die. RK3.

other information: Probably a lethal allele of *bb*.

****l(1)X2: lethal (1) X ray induced***

location: 1- (near forked).

origin: X ray induced.

discoverer: Auerbach.

references: Ede, 1956, Arch. Entwicklungsmech. Organ. 148: 437-51 (fig.).

phenotype: Embryos die in advanced stage of development. They live beyond normal hatching time, move actively, but do not hatch. Embryo distorted; head material not involuted and pharyngeal material external; body wall has disarranged segmentation in medial region. Mutant disrupts mechanism controlling mitosis in early

stages of gastrulation, occasionally as early as blastoderm formation. RK2.

cytology: Salivary chromosomes normal.

*I(1)X10

location: 1-0.0 (near *sc*).

origin: X ray induced.

discoverer: Auerbach.

references: Ede, 1956, Arch. Entwicklungsmech. Organ. 149: 247-58 (fig.).

phenotype: Variation in expression of factors discontinuous. There are three types of lethal embryos; some may survive into larval stage. Type 1 stops development after formation of a cap of undifferentiated cells. Type 2 has limited differentiation, often the nervous tissue exclusively, but no organ formation. Type 3 survives beyond normal hatching time, has no gross abnormalities, but does not hatch. RK2.

*I(1)X20

location: 1- (near *sc*).

origin: X ray induced.

discoverer: Auerbach.

references: Ede, 1956, Arch. Entwicklungsmech. Organ. 149: 101-14 (fig.).

phenotype: Four types of defective embryos produced. Types 1 and 2 reach late stage of development and are alive at time larvae normally hatch. Type 1 has a complete nervous system but incomplete hypoderm. Type 2 has hypoderm but a deficient nervous system. Types 3 and 4 stop developing at early stages. Type 3 has no development beyond gastrulation, and type 4 forms no blastoderm. RK2.

*I(1)X27

location: 1-63.4.

origin: X ray induced.

discoverer: Auerbach.

references: Ede, 1956, Arch. Entwicklungsmech. Organ. 149: 88-100 (fig.).

phenotype: Embryos alive in a late stage of development at normal hatching time but do not hatch. Degeneration begins at about 25 hr. Germ band irregular at beginning of gastrulation, apparently the result of defective ventral furrow formation. Consequently, hindgut is open dorsally, nervous system irregularly developed, and ventral nerve cord interrupted in region of midgut. Other abnormalities from different causes are: (1) gut remains sac-like, (2) ectoderm remains unsegmented, and (3) musculature of body wall is underdeveloped. RK2.

I(1)Y

A series of sex-linked recessive lethals induced by ethyl methanesulfonate by Bryant and Zornetzer (1973, Genetics 75: 623-37). They were characterized with respect to lethal stage and survival in gynandromorphs.

mutant	lethal stage	Gynandromorphs		
		relative survival (%)	amount of male tissue (%)	phenotype of male tissue
<i>I(1)Y8</i>	L1	0		
<i>I(1)Y12</i>	E	3	12	wings incompletely expanded
<i>I(1)Y80</i>	E	20	21	+
<i>I(1)Y107</i>	L1	2	15	abnormal bristles
<i>I(1)Y110</i>	LP	6	17	degenerated ommatidia

The following table refers to a number of series of sex-linked-lethal mutations induced or spontaneous, many of which were mapped genetically, and some of which were analysed cytologically. Virtually all have long since been discarded; they are cited here for completeness of the record (see also CP627).

series	number of lethals	genetic mapping	cytology	origin	ref ^α
<i>I(1)291-</i>	13	no	yes	spont	13
<i>I(1)294-</i>	4	yes	yes	X ray	13
<i>I(1)296-</i>	6	yes	yes	spont	13
<i>I(1)302-</i>	4	yes	yes	neutron	13
<i>I(1)304-</i>	4	yes	yes	X ray	13
<i>I(1)DM</i>	5	rough	no	X ray	7,8
<i>I(1)GSB</i>	51	yes	no	X ray	4
<i>I(1)I</i>	13	no	no	heat	10, 11
<i>I(1)K</i>	4	yes	no	32 p	5
<i>I(1)LB</i>	500	yes	no	various	1, 2
<i>I(1)MA</i>	50	few	no	spont	9
<i>I(1)Q</i>	64	yes	no	ICR170	3
<i>I(1)R</i>	71	yes	no	various	12
<i>I(1)S</i>	4			spont	6, 14

^α 1 = Belitz, 1954, Z. Indukt. Abstamm. Vererbungsl. 86: 173-84; 2 = Belitz, 1956, DIS 30: 104; 3 = Carlson, Sederoff, and Cogan, 1967, Genetics 55: 295-313; 4 = Gershenson, 1934, DIS 1: 54; 5 = King, 1950, DIS 24: 58; 6 = Morgan and Bridges, 1916, Carnegie Inst. Wash. Publ. No. 237: 64, 79; 7 = Moriwaki, Jpn. J. Zool., 5: 585-602; 8 = Moriwaki, 1940, DIS 13: 50; 9 = Muller and Altenburg, 1919, Proc. Soc. Exp. Biol. Med. 17: 10-14; 10 = Plough and Ives, 1934, DIS 1: 32; 11 = Plough and Ives, 1934, Genetics 20: 42-69; 12 = Röhrborn, 1959, Z. Vererbungsl. 90: 116-31; 13 = Slizynski, 1938, Genetics 23: 283-90; 14 = Stark, 1915, J. Exp. Zool. 19: 531-38.

I(2)21B

Four lethally mutable loci, including *Gsl*, which encodes glutamine synthetase I, based on twenty recessive lethal mutations recovered following various mutagenic treatments (Caggese, Caizzi, Bozzetti, Barsanti, and Ritossa, 1988, Biochem. Genet. 26: 571-84).

locus	genetic location		cytological location		synonym
	included in	excluded from	included in	excluded from	
<i>I(2)21Ba</i>	2-(0)	21B2-5	<i>Df(2L)PM1</i>	<i>Df(2L)PM44</i>	
<i>I(2)21Bb</i>	2-(0)	21B2-5	<i>Df(2L)PM1</i>	<i>Df(2L)PM44</i>	
<i>I(2)21Bc</i>	2-0.1	21B3-6	<i>Df(2L)PM45</i>	<i>Df(2L)PM1</i>	Gsl
<i>I(2)21Bd</i>	2-(0)	21B4-6	<i>Df(2L)TE75</i>	<i>Df(2L)PM45</i>	

allele	origin	synonym
<i>I(2)21Ba</i> ¹	EMS	<i>I(2)M5</i>
<i>I(2)21Ba</i> ²	ENU	<i>I(2)R35</i>
<i>I(2)21Ba</i> ³	HD	<i>I(2)PM10</i>
<i>I(2)21Bb</i> ¹	EMS	<i>I(2)M10</i>
<i>I(2)21Bb</i> ²	EMS	<i>I(2)M11</i>
<i>I(2)21Bb</i> ³	ENU	<i>I(2)R31</i>
<i>I(2)21Bb</i> ⁴	ENU	<i>I(2)R212</i>
<i>I(2)21Bb</i> ⁵	HD	<i>I(2)PM58</i>
<i>I(2)21Bb</i> ⁶	HD	<i>I(2)PM59</i>
<i>I(2)21Bb</i> ⁷	HD	<i>I(2)PM158</i>
<i>I(2)21Bd</i> ¹	EMS	<i>I(2)M3</i>
<i>I(2)21Bd</i> ²	EMS	<i>I(2)M8</i>
<i>I(2)21Bd</i> ³	EMS	<i>I(2)M12</i>
<i>I(2)21Bd</i> ⁴	EMS	<i>I(2)C2</i>

I(2)21D-22A

Nineteen lethally mutable loci, including *S*, defined by complementation analysis of 55 ethyl-methanesulfonate-induced recessive lethal mutations uncovered by *Df(2L)S2* (Roberts, Brock, Rudden, and Evans-Roberts, 1985, Genetics 109: 145-56).

locus	genetic location	cytological location	included in	excluded from	synonym
I(2)21Da	21D1-2	<i>Df(2L)S3</i>	<i>Df(2L)ast3</i>	<i>l(2)neh1</i>	
I(2)21Db	21D1-2	<i>Df(2L)ast3</i>	<i>Df(2L)ast4</i>	<i>l(2)neh2</i>	
I(2)21Dc	21D1-2	<i>Df(2L)ast3</i>	<i>Df(2L)ast4</i>	<i>l(2)neh3</i>	
I(2)21Dd	21D1-E1	<i>Df(2L)ast4</i>	<i>Df(2L)ast6</i>	<i>l(2)neh4</i>	
I(2)21De	21D1-E1	<i>Df(2L)ast4</i>	<i>Df(2L)ast6</i>	<i>l(2)neh5</i>	
I(2)21Df	21D1-E1	<i>Df(2L)ast4</i>	<i>Df(2L)ast6</i>	<i>l(2)neh6</i>	
I(2)21Dg	21D1-E1	<i>Df(2L)ast4</i>	<i>Df(2L)ast6</i>	<i>l(2)neh7</i>	
I(2)21Ea	21E1-2	<i>Df(2L)ast6</i>	<i>Df(2L)ast5</i>	<i>l(2)neh8</i>	
I(2)21Eb	2-1.3	21E1-2	<i>Df(2L)ast5</i>	<i>S</i>	
I(2)21Fa	21E2-22A1	<i>Df(2L)ast5</i>	<i>Df(2L)ast6</i>	<i>l(2)neh9</i>	
I(2)21Fb	21E2-22A1	<i>Df(2L)ast5</i>	<i>Df(2L)ast6</i>	<i>l(2)neh10</i>	
I(2)21Fc	21E2-22A1	<i>Df(2L)ast5</i>	<i>Df(2L)ast6</i>	<i>l(2)neh11</i>	
I(2)21Fd	21E2-22A1	<i>Df(2L)ast5</i>	<i>Df(2L)ast6</i>	<i>l(2)neh12</i>	
I(2)21Fe	22A1-2	<i>Df(2L)ast10</i>	<i>Df(2L)S3</i>	<i>l(2)neh13</i>	

allele	origin	ref. ^α	synonym
I(2)21Da¹	EMS	1	<i>l(2)neh-1</i> ^{37-L}
I(2)21Db¹	EMS	1	<i>l(2)neh-2</i> ^{J4-4}
I(2)21Dc¹	EMS	1	<i>l(2)neh-3</i> ^{S3-1}
I(2)21Dd¹	EMS	1	<i>l(2)neh-4</i> ^{27-A}
I(2)21Dd²	EMS	1	<i>l(2)neh-4</i> ¹⁴⁻⁴
I(2)21De¹	EMS	1	<i>l(2)neh-5</i> ¹¹⁻³
I(2)21De²	EMS	1	<i>l(2)neh-5</i> ^{S3-2}
I(2)21De³	EMS	1	<i>l(2)neh-5</i> ^{14-C}
I(2)21De⁴	EMS	1	<i>l(2)neh-5</i> ^{35-A}
I(2)21De⁵	EMS	1	<i>l(2)neh-5</i> ^{12-L}
I(2)21Df¹	EMS	1	<i>l(2)neh-6</i> ^{J4-1}
I(2)21Dg¹	EMS	1	<i>l(2)neh-7</i> ^{24-B}
I(2)21Dg²	EMS	1	<i>l(2)neh-7</i> ¹⁴⁻⁵
I(2)21Ea¹	EMS	1	<i>l(2)neh-8</i> ^{Y2-1}
I(2)21Ea²	EMS	1	<i>l(2)neh-8</i> ^{Y5-2}
I(2)21Ea³	EMS	1	<i>l(2)neh-8</i> ^{4K}
I(2)21Ea⁴	EMS	1	<i>l(2)neh-8</i> ^F
I(2)21Fa¹	EMS	1	<i>l(2)neh-9</i> ^{J2-6}
I(2)21Fa²	EMS	1	<i>l(2)neh-9</i> ^{H1-3}
I(2)21Fa³	EMS	1	<i>l(2)neh-9</i> ^{20-M}
I(2)21Fa⁴	EMS	1	<i>l(2)neh-9</i> ^{S2-2}
I(2)21Fa⁵	EMS	1	<i>l(2)neh-9</i> ^{E-2}
I(2)21Fb¹	EMS	1	<i>l(2)neh-10</i> ²⁴⁻¹
I(2)21Fc¹	EMS	1	<i>l(2)neh-11</i> ^{28-K}
I(2)21Fd¹	EMS	1	<i>l(2)neh-12</i> ^{Y5-1}
I(2)21Fd²	EMS	1	<i>l(2)neh-12</i> ^{M1-4}
I(2)21Fd³	EMS	1	<i>l(2)neh-12</i> ^{27-K}
I(2)21Fd⁴	EMS	1	<i>l(2)neh-12</i> ^{24-K}
I(2)21Fd⁵	EMS	1	<i>l(2)neh-12</i> ^{J3-3}
I(2)21Fe¹	EMS	1	<i>l(2)neh-13</i> ^{X1-1}

^α 1 = Roberts, Brock, Rudden, and Evans-Roberts, 1985, Genetics 109: 145-56.

locus	cytological location	included in	excluded from	synonym
I(2)22Aa	22A1-B1	<i>Df(2L)S2</i>	<i>Df(2L)ast10</i>	<i>l(2)neh14</i>
I(2)22Ab	22A1-B1	<i>Df(2L)S2</i>	<i>Df(2L)ast10</i>	<i>l(2)neh15</i>
I(2)22Ac	22A1-B1	<i>Df(2L)S2</i>	<i>Df(2L)ast10</i>	<i>l(2)neh16</i>
I(2)22Ad	22A1-B1	<i>Df(2L)S2</i>	<i>Df(2L)ast10</i>	<i>l(2)neh17</i>
I(2)22Ae	22A1-B1	<i>Df(2L)S2</i>	<i>Df(2L)ast10</i>	<i>l(2)neh18</i>

allele	origin	synonym
I(2)22Aa¹	EMS	<i>l(2)neh-14</i> ^{25-K}
I(2)22Aa²	EMS	<i>l(2)neh-14</i> ^{J4-3}
I(2)22Aa³	EMS	<i>l(2)neh-14</i> ^{H5-2}
I(2)22Aa⁴	EMS	<i>l(2)neh-14</i> ^{26-K}
I(2)22Aa⁵	EMS	<i>l(2)neh-14</i> ^{5-K}
I(2)22Aa⁶	EMS	<i>l(2)neh-14</i> ^{37K}
I(2)22Ab¹	EMS	<i>l(2)neh-15</i> ^V
I(2)22Ab²	EMS	<i>l(2)neh-15</i> ^{L2-1}
I(2)22Ab³	EMS	<i>l(2)neh-15</i> ^{L1-2}
I(2)22Ab⁴	EMS	<i>l(2)neh-15</i> ^{F2}
I(2)22Ab⁵	EMS	<i>l(2)neh-15</i>
I(2)22Ab⁶	EMS	<i>l(2)neh-15</i> ^{X3-2}
I(2)22Ac¹	EMS	<i>l(2)neh-16</i> ^{21Q}
I(2)22Ac²	EMS	<i>l(2)neh-16</i> ^{38K}
I(2)22Ac³	EMS	<i>l(2)neh-16</i> ^{38L}
I(2)22Ac⁴	EMS	<i>l(2)neh-16</i> ^G
I(2)22Ac⁵	EMS	<i>l(2)neh-16</i> ^H
I(2)22Ad¹	EMS	<i>l(2)neh-17</i> ^{J2-8}
I(2)22Ad²	EMS	<i>l(2)neh-17</i> ^{5L}
I(2)22Ae¹	EMS	<i>l(2)neh-18</i> ^{X1-3}

I(2)22F

A series of eighteen ethyl methanesulfonate-induced recessive lethals falling into three complementation groups in the region uncovered by *Df(2L)dpp14* = *Df(2L)22E4-F1;22F3-23A1* (Spencer, Hoffman, and Gelbart, 1982, Cell 28: 451-61).

locus	genetic location	cytological location	included in	excluded from	synonym
I(2)22Fa	2-4.0	22F1-2	<i>Df(2L)dpp19</i>		dpp
I(2)22Fb	2-{4}	22F3	<i>Df(2L)dpp19</i>		<i>l(2)ND1</i>
I(2)22Fc	2-{4}	22F4-23A1	<i>Df(2L)dpp14</i>	<i>Dp(2;2)DTD48</i>	<i>l(2)ND2</i>
I(2)22Fd	2-{4}	22F4-23A1	<i>Df(2L)dpp14</i>	<i>Dp(2;2)DTD48</i>	<i>l(2)ND3</i>

DEFICIENCY MAP OF REGION 21D-22A

side	breakpoint	variant
		net
right	21B2-4	<i>Df(2L)PM44</i>
right	21B2-4	<i>Df(2L)PM82</i>
		I(2)21Ba
		I(2)21Bb
right	21B3-5	<i>Df(2L)PM1</i>
right	21B3-5	<i>Df(2L)PM51</i>
right	21B3-5	<i>Df(2L)PM59</i>
		Gsl
right	21B4-6	<i>Df(2L)PMG</i>
right	21B4-6	<i>Df(2L)PM91</i>
right	21B4-6	<i>Df(2L)PM45</i>
		I(2)21Bd
right	21B4-6	<i>Df(2L)TE75</i>
		ai
left	21C6-D1	<i>Df(2L)S2</i>
left	21C7-8	<i>Df(2L)ast1</i>
left	21D1-2	<i>Df(2L)ast2</i>
		ds
left	21D2-3	<i>Df(2L)ast10</i>

side	breakpoint	variant
left	21D2	<i>Df(2L)S3</i> I(2)21Da
left	21D1-2	<i>Df(2L)ast3</i> I(2)21Db I(2)21Dc
left	21D1-2	<i>Df(2L)ast4</i> I(2)21Dd I(2)21De I(2)21Df Lsp1β I(2)21Dg
left	21E1-2	<i>Df(2L)ast6</i> I(2)21Ea
left	21E1-2	<i>Df(2L)ast5</i> S
right	21E1-2	<i>Df(2L)ast3</i>
right	21E1-2	<i>Df(2L)ast4</i>
right	21E1-2	<i>Df(2L)ast6</i> I(2)21Fa I(2)21Fb I(2)21Fc I(2)21Fd
right	21F3-22A1	<i>Df(2L)ast5</i>
right	21F3-22A1	<i>Df(2L)S3</i> I(2)21Fe
right	22A1-2	<i>Df(2L)ast10</i> I(2)22Aa I(2)22Ab I(2)22Ac I(2)22Ad I(2)22Ae
right	22A6-B1	<i>Df(2L)S2</i>
right	22B2-3	<i>Df(2L)ast2</i>
left	22E2-4	<i>Dp(2;2)DTD48</i>
left	22F1-2	dpp^{sv} dpp^{Hln}
left	22F1-2	<i>Df(2L)dpp14</i>
right	22F1-2	<i>Dp(2;2)F21</i>
left	22F2-3	<i>Df(2L)dpp19</i> dpp I(2)22Fb
right	22F3-4	<i>Df(2L)dpp19</i>
right	22F4-23A1	<i>Dp(2;2)DTD48</i> I(2)22Fc I(2)22Fd
right	22F4-23A1	I(2)22Fc
right	22F4-23A1	I(2)22Fd
right	22F3-23A1	<i>Df(2L)dpp14</i>

I(2)24D-F

A series of lethal complementation groups covered by *Dp(2;1)B19* by Szidonya and Reuter (1988, Genet. Res. 51: 197-208). These lethals and those in the same region [*I(2)24EF*] recovered by Roy, Manna, and Duttagupta (1984, J. Biosci. 6: 87-95) not tested for complementation.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>I(2)24Da</i>	2-12.0	24D4-8	<i>Df(2L)sc19-1</i>	<i>Df(2L)M24F</i>	ft. I(2)fd
<i>I(2)24Db</i>	2-{12}	24D4-8	<i>Df(2L)sc19-1</i>	<i>Df(2L)M24F</i>	M(2)24D
I(2)24Dc	2-{12}	24D4-8	<i>Df(2L)sc19-1</i>	<i>Df(2L)M24F</i>	<i>I(2)jif1</i>
I(2)24Dd	2-{12}	24D4-8	<i>Df(2L)sc19-1</i>	<i>Df(2L)M24F</i>	<i>I(2)jif2</i>
I(2)24De	2-{12}	24D4-8	<i>Df(2L)sc19-1</i>	<i>Df(2L)M24F</i>	<i>I(2)jif3</i>
I(2)24Df	2-{12}	24D8-E3	<i>Df(2L)M24F</i>	<i>Df(2L)sc19-3</i>	<i>I(2)jif4</i>
I(2)24Ea	2-{13}	24E5-F2	<i>Df(2L)dp-h19</i>	<i>Df(2L)sc19-6</i>	<i>I(2)jif5</i>
I(2)24Fa	2-{13}	24F1-25A1	<i>Df(2L)sc19-6</i>	<i>Df(2L)dp-h24</i>	<i>I(2)jif6</i>
<i>I(2)24Fb</i>	2-12.9	24F1-25A1	<i>Df(2L)dp-h24</i>	<i>Df(2L)dp-cl-h3</i>	M(2)24F
I(2)24Fc	2-{13}	24F1-25A1	<i>Df(2L)dp-h24</i>	<i>Df(2L)dp-cl-h3</i>	<i>I(2)jif7</i>
<i>I(2)24Fd</i>	2-13.0	24F7-25A1	<i>Df(2L)dp-cl-h3</i>	<i>Df(2L)M24F</i>	dp

allele	synonym
I(2)24Dc¹	<i>I(2)jif1</i> <i>sz5</i>
I(2)24De¹	<i>I(2)jif2</i> <i>a6</i>
I(2)24De²	<i>I(2)jif2</i> <i>b8</i>
I(2)24Df¹	<i>I(2)jif3</i> <i>b25</i>
I(2)24Df²	<i>I(2)jif3</i> <i>sz11</i>
I(2)24Df³	<i>I(2)jif3</i> <i>sz49</i>
I(2)24Df⁴	<i>I(2)jif3</i> <i>sz56</i>
I(2)24Ea¹	<i>I(2)jif4</i> <i>b11</i>
I(2)24Fa	<i>I(2)jif6</i> <i>sz3</i>
I(2)24Fc¹	<i>I(2)jif7</i> <i>h6</i>
I(2)24Fc²	<i>I(2)jif7</i> <i>h32</i>
I(2)24Fc³	<i>I(2)jif7</i> <i>h36</i>
I(2)24Fc⁴	<i>I(2)jif7</i> <i>h39</i>

I(2)24EF

A series of mutants recovered by virtue of their lethal phenotype in combination with *Df(2L)M24F* by Roy, Manna, and Duttagupta (1984, J. Biosci. 6: 87-95). Not tested against alleles recovered by Szidonya and Reuter. Deficiencies inferred by failure to complement more than a single complementation group not detected cytologically. The order of loci *a*, *b*, *c*, and *d* with respect to each other, but not their polarity, inferred from these deficiencies; the order of the remaining loci with respect to both *a* through *d* and each other unknown. Despite the presence of *dw-24E* and *dp* in region monitored, the authors report recovery of no visible mutations.

locus	genetic location		cytological location		synonym
	location	location	included in	excluded from	
I(2)24EFa¹	1-{13}	24E1-25A1	<i>Df(2L)RMD202</i>	<i>Df(2L)RMD239</i>	<i>I(2)Gr.I</i>
I(2)24EFa²	1-{13}	24E1-25A1	<i>Df(2L)RMD239</i>	<i>Df(2L)RMD113</i>	<i>I(2)Gr.II</i>
I(2)24EFc	1-{13}	24E1-25A1	<i>Df(2L)RMD239</i> <i>Df(2L)RMD113</i>		<i>I(2)Gr.III</i>
I(2)24EFd	1-{13}	24E1-25A1	<i>Df(2L)RMD113</i>	<i>Df(2L)RMD239</i>	<i>I(2)Gr.IV</i>
I(2)24EFe	1-{13}	24E1-25A1	<i>Df(2L)M24F</i>	<i>Df(2L)RMD42</i>	<i>I(2)Gr.V</i>
I(2)24EFf	1-{13}	24E1-25A1	<i>Df(2L)M24F</i>	<i>Df(2L)RMD42</i>	<i>I(2)Gr.VI</i>
I(2)24EFg	1-{13}	24E1-25A1	<i>Df(2L)M24F</i>	<i>Df(2L)RMD42</i>	<i>I(2)Gr.VII</i>
<i>I(2)24EFh</i>	2-12.9	24E1-25A1	<i>Df(2L)M24F</i>	<i>Df(2L)RMD42</i>	M(2)24F

allele	origin	synonym
I(2)24EFa¹	EMS	<i>I(2)j32</i>
I(2)24EFa²	EMS	<i>I(2)j55</i>
I(2)24EFa³	EMS	<i>I(2)j103</i>
I(2)24EFb¹	EMS	<i>I(2)j378</i>
I(2)24EFb²	EMS	<i>I(2)j387</i>
I(2)24EFc¹	X ray	<i>I(2)j03</i>
I(2)24EFc²	EMS	<i>I(2)j05</i>
I(2)24EFc³	EMS	<i>I(2)j71</i>
I(2)24EFc⁴	EMS	<i>I(2)j72</i>
I(2)24EFc⁵	EMS	<i>I(2)j83</i>
I(2)24EFc⁶	EMS	<i>I(2)j92</i>
I(2)24EFc⁷	EMS	<i>I(2)j112</i>
I(2)24EFc⁸	EMS	<i>I(2)j125</i>
I(2)24EFc⁹	EMS	<i>I(2)j126</i>
I(2)24EFc¹⁰	EMS	<i>I(2)j144</i>
I(2)24EFc¹¹	EMS	<i>I(2)j193</i>
I(2)24EFc¹²	EMS	<i>I(2)j241</i>
I(2)24EFc¹³	EMS	<i>I(2)j243</i>
I(2)24EFc¹⁴	EMS	<i>I(2)j256</i>
I(2)24EFc¹⁵	EMS	<i>I(2)j257</i>
I(2)24EFc¹⁶	EMS	<i>I(2)j268</i>
I(2)24EFc¹⁷	EMS	<i>I(2)j279</i>

allele	origin	synonym
<i>l(2)24EFc</i>¹⁸	EMS	<i>l(2)320</i>
<i>l(2)24EFc</i>¹⁹	X ray	<i>l(2)336</i>
<i>l(2)24EFc</i>²⁰	EMS	<i>l(2)346</i>
<i>l(2)24EFc</i>²¹	EMS	<i>l(2)785</i>
<i>l(2)24EFd</i>¹	X ray	<i>l(2)381</i>
<i>l(2)24EFe</i>¹	EMS	<i>l(2)2296</i>
<i>l(2)24EFf</i>¹	EMS	<i>l(2)162</i>
<i>l(2)24EFg</i>¹	EMS	<i>l(2)178</i>

DEFICIENCY MAP OF REGION 24D, E, and F

side	breakpoint	variant ^α	variant ^β
right	24D4	<i>Dp(2;1)B19</i> ed	
left	24C3-5	<i>Df(2L)jed-dp-h1</i>	
left	24D2-4	<i>Df(2L)sc19-11</i>	
left	24D3-4	<i>Df(2L)M11</i>	
left	24D4-5	<i>Df(2L)sc19-1</i> ft M(2)24D <i>l(2)24Dc</i> <i>l(2)24Dd</i> <i>l(2)24De</i>	
left	24D7-8	<i>Df(2L)dp-h28</i>	
left	24D8-E1	<i>Df(2L)M24F</i>	<i>Df(2L)M24F</i>
left			<i>Df(2L)RMD42</i>
left			<i>Df(2L)RMD202</i>
left			<i>Df(2L)RMD261</i>
left			<i>Df(2L)RMD269</i>
left			<i>l(2)24EFa</i>
left			<i>Df(2L)RMD149</i>
left			<i>Df(2L)RMD239</i>
left			<i>l(2)24EFb</i>
left			<i>Df(2L)RMD113</i>
left			<i>l(2)24EFc</i>
right			<i>Df(2L)RMD113</i>
right			<i>Df(2L)RMD239</i>
right			<i>Df(2L)RMD202</i>
right			<i>Df(2L)RMD261</i>
right			<i>l(2)24EFd</i>
right			<i>Df(2L)RMD42</i>
right			<i>Df(2L)RMD149</i>
right			<i>l(2)24EFe</i>
right			<i>l(2)24EFf</i>
left	24E2-3	<i>l(2)24Df</i> <i>Df(2L)sc19-3</i>	
left	24E2-4	<i>Df(2L)dp-h25</i>	
left	24E2-4	<i>Df(2L)sc19-13</i> dw24E	
left	24E5-F1	<i>Df(2L)dp-h19</i> <i>l(2)24Ea</i>	
left	24F1-2	<i>Df(2L)sc19-6</i> <i>l(2)24Fa</i>	
left		<i>Df(2L)dp-h24</i> M(2)24F <i>l(2)24Fc</i>	M(2)24F
left	24F7-25A1	<i>Df(2L)dp-cl-h3</i> Su(var)2-8 dp	
right	24F7-25A1	<i>Df(2L)dp-h28</i>	
right	24F7-25A1	<i>Df(2L)M24F</i>	<i>Df(2L)M24F</i>
right			<i>Df(2L)RMD269</i>
right	24F7-25A1	<i>Df(2L)dp-h19</i>	
right		<i>Df(2L)dp-h24</i>	

^α Search of Szidonya and Reuter (1988, Genet. Res. 51: 197-208).
^β Search of Roy, Manna, and Duttagupta (1984, J. Biosci. 6: 87-95).
 Variants common to the two searches occupy the same lines in the table.

l(2)25

The entire region screened extensively for lethal mutations by Szidonya and Reuter (1988, Genet. Res. 51: 197-208). In addition, the region deleted by *Df(2L)GpdhA*, which is deficient for 25E through 26A has yielded seventeen lethally mutable loci in the region (Kotarski, Pickert, and MacIntyre, 1983, Genetics 105: 371-86). Complementation tests have not been carried out between mutations isolated in the two screens.

l(2)25A & B

Nine lethally mutable loci, the most distal of which is *slf*.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(2)25Aa</i>	2-15	25A4-B1	<i>Df(2L)sc19-4</i> <i>Df(2L)sc19-3</i>		<i>slf</i>
<i>l(2)25Ab</i>	2-{13}	25A4-B1	<i>Df(2L)sc19-4</i> <i>Df(2L)sc19-3</i>		<i>l(2)jif9</i>
<i>l(2)25Ba</i>	2-{13}	25A7-B4	<i>Df(2L)sc19-11</i>	<i>Df(2L)sc19-3</i>	<i>l(2)jif10</i>
<i>l(2)25Bb</i>	2-{13}	25A7-B4	<i>Df(2L)sc19-11</i>	<i>Df(2L)sc19-3</i>	<i>l(2)jif11</i>
<i>l(2)25Bc</i>	2-{13}	25B2-B5	<i>Df(2L)sc19-13</i>	<i>Df(2L)sc19-11</i>	<i>l(2)jif12</i>
<i>l(2)25Bd</i>	2-{13}	25B2-C1	<i>Df(2L)sc19-11</i>	<i>Df(2L)sc19-3</i>	<i>l(2)jif13</i>
<i>l(2)25Be</i>	2-{13}	25B2-C1	<i>Df(2L)sc19-11</i>	<i>Df(2L)sc19-3</i>	<i>l(2)jif14</i>
<i>l(2)25Bf</i>	2-{13}	25B2-C1	<i>Df(2L)sc19-11</i>	<i>Df(2L)sc19-3</i>	<i>l(2)jif15</i>
<i>l(2)25Bg</i>	2-{13}	25B2-C1	<i>Df(2L)sc19-11</i>	<i>Df(2L)sc19-3</i>	<i>l(2)jif16</i>

allele	origin	synonym
<i>l(2)25Ab</i>¹	EMS	<i>l(1)jif9</i> ^{b26}
<i>l(2)25Ba</i>¹	EMS	<i>l(2)jif10</i> ^{a9}
<i>l(2)25Bb</i>¹	EMS	<i>l(1)jif11</i> ^{sz59}
<i>l(2)25Bc</i>¹	EMS	<i>l(1)jif12</i> ^{sz17}
<i>l(2)25Bc</i>²	EMS	<i>l(1)jif12</i> ^{sz27}
<i>l(2)25Bd</i>¹	EMS	<i>l(1)jif13</i> ^{h4}
<i>l(2)25Bd</i>²	EMS	<i>l(1)jif13</i> ^{sz18}
<i>l(2)25Bd</i>³	EMS	<i>l(1)jif13</i> ^{sz19}
<i>l(2)25Be</i>¹	EMS	<i>l(1)jif14</i> ^{h7}
<i>l(2)25Be</i>²	EMS	<i>l(1)jif14</i> ^{h21}
<i>l(2)25Bf</i>¹	EMS	<i>l(1)jif15</i> ^{a4}
<i>l(2)25Bf</i>²	EMS	<i>l(1)jif15</i> ^{h12}
<i>l(2)25Bg</i>¹	EMS	<i>l(1)jif16</i> ^{a11}
<i>l(2)25Bg</i>²	EMS	<i>l(1)jif16</i> ^{b15}

l(2)25C & D

Eleven lethally mutable loci including *M(2)25C* and *tkv*; also, *l(2)25Ca* has dominant temperature-sensitive alleles and was described originally as *DTS*.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(2)25Ca</i>	2-{15}	25B9-C3	<i>Dp(2;2)B17</i>	<i>Df(2L)sc19-10</i>	<i>DTS</i>
<i>l(2)25Cb</i>	2-{15}	25B9-C3	<i>Df(1)sc19-7</i>	<i>Dp(2;2)B17</i>	<i>M(2)25C</i>
<i>l(2)25Cc</i>	2-{15}	25B9-C3	<i>Df(1)sc19-7</i>	<i>Dp(2;2)B17</i>	<i>l(2)jif17</i>
<i>l(2)25Cd</i>	2-{15}	25B9-C3	<i>Df(1)sc19-7</i>	<i>Dp(2;2)B17</i>	<i>l(2)jif18</i>
<i>l(2)25Ce</i>	2-{15}	25C3-5	<i>Df(2L)sc19-6</i>	<i>Df(1)sc19-7</i>	<i>l(2)jif19</i>
<i>l(2)25Cf</i>	2-{15}	25C3-5	<i>Df(2L)sc19-6</i>	<i>Df(1)sc19-7</i>	<i>l(2)jif20</i>
<i>l(2)25Cg</i>	2-{15}	25C3-8	<i>Df(2L)sc19-8</i>	<i>Df(1)sc19-6</i>	<i>l(2)jif21</i>
<i>l(2)25Ch</i>	2-{15}	25C8-9	<i>Df(2L)sc19-1</i>	<i>Df(1)sc19-8</i>	<i>l(2)jif22</i>
<i>l(2)25Da</i>	2-16	25D2-6	<i>Df(2L)cl2</i> <i>Df(2L)tkv-sz3</i>		<i>tkv</i>
<i>l(2)25Db</i>	2-{16}	25D2-6		<i>Df(2L)tkv-sz3</i> <i>Df(1)cl7</i>	<i>l(2)jif23</i>

locus	genetic cytological		included in	excluded from	synonym
	location	location			
l(2)25Dc	2-16}	25D5-E1	Df(2L)c17	Df(1)c4	l(2)jf24

l(2)25Ca

location: 2-15}.

origin: Induced by ethyl methanesulfonate; selected as dominant temperature-sensitive, dominant cold-sensitive lethal, or recessive lethal mutations.

references: Suzuki and Procnier, 1969, Proc. Nat. Acad. Sci. USA 62: 369-76.

Rosenbluth, Ezell, and Suzuki, 1972, Genetics 70: 75-86.

Szidonya and Reuter, 1988, Genet. Res. 51: 197-208.

phenotype: Heterozygotes for dominant temperature-sensitive alleles lethal or nearly so when raised at 29° but survive at 25°; +/- heterozygotes survive development at 29°. Heterozygotes for dominant cold-sensitive alleles lethal or nearly so when raised at 17° but survive at 22°; adults insensitive to 17°. Heat-sensitive period 18-24 hr after ovoposition; lethal phase varies, but most frequently at 75-90 hr for heat-sensitive alleles and late in development, but highly variable for cold-sensitive alleles. Heterozygotes for alleles selected as recessive lethals are mostly not dominant temperature-sensitives.

alleles:

allele	origin	synonym	ref ^α	comments
l(2)25Ca¹	EMS	DTS-L2	2	dominant heat sensitive
l(2)25Ca²	EMS	DTS-L3	2	dominant heat sensitive
l(2)25Ca³	EMS	DTS-L4	2	dominant heat sensitive
l(2)25Ca⁴	EMS	DTS-L5	2	dominant heat sensitive
l(2)25Ca⁵	EMS	DTS-L7	2	dominant heat sensitive
l(2)25Ca⁶	EMS	DTS-L10	2	dominant heat sensitive
l(2)25Ca⁷	EMS	DTS-L11	2	dominant heat sensitive
l(2)25Ca⁸	EMS	DTS-L12	2	dominant heat sensitive
l(2)25Ca⁹	EMS	DTS-L13	2	dominant heat sensitive
l(2)25Ca¹⁰	EMS	DTS-L14	2	dominant heat sensitive
l(2)25Ca¹¹	EMS	DTS-L16	2	dominant heat sensitive
l(2)25Ca¹²	EMS	DCS-L1	1	dominant cold sensitive
l(2)25Ca¹³	EMS	DCS-L2	1	dominant cold sensitive
l(2)25Ca¹⁴	EMS	DCS-L3	1	dominant cold sensitive
l(2)25Ca¹⁵	EMS	DCS-L4	1	dominant cold sensitive
l(2)25Ca¹⁶	EMS	DCS-L5	1	dominant cold sensitive
l(2)25Ca¹⁷	EMS	DCS-L6	1	dominant cold sensitive
l(2)25Ca¹⁸	EMS	DCS-L7	1	dominant cold sensitive
l(2)25Ca¹⁹	EMS	DCS-L8	1	dominant cold sensitive
l(2)25Ca²⁰	EMS	DCS-L9	1	dominant cold sensitive
l(2)25Ca²¹	EMS	DCS-L10	1	dominant cold sensitive
l(2)25Ca²²	EMS	DCS-L11	1	dominant cold sensitive
l(2)25Ca²³	EMS	DCS-L12	1	dominant cold sensitive
l(2)25Ca²⁴	EMS	DCS-L13	1	dominant cold sensitive
l(2)25Ca²⁵	EMS	DCS-L14	1	dominant cold sensitive
l(2)25Ca²⁶	EMS	DCS-L15	1	dominant cold sensitive
l(2)25Ca²⁷	EMS	DCS-L16	1	dominant cold sensitive
l(2)25Ca²⁸	EMS	DCS-L17	1	dominant cold sensitive
l(2)25Ca²⁹	EMS	DCS-L18	1	dominant cold sensitive
l(2)25Ca³⁰	EMS	DCS-L19	1	dominant cold sensitive
l(2)25Ca³¹	EMS	DCS-L20	1	dominant cold sensitive
l(2)25Ca³²	EMS	DCS-L21	1	dominant cold sensitive
l(2)25Ca³³	EMS	DCS-L22	1	dominant cold sensitive
l(2)25Ca³⁴	EMS	DCS-L23	1	dominant cold sensitive
l(2)25Ca³⁵	EMS	l(2)a30	1	dominant heat sensitive
l(2)25Ca³⁶	EMS	l(2)b9	3	slight dominant heat sensitive
l(2)25Ca³⁷	EMS	l(2)ab14	3	not dominant conditional
l(2)25Ca³⁸	EMS	l(2)b17	3	slight dominant heat sensitive
l(2)25Ca³⁹	EMS	l(2)b29	3	not dominant conditional
l(2)25Ca⁴⁰	EMS	l(2)b32	3	not dominant conditional
l(2)25Ca⁴¹	EMS	l(2)b35	3	not dominant conditional
l(2)25Ca⁴²	EMS	l(2)h12	3	not dominant conditional
l(2)25Ca⁴³	EMS	l(2)h30	3	not dominant conditional

allele	origin	synonym	ref ^α	comments
l(2)25Ca⁴⁴	EMS	l(2)sz6	3	not dominant conditional
l(2)25Ca⁴⁵	EMS	l(2)sz14	3	not dominant conditional
l(2)25Ca⁴⁶	EMS	l(2)sz74	3	not dominant conditional
l(2)25Ca⁴⁷	EMS	l(2)sz78	3	not dominant conditional

^α 1 = Rosenbluth, Ezell, and Suzuki, 1972, Genetics 70: 75-86; 2 = Suzuki and Procnier, 1969, Proc. Nat. Acad. Sci. USA 62: 369-76; 3 = Szidonya and Reuter, 1988, Genet. Res. 51: 197-208.

other information: Interallelic complementation of heat-sensitive alleles observed at 25°; complementation map circular with a tail. Circular complementation map also observed for cold-sensitive alleles at 22°. Cold-sensitive alleles fail to complement heat-sensitive allele **l(2)25Ca³**. Alleles selected as recessive lethals form a linear complementation map with dominant heat-sensitive alleles (Szidonya and Reuter, 1988).

allele	origin	synonym
l(2)25Cc¹	EMS	l(2)jf17 ^{b60}
l(2)25Cd¹	EMS	l(2)jf18 ^{sz77}
l(2)25Ce¹	EMS	l(2)jf19 ^{sz53}
l(2)25Cf¹	EMS	l(2)jf20 ^{sz70}
l(2)25Cg¹	EMS	l(2)jf21 ^{a2}
l(2)25Cg²	EMS	l(2)jf21 ^{a8}
l(2)25Cg³	EMS	l(2)jf21 ^{a16}
l(2)25Ch¹	EMS	l(2)jf22 ^{sz75}
l(2)25Db¹	EMS	l(2)jf23 ^{sz7}
l(2)25Db²	EMS	l(2)jf23 ^{sz36}
l(2)25Dc¹	EMS	l(2)jf24 ^{a1}
l(2)25Dc²	EMS	l(2)jf24 ^{a9}
l(2)25Dc³	EMS	l(2)jf24 ^{b19}
l(2)25Dc⁴	EMS	l(2)jf24 ^{h25}
l(2)25Dc⁵	EMS	l(2)jf24 ^{sz20}

l(2)25E

Seven lethally mutable loci including midline (*mid*). Isolated in a saturation study of the region uncovered by *Df(2L)GpdhA* by Kotarski, Pickert, and MacIntyre (1983, Genetics 105: 371-86). The mutation study of Szidonya and Reuter (1988, Genet. Res. 51: 197-208) also extends into the region. The deficiency maps from the two studies are highly concordant; however, there have been no complementation crosses between mutants from the two studies. Although each study groups six loci together as **l(2)25Ea** to **l(2)25Ef**, there is no reason to believe that they have been designated in the same order; nevertheless, we have designated them *Ea* and *Ea*, *Eb* and *Eb*, etc. with the understanding that complementation tests are required in order to establish that the primed and nonprimed alleles are in fact allelic.

locus	genetic cytological		included in	excluded from	synonym
	location	location			
l(2)25Ea	25D7-E2	Df(2L)GpdhA Y ^P 2 ^D B236			l(2)gdh11
l(2)25Eb	25D7-E2	Df(2L)GpdhA Y ^P 2 ^D B236			l(2)gdh12
l(2)25Ec	25D7-E4	Y ^P 2 ^D H164 Y ^P 2 ^D B236			l(2)gdh7
l(2)25Ed	25D7-E4	Y ^P 2 ^D H164 Y ^P 2 ^D B236			l(2)gdh8
l(2)25Ee	25D7-E4	Y ^P 2 ^D H164 Y ^P 2 ^D B236			l(2)gdh9
l(2)25Ef	25D7-E4	Y ^P 2 ^D H164 Y ^P 2 ^D B236			l(2)gdh10

THE GENOME OF *DROSOPHILA MELANOGASTER*

locus	genetic cytological		included in	excluded from	synonym
	location	location			
<i>l(2)25Eg</i>	2-17.91	25E1-F1		<i>Y^P 2^D H164</i>	<i>mid, l(2)gdh1 Df(2L)sc19-4</i>
allele	origin	synonym	ref	α	
<i>l(2)25Ea¹</i>	EMS	<i>l(2)gdh-11⁰⁷</i>	1		
<i>l(2)25Ea²</i>	EMS	<i>l(2)jif25^{b24}</i>	2		
<i>l(2)25Eb¹</i>	EMS	<i>l(2)gdh-12⁴⁰</i>	1		
<i>l(2)25Eb²</i>	EMS	<i>l(2)gdh-12³⁷⁰¹</i>	1		
<i>l(2)25Eb³</i>	EMS	<i>l(2)jif26^{sz15}</i>	2		
<i>l(2)25Ec¹</i>	EMS	<i>l(2)gdh-7^{25A}</i>	1		
<i>l(2)25Ec²</i>	EMS	<i>l(2)gdh-7⁹⁰¹</i>	1		
<i>l(2)25Ec³</i>	EMS	<i>l(2)jif27^{a7}</i>	2		
<i>l(2)25Ec⁴</i>	EMS	<i>l(2)jif27^{sz9}</i>	2		
<i>l(2)25Ec⁵</i>	EMS	<i>l(2)jif27^{sz18}</i>	2		
<i>l(2)25Ed¹</i>	EMS	<i>l(2)gdh-8⁰⁰¹</i>	1		
<i>l(2)25Ed²</i>	EMS	<i>l(2)gdh-8²⁶</i>	1		
<i>l(2)25Ed³</i>	EMS	<i>l(2)gdh-8²⁹</i>	1		
<i>l(2)25Ed⁴</i>	EMS	<i>l(2)gdh-8³⁸</i>	1		
<i>l(2)25Ed⁵</i>	EMS	<i>l(2)gdh-8¹⁵⁰¹</i>	1		
<i>l(2)25Ed⁶</i>	EMS	<i>l(2)jif28^{b2}</i>	2		
<i>l(2)25Ed⁷</i>	EMS	<i>l(2)jif28^{sz63}</i>	2		
<i>l(2)25Ee¹</i>	EMS	<i>l(2)gdh-9⁰¹</i>	1		
<i>l(2)25Ee²</i>	EMS	<i>l(2)gdh-9¹⁶</i>	1		
<i>l(2)25Ee³</i>	EMS	<i>l(2)gdh-9⁷⁰¹</i>	1		
<i>l(2)25Ee⁴</i>	EMS	<i>l(2)gdh-9²⁹⁰¹</i>	1		
<i>l(2)25Ee⁵</i>	EMS	<i>l(2)jif29^{b7}</i>	2		
<i>l(2)25Ef¹</i>	EMS	<i>l(2)gdh-10⁰³</i>	1		
<i>l(2)25Ef²</i>	EMS	<i>l(2)gdh-10⁴¹</i>	1		
<i>l(2)25Ef³</i>	EMS	<i>l(2)gdh-10²⁰¹</i>	1		
<i>l(2)25Ef⁴</i>	EMS	<i>l(2)jif30^{a26}</i>	2		
<i>l(2)25Ef⁵</i>	EMS	<i>l(2)jif30^{b23}</i>	2		

α 1 = Kotarski, Pickert, and MacIntyre, 1983, Genetics 105: 371-86;
2 = Szidonya and Reuter, 1988, Genet. Res. 51: 197-208.

l(2)25F

locus	genetic cytological		included in	excluded from	synonym
	location	location			
<i>l(2)25Fa</i>	2-{17}	25F2-3	<i>Df(2L)H151</i>	<i>Df(2L)50075a</i>	<i>l(2)gdh-14</i>
<i>l(2)25Fb</i>	2-{17}	25F2-3	<i>Df(2L)H151</i>	<i>Df(2L)50075a</i>	<i>l(2)gdh-17</i>
<i>l(2)25Fc</i>	2-{17}	25F2-26A1	<i>Df(2L)50075a</i>		<i>l(2)gdh-15</i>
<i>l(2)25Fd</i>	2-{17}	25F2-26A1	<i>Df(2L)2802</i>		<i>l(2)gdh-16</i>
<i>l(2)25Fe</i>	2-18.46	25F4-26A1	<i>Df(2L)50075a</i>	<i>Df(2L)2802</i>	<i>l(2)gdh-3</i>
allele	origin	synonym	ref	α	
<i>l(2)25Fa¹</i>	EMS	<i>l(2)gdh-14¹³</i>	1		
<i>l(2)25Fa²</i>	EMS	<i>l(2)gdh-14¹⁵</i>	1		
<i>l(2)25Fa³</i>	EMS	<i>l(2)gdh-14^{33B}</i>	1		
<i>l(2)25Fa⁴</i>	EMS	<i>l(2)gdh-14³⁰¹</i>	1		
<i>l(2)25Fa⁵</i>	EMS	<i>l(2)gdh-14⁸⁰²</i>	1		
<i>l(2)25Fa⁶</i>	EMS	<i>l(2)gdh-14²⁰⁰¹</i>	1		
<i>l(2)25Fa⁷</i>	EMS	<i>l(2)gdh-14³¹⁰²</i>	1		
<i>l(2)25Fa⁸</i>	EMS	<i>l(2)TE9</i>	2		
<i>l(2)25Fb¹</i>	EMS	<i>l(2)gdh-17¹⁰</i>	1		
<i>l(2)25Fc¹</i>	EMS	<i>l(2)gdh-15³⁰⁰²</i>	1		
<i>l(2)25Fd¹</i>	EMS	<i>l(2)gdh-16^{25B}</i>	1		
<i>l(2)25Fe¹</i>	EMS	<i>l(2)gdh-3³³⁰¹</i>	1		

α 1 = Kotarski, Pickert, and MacIntyre, 1983, Genetics 105: 371-86;
2 = Szidonya and Reuter, 1988, Genet. Res. 51: 197-208.

l(2)26A

Five lethally mutable genes isolated by virtue of their failure to complement *Df(2L)GpdhA* (Kotarski, Pickert, and MacIntyre, 1983, Genetics 105: 371-86).

locus	genetic cytological		included in	excluded from	synonym
	location	location			
<i>l(2)26Aa¹</i>	2-{20}	25F4-26A8	<i>Df(2L)cl7</i>	<i>Df(2L)50075a</i>	<i>l^x</i>
<i>l(2)26Ab</i>	2-20.55	26A7-9	<i>Df(2L)GpdhA</i>	<i>Df(2L)cl7</i>	<i>l(2)gdh-2</i>
<i>l(2)26Ac</i>	2-{20}	26A7-9	<i>Df(2L)GpdhA</i>	<i>Df(2L)cl7</i>	<i>l(2)gdh-4</i>
<i>l(2)26Ad</i>	2-{20}	26A7-9	<i>Df(2L)GpdhA</i>	<i>Df(2L)cl7</i>	<i>l(2)gdh-5</i>
<i>l(2)26Ae</i>	2-{20}	26A7-9	<i>Df(2L)GpdhA</i>	<i>Df(2L)cl7</i>	<i>l(2)gdh-6</i>

l(2)26Cf 2-{20} 26B9-27A1 *Y^P 2^D J136* *Y^P 2^D J70* *l(2)gdh-13*

α At least one lethally mutable locus inferred from the lethality of *Y^P 2^D D222/A^P Y^D H69/Df(2L)GpdhA* as well as the normal survival of *T(Y;2)D222/Df(2L)GpdhA* and *T(Y;2)H69/Df(2L)GpdhA*; no mutant alleles recovered.

allele	origin	synonym	comments
<i>l(2)26Ab¹</i>	EMS	<i>l(2)gdh-2⁸⁰¹</i>	
<i>l(2)26Ab²</i>	EMS	<i>l(2)gdh-2¹³⁰¹</i>	
<i>l(2)26Ab³</i>	EMS	<i>l(2)gdh-2²⁰⁰²</i>	
<i>l(2)26Ac¹</i>	EMS	<i>l(2)gdh-4^{401a}</i>	also <i>l(2)26Cf¹</i>
<i>l(2)26Ac²</i>	EMS	<i>l(2)gdh-4¹⁶⁰¹</i>	
<i>l(2)26Ac³</i>	EMS	<i>l(2)gdh-4³¹⁰¹</i>	
<i>l(2)26Ad¹</i>	EMS	<i>l(2)gdh-5²²⁰¹</i>	
<i>l(2)26Ae¹</i>	EMS	<i>l(2)gdh-6⁵⁰²</i>	
<i>l(2)26Ae²</i>	EMS	<i>l(2)gdh-6¹⁴⁰¹</i>	
<i>l(2)26Ae³</i>	EMS	<i>l(2)gdh-6³⁰⁰¹</i>	
<i>l(2)26Ae⁴</i>	EMS	<i>l(2)gdh-6³⁵⁰¹</i>	
<i>l(2)26Cf¹</i>	EMS	<i>l(2)gdh-13^{401b}</i>	also <i>l(2)26Ac¹</i>

DEFICIENCY MAP OF REGION 25 & 26A

side	breakpoint	variant α	variant β
right	25A2-3	<i>Df(2L)ed-dp-h1</i>	
right	25A2-3	<i>Df(2L)M11</i>	
left	25A4-5	<i>Df(2L)sc19-10</i>	
left	25A4-5	<i>Df(2L)sc19-5</i>	
left	25A4-5	<i>Df(2L)sc19-4</i>	
left	25A4-5	<i>Df(2L)sc19-12</i>	
left	25A4-5	<i>Df(2L)tkv-sz3</i>	
		sif	
right	25A7 B1	<i>Df(2L)sc19-3</i>	<i>l(2)25Ab</i>
			<i>l(2)25Ba</i>
			<i>l(2)25Bb</i>
right	25B2-4	<i>Df(2L)sc19-11</i>	
right	25B2-5	<i>Df(2L)dp-h25</i>	
		<i>l(2)25Bc</i>	
right	25B2-5	<i>Df(2L)sc19-13</i>	
		<i>l(2)25Bd</i>	
		<i>l(2)25Be</i>	
		<i>l(2)25Bf</i>	
		<i>l(2)25Bg</i>	
right	25B9-C1	<i>Df(2L)sc19-10</i>	
		<i>l(2)25Ca</i>	
right	25C3-8	<i>Dp(2;2)B17</i>	
		<i>M(2)25C</i>	
		<i>l(2)25Cc</i>	
		<i>l(2)25Cd</i>	
right	25C2-3	<i>Df(2L)sc19-7</i>	
		<i>l(2)25Ce</i>	
		<i>l(2)25Cf</i>	

side	breakpoint	variant α	variant β
right	25C3-5	<i>Df(2L)jsc19-6</i> I(2)25Cg	
right	25C8-9	<i>Df(2L)jsc19-8</i> I(2)25Ch	
right	25C8-9	<i>Df(2L)jsc19-1</i>	
left	25D2-4	<i>Df(2L)cl1</i>	
left	25D2-4	<i>Df(2L)cl2</i>	
left	25D2-4	<i>Df(2L)cl-h1</i>	
left	25D2-4	<i>Df(2L)cl-h3</i>	
left	25D2-4	<i>Df(2L)tkv-sz2</i> tkv	
right	25D5-E1	<i>Df(2L)tkv-sz-3</i> I(2)25Db	
left	25D5-6	<i>Df(2L)cl7</i>	
left	25D5-6	<i>Df(2L)cl-h2</i>	
right	25D5-7	<i>Df(2L)jsc19-5</i> I(2)25Dc	
left	25D6-E1	<i>Df(2L)cl-h4</i>	
left	25D7-E1	<i>Df(2L)GpdhA</i> I(2)25Ea α I(2)25Eb α	I(2)25Ea I(2)25Eb
	25D7-E2	<i>T(Y;2)B236</i> cl I(2)25Ec α I(2)25Ed α I(2)25Ee α I(2)25Ef α	cl I(2)25Ec I(2)25Ed I(2)25Ee I(2)25Ef
right	25E1-2	<i>Df(2L)D6</i>	
	25E1-4	<i>T(Y;2)H164</i>	
right	25D6-E1	<i>Df(2L)tkv-sz2</i>	
right	25E5-F1	<i>Df(2L)cl-h4</i> mid	I(2)25Eg
right	25E5-F1	<i>Df(2L)jsc19-4</i>	<i>Df(2L)jsc19-4</i>
right	25F1-2	<i>Df(2L)cl-h1</i> E(var)	
right	25F2-4	<i>Df(2L)cl2</i>	<i>Df(2L)cl2</i>
right	25F2-4	<i>Df(2L)cl1</i>	<i>Df(2L)cl1</i>
right	25F2	<i>Dp(2;1)B19</i>	
left	25F2-3	<i>Df(2L)j2802</i>	
left	25F2-3	<i>Df(2L)H151</i> I(2)25Fa α	I(2)25Fa
left	25F2-3	<i>Df(2L)J50075a</i>	
left	25F2-3	<i>Df(2L)J50078a</i> I(2)25Fb I(2)25Fc	
right	25F4-26A1	<i>Df(2L)j2802</i>	
	25F4-5	<i>Df(2L)cl-h2</i> I(2)25Fd	
	25F5	α Gpdh	
right	25F4-26A1	<i>Df(2L)J50075a</i>	
right	25F4-26A1	<i>Df(2L)J50078a</i>	
	26A2-3	<i>T(Y;2)D222</i> I(2)26Aa	
	26A4-5	<i>T(Y;2)H69</i>	
right	26A7-8	<i>Df(2L)cl7</i>	<i>Df(2L)cl7</i>
	26A8	β Gal	
	26A8	I(2)26Ab	
	26A8	I(2)26Ac	
	26A8	I(2)26Ad	
	26A8	I(2)26Ae	
right	26A8-9	<i>Df(2L)GpdhA</i>	
right	26B2-5	<i>Df(2L)cl-h3</i>	
	26B2-5	<i>T(Y;2)D211</i>	
	26B9-C1	<i>T(Y;2)J70</i> I(2)26Cf	
	26E4-27A1	<i>T(Y;2)J136</i>	
	27C1-E1	spd	
	27C9-E1	<i>T(Y;2)A171</i>	

α Szidonya and Reuter, 1988, Genet. Res. 51: 197-208.
 β Kotarski, Pickert, and MacIntyre, 1983, Genetics 103: 371-86.

I(2)34D

locus	genetic		cytological		synonym
	location	location	included in	excluded from	
I(2)34Da	2-48.3	34D1-2	<i>Df(2L)b82a1</i>	<i>Df(2L)b75</i>	<i>I(2)br38</i>
I(2)34Db	2-{48.3}	34D3	<i>Df(2L)b75</i>	<i>Df(2L)jSco7</i>	<i>I(2)br20</i> <i>I(2)br18</i>
I(2)34Dc	2-{48.4}	34D4-5	<i>Df(2L)Sco7</i>	<i>Df(2L)j82a3</i>	<i>I(2)br32</i>
I(2)34Dd	2-{48.3}	34D4-5	<i>Df(2L)b82a3</i> <i>Df(2L)b81h1</i>		<i>I(2)br5</i>
I(2)34De	2-{48.6}	34D6-8	<i>Df(2L)b8311</i>	<i>Df(2L)b81h1</i>	<i>I(2)br16</i>
I(2)34Df	2-{48.6}	34D6-8	<i>Df(2L)b8311</i>	<i>Df(2L)b81h1</i>	<i>I(2)br39</i>
I(2)34Dg	2-{48.6}	34D6-8	<i>Df(2L)b8311</i>	<i>Df(2L)b81h1</i>	<i>I(2)br17</i>
I(2)34Ea	2-{48.6}	34E1-2	<i>Df(2L)b84a9</i>	<i>Df(2L)b8311</i>	<i>I(2)br24</i> <i>I(2)br25</i>
I(2)34Eb	2-{48.6}	34E3	<i>Df(2L)b80f1</i>	<i>Df(2L)b84a9</i> <i>Df(2L)jfn7</i>	<i>I(2)br31</i>
I(2)34Fa	2-{48.8}	34F1-2	<i>Df(2L)jfn7</i>	<i>Df(2L)jfn1</i>	<i>I(2)br30</i>
I(2)34Fb	2-{48.9}	34F3-4	<i>Df(2L)jfn1</i>	<i>Df(2L)A217</i>	wb <i>I(2)br1</i>
I(2)34Fc	2-{48.9}	34F4-35A1	<i>Df(2L)A217</i>	<i>Df(2L)TE35A-7</i>	<i>I(2)br8</i>
I(2)34Fd	2-{49.0}	34F4-35A1	<i>Df(2L)A217</i>	<i>Df(2L)TE35A-7</i>	<i>I(2)br15</i>

I(2)34Da

allele	origin	discoverer	synonym
I(2)34Da ¹	TE	Ising	TE13
I(2)34Da ²	TE	Ising	TE60
I(2)34Da ³	TE	Ising	TE94

I(2)34Db

phenotype: Some combinations of alleles [*i.e.*, *I(2)34Db*⁸/*I(2)34Db*⁹] may escape; escapers have rough eyes (variable), etched wing costae, and show loss of ocellar, anterior alar and posterior scutellar bristles; bristles thin. Male and female sterile.

allele	origin	discoverer	synonym	ref α
I(2)34Db ¹	EMS	Ashburner	SF2	1
I(2)34Db ²	EMS	Ashburner	SF14	1
I(2)34Db ³	EMS	Ashburner	SF29	1
I(2)34Db ⁴	EMS	Maroni	GM14	
I(2)34Db ⁵	EMS	Maroni	GM15	
I(2)34Db ⁶	EMS	O'Donnell	CH5	
I(2)34Db ⁷	EMS	O'Donnell	CH7	
I(2)34Db ⁸	EMS	O'Donnell	CH13	
I(2)34Db ⁹	EMS	O'Donnell	CH16	
I(2)34Db ¹⁰	EMS	Lindsley	64-639	
I(2)34Db ¹¹	EMS	Lindsley	64-668	
I(2)34Db ¹²	EMS	Lindsley	64-1316	

α 1 = Woodruff and Ashburner, 1979, Genetics 92: 133-49.

I(2)34Dc

phenotype: *I(2)34Dc*³ produces about 1% escapers in heterozygous combination with both *I(2)34Dc*¹ and *I(2)34Dc*².

allele	origin	discoverer	synonym	ref α	comments
I(2)34Dc ¹	EMS	O'Donnell	CH60		
I(2)34Dc ²	EMS	O'Donnell	CH69		
I(2)34Dc ³	spont	Woodruff	SW27		
I(2)34Dd ¹	EMS	Bodmer and Walker	BMW29		
I(2)34Dd ²	EMS	Ashburner	SF22	1	
I(2)34Dd ³	EMS	Ashburner	SF23	1	
I(2)34Dd ⁴	EMS	Ashburner	SF28	1	
I(2)34Dd ⁵	EMS	O'Donnell	CH28		
I(2)34Dd ⁶	EMS	O'Donnell	CH40		
I(2)34Dd ⁷	EMS	O'Donnell	CH58		

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)34Dd</i>⁸	EMS	Lindsley	<i>64-449</i>		
<i>l(2)34De</i>¹	EMS	Ashburner	<i>SF1</i>	1	
<i>l(2)34De</i>²	spont?	Ashburner	<i>SF16</i>	1	on <i>ln(2LR)Gla</i>
<i>l(2)34De</i>³	EMS	O'Donnell	<i>CH30</i>		
<i>l(2)34Di</i>¹	spont	Woodruff	<i>BG1</i>		
<i>l(2)34Di</i>²	EMS	Lindsley	<i>64-480</i>		
<i>l(2)34Dg</i>¹	EMS	Ashburner	<i>SF10</i>	1	
<i>l(2)34Dg</i>¹	EMS	O'Donnell	<i>CH17</i>		

^α 1 = Woodruff and Ashburner, 1979, Genetics 92: 133-49.

l(2)34Ea

phenotype: Recessive lethal or semilethal; heteroallelic or hemizygous escapers have small rough eyes; heterozygotes between two weak alleles viable with rough eyes.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)34Ea</i>¹	EMS	Bodmer and Walker	<i>BMW7</i>		strong allele
<i>l(2)34Ea</i>²	EMS	Bodmer and Walker	<i>BMW19</i>		strong allele
<i>l(2)34Ea</i>³	EMS	Harrington	<i>HG14</i>		strong allele
<i>l(2)34Ea</i>⁴	EMS	Harrington	<i>HG16</i>		strong allele
<i>l(2)34Ea</i>⁵	EMS	Ashburner	<i>SF4</i>	1	weaker allele
<i>l(2)34Ea</i>⁶	EMS	Ashburner	<i>SF15</i>	1	strong allele
<i>l(2)34Ea</i>⁷	EMS	O'Donnell	<i>DM7</i>		strong allele
<i>l(2)34Ea</i>⁸	EMS	O'Donnell	<i>CH18</i>		strong allele
<i>l(2)34Ea</i>⁹	EMS	O'Donnell	<i>CH14</i>		strong allele
<i>l(2)34Ea</i>¹⁰	EMS	O'Donnell	<i>CH24</i>		weaker allele
<i>l(2)34Ea</i>¹¹	EMS	Maroni	<i>GM8</i>		weaker allele
<i>l(2)34Ea</i>¹²	EMS	Maroni	<i>GM9</i>		weaker allele
<i>l(2)34Ea</i>¹³	EMS	Lindsley	<i>64-408</i>		strong allele
<i>l(2)34Ea</i>¹⁴	EMS	Lindsley	<i>64-460</i>		weaker allele
<i>l(2)34Ea</i>¹⁵	EMS	Lindsley	<i>64-465</i>		strong allele
<i>l(2)34Ea</i>¹⁶	EMS	Lindsley	<i>64-504</i>		strong allele
<i>l(2)34Ea</i>¹⁷	EMS	Lindsley	<i>64-509</i>		strong allele
<i>l(2)34Ea</i>¹⁸	EMS	Lindsley	<i>64-734</i>		strong allele
<i>l(2)34Ea</i>¹⁹	EMS	Lindsley	<i>64-1216</i>		strong allele
<i>l(2)34Ea</i>²⁰	EMS	Lindsley	<i>64-1253</i>		strong allele

^α 1 = Woodruff and Ashburner, 1979, Genetics 92: 133-49.

l(2)34Eb

phenotype: Heteroallelic combinations escape about 10% with thin bristles; female fertile; male sterile.

allele	origin	discoverer	synonym
<i>l(2)34Eb</i>¹	EMS	O'Donnell	<i>CH61</i>
<i>l(2)34Eb</i>²	EMS	O'Donnell	<i>DM12</i>

l(2)34F

allele	origin	discoverer	synonym	ref ^α
<i>l(2)34Fa</i>¹	EMS	Harrington	<i>HG15</i>	
<i>l(2)34Fc</i>¹	EMS	Bodmer and Walker	<i>BMW18</i>	
<i>l(2)34Fc</i>²	EMS	Bodmer and Walker	<i>BMW23</i>	
<i>l(2)34Fc</i>³	EMS	Ashburner	<i>CR3</i>	2
<i>l(2)34Fc</i>⁴	EMS	Harrington	<i>HG20</i>	
<i>l(2)34Fc</i>⁵	EMS	Harrington	<i>HG28</i>	
<i>l(2)34Fc</i>⁶	EMS	Ashburner	<i>SF9</i>	2
<i>l(2)34Fc</i>⁷	EMS	Ashburner	<i>SF13</i>	2
<i>l(2)34Fc</i>⁸	EMS	O'Donnell	<i>CH43</i>	1
<i>l(2)34Fc</i>⁹	EMS	O'Donnell	<i>CH45</i>	1

^α 1 = O'Donnell, Mandel, Krauss, and Sofer, 1977, Genetics 86: 553-66; 2 = Woodruff and Ashburner, 1979, Genetics 92: 133-49.

l(2)34Fd

phenotype: Recessive lethal, but some heteroallelic combinations [e.g., *l(2)34Fd*³/*l(2)34Fd*⁶] complement for viability but emerge very late.

allele	origin	discoverer	synonym	ref ^α
<i>l(2)34Fd</i>¹	EMS	Bodmer and Walker	<i>BMW10</i>	
<i>l(2)34Fd</i>²	EMS	Ashburner	<i>CR5</i>	2
<i>l(2)34Fd</i>³	EMS	Harrington	<i>HG6</i>	
<i>l(2)34Fd</i>⁴	EMS	Harrington	<i>HG11</i>	
<i>l(2)34Fd</i>⁵	EMS	Harrington	<i>HG13</i>	
<i>l(2)34Fd</i>⁶	EMS	Harrington	<i>HG23</i>	
<i>l(2)34Fd</i>⁷	EMS	O'Donnell	<i>DM4</i>	1
<i>l(2)34Fd</i>⁸	EMS	O'Donnell	<i>OK15</i>	1
<i>l(2)34Fd</i>⁸	EMS	Lindsley	<i>64-1589</i>	

^α 1 = O'Donnell, Mandel, Krauss, and Sofer, 1977, Genetics 86: 553-66; 2 = Woodruff and Ashburner, 1979, Genetics 92: 133-49.

DEFICIENCY MAP OF REGION 34

side	breakpoint	variant
left		<i>Df(2L)b82a1</i>
left		<i>Df(2L)b84h50</i>
left	34C3	<i>Df(2L)b80f1</i>
left	34C3	<i>Df(2L)b84a2</i>
left	34C4	<i>Df(2L)b80e3</i>
left	34C4	<i>Df(2L)b80e4</i>
left	34D1-2	<i>Df(2L)b82a2</i>
	34C4-D1	<i>l(2)34Da</i>
left		<i>Df(2L)b74c6</i>
left		<i>Df(2L)b78j</i>
left		<i>Df(2L)b79b3</i>
left		<i>Df(2L)b79b4</i>
left		<i>Df(2L)b79a5</i>
left		<i>Df(2L)b81h1</i>
left		<i>Df(2L)b81l42</i>
left		<i>Df(2L)b83d29a</i>
left		<i>Df(2L)b83l1</i>
left		<i>Df(2L)b84h14</i>
left		<i>Df(2L)el28</i>
left	34C1	<i>Df(2L)b81l1</i>
left	34C4-5	<i>Df(2L)85fa</i>
left	34C6-7	<i>Df(2L)jfn30</i>
left	34D1-2	<i>Df(2L)64j</i>
left	34D1-2	<i>Df(2L)TE35BC-23</i>
left	34D2	<i>Df(2L)TE35A-5</i>
left	34D3	<i>Df(2L)jfn31</i>
left	34D3	<i>Df(2L)b-L</i>
left	34D3	<i>Df(2L)b80c1</i>
left	34D3	<i>Df(2L)b81a1</i>
left	34D3	<i>Df(2L)b81a6</i>
left	34D3	<i>Df(2L)b81f1</i>
left	34D3	<i>Df(2L)b84a3</i>
left	34D3	<i>Df(2L)b84a4</i>
left	34D3	<i>Df(2L)b84a8</i>
left	34D3-4	<i>Df(2L)b75</i>
left	34D3	<i>Df(2L)b84a5</i>
left	34D3-6	<i>Df(2L)TE35BC-6</i>
	34D3	<i>l(2)34Db</i>
left		<i>Df(2L)84h1</i>
left	34D3	<i>Df(2L)b84a9</i>
left	34D5	<i>Df(2L)Sco7</i>
left	34D7-E1	<i>Df(2L)b36f</i>
	34D4-5	<i>l(2)34Dd</i>
left		<i>Df(2L)b82a3</i>
	34D4-5	<i>l(2)34Dc</i>
		b
right		<i>Df(2L)b79d5</i>
right		<i>Df(2L)b81h1</i>
right		<i>Df(2L)b82a3</i>
	34D6-8	<i>l(2)34De</i>
	34D6-8	<i>l(2)34Df</i>
	34D6-8	<i>l(2)34Dg</i>
right		<i>Df(2L)b83l1</i>

side	breakpoint	variant
	34E1-2	I(2)34Ea
right		<i>Df(2L)b81142</i>
right		<i>Df(2L)b84a9</i>
right	34D7-E1	<i>Df(2L)b36f</i>
left	34E1	<i>Df(2L)A47</i>
left	34E1-2	<i>Df(2L)el80i1</i>
left	34E1-2	<i>Df(2L)el82f1</i>
	34E3-5	I(2)34Eb
right		<i>Df(2L)b82a1</i>
right	34D8-E1	<i>Df(2L)b80f1</i>
right	34D8-E1	<i>Df(2L)b80c1</i>
left	34E1-2	<i>Df(2L)jfn7</i>
left	34E3	<i>Df(2L)jfn26</i>
		j
left	34D3	<i>Df(2L)jfn12</i>
right	34E1-2	<i>Df(2L)b82a2</i>
left	34E3	<i>Df(2L)A376</i>
left	34E3	<i>Df(2L)el80f1</i>
left	34E4-5	<i>Df(2L)TE35A-8</i>
	34E3-5	rk
right	34E4-5	<i>Df(2L)jb-L</i>
left		<i>Df(2L)Adh-BR41</i>
left	34E3	<i>Df(2L)noc11</i>
left	34E5-F1	<i>Df(2L)A263</i>
left	34F1	<i>Df(2L)TE35A-9</i>
left	34F1-2	<i>Df(2L)noc10</i>
left	34F1-2	<i>Df(2L)noc20</i>
left	34F1-2	<i>Df(2L)Sco23</i>
left	34F1-2	<i>Df(2L)TE35A-4</i>
left	34F3	<i>Df(2L)GT4</i>
left	34F5	<i>Df(2L)TE35A-3</i>
left	34F5	<i>Df(2L)osp141</i>
left	35A3	<i>Df(2L)W</i>
left	34F1-2	I(2)34Fa
right		<i>Df(2L)b79b3</i>
right		<i>Df(2L)b84h14</i>
right	34E5-6	<i>Df(2L)b75</i>
left	34F1-4	<i>Df(2L)A377</i>
left	34F4	<i>Df(2L)A246</i>
left	34E4	<i>Df(2L)el20</i>
left	34F4	<i>Df(2L)TE35A-2</i>
left	34F4	<i>Df(2L)TE35A-15</i>
left	34F4-35A1	<i>Df(2L)jfn1</i>
left	34F5	<i>Df(2L)el81i1</i>
left	34F5	<i>Df(2L)jfn5</i>
left	34F5	<i>Df(2L)TE35A-10</i>
left	34F5	<i>Df(2L)TE35BC-31</i>
left	35A1-2	<i>Df(2L)noc13</i>
left	35A1-2	<i>Df(2L)osp38</i>
left	35A1-3	<i>Df(2L)el77</i>
left	35A3	<i>Df(2L)A215</i>
left	35A3	<i>Df(2L)jfn36</i>
left	34F5	<i>Df(2L)TE35BC-29</i>
left	35B	<i>Df(2L)el18</i>
	34F3	wb
right		<i>Df(2L)b78j</i>
right		<i>Df(2L)b79b4</i>
right	34F5	<i>Df(2L)b85f1</i>
left	34F5	<i>Df(2L)A217</i>
left	35B1	<i>Df(2L)do1</i>
left	35B1-2	<i>Df(2L)el15</i>
right	35B2	<i>Df(2L)b81f1A</i>
	34F4-35A1	I(2)34Fc
	34F4-35A1	I(2)34Fd
	34F4-35A1	ms(2)34F
left	35A3-4	<i>Df(2L)TE35A-7</i>

I(2)35

Extensive mutagenesis experiments carried out in this region by Ashburner and his colleagues and to a lesser extent in the laboratory of Sofer.

references: O'Donnell, Mandel, Krauss, and Sofer, 1977, Genetics 86: 553-66.

Woodruff and Ashburner, 1979, Genetics 92: 133-49.

Ashburner, Aaron, and Tsubota, 1982, Genetics 102: 421-35.

Ashburner, Tsubota, and Woodruff, 1982, Genetics 102: 401-20.

Gubb, Shelton, Roote, McGill, and Ashburner, 1984, Chromosoma 91: 54-64.

locus	genetic location	cytological location	included in	excluded from	synonym
I(2)35Aa	2-{49.0}	35A3-4	<i>Df(2L)TE35A-7</i>	<i>Df(2L)A400</i>	<i>l(2)br12</i>
I(2)35Ba	2-{50.0}	35B1	<i>Df(2L)A400</i>	<i>Df(2L)A446</i>	<i>l(2)br22</i>
I(2)35Bb	2-{50.36}	35B4	<i>Df(2L)A446</i>		<i>l(2)br3</i>
			<i>Df(2L)jfn3</i>		<i>l(2)br23</i>
I(2)35Bc	2-{50.5}	35B5-8	<i>Df(2L)jfn31</i>	<i>Df(2L)jfn3</i>	<i>l(2)br4</i>
					<i>l(2)br11</i>
					<i>l(2)br13</i>
I(2)35Bd	2-{50.6}	35B5-8	<i>Df(2L)jfn31</i>	<i>Df(2L)jfn3</i>	<i>l(2)br9</i>
I(2)35Be	2-{50.6}	35B5-8	<i>Df(2L)jfn31</i>	<i>Df(2L)jfn3</i>	<i>l(2)br10</i>
I(2)35Bf	2-{50.6}	35B5-8	<i>Df(2L)jfn31</i>	<i>Df(2L)jfn3</i>	<i>l(2)br2</i>
I(2)35Bg	2-{50.9}	35B9-10	<i>Df(2L)AR-R1</i>	<i>Df(2L)jfn31</i>	<i>l(2)br26</i>
I(2)35Bh	2-{50.5}	35B9-10	<i>Df(2L)AR-R1</i>	<i>Df(2L)jfn31</i>	Su(H) , <i>l(2)br7</i>

I(2)35A

allele	origin	discoverer	synonym	ref ^α
I(2)35Aa¹	EMS	Harrington	<i>HG8</i>	
I(2)35Aa²	EMS	Harrington	<i>HG12</i>	
I(2)35Aa³	EMS	Ashburner	<i>SF12</i>	2
I(2)35Aa⁴	EMS	Ashburner	<i>SF32</i>	2
I(2)35Aa⁵	EMS	O'Donnell	<i>CH32</i>	1
I(2)35Aa⁶	EMS	O'Donnell	<i>DM16</i>	1
I(2)35Aa⁷	EMS	O'Donnell	<i>DM18</i>	1

^α 1 = O'Donnell, Mandel, Krauss, and Sofer, 1977, Genetics 86: 553-66; 2 = Woodruff and Ashburner, 1979, Genetics 92: 133-49.

I(2)35Ba

phenotype: *l(2)35Ba¹* may escape in combination with deficiency for the locus; escapers are weak elbow in phenotype if deficiency includes *el*, but not if it does not. All alleles express weak elbow in combination with *el¹*; heteroallelic escapers [*i.e.*, *l(2)35Ba⁴* in combination with other alleles] are weak elbow. Semilethal with *In(2LR)Sco^{rv1}* and *Sco^{rv27}*; also show weak *noc* phenotype in combination with strong *noc* alleles.

allele	origin	discoverer	synonym	ref ^α
I(2)35Ba¹	EMS	Ashburner	<i>AR10</i>	1, 2
I(2)35Ba²	EMS	Ashburner	<i>FT1</i>	1
I(2)35Ba³	EMS	Harrington	<i>HG33</i>	1
I(2)35Ba⁴	EMS	Harrington	<i>HG46</i>	1

^α 1 = Ashburner, Tsubota, and Woodruff, 1982, Genetics 102: 401-20; 2 = Woodruff and Ashburner, 1979, Genetics 92: 133-49.

I(2)35Bb

phenotype: Rare survivors to adulthood in *l(2)35Bb⁵/Df(2L)jfn3*.

allele	origin	discoverer	synonym	ref ^α	comments
I(2)35Bb¹	EMS	Ashburner	<i>AR2</i>	2	
I(2)35Bb²	EMS	Bodmer and Walker	<i>BMW33</i>		
I(2)35Bb³	EMS	Harrington	<i>HG5</i>		
I(2)35Bb⁴	EMS	Littlewood	<i>LT4</i>		
I(2)35Bb⁵	EMS	Ashburner	<i>SF21</i>	2	
I(2)35Bb⁶	spont	Ashburner	<i>TA2</i>	2	in <i>In(2LR)Gla</i>
I(2)35Bb⁷	EMS	O'Donnell	<i>DM11</i>	1	
I(2)35Bb⁸	EMS	O'Donnell	<i>OK19</i>	1	
I(2)35Bb⁹	EMS	Lindsley	<i>64-692</i>		

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)35Bc</i> ¹	EMS	Ashburner	<i>AR1</i>	2	noncomplementing
<i>l(2)35Bc</i> ²	EMS	Ashburner	<i>AR6</i>	2	complements <i>l(2)35Bc</i> ³
<i>l(2)35Bc</i> ³	EMS	Ashburner	<i>AR7</i>	2	
<i>l(2)35Bc</i> ⁴	EMS	Harrington	<i>HG1</i>		noncomplementing
<i>l(2)35Bc</i> ⁵	EMS	Harrington	<i>HG94</i>		noncomplementing
<i>l(2)35Bc</i> ⁶	EMS	Ashburner	<i>SF17</i>	2	noncomplementing
<i>l(2)35Bc</i> ⁷	EMS	O'Donnell	<i>CH23</i>		complements <i>l(2)35Bc</i> ³
<i>l(2)35Bc</i> ⁸	EMS	Lindsley	<i>64-605</i>		complements <i>l(2)35Bc</i> ³
<i>l(2)35Bd</i> ¹	EMS	Ashburner	<i>AR4</i>	2	
<i>l(2)35Bd</i> ²	EMS	Ashburner	<i>AR13</i>	2	
<i>l(2)35Bd</i> ³	EMS	Littlewood	<i>LT8</i>		
<i>l(2)35Bd</i> ⁴	EMS	Ashburner	<i>SF6</i>	2	
<i>l(2)35Bd</i> ⁵	EMS	O'Donnell	<i>CH48</i>	1	
<i>l(2)35Bd</i> ⁶	EMS	O'Donnell	<i>OK4</i>	1	
<i>l(2)35Be</i> ¹	EMS	Ashburner	<i>AR3</i>	2	noncomplementing
<i>l(2)35Be</i> ²	EMS	Ashburner	<i>AR5</i>	2	noncomplementing
<i>l(2)35Be</i> ³	EMS	Harrington	<i>HG37</i>		
<i>l(2)35Be</i> ⁴	EMS	Ashburner	<i>SF5</i>	2	complements <i>l(2)35Be</i> ³
<i>l(2)35Be</i> ⁵	EMS	O'Donnell	<i>CH55</i>	1	complements <i>l(2)35Be</i> ³
<i>l(2)35Be</i> ⁶	EMS	O'Donnell	<i>OK20</i>	1	
<i>l(2)35Be</i> ⁷	EMS	Lindsley	<i>64-346</i>		noncomplementing

^α 1 = O'Donnell, Mandel, Krauss, and Sofer, 1977, Genetics 86: 553-66; 2 = Woodruff and Ashburner, 1979, Genetics 92: 133-149.

l(2)35Bf

phenotype: Escapers show thin, short bristles and outstretched wings. *l(2)35Bf*³ partially complements *l(2)35Bf*¹ and *l(2)35Bf*² for viability; female-sterile alleles recovered by Lindsley.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)35Bf</i> ¹	EMS	Bodmer and Walker	<i>BMW12</i>		
<i>l(2)35Bf</i> ²	EMS	Bodmer and Walker	<i>BMW20</i>		
<i>l(2)35Bf</i> ³	EMS	Harrington	<i>HG27</i>		
<i>l(2)35Bf</i> ⁴	EMS	Littlewood	<i>LT1</i>	2	
<i>l(2)35Bf</i> ⁵	EMS	Ashburner	<i>SF18</i>	2	
<i>l(2)35Bf</i> ⁶	EMS	Ashburner	<i>SF19B</i>	2	
<i>l(2)35Bf</i> ⁷	EMS	Maroni	<i>GM11</i>		
<i>l(2)35Bf</i> ⁸	EMS	Lindsley	<i>64-720</i>		female sterile

<i>l(2)35Bg</i> ¹	EMS	Harrington	<i>HG21</i>		
<i>l(2)35Bg</i> ²	EMS	O'Donnell	<i>OK5</i>	1	

^α 1 = O'Donnell, Mandel, Krauss, and Sofer, 1977, Genetics 86: 553-66; 2 = Woodruff and Ashburner, 1979, Genetics 92: 133-49.

l(2)35C

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(2)35Ca</i>	2-[51.0]	35C1-2	<i>Df(2L)A267</i>	<i>Df(2L)AR-R1</i>	ck , <i>l(2)br27</i>
<i>l(2)35Cb</i>	2-[51.2]	35C1-2	<i>Df(2L)A400</i>	<i>Df(2L)A267</i>	<i>l(2)br33</i>
<i>l(2)35Cc</i>	2-[51.2]	35C3	<i>Df(2L)A215</i>	<i>Df(2L)A400</i>	<i>l(2)br50</i>
<i>l(2)35Cd</i>	2-[51.2]	35C4-5	<i>Df(2L)A263</i>	<i>Df(2L)A215</i>	<i>l(2)br34</i>
<i>l(2)35Ce</i>	2-[51.3]	35C4-5	<i>Df(2L)A376</i>	<i>Df(2L)A263</i>	<i>l(2)br43</i>

l(2)35Cb

phenotype: *l(2)35Cb*¹ escapes in heterozygous combination with *Df(2L)Sco4*, but not with other *l(2)35Cb* deficiencies.

allele	origin	discoverer	synonym	comments
<i>l(2)35Cb</i> ¹	EMS	Harrington	<i>HG38</i>	
<i>l(2)35Cb</i> ²	EMS	Simpson	<i>A12</i>	
<i>l(2)35Cb</i> ³	EMS	Simpson	<i>A14</i>	
<i>l(2)35Cb</i> ⁴	EMS	Simpson	<i>B12</i>	
<i>l(2)35Cb</i> ⁵	EMS	Simpson	<i>B21</i>	

allele	origin	discoverer	synonym	comments
<i>l(2)35Cb</i> ⁶	X ray	Ashburner	<i>AM9</i>	with <i>ln(2L)shv</i> ²²

l(2)35Cc

phenotype: No known alleles; existence inferred from lethality associated with *Df(2L)rd9*.

l(2)35Cd

phenotype: *l(2)35Cd*², but not other alleles, fails to complement *ln(2LR)Sco*^{rv1}.

allele	origin	discoverer	synonym
<i>l(2)35Cd</i> ¹	EMS	Harrington	<i>HG43</i>
<i>l(2)35Cd</i> ²	EMS	Harrington	<i>HG39</i>
<i>l(2)35Cd</i> ³	EMS	Simpson	<i>B8</i>
<i>l(2)35Cd</i> ⁴	EMS	Simpson	<i>B14</i>
<i>l(2)35Cd</i> ⁵	EMS	Simpson	<i>B29</i>
<i>l(1)35Ce</i> ¹	EMS	Simpson	<i>VS2</i>
<i>l(1)35Ce</i> ²	EMS	Simpson	<i>VS4</i>
<i>l(1)35Ce</i> ³	EMS	Simpson	<i>VS8</i>

l(2)35D

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(2)35Da</i>	2-[51.3]	35D1-2	<i>Df(2L)el20</i>	<i>Df(2L)A376</i>	<i>l(2)br35</i>
<i>l(2)35Db</i>	2-[51.3]	35D1-2	<i>Df(2L)do1</i>	<i>Df(2L)el20</i>	sna ; <i>l(2)br28</i>
<i>l(2)35Dc</i>	2-[51.4]	35D3-4	<i>Df(2L)fn27</i>	<i>Df(2L)do1</i>	lace ; <i>l(2)br36</i>
<i>l(2)35Dd</i>	2-[51.4]	35D5-7	<i>Df(2L)TE35BC-34</i>	<i>Df(2L)fn27</i>	<i>l(2)br37</i>
<i>l(2)35De</i>	2-[51.4]	35D3-4	<i>Df(2L)A246</i>	<i>Df(2L)TE35BC-34</i>	<i>l(2)br46</i>
<i>l(2)35Df</i>	2-[51.4]	35D5-7	<i>Df(2L)A246</i>	<i>Df(2L)TE35BC-34</i>	<i>l(2)br44</i>
<i>l(2)35Dg</i>	2-[51.4]	35D4	<i>Df(2L)A246</i>	<i>Df(2L)TE35BC-34</i>	<i>l(2)br45</i>

allele	origin	discoverer	synonym	comments
<i>l(2)35Da</i> ¹	EMS	Harrington	<i>HG35</i>	
<i>l(2)35Da</i> ²	EMS	Simpson	<i>A4</i>	
<i>l(2)35Da</i> ³	EMS	Simpson	<i>B7</i>	
<i>l(2)35Da</i> ⁴	EMS	Simpson	<i>B27</i>	
<i>l(2)35Dd</i> ¹	X ray	Ashburner	<i>GE1</i>	associated with breakpoint of <i>T(2;3)Ha16</i>
<i>l(2)35Dd</i> ²	EMS	El Messal	<i>P28</i>	
<i>l(2)35Dd</i> ³	EMS	El Messal	<i>P41</i>	
<i>l(2)35Dd</i> ⁴	insert	Ising	<i>TE116</i>	
<i>l(2)35De</i> ¹		Ashburner	<i>AM2</i>	associated with breakpoint of <i>ln(2L)NS</i>
<i>l(2)35Df</i> ¹	EMS	El Messal	<i>P15</i>	
<i>l(2)35Df</i> ²	EMS	El Messal	<i>P32</i>	
<i>l(2)35Df</i> ³	EMS	El Messal	<i>P36</i>	
<i>l(2)35Df</i> ⁴	EMS	Grau	<i>RX1</i>	
<i>l(2)35Dg</i> ¹	EMS	El Messal	<i>P29</i>	
<i>l(2)35Dg</i> ²	EMS	El Messal	<i>P31</i>	
<i>l(2)35Dg</i> ³	EMS	El Messal	<i>P34</i>	
<i>l(2)35Dg</i> ⁴	EMS	El Messal	<i>P40</i>	

l(2)35E

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(2)35Ea</i>	2-[51]	35E1-2	<i>Df(2L)fn26</i>	<i>Df(2L)A246</i>	<i>l(2)br47</i>
<i>l(2)35Eb</i>	2-[51]	35E3-6	<i>Df(2L)A446</i>	<i>Df(2L)fn26</i>	<i>l(2)br48</i>
<i>l(2)35Ec</i>	2-[51]	35E3-6	<i>Df(2L)PA4</i>	<i>Df(2L)A446</i>	<i>l(2)br49</i>

locus	genetic location		cytological location	
	included in	excluded from	synonym	

allele	discoverer	synonym	comments
I(2)35Ea¹	Ashburner	AM3	associated with right breakpoint of <i>1n(2L)dpp36</i> ; semilethal
I(2)35Eb¹	Ashburner	AM4	associated with but proximal to <i>1n(2L)C163.41</i> ; semilethal
I(2)35Ec¹	Ashburner	AM5	associated with breakpoint of <i>T(2;3)DTD14</i>

DEFICIENCY MAP OF REGION 35A-E

side	breakpoint	variant	molecular coordinates (kb) α
left	35A3-4	<i>Df(2L)TE35A-7</i>	
	35A3-4	I(2)35Aa	
left		<i>Df(2L)TE35A-12</i>	
left	35A1-4	<i>Df(2L)A400</i>	
right	35A4	<i>Df(2L)b80e3</i>	
right	35A4	<i>Df(2L)b84h1</i>	
left	35B1-2	<i>Df(2L)TE35A-13</i>	
	35A4	pu	
right		<i>Df(2L)b83d29a</i>	
left		<i>Df(2L)jfn15</i>	
left	35A3-4	<i>Df(2L)ARR1</i>	
left	35A3-4	<i>Df(2L)jfn2</i>	
left	35A4	<i>Df(2L)TE35A-14</i>	
left	35B1	<i>Df(2L)TE35A-6</i>	
left	35B1-2	<i>Df(2L)TE35A-1</i>	
left	35B1-2	<i>Df(2L)TE35BC-8</i>	
		el	
left		<i>Df(2L)el16</i>	
left	35A4	<i>Df(2L)A245</i>	-137.8 to -134.8
left	35B1	<i>Df(2L)jfn3</i>	-126 to -125
right	35A4	<i>Df(2L)b84a2</i>	-125 to -121.8
right	35B	<i>Df(2L)el18</i>	
left	35B1	<i>Df(2L)Adh78I3</i>	-120 to -117.8
left	35B1-2	<i>Df(2L)A260</i>	
left	35A2-3	<i>Df(2L)A266</i>	
left	35A4	<i>Df(2L)A245</i>	
	35B1	I(2)35Ba	
left		<i>Df(2L)A178</i>	-113 to -110.8
left	35B1-2	<i>Df(2L)osp204</i>	
left	35B1-3	<i>Df(2L)A446</i>	-117 to -114
left	35B1	<i>Df(2L)jfn27</i>	-103
left	invisible	<i>Df(2L)A445</i>	-73
left	35B3	<i>Df(2L)TE35BC-7</i>	-70.8 to -68.3
left		<i>Df(2L)osp144</i>	-68.3 to -67.1
left	35B1-3	<i>Df(2L)A379</i>	-66.2 to -61.8
left	35B2-3	<i>Df(2L)A267</i>	-66.2 to -61.8
		noc	
right	35B2	<i>Df(2L)GT4</i>	
right	invisible	<i>Df(2L)TE35A-12</i>	-103 to -98
left	35B3	<i>Df(2L)mNXF1</i>	-86.6 to -79
right	35A1-2	<i>Df(2L)TE35A-4</i>	-79 to -75.5
right	35B1-2	<i>Df(2L)b81a1</i>	-79 to -75.5
right	35B2	<i>Df(2L)TE35A-10</i>	-79 to -75.5
left	35B3	<i>Df(2L)mNXF2</i>	-73
left		<i>Df(2L)jfn52</i>	-58
right	35B1-2	<i>Df(2L)b84a8</i>	-52.8 to -43.8
right	35B1-2	<i>Df(2L)b84a4</i>	-42.5 to -37.5
left	35B1-2	<i>Df(2L)osp18</i>	-37.5
left	35B1-2	<i>Df(2L)A220</i>	-28.4 to -24.6
left	35B1-3	<i>Df(2L)A264</i>	-24.8 to -24.6
left	invisible	<i>Df(2L)A63</i>	-18.2 to -13.1
left	35B2-3	<i>Df(2L)A48</i>	-0.7 to 0
left	35B2-3	<i>Df(2L)A72</i>	+0.7 to +1.3
right	35B3-4	<i>Df(2L)b84a5</i>	
	35B1-2	osp	
right		<i>Df(2L)osp204</i>	
right	35B1-2	<i>Df(2L)TE35A-8</i>	-15.9 to -11.2
right	invisible	<i>Df(2L)osp144</i>	-8.3
	35B3	Adh	
right		<i>Df(2L)A63</i>	

side	breakpoint	variant	molecular coordinates (kb) α
right		<i>Df(2L)A178</i>	
right		<i>Df(2L)A445</i>	
right		<i>Df(2L)Adh-BR41</i>	
right		<i>Df(2L)jfn52</i>	
right		<i>Df(2L)PA4</i>	
right		<i>Df(2L)TE35A-11</i>	
right		<i>Df(2L)TE35A-50</i>	
left		<i>Df(2L)TE35A-48</i>	
left		<i>Df(2L)TE35A-54</i>	+10 to +30
right	35A4	<i>Df(2L)el17</i>	
right	35B1	<i>Df(2L)A47</i>	
right	35B1-2	<i>Df(2L)A260</i>	
right	35B1-2	<i>Df(2L)el81i1</i>	
right	35B1-2	<i>Df(2L)noc10</i>	
right	35B1-2	<i>Df(2L)b84a1</i>	
right	35B1-2	<i>Df(2L)b84a7</i>	
right	35B1-3	<i>Df(2L)A379</i>	
left	35B1-3	<i>Df(2L)osp29</i>	
right	35B2	<i>Df(2L)A245</i>	
right	35B2	<i>Df(2L)jfn2</i>	
right	35B2	<i>Df(2L)noc13</i>	
right	35B2	<i>Df(2L)noc20</i>	+11.8 to +13.7
left	35B2	<i>Df(2L)TE35BC-28</i>	
left	35B2	<i>Df(2L)TE35BC-35</i>	
right	35B2	<i>Df(2L)TE35A-9</i>	
right	35B2	<i>Df(2L)TE35A-15</i>	
right	35B2	<i>Df(2L)TE35A-7</i>	
right	35B2-3	<i>Df(2L)A266</i>	
right	35B3	<i>Df(2L)A217</i>	
right	35B3	<i>Df(2L)el77</i>	
right	35B3	<i>Df(2L)TE35A-3</i>	
right	35B3-5	<i>Df(2L)jfn7</i>	
right	35B3-5	<i>Df(2L)W</i>	
right	35B4	<i>Df(2L)b8111</i>	
right	35B4	<i>Df(2L)jfn36</i>	
left	35B4	<i>Df(2L)TE35BC-4</i>	
left	35B4	<i>Df(2L)TE35BC-34</i>	
right	35B5	<i>Df(2L)osp141</i>	
	35B4	I(2)35Bb	
		<i>Df(2L)b74c6?</i>	
right	35B3-4	<i>Df(2L)jfn3</i>	
right	35B4-8	I(2)35Bc	
	35B4-8	I(2)35Bd	
	35B5-8	I(2)35Be	
	35B5-8	I(2)35Bf	
right	35B3-4	<i>Df(2L)b84a3</i>	
right	35B4	<i>Df(2L)m1a1</i>	
left	35B4-6	<i>Df(2L)TE35BC-24</i>	
right	35B3-5	<i>Df(2L)jfn31</i>	
right	35B6-7	<i>Df(2L)b84a6</i>	
left	35C1	<i>Df(2L)TE35BC-1</i>	
	35B9-10	I(2)35Bg	
	35B9-10	Su(H)	
right	35B9-C1	<i>Df(2L)ARR1</i>	
left	35C1	<i>Df(2L)TE35BC-3</i>	
	35C1-2	ck	
right	35B7	<i>Df(2L)TE35BC-28</i>	
right	35B7-C5	<i>Df(2L)jfn15</i>	
right	35B8-C1	<i>Df(2L)64j</i>	
right	35B9-10	<i>Df(2L)TE35BC-7</i>	
right	35B10	<i>Df(2L)A267</i>	
right	35B10	<i>Df(2L)jfn12</i>	
right	35B10	<i>Df(2L)TE35BC-31</i>	
right	35B10-11	<i>Df(2L)TE35BC-23</i>	
right	35C1	<i>Df(2L)TE35BC-29</i>	
right	35D1	<i>Df(2L)TE35BC-1</i>	
	35C1-2	vasa	
right	35B9-C1	<i>Df(2L)jfn30</i>	
	35C1-2	I(2)35Cb	
left		<i>Df(2L)rd9</i>	
right	35B7-8	<i>Df(2L)A72</i>	
right	35B8-9	<i>Df(2L)A264</i>	
right	35B9	<i>Df(2L)A220</i>	
right	35B10	<i>Df(2L)A400</i>	
right	35B10	<i>Df(2L)mNXF1</i>	
right	35B10	<i>Df(2L)mNXF2</i>	

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side	breakpoint	variant	molecular coordinates (kb) α
right	35C1	<i>Df(2L)TE35A-5</i>	
right	35C1	<i>Df(2L)TE35A-6</i>	
right	35C1	<i>Df(2L)TE35BC-6</i>	
right	35C2-3	<i>Df(2L)e180i1</i>	
right	35C3	<i>Df(2L)jfn5</i>	
right	35C5	<i>Df(2L)e115</i>	
	35C3	I(2)35Cc	
left		<i>Df(2L)snaS1</i>	
	35C3	rd	
right		<i>Df(2L)rd9</i>	
right	35B7-8	<i>Df(2L)A215</i>	
	35C4-5	I(2)35Cd	
right	35A2	<i>Df(2L)noc11</i>	
right	35C3-5	<i>Df(2L)A263</i>	
right	35C4-5	<i>Df(2L)osp38</i>	
	35C4-5	I(2)35Ce	
right	35C3-5	<i>Df(2L)e182f1</i>	
	35C4-5	dgl	
right	35C3	<i>Df(2L)TE35BC-4</i>	
right	35C4-5	<i>Df(2L)A376</i>	
right	35C3-4	<i>Df(2L)osp18</i>	
right	35C4-5	<i>Df(2L)TE35A-1</i>	
	35D1-2	I(2)35Da	
right	35C5	<i>Df(2L)e120</i>	
left	35D1-2	<i>Df(2L)PA4</i>	
	35D1-2	sna	
right	35D1-2	<i>Df(2L)b80e4</i>	
right	35D2	<i>Df(2L)do1</i>	
	35D3-4	lace	
right	35D1-2	<i>Df(2L)jfn27</i>	
right	35D2	<i>Df(2L)TE35BC-3</i>	
right	35D4	<i>Df(2L)TE35A-14</i>	
right	35D4	<i>Df(2L)TE35BC-35</i>	
right	35D5-7	<i>Df(2L)A48</i>	
right	35D5-7	<i>Df(2L)jfn1</i>	
right	35D7	<i>Df(2L)e180f1</i>	
		I(2)35Dd	
right	35D4	<i>Df(2L)TE35BC-34</i>	
	35D5-7	I(2)35De	
	35D5-7	I(2)35Df	
	35D5-7	I(2)35Dg	
right		<i>Df(2L)sna-S1</i>	
right		<i>Df(2L)TE35A-13</i>	
right	35D3-4	<i>Df(2L)A246</i>	
right	35D5-7	<i>Df(2L)Adh7813</i>	
right	35D7	<i>Df(2L)TE35A-2</i>	
right	35E1-2	<i>Df(2L)TE35BC-8</i>	
right	35F1-2	<i>Df(2L)A377</i>	
	35E1-2	I(2)35Ea	
right	35D8-E1	<i>Df(2L)jfn26</i>	
right	35E1-2	<i>Df(2L)TE35BC-24</i>	
	35E3-6	I(2)35Eb	
right	35E6	<i>Df(2L)osp29</i>	
right	35F1-2	<i>Df(2L)A446</i>	
	35E3-6	I(2)35Ec	
right		<i>Df(2L)PA4</i>	
left	36A7-10	<i>Df(2L)H20</i>	

α Origin of coordinates at *EcoRI* site 5' to *Adh*; coordinates are negative in the direction of the telomere and positive in the direction of the centromere (McGill, Chia, Karp, and Ashburner, 1988, Genetics 119: 647-61) and are not necessarily correlated with cytological breakpoints.

I(2)36

references: Steward and Nüsslein-Volhard, 1986, Genetics 113: 665-78.

I(2)36A

Five lethally mutable loci, including *Mhc*; map order not determined.

locus	genetic location	cytological location	included in	excluded from	synonym
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locus	genetic location	cytological location	included in	excluded from	synonym
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I(2)36Aa	2-{52}	36A7-B2	<i>Df(2L)H20</i>	<i>Df(2L)H68</i>	<i>l(2L)HT-5</i>
I(2)36Ab	2-{52}	36A7-B2	<i>Df(2L)H20</i>	<i>Df(2L)H68</i>	<i>l(2L)HT-6</i>
I(2)36Ac	2-{52}	36A7-B2	<i>Df(2L)H20</i>	<i>Df(2L)H68</i>	<i>l(2L)HT-9</i>
I(2)36Ad	2-{52}	36A7-B2	<i>Df(2L)H20</i>	<i>Df(2L)H68</i>	<i>l(2L)HT-11</i>
<i>l(2)36Ae</i>	2-52.2	36A7-B2	<i>Df(2L)H20</i>	<i>Df(2L)H68</i>	Mhc

I(2)36Aa, b, and c

phenotype: Larval lethals.

allele	origin
I(2)36Aa ¹	EMS
I(2)36Ab ¹	EMS
I(2)36Ab ²	EMS
I(2)36Ab ³	EMS
I(2)36Ac ¹	EMS
I(2)36Ac ²	EMS
I(2)36Ad ¹	EMS
I(2)36Ad ²	EMS

I(2)36B, C, and D

locus	genetic location	cytological location	included in	excluded from	synonym
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I(2)36Ba	2-{52}	36B3-C4	<i>Df(2L)H68</i>	<i>Df(2L)T317</i>	<i>l(2L)HT-4</i>
I(2)36Bb	2-{52}	36B3-C4	<i>Df(2L)H68</i>	<i>Df(2L)T317</i>	<i>l(2L)HT-7</i>
I(2)36Bc	2-{52}	36B3-C4	<i>Df(2L)H68</i>	<i>Df(2L)T317</i>	<i>l(2L)HT-10</i>
I(2)36Bd	2-{52}	36B3-C4	<i>Df(2L)T317</i>	<i>Df(2L)TW137</i>	<i>l(2L)HT-2</i>
I(2)36Ca	2-{52}	36C2-D1	<i>Df(2L)TW137</i>	<i>Df(2L)VA18</i>	<i>l(2L)HT-1</i>
I(2)36Da	2-53.1	36D	<i>Df(2L)VA18</i>	<i>Df(2L)VA22</i>	<i>l(2)Bld</i>
I(2)36Db	2-{53}	36D1-2	<i>Df(2L)b10-1</i>		

I(2)36Ba

phenotype: Larval lethal.

allele	origin
I(2)36Ba ¹	EMS
I(2)36Ba ²	EMS
I(2)36Ba ³	EMS

I(2)36Bb

phenotype: Rare survivors to adulthood show a naked phenotype with reduced bristles on the scutum and scutellum.

allele	origin
I(2)36Bb ¹	EMS
I(2)36Bb ²	EMS
I(2)36Bb ³	EMS
I(2)36Bb ⁴	EMS
I(2)36Bb ⁵	EMS
I(2)36Bb ⁶	EMS
I(2)36Bb ⁷	EMS

I(2)36Bc

phenotype: Larval lethal.

allele	origin
I(2)36Bc ¹	EMS

l(2)36Bd

phenotype: Larval lethal.

allele	origin
<i>l(2)36Bd</i> ¹	EMS
<i>l(2)36Bd</i> ²	EMS
<i>l(2)36Bd</i> ³	EMS
<i>l(2)36Bd</i> ⁴	EMS
<i>l(2)36Bd</i> ⁵	EMS
<i>l(2)36Bd</i> ⁶	EMS
<i>l(2)36Bd</i> ⁷	EMS
<i>l(2)36Bd</i> ⁸	EMS
<i>l(2)36Bd</i> ⁹	EMS

l(2)36Ca

phenotype: Larval lethal.

allele	origin
<i>l(2)36Ca</i> ¹	EMS
<i>l(2)36Ca</i> ²	EMS
<i>l(2)36Ca</i> ³	EMS
<i>l(2)36Ca</i> ⁴	EMS
<i>l(2)36Ca</i> ⁵	EMS

l(2)36Da

phenotype: Larval lethal.

allele	origin	discoverer	synonym	ref ^α
<i>l(2)36Da</i> ¹	spont	Bridges	<i>l(2)Bld</i>	1
<i>l(2)36Da</i> ²	EMS			2
<i>l(2)36Da</i> ³	EMS			2
<i>l(2)36Da</i> ⁴	EMS			2
<i>l(2)36Da</i> ⁵	EMS			2
<i>l(2)36Da</i> ⁶	EMS			2

^α 1 = CP627; 2 = Steward and Nüsslein-Volhard, 1986, Genetics 113: 665-78.

l(2)36D6phenotype: Inferred from the lethality of *Df(2L)b10-1*.***l(2)36F***

A series of ethyl-methanesulfonate-induced mutants comprising nine complementation groups, including *M(2)36F* recovered by Wright and co-workers (Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310).

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(2)36Fa</i>	2-54	36F2-6	<i>Df(2L)TW202</i>	<i>Df(2L)hk18</i>	<i>M(2)36F</i> , <i>M(2)m</i>
<i>l(2)36Fb</i>	2-53.9	36F4-6	<i>Df(2L)hk18</i>	<i>Df(2L)E71</i>	<i>l(2)E70</i>
<i>l(2)36Fc</i>	2-53.9	36F4-37A1	<i>Df(2L)E71</i>	<i>Df(2L)TW3</i>	<i>l(2)E53</i>
<i>l(2)36Fd</i>	2-53.9	36F7-9	<i>Df(2L)TW3</i>		<i>l(2)E51</i>
<i>l(2)36Fe</i>	2-53.9	36F7-9	<i>Df(2L)M36F-S5</i> <i>Df(2L)TW3</i>		<i>l(2)E134</i>
<i>l(2)36Ff</i>	2-[54]	36F7-37A1	<i>Df(2L)M36F-S6</i>	<i>Df(2L)M36F-S5</i>	<i>l(2)E47</i>
<i>l(2)36Fg</i>	2-[54]	36F7-37A1	<i>Df(2L)M36F-S6</i>	<i>Df(2L)M36F-S5</i>	<i>l(2)E20</i>
<i>l(2)36Fh</i>	2-[54]	36F7-37A1	<i>Df(2L)M36F-S6</i>	<i>Df(2L)M36F-S5</i>	<i>l(2)E50</i>
<i>l(2)36Fi</i>	2-[54]	36F7-37A1	<i>Df(2L)M36F-S6</i>	<i>Df(2L)M36F-S5</i>	<i>l(2)E105</i>

allele	synonym
<i>l(2)36Fb</i> ¹	<i>l(2)E70</i>
<i>l(2)36Fb</i> ²	<i>l(2)E36</i>
<i>l(2)36Fb</i> ³	<i>l(2)E48</i>
<i>l(2)36Fb</i> ⁴	<i>l(2)E64</i>
<i>l(2)36Fc</i> ¹	<i>l(2)E122</i>
<i>l(2)36Fc</i> ²	<i>l(2)E31</i>

allele synonym

<i>l(2)36Fd</i> ¹	<i>l(2)E51</i>
<i>l(2)36Fe</i> ¹	<i>l(2)E134</i>
<i>l(2)36Ff</i> ¹	<i>l(2)E47</i>
<i>l(2)36Ff</i> ²	<i>l(2)E52</i>
<i>l(2)36Fg</i> ¹	<i>l(2)E20</i>
<i>l(2)36Fh</i> ¹	<i>l(2)E50</i>
<i>l(2)36Fi</i> ¹	<i>l(2)E105</i>

DEFICIENCY MAP OF REGION 36

side	breakpoint	variant
left	36A7-10	<i>Df(2L)H20</i> <i>l(2)36Aa</i> <i>l(2)36Ab</i> <i>l(2)36Ac</i> <i>l(2)36Ad</i> <i>Mhc</i> <i>plo</i>
left	36B2-C1	<i>Df(2L)H68</i>
left	36B3-8	<i>Df(2L)M18</i> <i>l(2)36Ba</i> <i>l(2)36Bb</i> <i>l(2)36Bc</i> <i>Df(2L)T317</i> <i>l(2)36Bd</i>
left		<i>Df(2L)A92</i>
left	36C2-4	<i>Df(2L)B7</i>
left		<i>Df(2L)B131</i>
left	invisible	<i>Df(2L)TW119</i>
left	36C2-4	<i>Df(2L)TW137</i> <i>l(2)36Ca</i> <i>dl</i> <i>Bic-D</i> <i>qua</i> <i>Df(2L)A33</i> <i>Df(2L)D143</i>
left		<i>Df(2L)I131</i>
left	36D	<i>Df(2L)M36F-S5</i>
left	36E4-F1	<i>Df(2L)TW203</i>
left	36C4-D1	<i>Df(2L)VA18</i>
left		<i>Df(2L)VA21</i>
left		<i>Df(2L)VA22</i> <i>l(2)36Da</i> <i>Df(2L)M18</i>
right	36D	<i>Df(2L)M18</i>
left		<i>Df(2L)B11</i>
left	36E-F	<i>Df(2L)TW201</i>
left		<i>Df(2L)TW330</i> <i>rdo</i>
left	5' or 3'	<i>Df(2L)B10-1</i>
left	3' or 5'	<i>rdo</i>
right	invisible	<i>Df(2L)TW119</i>
right	36D1-2	<i>Arr1</i> <i>l(2)36Db</i> <i>Df(2L)B10-1</i>
right ^α	36E1-2	<i>Fas3</i> <i>kel</i>
right		<i>Df(2L)I131</i>
right	36D1-E4	<i>nina-D</i>
right	36E4-F1	<i>Df(2L)H20</i>
right		<i>Df(2L)T317</i>
left	36E4-F1	<i>Df(2L)TW50</i>
left ^β	34E6-F1	<i>Df(2L)M36F-S6</i>
left	37F2-5	<i>Df(2L)TW202</i> <i>M(2)36F</i>
left ^β	36E4-6	<i>Df(2L)hk18</i> <i>l(2)36Fb</i>
left	36F2-6	<i>Df(2L)E71</i>
left		<i>Df(2L)E53</i> <i>l(2)36Fc</i>

side	breakpoint	variant
left	36F7-37A1	<i>Df(2L)TW3</i> <i>msl-1</i>
right	36F7-37A1	<i>Df(2L)ES3</i> <i>Df(2L)OD15</i> <i>l(2)36Fd</i> <i>l(2)36Fe</i>
right	36F7-9	<i>Df(2L)M36F-S6</i> <i>Df(2L)VA16</i>
left		
right	37A	<i>Df(2L)TW201</i>
right	36F6-37A1	<i>Df(2L)hk39</i>

^α Positions of *Fas3* and *kel* with respect to the right breakpoint of *Df(2L)B10-1* are not determined.

^β Breakpoint not localized with respect to neighboring lethal mutations.

l(2)37

More than eighty lethal alleles at 37 loci, including *Dox-A2*, *Amd*, and *Ddc*, in the region uncovered by *Df(2L)TW50 = Df(2L)36E4-F2;38A6-7* recovered and deficiency mapped by Wright, Bewley and Sherald (1976, Genetics 84: 287-310). Detailed analysis of those under *Df(2L)TW130 = Df(2L)37B9-C1;37D1-2* presented in other work involving 204 mutants (Wright, Beerman, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, Chromosoma 83: 45-58). Additional mutants in more proximal part of region 37 recovered and analyzed by Gay and Contamine. Allelic information for this region are as yet incomplete.

l(2)37A

Three complementation groups represented by one ethyl-methanesulfonate-induced allele each; recovered by Wright and coworkers (Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310).

locus	genetic		cytological		synonym
	location	location	included in	excluded from	
<i>l(2)37Aa</i>	2-53.1-53.9	<i>36F8-37B7</i>	<i>Df(2L)TW3</i>	<i>Df(2L)M36F-S6</i>	<i>l(2)E41</i>
<i>l(2)37Ab</i>	2-53.1-53.9	<i>36F8-37B7</i>	<i>Df(2L)TW3</i>	<i>Df(2L)M36F-S6</i>	<i>l(2)E129</i>
<i>l(2)37Ac</i>	2-53.1-53.9	<i>36F8-37B7</i>	<i>Df(2L)TW3</i>	<i>Df(2L)M36F-S6</i>	<i>l(2)E145</i>

l(2)37B

Ten complementation groups located between the proximal breakpoint of *Df(2L)TW203* and the distal breakpoint of *Df(2L)VA17* in the distal half of the Dopa-decarboxylase region. *Amd*, which is listed in 37B could be in 37C1-2 (Wright, 1987, Adv. Genet. 24: 127-222).

locus	genetic		cytological		synonym
	location	location	included in	excluded from	
<i>l(2)37Ba</i>	2-53.9	<i>37B10-13</i>	<i>Df(2L)OD15</i>	<i>Df(2L)TW203</i>	
<i>l(2)37Bb</i>	2-53.9	<i>37B10-13</i>	<i>Df(2L)hk-UC1</i>	<i>Df(2L)OD15</i>	<i>l(2)E58</i>
<i>l(2)37Bc</i>	2-53.9	<i>37B10-13</i>	<i>Df(2L)OD15</i>	<i>Df(2L)TW203</i>	<i>l(2)E13</i>
<i>l(2)37Bd</i>	2-53.9+	<i>37B13-C1</i>	<i>Df(2L)NST</i>	<i>Df(2L)VA17</i>	
<i>l(2)37Be</i>	2-53.9	<i>37B10-13</i>	<i>Df(2L)OD15</i>	<i>Df(2L)TW203</i>	
<i>l(2)37Bf</i>	2-53.9	<i>37B10-13</i>	<i>Df(2L)hk-UC1</i>	<i>Df(2L)OD15</i>	<i>Dox-A2</i>
<i>l(2)37Bg</i>	2-53.9	<i>37B13-C1</i>	<i>Df(2L)VA18</i>	<i>Df(2L)hk18</i> <i>Df(2L)NST</i>	
<i>l(2)37Bi</i>	2-53.9	<i>37B3-7</i>	<i>Df(2L)TW137</i>	<i>Df(2L)TW3</i> <i>Df(2L)TW158</i>	<i>l(2)E131</i>
<i>l(2)37Bj</i>	2-53.9	<i>37B3-7</i>	<i>Df(2L)TW137</i>	<i>Df(2L)TW3</i> <i>Df(2L)TW158</i>	<i>l(2)E35</i>
<i>l(2)37Bk</i>	2-53.9+	<i>37B13-C1</i>	<i>Df(2L)NST</i>	<i>Df(2L)VA17</i>	<i>Amd</i>

l(2)37Ba

phenotype: Variably late lethal; death occurs at larval [*l(2)37Ba*²], or pupal [*l(2)37Ba*², *l(2)37Ba*⁴, *l(2)37Ba*⁸, *l(2)37Ba*⁹] stage with pharate adults; *l(2)37Ba*² produces some adults, which are male sterile or female sterile; *l(2)37Ba*⁴ produces many adults, which are male or female sterile; *l(2)37B*^{*fs1*} is a normally viable, female-sterile allele. Melanotic tumors noted in *l(2)37Ba*⁴ and *l(2)37Ba*⁸; pharate adults display incompletely developed abdominal cuticle in *l(2)37Ba*², *l(2)37Ba*⁴, and *l(2)37Ba*⁸ (Wright, 1987, Adv. Genet. 24: 127-222).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)37Ba</i>¹	EMS	Wright	<i>l(2)261</i>	1	
<i>l(2)37Ba</i>²	EMS+HCOH	Wright	<i>l(2)342</i>	1	
*<i>l(2)37Ba</i>³	EMS+γ ray	Wright	<i>l(2)501</i>	1	
<i>l(2)37Ba</i>⁴	EMS	Wright	<i>l(2)606</i>	1	
*<i>l(2)37Ba</i>⁵	EMS	Wright	<i>l(2)619</i>	1	
*<i>l(2)37Ba</i>⁶	EMS	Wright	<i>l(2)624</i>	1	
<i>l(2)37Ba</i>⁷	EMS+γ ray	Wright	<i>l(2)7101</i>	2	
<i>l(2)37Ba</i>⁸	EMS	Lindsley	<i>l(2)50-584</i>		
*<i>l(2)37Ba</i>⁹	EMS	Lindsley	<i>l(2)50-283</i>		
<i>l(2)37Ba</i>¹¹	EMS+γ ray	Cecil	<i>l(2)BB45</i>		
<i>l(2)37Ba</i>¹²	EMS+γ ray	Cecil	<i>l(2)A6</i>		
<i>l(2)37Ba</i>^{<i>fs1</i>}	EMS	Wright	<i>fs(2)R</i>		female sterile

^α 1 = Wright, Beerman, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, Chromosoma 83: 45-58; 2 = Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26.

l(2)37Bb

phenotype: Larval lethal; third instar larvae survive until day eight to thirteen, and at least in the case of *l(2)37Bb*¹ they are small with small imaginal discs, salivary glands, and fat bodies.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)37Bb</i>¹	EMS	Wright	<i>l(2)E58</i>	2	
<i>l(2)37Bb</i>²	EMS	Wright	<i>l(2)E138</i>	2	
*<i>l(2)37Bb</i>³	EMS	Wright	<i>l(2)E152</i>	2	
*<i>l(2)37Bb</i>⁴	EMS	Wright	<i>l(2)E154</i>	2	
<i>l(2)37Bb</i>⁵	EMS	Wright	<i>l(2)235</i>	1	
*<i>l(2)37Bb</i>⁶	EMS	Wright	<i>l(2)242</i>	1	
*<i>l(2)37Bb</i>⁷	EMS	Wright	<i>l(2)252</i>	1	
<i>l(2)37Bb</i>⁸	EMS	Wright	<i>l(2)278</i>	1	heat sensitive
<i>l(2)37Bb</i>⁹	EMS+HCOH	Wright	<i>l(2)393</i>	1	
<i>l(2)37Bb</i>¹⁰	EMS	Wright	<i>l(2)647</i>	1	
<i>l(2)37Bb</i>¹¹	EMS+γ ray	Wright	<i>l(2)7447</i>	3	400 base-pair intragenic deletion

^α 1 = Wright, Beerman, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, Chromosoma 83: 45-58; 2 = Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310; 3 = Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26.

l(2)37Bc

phenotype: Larval lethal. *l(2)37Bc*¹ produces many dead collapsed larvae at 48 h; *l(2)37Bc*²⁰ displays small, still-living larvae at 144 h; *l(2)37Bc*¹¹ pupal lethal with melanized puparia and pharate adults, with incomplete head and thoracic cuticle, no abdominal cuticle, and no eyes or bristles. Melanotic tumors in *l(2)37Bc*⁹-*l(2)37Bc*²⁰ and *l(2)37Bc*⁹/*l(2)37Bc*^{*fs1*} adults.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)37Bc</i> ¹	EMS	Wright	<i>l(2)E13</i>	2	
<i>l(2)37Bc</i> ²	EMS	Wright	<i>l(2)E59</i>	2	
<i>l(2)37Bc</i> ³	EMS	Wright	<i>l(2)E130</i>	2	
<i>l(2)37Bc</i> ⁴	EMS	Wright	<i>l(2)E132</i>	2	
<i>l(2)37Bc</i> ⁵	EMS	Wright	<i>l(2)216</i>	1	
<i>l(2)37Bc</i> ⁶	EMS	Wright	<i>l(2)225</i>	1	
<i>l(2)37Bc</i> ⁷	EMS	Wright	<i>l(2)257</i>	1	
<i>l(2)37Bc</i> ⁸	EMS	Wright	<i>l(2)271</i>	1	
<i>l(2)37Bc</i> ⁹	EMS	Wright	<i>l(2)290</i>	1	
<i>l(2)37Bc</i> ¹⁰	spont	Wright	<i>l(2)BL106</i>	1	
<i>l(2)37Bc</i> ¹¹	EMS+HCOH	Wright	<i>l(2)316</i>	1	
<i>l(2)37Bc</i> ¹²	EMS+HCOH	Wright	<i>l(2)380</i>	1	
<i>l(2)37Bc</i> ¹³	EMS+HCOH	Wright	<i>l(2)386</i>	1	
<i>l(2)37Bc</i> ¹⁴	EMS+HCOH	Wright	<i>l(2)399</i>	1	
<i>l(2)37Bc</i> ¹⁵	EMS	Wright	<i>l(2)614</i>	1	
<i>l(2)37Bc</i> ¹⁶	EMS	Wright	<i>l(2)625</i>	1	
<i>l(2)37Bc</i> ¹⁷	EMS	Wright	<i>l(2)626</i>	1	
<i>l(2)37Bc</i> ¹⁸	EMS	Wright	<i>l(2)641</i>	1	
<i>l(2)37Bc</i> ¹⁹	EMS	Wright	<i>l(2)642</i>	1	semilethal
<i>l(2)37Bc</i> ²⁰	EMS+γ ray	Wright	<i>l(2)7417</i>	3	
<i>l(2)37Bc</i> ²¹	EMS+γ ray	Wright	<i>l(2)7444</i>	3	
<i>l(2)37Bc</i> ²²	EMS	Lindsley	<i>l(2)158-1559</i>		
<i>l(2)37Bc</i> ²³	EMS+γ ray	Cecil	<i>l(2)B4</i>		
<i>l(2)37Bc</i> ²⁴	EMS+γ ray	Cecil	<i>l(2)B90</i>		
<i>l(2)37Bc</i> ²⁵	EMS+γ ray	Cecil	<i>l(2)BB5</i>		
<i>l(2)37Bc</i> ^{fs1}	EMS+γ ray	Wright	<i>l(2)7602</i>		female sterile
<i>l(2)37Bc</i> ^{fs2}	EMS+γ ray	Wright	<i>l(2)7607</i>		female sterile
<i>l(2)37Bc</i> ^{fs3}	EMS	Schüpbach	<i>l(2)QT50</i>		female sterile
<i>l(2)37Bc</i> ^{ts1}	EMS	Wright	<i>l(2)202</i> ^{ts}		temperature sensitive

^α 1 = Wright, Beerman, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, Chromosoma 83: 45-58; 2 = Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310; 3 = Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26, 45-58.

^β Homozygous females lay tiny eggs which remain unfertilized.

l(2)37Bd

phenotype: Larval-pupal lethal; *l(2)37Bd*² and *l(2)37Bd*⁶ are strong alleles in which second and third instar larvae persist until days eight and nine; few survive to pharate-adult stage with incomplete sclerotization; *l(2)37Bd*⁷ similar, but with more pharate adults. *l(2)37Bd*⁴ semilethal; *l(2)37Bd*⁴/*l(2)37Bd*⁷ phenotype similar to that of *l(2)37Bd*¹.

allele	origin	discoverer	synonym	ref ^α
<i>l(2)37Bd</i> ¹	EMS	Wright	<i>l(2)285</i>	1
<i>l(2)37Bd</i> ²	EMS+HCOH	Wright	<i>l(2)322</i>	1
<i>l(2)37Bd</i> ³	EMS+HCOH	Wright	<i>l(2)331</i>	1
<i>l(2)37Bd</i> ⁴	EMS+HCOH	Wright	<i>l(2)385</i>	1
<i>l(2)37Bd</i> ⁵	EMS+HCOH	Wright	<i>l(2)401</i>	1
<i>l(2)37Bd</i> ⁶	EMS	Wright	<i>l(2)621</i>	1
<i>l(2)37Bd</i> ⁷	EMS+γ ray	Wright	<i>l(2)7408</i>	2
<i>l(2)37Bd</i> ⁸	EMS	Lindsley	<i>l(2)158-1440</i>	
<i>l(2)37Bd</i> ⁹	EMS	Lindsley	<i>l(2)158-2032</i>	

^α 1 = Wright, Beerman, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, Chromosoma 83: 45-58; 2 = Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26.

l(2)37Be

phenotype: Embryonic lethal *l(2)37Be*¹ and *l(2)37Be*² uniformly die as embryos; *l(2)37Be*³, on the other hand, dies as embryos or larvae, with a few reaching the pharate adult stage with incomplete abdominal cuticle, and some even surviving as fertile adults.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)37Be</i> ¹	EMS	Wright	<i>l(2)601</i>	1	
<i>l(2)37Be</i> ²	EMS	Wright	<i>l(2)665</i>	1	

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)37Be</i> ³	EMS+γ ray	Wright	<i>l(2)7401</i>	2	
<i>l(2)37Be</i> ⁴	EMS+γ ray	Wright	<i>l(2)7446</i>	2	
<i>l(2)37Be</i> ⁵	EMS+γ ray	Cecil	<i>l(2)A1</i>		0.8-1.0-kb deletion

^α 1 = Wright, Beerman, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, Chromosoma 83: 45-58; 2 = Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26.

l(2)37Bg

phenotype: Pupal lethal; on the seventh day large third-instar larvae begin to form large, darker tan pupae; eye-antennal discs evert, but only the genital disc derivatives form cuticle; eyes, antennae, legs, and wing discs form no cuticle. Pupae form a large [*l(2)37Bg*¹] or numerous small [*l(2)37Bg*²] melanotic tumors.

allele	origin	discoverer	synonym	ref ^α
<i>l(2)37Bg</i> ¹	EMS+γ ray	Wright	<i>l(2)7420</i>	1
<i>l(2)37Bg</i> ²	EMS	Lindsley	<i>l(2)50-564</i>	

^α 1 = Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26.

allele	origin	discoverer	synonym	ref ^α
<i>l(2)37Bj</i> ¹	EMS	Wright	<i>l(2)E131</i>	1
<i>l(2)37Bj</i> ²	EMS	Wright	<i>l(2)E46</i>	1
<i>l(2)37Bj</i> ³	EMS	Wright	<i>l(2)E133</i>	1

^α 1 = Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26.

^α 1 = Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310.

l(2)37C

locus	genetic		cytological		synonym
	location	location	included in	excluded from	
<i>l(2)37Ca</i>	2-53.9+	37C2-4	<i>Df(2L)VA21</i> <i>Df(2L)VA17</i>	<i>Df(2L)hk-UC2</i> <i>Df(2L)VA12</i>	<i>l(2)E11</i>
<i>l(2)37Cb</i>	2-53.9+	37C2-4	<i>Df(2L)hk-UC2</i>	<i>Df(2L)VA18</i>	<i>l(2)E104</i>
<i>l(2)37Cc</i>	2-53.9+	37C2-4	<i>Df(2L)VA17</i> <i>Df(2L)VA18</i>		
<i>l(2)37Cd</i>	2-53.9+	37C2-4	<i>Df(2L)VA21</i> <i>Df(2L)VA17</i>	<i>Df(2L)hk-UC2</i> <i>Df(2L)VA12</i>	
<i>l(2)37Ce</i>	2-53.9+	37C2-4	<i>Df(2L)VA21</i> <i>Df(2L)VA17</i>	<i>Df(2L)hk-UC2</i> <i>Df(2L)VA12</i>	<i>l(2)E62</i>
<i>l(2)37Cf</i>	2-53.95	37C3-7	<i>Df(2L)TE37C-B7</i> <i>Df(2L)Sd57</i>	<i>Df(2L)VA21</i> <i>Df(2L)Sd77</i>	<i>l(2)E60</i>
<i>l(2)37Cg</i>	2-53.9+	37C2-4	<i>Df(2L)VA21</i> <i>Df(2L)VA17</i>	<i>Df(2L)hk-UC2</i> <i>Df(2L)VA12</i>	
<i>l(2)37Ch</i>	2-53.9+	37C1-2	<i>Df(2L)VA17</i>	<i>Df(2L)VA12</i>	Ddc

l(2)37Ca

phenotype: *l(2)37Ca*^{ts1}/*Df(2L)TW130* eclosed adults at 25° have incompletely sclerotized thoracic cuticle, which deforms when the indirect flight muscles contract; wings rarely completely expanded. X-ray-induced abdominal clones hemizygous for the amorphic allele *l(2)37Ca*⁴, produce bristles one-third wild-type size.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)37Ca</i> ¹	EMS	Wright	<i>l(2)E11</i>	2	
<i>l(2)37Ca</i> ²	EMS	Wright	<i>l(2)E56</i>	2	
<i>l(2)37Ca</i> ³	EMS	Wright	<i>l(2)E125</i>	2	larval lethal
<i>l(2)37Ca</i> ⁴	EMS	Wright	<i>l(2)217</i>	1	
<i>l(2)37Ca</i> ⁵	EMS	Wright	<i>l(2)230</i>	1	
<i>l(2)37Ca</i> ⁶	EMS	Wright	<i>l(2)240</i>	1	
<i>l(2)37Ca</i> ⁷	EMS	Wright	<i>l(2)246</i>	1	
<i>l(2)37Ca</i> ⁸	EMS	Wright	<i>l(2)253</i>	1	heat sensitive; leaky at 30°

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allele	origin	discoverer	synonym	ref ^α	comments
*I(2)37Ca⁹	EMS	Wright	I(2)264	1	
I(2)37Ca¹⁰	EMS	Wright	I(2)266	1	larval lethal
*I(2)37Ca¹¹	EMS	Wright	I(2)267	1	heat sensitive; leaky at 30°
*I(2)37Ca¹²	EMS	Wright	I(2)268	1	
*I(2)37Ca¹³	EMS	Wright	I(2)269	1	
I(2)37Ca¹⁴	EMS+HCOH	Wright	I(2)312	1	
*I(2)37Ca¹⁵	EMS+HCOH	Wright	I(2)313	1	
I(2)37Ca¹⁶	EMS+HCOH	Wright	I(2)314	1	
I(2)37Ca¹⁷	EMS+HCOH	Wright	I(2)319	1	
I(2)37Ca¹⁸	EMS+HCOH	Wright	I(2)334	1	
I(2)37Ca¹⁹	EMS+HCOH	Wright	I(2)3452	1	
*I(2)37Ca²⁰	EMS+HCOH	Wright	I(2)3462	1	
*I(2)37Ca²¹	EMS+HCOH	Wright	I(2)352	1	
*I(2)37Ca²²	EMS+HCOH	Wright	I(2)402	1	
*I(2)37Ca²³	EMS+HCOH	Wright	I(2)403	1	
*I(2)37Ca²⁴	EMS+HCOH	Wright	I(2)407	1	
*I(2)37Ca²⁵	EMS	Wright	I(2)610	1	
*I(2)37Ca²⁶	EMS	Wright	I(2)612	1	
I(2)37Ca²⁷	EMS	Wright	I(2)613	1	
I(2)37Ca²⁸	EMS	Wright	I(2)622	1	larval lethal
*I(2)37Ca²⁹	EMS	Wright	I(2)629	1	
*I(2)37Ca³⁰	EMS	Wright	I(2)639	1	
I(2)37Ca³¹	EMS	Wright	I(2)680	1	
*I(2)37Ca³²	EMS+γ ray	Wright	I(2)7303	3	
*I(2)37Ca³³	EMS+γ ray	Wright	I(2)7404	3	
*I(2)37Ca³⁴	EMS+γ ray	Wright	I(2)7425	3	
*I(2)37Ca³⁵	EMS	Brittnacher	I(2)L2		
I(2)37Ca³⁶	γ ray	Cecil	I(2)PC		
*I(2)37Ca³⁷	EMS	L. Hall	I(2)P3		
I(2)37Ca³⁸	EMS	Wright	I(2)620		chromosome lethal

I(2)37Ca^{39β}	EMS+γ ray	Wright	I(2)7431		over <i>Ddc</i> alleles
*I(2)37Ca⁴⁰	EMS+γ ray	Cecil	I(2)AA7		
*I(2)37Ca⁴¹	EMS+γ ray	Cecil	I(2)A2		
*I(2)37Ca^{ts1}	EMS	Wright	I(2)209 ^{ts}	1	temperature sensitive
I(2)37Ca^{ts2}	EMS+γ ray	Wright	I(2)7432r ^{ts}	3	temperature sensitive
I(2)37Ca^{ts3}	EMS+HCOH	Wright	I(2)3500 ^{ts}	1	temperature sensitive

^α 1 = Wright, Beerman, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, Chromosoma 83: 45-58; 2 = Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310; 3 = Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26.

^β Induced simultaneously with and not separated from I(2)37Cb³⁰; lethal over many I(2)37Ca and I(2)37Cb alleles; male and female sterile in those combinations that complement.

I(2)37Cb

allele	origin	discoverer	synonym	ref ^α	comments
I(2)37Cb¹	EMS	Wright	I(2)E104	2	larval lethal
I(2)37Cb²	EMS	Wright	I(2)E118	2	
I(2)37Cb³	EMS	Wright	I(2)222	1	heat sensitive
*I(2)37Cb⁴	EMS	Wright	I(2)241	1	
*I(2)37Cb⁵	EMS	Wright	I(2)274	1	
*I(2)37Cb⁶	EMS+HCOH	Wright	I(2)3488	1	
*I(2)37Cb⁷	EMS+HCOH	Wright	I(2)3510	1	
I(2)37Cb⁸	EMS+HCOH	Wright	I(2)394	1	larval lethal
I(2)37Cb⁹	EMS+γ ray	Wright	I(2)503	1	
*I(2)37Cb¹⁰	EMS	Wright	I(2)603	1	
*I(2)37Cb¹¹	EMS	Wright	I(2)609	1	
*I(2)37Cb¹²	EMS	Wright	I(2)611	1	
*I(2)37Cb¹³	EMS	Wright	I(2)618	1	
I(2)37Cb¹⁴	EMS	Wright	I(2)627	1	
*I(2)37Cb¹⁵	EMS	Wright	I(2)630	1	
*I(2)37Cb¹⁶	EMS	Wright	I(2)631	1	
*I(2)37Cb¹⁷	EMS	Wright	I(2)648	1	
I(2)37Cb¹⁸	EMS+γ ray	Wright	I(2)7406	3	170-bp intra-genic deletion
I(2)37Cb¹⁹	EMS+γ ray	Wright	I(2)7419	3	
I(2)37Cb²⁰	EMS+γ ray	Wright	I(2)7424	3	larval lethal

allele	origin	discoverer	synonym	ref ^α	comments
*I(2)37Cb²¹	EMS+γ ray	Wright	I(2)7427	3	
*I(2)37Cb²²	EMS+γ ray	Wright	I(2)7428	3	
I(2)37Cb²³	EMS+γ ray	Wright	I(2)7448	3	
I(2)37Cb²⁴	EMS	Lindsley	I(2)158-1652		
*I(2)37Cb²⁵	EMS	Lindsley	I(2)50-569		
*I(2)37Cb²⁶	EMS	Lindsley	I(2)50-586		
I(2)37Cb²⁷	EMS	Brittnacher	I(2)L1		
*I(2)37Cb²⁸	EMS	Brittnacher	I(2)L12		
*I(2)37Cb²⁹	EMS	Brittnacher	I(2)L16		
I(2)37Cb^{30β}	EMS+γ ray	Wright	I(2)7431		
I(2)37Cb^{ts1}	EMS	Wright	I(2)232 ^{ts}	1	temperature sensitive
*I(2)37Cb^{ts2}	EMS+HCOH	Wright	I(2)383 ^{ts}	1	temperature sensitive

^α 1 = Wright, Beerman, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, Chromosoma 83: 45-58; 2 = Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310; 3 = Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26.

^β Induced simultaneously with and not separated from I(2)37Ca³⁹; lethal over many I(2)37Ca and I(2)37Cb alleles; male and female sterile in those combinations that complement.

I(2)37Cc

phenotype: Many different complementing heteroallelic genotypes exhibit opaque wings, often incompletely expanded, moderate Minute-like bristles, and a lighter tan body color.

allele	origin	discoverer	synonym	ref ^α	comments
I(2)37Cc¹	EMS	Wright	I(2)236	1	
I(2)37Cc²	EMS	Wright	I(2)244	1	
I(2)37Cc³	EMS	Wright	I(2)284	1	
I(2)37Cc⁴	EMS	Wright	I(2)286	1	
*I(2)37Cc⁵	EMS	Wright	I(2)289	1	
I(2)37Cc⁶	EMS+HCOH	Wright	I(2)395	1	heat sensitive; posterior half of unexpanded wings heavily melanized
I(2)37Cc⁷	EMS	Wright	I(2)658	1	larval lethal
*I(2)37Cc⁸	EMS	Wright	I(2)686	1	
I(2)37Cc⁹	EMS	Steward	I(2)RS1		
I(2)37Cc¹⁰	EMS+γ ray	Wright	I(2)7421	2	larval lethal —L2 and L3
*I(2)37Cc¹¹	EMS+γ ray	Wright	I(2)7434	2	
I(2)37Cc¹²	EMS+γ ray	Wright	I(2)7449	2	
I(2)37Cc¹³	EMS+γ ray	Wright	I(2)7450	2	dominant female sterile
I(2)37Cc¹⁴	EMS	Lindsley	I(2)50-947		larval lethal
I(2)37Cc¹⁵	EMS	Lindsley	I(2)158-1683		larval-pupal lethal; leaky; adult escapers female sterile and Minute
*I(2)37Cc¹⁶	EMS	Lindsley	I(2)50-546		
*I(2)37Cc¹⁷	EMS	Lindsley	I(2)50-5		
*I(2)37Cc¹⁸	EMS	Brittnacher	I(2)L4		
*I(2)37Cc¹⁹	EMS	Brittnacher	I(2)L13		
I(2)37Cc²⁰	DEB	Cecil	I(2)DEB12		30-bp intragenic deletion
I(2)37Cc^{ts1}	EMS+HCOH	Wright	I(2)350 ^{ts}	1	temperature sensitive
I(2)37Cc^{ts2}	EMS+HCOH	Wright	I(2)392 ^{ts}	1	temperature sensitive
I(2)37Cc^{fs1}	EMS	Schüpbach	fs(2)HH32		female sterile

^α 1 = Wright, Beerman, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, Chromosoma 83: 45-58; 2 = Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26.

l(2)37Cd

phenotype: Late third-instar larval lethal; some form unsclerotized pseudopupae after ten days; imaginal discs and central nervous system half normal size; melanotic tumors [*l(2)37Cd*⁴].

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)37Cd</i> ¹	EMS	Wright	<i>l(2)234</i>	1	melanotic tumors
<i>l(2)37Cd</i> ²	EMS+HCOH	Wright	<i>l(2)335</i>	1	
<i>l(2)37Cd</i> ³	EMS+HCOH	Wright	<i>l(2)343</i>	1	
<i>l(2)37Cd</i> ⁴	EMS+HCOH	Wright	<i>l(2)369</i>	1	
<i>l(2)37Cd</i> ⁵	EMS	Wright	<i>l(2)636</i>	1	pupal lethal
<i>l(2)37Cd</i> ⁶	EMS	Wright	<i>l(2)678</i>	1	melanotic tumors
<i>l(2)37Cd</i> ⁷	EMS+γ ray	Wright	<i>l(2)7001</i>	2	larval lethal; melanotic tumors; 600 bp deletion
<i>l(2)37Cd</i> ⁸	EMS+γ ray	Wright	<i>l(2)7405</i>	2	
<i>l(2)37Cd</i> ⁹	EMS+γ ray	Wright	<i>l(2)7451</i>	2	
<i>l(2)37Cd</i> ¹⁰	EMS+γ ray	Cecil	<i>l(2)B10</i>		
<i>l(2)37Cd</i> ^{ts1}	EMS+HCOH	Wright	<i>l(2)321</i> ^{ts}	1	

^α 1 = Wright, Beerman, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, Chromosoma 83: 45-58; 2 = Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26.

l(2)37Ce

phenotype: Most alleles are pupal lethals; abdominal cuticle absent and melanotic tumors recorded for *l(2)37Ce*¹ and *l(2)37Ce*⁵.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)37Ce</i> ¹	EMS	Wright	<i>l(2)E62</i>	2	
<i>l(2)37Ce</i> ²	EMS	Wright	<i>l(2)E111</i>	2	
<i>l(2)37Ce</i> ³	EMS	Wright	<i>l(2)255</i>	1	semilethal
<i>l(2)37Ce</i> ⁴	EMS+HCOH	Wright	<i>l(2)3514</i>	1	
<i>l(2)37Ce</i> ⁵	EMS	Wright	<i>l(2)635</i>	1	
<i>l(2)37Ce</i> ⁶	EMS	Britnacher	<i>l(2)L18</i>		
<i>l(2)37Ce</i> ⁷	EMS+γ ray	Cecil	<i>l(2)AA5</i>		
<i>l(2)37Ce</i> ⁸	EMS+γ ray	Cecil	<i>l(2)BB6</i>		

^α 1 = Wright, Beerman, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, Chromosoma 83: 45-58; 2 = Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310.

l(2)37Cf

phenotype: Lethality occurs as pharate adults. Third-instar larvae of 13 of 14 alleles examined display enlarged brains—up to eight times normal volume; greatest in presumptive optic center (Hankins and Wright). Brain fragments transplanted into normal adults show uncontrolled proliferation and metastatic invasion of other tissues (Gateff). Escapers [*l(2)37Cf*¹-/*l(2)37Cf*^{ts3}] lethargic and sterile in both sexes [Wright, 1987, Results and Problems in Cell Differentiation (W. Hennig, ed.). Springer-Verlag, Berlin, Heidelberg, pp. 95-120]. Homozygous females [*l(2)37Cf*^{ts2}] retain eggs in ovaries (Schüpbach).

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)37Cf</i> ¹	EMS	Wright	<i>l(2)E60</i>	2	
<i>l(2)37Cf</i> ²	EMS	Wright	<i>l(2)E127</i>	2	
<i>l(2)37Cf</i> ³	EMS	Wright	<i>l(2)223</i>	1	
<i>l(2)37Cf</i> ⁴	EMS	Wright	<i>l(2)243</i>	1	
<i>l(2)37Cf</i> ⁵	EMS	Wright	<i>l(2)251</i>	1	
<i>l(2)37Cf</i> ⁶	EMS	Wright	<i>l(2)259</i>	1	
<i>l(2)37Cf</i> ⁷	EMS	Wright	<i>l(2)291</i>	1	
<i>l(2)37Cf</i> ⁸	EMS+HCOH	Wright	<i>l(2)320A</i>	1	
<i>l(2)37Cf</i> ⁹	EMS+HCOH	Wright	<i>l(2)3450</i>	1	semilethal
<i>l(2)37Cf</i> ¹⁰	EMS+HCOH	Wright	<i>l(2)3454</i>	1	
<i>l(2)37Cf</i> ¹¹	EMS+HCOH	Wright	<i>l(2)347</i>	1	
<i>l(2)37Cf</i> ¹²	EMS	Wright	<i>l(2)608</i>	1	few adults

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)37Cf</i> ¹³	EMS	Wright	<i>l(2)643</i>	1	
<i>l(2)37Cf</i> ¹⁴	EMS	Wright	<i>l(2)649</i>	1	few adults
<i>l(2)37Cf</i> ¹⁵	EMS	Wright	<i>l(2)684</i>	1	
<i>l(2)37Cf</i> ¹⁶	EMS+γ ray	Wright	<i>l(2)7403</i>	3	some adults
<i>l(2)37Cf</i> ¹⁷	EMS+γ ray	Wright	<i>l(2)7418</i>	3	few adults
<i>l(2)37Cf</i> ¹⁸	EMS+γ ray	Wright	<i>l(2)7429</i>	3	1-kb deletion
<i>l(2)37Cf</i> ¹⁹	EMS	Lindsley	<i>l(2)158-1725</i>		
<i>l(2)37Cf</i> ²⁰	DEB	Cecil	<i>l(2)DEB18</i>		
<i>l(2)37Cf</i> ²¹	EMS	Schüpbach	<i>l(2)RSS3</i>		
<i>l(2)37Cf</i> ²²	EMS+γ ray	Cecil	<i>l(2)AA2</i>		
<i>l(2)37Cf</i> ²³	EMS+γ ray	Cecil	<i>l(2)C2</i>		
<i>l(2)37Cf</i> ²⁴	EMS+γ ray	Cecil	<i>l(2)CC5</i>		
<i>l(2)37Cf</i> ^{ts1}	EMS	Wright	<i>fs(2)E60</i>	1	female-sterile allele
<i>l(2)37Cf</i> ^{ts2}	EMS	Schüpbach	<i>fs(2)P1123</i>		female-sterile allele larval brains normal size
<i>l(2)37Cf</i> ^{ts3}	EMS	Schüpbach	<i>fs(2)PM43</i>		female-sterile allele
<i>l(2)37Cf</i> ^{ts1}	EMS	Wright	<i>l(2)E29</i> ^{ts}	2	temperature sensitive
<i>l(2)37Cf</i> ^{ts2}	EMS+HCOH	Wright	<i>l(2)336</i> ^{ts}	1	temperature sensitive
<i>l(2)37Cf</i> ^{ts3}	EMS	Lindsley	<i>l(2)50-899</i>		temperature sensitive

^α 1 = Wright, Beerman, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, Chromosoma 83: 45-58; 2 = Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310; 3 = Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26.

l(2)37Cg

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)37Cg</i> ¹	EMS+γ ray	Wright	<i>l(2)7414</i>	3	lethal in L2 after 7 days
<i>l(2)37Cg</i> ²	EMS	Wright	<i>l(2)615</i>	2	larval lethal; melanotic tumors
<i>l(2)37Cg</i> ^{ts1}	EMS+HCOH	Wright	<i>l(2)354</i> ^{ts}	1	temperature sensitive

^α 1 = Wright, Beerman, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, Chromosoma 83: 45-58; 2 = Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310; 3 = Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26.

l(2)37D-E

Eleven lethally mutable loci identified, ten in 37D and one in 37E. In addition to the alleles identified below, Wright, Bewley, and Sherald (1976, Genetics 84: 287-310) report seven mutants between the right break of *Df(2L)TW130* and the left break of *Df(2L)TW2* (designated *E27*, *E29*, *E56*, *E107*, *E137*, *E142M*, and *E151*) plus four between the left breakpoint of *Df(2L)TW2* and the right breakpoint of *Df(2L)TW158* (designated *E12*, *E34*, *E68*, and *E144M*) that were not tested for complementation.

locus	genetic location		included in	excluded from	synonym
	location	location			
<i>l(2)37Da</i>	2-[54]	37D2-7	<i>Df(2L)prA14</i>	<i>Df(2L)Sd2</i>	<i>l(2)OE23</i>
<i>l(2)37Db</i>	2-[54]	37D2-E1	<i>Df(2L)Sd2</i> <i>Df(2L)VA15</i>	<i>Df(2L)TW330</i>	<i>l(2)E19</i>
<i>l(2)37Dc</i>	2-[54]	37D2-E1	<i>Df(2L)Sd37</i>	<i>Df(2L)pr26</i>	<i>l(2)E106</i>
<i>l(2)37Dd</i>	2-[54]	37D2-E1	<i>Df(2L)Sd37</i>	<i>Df(2L)pr26</i>	<i>l(2)OE36</i>
<i>l(2)37De</i>	2-[54]	37D2-E1	<i>Df(2L)Sd37</i>	<i>Df(2L)pr26</i>	<i>l(2)OE30</i>
<i>l(2)37Df</i>	2-[54]	37D2-E1	<i>Df(2L)Sd37</i>	<i>Df(2L)pr26</i>	<i>l(2)OD8</i>
<i>l(2)37Dg</i>	2-[54]	37D2-E1	<i>Df(2L)pr26</i>	<i>Df(2L)TW2</i>	<i>l(2)OE39</i>
<i>l(2)37Dh</i>	2-[54]	37D2-F4	<i>Df(2L)TW2</i>	<i>Df(2L)TW9</i>	<i>l(2)E1</i>
<i>l(2)37Di</i>	2-[54]	37D2-E1	<i>Df(2L)TW2</i>	<i>Df(2L)TW9</i>	<i>l(2)E103</i>
<i>l(2)37Dj</i>	2-[54]	37D2-E1	<i>Df(2L)TW2</i>	<i>Df(2L)TW9</i>	<i>l(2)OE31</i>
<i>l(2)37Ea</i>	2-[54]	37E2-F4	<i>Df(2L)TW9</i> <i>Df(2L)TW158</i>		<i>l(2)E124</i>

l(2)37Da

phenotype: Semilethal, temperature-sensitive alleles. Escapers have a straight body and extended genital plates.

allele	origin	discoverer	synonym
<i>l(2)37Da</i> ¹	EMS	Gay, Contamine	<i>l(2)OE23</i>
<i>l(2)37Da</i> ²	EMS	Gay, Contamine	<i>l(2)OE68</i>
<i>l(2)37Da</i> ³	EMS	Gay, Contamine	<i>l(2)OE78</i>
<i>l(2)37Da</i> ⁴	EMS	Gay, Contamine	<i>l(2)OE80</i>
<i>l(2)37Da</i> ⁵	DEB	Gay, Contamine	<i>l(2)OD5</i>
<i>l(2)37Da</i> ⁶	DEB	Gay, Contamine	<i>l(2)OD32</i>

allele	origin	discoverer	synonym	ref ^α
<i>l(2)37Db</i> ¹	EMS	Wright	<i>l(2)E19</i>	1
<i>l(2)37Db</i> ²	EMS	Wright	<i>l(2)E63</i>	1
<i>l(2)37Db</i> ³	EMS	Wright	<i>l(2)E109</i>	1
<i>l(2)37Db</i> ⁴	EMS	Wright	<i>l(2)E139</i>	1
<i>l(2)37Db</i> ⁵	EMS	Gay, Contamine	<i>l(2)OE34</i>	
<i>l(2)37Db</i> ⁶	EMS	Gay, Contamine	<i>l(2)OE43</i>	
<i>l(2)37Db</i> ⁷	EMS	Gay, Contamine	<i>l(2)OE44</i>	
<i>l(2)37Db</i> ⁸	EMS	Gay, Contamine	<i>l(2)OE47</i>	
<i>l(2)37Db</i> ⁹	EMS	Gay, Contamine	<i>l(2)OE48</i>	
<i>l(2)37Db</i> ¹⁰	EMS	Gay, Contamine	<i>l(2)OE65</i>	
<i>l(2)37Db</i> ¹¹	EMS	Gay, Contamine	<i>l(2)OE87</i>	
<i>l(2)37Db</i> ¹²	EMS	Gay, Contamine	<i>l(2)OE90</i>	
<i>l(2)37Db</i> ¹³	EMS	Gay, Contamine	<i>l(2)OE95</i>	
<i>l(2)37Db</i> ¹⁴	EMS	Gay, Contamine	<i>l(2)OE97</i>	
<i>l(2)37Db</i> ¹⁵	DEB	Gay, Contamine	<i>l(2)OD2</i>	
<i>l(2)37Db</i> ¹⁶	DEB	Gay, Contamine	<i>l(2)OD26</i>	
<i>l(2)37Db</i> ¹⁷	DEB	Gay, Contamine	<i>l(2)OD33</i>	
<i>l(2)37Db</i> ¹⁸	EMS	Gay, Contamine	<i>l(2)PE1</i>	
<i>l(2)37Db</i> ¹⁹	EMS	Gay, Contamine	<i>l(2)PE6</i>	

^α 1 = Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)37Dc</i> ¹	EMS	Wright	<i>l(2)E106</i>	1	
<i>l(2)37Dc</i> ²	EMS	Gay, Contamine	<i>l(2)OE11</i>		
<i>l(2)37Dc</i> ³	EMS	Gay, Contamine	<i>l(2)OE20</i>		
<i>l(2)37Dc</i> ⁴	EMS	Gay, Contamine	<i>l(2)OE24</i>		
<i>l(2)37Dc</i> ⁵	EMS	Gay, Contamine	<i>l(2)OE28</i>		
<i>l(2)37Dc</i> ⁶	EMS	Gay, Contamine	<i>l(2)OE53</i>		
<i>l(2)37Dc</i> ⁷	EMS	Gay, Contamine	<i>l(2)OE64</i>		
<i>l(2)37Dc</i> ⁸	EMS	Gay, Contamine	<i>l(2)OE74</i>		
<i>l(2)37Dc</i> ⁹	EMS	Gay, Contamine	<i>l(2)OE93</i>		
<i>l(2)37Dc</i> ¹⁰	DEB	Gay, Contamine	<i>l(2)OD18</i>		
<i>l(2)37Dc</i> ¹¹	DEB	Gay, Contamine	<i>l(2)OD23</i>		

*l(2)37Dd*¹ EMS Gay, Contamine *l(2)OE36* 1 semilethal
^α 1 = Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310.

l(2)37De

alleles: Interallelic complementation between all Gay and Contamine alleles, except *l(2)De*¹⁸, and *l(2)De*¹, *l(2)De*², and *l(2)De*⁴.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)37De</i> ¹	EMS	Wright	<i>l(2)E2</i>	1	
<i>l(2)37De</i> ²	EMS	Wright	<i>l(2)E8</i>	1	
<i>l(2)37De</i> ³	EMS	Wright	<i>l(2)E39</i>	1	
<i>l(2)37De</i> ⁴	EMS	Wright	<i>l(2)E102</i>	1	
<i>l(2)37De</i> ⁵	EMS	Wright	<i>l(2)E108</i>	1	
<i>l(2)37De</i> ⁶	EMS	Gay, Contamine	<i>l(2)OE1</i>		
<i>l(2)37De</i> ⁷	EMS	Gay, Contamine	<i>l(2)OE4</i>		
<i>l(2)37De</i> ⁸	EMS	Gay, Contamine	<i>l(2)OE6</i>		
<i>l(2)37De</i> ⁹	EMS	Gay, Contamine	<i>l(2)OE7</i>		
<i>l(2)37De</i> ¹⁰	EMS	Gay, Contamine	<i>l(2)OE8</i>		
<i>l(2)37De</i> ¹¹	EMS	Gay, Contamine	<i>l(2)OE9</i>		
<i>l(2)37De</i> ¹²	EMS	Gay, Contamine	<i>l(2)OE10</i>		
<i>l(2)37De</i> ¹³	EMS	Gay, Contamine	<i>l(2)OE15</i>		
<i>l(2)37De</i> ¹⁴	EMS	Gay, Contamine	<i>l(2)OE19</i>		

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)37De</i> ¹⁵	EMS	Gay, Contamine	<i>l(2)OE21</i>		
<i>l(2)37De</i> ¹⁶	EMS	Gay, Contamine	<i>l(2)OE25</i>		
<i>l(2)37De</i> ¹⁷	EMS	Gay, Contamine	<i>l(2)OE29</i>		complex aberration
<i>l(2)37De</i> ¹⁸	EMS	Gay, Contamine	<i>l(2)OE30</i>		
<i>l(2)37De</i> ¹⁹	EMS	Gay, Contamine	<i>l(2)OE35</i>		
<i>l(2)37De</i> ²⁰	EMS	Gay, Contamine	<i>l(2)OE37</i>		
<i>l(2)37De</i> ²¹	EMS	Gay, Contamine	<i>l(2)OE38</i>		temperature sensitive
<i>l(2)37De</i> ²²	EMS	Gay, Contamine	<i>l(2)OE41</i>		temperature sensitive
<i>l(2)37De</i> ²³	EMS	Gay, Contamine	<i>l(2)OE42</i>		
<i>l(2)37De</i> ²⁴	EMS	Gay, Contamine	<i>l(2)OE46</i>		Glued-like phenotype
<i>l(2)37De</i> ²⁵	EMS	Gay, Contamine	<i>l(2)OE50</i>		temperature sensitive
<i>l(2)37De</i> ²⁶	EMS	Gay, Contamine	<i>l(2)OE54</i>		
<i>l(2)37De</i> ²⁷	EMS	Gay, Contamine	<i>l(2)OE57</i>		
<i>l(2)37De</i> ²⁸	EMS	Gay, Contamine	<i>l(2)OE69</i>		
<i>l(2)37De</i> ²⁹	EMS	Gay, Contamine	<i>l(2)OE79</i>		
<i>l(2)37De</i> ³⁰	EMS	Gay, Contamine	<i>l(2)OE82</i>		
<i>l(2)37De</i> ³¹	EMS	Gay, Contamine	<i>l(2)OE85</i>		
<i>l(2)37De</i> ³²	EMS	Gay, Contamine	<i>l(2)OE91</i>		
<i>l(2)37De</i> ³³	EMS	Gay, Contamine	<i>l(2)OE98</i>		
<i>l(2)37De</i> ³⁴	EMS	Gay, Contamine	<i>l(2)OE99</i>		
<i>l(2)37De</i> ³⁵	EMS	Gay, Contamine	<i>l(2)OE100</i>		
<i>l(2)37De</i> ³⁶	DEB	Gay, Contamine	<i>l(2)OD1</i>		
<i>l(2)37De</i> ³⁷	DEB	Gay, Contamine	<i>l(2)OD19</i>		
<i>l(2)37De</i> ³⁸	DEB	Gay, Contamine	<i>l(2)OD27</i>		
<i>l(2)37De</i> ³⁹	DEB	Gay, Contamine	<i>l(2)OD28</i>		
<i>l(2)37De</i> ⁴⁰	EMS	Gay, Contamine	<i>l(2)PE3</i>		
<i>l(2)37De</i> ⁴¹	EMS	Gay, Contamine	<i>l(2)PE4</i>		
<i>l(2)37De</i> ⁴²	EMS	Gay, Contamine	<i>l(2)PE5</i>		

^α 1 = Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310.

allele	origin	discoverer	synonym	comments
<i>l(2)37Df</i> ¹	DEB	Gay, Contamine	<i>l(2)OD8</i>	semilethal
<i>l(2)37Dg</i> ¹	EMS	Gay, Contamine	<i>l(2)OE39</i>	
<i>l(2)37Dg</i> ²	EMS	Gay, Contamine	<i>l(2)OE62</i>	

l(2)37Dh

phenotype: Interferes with *M* and *M*-like mutants.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)37Dh</i> ¹	EMS	Wright	<i>l(2)E1</i>	1	
<i>l(2)37Dh</i> ²	EMS	Gay, Contamine	<i>l(2)OE2</i>		
<i>l(2)37Dh</i> ³	EMS	Gay, Contamine	<i>l(2)OE13</i>		
<i>l(2)37Dh</i> ⁴	EMS	Gay, Contamine	<i>l(2)OE22</i>		
<i>l(2)37Dh</i> ⁵	EMS	Gay, Contamine	<i>l(2)OE27</i>		
<i>l(2)37Dh</i> ⁶	EMS	Gay, Contamine	<i>l(2)OE77</i>		
<i>l(2)37Dh</i> ⁷	EMS	Gay, Contamine	<i>l(2)OE83</i>		
<i>l(2)37Dh</i> ⁸	DEB	Gay, Contamine	<i>l(2)OD10</i>		
<i>l(2)37Dh</i> ⁹	DEB	Gay, Contamine	<i>l(2)OD30</i>		

^α 1 = Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(2)37DI</i> ¹	EMS	Wright	<i>l(2)E103</i>	1	
<i>l(2)37DI</i> ²	EMS	Gay, Contamine	<i>l(2)OE16</i>		
<i>l(2)37DI</i> ³	EMS	Gay, Contamine	<i>l(2)OE32</i>		
<i>l(2)37DI</i> ⁴	EMS	Gay, Contamine	<i>l(2)OE49</i>		
<i>l(2)37DI</i> ⁵	EMS	Gay, Contamine	<i>l(2)OE51</i>		
<i>l(2)37DI</i> ⁶	EMS	Gay, Contamine	<i>l(2)OE52</i>		
<i>l(2)37DI</i> ⁷	EMS	Gay, Contamine	<i>l(2)OE61</i>		
<i>l(2)37DI</i> ⁸	EMS	Gay, Contamine	<i>l(2)OE63</i>		
<i>l(2)37DI</i> ⁹	EMS	Gay, Contamine	<i>l(2)OE66</i>		
<i>l(2)37DI</i> ¹⁰	EMS	Gay, Contamine	<i>l(2)OE67</i>		
<i>l(2)37DI</i> ¹¹	EMS	Gay, Contamine	<i>l(2)OE73</i>		
<i>l(2)37DI</i> ¹²	EMS	Gay, Contamine	<i>l(2)OE81</i>		
<i>l(2)37DI</i> ¹³	EMS	Gay, Contamine	<i>l(2)OE86</i>		
<i>l(2)37DI</i> ¹⁴	EMS	Gay, Contamine	<i>l(2)OE89</i>		
<i>l(2)37DI</i> ¹⁵	EMS	Gay, Contamine	<i>l(2)OE94</i>		
<i>l(2)37DI</i> ¹⁶	DEB	Gay, Contamine	<i>l(2)OD4</i>		
<i>l(2)37DJ</i> ¹	EMS	Gay, Contamine	<i>l(2)OE31</i>		

allele	origin	discoverer	synonym	ref ^α	comments
I(2)37D¹	EMS	Gay, Contamine	<i>I(2)OE40</i>		
I(2)37D²	EMS	Gay, Contamine	<i>I(2)OE58</i>		
I(2)37D³	EMS	Gay, Contamine	<i>I(2)OE59</i>		
I(2)37D⁴	EMS	Gay, Contamine	<i>I(2)OE72</i>		
I(2)37D⁵	DEB	Gay, Contamine	<i>I(2)OD25</i>		
I(2)37D⁶	DEB	Gay, Contamine	<i>I(2)OD31</i>		
I(2)37Ea¹	EMS		<i>I(2)E124</i>	1	
I(2)37Ea²	EMS	Gay, Contamine	<i>I(2)OE17</i>		
I(2)37Ea³	EMS	Gay, Contamine	<i>I(2)OE56</i>		temperature sensitive
I(2)37Ea⁴	EMS	Gay, Contamine	<i>I(2)OE75</i>		temperature sensitive
I(2)37Ea⁵	EMS	Gay, Contamine	<i>I(2)OE88</i>		
I(2)37Ea⁶	EMS	Gay, Contamine	<i>I(2)OE102</i>		
I(2)37Ea⁷	DEB	Gay, Contamine	<i>I(2)OD3</i>		

^α 1 = Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310.

I(2)37F

locus	genetic location	cytological location	included in	excluded from	synonym
I(2)37Fa	2-[54]	37E3-38A1	<i>Df(2L)TW12</i>	<i>Df(2L)OD21</i>	<i>I(2)OE92</i>
I(2)37Fb	2-[54]	37E3-38A1	<i>Df(2L)TW12</i>	<i>Df(2L)OD21</i>	<i>I(2)E146</i>
I(2)37Fc	2-[54]	37E3-38A1	<i>Df(2L)OD21</i>	<i>Df(2L)TW84</i>	<i>I(2)E42</i>
I(2)37Fd	2-[54]	37E3-38A1	<i>Df(2L)TW84</i>	<i>Df(2L)E55</i>	<i>I(2)OE55</i>
I(2)37Fe	2-[54]	37F5-38A1	<i>Df(2L)TW150</i>	<i>Df(2L)E55</i>	<i>I(2)E3</i>
I(2)37Ff	2-[54]	37F5-38A1	<i>Df(2L)TW65</i>	<i>Df(2L)H1</i>	<i>I(2)E43</i>
I(2)37Fg	2-[54]	37F5-38A1	<i>Df(2L)TW150</i>	<i>Df(2L)E55</i>	<i>I(2)O9</i>
I(2)37Fh	2-[54]	37F5-38A1	<i>Df(2L)E55</i>	<i>Df(2L)TW65</i>	<i>I(2)C188</i>

allele	origin	discoverer	synonym	ref ^α
I(2)37Fa¹	EMS	Gay, Contamine	<i>I(2)OE92</i>	
I(2)37Fa²	DEB	Gay, Contamine	<i>I(2)OD7</i>	
I(2)37Fa³	DEB	Gay, Contamine	<i>I(2)OD24</i>	
I(2)37Fb¹	EMS	Wright	<i>I(2)E146</i>	1
I(2)37Fb²	EMS	Gay, Contamine	<i>I(2)OE18</i>	
I(2)37Fb³	EMS	Gay, Contamine	<i>I(2)OE26</i>	
I(2)37Fb⁴	EMS	Gay, Contamine	<i>I(2)OE33</i>	
I(2)37Fb⁵	EMS	Gay, Contamine	<i>I(2)OE60</i>	
I(2)37Fb⁶	EMS	Gay, Contamine	<i>I(2)OE71</i>	
I(2)37Fb⁷	EMS	Gay, Contamine	<i>I(2)OE101</i>	
I(2)37Fb⁸	EMS	Gay, Contamine	<i>I(2)PE7</i>	
I(2)37Fb⁹	DEB	Gay, Contamine	<i>I(2)OD29</i>	
I(2)37Fc¹	EMS	Wright	<i>I(2)E42</i>	1
I(2)37Fc²	EMS	Wright	<i>I(2)E72</i>	1
I(2)37Fc³	EMS	Gay, Contamine	<i>I(2)OE14</i>	
I(2)37Fc⁴	EMS	Gay, Contamine	<i>I(2)OE76</i>	
I(2)37Fc⁵	EMS	Gay, Contamine	<i>I(2)OE96</i>	
I(2)37Fc⁶	DEB	Gay, Contamine	<i>I(2)OD6</i>	

^α 1 = Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310.

I(2)37Fd

phenotype: Semilethal with opaque wings.

allele	origin	discoverer	synonym
I(2)37Fd¹	EMS	Gay, Contamine	<i>I(2)OE55</i>
I(2)37Fd²	EMS	Gay, Contamine	<i>I(2)OE103</i>
I(2)37Fd³	EMS	Gay, Contamine	<i>I(2)OE104</i>
I(2)37Fd⁴	DEB	Gay, Contamine	<i>I(2)OD11</i>

allele	origin	discoverer	synonym	ref ^α
I(2)37Fe	EMS	Wright	<i>I(2)E3</i>	1
I(2)37Ff	EMS	Wright	<i>I(2)E43</i>	1
I(2)37Fg		Gay, Contamine	<i>I(2)O9</i>	
I(2)37Fh¹		Gay, Contamine	<i>I(2)C188</i>	
I(2)37Fh²		Gay, Contamine	<i>I(2)OD11</i>	

^α 1 = Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310.

I(2)38A

locus	genetic location	cytological location	included in	synonym
I(2)38Aa	2-[54]	38A6-C1	<i>Df(2L)pr49</i>	<i>I(2)E124P</i>
			<i>Df(2L)TW150</i>	
I(2)38Ab	2-[54]	38A6-C1	<i>Df(2L)pr49</i>	<i>I(2)A113</i>
			<i>Df(2L)TW150</i>	

allele	origin	discoverer	synonym	ref ^α
I(2)38Aa¹	EMS	Wright	<i>I(2)E124P</i>	1
I(2)38Ab¹		Gay, Contamine	<i>I(2)A113</i>	

^α 1 = Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310.

DEFICIENCY MAP OF REGION 37-38

side	breakpoint	variant	DNA coordinates ^α
right	36F1-37A1	<i>Df(2L)M36F-55</i>	
left		<i>Df(2L)VA20</i>	
right	37A	<i>Df(2L)TW202</i>	
left		<i>Df(2L)VA14</i>	
left		<i>Df(2L)VA15</i>	
left		<i>Df(2L)VA25</i>	
		I(2)37Aa	
		I(2)37Ab	
		I(2)37Ac	
right	37A1-B1	<i>Df(2L)H68</i>	
right	37B2-8	<i>Df(2L)TW3</i>	
		I(2)37Bi	
		I(2)37Bj	
left	37B2-8	<i>Df(2L)TW158</i>	
left		<i>Df(2L)VA24</i>	
left		<i>Df(2L)prA16</i>	
right	37B9-C1	<i>Df(2L)TW137</i>	
left		<i>Df(2L)hk-UC1</i>	
left		<i>Df(2L)hk-UC2</i>	
left	37B3-7	<i>Df(2L)Sd68</i>	
left	37B9-C1	<i>Df(2L)TW130</i>	
left	37B9-C1	<i>Df(2L)VA23</i>	
		hk	-84.95
right	37B9-C1	<i>Df(2L)TW203</i>	-83.25 to -81.35
		I(2)37Be	-79.35 to -74.75
		I(2)37Bc	-74.05 to -72.25
		I(2)37Ba	-72.35 to -66.60
right		<i>Df(2L)OD15</i>	-67.37 to -67.25
		TU37B2^Y	-67.36 to -66.63
		I(2)37Bb	-66.68 to -64.92
		Dox-A2	-64.63 to -62.92
		TU37B1^Y	-62.15 to -57.15
right		<i>Df(2L)hk-UC1</i>	-56.75 to -54.95
right	37B9-C1	<i>Df(2L)hk18</i>	-52.15 to -50.55
		I(2)37Bg	-43.35 to -34.95
		I(2)37Bd	-39.35 to -13.65
left		<i>Df(2L)NST</i>	-38.35 to -29.85
		TU37B3^Y	-15.05 to -13.65
		amd	-4.85 to -2.42
		TU37C1^Y	-1.74 to +0.02
left	37B9-C1	<i>Df(2L)VA17</i>	+2.75 to +3.35
	37C1-2	Ddc	-0.06 to +3.76
		I(2)37Cc	+5.85 to +8.15

THE GENOME OF *DROSOPHILA MELANOGASTER*

side	breakpoint	variant	DNA coordinates ^α
right	37C2-5	<i>Df(2L)VA18</i>	+6.05 to +6.55
		I(2)37Cb	+8.15 to +10.95
right		<i>Df(2L)hk-UC2</i>	+8.15 to +10.95
		I(2)37Cd	+10.25 to +12.95
		I(2)37Ca	+14.25 to +20.95
		I(2)37Cg	+14.25 to +20.95
		I(2)37Ce	+14.25 to +20.95
left	37C2-5	<i>Df(2L)TE42-1</i>	+26.45 to +29.05
left		<i>Df(2L)TE42B7</i>	
left	37C2-5	<i>Df(2L)VA12</i>	+26.45 to +29.05
right		<i>Df(2L)VA21</i>	+27.95 to +30.75
left	37C2-D1	<i>Df(2L)VA19</i>	+30.75 to +31.75
left		<i>Df(2L)VA13</i>	+31.65 to +36.35
left	37D1-2	<i>Df(2L)Sd57</i>	+38.55 to +40.55
		I(2)37Cf	
left	37D1-2	<i>Df(2L)Sd77</i>	+47.05 to +52.55
		fs(2)TW1	+47.05 to +58.25
right		<i>Df(2L)TE42B7</i>	
right		<i>Df(2L)NST</i>	
right	37C6-D1	<i>Df(2L)E71</i>	
right	37D1-2	<i>Df(2L)hk39</i>	
right	37D1-2	<i>Df(2L)TW130</i>	
right		<i>Df(2L)VA14</i>	
right		<i>Df(2L)VA16</i>	
left		<i>Df(2L)E55</i>	+52.55 to +58.25
left		<i>Df(2L)VA6</i>	+52.55 to +58.25
right		<i>Df(2L)VA25</i>	
left		<i>Df(2L)VA8</i>	
right		<i>Df(2L)VA22</i>	
left	37D2-E1?	<i>Df(2L)prA14</i>	
left	37D1-2	<i>Df(2L)Sd37</i>	
right		<i>Df(2L)TW330</i>	
		I(2)37Da	
left	37D1-2	<i>Df(2L)Sd2</i>	
left	37D1-2	<i>Df(2L)Sd14</i>	
	37D2-6	Sd	
		I(2)37Db	
right	37D2-E1	<i>Df(2L)VA15</i>	
right	37D2-E1	<i>Df(2L)VA20</i>	
right	37D2-E1	<i>Df(2L)VA23</i>	
	37D2-6	Top2	
		I(2)37Dc	
		I(2)37Dd	
		I(2)37De	
		I(2)37Df	
left	37D5-6	<i>Df(2L)pr26</i>	
		I(2)37Dg	
left	37D2-E1	<i>Df(2L)TW2</i>	
		fs(2)37D	
		mfs(2)37D	
		I(2)37Dh	
		I(2)37Di	
		I(2)37Dj	
right		<i>Df(2L)OD9</i>	
left		<i>Df(2L)ODP5</i>	
left		<i>Dp(2;Y)G-F280</i>	
left	37E2-F4	<i>Df(2L)TW9</i>	
		I(2)37Ea	
right	37E2-F4	<i>Df(2L)TW158</i>	
	37E3-F1	<i>Dp(1;Y)G-M15</i>	
	37E3-F1	ref(2)P	
left	37E3-F1	<i>Df(2L)CPP2R</i>	
left		<i>Dp(2;Y)G-H3</i>	
left	37E3-F1	<i>Df(2L)pr21</i>	
left	37E2-F4	<i>Df(2L)TW12</i>	
left		<i>Df(2L)OD12</i>	
		I(2)37Fa	
		I(2)37Fb	
left		<i>Df(2L)OD21</i>	
		I(2)37Fc	
left	37F5-38A1	<i>Df(2L)TW84</i>	
left	37F5-38A1	<i>Df(2L)TW150</i>	
left		<i>Df(2L)OD16</i>	
		I(2)37Fd	
right		<i>Df(2L)ODP5</i>	
right		<i>Df(2L)VA17</i>	

side	breakpoint	variant	DNA coordinates ^α
		I(2)37Fh	
right	37F5-38A1	<i>Df(2L)E55</i>	
		I(2)37Fe	
		I(2)37Fi	
left	37F5-38A1	<i>Df(2L)TW65</i>	
		I(2)37Fg	
left		<i>Df(2L)H1</i>	
right		<i>Df(2L)CPP2R</i>	
right		<i>Df(2L)OD16</i>	
left	38A3-4	<i>Df(2L)prA20</i>	
left	38A3-5	<i>Df(2L)pr65</i>	
left	38A5-8	<i>Df(2L)pr67</i>	
right	38A6-7	<i>Df(2L)TW50</i>	
right	38A6-B2	<i>Df(2L)Sd37</i>	
left		<i>Df(2L)pr-B12</i>	
left	38A6-B1	<i>Df(2L)TW161</i>	
left	38A6-B1	<i>Df(2L)TW1</i>	
left	38B1-2	<i>Df(2L)pr47</i>	
left	38B1-2	<i>Df(2L)pr69</i>	
left	38B3-6	<i>Df(2L)pr49</i>	
	38B1-2	pr	
right ^β	38A6-C1	<i>Df(2L)TW9</i>	
		I(2)38Ba	
		I(2)38Bb	
right	38B2-C1	<i>Df(2L)TW150</i>	
right	38B2-C1	<i>Df(2L)VA12</i>	
right	38B6-C1	<i>Df(2L)pr-A20</i>	
right	38C1-2	<i>Df(2L)pr47</i>	
right	38C1-2	<i>Df(2L)Sd14</i>	
right	38C1-2	<i>Df(2L)Sd57</i>	
right	38C1-2	<i>Df(2L)Sd77</i>	
right	38C5-6	<i>Df(2L)pr69</i>	
	38C6-9	ms(2)38C	
right	38C6-10	<i>Df(2L)pr21</i>	
right	38C6-10	<i>Df(2L)pr49</i>	
right	38C8-10	<i>Df(2L)pr26</i>	
right	38D3-5	<i>Df(2L)pr65</i>	
right	38D2-E1	<i>Df(2L)pr-A16</i>	
right	38D2-E1	<i>Df(2L)Sd2</i>	
right	38E3-5	<i>Df(2L)Sd68</i>	
right	38F2-39A1	<i>Df(2L)VA6</i>	
		BI	
right	38E6-9	<i>Df(2L)TW2</i>	
right	38F5-39A1	<i>Df(2L)VA8</i>	
right	38F5-39A1	<i>Df(2L)TE37C-1</i>	

^α Coordinates of restriction fragments containing the genes and breakpoints in question; 0 is the axis of symmetry of the *HpaI* site near the terminus of the *Ddc* coding sequence. The origin of this set of coordinates is approximately 950 base pairs to the left of that used by Hersh and Wright, and their coordinates have been adjusted accordingly.

^β Breakpoint not localized with respect to neighboring lethal mutations.

^γ TU = transcription unit.

I(2)39a

location: 2-50 (right of *BI* ?).

origin: Spontaneous.

discoverer: Curry, 39a.

references: 1939, DIS 12: 45.

I(2)40F

Twenty-eight ethyl-methanesulfonate-induced recessive lethal mutations that are not complemented by *Df(2L)C'*, which is thought to delete only 2L heterochromatin (Hilliker, 1976, Genetics 83: 765-82). All seven loci are localized to heterochromatic segment h35 (Pimpinelli).

locus	genetic location	cytological location	included in	excluded from	synonym
I(2)40Fa	2-55.0	40F	<i>Df(2L)C'</i>	<i>Df(2L)C</i>	

locus	genetic location	cytological location	included in	excluded from	synonym
<i>I(2)40Fb</i>	2-55.0	40F	<i>Df(2L)C</i>	<i>Df(2L)D</i>	It
<i>I(2)40Fc</i>	2-55.0	40F	<i>Df(2L)C</i>	<i>Df(2L)D</i>	
<i>I(2)40Fd</i>	2-55.0	40F	<i>Df(2L)C</i>	<i>Df(2L)D</i>	
<i>I(2)40Fe</i>	2-55.0	40F	<i>Df(2L)D</i>	<i>Df(2L)D</i>	
<i>I(2)40Ff</i>	2-55.0	40F	<i>Df(2L)D</i>	<i>Df(2L)D</i>	
<i>I(2)40Fg</i>	2-55.0	40F	<i>Df(2L)D</i>	<i>Df(2L)D</i>	

I(2)40Fa

phenotype: Hemizygotes pupate, but subsequently undergo complete autolysis with no adult structures present and larval tissues being reduced to an oily mass at the bottom of the pupal case.

allele	synonym
<i>I(2)40Fa</i>¹	<i>I(2)EMS40-18</i>
<i>I(2)40Fa</i>²	<i>I(2)EMS56-09</i>
<i>I(2)40Fc</i>¹	<i>I(2)EMS40-02</i>
<i>I(2)40Fc</i>²	<i>I(2)EMS56-06</i>
<i>I(2)40Fc</i>³	<i>I(2)EMS56-32</i>
<i>I(2)40Fd</i>¹	<i>I(2)EMS40-06</i>
<i>I(2)40Fd</i>²	<i>I(2)EMS40-07</i>
<i>I(2)40Fd</i>³	<i>I(2)EMS40-08</i>
<i>I(2)40Fd</i>⁴	<i>I(2)EMS40-20</i>
<i>I(2)40Fd</i>⁵	<i>I(2)EMS40-21</i>
<i>I(2)40Fd</i>⁶	<i>I(2)EMS56-01</i>
<i>I(2)40Fd</i>⁷	<i>I(2)EMS56-02</i>
<i>I(2)40Fd</i>⁸	<i>I(2)EMS56-05</i>
<i>I(2)40Fd</i>⁹	<i>I(2)EMS56-07</i>
<i>I(2)40Fd</i>¹⁰	<i>I(2)EMS56-09</i>
<i>I(2)40Fd</i>¹¹	<i>I(2)EMS56-10</i>
<i>I(2)40Fd</i>¹²	<i>I(2)EMS56-16</i>
<i>I(2)40Fd</i>¹³	<i>I(2)EMS56-20</i>
<i>I(2)40Fd</i>¹⁴	<i>I(2)EMS56-27</i>

I(2)40Fe

Only one allele; complements all other mutations in the region. Hemizygote shows 2% viability.

allele	synonym
<i>I(2)40Fe</i>¹	<i>I(2)EMS56-24</i>

I(2)40Ff

*I(2)40Ff*⁴ semilethal; exhibits 20%, 33%, 26%, and 35% survival in combination with a deficiency, *I(2)40Ff*¹, *I(2)40Ff*², and *I(2)40Ff*³, respectively; these combinations late eclosing.

allele	synonym	comments
<i>I(2)40Ff</i>¹	<i>I(2)EMS56-04</i>	
<i>I(2)40Ff</i>²	<i>I(2)EMS56-14</i>	
<i>I(2)40Ff</i>³	<i>I(2)EMS56-15</i>	
<i>I(2)40Ff</i>⁴	<i>I(2)EMS40-22</i>	semilethal

I(2)40Fg

phenotype: Rare escapers are late eclosing and although normal in external phenotype are greatly reduced in size.

allele	synonym
<i>I(2)40Fg</i>¹	<i>I(2)EMS40-05</i>
<i>I(2)40Fg</i>²	<i>I(2)EMS56-19</i>

DEFICIENCY MAP OF REGION 40

side	breakpoint	variant
left	40F	<i>Df(2L)F</i>
left	40F	<i>Df(2L)C</i>
		<i>I(2)40Fa</i>
right	40F	<i>Df(2L)F</i>
left	40F	<i>Df(2L)C</i>
		It
		<i>I(2)40Fc</i>
		<i>I(2)40Fd</i>
		<i>mat(2)cta</i>
left	40F	<i>Df(2L)D</i>
		<i>I(2)40Fe</i>
		<i>I(2)40Ff</i>
left	40F	<i>Df(2L)D</i>
		<i>I(2)40Fg</i>
right	40F	<i>Df(2L)C</i>
right	40F	<i>Df(2L)C</i>
right	40F	<i>Df(2L)D</i>
right	40F	<i>Df(2L)D</i>

I(2)41A

A series of 85 ethyl-methanesulfonate-induced recessive lethal mutations comprising seven complementation groups, including *rl*, *uex*, and *M(2)41A*, detected by virtue of their inability to complement *Df(2R)M41A* and other deficiencies for the proximal heterochromatin of 2R. All mutants occupy but a single complementation group, and no mutant alleles of *M(2)41A*, which lies in the region, were recovered (Hilliker, 1976, Genetics 83: 765-82).

locus	genetic location	cytological location	included in	excluded from	synonym
<i>I(2)41Aa</i>	2-55.1	41A	<i>Df(2R)B</i>		
<i>I(2)41Ab</i>	2-55.1	41A	<i>Df(2R)B</i>		
<i>I(2)41Ac</i>	2-55.1	41A	<i>Df(2R)A</i>	<i>Df(2R)B</i>	rl
<i>I(2)41Ad</i>	2-55.1	41A	<i>Df(2R)A</i>	<i>Df(2R)A</i>	uex
<i>I(2)41Ae</i>	2-55.1	41A	<i>Df(2R)A</i>	<i>Df(2R)A</i>	
<i>I(2)41Af</i>	2-55.1	41A	<i>Df(2R)A</i>	<i>Df(2R)A</i>	
<i>I(2)41Ag</i>	2-55.1	41A	<i>Df(2R)A</i>	<i>Df(2R)A</i>	M(2)41A
<i>I(2)41Ah</i>	2-55.1	41A	<i>Df(2R)M41A</i>	<i>Df(2R)A</i>	

I(2)41Aa

phenotype: In heterozygous combination with *Df(2L)B* dies as late third-instar larvae exhibiting one or more large and numerous small melanotic masses in the hemocoel.

allele	synonym
<i>I(2)41Aa</i>¹	<i>I(2)EMS31</i>

I(2)41Ab

phenotype: Late larval lethal, but without melanotic masses. Alleles apparently form a circular complementation map.

allele	synonym
<i>I(2)41Ab</i>¹	<i>I(2)EMS45-10</i>
<i>I(2)41Ab</i>²	<i>I(2)EMS45-84</i>
<i>I(2)41Ab</i>³	<i>I(2)EMS45-87</i>
<i>I(2)41Ab</i>⁴	<i>I(2)EMS45-91</i>

I(2)41Ae

phenotype: Heterozygotes not Minute. Complementation map shaped like a figure eight and highly complex. Four heteroallelic combinations exhibit partial complementa-

tion: *l(2)41Ae*¹⁰/*l(2)41Ae*¹⁵ shows 17% survival, *l(2)41Ae*¹⁰/*l(2)41Ae*¹⁹ 8% survival, *l(2)41Ae*⁹/*l(2)41Ae*²⁸ 10% survival, and *l(2)41Ae*²¹/*l(2)41Ae*²⁸ 21% viability. The remaining combinations exhibit non-complementation or complete complementation. In spite of the fact that among alleles *l(2)41Ae*³⁵ to *l(2)41Ae*⁴⁰, there is only one noncomplementing pair of alleles, *l(2)41Ae*³⁵ and *l(2)41Ae*³⁶, these mutants are designated alleles of *l(2)41Ae* on the basis of their inclusion in *Df(2R)M41A-4*, *Df(2R)M41A-8*, *Df(2R)M41A-10*, and *Df(2R)M41A-50j* and the extensive complementation characteristic of *l(2)41Ae* alleles.

allele	synonym	ref ^α
<i>l(2)41Ae</i>¹	<i>l(2)EMS34-02</i>	1
<i>l(2)41Ae</i>²	<i>l(2)EMS34-03</i>	1
<i>l(2)41Ae</i>³	<i>l(2)EMS34-08</i>	1
<i>l(2)41Ae</i>⁴	<i>l(2)EMS34-10</i>	1
<i>l(2)41Ae</i>⁵	<i>l(2)EMS34-11</i>	1
<i>l(2)41Ae</i>⁶	<i>l(2)EMS34-13</i>	1
<i>l(2)41Ae</i>⁷	<i>l(2)EMS34-14</i>	1
<i>l(2)41Ae</i>⁸	<i>l(2)EMS34-25</i>	1
<i>l(2)41Ae</i>⁹	<i>l(2)EMS34-26</i>	1
<i>l(2)41Ae</i>¹⁰	<i>l(2)EMS34-28</i>	1
<i>l(2)41Ae</i>¹¹	<i>l(2)EMS45-03</i>	1
<i>l(2)41Ae</i>¹²	<i>l(2)EMS45-04</i>	1
<i>l(2)41Ae</i>¹³	<i>l(2)EMS45-09</i>	1
<i>l(2)41Ae</i>¹⁴	<i>l(2)EMS45-11</i>	1
<i>l(2)41Ae</i>¹⁵	<i>l(2)EMS45-16</i>	1
<i>l(2)41Ae</i>¹⁶	<i>l(2)EMS45-20</i>	1
<i>l(2)41Ae</i>¹⁷	<i>l(2)EMS45-23</i>	1
<i>l(2)41Ae</i>¹⁸	<i>l(2)EMS45-27</i>	1
<i>l(2)41Ae</i>¹⁹	<i>l(2)EMS45-28</i>	1
<i>l(2)41Ae</i>²⁰	<i>l(2)EMS45-33</i>	1
<i>l(2)41Ae</i>²¹	<i>l(2)EMS45-34</i>	1
<i>l(2)41Ae</i>²²	<i>l(2)EMS45-35</i>	1
<i>l(2)41Ae</i>²³	<i>l(2)EMS45-53</i>	1
<i>l(2)41Ae</i>²⁴	<i>l(2)EMS45-60</i>	1
<i>l(2)41Ae</i>²⁵	<i>l(2)EMS45-61</i>	1
<i>l(2)41Ae</i>²⁶	<i>l(2)EMS45-64</i>	1
<i>l(2)41Ae</i>²⁷	<i>l(2)EMS45-70</i>	1
<i>l(2)41Ae</i>²⁸	<i>l(2)EMS45-71</i>	1
<i>l(2)41Ae</i>²⁹	<i>l(2)EMS45-83</i>	1
<i>l(2)41Ae</i>³⁰	<i>l(2)EMS45-86</i>	1
<i>l(2)41Ae</i>³¹	<i>l(2)EMS187</i>	1
<i>l(2)41Ae</i>³²	<i>l(2)EMS693</i>	1
<i>l(2)41Ae</i>³³	<i>l(2)EMS788</i>	1
<i>l(2)41Ae</i>³⁴	<i>l(2)EMS885</i>	1
<i>l(2)41Ae</i>³⁵	<i>l(2)C113</i>	3
<i>l(2)41Ae</i>³⁶	<i>l(2)C114</i>	3
<i>l(2)41Ae</i>³⁷	<i>l(2)C143</i>	3
<i>l(2)41Ae</i>³⁸	<i>l(2)CS15</i>	3
<i>l(2)41Ae</i>³⁹	<i>l(2)Sp9b</i> ^β	2
<i>l(2)41Ae</i>⁴⁰	<i>l(2)Sp11</i> ^γ	2

^α 1 = Hilliker, 1976, Genetics 83: 765-82; 2 = Spiess, Helling, and Capenos, 1963, Genetics 48: 1377-88; 3 = Tano, 1966, Jpn. J. Genet. 41: 299-308.

^β *l(2)C147* of Tano.

^γ *l(2)C124* of Tano.

l(2)41Af

A single mutation that deficiency maps to the same region as *M(2)41A* and *l(2)41Ae*, but complements all alleles of the latter, and exhibits no Minute phenotype.

allele	synonym
<i>l(2)41Af1</i>	<i>l(2)EMS4--72</i>

l(2)41Ah

Located very close to the heterochromatic-euchromatic junction of 2R. Complementation map circular, with the majority of mutants falling into a single complementation group that is complemented by only three of the thirty alleles. *l(2)41Ah*⁸/*l(2)41Ah*¹² survives and has wings uniformly spread from the body at a 45° angle; *l(2)41Ah*³/*l(2)41Ah*¹² survivors have in addition misshapen, unpigmented or absent ocelli. *l(2)41Ah*³¹ to *l(2)41Ah*³⁴ not tested against the remaining alleles; designated alleles because they are mutually noncomplementing, and they are included in *Df(2R)M41A-4*, *Df(2R)M41A-8*, and *Df(2R)M41A-10*, but not *Df(2R)M41A-50j*.

allele	synonym	ref ^α
<i>l(2)41Ah</i>¹	<i>l(2)EMS34-04</i>	1
<i>l(2)41Ah</i>²	<i>l(2)EMS34-12</i>	1
<i>l(2)41Ah</i>³	<i>l(2)EMS34-20</i>	1
<i>l(2)41Ah</i>⁴	<i>l(2)EMS34-21</i>	1
<i>l(2)41Ah</i>⁵	<i>l(2)EMS34-22</i>	1
<i>l(2)41Ah</i>⁶	<i>l(2)EMS34-23</i>	1
<i>l(2)41Ah</i>⁷	<i>l(2)EMS34-27</i>	1
<i>l(2)41Ah</i>⁸	<i>l(2)EMS45-08</i>	1
<i>l(2)41Ah</i>⁹	<i>l(2)EMS45-15</i>	1
<i>l(2)41Ah</i>¹⁰	<i>l(2)EMS45-19</i>	1
<i>l(2)41Ah</i>¹¹	<i>l(2)EMS45-21</i>	1
<i>l(2)41Ah</i>¹²	<i>l(2)EMS45-26</i>	1
<i>l(2)41Ah</i>¹³	<i>l(2)EMS45-29</i>	1
<i>l(2)41Ah</i>¹⁴	<i>l(2)EMS45-45</i>	1
<i>l(2)41Ah</i>¹⁵	<i>l(2)EMS45-48</i>	1
<i>l(2)41Ah</i>¹⁶	<i>l(2)EMS45-49</i>	1
<i>l(2)41Ah</i>¹⁷	<i>l(2)EMS45-50</i>	1
<i>l(2)41Ah</i>¹⁸	<i>l(2)EMS45-57</i>	1
<i>l(2)41Ah</i>¹⁹	<i>l(2)EMS45-58</i>	1
<i>l(2)41Ah</i>²⁰	<i>l(2)EMS45-59</i>	1
<i>l(2)41Ah</i>²¹	<i>l(2)EMS45-62</i>	1
<i>l(2)41Ah</i>²²	<i>l(2)EMS45-67</i>	1
<i>l(2)41Ah</i>²³	<i>l(2)EMS45-68</i>	1
<i>l(2)41Ah</i>²⁴	<i>l(2)EMS45-75</i>	1
<i>l(2)41Ah</i>²⁵	<i>l(2)EMS45-78</i>	1
<i>l(2)41Ah</i>²⁶	<i>l(2)EMS45-80</i>	1
<i>l(2)41Ah</i>²⁷	<i>l(2)EMS45-89</i>	1
<i>l(2)41Ah</i>²⁸	<i>l(2)EMS45-90</i>	1
<i>l(2)41Ah</i>²⁸	<i>l(2)EMS45-93</i>	1
<i>l(2)41Ah</i>³⁰	<i>l(2)EMS172</i>	1
<i>l(2)41Ah</i>³¹	<i>l(2)C31</i>	3
<i>l(2)41Ah</i>³²	<i>l(2)C404</i>	3
<i>l(2)41Ah</i>³³	<i>l(2)Sp15</i> ^β	2, 3

^α 1 = Hilliker, 1976, Genetics 83: 765-82; 2 = Spiess, Helling, and Capenos, 1963, Genetics 48: 1377-88; 3 = Tano, 1966, Jpn. J. Genet. 41: 299-308.

^β *l(2)C123* of Tano.

DEFICIENCY MAP OF REGION 41

side	breakpoint	variant
left	41A	<i>Df(2R)A</i> ∼
left	41A	<i>Df(2R)Rsp11</i>
left	41A	<i>Df(2R)Rsp31</i>
	h39	Rsp
left	41A	<i>Df(2R)M41A10</i>
left	41A	<i>Df(2R)A</i>
left	41A	<i>Df(2R)A</i> ∼
left	41A	<i>Df(2R)B</i>
		<i>l(2)41Aa</i>
		<i>l(2)41Ab</i>
right	41A	<i>Df(2R)B</i>
left	41A	<i>Df(2R)Rsp1</i>
		rl
right	41A	<i>Df(2R)A</i>
right	41A	<i>Df(2R)Rsp1</i>
right	41A	<i>Df(2R)Rsp11</i>

side	breakpoint	variant
		uex
right	41A	<i>Df(2R)Rsp31</i>
right	41A	<i>Df(2R)A</i> "
left	41A	<i>Df(2R)M41A4</i>
left	41A	<i>Df(2R)M41A8</i>
		I(2)41Ae
		I(2)41Af
		M(2)41A
right	41A	<i>Df(2R)A</i> '
		I(2)41Ah
right	41A	<i>Df(2R)M41A10</i>
		stw
right	41A	<i>Df(2R)M41A8</i>
		ap
right	41A	<i>Df(2R)M41A4</i>

I(2)49D-F

Seventeen complementation groups defined by fifty-eight recessive alleles uncovered by *Df(2R)vg-B* (Lasko and Pardue, 1988, Genetics 120: 495-502).

locus	genetic location	cytological location	included in	excluded from	synonym
I(2)49Da	2-{66}	49D3-E1	<i>Df(2R)vg-B</i>	<i>Df(2R)vg56</i>	<i>l(2)vr22</i>
I(2)49Db	2-{66}	49D3-E1	<i>Df(2R)vg-B</i>	<i>Df(2R)vg56</i>	<i>l(2)vr1</i>
I(2)49Dc	2-67.1	49D3-E1	<i>Df(2R)vg56</i>	<i>Df(2R)vg135</i>	<i>l(2)C</i>
I(2)49Ea	2-{67}	49E7-F2	<i>Df(2R)vg-D</i>	<i>Df(2R)vg-C</i>	<i>l(2)vr14</i>
I(2)49Fa	2-{68}	49E7-F13	<i>Df(2R)vg56</i>	<i>Df(2R)vg-D</i>	<i>l(2)vr3</i>
I(2)49Fb	2-{68}	49E7-F13	<i>Df(2R)vg56</i>	<i>Df(2R)vg-D</i>	<i>l(2)vr4</i>
I(2)49Fc	2-{68}	49E7-F13	<i>Df(2R)vg56</i>	<i>Df(2R)vg-D</i>	<i>l(2)vr5</i>
I(2)49Fd	2-{68}	49E7-F13	<i>Df(2R)vg56</i>	<i>Df(2R)vg-D</i>	<i>l(2)vr6</i>
I(2)49Ff	2-{68}	49E7-F13	<i>Df(2R)vg56</i>	<i>Df(2R)vg-D</i>	<i>l(2)vr9</i>
I(2)49Fg	2-{68}	49E7-F13	<i>Df(2R)vg56</i>	<i>Df(2R)vg-D</i>	<i>l(2)vr11</i>
I(2)49Fh	2-{68}	49E7-F13	<i>Df(2R)vg56</i>	<i>Df(2R)vg-D</i>	<i>l(2)vr13</i>
I(2)49Fi	2-{68}	49E7-F13	<i>Df(2R)vg56</i>	<i>Df(2R)vg-D</i>	<i>l(2)vr20</i>
I(2)49Fj	2-{68}	49E7-F13	<i>Df(2R)vg106</i>	<i>Df(2R)vg56</i>	<i>l(2)vr19</i>
I(2)49Fk	2-{68}	49E7-F13	<i>Df(2R)vg104</i>	<i>Df(2R)vg106</i>	<i>l(2)vr10</i>
I(2)49Fl	2-{68}	49F13-50A3	<i>Df(2R)vg33</i>	<i>Df(2R)vg104</i>	<i>l(2)vr15</i>
I(2)49Fm	2-{68}	49F13-50A3	<i>Df(2R)vg33</i>	<i>Df(2R)vg104</i>	<i>l(2)vr18</i>
I(2)49Fn	2-67.2	49F13-50A3	<i>Df(2R)vg-B</i>	<i>Df(2R)vg33</i>	cos1
I(2)49Fo	2-{68}	49F13-50A3	<i>Df(2R)vg-B</i>	<i>Df(2R)vg33</i>	<i>l(2)vr16</i>

I(2)49D

allele	origin	discoverer	synonym	ref ^α	comments
I(2)49Da¹	HD	Lasko	<i>l(2)P3</i>	1, 2	dominant enhancer of maternal effect of <i>BicD</i>
I(2)49Db¹	EMS	Lasko	<i>l(2)20</i>	1	
I(2)49Db²	EMS	Lasko	<i>l(2)27</i>	1	
I(2)49Dc¹	spont	Curry, 34a21	<i>l(2)C</i>	3	lethal before pupation
I(2)49Dc²	EMS	Lasko	<i>l(2)C¹⁰</i>	1	
I(2)49Dc³	EMS	Lasko	<i>l(2)C³⁹</i>	1	
I(2)49Dc⁴	EMS	J. Mohler	<i>l(2)C^{1F34}</i>		

^α 1 = Lasko and Pardue, 1988, Genetics 120: 495-502; 2 = Mohler and Wieschaus, 1986, Genetics 112: 803-22; 3 = Morgan, Bridges, and Schultz, 1938, Carnegie Inst. Wash. Year Book 37: 306.

I(2)49Ea

A member of the *Su(z)2* complex (Wu, Jones, Lasko, and Gelbart, 1989, Mol. Gen. Genet. 218: 559-64). Alleles recovered by Lasko and Pardue (1988, Genetics 120: 495-502).

allele	origin	discoverer	synonym	comments
I(2)49Ea¹	EMS	Lasko	<i>l(2)8</i>	recessive semilethal
I(2)49Ea²	EMS	Lasko	<i>l(2)33</i>	recessive semilethal;

allele	origin	discoverer	synonym	comments
I(2)49Ea³	EMS	Lasko	<i>l(2)45</i>	dominant bristle loss
I(2)49Ea⁴	HD	Lasko	<i>l(2)P4</i>	weak dominant bristle loss recessive semilethal; dominant bristle loss

I(2)49F

alleles: Recovery of alleles described by Lasko and Pardue (1988, Genetics 120: 495-502).

allele	origin	discoverer	synonym	comments
I(2)49Fa¹	EMS	Lasko	<i>l(2)2</i>	
I(2)49Fa²	EMS	Lasko	<i>l(2)4</i>	
I(2)49Fa³	EMS	Lasko	<i>l(2)16</i>	
I(2)49Fa⁴	EMS	Lasko	<i>l(2)21</i>	
I(2)49Fa⁵	EMS	Lasko	<i>l(2)25</i>	temperature-sensitive semilethal
I(2)49Fa⁶	EMS	Lasko	<i>l(2)31</i>	
I(2)49Fa⁷	EMS	Lasko	<i>l(2)38</i>	
I(2)49Fa⁸	EMS	Lasko	<i>l(2)54</i>	
I(2)49Fa⁹	EMS	Lasko	<i>l(2)55</i>	
I(2)49Fa¹⁰	EMS	Lasko	<i>l(2)56</i>	
I(2)49Fb¹	EMS	Lasko	<i>l(2)3</i>	
I(2)49Fb²	EMS	Lasko	<i>l(2)28</i>	homozygous viable; lethal in combination with <i>l(2)49Fb¹</i> or a deficiency

I(2)49Fc

phenotype: Embryonic lethal.

alleles: Recovery of alleles described by Lasko and Pardue (1988, Genetics 120: 495-502).

allele	origin	discoverer	synonym
I(2)49Fc¹	EMS	Lasko	<i>l(2)5</i>
I(2)49Fc²	EMS	Lasko	<i>l(2)32</i>
I(2)49Fc³	EMS	Lasko	<i>l(2)40</i>
I(2)49Fc⁴	EMS	Lasko	<i>l(2)48</i>
I(2)49Fc⁵	EMS	Lasko	<i>l(2)57</i>
I(2)49Fd¹	EMS	Lasko	<i>l(2)6</i>
I(2)49Fd²	EMS	Lasko	<i>l(2)7</i>
I(2)49Fd³	EMS	Lasko	<i>l(2)35</i>
I(2)49Fd⁴	γ ray	Lasko	<i>l(2)R4</i>
I(2)49Fd⁵	γ ray	Lasko	<i>l(2)R5</i>
I(2)49Fd⁶	γ ray	Lasko	<i>l(2)R6</i>
I(2)49Fd⁷	γ ray	Lasko	<i>l(2)R8</i>

I(2)49Ff

phenotype: Embryonic lethal.

alleles: Recovery of alleles described by Lasko and Pardue (1988, Genetics 120: 495-502).

allele	origin	discoverer	synonym	comments
I(2)49Ff¹	EMS	Lasko	<i>l(2)11</i>	
I(2)49Ff²	EMS	Lasko	<i>l(2)22</i>	temperature-sensitive lethal
I(2)49Ff³	EMS	Lasko	<i>l(2)23</i>	
I(2)49Ff⁴	EMS	Lasko	<i>l(2)37</i>	
I(2)49Ff⁵	EMS	Lasko	<i>l(2)42</i>	
I(2)49Ff⁶	EMS	Lasko	<i>l(2)43</i>	
I(2)49Ff⁷	EMS	Lasko	<i>l(2)46</i>	
I(2)49Ff⁸	γ ray	Lasko	<i>l(2)R2</i>	
I(2)49Ff⁹	EMS	J. Mohler	<i>l(2)IIIE94</i>	

I(2)49Fg¹	EMS	Lasko	<i>l(2)14</i>
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I(2)49Fh¹	EMS	Lasko	<i>l(2)24</i>	embryonic lethal
I(2)49Fh²	EMS	Lasko	<i>l(2)47</i>	embryonic lethal

I(2)49Fi¹	EMS	Lasko	<i>l(2)53</i>
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I(2)49Fj¹	EMS	Lasko	<i>l(2)41</i>	recessive semilethal
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allele	origin	discoverer	synonym	comments
<i>l(2)49Fk</i>¹	EMS	Lasko	<i>l(2)13</i>	
<i>l(2)49Fk</i>²	EMS	Lasko	<i>l(2)50</i>	
<i>l(2)49Fk</i>³	EMS	Lasko	<i>l(2)51</i>	fails to complement all alleles of <i>l(2)49Fm</i>
<i>l(2)49F1</i>¹	EMS	Lasko	<i>l(2)29</i>	
<i>l(2)49Fm</i>¹	EMS	Lasko	<i>l(2)51</i>	fails to complement all alleles of <i>l(2)49Fm</i>
<i>l(2)49Fm</i>²	EMS	Lasko	<i>l(2)52</i>	
<i>l(2)49Fo</i>¹	EMS	Lasko	<i>l(2)30</i>	recessive semilethal
<i>l(2)49Fo</i>²	EMS	Lasko	<i>l(2)44</i>	
<i>l(2)49Fo</i>³	HD	Lasko	<i>l(2)P1</i>	

DEFICIENCY MAP OF REGION 49D-F

side	breakpoint	variant
left	49D3-5	<i>Df(2R)vg-B</i> <i>l(2)49Da</i> <i>l(2)49Db</i>
left	49D	<i>Df(2R)vg56</i>
left		<i>Df(2R)vg136</i> vg <i>l(2)49Dc</i>
left		<i>Df(2R)R9</i>
right		<i>Df(2R)vg133</i>
right	49D6-E1	<i>Df(2R)vg135</i>
right	49E7-F1	<i>Df(2R)vg-C</i> <i>l(2)49Ea</i> Psc <i>Su(z)2</i>
right	49E1-F2	<i>Df(2R)vg62</i>
right	49E2	<i>Df(2R)vg79a</i>
right	49E2	<i>Df(2R)vg79b3</i>
right		<i>Df(2R)vg79d8</i>
right		<i>Df(2R)vg81</i>
right	49E7-F1	<i>Df(2R)vg83f15</i>
right		<i>Df(2R)vg107</i>
right		<i>Df(2R)vg120</i>
right		<i>Df(2R)vg124</i>
right		<i>Df(2R)vg136</i>
right	49E2-7	<i>Df(2R)vg-D</i>
right		<i>Df(2R)vg-R7</i>
right		<i>Df(2R)vg-R9</i> <i>l(2)49Fa</i> <i>l(2)49Fb</i> <i>l(2)49Fc</i> <i>l(2)49Fd</i> <i>l(2)49Ff</i> <i>l(2)49Fg</i> <i>l(2)49Fh</i> <i>l(2)49Fi</i>
right	49F	<i>Df(2R)vg56</i> <i>l(2)49Fj</i>
right		<i>Df(2R)vg106</i> <i>l(2)49Fk</i> Mp20
right	49F9-13	<i>Df(2R)vg104</i> <i>l(2)49F1</i> <i>l(2)49Fm</i>
right		<i>Df(2R)P2</i>
right	50A	<i>Df(2R)vg33</i> cos1 <i>l(2)49Fo</i>
right	50A2-3	<i>Df(2R)vg-B</i>

l(2)52

Nine lethally mutable complementation groups identified in the region uncovered by *Df(2R)WMG* = *Df(2R)52A2-B4;52D7-E1* and one in the region uncovered by *Df(2R)KL32* = *Df(2R)52C5-D1;52E2-5*,

but not by *Df(2R)WMG*. Two additional complementation groups inferred from the lethality of overlapping deficiencies. Eight of the nine uncovered by *Df(2R)WMG* were also uncovered by *Df(2R)XTE18* = *Df(2R)51E3-4;52C9-D1*, and were not further studied (Davis and MacIntyre, 1988, Genetics 120: 755-66).

l(2)52ACa-l(2)52ACh

Eight complementation groups identified but not further characterized.

l(2)52Da

location: 2-{75}.

origin: Induced by ethyl methanesulfonate.

synonym: Group 1.

alleles: Nine.

cytology: Placed in 52D3 based on its inclusion in *Df(2R)KL9* = *Df(2R)52D3;53D7-9* but not *Df(2R)KL69* = *Df(2R)52D2-3;52D9-E1*.

l(2)52Db

location: 2-{75}.

alleles: None recovered.

cytology: Inferred to be in 52D7-9 based on the inviability of the heterozygote between *Df(2R)KL9* = *Df(2R)52D3;-53D7-9* and *Df(2R)KL69* = *Df(2R)52D3;52D9-E1* but not of that between *Df(2R)KL9* and *Df(2R)WMG* = *Df(2R)52A4-B2;53D7-E1*.

l(2)52Dc

location: 2-{75}.

alleles: None recovered.

cytology: Inferred to be in 52D6-E1 based on the inviability of the heterozygote between *Df(2R)l30* = *Df(2R)52D6-7;52E1* and *Df(2R)KL69* = *Df(2R)52D3;-52D9-E1*.

l(2)52Ea

location: 2-{75}.

origin: Induced by ethyl methanesulfonate.

alleles: Two.

cytology: Placed in 52D9-E5 based on its inclusion in *Df(2R)KL32* = *Df(2R)52C5-D1;52E2-5* but not *Df(2R)KL69* = *Df(2R)52D3;52D9-E1*.

DEFICIENCY MAP OF REGION 52

side	breakpoint	variant
left	50D	<i>Df(2R)L7</i>
left	50F6-51A1	<i>Df(2R)L48</i>
left	51A2	<i>Df(2R)L4</i>
right	51A12-B1	<i>Df(2R)L4</i>
left	51B3-4	<i>Df(2R)sf</i>
right	51B4-7	<i>Df(2R)L48</i>
right	51B5-C2	<i>Df(2R)L7</i>
right	51C7-E2	<i>Df(2R)sf</i>
left	51E3-4	<i>Df(2R)XTE11</i>
left	52B2-C1	<i>Df(2R)KL99</i>
left	51E3-4	<i>Df(2R)XTE18</i>
left	52A4-B2	<i>Df(2R)WMG</i>
right	52A6-10	<i>Df(2R)XTE11</i> <i>l(2)52ACa</i> <i>l(2)52ACb</i> <i>l(2)52ACc</i> <i>l(2)52ACd</i> <i>l(2)52ACe</i> <i>l(2)52ACf</i> <i>l(2)52ACg</i> <i>l(2)52ACh</i>
left	52C5-D1	<i>Df(2R)KL32</i>

side	breakpoint	variant
right	52C9-D1	<i>Df(2R)XTE18</i> α Gpo
left	52D3	<i>Df(2R)KL9</i> l(2)52Da
left	52D2-3	<i>Df(2R)KL69</i>
right	52D7-E1	<i>Df(2R)WVG</i> c l(2)52Db α
right	52D7-9	<i>Df(2R)KL9</i>
left	52D6-7	<i>Df(2R)l30</i> l(2)52Dc α
right	52D9-E1	<i>Df(2R)KL69</i> l(2)52Ea
right	52E2-5	<i>Df(2R)KL32</i>
right	52E2-5	<i>Df(2R)KL99</i>
right	52E1	<i>Df(2R)l30</i>

α Loci inferred from lethality of overlapping deficiencies.

l(2)54

location: 2-41 (not separated from *J*: 2-41.0).

origin: Induced by ethyl methanesulfonate.

discoverer: Sandler, 1976.

references: 1977, *Genetics* 86: 567-82.

phenotype: Recessive lethal.

cytology: Located to 31B-F based on its inclusion in *Df(2L)J-der-27 = Df(2L)31B-D;31F*.

other information: Not allelic to *da* or *mfs(2)48*.

l(2)55i

location: 2-55.0 (probably to the left of the centromere).

origin: Spontaneous.

discoverer: Burdick, 55i.

references: 1956, *DIS* 30: 69.

Mukai and Burdick, 1959, *Genetics* 44: 211-32.

1960, *Genetics* 45: 1581-93.

Schnick, Mukai, and Burdick, 1960, *Genetics* 45: 315-29.

Mukai and Burdick, 1961, *Japan J. Genetics* 36: 97-104.

phenotype: Larvae hatch but die before pupation. Females heterozygous for *l(1)55i* have higher fecundity than homozygous, wild-type females. The lethal is therefore not eliminated from laboratory populations. RK3.

l(2)56a

location: 2-90.

origin: Spontaneous.

discoverer: Burdick, 56a.

references: 1956, *DIS* 30: 69.

phenotype: Homozygous lethal; heterozygote shows normal viability. RK3.

other information: Crossing over normal.

l(2)56F

A series of mostly recessive lethals recovered based on their lethality in combination with *Df(2R)l73* (cytology normal) and *Df(2R)017 = Df(2R)56F5;56F15* [Shellenbarger and Dutttagupta, 1978, *Mutat. Res.* 52: 395-407; Dutttagupta and Shellenbarger, 1980, *Development and Neurobiology of Drosophila* (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York and London, pp. 25-33]. Four complementation groups within *Df(2R)017* but not *Df(2R)l73* (not further described) plus five sites uncovered by both deficiencies, which are separable by complementation analysis, but arbitrarily considered to represent three gene loci, two of which have complementing alleles, and the third of which interacts in *trans*

with the other two. Except for the fact that *l(2)56Fa* maps to the left of *M(2)56F* and the inference that *l(2)56Fb* lies between them, nothing known of order of loci.

locus	genetic location		cytological location		included in	excluded from	synonym
	location	location	location	location			
l(2)56Fa	2-92.3	56F5-15	<i>Df(2R)l73</i>				
l(2)56Fb	2-92.3	56F5-15	<i>Df(2R)l73</i>				
<i>l(2)56Fc</i>	2-92.3	56F5-15	<i>Df(2R)l73</i>				M(2)56F
l(2)56Fd	2-92.3	56F5-15	<i>Df(2R)017</i>	<i>Df(2R)l73</i>			
l(2)56Fe	2-92.3	56F5-15	<i>Df(2R)017</i>	<i>Df(2R)l73</i>			
l(2)56Ff	2-92.3	56F5-15	<i>Df(2R)017</i>	<i>Df(2R)l73</i>			
l(2)56Fg	2-92.3	56F5-15	<i>Df(2R)017</i>	<i>Df(2R)l73</i>			

l(2)56Fa

phenotype: Recessive lethal; no phenotype in heterozygote, except when heterozygous to *l(2)56Fb* in which case short thin bristles are observed.

alleles: Nine alleles, two complementing and seven non-complementing. Shown to map to the left of *M(2)56F* by recombination.

allele	origin	synonym	comments
l(2)56Fa ¹	EMS	<i>l(2)36</i>	noncomplementing
l(2)56Fa ²	EMS	<i>l(2)157</i>	noncomplementing
l(2)56Fa ³	EMS	<i>l(2)775</i>	noncomplementing
l(2)56Fa ⁴	EMS	<i>l(2)1991</i>	noncomplementing
l(2)56Fa ⁵	EMS	<i>l(2)2735</i>	complementing
l(2)56Fa ⁶	EMS	<i>l(2)D91</i>	noncomplementing
l(2)56Fa ⁷	EMS	<i>l(2)D292</i>	noncomplementing
l(2)56Fa ⁸	EMS	<i>l(2)D932</i>	noncomplementing
l(2)56Fa ⁹	EMS	<i>l(2)D1368</i>	complementing

l(2)56Fb

location: Position between *l(2)56Fa* and *M(2)56F* provisional; based on interactions with mutants at both loci.

synonym: *l(2)l2*.

phenotype: A single allele, which is lethal in homozygotes but produces an extreme Minute, semilethal phenotype in trans heterozygotes with *Df(2R)017* and *Df(2R)l73*. Complements all other *l(2)56F* mutants for viability; interacts with the the noncomplementing alleles of *l(2)56Fa* and *M(2)56F* to produce a Minute-like phenotype in the case of *l(2)56Fa* and an enhanced Minute phenotype in combination with *M(2)56F*. Trans heterozygotes of *l(2)56Fb* with the complementing alleles of the other two loci are wild type.

*l(2)57

origin: Spontaneous.

discoverer: Paik.

references: 1960, *Evolution* 14: 293-303.

other information: A series of 11 lethals selected from Korean wild populations.

l(2)57

Two lethal-saturation studies carried out in the region; tests against *Df(2R)D17 = Df(2R)57B5;58B1-2* or against smaller deficiencies including *Pu* and *tud* in 57C were carried out by O'Donnell, Boswell, Reynolds, and Mackay, (1989, *Genetics* 121: 273-80); they recovered 76 lethal mutations identifying sixteen complementation groups between 57B17 and 57F6; in addition, there were fourteen mutations outside this region, but still uncovered by *Df(2R)D17*. The second study by Schejter and Shilo (1989, *Cell* 56: 1093-1104) identified eight complemen-

tation groups among sixteen lethal mutations uncovered by *Df(2R)PK1 = Df(2R)57C5,57F6*, the region containing *Egfr*. Few complementation crosses were carried out between mutations from the two screens.

1(2)57B

locus	genetic location	cytological location	included in	excluded from
1(2)58Ba	2-[97]	57B16-20	<i>Df(2R)PC18</i>	<i>Df(2R)PL3</i>
1(2)58Bb	2-[97]	57B16-20	<i>Df(2R)PC18</i>	<i>Df(2R)PL3</i>
1(2)58Bc	2-[97]	57B16-20	<i>Df(2R)PC18</i>	<i>Df(2R)PL3</i>
1(2)58Bd	2-[97]	57B16-20	<i>Df(2R)PC18</i>	<i>Df(2R)PL3</i>

allele	origin	discoverer	synonym
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1(2)57Ba¹	EMS	O'Donnell	<i>1(2)JE7</i>
1(2)57Bb¹	EMS	O'Donnell	<i>1(2)JE6</i>
1(2)57Bc¹	EMS	Mackay	<i>1(2)WE188</i>
1(2)57Bd¹	ENU	Boswell	<i>1(2)E14B</i>
1(2)57Bd²	EMS	Boswell	<i>1(2)SHB1</i>

locus	genetic location	cytological location	included in	excluded from	synonym
1(2)57Ca	2-[97]	57B18-C4	<i>Df(2R)PL3</i>	<i>Df(2R)MP1</i>	
1(2)57Cb	2-[97]	57C3-4	<i>Df(2R)MP1</i>	<i>Df(2R)K11</i>	
1(2)57Cc	2-[97]	57C5-7	<i>Df(2R)PC18</i>	<i>Df(2R)PF1</i>	
1(2)57Cd	2-[97]	57C5-7	<i>Df(2R)PC18</i>	<i>Df(2R)PF1</i>	
1(2)57Ce	2-[97]	57C6-D1	<i>Df(2R)PF1</i>	<i>Df(2R)PC18</i>	
1(2)57Cf	2-97	57C4-6	<i>Df(2R)PI12</i>	<i>Df(2R)CC2</i>	Pu

1(2)57C

allele	origin	discoverer	synonym
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1(2)57Ca¹	ENU	Boswell	<i>1(2)E19A</i>
1(2)57Ca²	EMS	Boswell	<i>1(2)SHL1</i>
1(2)57Ca³	EMS	Reynolds	<i>1(2)RE2</i>
1(2)57Ca⁴	EMS	Reynolds	<i>1(2)RE7</i>
1(2)57Ca⁵	EMS	Mackay	<i>1(2)WE7</i>
1(2)57Cb¹	EMS	Mackay	<i>1(2)WESA</i>
1(2)57Cb²	EMS	Reynolds	<i>1(2)RE6</i>
1(2)57Cc¹	ENU	Boswell	<i>1(2)E12A</i>
1(2)57Cc²	EMS	Mackay	<i>1(2)WE12</i>
1(2)57Cc³	EMS	Mackay	<i>1(2)WE9</i>
1(2)57Cc⁴	EMS	Reynolds	<i>1(2)RE4</i>
1(2)57Cc⁵	EMS	Reynolds	<i>1(2)RE3</i>
1(2)57Cc⁶	EMS	Reynolds	<i>1(2)RE8</i>
1(2)57Cc⁷	EMS	Reynolds	<i>1(2)RE11</i>
1(2)57Cc⁸	EMS	Boswell	<i>1(2)K8-1</i>
1(2)57Cc⁹			<i>1(2)RE10</i>
1(2)57Cd¹	EMS	Boswell	<i>1(2)K1b2</i>
1(2)57Cd²	EMS	Boswell	<i>1(2)FAMII-14</i>
1(2)57Ce¹	EMS	Mackay	<i>1(2)WE8</i>
1(2)57Ce²	EMS	Mackay	<i>1(2)WE10</i>
1(2)57Ce³	EMS	Mackay	<i>1(2)WE5</i>
1(2)57Ce⁴	EMS	Mackay	<i>1(2)WE2</i>
1(2)57Ce⁵	EMS	Reynolds	<i>1(2)RE1</i>
1(2)57Ce⁶	EMS	Reynolds	<i>1(2)RES</i>
1(2)57Ce⁷	EMS	Boswell	<i>1(2)FAMII-12</i>
1(2)57Ce⁸	EMS	Boswell	<i>1(2)K10b2</i>

1(2)57CD

Three complementation groups were identified by Schejter and Shilo in 57C5-D9, the region of overlap between *Df(2R)PK1* and *Df(2R)PI12*; O'Donnell *et al.* identified five complementation groups in the same region, and no complementation tests have been carried out. Until they are, these mutations are considered independently of those of O'Donnell *et al.*

locus	genetic location	cytological location	included in
1(2)57CDa	2-[97]	57C5-D9	<i>Df(2R)PK1</i> <i>Df(2R)PI12</i>
1(2)57CDb	2-[97]	57C5-D9	<i>Df(2R)PK1</i> <i>Df(2R)PI12</i>
1(2)57CDc	2-[97]	57C5-D9	<i>Df(2R)PK1</i> <i>Df(2R)PI12</i>

allele	discoverer	synonym
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1(2)57CDa¹	Schejter	<i>1(2)ES16</i>
1(2)57CDa²	Schejter	<i>1(2)ES27</i>
1(2)57CDa³	Schejter	<i>1(2)ES43</i>
1(2)57CDa⁴	Schejter	<i>1(2)ES46</i>
1(2)57CDb¹	Schejter	<i>1(2)ES20</i>
1(2)57CDc¹	Schejter	<i>1(2)ES26</i>

1(2)57D

Two sets of three complementation groups found in this region; they have not been cross checked for complementation; two of the three isolated by O'Donnell *et al.* are within *Df(2R)PI12*, whereas those isolated by Schejter *et al.* are not; the latter could be in 57E as well. Further complementation and deficiency analyses are required to determine the actual number of loci identified.

locus	genetic location	cytological location	included in	excluded from
1(2)57Da	2-[98]	57D6-8	<i>Df(2R)K11</i>	<i>Df(2R)MP1</i>
1(2)57Db	2-[98]	57D7-9	<i>Df(2R)PI12</i>	<i>Df(2R)K11</i>
1(2)57Dc	2-[98]	57D8-12	<i>Df(2R)AA21</i>	<i>Df(2R)PI12</i>
1(2)57Dd	2-[99]	57D8-E		<i>Df(2R)BP7</i>
1(2)57De	2-[99]	57D8-E		<i>Df(2R)PI12</i>
1(2)57Df	2-[99]	57D8-E		<i>Df(2R)BP7</i>

allele	origin	discoverer	synonym	ref ^α	comments
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1(2)57Da¹	EMS	Mackay	<i>1(2)WE76</i>	1	
1(2)57Da²	EMS	Reynolds	<i>1(2)RE12</i>	1	
1(2)57Db¹	EMS	Boswell	<i>1(2)SH1</i>	1	
1(2)57Db²	EMS	Boswell	<i>1(2)SH10</i>	1	
1(2)57Db³	EMS	Boswell	<i>1(2)SH12</i>	1	
1(2)57Db⁴	EMS	O'Donnell	<i>1(2)JE4</i>	1	
1(2)57Db⁵	EMS	O'Donnell	<i>1(2)JE9</i>	1	
1(2)57Db⁶	EMS	Boswell	<i>1(2)SH6B</i>	1	
1(2)57Db⁷			<i>1(2)WEC</i>	1	
1(2)57Dc¹	EMS	O'Donnell	<i>1(2)JE8</i>	1, 2	<i>1(2)57Ed</i> of Schejter and Shilo
1(2)57Dd¹		Schejter	<i>1(2)ES1</i>	2	
1(2)57Dd²		Schejter	<i>1(2)ES4</i>	2	
1(2)57Dd³		Schejter	<i>1(2)ES38</i>	2	
1(2)57De¹		Schejter	<i>1(2)ES2</i>	2	

allele	origin	discoverer	synonym	ref ^α	comments
I(2)57D1¹		Schejter	<i>I(2)ES15</i>	2	
^α	1 = O'Donnell, Boswell, Reynolds, and Mackay, 1989, Genetics 121: 273-80; 2 = Schejter and Shilo, 1989, Cell 56: 1093-1104.				

I(2)57E

Three complementation groups were localized between 57D11 and 57F6 by O'Donnell *et al.*; two of these, including *Egfr*, were further localized to 57E by Price *et al.* Schejter *et al.* localized *I(2)57Dc* of O'Donnell to 57E-F, whereas O'Donnell placed it to the left of 57D11-12.

locus	genetic location		cytological location		included in	excluded from	synonym
	location	location	location	location			
<i>I(2)57Ea</i>	2-100	57E4-F1	<i>Df(2R)Egfr18</i>				Egfr , <i>I(2)57DEFa</i>
I(2)57Eb	2-[99]	57E1-11	<i>Df(2R)Egfr3</i>	<i>Df(2R)Egfr18</i>	<i>I(2)57DEFb</i>		
I(2)57Ec	2-[99]	57D11-F6	<i>Df(2R)PK1</i>	<i>Df(2R)AA21</i>	<i>I(2)57DEFc</i>		
I(2)57Ed	2-[99]	57E-F	<i>Df(2R)BP7</i>	<i>Df(2R)PK1</i>			

allele	origin	discoverer	synonym	ref ^α	comments
I(2)57Eb¹	EMS	Boswell	<i>I(2)SH7</i>	1, 2, 3	
I(2)57Eb²		Schejter	<i>I(2)ES37</i>	3	
I(2)57Ec¹	EMS	Boswell	<i>I(2)SH14</i>	1	
I(2)57Ed¹	EMS	O'Donnell	<i>I(2)JE8</i>	1, 3	<i>I(2)57Dc¹</i> according to O'Donnell <i>et al.</i>
^α	1 = O'Donnell, Boswell, Reynolds, and Mackay, 1989, Genetics 121: 273-80; 2 = Price, Clifford, and Schüpbach, 1989, Cell 56: 1085-92; 3 = Schejter and Shilo, 1989, Cell 56: 1093-1104.				

DEFICIENCY MAP OF REGION 57

side	breakpoint	variant ^α	variant ^β	variant ^γ
left	56F9-17	<i>Df(2R)AA21</i>		
left	57B5	<i>Df(2R)D17</i>		<i>Df(2R)D17</i>
left	57B16-17	<i>Df(2R)PC18</i>		
left	57B16-17	<i>Df(2R)F36</i>		
		I(2)57Ba		
		I(2)57Bb		
		I(2)57Bc		
		I(2)57Bd		
left	57B18-20	<i>Df(2R)PL3</i>		<i>Df(2R)PL3</i>
		I(2)57Ca		
left	57C3	<i>Df(2R)CC2</i>		
left	57C3-4	<i>Df(2R)MP1</i>		
		I(2)57Cb		
left	57C3-4	<i>Df(2R)K11</i>		
left	57C4	<i>Df(2R)P112</i>	<i>Df(2R)P112</i>	<i>Df(2R)P112</i>
left		Pu		
left	57C5	<i>Df(2R)PK1</i>	<i>Df(2R)PK1</i>	<i>Df(2R)PK1</i>
left	57C5	<i>Df(2R)PF1</i>		
right		Pu		
		I(2)57Cc		
		I(2)57Cd		
right	57C6	<i>Df(2R)CC2</i>		
right	57C6-7	<i>Df(2R)PC18</i>		
right	57C6-7	<i>Df(2R)F36</i>		
		I(2)57Ce		
		tud		
		I(2)57Cda		
		I(2)57Cdb		
		I(2)57Cdc		
right	57D1	<i>Df(2R)PF1</i>		
left	57D2-8			<i>Df(2R)Egfr5</i>
right	57D6-7	<i>Df(2R)MP1</i>		
		I(2)57Da		
right	57D7-8	<i>Df(2R)K11</i>		
		I(2)57Db		

side	breakpoint	variant ^α	variant ^β	variant ^γ
right	57D8-9	<i>Df(2R)PL3</i>		<i>Df(2R)PL3</i>
right	57D8-9	<i>Df(2R)P112</i>	<i>Df(2R)P112</i>	<i>Df(2R)P112</i>
		I(2)57Dc		
			I(2)57Dd	
			I(2)57De	
			I(2)57Df	
right	57D11-12	<i>Df(2R)AA21</i>		
left	57E		<i>Df(2R)BP7</i>	
left	57E1			<i>Df(2R)Egfr3</i>
	57F		<i>T(Y:2)NL11</i>	
		I(2)57Eb	I(2)57Eb	I(2)57Eb
			mat(2)N	<i>Df(2R)Egfr18</i>
left	57E4-11	Egfr	Egfr	Egfr
		I(2)57Ec	I(2)57Ed	
right	57F1			<i>Df(2R)Egfr18</i>
right	57F5-6	<i>Df(2R)PK1</i>	<i>Df(2R)PK1</i>	<i>Df(2R)PK1</i>
right	57F11			<i>Df(2R)Egfr3</i>
right	58A1		<i>Df(2R)BP7</i>	
right	58B1-2	<i>Df(2R)D17</i>		<i>Df(2R)D17</i>
right	58D1			<i>Df(2R)Egfr5</i>

^α O'Donnell, Boswell, Reynolds, and Mackay, 1989, Genetics 121: 273-80.
^β Schejter and Shilo, 1989, Cell 56: 1093-1104.
^γ Price, Clifford, and Schüpbach, 1989, Cell 56: 1085-92.

I(2)64

location: 2-70.

origin: X ray induced.

references: Ytterborn, 1967, Hereditas 58: 165-90.

phenotype: Homozygous lethal. Allele frequency maintained in experimental populations in higher-than-expected frequencies; this "overdominance" effect lost in later generations (Ytterborn, 1968, Hereditas 60: 33-71).

cytology: Polytene configuration normal.

I(2)74I

location: 2R.

origin: Spontaneous.

references: Wright, Hodgetts, and Sherald, 1976, Genetics 84: 267-85.

I(2)85

location: 2-54.8.

origin: X ray induced.

references: Ytterborn, 1967, Hereditas 58: 165-90.

phenotype: Homozygous lethal; eliminated from experimental populations less rapidly than expected.

cytology: No aberrations observed.

***I(2)1076**

location: 2-15 (about 40 units from *Bl*).

origin: Spontaneous.

discoverer: Ives, 49h.

references: 1951, DIS 25: 70.

phenotype: Lethal homozygous and in combination with *In(2L)Cy*. RK3.

***I(2)1323**

location: 2-55 (0/162 crossovers with *Bl*).

origin: Spontaneous.

discoverer: Ives, 51g.

references: 1951, DIS 25: 70.

phenotype: Lethal homozygous and in combination with *In(2L)Cy* + *In(2R)Cy*. RK3.

l(2)1542

location: 2-unmapped.
origin: Induced by ethyl methanesulfonate.
references: Gateff, 1978, *Biol. Rev.* 53: 123-68.
 1978, *Science*, 200: 1448-59.
phenotype: Causes malignant neuroblastoma of the adult neuroblasts and ganglion mother cells; also displays intermediate imaginal disc neoplasm with compact mode of growth.

l(2)a

location: 2-64.7.
origin: Spontaneous.
discoverer: Bridges, 16a15.
references: Bridges and Morgan, 1919, *Carnegie Inst. Washington Publ. No. 278*: 286, 302.
phenotype: Almost completely lethal; body color of rare survivor pale. RK3.

l(2)ax

location: 2-106.9.
origin: Spontaneous.
discoverer: Bridges, 19b28.
references: 1937, *Cytologia (Tokyo)*, Fujii Jub., Vol. 2: 745-55.
phenotype: Lethal in very early larval stage. RK3.
cytology: Located in 60B on salivary chromosome by Bridges but not included in *Df(2R)Px = Df(2R)60B8-10;60D1-2*.

l(2)ay

location: 2-8.3.
origin: Spontaneous.
discoverer: Bridges, 30d5.

l(2)B

location: 2- [in 2L of *In(2L)t*].
discoverer: Bridges, 1930.

l(2)bl: lethal (2) bluter

location: 2-43.8.
origin: X ray induced.
discoverer: Käfer, 50b.
references: Benz, 1953, *DIS* 27: 55.
 1957, *Z. Indukt. Abstamm. Vererbungsl.* 88: 78-114 (fig.).
phenotype: Lethal at end of pupal stage. Homozygotes make emerging movements, but puparia have abnormally thick protein layer so that imaginal hypodermis is punctured in attempt to eclose. Hemolymph is lost, and flies die. Apparently-normal homozygotes may be obtained by artificially opening puparium. Occasionally, a fly spontaneously escapes puparium without serious injury. Differences in content of free amino acids and peptides between *l(2)bl* and wild type can be distinguished in third-instar larvae, prepupae, and early pupae. RK3.

l(2)Bld: see *l(2)36Da*

l(2)bw: lethal (2) with brown

location: 2-104.
origin: Spontaneous in *bw^{2b}mr* chromosome.
discoverer: Curry, 36i.
cytology: Salivary chromosomes seem to show slight deficiency or disturbance in 59C and D (Bridges).

l(2)C: see *l(2)49Dc*

l(2)CA6

location: 2-distal to *Df(2L)64j*.
synonym: *l(1)br6*.
references: Woodruff and Ashburner, 1979, *Genetics* 92: 133-49.
alleles: Three ethyl-methanesulfonate-induced alleles (*SF3*, *SF24*, *SF27*).

l(2)cg: lethal (2) with comb gap

location: 2-15 (between *dp* and *cl*).
origin: Spontaneous.
discoverer: Nichols-Skoog, 33d19.
references: Curry, 1939, *DIS* 12: 46.

l(2) cn bw^{co-3a}: see *l(2)S3a*

l(2) cn bw^{co-7}: see *l(2)S7*

l(2)cos: see *cos*

l(2)crc: see *crc*

l(2)DTS

origin: Induced with ethyl methanesulfonate; selected as dominant temperature-sensitive lethal.
references: Suzuki and Procnier, 1969, *Proc. Nat. Acad. Sci. USA* 62: 369-76.
phenotype: Homozygotes lethal at all temperatures; *l(2)DTS/+* flies lethal when raised at 29°, but survive development at 25°. *l(2)DTS/++* heterozygotes survive development at 29°.

locus	genetic location	T.S.P. ^α	lethal phase	synonym
l(2)DTS1	just to left of <i>px</i>	115-120 hr	115-144 hr	<i>DTS-L1, DTS-L15</i>
l(2)DTS6	<i>b-pr</i>	144-192 hr	190-210 hr	<i>DTS-L6</i>
l(2)DTS8	synthetic			<i>DTS-L8</i>
l(2)DTS9	FS ^β			<i>DTS-L9</i>
l(2)DTS18	FS			<i>DTS-L18</i>
l(2)DTS19	<i>pr-c</i>	115-120hr	>210 hr; eclosion incomplete	<i>DTS-L19</i>
l(2)DTS20^γ	FS		late pupal stage	<i>DTS-L20</i>

^α T.S.P. = temperature sensitive period of heterozygote.

^β FS = dominant female sterile and therefore not genetically localizable.

^γ Heterozygotes raised at 25° show abnormal chitin development on dorsal surface of abdomen.

locus ^α	chromosome	ref ^β
l(2)DTS18	<i>In(2LR)bw^{VI}</i>	1
l(2)DTS100	<i>Cy0</i>	1
l(2)DTS486	<i>Cy0</i>	1
l(2)DTS513	<i>Cy0</i>	1

^α Not tested for allelism with *l(2)25Ca*.

^β 1 = Falke and Wright, 1972, *DIS* 48: 89-91.

other information: Many dominant temperature-sensitive lethal mutations on chromosome 2 are allelic and are described under *l(2)25Ca*.

l(2)ff10

location: 2R.
origin: Induced by ethyl methanesulfonate.
discoverer: Ransom.
references: Campos-Ortega, 1980, *Current Topics in Developmental Biology*, (R.K. Hunt, ed.). Academic Press, New York, Vol. 15, pp. 347-71.

phenotype: Recessive lethal; in homozygous mutant clones in mosaic eyes, cone cells are absent; thus, lenses of eye facets do not form and their place is occupied by cuticle with bristles; other constituents of ommatidia are present, though abnormal.

l(2)G: lethal (2) of Golubovsky

A group of mapped lethals isolated from natural populations in the vicinity of Unmanj, the Ukraine.

references: Golubovsky, 1971, *Genetika* 7: 77.

locus	location
<i>l(2)G78</i>	2-9.4
<i>l(2)G80</i>	2-54.8
<i>l(2)G84</i>	2-73.4
<i>l(2)G93</i>	2-53.9
<i>l(2)G94</i>	2-53.9
<i>l(2)G97</i>	2-73.1
<i>l(2)G99</i>	2-9.3
<i>l(2)G121</i>	2-76.2
<i>l(2)G129</i>	2-0.0
<i>l(2)G137</i>	2-51.9
<i>l(2)G145</i>	2-51.8
<i>l(2)G151</i>	2-54.0
<i>l(2)G156</i>	2-78.6
<i>l(2)G305</i>	2-73.9
<i>l(2)G341</i>	2-103.5
<i>l(2)G552</i>	2-52.2
<i>l(2)G559</i>	2-32.8
<i>l(2)G560</i>	2-81.8
<i>l(2)G562</i>	2-24.2
<i>l(2)G563</i>	2-26.7
<i>l(2)G566</i>	2-54.3
<i>l(2)G571</i>	2-11.3
<i>l(2)G572</i>	2-18.8
<i>l(2)G583</i>	2-49.0

l(2)gd1: lethal (2) giant discs

location: 2-47.7.

origin: Spontaneous.

references: Bryant, 1969, *DIS* 44: 47.

Bryant and Schubiger, 1971, *Dev. Biol.* 24: 233-63.

phenotype: Pupariation delayed, up to nine days in crowded cultures; pupariation is but one day in uncrowded cultures. Discs continue to grow, becoming very large with long delays. Mutant discs transplanted into normal larval hosts metamorphose; cuticular patterns normal but bristles and to a greater degree sensillae and bracts missing; unguis missing from tarsal claws; tarsi two jointed. Mutant discs yield low incidence of trans-determination.

cytology: Chromosomes normal (Ashburner).

l(2)gd2

location: 2-10.4 (Jürgens).

origin: Induced by ethyl methanesulfonate.

synonym: *l(2)gd-l*.

references: Gateff, 1977, *DIS* 52: 4.

phenotype: Lethal shortly after pupation; imaginal discs two to four times normal size. Usually display abnormal shapes and folding patterns; occasionally appear normal. Metamorphosed fragments in normal larval hosts exhibit hair patterns; occasional single bristles seen; often only naked cuticle forms.

l(2)gl: lethal (2) giant larvae

location: 2-0.0.

synonym: *lgl*.

references: Hadorn, 1937, *Proc. Soc. Exp. Biol. Med.*

36: 632-34.

1937, *Proc. Nat. Acad. Sci. USA* 23: 478-84.

1938, *Rev. Suisse Zool.* 45: 425-29.

Vogt, 1947, *Z. Naturforsch* 26: 292-94.

Gateff and Schneiderman, 1974, *Wilhelm Roux's Arch. Dev. Biol.* 176: 23-65.

Gateff, 1978, *Biol. Rev. Cambridge Phil. Soc.* 53: 123-68.

Mechler, McGinnis, and Gehring, 1985, *EMBO J.* 4: 1551-57.

phenotype: Homozygotes undergo embryogenesis and the first three larval instars; larvae reach normal maximum size; then for some alleles most homozygotes fail to pupate, becoming bloated and 1.5-2 times normal size, whereas for others the majority form prepupae but fail to progress into morphogenesis. Ring gland small and appears immature in third-instar larvae (Scharrer and Hadorn, 1938, *Proc. Nat. Acad. Sci. USA* 24: 236-42); third-instar *l(2)gl* larvae implanted with a normal ring gland pupate but do not metamorphose; injection of ecdysone elicits the same result (Karlson and Hanser, 1952, *Z. Naturforsch* 76: 80-83); thus a deficiency of hormones from the ring gland is probably one, but not the only, result of *l(2)gl*. Homozygotes that die as prepupae have underdeveloped corpora allata and prothoracic glands, whereas larval lethals have underdeveloped prothoracic glands but normal corpora allata (Korochkina and Nazarova, 1977, *Chromosoma* 62: 175-90). Prothoracic glands contain approximately 1% the normal quantity of smooth endoplasmic reticulum (Aggarwal and King, 1969, *J. Morph.* 129: 171-99). Alleles range from 98% larval and 2% pupal death to 18% larval and 82% pupal death (Gateff, Golubovsky, and Sokolova, 1977, *DIS* 52: 128-29). In the most extreme phenotypes (+++), the larval brain and optic lobes become enlarged and disorganized and the imaginal discs large and clumped; when discs of such larvae are transplanted into wild-type-female abdomens, they form large contained tumors, whereas transplanted optic primordia from larval brains form invasive neuroblastomas, which grow rapidly, killing the host within 7-14 days; they can be serially cultured in adult abdomens (Gateff and Schneiderman, 1974). These observations have led to *l(2)gl*'s being designated a *Drosophila* oncogene. Intermediate alleles (++) exhibit moderately enlarged brain and discs, which show enhanced growth when transplanted into wild-type females and death of host is delayed. In weak alleles (+) the brain and discs are small and rudimentary and grow slowly in transplants. One allele normal in disc morphology and behavior in transplants (-). The lethal phase is not well correlated with the phenotypic expression.

Most abundant transcription noted in early (0-6 h) embryos and late third-instar larvae, with the smaller transcript more abundant in embryos and the larger in larvae (Mechler, McGinnis, and Gehring). Immunocytochemistry shows localization of *l(2)gl* product at cell surfaces, specifically at the interfaces between proliferating cells (Klämbt and Schmidt, 1986, *EMBO J.* 5: 2955-61; Klämbt, Müller, Lützelshwab, Rossa, Totzke, and Schmidt, 1989, *Dev. Biol.* 133: 425-36; Lützelshwab, Klämbt, Rossa, and Schmidt, 1987, *EMBO J.* 6: 1791-97). During later embryogenesis relatively high amounts of *l(2)gl* protein is detected in pole cells and cells of the

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allele	origin	discoverer	ref ^α	lethal phase (%)		phenotypic grade	coordinates ^β
				larvae	pupae		
<i>l(2)gl</i> ¹	spont	Bridges, 33e9	5, 8	98.0	2.0	+++	19
<i>l(2)gl</i> ²	UV	Meyer, 51a	9				
<i>l(2)gl</i> ³	spont	Meyer, 51a	9				
<i>l(2)gl</i> ⁴	spont	Gateff	3, 8	89.1	10.9	+++	22.4
<i>l(2)gl</i> ⁶	EMS	Gateff	8				11.1
<i>l(2)gl</i> ²⁵	spont		8				22.4
<i>l(2)gl</i> ²⁶	spont		4, 8				17.4
<i>l(2)gl</i> ⁵²	spont		4, 8				12.8
<i>l(2)gl</i> ¹¹⁰	spont		2, 8	53.6	43.4	+++	35.6
<i>l(2)gl</i> ¹³⁸	spont		2, 8	98.0	2.0	+	22.4
<i>l(2)gl</i> ¹⁵⁰	spont		8				17.4
<i>l(2)gl</i> ²⁵⁸	spont		8				23.9
<i>l(2)gl</i> ²⁷¹	spont		2, 8	81.4	18.6	+	34.7
<i>l(2)gl</i> ²⁷⁵	spont		2, 8	79.1	20.9	++	11.4 to 19.3
<i>l(2)gl</i> ³⁰⁹	spont		2	54.0	46.0	++	
<i>l(2)gl</i> ³¹⁴	spont		2, 8	57.3	42.7	+	10.2
<i>l(2)gl</i> ³³⁴	spont		2, 8	18.4	81.6	+	
<i>l(2)gl</i> ³⁵¹	spont		2	17.5	82.5	++	
<i>l(2)gl</i> ³⁵³	spont		2, 8	19.5	80.5	+	27.2
<i>l(2)gl</i> ⁴³²	spont		8				32.1
<i>l(2)gl</i> ⁵⁵⁸	spont		2, 8	97.2	2.8	-	27.2
<i>l(2)gl</i> ⁷⁰⁵	spont		2	89.7	10.3	++	
<i>l(2)gl</i> ^B	spont	Burnet	1				
<i>l(2)gl</i> ^{D150}	spont		2	97.3	2.7	+++	
<i>l(2)gl</i> ^{M119}	spont		2	85.0	15.0	+	
<i>l(2)gl</i> ^{ts1}	EMS	Hanratty	6, 7				
<i>l(2)gl</i> ^{ts2}	EMS	Hanratty	6, 7				

^α 1 = Burnet, 1968, DIS 43: 61; 2 = Gateff, Golubovsky, and Sokolova, 1977, DIS 52: 128-29; 3 = Gateff and Schneiderman, 1974, Wilhelm Roux's Arch. Dev. Biol. 176: 23-65; 4 = Green and Shepherd, 1979, Genetics 92: 823-32; 5 = Hadorn, 1937, Proc. Nat. Acad. Sci. USA 23: 478-84; 6 = Hanratty, 1984, Dev. Biol. 193: 90-97; 7 = Hanratty, 1984, Dev. Biol. 193: 98-107; 8 = Mechler, McGinnis, and Gehring, 1985, EMBO J. 4: 1551-57; 9 = Meyer and Edmondson, 1951, DIS 25: 72.

^β DNA coordinates with respect to an arbitrarily chosen origin close to the tip of 2L; positive values to the right. Most tested alleles are molecular deficiencies with one breakpoint distal to the origin of the 40 kb walk and the other as indicated in the table. Only *l(2)gl*⁵², which has a 10 kb insertion, and *l(2)gl*²⁷⁵, in which 7.9 kb have been deleted and replaced by an insert of 6.5 kb, are exceptions.

developing nervous system; specifically neurons in the peripheral nervous system undergoing axogenesis express the protein. Monoclonal antibodies specifically stain junctions between mammalian cells in culture as though they are recognizing either membrane or intercellular-matrix proteins (Klämbt *et al.*, 1989).

Ten-to-eleven-day-old larvae homozygous for larval-lethal alleles exhibit remarkably few puff sites, only 63BC and occasionally 88D and 89B; however, heat shock induced puffs develop normally (Ashburner, 1970, Chromosoma 31: 356-76). Homozygotes able to form prepupae exhibit more nearly normal puffing patterns; puffing in response to administration of ecdysone also appears normal (Richards, 1976, Wilhelm Roux's Arch. Dev. Biol. 179: 339-48).

alleles: A high incidence of mutant alleles found in natural populations in the Soviet Union (Golubovsky and Sokolova, 1973, DIS 50: 124). More than fifty independent alleles recorded.

cytology: Placed in 21A3-4 by *in situ* hybridization (Mechler, McGinnis, and Gehring).

molecular biology: Region from tip of 2L cloned and a 40 kb walk and restriction mapping carried out. Coordinate 0 at the left end of the walk, with positive coordinate values extending to the right toward the centromere. A transcribed region between coordinates 7.3 and 19.4 kb, which is interrupted or deleted by every *l(2)gl* allele tested, is able to rescue *l(2)gl* homozygotes by transformation (Opper, Schuler, and Mechler, 1987, Oncogene 1: 91-96); it produces transcripts of 4.3 and 5.5-6 kb in length, which originate from the same sequence through alternative splicing. The larger RNA encodes a polypep-

ptide of 1114 amino acids and approximately 130 kd, whereas the smaller one contains only the 708 N-terminal residues of the larger for a molecular weight of 780 kd. Comparison of genomic and cDNA sequences show *l(2)gl* to contain ten exons and to comprise two major blocks of coding exons, the core segment, comprising predominantly exons IV and V, which encodes the majority of the smaller polypeptide and the N-terminal portion of the larger one, and the tail segment comprising exons VII and VIII, which encodes the C-terminal portion of the larger polypeptide. Alternative splicing takes place in and around exon VI (Jacob, Opper, Metzroth, Phannavong, and Mechler, 1987, Cell 50: 215-25). The conceptual sequence of the 130 kd protein shares many features with mammalian cell adhesion proteins, specifically members of the vertebrate cadherin family of proteins, suggesting a function in cell to cell interaction (Lützeltschwab *et al.*, 1987; Klämbt *et al.*, 1989). *l(2)gl* protein lacks the transmembrane domain found in cadherins, but it does have an internally located signal sequence near the N-terminus. It contains four kinds of sequences that show 30 to 40% homology, and half that level of identity with similar sequences in mammalian cadherins (*e.g.* L-CAM): sequence A of some 60 residues has three characteristically spaced N-glycosylation sites; sequence B is around 140 amino acids in length with two cysteine residues and all N-glycosylation sites conserved; sequence C is 70 residues in length and has a high concentration of negative charges; and sequence D contains 45 residues and is positively charged. The sequence of L-CAM can be represented as cytoplasmic domain-transmembrane domain-B—A-C-D— and that of *l(2)gl*

as AA-B-AD—CC-D—.

other information: At least one allele of *bhe* (broad head) fails to complement *l(2)gl* alleles and is covered by duplications for the 2L terminus (Kennison).

l(2)H: lethal (2) of Humphrey

location: 2-50.

origin: Spontaneous.

discoverer: Humphrey, 32k.

references: Dunn, 1934, DIS 1: 30.
1935, DIS 4: 9.

phenotype: Usually dies as pupa; 10-15% of flies survive and look normal but are weak. Homozygote usually sterile when inbred but fertile in outcrosses. RK3.

l(2)hst: lethal (2) histolytic

location: 2-56.

origin: X ray induced.

discoverer: Thompson, 59k.

phenotype: Homozygote dies in early pupal stage. Heterozygous viability good. RK3.

l(2)K: lethals from Kofu-Katsunama, Japan

origin: Repeatedly isolated from natural population in Kofu-Katsunama, Japan.

references: Watanabe and Oshima, 1970, Genetics 64: 93-106.

locus	genetic location
<i>l(2)K201</i>	2-47.9
<i>l(2)K202</i>	2-33.7
<i>l(2)K203</i>	2-5.5
<i>l(2)K204</i>	2-32.1
<i>l(2)K207</i>	2-58.4
<i>l(2)K208</i>	2-16.7
<i>l(2)K215</i>	2-1.9
<i>l(2)K224</i>	2-46.2
<i>l(2)K225</i>	2-27.5
<i>l(2)K234</i>	2-57.9
<i>l(2)K255</i>	2-65.1
<i>l(2)K300</i>	2-56.6
<i>l(2)K305</i>	2-53.0
<i>l(2)K326</i>	2-37.7
<i>l(2)K327</i>	2-101.8
<i>l(2)K333</i>	2-65.4
<i>l(2)K335</i>	2-59.9
<i>l(2)K483</i>	2-53.7
<i>l(2)K508</i>	2-69.7
<i>l(2)K513</i>	2-57.8
<i>l(2)K534</i>	2-1.0

l(2)M: lethal (2) from Mohr

location: 2- (between *dp* and *b*).

origin: Spontaneous.

discoverer: Bridges, 33118.

l(2)Mad51ⁿ⁻: see *l(2)S*

l(2)Mass38^{x-}: see *l(2)S*

l(2)mat: lethal (2) maternal

location: 2- (near *pr*).

origin: Spontaneous.

discoverer: Redfield, 23b.

references: 1924, Am. Naturalist 58: 566-69.
1926, Genetics 11: 482-502.

phenotype: Homozygous females produce 1 daughter to 5.5 sons. Abnormal sex ratio caused by inviability of

females. *l(2)mat* does not seem to be allelic to *da*, which has a similar effect. RK3.

l(2)mbn: lethal (2) malignant blood neoplasm

origin: Induced by ethyl methanesulfonate.

references: Gateff, 1977, DIS 52: 4.

1978, Biol. Rev. Cambridge Philos. Soc. 53: 123-68.

1978, Nature 200: 1448-59.

phenotype: Hyperplasia of cellular components of larval hemolymph; cell counts 30-40 times normal; cells resemble immature plasmatocytes. In premortal stages the larval tissues have mostly disintegrated and larvae appear to be sacks filled with blood cells. Cells do not proliferate when transplanted to normal larvae.

l(2)me: lethal (2) meander

location: 2-72 (71-73).

origin: Spontaneous.

discoverer: Hadorn, 44g20.

synonym: *lme*.

references: 1947, Exptl. Biol. Symp., Vol. 2: 177-95, Cambridge Univ. Press.

1947, DIS 21: 68.

Schmid, 1949, Z. Indukt. Abstamm. Vererbungslehre 83: 220-53 (fig.).

Chen and Hadorn, 1954, Rev. Suisse Zool. 61: 437-51.

1955, Rev. Suisse Zool. 62: 338-47.

phenotype: Larvae do not grow normally, die while small. Body length remains relatively shorter than tracheal stems, which become convoluted in a meandering manner. Salivary glands reach 30% normal size; pharyngeal development normal. Intestines lack proteolytic enzymes. RK3. Homozygotes show altered charged tRNA^{Glu} isoacceptor pattern. Kubli (1978, Rev. Suisse Zool. 85: 790) suggests a deletion for *tRNA-Glu* and *HexC*.

l(2)mr²: lethal (2) with morula

location: 2-70.

origin: Spontaneous.

discoverer: Bridges, 25k24.

l(2)MV: lethal (2) of Mglinetz and Vikulova

location: 2-82 (not separated from *wt*).

references: Mglinetz and Vikulova, 1977, Genetika 13: 1318-20.

phenotype: Homozygotes die after head eversion at 29°, but viable and fertile adults produced at 17° and 25°. Transplanted mutant disks into wild-type hosts develop normally.

****l(2)NS: lethal (2) Nova Scotia***

location: 2-107.0 [to the right of *l(2)ax* and to the left of *sp*].

discoverer: Bridges, 23j31.

references: 1937, Cytologia (Tokyo), Fujii Jub., Vol. 2: 745-55.

phenotype: Lethal when larvae are about 2 mm long. Development of tracheae and other chitinized parts abnormal. RK3A.

cytology: Exists only as *In(2R)NS, px l(2)NS sp*. Salivary chromosome locus in 60B10-12 on the basis of its inclusion in *Df(2R)Px = Df(2R)60B8-10;60D1-2* but not in the *2R^{PX}D* element of *T(1;2)Bld = T(1;2)1C3-4;60B12-13* (Bridges).

l(2)pm: lethal (2) polymorph

location: 2-30.3.

origin: X ray induced.

discoverer: Käfer, 50b.

references: Benz, 1953, DIS 27: 55.

1957, Z. Indukt. Abstamm. Vererbungs. 88: 78-114 (fig.).

phenotype: Flies die throughout larval and pupal stages. Larvae do not contract before pupation; hence, pupae are long and thin. Imagos often cryptocephalic. Chief characteristic is a severe muscular dystrophy. Protein metabolism extremely disturbed. In larval stage, free amino acids and one peptide are in abnormally high concentration. Prepupae only slightly different from normal in this respect. Occasional survivors viable and fertile. RK3.

****l(2)pup: lethal (2) pupal***

location: 2-47.

origin: Spontaneous.

discoverer: Ives, 38j25.

references: 1945, Genetics 30: 175.

1945, DIS 19: 46.

phenotype: Dies during middle or late pupal stage. External anatomy appears normal except for heavy melanization of wings and legs. RK3.

l(2)R: lethal (2) of Redfield

location: 2- [in 2L with *In(2L)t*].

discoverer: Redfield, 1933.

l(2)S: lethal (2) of Seto

A series of late larval and pupal lethals recovered and examined developmentally by Seto. All are lost; more thorough phenotypic descriptions may be found in the references.

references: 1954, J. Exp. Zool. 126: 17-32.

1954, Am. Nat. 88: 373-78.

1956, J. Hered. 47: 21-27 (fig.).

1957, DIS 31: 160-62.

1958, DIS 32: 157-58.

1961, DIS 35: 94-95.

1963, DIS 37: 128-229.

locus	genetic location	origin
* <i>l(2)S1</i>		spont
* <i>l(2)S1A</i>	<i>Sp-b</i>	spont
* <i>l(2)S3</i>	<i>c-px</i>	X ray
* <i>l(2)S3a</i>	<i>dp-Sp</i>	γ ray
* <i>l(2)S4</i>	near <i>pr</i>	spont
* <i>l(2)S7</i>	<i>Sp-b</i>	γ ray
* <i>l(2)S11</i>		X ray
* <i>l(2)S13</i>		spont
* <i>l(2)S32</i>	<i>dp-Sp</i>	spont
* <i>l(2)S42</i>	<i>Bl-L</i>	spont
* <i>l(2)S42A</i>		spont
* <i>l(2)S45</i>	<i>Sp-b</i>	spont
* <i>l(2)S50</i>		spont
* <i>l(2)S51</i>	near <i>pr</i>	spont
* <i>l(2)S55</i>	<i>dp-Sp</i>	spont
* <i>l(2)S59</i>	<i>h</i>	spont
* <i>l(2)S61</i>	near <i>pr</i>	spont

l(2)Sp: lethal (2) of Spiess

A series of recessive lethal mutations extracted from a long term (170 generations) laboratory population.

references: Spiess, Helling, and Capenos, 1963, Genetics

48: 1377-86.

locus	genetic location	synonym	comments
* <i>l(2)Sp1</i>	2-35.0		
* <i>l(2)Sp2b</i>	2-49		
* <i>l(2)Sp6b</i>	2-50.0		
<i>l(2)Sp7</i>	2-3.2		
* <i>l(2)Sp18</i>	2-61.5		
* <i>l(2)Sp19a</i>	2-1.9		
<i>l(2)Sp9b</i>	2-49		
* <i>l(2)Sp9c</i>	2-55.1	<i>l(2)41Ae³⁹</i>	between <i>ri</i> and <i>stw</i>
* <i>l(2)Sp9d</i>	2-55.1		to the right of <i>stw</i>
* <i>l(2)Sp10</i>	2-37.5		
<i>l(2)Sp11</i>	2-55.1	<i>l(2)41Ae⁴⁰</i>	
* <i>l(2)Sp12</i>	2-61.5		
* <i>l(2)Sp14</i>	2-32.0		
<i>l(2)Sp15</i>	2-55.1	<i>l(2)41Ah³³</i>	
* <i>l(2)Sp18</i>	2-65.3		

l(2)Su(H): lethal (2) from Suppressor of Hairless

location: 2-99.

origin: Spontaneous.

discoverer: Bridges, 3717.

cytology: Located in salivary region 58A1 through 58F8 on the basis of its inclusion in *Df(2R)M-1 = Df(2R)57F11-58A1,58F8-59A1*.

l(2)Stn: lethal (2) of Steinmetz

location: 2-50.

origin: Naturally occurring.

discoverer: Steinmetz.

references: Liebenguth and Steinmetz, 1976, Biochem. Genet. 14: 299.

phenotype: Late pupal lethal; pigmentation of eyes and cuticular tissues retarded.

cytology: Not included in *Df(2L)64j*, *Df(2L)75c*, or *Df(2L)fn1* (Woodruff and Ashburner, 1979, Genetics 92: 117-32).

****l(2)T: lethal (2) of Thompson***

origin: Spontaneous in normal chromosome of *SM1/+* heterozygote.

discoverer: Thompson, 1956, 1957.

synonym: *l(2)56i24* through *l(2)57h10*.

other information: A series of 13 independently-occurring and genetically-located lethals.

l(2)Wau55ⁿ⁻: see *l(2)S*

l(3)I: see *l(3)a*

l(3)26: see *Ace¹*

l(3)36d10

location: 3- (close to *D*, or rearrangement).

origin: Spontaneous.

discoverer: Bridges, 36d10.

****l(3)36d24***

location: 3- (near centromere).

origin: Spontaneous.

discoverer: Bridges, 36d24.

references: 1937, DIS 7: 13.

Bridges and Bridges, 1938, Genetics 23: 111-14.

l(3)62

Lethals in thirteen complementation groups isolated in the region uncovered by $Df(3L)R-R2 = Df(3L)62B2-4;62D3-5$ (Sliter, Henrich, Tucker, and Gilbert, 1989, Genetics 123: 327-36).

locus	genetic location	cytological location	included in	excluded from	synonym
l(3)62Ba	3-{1.5}	62B2-9	<i>Df(3L)R-R2</i>	<i>Df(3L)R-G7</i>	<i>l(3)dre1</i>
l(3)62Bb	3-{1.5}	62B2-9	<i>Df(3L)R-R2</i>	<i>Df(3L)R-G7</i>	<i>l(3)dre9</i>
l(3)62Bc	3-{1.5}	62B2-9	<i>Df(3L)R-R2</i>	<i>Df(3L)R-G7</i>	<i>l(3)dre2</i>
l(3)62Bd	3-{1.5}	62B2-9	<i>Df(3L)R-R2</i>	<i>Df(3L)R-G7</i>	<i>l(3)dre3</i>
l(3)62Be	3-{1.5}	62B2-9	<i>Df(3L)R-R2</i>	<i>Df(3L)R-G7</i>	<i>l(3)neo7</i>
l(3)62Bf	3-1.4	62B8-9	<i>Df(3L)R-G7</i>	<i>Df(3L)R-G2</i>	R
l(3)62Bg	3-{1.5}	62B8-12	<i>Df(3L)R-G7</i>	<i>Df(3L)R-G2</i>	<i>l(3)dre4</i>
l(3)62Bh	3-{1.5}	62B8-12	<i>Df(3L)R-G7</i>	<i>Df(3L)R-G2</i>	<i>l(3)dre5</i>
l(3)62Bi	3-{1.5}	62B11-C1	<i>Df(3L)R-G2</i>	<i>Df(3L)R-E</i>	<i>l(3)dre6</i>
l(3)62Bj	3-{1.5}	62B11-C1	<i>Df(3L)R-G2</i>	<i>Df(3L)R-E</i>	<i>l(3)dre7</i>
l(3)62Ca	3-{1.5}	62B12-D1	<i>Df(3L)R-E</i>	<i>Df(3L)R-G5</i>	<i>l(3)dre8</i>
l(3)62Da	3-{1.5}	62B11-C1	<i>Df(3L)R-G5</i>	<i>Df(3L)R-R2</i>	<i>l(3)dre10</i>
l(3)62Db	3-{1.5}	62B11-C1	<i>Df(3L)R-G5</i>	<i>Df(3L)R-R2</i>	ecd

allele	origin	comments
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l(3)62Ba¹	EMS	L3-P
l(3)62Ba²	EMS	also <i>l(3)Bb¹</i>
l(3)62Ba³	γ ray	L3-P
l(3)62Bb¹	EMS	L2; also <i>l(3)Ba²</i>
l(3)62Bb²	EMS	L2
l(3)62Bb³	EMS	L2

l(3)62Bc

phenotype: Hemizygous lethal; larvae die in the third instar, with one allele occasionally able to proceed into the prepupal stage. Mature larvae discless; in addition, larval brain, but not the ventral ganglion, greatly reduced in size.

allele	origin	synonym	comments
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l(3)62Bc¹	EMS		
l(3)62Bc²	EMS		
l(3)62Bc³	γ ray	<i>dre2^{g17}</i>	<i>ln(3L)62B2-7;63F</i>

l(3)62Bd

phenotype: Hemizygotes die in first larval instar, with one of seven alleles tested occasionally surviving until the second instar.

allele	origin
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l(3)62Bd¹⁻⁵	EMS
l(3)62Bd⁶⁻⁷	γ ray

allele	origin	synonym	comments
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l(3)62Be¹	P	<i>l(3)neo7^{lα}</i>	L2; L2/3
l(3)62Be²	EMS	<i>l(3)neo7^{e43}</i>	P-A

^α Cooley, Kelly, and Spradling, 1988, Science 239: 1121-28.

l(3)62Bg

phenotype: Locus displays *ca* 80 times the mutation rate of most of the other loci in the region, and it is relatively more sensitive to γ irradiation than to EMS mutagenesis than other loci (by a factor of six); in addition, none of the alleles is associated with a gross chromosome rearrangement. Hemizygotes are arrested in the first larval

instar, where they remain robust and active. One allele temperature sensitive; developmental arrest takes place shortly after shift up in temperature at several stages.

allele	origin
--------	--------

l(3)62Bg¹⁻¹⁸	EMS
l(3)62Bg¹⁹⁻⁷⁴	γ ray

l(3)62Bh

phenotype: Hemizygous larvae die during the third instar; one allele displays occasional escapers into the second instar.

allele	origin	comments
--------	--------	----------

l(3)62Bh¹	EMS
l(3)62Bh²	EMS
l(3)62Bh³	EMS
l(3)62Bh⁴	EMS
l(3)62Bh⁵	γ ray

l(3)62Bi¹	EMS	temperature sensitive; dies as pharate adult
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l(3)62Bj¹	γ ray	L1
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l(3)62Ca

phenotype: Hemizygotes for seventeen of nineteen alleles die during the first larval instar; four of these show some escape into the second instar; two alleles cause lethality in the pupal-adult stage.

allele	origin	comments
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l(3)62Ca¹⁻¹²	EMS	L1
l(3)62Ca¹³	γ ray	L1; <i>T(2;3)27E-F;62C2-D1</i>
l(3)62Ca¹⁴	γ ray	P-A
l(3)62Ca¹⁵	γ ray	P-A
l(3)62Ca¹⁶⁻¹⁹	γ ray	L1

l(3)62Da¹	HD	L2
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***l(3)62g**

origin: Spontaneous.

discoverer: Paik.

references: 1963, Proc. Intern. Congr. Genet., 11th., Vol. 1: 163-64.

other information: A series of 65 lethals recovered from Korean wild populations.

DEFICIENCY MAP OF REGION 62

side	breakpoint	variant
left	62A10-B1	<i>Df(3L)R-G5</i>
left	62B2-4	<i>Df(3L)R-R2</i>
left	62B2-4	<i>Df(3L)R-G2</i>
		l(3)62Ba
		l(3)62Bb
		l(3)62Bc
		l(3)62Bd
		l(3)62Be
left	62B8-9	<i>Df(3L)R-E</i>
left	62B8-9	<i>Df(3L)R-G7</i>
		R
		Aprt
		l(3)62Bf
		l(3)62Bg
right	62B11-12	<i>Df(3L)R-G2</i>
		l(3)62Bh
		l(3)62Bi
right	62B12-C1	<i>Df(3L)R-E</i>
		l(3)62Ca

THE GENOME OF *DROSOPHILA MELANOGASTER*

side	breakpoint	variant
right	62C4-D1	<i>Df(3L)R-G5</i> I(3)62Da <i>ecd</i>
right	62D3-5	<i>Df(3L)R-R2</i>
right	62F2-5	<i>Df(3L)R-G7</i>

I(3)63

Results of saturation mutagenesis of the region of *Hsp83* (Wohlwill and Bonner).

locus	cytological		excluded from	synonym
	location	included in		
I(3)63Aa	63A8-B6	<i>Df(3L)HR218</i>	<i>Df(3L)HR298</i>	
I(3)63Ab	63A8-B6	<i>Df(3L)HR218</i>	<i>Df(3L)HR298</i>	
I(3)63Ba	63B6-C1	<i>Df(3L)HR218</i>	<i>Df(3L)HR232</i>	
I(3)63Bb	63B6-C1	<i>Df(3L)HR218</i>	<i>Df(3L)HR232</i>	
I(3)63Bc	63B6-C1	<i>Df(3L)HR218</i>	<i>Df(3L)HR232</i>	
I(3)63Bd	63B6-C1	<i>Df(3L)HR218</i>	<i>Df(3L)HR232</i>	
I(3)63Be	63B6-C1	<i>Df(3L)HR218</i>	<i>Df(3L)HR232</i>	
I(3)63Bf	63B6-C1	<i>Df(3L)HR218</i>	<i>Df(3L)HR232</i>	M(3)63B , <i>M(3)LS3</i>
I(3)63Ea	63D3-F1	<i>Df(3L)HR119</i>	<i>Df(3L)HR232</i>	
I(3)63Eb	63D3-F1	<i>Df(3L)HR119</i>	<i>Df(3L)HR232</i>	

allele	origin	discoverer	synonym	comments
I(3)63Aa¹	EMS	Wohlwill	<i>E-33</i>	
I(3)63Ab¹	EMS	Wohlwill	<i>E-13 995</i>	
I(3)63Ab²	EMS	Wohlwill	<i>18-4</i>	
I(3)63Ba¹	EMS	Wohlwill	<i>E25-299</i>	
I(3)63Bb¹	DEB	Wohlwill	<i>D32-187</i>	
I(3)63Bb²	DEB	Wohlwill	<i>D27-1159</i>	
I(3)63Bb³	EMS	Wohlwill	<i>9-9</i>	
I(3)63Bb⁴	EMS	Wohlwill	<i>9-1</i>	
I(3)63Bb⁵	EMS	Wohlwill	<i>1-7</i>	
I(3)63Bc¹	EMS	Wohlwill	<i>E-13 564</i>	
I(3)63Bc²	EMS	Wohlwill	<i>29-12</i>	
I(3)63Bc³	EMS	Wohlwill	<i>79</i>	
I(3)63Bc⁴	EMS	Wohlwill	<i>14-7</i>	
I(3)63Bc⁵	EMS	Wohlwill	<i>23-16</i>	leaky
I(3)63Bd¹	EMS	Wohlwill	<i>5-5</i>	
I(3)63Bd²	EMS	Wohlwill	<i>12-1</i>	
I(3)63Be¹	EMS	Wohlwill	<i>8-1</i>	
I(3)63Be²	EMS	Wohlwill	<i>1-1</i>	
I(3)63Be³	EMS	Wohlwill	<i>29-17</i>	
I(3)63Be⁴	EMS	Wohlwill	<i>10-5</i>	leaky
I(3)63Ea¹	EMS	Wohlwill	<i>164</i>	
I(3)63Eb¹	EMS	Wohlwill	<i>203</i>	

DEFICIENCY MAP OF REGION 63A-C

side	breakpoint	variant
left	62B	<i>Dp(3;3)BK14</i>
left	62C1	<i>Dp(3;3)BK7</i>
left	63A1	<i>Df(3L)HR370</i>
left	63A1	<i>Dp(3;3)BK116</i>
left	63B6	<i>Df(3L)HR218</i> I(3)63Aa I(3)63Ab
left	63B6	<i>Df(3L)HR298</i> I(3)63Ba

side	breakpoint	variant
		I(3)63Bb
		I(3)63Bc
		I(3)63Bd
		I(3)63Be
		M(3)63B
left	63C1	<i>Df(3L)HR232</i>
left	63B12-C1	<i>Df(3L)HR277</i>
left	63C6	<i>Df(3L)HR119</i>
left	63C6	<i>Dp(3;3)M2</i>
left	63C6	<i>Dp(3;3)M28</i>
right	63D1	<i>Df(3L)HR218</i>
right	63D1	<i>Df(3L)HR370</i>
right	63D3	<i>Df(3L)HR232</i> I(3)63Ea I(3)63Eb
right	63E9	<i>Df(3L)HR119</i>
right	64A6	<i>Df(3L)HR298</i>
right	64B7	<i>Dp(3;3)M2</i>
right	64B10	<i>Dp(3;3)BK14</i>
right	64B10-12	<i>Dp(3;3)BK116</i>
right	64B10-12	<i>Dp(3;3)BK7</i>
right	64B12	<i>Df(3L)HR277</i>
right	65A	<i>Dp(3;3)M28</i>

I(3)67

Results of saturation mutagenesis experiments in the region uncovered by *Df(3L)AC1 = Df(3L)67A2;67D11-13* plus additional saturation of its region of overlap with *Df(3L)29A6 = Df(3L)66F3;67B1* (Leicht and Bonner, 1988, Genetics 119: 579-93).

I(3)67A

Five complementation groups of unknown order; two are included in the cytologically invisible deficiency, *Df(3L)e146*, whose position in 67A is undetermined.

locus	genetic		cytological	
	location	location	included in	excluded from
I(3)67Aa	3-{27}	67A2-B1	<i>Df(3L)29A6</i> <i>Df(3L)AC1</i>	<i>Df(3L)e146</i>
I(3)67Ab	3-{27}	67A2-B1	<i>Df(3L)29A6</i> <i>Df(3L)AC1</i>	<i>Df(3L)e146</i>
I(3)67Ac	3-{27}	67A2-B1	<i>Df(3L)e146</i>	
I(3)67Ad	3-{27}	67A2-B1	<i>Df(3L)e146</i>	
I(3)67Ae	3-{27}	67A2-B1	<i>Df(3L)29A6</i> <i>Df(3L)AC1</i>	<i>Df(3L)e146</i>
I(3)67Af	3-{27}	67A2-B1	<i>Df(3L)29A6</i> <i>Df(3L)AC1</i>	<i>Df(3L)e146</i>

allele	origin	discoverer	synonym	comments ^α
I(3)67Aa¹	DEB	Leicht	<i>l(3)d37A1</i>	P-A
I(3)67Aa²	DEB	Leicht	<i>l(3)d174B5</i>	P-A
I(3)67Aa³	DEB	Leicht	<i>l(3)d232A2</i>	P-A
I(3)67Aa⁴	EMS	Leicht	<i>l(3)e73B3</i>	P-A
I(3)67Aa⁵	EMS	Leicht	<i>l(3)e106C3</i>	P-A
I(3)67Aa⁶	EMS	Matthews	<i>l(3)E1.1</i>	P-A
I(3)67Ab¹	DEB	Leicht	<i>l(3)d236A1</i>	pharate adults
I(3)67Ab²	EMS	Leicht	<i>l(3)e61c</i>	pharate adults
I(3)67Ab³	EMS	Leicht	<i>l(3)e76A1</i>	pharate adults
I(3)67Ac¹	EMS	Leicht	<i>l(3)e78B4</i>	young adult ^β
I(3)67Ad¹	DEB	Leicht	<i>l(3)d189B2</i>	L
I(3)67Ad²	EMS	Leicht	<i>l(3)e152C4</i>	L
I(3)67Ae¹	EMS	Leicht	<i>l(3)e72A2</i>	L
I(3)67Af¹	EMS	Matthews	<i>l(3)e17</i>	E

α Lethal phase of hemizygotes.
 β Smaller and rougher eyes than wild type; some individuals also have held-back wings.

I(3)67BD

Eighteen lethally mutable complementation groups including *M(3)67C*. Linear order of loci undetermined.

locus	genetic location	cytological location	included in	excluded from	synonym
I(3)67BDa	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	
I(3)67BDb	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	
I(3)67BDc	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	
I(3)67BDd	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	
I(3)67BDe	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	
I(3)67BDf	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	
I(3)67BDg	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	
I(3)67BDh	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	
I(3)67BDi	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	
I(3)67BDj	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	
I(3)67BDk	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	
I(3)67BDl	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	
I(3)67BDm	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	
I(3)67BDn	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	
I(3)67BDo	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	M(3)67C, M(3)I
I(3)67BDp	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	
I(3)67BDq	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	
I(3)67BDr	3-[28]	67B1-D13	<i>Df(3L)AC1</i>	<i>Df(3L)29A6</i>	

allele	origin	discoverer	synonym	comments α
I(3)67BDa¹	DEB	Leicht	<i>l(3)d442</i>	
I(3)67BDa²	DEB	Leicht	<i>l(3)d7A5</i>	
I(3)67BDa³	DEB	Leicht	<i>l(3)d17A6</i>	L
I(3)67BDa⁴	DEB	Leicht	<i>l(3)d36A7</i>	
I(3)67BDa⁵	DEB	Leicht	<i>l(3)d38A5</i>	
I(3)67BDa⁶	DEB	Leicht	<i>l(3)d38B5</i>	
I(3)67BDa⁷	DEB	Leicht	<i>l(3)d54A7</i>	
I(3)67BDa⁸	DEB	Leicht	<i>l(3)d58A7</i>	weak allele
I(3)67BDa⁹	EMS	Leicht	<i>l(3)e64A1</i>	P-A
I(3)67BDa¹⁰	EMS	Leicht	<i>l(3)e77A5</i>	P-A
I(3)67BDa¹¹	EMS	Leicht	<i>l(3)e81B4</i>	P-A
I(3)67BDa¹²	EMS	Leicht	<i>l(3)e86D1-1</i>	
I(3)67BDa¹³	EMS	Leicht	<i>l(3)e105B2</i>	L
I(3)67BDa¹⁴	EMS	Leicht	<i>l(3)e122A7</i>	
I(3)67BDa¹⁵	EMS	Leicht	<i>l(3)e138A6</i>	
I(3)67BDa¹⁶	EMS	Leicht	<i>l(3)e142A2</i>	L
I(3)67BDa¹⁷	EMS	Leicht	<i>l(3)e143A1</i>	
I(3)67BDa¹⁸	EMS	Leicht	<i>l(3)e153A6</i>	
I(3)67BDa¹⁹	EMS	Leicht	<i>l(3)e154B3</i>	P-A
I(3)67BDb¹	DEB	Leicht	<i>l(3)d5A1</i>	
I(3)67BDb²	DEB	Leicht	<i>l(3)d7A6</i>	
I(3)67BDb³	DEB	Leicht	<i>l(3)d10A3</i>	E
I(3)67BDb⁴	DEB	Leicht	<i>l(3)d49A5</i>	E
I(3)67BDb⁵	EMS	Leicht	<i>l(3)e66A5</i>	
I(3)67BDb⁶	EMS	Leicht	<i>l(3)e75B4</i>	P-A
I(3)67BDb⁷	EMS	Leicht	<i>l(3)e86D1-2</i>	E
I(3)67BDb⁸	EMS	Leicht	<i>l(3)e113A1-2</i>	L-P
I(3)67BDb⁹	EMS	Leicht	<i>l(3)e132A7</i>	pharate adults
I(3)67BDc¹	DEB	Leicht	<i>l(3)d8A1</i>	
I(3)67BDc²	DEB	Leicht	<i>l(3)d14A3</i>	L
I(3)67BDc³	DEB	Leicht	<i>l(3)d49A3</i>	
I(3)67BDc⁴	EMS	Leicht	<i>l(3)e90C3</i>	L-P
I(3)67BDc⁵	EMS	Leicht	<i>l(3)e97B3</i>	L
I(3)67BDd¹	DEB	Leicht	<i>l(3)d44A2</i>	
I(3)67BDd²	DEB	Leicht	<i>l(3)d48A6</i>	L
I(3)67BDd³	EMS	Leicht	<i>l(3)e78A1</i>	L
I(3)67BDd⁴	EMS	Leicht	<i>l(3)e113A1-1</i>	L
I(3)67BDd⁵	EMS	Leicht	<i>l(3)e154A1</i>	L
I(3)67BDe¹	DEB	Leicht	<i>l(3)d10A5</i>	L
I(3)67BDe²	DEB	Leicht	<i>l(3)d14A5</i>	
I(3)67BDe³	DEB	Leicht	<i>l(3)d49A4</i>	

allele	origin	discoverer	synonym	comments α
I(3)67BDe⁴	EMS	Leicht	<i>l(3)E150C4</i>	L
I(3)67BDf¹	DEB	Leicht	<i>l(3)d23A3</i>	
I(3)67BDf²	DEB	Leicht	<i>l(3)d35A6</i>	E
I(3)67BDf³	EMS	Leicht	<i>l(3)e2J187</i>	L
I(3)67BDg¹	DEB	Leicht	<i>l(3)d7A3</i>	
I(3)67BDg²	DEB	Leicht	<i>l(3)d19A3</i>	L-P
I(3)67BDg³	EMS	Leicht	<i>l(3)e88B2</i>	P-A
I(3)67BDg⁴	EMS	Leicht	<i>l(3)e94A5</i>	L-P
I(3)67BDh¹	DEB	Leicht	<i>l(3)d14A7</i>	
I(3)67BDh²	DEB	Leicht	<i>l(3)d45C2</i>	L
I(3)67BDh³	EMS	Leicht	<i>l(3)e94D1</i>	homozygotes die as adults
I(3)67BDh⁴	EMS	Leicht	<i>l(3)e140B1</i>	homozygotes die as adults
I(3)67BDi¹	DEB	Leicht	<i>l(3)d44A3</i>	
I(3)67BDi²	DEB	Leicht	<i>l(3)d51B2</i>	L-P
I(3)67BDj¹	DEB	Leicht	<i>l(3)e73B2</i>	P-A
I(3)67BDk¹	DEB	Leicht	<i>l(3)d11A5</i>	
I(3)67BDl¹	DEB	Leicht	<i>l(3)d33A3</i>	
I(3)67BDl²	EMS	Leicht	<i>l(3)e154A2</i>	L
I(3)67BDm¹	DEB	Leicht	<i>l(3)d47B5</i>	L-P
I(3)67BDn¹	DEB	Leicht	<i>l(3)d50A1</i>	P-A
I(3)67BDp¹	EMS	Leicht	<i>l(3)e105A3</i>	
I(3)67BDq¹	EMS	Leicht	<i>l(3)e83A1</i>	P-A
I(3)67BDr¹	EMS	Leicht	<i>l(3)e97C2</i>	P-A

α Lethal phase of hemizygotes.

DEFICIENCY MAP OF REGION 67

side	breakpoint	variant
left	66F3	<i>Df(3L)29A6</i>
left	67A2	<i>Df(3L)AC1</i>
		I(3)67Aa
		I(3)67Ab
		I(3)67Ac
		I(3)67Ad
		I(3)67Ae
		I(3)67Af
right	67B1	<i>Df(3L)29A6</i>
		I(3)67BDa
		I(3)67BDb
		I(3)67BDc
		I(3)67BDd
		I(3)67BDe
		I(3)67BDf
		I(3)67BDg
		I(3)67BDh
		I(3)67BDi
		I(3)67BDj
		I(3)67BDk
		I(3)67BDl
		I(3)67BDm
		I(3)67BDn
		M(3)67C
		I(3)67BDp
		I(3)67BDq
		I(3)67BDr
right	67D11-13	<i>Df(3L)AC1</i>
left	67F	<i>Df(3L)P20</i>
left	67E1-2	<i>Df(3L)Ez6</i>
	67E1-4	E(z)
left	67E3-4	<i>Df(3L)Ez3</i>

side	breakpoint	variant
right	67E3-5	<i>Df(3L)Ez6</i>
right	67E6-7	<i>Df(3L)Ez3</i>
left	67F2-3	<i>Df(3L)vin2</i>

l(3)68

A collection of chemically-induced lethals in the region flanking the 68C *glu* gene cluster on chromosome three (Crosby and Meyerowitz, 1986, Genetics 112: 785-802). They have been mapped with deficiencies as well as recombinationally. The order of loci reflects the map order of the mutants except for *l(3)68Am*, which has not been recombinationally mapped. Hoogwerf, Akam, and Roberts (unpublished) have extended the analysis through 68F. An independent sample of chemically-induced lethal mutations uncovered by *Df(3L)lxd9* was selected and complementation mapped by Campbell, Hilliker, and Phillips (1986, Genetics, 112: 205-15). The latter sample of mutants has not been tested against those of Crosby and Meyerowitz, and the breakpoints of *Df(3L)lxd9* and the *lxd* locus are the only points shared by the analysis with the other two. Since gene order was determined using recombination by Crosby and Meyerowitz, their map is used as standard; the findings of Campbell, Hilliker, and Phillips are noted in the deficiency map of the region.

locus	genetic		cytological		synonym
	location	location	included in	excluded from	
<i>l(3)68Aa</i>	3-[35]	68A3-4	<i>Df(3L)vin5</i>	<i>Df(3L)lxd9</i>	<i>l(3)C288</i>
<i>l(3)68Ab</i>	3-[35]	68A3-4	<i>Df(3L)vin5</i>	<i>Df(3L)lxd9</i>	<i>l(3)C493</i>
<i>l(3)68Ac</i>	3-[35]	68A4-6	<i>Df(3L)lxd8</i>	<i>Df(3L)lxd9</i>	<i>l(3)C117</i>
<i>l(3)68Ad</i>	3-35.7	68A5-9	<i>Df(3L)lxd9</i>	<i>Df(3L)lxd8</i>	<i>l(3)517</i>
<i>l(3)68Ae</i>	3-[35]	68A5-9	<i>Df(3L)lxd9</i>	<i>Df(3L)lxd8</i>	<i>l(3)C178</i>
<i>l(3)68Af</i>	3-[35]	68A5-9	<i>Df(3L)lxd9</i>	<i>Df(3L)lxd8</i>	<i>l(3)C206</i>
<i>l(3)68Ag</i>	3-[35]	68A5-9	<i>Df(3L)lxd9</i>	<i>Df(3L)lxd8</i>	<i>l(3)C404</i>
<i>l(3)68Ah</i>	3-[35]	68A5-9	<i>Df(3L)lxd9</i>	<i>Df(3L)lxd8</i>	<i>l(3)C11</i>
<i>l(3)68Ai</i>	3-[35]	68A5-9	<i>Df(3L)lxd9</i>	<i>Df(3L)lxd8</i>	<i>l(3)C46</i>
<i>l(3)68Aj</i>	3-[35]	68A5-9	<i>Df(3L)lxd9</i>	<i>Df(3L)lxd8</i>	<i>l(3)C141</i>
<i>l(3)68Ak</i>	3-[35]	68A5-9	<i>Df(3L)lxd9</i>	<i>Df(3L)lxd8</i>	<i>l(3)C557</i>
<i>l(3)68Al</i>	3-[35]	68A5-9	<i>Df(3L)lxd9</i>	<i>Df(3L)lxd8</i>	<i>l(3)C28</i>
<i>l(3)68Am</i>	3-[35]	68A5-9	<i>Df(3L)lxd9</i>	<i>Df(3L)lxd8</i>	<i>l(3)B76</i>

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(3)68Aa</i> ¹	EMS	Crosby	<i>l(3)C288</i> ^a	1	L3
<i>l(3)68Aa</i> ²	EMS	Crosby	<i>l(3)C288</i> ^b	1	
<i>l(3)68Aa</i> ³	EMS	Crosby	<i>l(3)C288</i> ^c	1	
<i>l(3)68Aa</i> ⁴	EMS	Crosby	<i>l(3)C288</i> ^d	1	
<i>l(3)68Aa</i> ⁵	ENU	Villeneuve	<i>l(3)C288</i> ^e	1	semilethal; adults with thin bristles
<i>l(3)68Aa</i> ⁶	ENU	Villeneuve	<i>l(3)C288</i> ^f	1	
<i>l(3)68Aa</i> ⁷	ENU	Villeneuve	<i>l(3)C288</i> ^g	1	
<i>l(3)68Aa</i> ⁸	DEB	Crosby	<i>l(3)C288</i> ^h	1	
<i>l(3)68Aa</i> ⁹	DEB	Crosby	<i>l(3)C288</i> ⁱ	1	
<i>l(3)68Ab</i> ¹	DEB	Crosby	<i>l(3)C493</i> ^a	1	L1
<i>l(3)68Ab</i> ²	EMS	Crosby	<i>l(3)C493</i> ^b	1	
<i>l(3)68Ab</i> ³	EMS	Crosby	<i>l(3)C493</i> ^c	1	
<i>l(3)68Ab</i> ⁴	EMS	Crosby	<i>l(3)C493</i> ^d	1	semilethal; adults with rough eyes
<i>l(3)68Ac</i> ¹	EMS	Crosby	<i>l(3)C117</i> ^a	1	L2
<i>l(3)68Ac</i> ²	ENU	Crosby	<i>l(3)C117</i> ^b	1	
<i>l(3)68Ac</i> ³	ENU	Villeneuve	<i>l(3)C117</i> ^c	1	
<i>l(3)68Ac</i> ⁴	EMS	Crosby	<i>l(3)C117</i> ^d	1	

^α 1 = Crosby and Meyerowitz, 1986, Genetics 112: 785-802.

l(3)68Ad

phenotype: Homozygote lethal at prepupal stage; imaginal discs small. Mitotic figures normal (Gatti).

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(3)68Ad</i> ¹	NNG	Shearn	<i>l(1)517</i>	2	L3-EP
<i>l(3)68Ad</i> ²	EMS	Crosby	<i>l(3)517</i> ^b	1	L3
<i>l(3)68Ad</i> ³	EMS	Crosby	<i>l(3)517</i> ^c	1	
<i>l(3)68Ad</i> ⁴	EMS	Crosby	<i>l(3)517</i> ^d	1	semilethal; adults with dusky wings
<i>l(3)68Ae</i> ¹	EMS	Crosby	<i>l(3)C178</i> ^a	1	EP
<i>l(3)68Ae</i> ²	ENU	Villeneuve	<i>l(3)C178</i> ^b	1	
<i>l(3)68Ae</i> ³	ENU	Villeneuve	<i>l(3)C178</i> ^c	1	semilethal; adults with small wings, malformed legs; variable penetrance
<i>l(3)68Ae</i> ⁴	DEB	Crosby	<i>l(3)C178</i> ^d	1	
<i>l(3)68Ae</i> ⁵	DEB	Martin	<i>l(3)C178</i> ^e	1	
<i>l(3)68Af</i> ¹	ENU	Crosby	<i>l(3)C206</i> ^a	1	EP
<i>l(3)68Af</i> ²	ENU	Villeneuve	<i>l(3)C206</i> ^b	1	
<i>l(3)68Ag</i> ¹	ENU	Villeneuve	<i>l(3)C404</i> ^a	1	L2
<i>l(3)68Ag</i> ²	ENU	Villeneuve	<i>l(3)C404</i> ^b	1	
<i>l(3)68Ag</i> ³	ENU	Villeneuve	<i>l(3)C404</i> ^c	1	
<i>l(3)68Ag</i> ⁴	ENU	Villeneuve	<i>l(3)C404</i> ^d	1	
<i>l(3)68Ag</i> ⁵	EMS	Crosby	<i>l(3)C404</i> ^e	1	semilethal; adults with small, thin bristles
<i>l(3)68Ah</i> ¹	EMS	Crosby	<i>l(3)C11</i> ^a	1	E
<i>l(3)68Ah</i> ²	ENU	Crosby	<i>l(3)C11</i> ^b	1	
<i>l(3)68Ai</i> ¹	EMS	Crosby	<i>l(3)C46</i> ^a	1	L1-L2
<i>l(3)68Ai</i> ²	EMS	Crosby	<i>l(3)C46</i> ^b	1	
<i>l(3)68Ai</i> ³	EMS	Crosby	<i>l(3)C46</i> ^c	1	
<i>l(3)68Ai</i> ⁴	EMS	Crosby	<i>l(3)C46</i> ^d	1	semilethal; adults with upright or outspread wings; variable penetrance; female sterile
<i>l(3)68Ai</i> ⁵	ENU	Villeneuve	<i>l(3)C46</i> ^e	1	
<i>l(3)68Ai</i> ⁶	ENU	Villeneuve	<i>l(3)C46</i> ^f	1	
<i>l(3)68Ai</i> ⁷	ENU	Villeneuve	<i>l(3)C46</i> ^g	1	
<i>l(3)68Ai</i> ⁸	ENU	Villeneuve	<i>l(3)C46</i> ^h	1	same as <i>l(3)68Ai</i> ⁴
<i>l(3)68Ai</i> ⁹	DEB	Crosby	<i>l(3)C46</i> ⁱ	1	
<i>l(3)68Aj</i> ¹	EMS	Crosby	<i>l(3)C141</i> ^a	1	EP
<i>l(3)68Aj</i> ²	EMS	Crosby	<i>l(3)C141</i> ^b	1	
<i>l(3)68Aj</i> ³	DEB	Crosby	<i>l(3)C141</i> ^c	1	
<i>l(3)68Ak</i> ¹	ENU	Villeneuve	<i>l(3)C57</i> ^a	1	EP

^α 1 = Crosby and Meyerowitz, 1986, Genetics 112: 785-802. 2 = Shearn, Rice, Garen, and Gehring, 1971, Proc. Nat. Acad. Sci. USA 68: 2594-98.

l(3)68Ai

Alleles at this locus exhibit complex complementation relations.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(3)68Ai</i> ¹	EMS	Crosby	<i>l(3)C28</i> ^{a1}	1	
<i>l(3)68Ai</i> ²	EMS	Crosby	<i>l(3)C28</i> ^{a2}	1	
<i>l(3)68Ai</i> ³	EMS	Crosby	<i>l(3)C28</i> ^{b1}	1	E-L
<i>l(3)68Ai</i> ⁴	EMS	Crosby	<i>l(3)C28</i> ^{b2}	1	temperature sensitive
<i>l(3)68Ai</i> ⁵	EMS	Crosby	<i>l(3)C28</i> ^{c1}	1	E-L
<i>l(3)68Ai</i> ⁶	EMS	Crosby	<i>l(3)C28</i> ^{c2}	1	
<i>l(3)68Ai</i> ⁷	ENU	Villeneuve	<i>l(3)C28</i> ^{c3}	1	
<i>l(3)68Ai</i> ⁸	ENU	Crosby	<i>l(3)C28</i> ^{c4}	1	
<i>l(3)68Ai</i> ⁹	EMS	Crosby	<i>l(3)C28</i> ^{d1}	1	
<i>l(3)68Ai</i> ¹⁰	ENU	Villeneuve	<i>l(3)C28</i> ^{e1}	1	

^α 1 = Crosby and Meyerowitz, 1986, Genetics 112: 785-802.

l(3)68Am

phenotype: Semilethal; polyphasic. Adults with malformed legs, less often stunted wings; variable penetrance.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(3)68Am</i>¹	EMS	Roberts	<i>l(3)B76</i>	1, 3	L-P
<i>l(3)68Am</i>²	EMS	Crosby	<i>l(3)B76^b</i>	2	LP-A

^α 1 = Akam, Roberts, Richards, and Ashburner, 1978, Cell 13: 215-25; 2 = Crosby and Meyerowitz, 1986, Genetics 112: 785-802. 3 = Roberts and Evans-Roberts, 1976, Mol. Gen. Genet. 148: 57-64.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(3)68Ca</i>	3-[36]	68C8-11	<i>Df(3L)vin3</i>	<i>Df(3L)vin7</i>	<i>l(3)v4-2</i>
<i>l(3)68Cb</i>	3-[36]	68C8-11	<i>Df(3L)vin7</i>	<i>Df(3L)vin6</i>	<i>l(3)C605</i>
<i>l(3)68Cc</i>	3-[36]	68C8-11	<i>Df(3L)vin7</i>	<i>Df(3L)vin6</i>	<i>l(3)C70</i>
<i>l(3)68Cd</i>	3-[36]	68C8-D3	<i>Df(3L)vin6</i>		<i>l(3)rsg3</i>
<i>l(3)68Ce</i>	3-[36]	68C8-D3	<i>Df(3L)vin6</i>		<i>l(3)rsg4</i>
<i>l(3)68Cf</i>	3-[36]	68C8-D3	<i>Df(3L)vin6</i>		<i>l(3)rsg5</i>
<i>l(3)68Cg</i>	3-[36]	68C8-D3	<i>Df(3L)vin6</i>		<i>l(3)rsg6</i>
<i>l(3)68Ch</i>	3-[36]	68C8-D3	<i>Df(3L)vin6</i>		<i>l(3)rsg7</i>

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(3)68Ca</i>¹	EMS	Akam	<i>l(3)v4-2</i>	1	P-A; semilethal temperature sensitive
<i>l(3)68Cb</i>¹	EMS	Crosby	<i>l(3)C605</i>	2	L3
<i>l(3)68Cb</i>²	DEB	Martin	<i>l(3)C605</i>	2	
<i>l(3)68Cc</i>¹	EMS	Crosby	<i>l(3)C70</i>	2	P-A; semilethal; adults with dusky wings, thin bristles
<i>l(3)68Cd</i>¹	EMS		<i>l(3)V4-3</i>	1, 3	
<i>l(3)68Cd</i>²	EMS		<i>l(3)B1^D</i>	3	
<i>l(3)68Ce</i>¹	EMS		<i>l(3)V7-1</i>	1, 3	
<i>l(3)68Cf</i>¹	EMS		<i>l(3)I54</i>	3	
<i>l(3)68Cg</i>¹	EMS		<i>l(3)J27-2</i>	3	
<i>l(3)68Cg</i>²	EMS		<i>l(3)J90</i>	3	
<i>l(3)68Cg</i>³	EMS	Meyerowitz	<i>l(3)I13.178</i>	3	
<i>l(3)68Cg</i>⁴	EMS	Meyerowitz	<i>l(3)I13.253</i>	3	
<i>l(3)68Ch</i>¹	EMS	Meyerowitz	<i>l(3)I12.10</i>	3	
<i>l(3)68Ch</i>²	EMS	Meyerowitz	<i>l(3)I12.204</i>	3	
<i>l(3)68Ch</i>³	EMS	Meyerowitz	<i>l(3)I13.441</i>	3	

^α 1 = Akam, Roberts, Richards, and Ashburner, 1978, Cell 13: 215-25; 2 = Crosby and Meyerowitz, 1986, Genetics 112: 785-802; 3 = Hoogwerf, Akam, and Roberts, 1988, Genetics 118: 665-70.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(3)68Da</i>	3-[36]	68D3-6	<i>Df(3L)vin2</i>	<i>Df(3L)vin66</i>	<i>l(3)rsg8</i>
<i>l(3)68Db</i>	3-[36]	68D3-6	<i>Df(3L)vin2</i>	<i>Df(3L)vin66</i>	<i>l(3)rsg9</i>
<i>l(3)68Dc</i>	3-[36]	68D3-6	<i>Df(3L)vin2</i>	<i>Df(3L)vin66</i>	<i>l(3)rsg10</i>

allele	origin	synonym	ref ^α
<i>l(3)68Da</i>¹	EMS	<i>l(3)I.4</i>	1
<i>l(3)68Db</i>¹	EMS	<i>l(3)I2.5B</i>	1
<i>l(3)68Dc</i>¹	EMS	<i>l(3)I46</i>	1
<i>l(3)68Dc</i>²	EMS	<i>l(3)I55</i>	1

^α 1 = Hoogwerf, Akam, and Roberts, 1988, Genetics 118: 665-70.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(3)68Ea</i>	3-[36]	68D6-E4	<i>Df(3L)vin3</i>	<i>Df(3L)vin2</i>	<i>Cyca</i> , <i>l(3)rsg11</i>
<i>l(3)68Eb</i>	3-[36]	68D6-E4	<i>Df(3L)vin3</i>	<i>Df(3L)vin2</i>	<i>l(3)rsg12</i>
<i>l(3)68Ec</i>	3-[36]	68D6-E4	<i>Df(3L)vin3</i>	<i>Df(3L)vin2</i>	<i>l(3)rsg13</i>
<i>l(3)68Ed</i>	3-[36]	68D6-E4	<i>Df(3L)vin3</i>	<i>Df(3L)vin2</i>	<i>l(3)rsg14</i>
<i>l(3)68Ee</i>	3-[36]	68D6-E4	<i>Df(3L)vin3</i>	<i>Df(3L)vin2</i>	<i>l(3)rsg15</i>
<i>l(3)68Ef</i>	3-[37]	68E3-4	<i>Df(3L)P20</i>	<i>Df(3L)vin3</i>	<i>l(3)rsg16</i>
<i>l(3)68Eg</i>	3-[37]	68E3-4	<i>Df(3L)P20</i>	<i>Df(3L)vin3</i>	<i>l(3)rsg17</i>

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(3)68Eb</i>¹	EMS		<i>l(3)I07</i>	2	
<i>l(3)68Ec</i>¹	EMS	Meyerowitz	<i>l(3)I06.19</i>	2	
<i>l(3)68Ed</i>¹	EMS		<i>l(3)5</i>	2	
<i>l(3)68Ee</i>¹	EMS		<i>l(3)I37</i>	2	
<i>l(3)68Ee</i>²	EMS		<i>l(3)I41A</i>	2	
<i>l(3)68Ef</i>¹	EMS		<i>l(3)I6-2</i>	2	
<i>l(3)68Eg</i>¹	X ray		<i>l(3)P20</i>	2	associated with <i>Trp(3;3)Ubx^{P20}</i>

^α 1 = Akam, Roberts, Richards, and Ashburner, 1978, Cell 13: 215-25; 2 = Hoogwerf, Akam, and Roberts, 1988, Genetics 118: 665-70.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(3)68Fa</i>	3-[37]	68E3-6	<i>Df(3L)vin4</i>	<i>Df(3L)P20</i>	<i>l(3)rsg18</i>
<i>l(3)68Fb</i>	3-[37]	68E3-6	<i>Df(3L)vin4</i>	<i>Df(3L)P20</i>	<i>l(3)rsg19</i>
<i>l(3)68Fc</i>	3-[37]	68E3-6	<i>Df(3L)vin4</i>	<i>Df(3L)P20</i>	<i>l(3)rsg20</i>
<i>l(3)68Fd</i>	3-[37]	68E3-6	<i>Df(3L)vin4</i>	<i>Df(3L)P20</i>	<i>l(3)rsg21</i>
<i>l(3)68Fe</i>	3-[37]	68E3-6	<i>Df(3L)vin8</i>	<i>Df(3L)vin4</i>	<i>l(3)rsg22</i>
<i>l(3)68Ff</i>	3-[37]	68E3-6	<i>Df(3L)vin8</i>	<i>Df(3L)vin4</i>	<i>l(3)rsg23</i>

allele	origin	discoverer	synonym	ref ^α
<i>l(3)68Fa</i>¹	EMS		<i>l(3)F11</i>	2
<i>l(3)68Fa</i>²	EMS		<i>l(3)V4-1</i>	1, 2
<i>l(3)68Fa</i>³	EMS		<i>l(3)V4-5^{ts}</i>	1, 2
<i>l(3)68Fa</i>⁴	ICR170	Shearn	<i>l(3)V11-5</i>	2, 3
<i>l(3)68Fb</i>¹	EMS		<i>l(3)J49</i>	2
<i>l(3)68Fc</i>¹	EMS		<i>l(3)B1P</i>	2
<i>l(3)68Fc</i>²	spont		<i>l(3)igh</i>	2
<i>l(3)68Fd</i>¹	EMS		<i>l(3)IB</i>	2
<i>l(3)68Fe</i>¹	EMS		<i>l(3)69</i>	2
<i>l(3)68Fe</i>²	EMS	Meyerowitz	<i>l(3)I12.226</i>	2
<i>l(3)68Fe</i>³	EMS		<i>l(3)I13</i>	2
<i>l(3)68Ff</i>¹	EMS		<i>l(3)J21-2</i>	2
<i>l(3)68Ff</i>²	EMS	Meyerowitz	<i>l(3)I12.139</i>	2
<i>l(3)68Ff</i>³	EMS	Meyerowitz	<i>l(3)I12.143</i>	2
<i>l(3)68Ff</i>⁴	EMS	Meyerowitz	<i>l(3)I12.186</i>	2
<i>l(3)68Ff</i>⁵	EMS	Meyerowitz	<i>l(3)I12.25</i>	2

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	synonym	ref ^α
<i>I(3)68Ff</i>⁶	EMS		<i>l(3)D</i> ¹¹	2

^α 1 = Akam, Roberts, Richards, and Ashburner, 1978, Cell 13: 215-25;
2 = Hoogwerf, Akam, and Roberts, 1988, Genetics 118: 665-70; 3 = Shearn, 1974, Genetics 77: 115-25.

DEFICIENCY MAP OF REGION 68

side	breakpoint	variant ^α	variant ^β
left	67E	<i>Df(3L)lxd15</i>	
left		<i>Df(3L)lxd6</i>	
left	67F	<i>Df(3L)P20</i>	
left	67F2-3	<i>Df(3L)vin2</i>	
		rs	
left	68A2-3	<i>Df(3L)lxd2</i>	
left	68A2-3	<i>Df(3L)lxd8</i>	
left	68A2-3	<i>Df(3L)vin5</i>	
left	68A2-3	<i>Df(3L)vin66</i>	
		<i>I(3)68Aa</i>	
		<i>I(3)68Ab</i>	
left	68A3-4		<i>Df(3L)h1</i>
left	68A3-4	<i>Df(3L)lxd9</i>	<i>Df(3L)lxd9</i>
		<i>I(3)68Ac</i>	
			<i>H = I(3)68Ac</i> ⁻ (1)
left	68A3-4		<i>Df(3L)h5</i>
left	68A3-4		<i>Df(3L)h8</i>
left	invisible		<i>Df(3L)h9</i>
		ixd	ixd
left			<i>Df(3L)h2</i>
left			<i>Df(3L)h76</i>
		<i>I(3)68Ad</i>	
			<i>D = I(3)68Ad</i> ⁻ (4)
right	68A5-6	<i>Df(3L)lxd8</i>	
left	invisible		<i>Df(3L)h9</i>
			<i>J = I(3)68Ae</i> ⁻ (1)
			<i>cSod = I(3)68Af</i>⁻(1)
			<i>E = I(3)68Ag</i> ⁻ (4)
		<i>I(3)68Ad</i>	
		<i>I(3)68Ae</i>	
		<i>I(3)68Af</i>	
		<i>I(3)68Ag</i>	
		<i>I(3)68Ah</i>	
		<i>I(3)68Ai</i>	
		<i>I(3)68Aj</i>	
		<i>I(3)68Ak</i>	
		<i>I(3)68Al</i>	
		<i>I(3)68Am</i>	
left	68A8-B1		<i>Df(3L)l-3</i>
right			<i>Df(3L)h76</i>
			<i>A = I(3)68Ba</i> (2)
			<i>B = I(3)68Bb</i> (4)
			<i>I = I(3)68Bc</i> (2)
			<i>F = I(3)68Bd</i> (4)
right			<i>Df(3L)h2</i>
			<i>C = I(3)68Be</i> (4)
left	68A8-9 or 68B1-3?	<i>Df(3L)vin4</i>	
right	68B4-C1		<i>Df(3L)h1</i>
right	68B4-C1	<i>Df(3L)lxd9</i>	<i>Df(3L)lxd9</i>
right	68B4-C1		<i>Df(3L)h5</i>
right	68B4-C1		<i>Df(3L)h8</i>
right	68B4-C1		<i>Df(3L)l-3</i>
right	68C1-2	<i>Df(3L)lxd6</i>	
right		<i>Df(3L)lxd2</i>	
left	68C2-3	<i>Df(3L)vin8</i>	
		Sgs3	
left		<i>Df(3L)BK9</i>	
left		<i>Df(3L)Est6-1</i>	
left	68C5-6	<i>Df(3L)vin9</i>	
left	68C8-11	<i>Df(3L)vin3</i>	
		<i>I(3)68Ca</i>	
left	68C8-11	<i>Df(3L)vin7</i>	
		<i>I(3)68Cb</i>	
		rt	
		<i>I(3)68Cc</i>	

side	breakpoint	variant ^α	variant ^β
left	68C8-11	<i>Df(3L)vin6</i>	
right	68C10-15	<i>Df(3L)lxd15</i>	
		<i>I(3)68Cd</i>	
		<i>I(3)68Ce</i>	
		<i>I(3)68Cf</i>	
		<i>I(3)68Cg</i>	
		<i>I(3)68Ch</i>	
		vin	
right	68D3	<i>Df(3L)vin66</i>	
		<i>I(3)68Da</i>	
		<i>I(3)68Db</i>	
		<i>I(3)68Dc</i>	
right	68D6	<i>Df(3L)vin2</i>	
		<i>I(3)68Ea</i>	
		<i>I(3)68Eb</i>	
		<i>I(3)68Ec</i>	
		<i>I(3)68Ed</i>	
		<i>I(3)68Ee</i>	
right	68E3-4	<i>Df(3L)vin3</i>	
		<i>I(3)68Ef</i>	
		<i>I(3)68Eg</i>	
right	68E3-4	<i>Df(3L)P20</i>	
		<i>I(3)68Fa</i>	
		<i>I(3)68Fb</i>	
		<i>I(3)68Fc</i>	
		<i>I(3)68Fd</i>	
		Lsp2	
right	68F3-6	<i>Df(3L)vin4</i>	
		<i>I(3)68Fe</i>	
left	invisible		<i>Df(3L)vin10</i>
		<i>I(3)68Ff</i>	
right	<i>Df(3L)BK9</i>		
right		<i>Df(3L)Est6-1</i>	
right	68F3-6	<i>Df(3L)vin8</i>	

^α Crosby and Meyerowitz, 1986, Genetics 112: 785-802; Hoogwerf, Akam, and Roberts, 1988, Genetics 118: 665-70.
^β Campbell, Hilliker, and Phillips, 1986, Genetics 112: 205-15; the letters preceding mutant designations are the original symbols used by the authors, and the numbers in parentheses following represent the numbers of alleles recovered. Variants common to the two studies occupy the same lines.

I(3)69A-B

The extension of the saturation mutagenesis investigation of the region around *Lsp* of Hoogwerf, Akam, and Roberts (1988, Genetics 118: 665-70).

locus	genetic location	cytological location	included in	excluded from	synonym
<i>I(3)69Aa</i>	3-[37]	68F3-69A3	<i>Df(3L)vin5</i>	<i>Df(3L)vin8</i>	<i>l(3)rsg24</i>
<i>I(3)69Ab</i>	3-[37]	68F3-69A3	<i>Df(3L)vin5</i>	<i>Df(3L)vin8</i>	<i>l(3)rsg25</i>
<i>I(3)69Ac</i>	3-[38]	69A1-5	<i>Df(3L)vin6</i>	<i>Df(3L)vin5</i>	<i>l(3)rsg26</i>
<i>I(3)69Ad</i>	3-[38]	69A1-5	<i>Df(3L)vin6</i>	<i>Df(3L)vin5</i>	<i>l(3)rsg27</i>
<i>I(3)69Ae</i>	3-[38]	69A4-B1	<i>Df(3L)vin9</i>	<i>Df(3L)vin6</i>	<i>l(3)rsg28</i>
<i>I(3)69Af</i>	3-[38]	69A4-B1	<i>Df(3L)vin9</i>	<i>Df(3L)vin6</i>	<i>l(3)rsg29</i>
<i>I(3)69Ag</i> ^α	3-[38]	69A4-B1	<i>Df(3L)vin9</i>	<i>Df(3L)vin6</i>	<i>l(3)rsg30</i>

^α No mutant allele recovered; lethally-mutable locus inferred from the inability of *Df(3L)vin10* to complement *Df(3L)vin9*, although complementing both *l(3)69Ae* and *l(3)69Af* alleles.

allele	origin	synonym	ref ^α
<i>I(3)69Aa</i>¹	EMS	<i>l(3)145</i>	1
<i>I(3)69Ab</i>¹	EMS	<i>l(3)58-2</i>	1
<i>I(3)69Ac</i>¹	EMS	<i>l(3)163</i>	1
<i>I(3)69Ad</i>¹	EMS	<i>l(3)62-2</i>	1
<i>I(3)69Ae</i>¹	EMS	<i>l(3)N24</i>	1
<i>I(3)69Af</i>¹	EMS	<i>l(3)21b</i>	1

allele	origin	synonym	ref ^α		
α I = Hoogwerf, Akam, and Roberts, 1988, Genetics 118: 665-70.					
locus	genetic location	cytological location	included in	excluded from	synonym
l(3)69Ba	3-{38}	69A5-B5	<i>Df(3L)vin7</i>	<i>Df(3L)vin9</i>	<i>l(3)rsg31</i>
l(3)69Bb	3-{38}	69A5-B5	<i>Df(3L)vin7</i>	<i>Df(3L)vin9</i>	<i>l(3)rsg32</i>
l(3)69Bc	3-{38}	69A5-B5	<i>Df(3L)vin7</i>	<i>Df(3L)vin9</i>	<i>l(3)rsg33</i>
l(3)69Bd	3-{38}	69A5-B5	<i>Df(3L)vin7</i>	<i>Df(3L)vin9</i>	<i>l(3)rsg34</i>

allele	origin	synonym	ref ^α
l(3)69Ba¹	EMS	<i>l(3)74</i>	1
l(3)69Ba²	EMS	<i>l(3)141</i>	1
l(3)69Bb¹	EMS	<i>l(3)12-5</i>	1
l(3)69Bc¹	EMS	<i>l(3)47</i>	1
l(3)69Bd¹	EMS	<i>l(3)13</i>	1

α I = Hoogwerf, Akam, and Roberts, 1988, Genetics 118: 665-70.

DEFICIENCY MAP OF REGION 69A-B

side	breakpoint	variant
right	68F3-6	<i>Df(3L)vin8</i> l(3)69Aa
right	invisible	<i>Df(3L)vin10</i> l(3)69Ab
right	69A1-3	<i>Df(3L)vin5</i> app Est6 l(3)69Ac l(3)69Ad
right	69A4-5	<i>Df(3L)vin6</i> l(3)69Ae l(3)69Af
left	invisible	<i>Df(3L)vin11</i> l(3)69Ag
right	69A5-B1	<i>Df(3L)vin9</i> l(3)69Ba
right	invisible	<i>Df(3L)vin11</i> l(3)69Bb l(3)69Bc l(3)69Bd
right	69B4-5	<i>Df(3L)vin7</i> gv eyg

l(3)70Aa

Four interallelic complementation groups as follows: (alleles 5 and 7) (allele1) (allele3) (allele4); alleles 2 and 6 are noncomplementing except that *l(3)70Aa*⁶ complements *l(3)70Aa*⁴ at 30°.

locus	genetic location	cytological location	included in	synonym
l(3)70Aa	3-40.5	70A2-6	<i>Df(3L)Ly</i>	<i>l(3)ZF1</i>
l(3)70Ab	3-40.5	70A2-6	<i>Df(3L)Ly</i>	<i>l(3)ZF2</i>
l(3)70Ac	3-40.5	70A2-6	<i>Df(3L)Ly</i>	<i>l(3)ZF3</i>

allele	origin	synonym	ref ^α
l(3)70Aa¹	EMS	<i>l(3)ZF1</i> ^{A1}	1
l(3)70Aa²	EMS	<i>l(3)ZF1</i> ^{G1}	1
l(3)70Aa³	EMS	<i>l(3)ZF1</i> ^{G2}	1
l(3)70Aa⁴	EMS	<i>l(3)ZF1</i> ^{F15}	1
l(3)70Aa⁵	EMS	<i>l(3)ZF1</i> ^{F83}	1
l(3)70Aa⁶	EMS	<i>l(3)ZF1</i> ^{F85}	1

allele	origin	synonym	ref ^α
l(3)70Aa⁷	EMS	<i>l(3)ZF1</i> ^{F86}	1
l(3)70Ab¹	EMS	<i>l(3)ZF2</i> ^{F16}	1
l(3)70Ab²	EMS	<i>l(3)ZF2</i> ^{F22}	1
l(3)70Ac¹	EMS	<i>l(3)ZF3</i> ^{A2}	1

α I = Zhimulev and Feldman, 1982, DIS 58: 152.

l(3)72A-D (J.A. Kennison)

Twenty one lethally mutable loci, including *arl*, *brm* and *th* defined by complementation analysis of 95 ethyl-methanesulfonate-induced recessive lethal mutations uncovered by *Df(3L)th102* (Kennison and Brizuela).

locus	genetic location	cytological location	included in	excluded from	synonym
l(3)72Aa	3-43.0	<i>71F-72B1</i>	<i>Df(3L)th1-2</i>	<i>Df(3L)th117</i>	brm
l(3)72Ab	3-{43}	<i>71F-72B1</i>	<i>Df(3L)th1-2</i>	<i>Df(3L)th117</i>	
l(3)72Ac	3-{43}	<i>71F-72B1</i>	<i>Df(3L)th1-2</i>	<i>Df(3L)th117</i>	
l(3)72Ad	3-{43}	<i>71F-72B1</i>	<i>Df(3L)th1-2</i>	<i>Df(3L)th117</i>	
l(3)72Ae	3-{43}	<i>71F-72B1</i>	<i>Df(3L)th1-2</i>	<i>Df(3L)th117</i>	arl

allele	origin	discoverer	comments
l(3)72Ab¹	EMS	Kennison	
l(3)72Ab²	EMS	Kennison	
l(3)72Ab³	EMS	Kennison	
l(3)72Ab⁴	EMS	Kennison	
l(3)72Ab⁵	EMS	Kennison	
l(3)72Ab⁶	EMS	Kennison	
l(3)72Ac¹	EMS	Kennison	
l(3)72Ac²	EMS	Kennison	
l(3)72Ac³	EMS	Kennison	
l(3)72Ac⁴	EMS	Kennison	
l(3)72Ac⁵	EMS	Kennison	
l(3)72Ac⁶	EMS	Kennison	
l(3)72Ac⁷	EMS	Kennison	
l(3)72Ac⁸	EMS	Kennison	
l(3)72Ad¹	EMS	Kennison	
l(3)72Ad²	EMS	Brizuela	mutant for <i>brm</i>

locus	genetic location	cytological location	included in	excluded from	synonym
l(3)72Da	3-{43}	<i>72D1-5</i>	<i>Df(3L)st-g24</i> <i>Df(3L)brm11</i>	<i>Df(3L)st-e4</i>	
l(3)72Db	3-{43}	<i>72D1-5</i>	<i>Df(3L)st-g24</i> <i>Df(3L)brm11</i>	<i>Df(3L)st-e4</i>	
l(3)72Dc	3-43.2	<i>72D1-5</i>	<i>Df(3L)st-g24</i> <i>Df(3L)brm11</i>	<i>Df(3L)st-e4</i>	th
l(3)72Dd	3-{43}	<i>72D5</i>	<i>Df(3L)st-e4</i> <i>Df(3L)th117</i>	<i>Df(3L)brm11</i>	
l(3)72De	3-{43}	<i>72D5-11</i>	<i>Df(3L)st-e4</i>	<i>Df(3L)st4</i>	
l(3)72Df	3-{43}	<i>72D5-11</i>	<i>Df(3L)st-e4</i>	<i>Df(3L)st4</i>	
l(3)72Dg	3-{43}	<i>72D5-11</i>	<i>Df(3L)st-e4</i>	<i>Df(3L)st4</i>	
l(3)72Dh	3-{43}	<i>72D5-11</i>	<i>Df(3L)st-e4</i>	<i>Df(3L)st4</i>	
l(3)72Di	3-{43}	<i>72D5-11</i>	<i>Df(3L)st-e4</i>	<i>Df(3L)st4</i>	
l(3)72Dj	3-{43}	<i>72D5-11</i>	<i>Df(3L)st-e4</i>	<i>Df(3L)st4</i>	
l(3)72Dk	3-{43}	<i>72D5-11</i>	<i>Df(3L)st-e4</i>	<i>Df(3L)st4</i>	
l(3)72Dl	3-{43}	<i>72D5-11</i>	<i>Df(3L)st-e4</i>	<i>Df(3L)st4</i>	
l(3)72Dm	3-{43}	<i>72D5-11</i>	<i>Df(3L)st-e4</i>	<i>Df(3L)st4</i>	
l(3)72Dn	3-{43}	<i>72D7-11</i>	<i>Df(3L)st4</i>	<i>Df(3L)st-b11</i>	
l(3)72Do	3-{43}	<i>72D7-11</i>	<i>Df(3L)st4</i>	<i>Df(3L)st-b11</i>	
l(3)72Dp	3-{43}	<i>72D10-12</i>	<i>Df(3L)st-b11</i>	<i>Df(3L)th102</i>	

allele	origin	discoverer	comments
<i>I(3)72Da</i> ¹	EMS	Kennison	
<i>I(3)72Da</i> ²	EMS	Kennison	
<i>I(3)72Da</i> ³	EMS	Kennison	
<i>I(3)72Da</i> ⁴	EMS	Brizuela	semi-lethal
<i>I(3)72Db</i> ¹	EMS	Kennison	
<i>I(3)72Db</i> ²	EMS	Kennison	
<i>I(3)72Db</i> ³	EMS	Kennison	
<i>I(3)72Db</i> ⁴	EMS	Kennison	
<i>I(3)72Db</i> ⁵	EMS	Kennison	
<i>I(3)72Db</i> ⁶	EMS	Kennison	
<i>I(3)72Db</i> ⁷	EMS	Kennison	
<i>I(3)72Db</i> ⁸	EMS	Kennison	
<i>I(3)72Db</i> ⁹	EMS	Brizuela	
<i>I(3)72Db</i> ¹⁰	EMS	Brizuela	
<i>I(3)72Db</i> ¹¹	EMS	Brizuela	
<i>I(3)72Db</i> ¹²	EMS	Brizuela	
<i>I(3)72Db</i> ¹³	EMS	Brizuela	
<i>I(3)72Db</i> ¹⁴	EMS	Brizuela	
<i>I(3)72Db</i> ¹⁵	EMS	Brizuela	
<i>I(3)72Db</i> ¹⁶	EMS	Brizuela	
<i>I(3)72Db</i> ¹⁷	EMS	Brizuela	
<i>I(3)72Db</i> ¹⁸	EMS	Brizuela	
<i>I(3)72Db</i> ¹⁹	EMS	Brizuela	
<i>I(3)72Db</i> ²⁰	EMS	Brizuela	
<i>I(3)72Db</i> ²¹	EMS	Brizuela	
<i>I(3)72Db</i> ²²	EMS	Brizuela	
<i>I(3)72Db</i> ²³	Δ2-3	Kennison	haplo-specific lethal
<i>I(3)72Dd</i> ¹	EMS	Kennison	
<i>I(3)72Dd</i> ²	EMS	Kennison	
<i>I(3)72Dd</i> ³	EMS	Brizuela	
<i>I(3)72Dd</i> ⁴	EMS	Brizuela	
<i>I(3)72Dd</i> ⁵	EMS	Brizuela	
<i>I(3)72Dd</i> ⁶	EMS	Brizuela	
<i>I(3)72De</i> ¹	EMS	Kennison	
<i>I(3)72De</i> ²	EMS	Kennison	
<i>I(3)72De</i> ³	EMS	Kennison	
<i>I(3)72De</i> ⁴	EMS	Kennison	
<i>I(3)72De</i> ⁵	EMS	Kennison	
<i>I(3)72De</i> ⁶	EMS	Brizuela	
<i>I(3)72Df</i> ¹	EMS	Kennison	
<i>I(3)72Df</i> ²	EMS	Kennison	
<i>I(3)72Df</i> ³	EMS	Kennison	
<i>I(3)72Df</i> ⁴	EMS	Kennison	
<i>I(3)72Df</i> ⁵	EMS	Kennison	
<i>I(3)72Df</i> ⁶	EMS	Brizuela	
<i>I(3)72Dg</i> ¹	EMS	Kennison	
<i>I(3)72Dg</i> ²	EMS	Kennison	
<i>I(3)72Dg</i> ³	EMS	Kennison	
<i>I(3)72Dg</i> ⁴	EMS	Kennison	
<i>I(3)72Dh</i> ¹	EMS	Kennison	
<i>I(3)72Di</i> ¹	EMS	Kennison	
<i>I(3)72Dj</i> ¹	EMS	Kennison	
<i>I(3)72Dk</i> ¹	EMS	Brizuela	
<i>I(3)72Di</i> ¹	EMS	Kennison	
<i>I(3)72Di</i> ²	EMS	Brizuela	
<i>I(3)72Dm</i> ¹	EMS	Kennison	
<i>I(3)72Dm</i> ²	EMS	Kennison	
<i>I(3)72Dn</i> ¹	EMS	Kennison	
<i>I(3)72Dn</i> ²	EMS	Brizuela	
<i>I(3)72Do</i> ¹	EMS	Kennison	
<i>I(3)72Dp</i> ¹	EMS	Kennison	
<i>I(3)72Dp</i> ²	EMS	Kennison	

allele	origin	discoverer	comments
<i>I(3)72Dp</i> ³	EMS	Kennison	
<i>I(3)72Dp</i> ⁴	EMS	Kennison	

DEFICIENCY MAP OF REGION 72A-72D

side	breakpoint	variant
left	71F	<i>Df(3L)brm11</i>
left	71F-72A1	<i>Df(3L)th102</i> brm <i>I(3)72Aa</i> <i>I(3)72Ab</i> <i>I(3)72Ac</i> <i>I(3)72Ad</i> ari
left	72B1	<i>Df(3L)th117</i>
left	72C1-D1	<i>Df(3L)st-fl3</i>
left	72D1-2	<i>Df(3L)st-g24</i> <i>I(3)72Da</i> <i>I(3)72Db</i> th
right	72D2-5	<i>Df(3L)brm11</i>
left	72D5-10	<i>Df(3L)st-er</i> I(3)72Dd
right	72D5	<i>Df(3L)th117</i> <i>I(3)72De</i> <i>I(3)72Df</i> <i>I(3)72Dg</i> <i>I(3)72Dh</i> <i>I(3)72Di</i> <i>I(3)72Dj</i> <i>I(3)72Dk</i> <i>I(3)72Di</i> <i>I(3)72Dm</i>
left	72D7-11	<i>Df(3L)st4</i> I(3)72Dn <i>I(3)72Do</i>
left	72D10-11	<i>Df(3L)st-b11</i> I(3)72Dp
right	72D12	<i>Df(3L)th102</i>

I(3)73

Two groups active in inducing mutations in this region; McKeown and Belote in their studies of *tra*, and Hoffman in his investigation of *Abl* (Belote, Hoffmann, McKeown, Chorsky, and Baker, 1990, Genetics 125: 783-93).

locus	genetic		included in	excluded from	synonym
	location	location			
<i>I(3)73Aa</i>	3-{44}	73A2-3	<i>Df(3L)st-fl3</i>	<i>Df(3L)st-a20</i>	<i>I(3)std6</i>
<i>I(3)73Ab</i>	3-{44}	73A3-4	<i>Df(3L)st-a20</i> <i>Df(3L)st-fl3</i>		<i>I(3)133.37</i>
<i>I(3)73Ac</i>	3-{44}	73A4-7	<i>Df(3L)st7P</i>	<i>Df(3L)st-fl3</i>	<i>I(3)std2</i>
<i>I(3)73Ad</i>	3-{44}	73A4-7	<i>Df(3L)st7P</i>	<i>Df(3L)st-fl3</i>	<i>I(3)std8</i>
<i>I(3)73Ae</i>	3-{44}	73A4-7	<i>Df(3L)st7P</i>	<i>Df(3L)st-fl3</i>	<i>I(3)std18</i>
<i>I(3)73Af</i>	3-{44}	73A4-7	<i>Df(3L)st7P</i>	<i>Df(3L)st-fl3</i>	<i>I(3)133.52</i>
<i>I(3)73Ag</i>	3-{44}	73A4-7	<i>Df(3L)st7P</i>	<i>Df(3L)st-fl3</i>	<i>I(3)133.59</i>
<i>I(3)73Ah</i>	3-{44}	73A7-9	<i>Df(3L)st-E52</i>	<i>Df(3L)st7P</i>	<i>I(3)133.18</i>
<i>I(3)73Ai</i>	3-{44}	73A9-10	<i>Df(3L)st-g24</i>	<i>Df(3L)st-E52</i>	<i>I(3)DT55</i>
<i>I(3)73Aj</i>	3-{44}	73A10-B1	<i>Df(3L)st-j7</i>	<i>Df(3L)st-g24</i>	<i>I(3)std4</i>
<i>I(3)73Ba</i>	3-{44}	73A10-B1	<i>Df(3L)st-E34</i>	<i>Df(3L)st-j7</i>	Abl
<i>I(3)73Bb</i>	3-{44}	73B1-2	<i>Df(3L)st-E34</i>	<i>Df(3L)st-j7</i>	<i>I(3)std10</i>
<i>I(3)73Bc</i>	3-{44}	73B1-5	<i>Df(3L)std100.62</i>	<i>Df(3L)st-E34</i>	<i>I(3)std5</i>
<i>I(3)73Bd</i>	3-{44}	73C1	<i>Df(3L)st4</i>	<i>Df(3L)st100.62</i>	
<i>I(3)73Be</i>	3-{44}	73B1-5	<i>Df(3L)st100.62</i>	<i>Df(3L)st4</i>	dab
<i>I(3)73Ca</i>	3-{44}	73C1-D2	<i>Df(3L)st81k</i>	<i>Df(3L)st4</i>	
<i>I(3)73Cd</i>	3-{44}	73C1-D2	<i>Df(3L)st81k</i>	<i>Df(3L)st4</i>	

allele	origin	discoverer	synonym	comments
<i>l(3)73Aa</i>¹	X ray	Belote, McKeown	<i>l(3)std6</i>	P, PA
<i>l(3)73Aa</i>²	X ray	Belote, McKeown	<i>l(3)std12</i>	
<i>l(3)73Aa</i>³	X ray	Belote, McKeown	<i>l(3)std13</i>	
<i>l(3)73Aa</i>⁴	X ray	Belote, McKeown	<i>l(3)std15</i>	
<i>l(3)73Aa</i>⁵	X ray	Belote, McKeown	<i>l(3)std16</i>	
<i>l(3)73Aa</i>⁶	X ray	Belote, McKeown	<i>l(3)std17</i>	<i>T(2;3)40-41;</i> <i>73A2-3</i>
<i>l(3)73Aa</i>⁷	X ray	Belote, McKeown	<i>l(3)std19</i>	
<i>l(3)73Aa</i>⁸	X ray	Belote, McKeown	<i>l(3)std20</i>	
<i>l(3)73Aa</i>⁹	X ray	Belote, McKeown	<i>l(3)std21</i>	
<i>l(3)73Aa</i>¹⁰	EMS	Hoffmann	<i>l(3)132.23</i>	
<i>l(3)73Aa</i>¹¹	EMS	Hoffmann	<i>l(3)133.36</i>	
<i>l(3)73Aa</i>¹²	EMS	Hoffmann	<i>l(3)133.45</i>	
<i>l(3)73Aa</i>¹³	EMS	Hoffmann	<i>l(3)133.7</i>	
<i>l(3)73Aa</i>¹⁴	EMS			
<i>l(3)73Aa</i>¹⁵	EMS			
<i>l(3)73Ab</i>¹	EMS	Hoffmann	<i>l(3)132.10</i>	P, PA
<i>l(3)73Ab</i>²	EMS	Hoffmann	<i>l(3)133.1</i>	
<i>l(3)73Ab</i>³	EMS	Hoffmann	<i>l(3)133.12</i>	
<i>l(3)73Ab</i>⁴	EMS	Hoffmann	<i>l(3)133.37</i>	L3 ^α
<i>l(3)73Ab</i>⁵	EMS	Hoffmann	<i>l(3)134.22</i>	
<i>l(3)73Ab</i>⁶	EMS	Hoffmann	<i>l(3)134.43</i>	
<i>l(3)73Ab</i>⁷	EMS			
<i>l(3)73Ab</i>⁸	EMS			
<i>l(3)73Ac</i>¹	X ray	Belote, McKeown	<i>l(3)std2</i>	L
<i>l(3)73Ac</i>²	EMS	Hoffmann	<i>l(3)133.34</i>	
<i>l(3)73Ac</i>³	EMS			
<i>l(3)73Ac</i>⁴	EMS			
<i>l(3)73Ac</i>⁵	EMS			
<i>l(3)73Ad</i>¹	X ray	Belote, McKeown	<i>l(3)std8</i>	L
<i>l(3)73Ae</i>¹	X ray	Belote, McKeown	<i>l(3)std18</i>	L
<i>l(3)73Af</i>¹	EMS	Hoffmann	<i>l(3)133.52</i>	L
<i>l(3)73Af</i>²	EMS			
<i>l(3)73Af</i>³	EMS			
<i>l(3)73Af</i>⁴	EMS			
<i>l(3)73Ag</i>¹	EMS	Hoffmann	<i>l(3)133.59</i>	P, PA; tergites fail to develop
<i>l(3)73Ah</i>¹	EMS	Hoffmann	<i>l(3)133.18</i>	L3, P
<i>l(3)73Ai</i>¹	EMS	Suzuki	<i>l(3)DTS5</i>	L-P; <i>hfw</i> -like; dominant temperature sensitive
<i>l(3)73Ai</i>²	EMS	Hoffmann	<i>l(3)133.16</i>	L
<i>l(3)73Ai</i>³	EMS	Hoffmann	<i>l(3)133.29</i>	L
<i>l(3)73Aj</i>¹	X ray	Belote, McKeown	<i>l(3)std4</i>	E; small deletion
<i>l(3)73Bb</i>¹	X ray	Belote, McKeown	<i>l(3)std10</i>	E; <i>T(Y;2;3)Y;</i> <i>44C2-4;</i> <i>54B10-15;</i> <i>73B1-2</i>
<i>l(3)73Bb</i>²	EMS			
<i>l(3)73Bb</i>³	EMS			
<i>l(3)73Bb</i>⁴	EMS			
<i>l(3)73Bc</i>¹	X ray	Belote, McKeown	<i>l(3)std5</i>	E
<i>l(3)73Bc</i>²	EMS			
<i>l(3)73Bd</i>¹	EMS			
<i>l(3)73Bd</i>²	EMS			
<i>l(3)73Bd</i>³	EMS			
<i>l(3)73Ca</i>¹	EMS			

^α Abnormal mitosis; sex transformation in *tra2/+*; *tra/l(3)73Ab*⁴ females (Andrew, 1987, PhD Thesis, University of California, San Diego).

β Y^P |54B10—44C4|54B15—60.
Y^D |73B2—100|21—44C2|73B1—61.

DEFICIENCY MAP OF REGION 73A

side	breakpoint	variant	DNA ^α coordinates
	73A2-3	<i>l(1)73Aa</i>	< -90
left	73A3	<i>Df(3L)st-a20</i>	-40
	73A3-4	<i>l(3)73Ab</i>	
left	73A3-4	<i>In(3LR)st-a27</i>	0
	73A3-4	st	0
right	73A3-4	<i>Df(3L)st-fl3</i>	
left	73A4	<i>Df(3L)tra</i>	
right	73A3-4	<i>Df(3L)st-a20</i>	
right	73A5	<i>Df(3L)st-e4</i>	60
	73A5-9	<i>l(3)73Ac</i>	
	73A5-9	<i>l(3)73Ad</i>	
	73A5-9	<i>l(3)73Ae</i>	
	73A5-9	<i>l(3)73Af</i>	
right	73A5-9	<i>l(3)73Ag</i>	
right	73A7-9	<i>Df(3L)st7P</i>	80
	73A7-10	<i>l(3)73Ah</i>	
	73A7-10	tra	
right	73A7-9	<i>Df(3R)st-E52</i>	90
		<i>l(3)73Ai</i>	
	73A10-B1	<i>mfs(3)73A</i>	
right		<i>Df(3L)st-g24</i>	105
	73A10-B1	<i>l(3)73Aj</i>	
3'		<i>Abl</i>	
left	73A10-11	<i>Df(3R)std11</i>	
right	73B1-2	<i>Df(3R)st-E36</i>	110
right	73B1-2	<i>Df(3R)st-j7</i>	140
5'		<i>Abl</i>	
	73B1-2	<i>l(3)73Bb</i>	
right	73B3	<i>Df(3L)st-E34</i>	
	73B1-5	<i>l(3)73Bc</i>	
right	73B3-5	<i>Df(3L)st62</i>	
right	73B4	<i>Df(3L)st8P</i>	
left		dab	
right	73C1-2	<i>Df(3L)st100;62</i>	
right		dab	
		<i>l(3)73Bd</i>	
right	73C1	<i>Df(3L)st4</i>	
		<i>l(3)73Ca</i>	
		<i>l(3)73Cd</i>	
		db	
		<i>plk</i>	
right	73D1-2	<i>Df(3L)st81k</i>	

^α Coordinates in kilobases from the left breakpoint of *In(3L)st-a27*.

l(3)80F-81F

A series of ethyl-methanesulfonate-induced lethals detected by failure to complement proximal heterochromatic deficiencies in *3L* or *3R*, which resulted from detachments, *i.e.*, reconstitutions of normal third chromosomes, from irradiated *C(3L)RM/C(3R)RM*-bearing females; a large set of such proximal heterochromatic deficiencies was employed in deficiency mapping of these lethals (Marchant and Holm, 1988, Genetics 120: 519-32).

locus	genetic location	included in	excluded from	synonym
<i>l(3)80Fa</i>	3-[47]	<i>Df(3L)I-16</i>	<i>Df(3L)J5-84</i>	<i>l(3)Lh8</i>
<i>l(3)80Fb</i>	3-[47]	<i>Df(3L)J5-84</i>	<i>Df(3L)J10-58</i>	<i>l(3)Lh7</i>
<i>l(3)80Fc</i>	3-[47]	<i>Df(3L)J10-58</i>	<i>Df(3L)J10-39</i>	<i>l(3)Lh6</i>
<i>l(3)80Fd</i>	3-[47]	<i>Df(3L)J10-39</i>	<i>Df(3L)J8A-80</i>	<i>l(3)Lh5</i>
<i>l(3)80Fe</i>	3-[47]	<i>Df(3L)J10-39</i>	<i>Df(3L)J8A-80</i>	<i>l(3)Lh5</i>
<i>l(3)80Ff</i>	3-[47]	<i>Df(3L)J8A-80</i>	<i>Df(3L)J1-166</i>	<i>l(3)Lh4B</i>
<i>l(3)80Fg</i>	3-[47]	<i>Df(3L)J1-166</i>	<i>Df(3L)J2-66</i>	<i>l(3)Lh4A</i>
<i>l(3)80Fh</i>	3-[47]	<i>Df(3L)J2-66</i>	<i>Df(3L)J9-56</i>	<i>l(3)Lh3</i>
<i>l(3)80Fi</i>	3-[47]	<i>Df(3L)J9-56</i>	<i>Df(3L)J3-9</i>	<i>l(3)Lh2</i>
<i>l(3)80Fj</i>	3-[47]	<i>Df(3L)J3-9</i>		<i>l(3)Lh1</i>

THE GENOME OF *DROSOPHILA MELANOGASTER*

locus	genetic			synonym
	location	included in	excluded from	
<i>l(3)81Fa</i> ¹	3-[47]	<i>Df(3R)10-65</i>		<i>l(3)Rh1</i>
<i>l(3)81Fb</i>	3-[47]	<i>Df(3)4-7</i>	<i>Df(3R)10-65</i>	<i>l(3)Rh2</i>

allele	origin	synonym	comments	^α
<i>l(3)80Fa</i> ¹	EMS	1-16-2		
<i>l(3)80Fa</i> ²	EMS	1-16-10		
<i>l(3)80Fa</i> ³	EMS	1-16-30		
<i>l(3)80Fb</i> ¹	EMS	1-16-29	group 1	
<i>l(3)80Fb</i> ²	EMS	1-16-36	group 2 + 3	
<i>l(3)80Fb</i> ³	EMS	1-16-37	group 1	
<i>l(3)80Fb</i> ⁴	EMS	1-16-38	group 2	
<i>l(3)80Fb</i> ⁵	EMS	1-16-40	group 1 + 2	
<i>l(3)80Fc</i> ¹	EMS	1-16-18		
<i>l(3)80Fc</i> ²	EMS	1-16-28		
<i>l(3)80Fd</i> ¹	EMS	1-16-16		
<i>l(3)80Fd</i> ²	EMS	1-16-19		
<i>l(3)80Fd</i> ³	EMS	1-16-26		
<i>l(3)80Fd</i> ⁴	EMS	1-16-34		
<i>l(3)80Fe</i> ¹	EMS	1-16-27		
<i>l(3)80Ff</i> ¹	EMS	1-16-4		
<i>l(3)80Ff</i> ²	EMS	1-16-7		
<i>l(3)80Ff</i> ³	EMS	1-16-8		
<i>l(3)80Ff</i> ⁴	EMS	1-16-11		
<i>l(3)80Ff</i> ⁵	EMS	1-16-12		
<i>l(3)80Ff</i> ⁶	EMS	1-16-13		
<i>l(3)80Ff</i> ⁷	EMS	1-16-17		
<i>l(3)80Ff</i> ⁸	EMS	1-16-20		
<i>l(3)80Ff</i> ⁹	EMS	1-16-21		
<i>l(3)80Ff</i> ¹⁰	EMS	1-16-22		
<i>l(3)80Ff</i> ¹¹	EMS	1-16-23		
<i>l(3)80Ff</i> ¹²	EMS	1-16-24		
<i>l(3)80Ff</i> ¹³	EMS	1-16-25		
<i>l(3)80Ff</i> ¹⁴	EMS	1-16-31		
<i>l(3)80Ff</i> ¹⁵	EMS	1-16-32		
<i>l(3)80Ff</i> ¹⁶	EMS	1-16-35		
<i>l(3)80Ff</i> ¹⁷	EMS	1-16-41		
<i>l(3)80Ff</i> ¹⁸	EMS	1-16-43		
<i>l(3)80Ff</i> ¹⁹	EMS	1-16-44		
<i>l(3)80Ff</i> ²⁰	EMS	1-16-45		
<i>l(3)80Fg</i> ¹	EMS	1-16-15		
<i>l(3)80Fg</i> ²	EMS	1-16-42		
<i>l(3)80Fg</i> ³	EMS	1-166-5	heat-sensitive allele	
<i>l(3)80Fg</i> ⁴	EMS	1-166-8		
<i>l(3)80Fg</i> ⁵	EMS	1-166-9		
<i>l(3)80Fg</i> ⁶	EMS	1-166-14		
<i>l(3)80Fg</i> ⁷	EMS	1-166-18		
<i>l(3)80Fg</i> ⁸	EMS	1-166-26		
<i>l(3)80Fg</i> ⁹	EMS	1-166-32		
<i>l(3)80Fg</i> ¹⁰	EMS	1-166-44		
<i>l(3)80Fh</i> ¹	EMS	1-166-38		
<i>l(3)80Fh</i> ²	EMS	1-166-39		
<i>l(3)80Fi</i> ¹	EMS	1-166-13		
<i>l(3)80Fi</i> ²	EMS	1-166-33		
<i>l(3)80Fi</i> ³	EMS	1-166-37		
<i>l(3)80Fj</i> ¹	EMS	1-166-1		
<i>l(3)80Fj</i> ²	EMS	1-166-2		
<i>l(3)80Fj</i> ³	EMS	1-166-3		
<i>l(3)80Fj</i> ⁴	EMS	1-166-22		
<i>l(3)80Fj</i> ⁵	EMS	1-166-27		
<i>l(3)80Fj</i> ⁶	EMS	1-166-29		
<i>l(3)80Fj</i> ⁷	EMS	1-166-34		
<i>l(3)80Fj</i> ⁸	EMS	1-166-40		
<i>l(3)80Fj</i> ⁹	EMS	1-166-45		

allele	origin	synonym	comments	^α
<i>l(3)81Fa</i> ¹	EMS	4-75-12		
<i>l(3)81Fa</i> ²	EMS	4-75-17		
<i>l(3)81Fa</i> ³	EMS	10-65-2		
<i>l(3)81Fa</i> ⁴	EMS	10-65-6		

<i>l(3)81Fb</i> ¹	EMS	4-75-3	group 1	
<i>l(3)81Fb</i> ²	EMS	4-75-4	group 1 + 2	
<i>l(3)81Fb</i> ³	EMS	4-75-5	group 2	
<i>l(3)81Fb</i> ⁴	EMS	4-75-6	group 1 + 2	
<i>l(3)81Fb</i> ⁵	EMS	4-75-7	group 1 + 2	
<i>l(3)81Fb</i> ⁶	EMS	4-75-9	group 2	
<i>l(3)81Fb</i> ⁷	EMS	4-75-13	group 1 + 2	
<i>l(3)81Fb</i> ⁸	EMS	4-75-14	group 1 + 2	
<i>l(3)81Fb</i> ⁹	EMS	4-75-15	group 2	
<i>l(3)81Fb</i> ¹⁰	EMS	4-75-16	group 2	

^α Complementation groups indicated for cases of interallelic complementation.

DEFICIENCY MAP OF REGION 80F

side	variant
left	<i>Df(3L)1-16</i>
left	<i>Df(3L)1-104</i>
left	<i>Df(3L)8-15</i>
left	<i>Df(3L)9-52</i>
	<i>l(3)80Fa</i>
left	<i>Df(3L)2-85</i>
left	<i>Df(3L)3-52</i>
left	<i>Df(3L)5-84</i>
	<i>l(3)80Fb</i>
left	<i>Df(3L)10-58</i>
left	<i>Df(3L)10-33</i>
	<i>l(3)80Fc</i>
left	<i>Df(3L)1-17</i>
left	<i>Df(3L)3-10</i>
left	<i>Df(3L)3-109</i>
left	<i>Df(3L)3-135</i>
left	<i>Df(3L)3-164</i>
left	<i>Df(3L)4-134</i>
left	<i>Df(3L)6-53</i>
left	<i>Df(3L)6-61</i>
left	<i>Df(3L)8-68</i>
left	<i>Df(3L)9-2</i>
left	<i>Df(3L)9-7</i>
left	<i>Df(3L)10-14</i>
left	<i>Df(3L)10-39</i>
left	<i>Df(3L)10-210</i>
left	<i>Df(3L)12-2</i>
left	<i>Df(3L)12-123</i>
right	<i>Df(3L)10-33</i>
	<i>l(3)80Fd</i>
	<i>l(3)80Fe</i>
left	<i>Df(3L)4A-6</i>
left	<i>Df(3L)6-21</i>
left	<i>Df(3L)8A-80</i>
left	<i>Df(3L)9-37</i>
left	<i>Df(3L)10-26</i>
left	<i>Df(3L)10-109</i>
right	<i>Df(3L)1-104</i>
right	<i>Df(3L)2-85</i>
right	<i>Df(3L)3-109</i>
right	<i>Df(3L)3-164</i>
right	<i>Df(3L)5-84</i>
right	<i>Df(3L)6-53</i>
right	<i>Df(3L)6-61</i>
right	<i>Df(3L)9-2</i>
right	<i>Df(3L)9-7</i>
right	<i>Df(3L)9-52</i>
right	<i>Df(3L)10-14</i>
right	<i>Df(3L)10-39</i>
right	<i>Df(3L)10-210</i>
right	<i>Df(3L)12-2</i>
right	<i>Df(3L)12-123</i>
	<i>l(3)80Ff</i>

side	variant
left	<i>Df(3L)1-166</i>
left	<i>Df(3L)6-18</i>
	I(3)80Fg
left	<i>Df(3L)1-166-12</i> ^α
left	<i>Df(3L)1-166-46</i> ^α
left	<i>Df(3L)2-66</i>
left	<i>Df(3L)3-30</i>
left	<i>Df(3L)4-76</i>
left	<i>Df(3L)4-184</i>
left	<i>Df(3L)4A-48</i>
right	<i>Df(3L)1-16</i>
right	<i>Df(3L)1-17</i>
right	<i>Df(3L)3-10</i>
right	<i>Df(3L)3-52</i>
right	<i>Df(3L)3-135</i>
right	<i>Df(3L)4-134</i>
right	<i>Df(3L)4A-6</i>
right	<i>Df(3L)8-15</i>
right	<i>Df(3L)8-68</i>
right	<i>Df(3L)8A-80</i>
right	<i>Df(3L)10-26</i>
right	<i>Df(3L)10-58</i>
right	<i>Df(3L)10-109</i>
	I(3)80Fh
left	<i>Df(3L)9-10</i>
left	<i>Df(3L)9-56</i>
right	<i>Df(3L)3-30</i>
	I(3)80Fi
left	<i>Df(3L)2-30</i>
left	<i>Df(3L)3-9</i>
left	<i>Df(3L)8A-49</i>
left	<i>Df(3L)10-39</i>
right	<i>Df(3L)1-166-12</i> ^α
right	<i>Df(3L)4-76</i>
right	<i>Df(3L)4-184</i>
right	<i>Df(3L)4A-48</i>
	I(3)80Fj
right	<i>Df(3L)1-166</i>
right	<i>Df(3L)1-166-46</i> ^α
right	<i>Df(3L)2-30</i>
right	<i>Df(3L)2-66</i>
right	<i>Df(3L)3-9</i>
right	<i>Df(3L)8A-49</i>
right	<i>Df(3L)9-10</i>
right	<i>Df(3L)9-56</i>
right	<i>Df(3L)10-39</i>
	CENTROMERE
left	<i>Df(3R)4-75</i>
left	<i>Df(3R)4-75-08</i> ^α
left	<i>Df(3R)5-53</i>
left	<i>Df(3R)7-53</i>
left	<i>Df(3R)7-93</i>
left	<i>Df(3R)10-65</i>
left	<i>Df(3R)10-73</i>
left	<i>Df(3R)10-120</i>
left	<i>Df(3R)12-68</i>
left	<i>Df(3R)12-122</i>
	I(3)81Fa
right	<i>Df(3R)7-53</i>
right	<i>Df(3R)7-93</i>
right	<i>Df(3R)10-65</i>
right	<i>Df(3R)10-73</i>
right	<i>Df(3R)10-120</i>
right	<i>Df(3R)12-68</i>
right	<i>Df(3R)12-122</i>
	I(3)81Fb
right	<i>Df(3R)4-75</i>
right	<i>Df(3R)4-75-08</i> ^α
right	<i>Df(3R)5-53</i>

^α Deletions induced in normal third chromosomes with ethyl methanesulfonate.

I(3)84

Comprised primarily of components of the Antennapedia Complex, *ANTC*.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(3)84Aa</i>	3-{47}	84A1-6	<i>Df(3R)Scr</i>	<i>Df(3R)Antp17</i>	twr
I(3)84Ab	3-{47}	84A1-6	<i>Df(3R)Scr</i>	<i>Df(3R)Antp17</i>	<i>cel</i>
<i>l(3)84Ac</i>	3-{47}	84A1-6	<i>Df(3R)Scr</i>	<i>Df(3R)Antp17</i>	lab
<i>l(3)84Ad</i>	3-{47}	84A1-6	<i>Df(3R)Scr</i>	<i>Df(3R)Antp17</i>	zen
<i>l(3)84Ae</i>	3-47.5	84A6-B1	<i>Df(3R)Scr</i>	<i>Df(3R)Hu</i>	Dfd
			<i>Df(3R)Antp17</i>		
<i>l(3)84Af</i>	3-47.5	84A6-B1	<i>Df(3R)Scr</i>		Scr
			<i>Df(3R)Antp17</i>		
<i>l(3)84Ag</i>	3-47.5	84A6-B1	<i>Df(3R)Scr</i>	<i>Df(3R)Hu</i>	ftz
			<i>Df(3R)Antp17</i>		

I(3)84Ab

phenotype: Cell lethal; dies as normal-appearing second instar larva.

references: Lewis, Wakimoto, Denell, and Kaufman, 1980, *Genetics* 95: 367-81.

alleles:

allele	origin	discoverer	synonym
I(3)84Ab ¹	EMS	Wakimoto	<i>EjW1</i>
I(3)84Ab ²	EMS	Wakimoto	<i>EjW14</i>
I(3)84Ab ³	X ray	Abbot	<i>a12</i>
I(3)84Ab ⁴	EMS	Lambert	<i>c29</i>
I(3)84Ab ⁵	EMS	Lambert	<i>c59</i>
I(3)84Ab ⁶	EMS	Fornili	<i>f32</i>
I(3)84Ab ⁷	EMS	Fornili	<i>f35</i>
I(3)84Ab ⁸	EMS	Fornili	<i>f39</i>
I(3)84Ab ⁹	EMS	Fornili	<i>f44</i>
I(3)84Ab ¹⁰	EMS	Fornili	<i>f45</i>
I(3)84Ab ¹¹	EMS	Fornili	<i>f67</i>
I(3)84Ab ¹²	EMS	Fornili	<i>f72</i>
I(3)84Ab ¹³	EMS	Fornili	<i>f77</i>
I(3)84Ab ¹⁴	X ray	Lopez	<i>L5</i>
I(3)84Ab ¹⁵	EMS	Merrill	<i>V11</i>

I(3)84B-C

The region within *Df(3R)Antp17*; subjected to eight saturation mutagenesis experiments; mutants recovered subject to deficiency and complementation mapping.

references: Lewis, Kaufman, Denell, and Tallero, 1980, *Genetics* 95: 367-81.

Cavener, Otterson, and Kaufman, 1986, *Genetics* 114: 111-23.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(3)84Ba</i>	3-47.5	84B1-2	<i>Df(3R)A41</i>		Antp
			<i>Df(3R)A3</i>		
I(3)84Bb	3-{47}	84B2-C2	<i>Df(3R)30c76</i>	<i>Df(3R)Scr</i>	
I(3)84Bc	3-{47}	84B2-C2	<i>Df(3R)Win3</i>	<i>Df(3R)30c76</i>	
<i>l(3)84Bd</i>	3-47.8	84B2-C2	<i>Df(3R)Scx2</i>	<i>Df(3R)Win3</i>	^α Tub84B
<i>l(3)84Be</i>	3-{47}	84B2-C2	<i>Df(3R)Scx2</i>	<i>Df(3R)Win3</i>	stk

allele	origin	discoverer	synonym
I(3)84Bb ¹	EMS	Denell	<i>l(3)d5</i>
I(3)84Bb ²	EMS	Kaufman	<i>l(3)k3</i>
I(3)84Bb ³	EMS	Kaufman	<i>l(3)k12</i>
I(3)84Bb ⁴	EMS	R. Lewis	<i>l(3)r7</i>
I(3)84Bb ⁵	EMS	R. Lewis	<i>l(3)r10</i>
I(3)84Bb ⁶	EMS	R. Lewis	<i>l(3)r15</i>
I(3)84Bc ¹	EMS	R. Lewis	<i>l(3)r14</i>

THE GENOME OF *DROSOPHILA MELANOGASTER*

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(3)84Ca</i>	3-[47]	84C1-6	<i>Df(3R)Antp1</i>	<i>Df(3R)Scx2</i>	hat
<i>l(3)84Cb</i>	3-[47]	84C1-6	<i>Df(3R)Antp1</i>	<i>Df(3R)Scx2</i>	
<i>l(3)84Cc</i>	3-[47]	84C1-6	<i>Df(3R)Antp1</i>	<i>Df(3R)Scx2</i>	
<i>l(3)84Cd</i>	3-[47]	84C1-6	<i>Df(3R)Antp1</i>	<i>Df(3R)Scx2</i>	sas
<i>l(3)84Ce</i>	3-[47]	84C5-8	<i>Df(3R)A40</i>	<i>Df(3R)Antp1</i> <i>Df(3R)dsx29</i>	
<i>l(3)84Cf</i>	3-[47]	84C5-8	<i>Df(3R)A40</i>	<i>Df(3R)Antp1</i> <i>Df(3R)dsx29</i>	ted
<i>l(3)84Cg</i>	3-48	84C8-D1	<i>Df(3R)dsx29</i>	<i>Df(3R)D6</i>	Gld

l(3)84Cb

phenotype: Pupal lethal.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(3)84Cb</i>¹	EMS	Cavener	<i>l(3)g6</i>	1	complements <i>l(3)84Cb</i> ³
<i>l(3)84Cb</i>²	EMS	R. Lewis	<i>l(3)r8</i>	1	non-complementing allele
<i>l(3)84Cb</i>³	EMS	R. Lewis	<i>l(3)r15</i>	1	complements <i>l(3)84Cb</i> ¹

^α 1 = Cavener, Otteson, and Kaufman, 1986, Genetics 114: 111-23.

l(3)84Cc

phenotype: Pupal lethal.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(3)84Cc</i>¹	EMS	Cavener	<i>l(3)g4</i>	1	
<i>l(3)84Cc</i>²	EMS		<i>l(3)k9</i>	1	
<i>l(3)84Cc</i>³	EMS	R. Lewis	<i>l(3)r6</i>	1	

^α 1 = Cavener, Otteson, and Kaufman, 1986, Genetics 114: 111-23.

l(3)84Ce

phenotype: *l(3)84Ce*¹ partially complements both *l(3)84Ce*² and *Df(3R)dsx2*; *l(3)84Ce*¹/*Df(3R)dsx2* eclose successfully, but cannot fly or walk normally. *l(3)84Ce*²/*Df(3R)Scx4* exhibit half-out phenotype characteristic of *hat*.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(3)84Ce</i>¹	EMS	Grigliatti	<i>l(3)j2.1</i>	1	partially complements <i>l(3)84Ce</i> ²
<i>l(3)84Ce</i>²	EMS	Kaufman	<i>l(3)k2</i>	1	

^α 1 = Cavener, Otteson, and Kaufman, 1986, Genetics 114: 111-23.

l(3)84D-F

The region within *Df(3R)dsx2D* subjected to saturation mutagenesis with ethyl methanesulfonate by Baker, Hoff, Kaufman, Wolfner, and Hazelrigg (Genetics, in press).

locus	genetic location	cytological location	included in	excluded from
<i>l(3)84Da</i>	3-[48]	84D11-12	<i>Df(3R)dsx2D</i>	<i>Df(3R)10D</i>
<i>l(3)84Db</i>	3-[48]	84D13-14	<i>Df(3R)dsx43</i> <i>Df(3R)Antp17</i>	
<i>l(3)84Dc</i>	3-[48]	84D13-14	<i>Df(3R)dsx43</i> <i>Df(3R)Antp17</i>	
<i>l(3)84Dd</i>	3-[48]	84D13-14	<i>Df(3R)dsx43</i> <i>Df(3R)Antp17</i>	

l(3)84Da

phenotype: Hemizygotes for *l(3)84Da*¹ semilethal when the test deficiency extends distally as far as 84F6-7 [e.g. *Df(3R)dsx29*] and lethal when the deficiency extends as far as 84F16 [e.g. *Df(3R)dsx2D*]. *l(3)84Da*² not extensively tested for this behavior.

alleles:

allele	synonym
<i>l(3)84Da</i>¹	k5
<i>l(3)84Da</i>²	k21
<i>l(3)84Db</i>¹	bD10
<i>l(3)84Db</i>²	bR24
<i>l(3)84Db</i>³	cK1
<i>l(3)84Db</i>⁴	dK4
<i>l(3)84Db</i>⁵	dR9
<i>l(3)84Db</i>⁶	dR11
<i>l(3)84Db</i>⁷	dR23
<i>l(3)84Db</i>⁸	H7
<i>l(3)84Db</i>⁹	H23
<i>l(3)84Db</i>¹⁰	Ns+R72
<i>l(3)84Dc</i>¹	bR4
<i>l(3)84Dc</i>²	bR14
<i>l(3)84Dc</i>³	cK3
<i>l(3)84Dc</i>⁴	K25
<i>l(3)84Dd</i>¹	H34

l(3)84E

locus	genetic location	cytological location	included in	excluded from
<i>l(3)84Ea</i>	3-[48]	84E1-8	<i>Df(3R)dsx43</i>	<i>Df(3R)dsx2M</i>
<i>l(3)84Eb</i>	3-[48]	84E1-8	<i>Df(3R)dsx43</i>	<i>Df(3R)dsx2M</i>
<i>l(3)84Ec</i>	3-[48]	84E6-9	<i>Df(3R)dsx21</i>	<i>Df(3R)dsx43</i>
<i>l(3)84Ed</i>	3-[48]	84E6-9	<i>Df(3R)dsx21</i>	<i>Df(3R)dsx43</i>
<i>l(3)84Ee</i>	3-[48]	84E6-9	<i>Df(3R)dsx21</i>	<i>Df(3R)dsx43</i>
<i>l(3)84Ef</i>	3-[48]	84E8-9		<i>Df(3R)dsx21</i> <i>Df(3R)p40</i>
<i>l(3)84Eg</i>	3-[48]	84E8-9		<i>Df(3R)dsx21</i> <i>Df(3R)p40</i>
<i>l(3)84Eh</i>	3-[48]	84E8-F1	<i>Df(3R)p40</i>	<i>Df(3R)p13</i>
<i>l(3)84Ei</i>	3-[48]	84E8-F1	<i>Df(3R)p40</i>	<i>Df(3R)p13</i>

allele synonym

<i>l(3)Ea</i>¹	H5
<i>l(3)Ea</i>²	H18
<i>l(3)Ea</i>³	H38
<i>l(3)Ea</i>⁴	H56
<i>l(3)Eb</i>¹	Es
<i>l(3)Eb</i>²	H4
<i>l(3)Eb</i>³	H20
<i>l(3)Eb</i>⁴	K2
<i>l(3)Ec</i>¹	H8
<i>l(3)Ed</i>¹	H13
<i>l(3)Ee</i>¹	H60
<i>l(3)Ef</i>¹	bR5
<i>l(3)Ef</i>²	H36
<i>l(3)Ef</i>³	K1
<i>l(3)Eg</i>¹	H
<i>l(3)Eg</i>²	223
<i>l(3)Eg</i>³	H12
<i>l(3)Eg</i>⁴	H19
<i>l(3)Eg</i>⁵	H24
<i>l(3)Eg</i>⁶	H61
<i>l(3)Eg</i>⁷	K41
<i>l(3)Eh</i>¹	K10
<i>l(3)Eh</i>²	K19
<i>l(3)Eh</i>³	K36
<i>l(3)Ei</i>¹	K40

allele	synonym			
locus	genetic location	cytological location	included in	excluded from
I(3)84Fa	3-{48}	84F1-2	<i>Df(3R)p13</i> <i>Df(3R)dsx10M</i>	
I(3)84Fb	3-{48}	84F1-2	<i>Df(3R)dsx3</i>	<i>Df(3R)dsx10M</i>
I(3)84Fc	3-{48}	84F1-2	<i>Df(3R)dsx3</i>	<i>Df(3R)dsx10M</i>
I(3)84Fd	3-{48}	84F1-2	<i>Df(3R)D7</i>	<i>Df(3R)dsx3</i>
I(3)84Fe	3-{48}	84F1-7	<i>Df(3R)dsx29</i>	<i>Df(3R)10M</i>
I(3)84Ff	3-{48}	84F1-7	<i>Df(3R)dsx29</i>	<i>Df(3R)D7</i>
I(3)84Fg	3-{48}	84F1-7	<i>Df(3R)dsx29</i>	<i>Df(3R)D7</i>
I(3)84Fh	3-{48}	84F10-16	<i>Df(3R)p30</i> <i>Df(3R)D6</i>	
I(3)84Fi	3-{48}	84F10-16	<i>Df(3R)p30</i> <i>Df(3R)D6</i>	
I(3)84Fj	3-{48}	84F10-16	<i>Df(3R)p30</i> <i>Df(3R)D6</i>	
I(3)84Fk	3-{48}	84F13-16	<i>Df(3R)2D</i>	<i>Df(3R)D6</i>
I(3)84Fi	3-{48}	84F13-16	<i>Df(3R)2D</i>	<i>Df(3R)D6</i>
I(3)84Fm	3-{48}	84F13-16	<i>Df(3R)2D</i>	<i>Df(3R)D6</i>

allele	synonym
I(3)84Fa¹	H10
I(3)84Fa²	K18
I(3)84Fb¹	H27
I(3)84Fb²	H28
I(3)84Fb³	H44
I(3)84Fc¹	H40
I(3)84Fd¹	K9
I(3)84Fd²	K13
I(3)84Fd³	K38
I(3)84Fe¹	H17
I(3)84Ff¹	H11
I(3)84Ff²	H57
I(3)84Fh¹	K23
I(3)84Fi¹	K29
I(3)84Fj¹	dR1
I(3)84Fj²	H15
I(3)84Fk¹	H2
I(3)84Fk²	H33
I(3)84Fi¹	dK13
I(3)84Fm¹	434.12
I(3)84Fm²	H37
I(3)84Fm³	K3

DEFICIENCY MAP OF REGION 84

side	breakpoint	variant
left	84A4-5	<i>Df(3R)Antp3</i>
left	84A4-5	<i>Df(3R)Antp4</i>
left	84A1-2	<i>Df(3R)Scr</i>
	84A1-6	twr
	84A1-6	I(3)84Ab
	84A1-6	lab
left	84A4-5	<i>Df(3R)Scx2</i>
	84A4-6	bcd
	84A4-6	pb
	84A1-6	zen
left		<i>Df(3R)29c76+Dp(3;3)Dfd^{rv1}</i>
left		<i>Df(3R)30c76+Dp(3;3)Dfd^{rv1}</i>

side	breakpoint	variant
left	84A6	<i>Df(3R)Antp17</i>
	84A6-B1	Dfd
right	84A6-B1	<i>Df(3R)26b77+Dp(3;3)Dfd^{rv1}</i>
	84A6-B1	Scr
right	84A4-5	<i>Df(3R)29c76+Dp(3;3)Dfd^{rv1}</i>
	84A6-B1	ftz
left	84B1-2	<i>Df(3R)A41</i>
5'	84B1-2	Antp
left		<i>Df(3R)A60</i>
left		<i>Df(3R)IP</i>
right		<i>Df(3R)Scr</i>
left		<i>Df(3R)A75</i>
left		<i>Df(3R)BDR3</i>
right		<i>Df(3R)1274</i>
3'		Antp
right	84B1-2	<i>Df(3R)Antp3</i>
right	84B1-2	<i>Df(3R)Antp4</i>
right	84B1-2	<i>Df(3R)30c76+Dp(3;3)Dfd^{rv1}</i>
right	84B2-3	<i>Df(3R)Antp73a</i>
	84B3	I(3)84Ba
right	84B3-6	<i>Df(3R)30c76</i>
	84B3	I(3)84Bb
right	84B1-2	<i>Df(3R)Win3</i>
	84B1-2	α Tub84B
		mab
left		<i>T(2;3)Ta¹</i>
		stk
left	84C1-2	<i>Df(3R)Antp1</i>
left	84C1-2	<i>Df(3R)dsx2M^a</i>
right	84B1-2	<i>Df(3R)Scx2^a</i>
	84C1-6	hat
right		<i>T(2;3)Ta^L</i>
	84C1-6	I(3)84Cb
	84C1-6	I(3)84Cc
	84C1-6	rue
	84C1-6	sas
right	84C5-6	<i>Df(3R)Antp73b</i>
	84C5-8	I(3)84Ce
	84C5-8	ted
left	84C8-D1	<i>Df(3R)dsx29</i>
	84C8-D1	Gld
right	84D1-2	<i>Df(3R)A41</i>
left	84D2-3	<i>Df(3R)D6</i>
		roe
left	84D3	<i>Df(3R)dsx10M</i>
		rn
		Est-C
left	84D5	<i>Df(3R)D7</i>
left	84D8-9	<i>Df(3R)dsx11</i>
left	84D11	<i>Df(3R)dsx2D</i>
left	84D11-12	<i>Df(3R)dsx21</i>
		I(3)84Da
left	84D11-12	<i>Df(3R)dsx10D</i>
left	84D10-11	<i>Df(3R)dsx15</i>
left	84D11-14	<i>Df(3R)dsx3</i>
left	84D13-14	<i>Df(3R)dsx43</i>
		I(3)84Db
		I(3)84Dc
		I(3)84Dd
right	84D13-14	<i>Df(3R)Antp17</i>
right	84E1-2	<i>Df(3R)dsx2M</i>
		dsx
		I(3)84Ea
		I(3)84Eb
right	84E6-8	<i>Df(3R)dsx43</i>
right	84E8	<i>Df(3R)dsx15</i>
		I(3)84Ec
		I(3)84Ed
		I(3)84Ee
right	84E8-9	<i>Df(3R)dsx21</i>
		I(3)84Ef
		I(3)84Eg
left	84E8-9	<i>Df(3R)p40</i>
		I(3)84Eh
		I(3)84Ei
left	84F1	<i>Df(3R)p13</i>

side	breakpoint	variant
right	84F1-2	I(3)84Fa <i>Df(3R)dsx10M</i>
		I(3)84Fb
		I(3)84Fc
right	84E12-13	<i>Df(3R)dsx3</i>
		I(3)84Fd
right	84F1-2	<i>Df(3R)D7</i>
		I(3)84Fe
		I(3)84Ff
		I(3)84Fg
right	84F6-7	<i>Df(3R)dsx29</i>
left	84F8-10	<i>Df(3R)p30</i>
		I(3)84Fh
		I(3)84Fi
		I(3)84Fj
right	84F13-16	<i>Df(3R)D6</i>
		I(3)84Fk
		I(3)84Fl
		I(3)84Fm
right	84F16	<i>Df(3R)dsx2D</i>

^α *Df(3R)Scx2* and *Df(3R)dsx2M* overlap by ten kb and the *trans* heterozygote is lethal. *T(2;3)Ta^L* breakpoint is in the 10 kb segment and may define a lethally mutable locus between *stk* and *hat*.

I(3)85A

A series of eight lethally mutable loci, all but one of which were first recorded in a saturation mutagenesis screen of the region uncovered by *Df(3R)p25* (Bender, Turner, and Kaufman, 1986, *Dev. Biol.* 119: 418-32). Either ethyl methanesulfonate or diepoxybutane used as the mutagenic agent. Two more loci inferred from the lethality of overlapping deficiencies by Jones and Rawls (1988, *Genetics* 120: 733-42). The right most of these loci is that of *hb*; the rest were previously unrecorded. Deficiency analysis of Bender *et al.* updated by Jones and Rawls with additional deficiencies.

locus	genetic location	cytological location	included in	excluded from	synonym
I(3)85Aa	3-[48]	85A1-4	<i>Df(3R)p25</i>	<i>Df(3R)dsx11</i>	L1
I(3)85Ab	3-[48]	85A3-4		<i>Df(3R)D8</i> <i>Df(3R)Dhod-G1</i>	L2
I(3)85Ac	3-[48]	85A6-11	<i>Df(3R)CA4B</i> <i>Df(3R)CA1</i>		L3
I(3)85Ad	3-[48]	85A6-11	<i>Df(3R)V8</i>	<i>Df(3R)CA1</i>	L4
I(3)85Ae	3-[48]	85A6-11	<i>Df(3R)p16</i>	<i>Df(3R)V8</i>	L5
I(3)85Af	3-[48]	85A6-11	<i>Df(3R)p16</i>	<i>Df(3R)V8</i>	L6
I(3)85Ag	3-[48]	85A6-11	<i>Df(3R)Dhod-G1</i>	<i>Df(3R)p16</i>	L7
<i>l(3)85Ah</i>	3-48	85A6-11	<i>Df(3R)p25</i>	<i>Df(3R)Dhod-G1</i>	hb , <i>Rg</i> , <i>pbx</i>
I(3)85Ai	3-[48]	85A4-5	<i>Df(3R)Dhod-G1</i> <i>Df(3R)V2</i>		
I(3)85Aj	3-[48]	85A6-11	<i>Df(3R)p5</i>		

alleles:

allele
I(3)85Aa¹⁻⁴
I(3)85Ab¹
I(3)85Ac¹⁻⁷
I(3)85Ad¹
I(3)85Ae¹⁻³
I(3)85Af¹⁻⁷
I(3)85Ag¹⁻³

I(3)85D-F

A collection of lethals recovered from saturation mutagenesis screens in three different laboratories (Che-

ney, Matthews, and Maroni).

locus	genetic location	cytological location	included in	excluded from
I(3)85Da	3-[49]	85D8-13	<i>Df(3R)by10</i>	<i>Df(3R)GB104</i>
I(3)85Db	3-[49]	85D11-12	<i>Df(3R)GB104</i>	<i>Df(3R)by416</i>
I(3)85Dc	3-[49]	85D10-14	<i>Df(3R)by416</i>	<i>Df(3R)by62</i>
I(3)85Dd	3-[49]	85D11-E3	<i>Df(3R)by62</i> <i>Df(3R)by416</i>	
I(3)85De	3-[49]	85D11-E3	<i>Df(3R)by62</i> <i>Df(3R)by416</i>	
I(3)85Df	3-[49]	85D11-E3	<i>Df(3R)by62</i> <i>Df(3R)by416</i>	
I(3)85Dg	3-[49]	85D11-E3	<i>Df(3R)by62</i> <i>Df(3R)by416</i>	
I(3)85Dh	3-[49]	85D11-E3	<i>Df(3R)by62</i> <i>Df(3R)by416</i>	
I(3)85Di	3-[49]	85D11-E3	<i>Df(3R)by62</i> <i>Df(3R)by416</i>	
I(3)85Dj	3-[49]	85D11-E3	<i>Df(3R)by62</i> <i>Df(3R)by416</i>	

alleles:

allele	origin	discoverer	synonym
*I(3)85Da¹	X ray	Maroni	<i>l(3)M7</i>
*I(3)85Da²	X ray	Maroni	<i>l(3)M13</i>
*I(3)85Da³	X ray	Maroni	<i>l(3)M16</i>
*I(3)85Db¹	X ray	Maroni	<i>l(3)M1</i>
*I(3)85Db²	X ray	Maroni	<i>l(3)M10</i>
*I(3)85Dc¹	X ray	Maroni	<i>l(3)M17</i>
*I(3)85Dd¹	EMS	Matthews	<i>l(3)E4</i>
*I(3)85De¹	EMS	Matthews	<i>l(3)E10</i>
*I(3)85Df¹	X ray	Maroni	<i>l(3)M4</i>
*I(3)85Dg¹	X ray	Maroni	<i>l(3)M5</i>
*I(3)85Dh¹	EMS	Matthews	<i>l(3)E7</i>
*I(3)85Dh²	X ray	Maroni	<i>l(3)M6</i>
*I(3)85Dh³	X ray	Maroni	<i>l(3)M9</i>
*I(3)85Di¹	EMS	Matthews	<i>l(3)E11</i>
*I(3)85Di²	X ray	Maroni	<i>l(3)M12</i>
*I(3)85Di³	X ray	Maroni	<i>l(3)M14</i>
I(3)85Dj¹	EMS	Matthews	<i>l(3)J2</i>

I(3)85E

These lethals fall into two segments of 85E; order *l(3)85Ec Scm l(3)85Eb l(3)85Ea* established by Cheney; *l(3)85Ed* and *l(3)85Ee* placed to the left of *l(3)85Ea* by Matthews. Neither *l(3)85Ed* nor *l(3)85Ee* tested for complementation with *l(3)85Eb*.

locus	genetic location	cytological location	included in	excluded from	synonym
I(3)85Ea	3-49.0	85E1-10	<i>Df(3R)GB104</i> <i>Df(3R)by62</i>	<i>Df(3R)by416</i>	<i>l(3)C43</i>
I(3)85Eb	3-49.2	85E1-10	<i>Df(3R)GB104</i> <i>Df(3R)by62</i>	<i>Df(3R)by416</i>	<i>l(3)J5</i>
I(3)85Ec	3-49.8	85E1-10	<i>Df(3R)GB104</i> <i>Df(3R)by62</i>	<i>Df(3R)by416</i>	<i>l(3)WG89</i>
I(3)85Ed	3-[49]	85E1-10	<i>Df(3R)GB104</i> <i>Df(3R)by62</i>	<i>Df(3R)by416</i>	<i>l(3)KM19</i>
I(3)85Ee	3-49	85E1-10	<i>Df(3R)GB104</i> <i>Df(3R)by62</i>	<i>Df(3R)by416</i>	
<i>l(3)85Ef</i>	3-48.5	85E1-10	<i>Df(3R)GB104</i> <i>Df(3R)by62</i>	<i>Df(3R)by416</i>	Scm

locus	genetic location		cytological location		included in	excluded from	synonym
	location	location	location	location			
<i>l(3)85Eg</i>	3-49	85E1-10	<i>Df(3R)GB104</i>	<i>Df(3R)by416</i>		<i>knk</i>	
<i>l(3)85Eh</i>	3-(49)	85E10-F1	<i>Df(3R)by10</i>	<i>Df(3R)GB104</i>			
<i>l(3)85Ei</i>	3-(49)	85E10-F1	<i>Df(3R)by10</i>	<i>Df(3R)GB104</i>			
<i>l(3)85Ej</i>	3-(49)	85E10-F1	<i>Df(3R)by10</i>	<i>Df(3R)GB104</i>			

l(3)85Ea

phenotype: *l(3)85Ea*¹ is a heat-sensitive allele. At 20° about half the expected number of homozygotes eclose, the remainder dying as pharate adults; those that emerge are sterile, ovaries failing to mature, testes appearing morphologically normal; they also exhibit other slight abnormalities such as extra sex-comb teeth and defective wing margins. At 22° all flies die as pharate adults with greatly reduced heads, often with duplicated antennal and palpal structures. No metamorphosis accompanies development at 25°; the eye disc is virtually missing, whereas the wing and haltere discs are severely hyperplastic; disc effects autonomous in transplants. Temperature-shift experiments demonstrate a temperature-sensitive period lasting from the beginning of the second instar until the end of pupation. The 24-hr pulses of 27° during development reveal that the various phenotypic consequences of homozygosity for this allele have different TSP's. *l(3)85Ea*¹ wing discs used to demonstrate that extra cellular proliferation is insufficient to induce transdetermination (Shearn, Martin, Davis, and Hersperger, 1984, Dev. Biol. 106: 135-46). Density of gap junctions in hyperplastic imaginal wing discs markedly reduced (Ryerse and Nagel, 1984, Dev. Biol. 105: 396-403); in shift-up experiments, reduction in gap junctions precedes the onset of tissue hyperplasia (Ryerse and Nagel, 1985, Wilhelm Roux's Arch. Dev. Biol. 194: 480-86). Cell death also observed in wing discs as early as twelve hours following application of restrictive temperature (Sedlak, 1986, Dev. Genet 6: 199-212). Mitosis normal (Gatti and Baker, 1989, Genes Dev. 3: 438-53).

alleles: In addition to those listed below a series of *P*-element induced alleles recovered by Cheney.

allele	origin	discoverer	synonym	ref ^α
<i>l(3)85Ea</i> ¹	EMS	Shearn	<i>l(3)C43^{hs}</i>	1, 2
<i>l(3)85Ea</i> ²	EMS	Shearn	<i>l(3)RD222</i>	
<i>l(3)85Ea</i> ³	EMS	Shearn	<i>l(3)RE230</i>	
<i>l(3)85Ea</i> ⁴	EMS	Shearn	<i>l(3)SB610</i>	
<i>l(3)85Ea</i> ⁵	EMS	Shearn	<i>l(3)SE924</i>	
<i>l(3)85Ea</i> ⁶	EMS	Shearn	<i>l(3)SJ105</i>	
<i>l(3)85Ea</i> ⁷	EMS	Shearn	<i>l(3)UL163</i>	
<i>l(3)85Ea</i> ⁸	EMS	Shearn	<i>l(3)UO571</i>	
<i>l(3)85Ea</i> ⁹	EMS	Shearn	<i>l(3)V1343</i>	
<i>l(3)85Ea</i> ¹⁰	EMS	Shearn	<i>l(3)VF202</i>	
<i>l(3)85Ea</i> ¹¹	EMS	Shearn	<i>l(3)VX267</i>	
<i>l(3)85Ea</i> ¹²	EMS	Shearn	<i>l(3)VY293</i>	
<i>l(3)85Ea</i> ¹³	EMS	Shearn	<i>l(3)WB123</i>	
<i>l(3)85Ea</i> ¹⁴	EMS	Shearn	<i>l(3)WC461</i>	
<i>l(3)85Ea</i> ¹⁵	EMS	Mathews	<i>l(3)I8</i>	
<i>l(3)85Ea</i> ¹⁶	EMS	Mathews	<i>l(3)J29</i>	
<i>l(3)85Ea</i> ¹⁷	EMS	Mathews	<i>l(3)J36</i>	
<i>l(3)85Ea</i> ¹⁸	EMS	Mathews	<i>l(3)J42</i>	
<i>l(3)85Ea</i> ¹⁹	EMS	Mathews	<i>l(3)D1A</i>	
<i>l(3)85Ea</i> ²⁰	EMS	Mathews	<i>l(3)E5</i>	
<i>l(3)85Ea</i> ²¹	EMS	Mathews	<i>l(3)E12</i>	

allele	origin	discoverer	synonym	ref ^α
<i>l(3)85Ea</i> ²²	X ray	Maroni	<i>l(3)M19</i>	
<i>l(3)85Ea</i> ²³	X ray	Maroni	<i>l(3)M20</i>	
<i>l(3)85Ea</i> ²⁴	X ray	Maroni	<i>l(3)M22</i>	
<i>l(3)85Ea</i> ²⁵	EMS	Maroni	<i>l(3)M31</i>	
<i>l(3)85Ea</i> ²⁶	EMS	Maroni	<i>l(3)M39</i>	
<i>l(3)85Ea</i> ²⁸	EMS	Maroni	<i>l(3)M43</i>	
<i>l(3)85Ea</i> ²⁹	EMS	Maroni	<i>l(3)M49</i>	
<i>l(3)85Ea</i> ³⁰	EMS	Maroni	<i>l(3)M52</i>	
<i>l(3)85Ea</i> ³¹	DEB	Maroni	<i>l(3)M55</i>	
<i>l(3)85Ea</i> ³²	DEB	Maroni	<i>l(3)M64</i>	
<i>l(3)85Ea</i> ³³	DEB	Maroni	<i>l(3)M68</i>	

^α 1 = Martin, Martin, and Shearn, 1977, Dev. Biol. 55: 213-32; 2 = Shearn, Rice, Garen, and Gehring, 1971, Proc. Nat. Acad. Sci. USA 68: 2594-98.

l(3)85Eb

phenotype: Lethal at prepupal stage; morphology of all imaginal discs normal.

alleles:

allele	origin	discoverer	synonym	ref ^α
<i>l(3)85Eb</i> ¹	EMS	Shearn	<i>l(3)J5</i>	2, 3
<i>l(3)85Eb</i> ²	EMS	Shearn	<i>l(3)J83</i>	2, 3

l(3)85Ec

phenotype: Lethal at prepupal stage; morphology of all imaginal discs normal.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(3)85Ec</i> ¹	EMS	Shearn	<i>l(3)WG89</i>	1	
<i>l(3)85Ec</i> ²	EMS	Mathews	<i>l(3)KM12</i>		
<i>l(3)85Ec</i> ³	DEB	Mathews	<i>l(3)D1B</i>		
<i>l(3)85Ed</i> ¹	EMS	Maroni	<i>l(3)M36</i>		
<i>l(3)85Ed</i> ²	EMS	Maroni	<i>l(3)M56</i>		
<i>l(3)85Ed</i> ³	EMS	Maroni	<i>l(3)M58</i>		
<i>l(3)85Ed</i> ⁴	EMS	Maroni	<i>l(3)M63</i>		
<i>l(3)85Ee</i> ¹	EMS	Mathews	<i>l(3)KM19</i>		
<i>l(3)85Ee</i> ²	EMS	Mathews	<i>l(3)I04</i>		
<i>l(3)85Ee</i> ³	EMS	Mathews	<i>l(3)I07</i>		
<i>l(3)85Ee</i> ⁴	EMS	Mathews	<i>l(3)I13</i>		
<i>l(3)85Ee</i> ⁵	EMS	Mathews	<i>l(3)I17</i>		
<i>l(3)85Ee</i> ⁶	DEB	Maroni	<i>l(3)M62</i>		

*l(3)85Eh*¹ *l(3)J3*

*l(3)85Ei*¹ *l(3)E43*

*l(3)85Ej*¹ *l(3)M2* noncomplementing
*l(3)85Ej*² *l(3)M8* complements *l(3)M15*
*l(3)85Ej*³ *l(3)M15* complements *l(3)M8*

^α 1 = Shearn, Rice, Garen, and Gehring, 1971, Proc. Nat. Acad. Sci. USA 68: 2594-98.

l(3)85F

A group of as many as eight and as few as four lethal complementation groups located in the region uncovered by *Df(3R)by62* but not *Df(3R)by10*. Complementation analysis incomplete; *l(3)85Fa* through *l(3)85Fd* tested *inter se*; *l(3)85Fe* through *l(3)85Fh* not tested against the first four loci and incompletely tested *inter se*.

locus	genetic location		cytological location	
	location	location	included in	excluded from
<i>l(3)85Fa</i>	3-(49)	85E15-F6	<i>Df(3R)by62</i>	<i>Df(3R)by10</i>
<i>l(3)85Fb</i>	3-(49)	85E15-F6	<i>Df(3R)by62</i>	<i>Df(3R)by10</i>
<i>l(3)85Fc</i>	3-(49)	85E15-F6	<i>Df(3R)by62</i>	<i>Df(3R)by10</i>

locus	genetic location	cytological location	included in	excluded from
<i>l(3)85Fd</i>	3-[49]	85E15-F6	<i>Df(3R)by62</i>	<i>Df(3R)by10</i>
<i>l(3)85Fe</i>	3-[49]	85E15-F6	<i>Df(3R)by62</i>	<i>Df(3R)by10</i>
<i>l(3)85Ff</i>	3-[49]	85E15-F6	<i>Df(3R)by62</i>	<i>Df(3R)by10</i>
<i>l(3)85Fg</i>	3-[49]	85E15-F6	<i>Df(3R)by62</i>	<i>Df(3R)by10</i>
<i>l(3)85Fh</i>	3-[49]	85E15-F6	<i>Df(3R)by62</i>	<i>Df(3R)by10</i>

alleles:

allele	origin	discoverer	synonym	comments
*<i>l(3)85Fa</i>¹	EMS	Matthews	<i>l(3)1</i>	
*<i>l(3)85Fb</i>¹	EMS	Matthews	<i>l(3)11</i>	
*<i>l(3)85Fb</i>²	EMS	Matthews	<i>l(3)26</i>	
*<i>l(3)85Fc</i>¹	EMS	Matthews	<i>l(3)15</i>	
*<i>l(3)85Fc</i>²	EMS	Matthews	<i>l(3)16</i>	
*<i>l(3)85Fd</i>¹	EMS	Matthews	<i>l(3)35</i>	
*<i>l(3)85Fe</i>¹	EMS	Matthews	<i>l(3)40</i>	complements <i>l(3)85Fh</i>
*<i>l(3)85Ff</i>¹	EMS	Matthews	<i>l(3)106</i>	complements <i>l(3)85Fg</i>
*<i>l(3)85Fg</i>¹	EMS	Matthews	<i>l(3)108</i>	complements <i>l(3)85Ff</i>
*<i>l(3)85Fh</i>¹	EMS	Matthews	<i>l(3)111</i>	complements <i>l(3)85Fe</i>

DEFICIENCY MAP OF REGION 85

side	breakpoint	variant	DNA coordinates ^α
left	84E12-13	<i>Df(3R)CA1</i>	
left	84D8-9	<i>Df(3R)dsx11</i>	
left	84E6-9	<i>Df(3R)V2</i>	
left	84F2-6	<i>Df(3R)CA3</i>	
		<i>l(3)84Fk</i>	
		<i>l(3)84Fm</i>	
right	85A1-2	<i>Df(3R)dsx11</i>	
left	85A5-7	<i>Df(3R)p25</i>	
		<i>l(3)85Aa</i>	
right	85A2-4	<i>Df(3R)D8</i>	-57 kb
right	85A4-5	<i>Df(3R)V5</i>	
		<i>l(3)85Ab</i>	
left	84A4-5	<i>Df(3R)Dhod-G1</i>	-49 kb
		<i>l(3)85Ai</i>^β	
		<i>Dhod</i>	+14 kb
right	85A3-5	<i>Df(3R)V2</i>	+19 kb
left	85A6-11	<i>Df(3R)CA4B</i>	+22 kb
left		<i>Df(3R)p5</i>	+24 kb
left		<i>Df(3R)p16</i>	+24 kb
left		<i>Df(3R)p19</i>	+24 kb
		<i>l(3)85Aj</i>^β	
		<i>p</i>	
right		<i>Df(3R)p5</i>	+29 kb
		<i>l(3)85Ac</i>	
right	85A6-11	<i>Df(3R)CA1</i>	+33 kb
right	85A6-11	<i>Df(3R)CA3</i>	+33 kb
right		<i>Df(3R)p19</i>	+33 kb
		<i>l(3)85Ad</i>	
right	85A6-11	<i>Df(3R)V8</i>	+37 kb
		<i>l(3)85Ae</i>	
		<i>l(3)85Af</i>	
right		<i>Df(3R)p16</i>	+45 kb
		<i>l(3)85Ag</i>	
right	85A6-11	<i>Df(3R)Dhod-G1</i>	+45 kb
		<i>hb</i>	+60 kb
right	85A11	<i>Df(3R)p25</i>	+60 kb
right	85C3-6	<i>Df(3R)CA4B</i>	
left	85D3-5	<i>Df(3R)D8</i>	
left	85D8-12	<i>Df(3R)by10</i>	
		<i>l(3)85Da</i>	
left	85D11-13	<i>Df(3R)GB104</i>	
		<i>l(3)85Db</i>	

side	breakpoint	variant	DNA coordinates ^α
left	85D10-12	<i>Df(3R)by416</i>	
		<i>l(3)85Dc</i>	
left	85D11-14	<i>Df(3R)by62</i>	
		<i>l(3)85Dd</i>	
		<i>l(3)85De</i>	
		<i>l(3)85Df</i>	
		<i>l(3)85Dg</i>	
		<i>l(3)85Dh</i>	
		<i>l(3)85Di</i>	
		<i>l(3)85Dj</i>	
right	85E1-3	<i>Df(3R)by416</i>	
		<i>l(3)85Ec</i>	
		<i>Scm</i>	
		<i>l(3)85Eb</i>	
		<i>l(3)85Ed</i>	
		<i>l(3)85Ee</i>	
		<i>knk</i>	
		<i>α Tub85E</i>	
right	85E10	<i>Df(3R)GB104</i>	
		<i>l(3)85Eg</i>	
		<i>l(3)85Eh</i>	
		<i>l(3)85Ei</i>	
right	85E7-F1	<i>Df(3R)by10</i>	
		<i>l(3)85Fa</i>	
		<i>l(3)85Fb</i>	
		<i>l(3)85Fc</i>	
		<i>l(3)85Fd</i>	
		<i>l(3)85Fe</i>	
		<i>l(3)85Ff</i>	
		<i>l(3)85Fg</i>	
		<i>l(3)85Fh</i>	
right	85F16	<i>Df(3R)by62</i>	

^α Approximate coordinates of Jones and Rawls (1988, Genetics 120: 733-42).

l(3)86F

Seven lethally mutable loci identified by saturation mutagenesis studies of Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz (1981, Genetics 98: 775-89).

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(3)86Fa</i>	3-[51]	86F1-7	<i>Df(3R)T07</i>		<i>l(3)ck1</i>
<i>l(3)86Fb</i>	3-[51]	86F1-7	<i>Df(3R)T07</i>		<i>l(3)ck2</i>
<i>l(3)86Fc</i>	3-[51]	86F4-7		<i>Df(3R)T07</i>	<i>l(3)ck3</i>
				<i>Df(3R)E229</i>	
<i>l(3)86Fd</i>	3-[51]	86F4-7		<i>Df(3R)T07</i>	<i>l(3)ck4</i>
				<i>Df(3R)E229</i>	
<i>l(3)86Fe</i>	3-[51]	86F6-87A2	<i>Df(3R)E229</i>	<i>Df(3R)kar-H5</i>	<i>l(3)ck5</i>
<i>l(3)86Ff</i>	3-[51]	86F6-87A2	<i>Df(3R)E229</i>	<i>Df(3R)kar-H5</i>	<i>l(3)ck6</i>
<i>l(3)86Fg</i>	3-[51]	86F6-87A2	<i>Df(3R)E229</i>	<i>Df(3R)kar-H5</i>	<i>l(3)ck7</i>

l(3)86Fa

phenotype: Hemizygous lethal; lethal phase in first and second larval instars. Intragenic complementation seen in some heteroallelic combinations; *l(3)86Fa*¹/*l(3)86Fa*² survivors exhibit low viability; adults weak with poor cuticular pigmentation and abnormal wings.

references: Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz, 1981, Genetics 98: 775-89.

alleles:

allele	origin	synonym
<i>l(3)86Fa</i>¹	EMS	<i>ck1</i> ^{e70}
<i>l(3)86Fa</i>²	EMS	<i>ck1</i> ^{e143}
<i>l(3)86Fa</i>³	EMS	<i>ck1</i> ^{e199}
<i>l(3)86Fa</i>⁴	EMS	<i>ck1</i> ^{e264}
<i>l(3)86Fa</i>⁵	EMS	<i>ck1</i> ^{nf20}

allele	origin	synonym
<i>l(3)86Fa</i>⁶	EMS	<i>ck1</i> <i>hs9</i>

l(3)86Fb

phenotype: Hemizygotes die as second or third instar larvae; larvae leave medium and die.

references: Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz, 1981, Genetics 98: 775-89.

alleles:

allele	origin	synonym	comments
<i>l(3)86Fb</i>¹	EMS	<i>ck2</i> <i>e2</i>	L2
<i>l(3)86Fb</i>²	EMS	<i>ck2</i> <i>e144</i>	L2-3
<i>l(3)86Fb</i>³	EMS	<i>ck2</i> <i>e202</i>	
<i>l(3)86Fb</i>⁴	EMS	<i>ck2</i> <i>e253</i>	
<i>l(3)86Fb</i>⁵	EMS	<i>ck2</i> <i>nf38</i>	

l(3)86Fc

phenotype: Hemizygotes surviving to adulthood exhibit abnormal morphology.

references: Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz, 1981, Genetics 98: 775-89.

Reuter, Gausz, Gyurkovics, Friede, Bang, Spierer, Hall, and Spierer, 1987, Mol. Gen. Genet. 210: 429-36.

phenotype: Fails to complement lethality of *Su(var)3-13*; however, *l(3)86Fc*⁴ and *l(3)86Fc*⁵ have no dominant variegation suppressing effects (Reuter *et al.*).

alleles:

allele	origin	synonym	comments
<i>l(3)86Fc</i>¹	EMS	<i>ck3</i> <i>e27</i>	P
<i>l(3)86Fc</i>²	EMS	<i>ck3</i> <i>e29</i>	
<i>l(3)86Fc</i>³	EMS	<i>ck3</i> <i>e51</i>	
<i>l(3)86Fc</i>⁴	EMS	<i>ck3</i> <i>e277</i>	P-A
<i>l(3)86Fc</i>⁵	EMS	<i>ck3</i> <i>e297</i>	
<i>l(3)86Fc</i>⁶	EMS	<i>ck3</i> <i>hs18</i>	
<i>l(3)86Fd</i>¹	EMS	<i>ck4</i> <i>e243</i>	E-L-P
<i>l(3)86Fe</i>¹	EMS	<i>ck5</i> <i>e88</i>	L1-2
<i>l(3)86Fe</i>²	X ray	<i>ck5</i> <i>t19</i>	
<i>l(3)86Fe</i>³	EMS	<i>ck5</i> <i>b221</i>	
<i>l(3)86Fe</i>⁴	EMS	<i>ck5</i> <i>b262</i>	
<i>l(3)86Ff</i>¹	EMS	<i>ck6e</i> <i>145</i>	L1-2
<i>l(3)86Ff</i>²	X ray	<i>ck6</i> <i>t07</i>	

l(3)86Fg

phenotype: Hemizygous larvae have uninflated tracheae.

references: Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz, 1981, Genetics 98: 775-89.

alleles:

allele	origin	synonym	comments
<i>l(3)86Fg</i>¹	EMS	<i>ck7</i> <i>nf12</i>	L1
<i>l(3)86Fg</i>²	EMS	<i>ck7</i> <i>nf16</i>	L1

DEFICIENCY MAP OF REGION 86F

side	breakpoint	variant
left	86E	<i>Df(3R)T45</i>
left	86E2-4	<i>Df(3R)T32</i>
left	86E16-18	<i>Df(3R)kar-D3</i>
left	86F1-2	<i>Df(3R)E079</i>
left	86F1-2	<i>Df(3R)T07</i>
left	86F2-4 (1-2)	<i>Df(3R)T10</i>

side	breakpoint	variant
left	86F1-2	<i>Df(3R)T41</i>
left	86F1-2	<i>Df(3R)T47</i>
	86F1-7	<i>l(3)86Fa</i>
	86F1-7	<i>l(3)86Fb</i>
left	86F1-2	<i>Df(3R)T55</i>
left	86F1-2	<i>Df(3R)T61</i>
left	86F1-2	<i>Df(3R)T63</i>
right	86F4-7	<i>Df(3R)T07</i>
	86F4-7	<i>l(3)86Fc</i>
	86F4-7	<i>l(3)86Fd</i>
left	86F6-7	<i>Df(3R)E229</i>
	86F6-87A2	<i>l(3)86Fe</i>
	86F6-87A2	<i>l(3)86Ff</i>
	86F6-87A2	<i>l(3)86Fg</i>
left	87A1-2	<i>Df(3R)kar-H5</i>

l(3)87

Three different studies have identified forty lethally-mutable loci in 87A-E. 87A and 87B with five and thirteen loci respectively (Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz, 1981, Genetics 98: 775-89); 87C with four loci (Gausz, Bencze, Gyurkovics, Ashburner, Ish-Horowicz, and Holden, 1979, Genetics 93: 917-34); and 87D and E with seven and eleven loci respectively (Hilliker, Clark, and Chovnick, 1980, Genetics 95: 95-110). Included among these loci are *pic* in 87D and *Ace* in 87E.

l(3)87A

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(3)87Aa</i>	3-[51]	87A1-7	<i>Df(3R)kar-H5</i>	<i>Df(3R)kar1W</i>	<i>l(3)ck8</i>
<i>l(3)87Ab</i>	3-[51]	87A1-7	<i>Df(3R)kar-H5</i>	<i>Df(3R)kar1W</i>	<i>l(3)ck9</i>
<i>l(3)87Ac</i>	3-[51]	87A7-9	<i>Df(3R)kar-D1</i>		<i>l(3)ck10</i>
			<i>Df(3R)T47</i>		
<i>l(3)87Ad</i>	3-[51]	87A9-B2	<i>Df(3R)E229</i>	<i>Df(3R)T47</i>	<i>l(3)ck11</i>
<i>l(3)87Ae</i>	3-[51]	87A9-B2	<i>Df(3R)E229</i>	<i>Df(3R)T47</i>	<i>l(3)ck12</i>

l(3)87Aa-Ab

phenotype: Hemizygotes for *l(3)87Aa*³ form tanned pseudopupae in late third instar.

references: Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz, 1981, Genetics 98: 775-89.

allele	origin	synonym	comments
<i>l(3)87Aa</i>¹	EMS	<i>ck8</i> <i>e210</i>	L3
<i>l(3)87Aa</i>²	EMS	<i>ck8</i> <i>e212</i>	
<i>l(3)87Aa</i>³	EMS	<i>ck8</i> <i>e296</i>	L3
<i>l(3)87Aa</i>⁴	EMS	<i>ck8</i> <i>a1</i>	
<i>l(3)87Aa</i>⁵	EMS	<i>ck8</i> <i>a2</i>	
<i>l(3)87Aa</i>⁶	EMS	<i>ck8</i> <i>a3</i>	
<i>l(3)87Aa</i>⁷	EMS	<i>ck8</i> <i>a6</i>	
<i>l(3)87Aa</i>⁸	EMS	<i>ck8</i> <i>nf15</i>	
<i>l(3)87Aa</i>⁹	EMS	<i>ck8</i> <i>hs7</i>	
<i>l(3)87Aa</i>¹⁰	EMS	<i>ck8</i> <i>hs15</i>	
<i>l(3)87Aa</i>¹¹	EMS	<i>ck8</i> <i>hs17</i>	
<i>l(3)87Aa</i>¹²	EMS	<i>ck8</i> <i>hs32</i>	

<i>l(3)87Ab</i>¹	EMS	<i>ck9</i> <i>e42</i>	
<i>l(3)87Ab</i>²	EMS	<i>ck9</i> <i>e47</i>	
<i>l(3)87Ab</i>³	EMS	<i>ck9</i> <i>e82</i>	E
<i>l(3)87Ab</i>⁴	EMS	<i>ck9</i> <i>e110</i>	semilethal
<i>l(3)87Ab</i>⁵	EMS	<i>ck9</i> <i>e259</i>	
<i>l(3)87Ab</i>⁶	EMS	<i>ck9</i> <i>e260</i>	E
<i>l(3)87Ab</i>⁷	EMS	<i>ck9</i> <i>a4</i>	
<i>l(3)87Ab</i>⁸	EMS	<i>ck9</i> <i>nf9</i>	
<i>l(3)87Ab</i>⁹	EMS	<i>ck9</i> <i>nf11</i>	
<i>l(3)87Ab</i>¹⁰	EMS	<i>ck9</i> <i>hs20</i>	

allele origin synonym comments

l(3)87Ac

phenotype: Hemizygotes pupate but form longer-than-normal pupae.

references: Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz, 1981, Genetics 98: 775-89.

allele	origin	synonym	comments
l(3)87Ac¹	EMS	<i>ck10</i> <i>e132</i>	semilethal
l(3)87Ac²	EMS	<i>ck10</i> <i>e160</i>	
l(3)87Ac³	EMS	<i>ck10</i> <i>e170</i>	
l(3)87Ac⁴	EMS	<i>ck10</i> <i>e200</i>	LP
l(3)87Ac⁵	EMS	<i>ck10</i> <i>e209</i>	LP
l(3)87Ac⁶	EMS	<i>ck10</i> <i>hs55</i>	

l(3)87Ad

phenotype: Hemizygotes die in late third instar; imaginal discs strongly reduced in size.

references: Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz, 1981, Genetics 98: 775-89.

allele	origin	synonym	comments
l(3)87Ad¹	X ray	<i>ck11</i> <i>x02</i>	L3
l(3)87Ad²	EMS	<i>ck11</i> <i>b42</i>	
l(3)87Ad³	EMS	<i>ck11</i> <i>b52</i>	
l(3)87Ad⁴	EMS	<i>ck11</i> <i>b284</i>	
l(3)87Ad⁵	EMS	<i>ck11</i> <i>b303</i>	

l(3)87Ae

phenotype: Hemizygotes die in late pupal stage; Malpighian tubules exhibit abnormal morphology.

references: Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz, 1981, Genetics 98: 775-89.

allele	origin	synonym	comments
l(3)87Ae¹	EMS	<i>ck12</i> <i>e77</i>	L-LP
l(3)87Ae²	EMS	<i>ck12</i> <i>e177</i>	LP, semilethal
l(3)87Ae³	EMS	<i>ck12</i> <i>e294</i>	
l(3)87Ae⁴	EMS	<i>ck12</i> <i>nf10</i>	
l(3)87Ae⁵	EMS	<i>ck12</i> <i>hs5</i>	
l(3)87Ae⁶	EMS	<i>ck12</i> <i>hs28</i>	
l(3)87Ae⁷	EMS	<i>ck12</i> <i>hs41</i>	
l(3)87Ae⁸	EMS	<i>ck12</i> <i>hs78</i>	temperature sensitive

l(3)87B

locus	genetic		included in	excluded from	synonym
	location	cytological location			
l(3)87Ba	3-[51]	87B1-4	<i>Df(3R)kar-Sz12</i>	<i>Df(3R)kar3Q</i>	<i>l(3)ck13</i>
l(3)87Bb	3-[51]	87B2-4	<i>Df(3)kar3Q</i>	<i>Df(3R)kar-H10</i>	<i>l(3)ck14</i>
l(3)87Bc	3-[51]	87B2-4	<i>Df(3)kar3Q</i>	<i>Df(3R)kar-H10</i>	<i>l(3)ck15</i>
l(3)87Bd	3-[51]	87B4-6	<i>Df(3R)kar-H10</i>		svp
			<i>Df(3R)T45</i>		<i>l(3)ck16</i>
l(3)87Be	3-[51]	87B5-9	<i>Df(3R)E079</i>	<i>Df(3R)T45</i>	<i>l(3)ck17</i>
l(3)87Bf	3-[51]	87B5-9	<i>Df(3R)E079</i>	<i>Df(3R)T45</i>	<i>l(3)ck18</i>
l(3)87Bg	3-[51]	87B5-9	<i>Df(3R)E079</i>	<i>Df(3R)T45</i>	<i>l(3)ck19</i>
l(3)87Bh	3-[51]	87B9-13		<i>Df(3R)E079</i>	<i>l(3)ck20</i>
				<i>Df(3R)ry615</i>	
l(3)87Bi	3-[51]	87B9-13		<i>Df(3R)E079</i>	<i>l(3)ck21</i>
				<i>Df(3R)ry615</i>	
l(3)87Bj	3-[51]	87B9-13		<i>Df(3R)E079</i>	<i>l(3)ck22</i>
				<i>Df(3R)ry615</i>	
l(3)87Bk	3-[51]	87B11-C1	<i>Df(3R)ry615</i>	<i>Df(3R)kar3J</i>	<i>l(3)ck23</i>
l(3)87Bl	3-[51]	87B11-C1	<i>Df(3R)ry615</i>	<i>Df(3R)kar3J</i>	<i>l(3)ck24</i>
l(3)87Bm	3-[51]	87B11-C1	<i>Df(3R)ry615</i>	<i>Df(3R)kar3J</i>	<i>l(3)ck25</i>

l(3)87Ba

phenotype: Some heteroallelic combinations survive.

references: Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz, 1981, Genetics 98: 775-89.

allele	origin	synonym	comments
l(3)87Ba¹	EMS	<i>ck13</i> <i>e17</i>	semilethal
l(3)87Ba²	EMS	<i>ck13</i> <i>e72</i>	
l(3)87Ba³	EMS	<i>ck13</i> <i>e152</i>	
l(3)87Ba⁴	EMS	<i>ck13</i> <i>e233</i>	L1-2
l(3)87Ba⁵	EMS	<i>ck13</i> <i>e248</i>	
l(3)87Ba⁶	EMS	<i>ck13</i> <i>e270</i>	L1-2
l(3)87Ba⁷	EMS	<i>ck13</i> <i>nf31</i>	
l(3)87Ba⁸	EMS	<i>ck13</i> <i>hs56</i>	
l(3)87Bb¹	EMS	<i>ck14</i> <i>e139</i>	L1-2
l(3)87Bb²	EMS	<i>ck14</i> <i>hs2</i>	
l(3)87Bc¹	EMS	<i>ck15</i> <i>e61</i>	L1-2
l(3)87Bc²	EMS	<i>ck15</i> <i>e298</i>	L1-2
l(3)87Bc³	EMS	<i>ck15</i> <i>nf18</i>	
l(3)87Bc⁴	EMS	<i>ck15</i> <i>nf28</i>	
l(3)87Bc⁵	EMS	<i>ck15</i> <i>hs11</i>	
l(3)87Bc⁶	EMS	<i>ck15</i> <i>hs12</i>	
l(3)87Bc⁷	EMS	<i>ck15</i> <i>hs29</i>	
l(3)87Bc⁸	EMS	<i>ck15</i> <i>hs38</i>	
l(3)87Be¹	EMS	<i>ck17</i> <i>e8</i>	
l(3)87Be²	EMS	<i>ck17</i> <i>e26</i>	L1-2
l(3)87Be³	EMS	<i>ck17</i> <i>e74</i>	
l(3)87Be⁴	EMS	<i>ck17</i> <i>e180</i>	L1-3
l(3)87Be⁵	EMS	<i>ck17</i> <i>e228</i>	
l(3)87Be⁶	EMS	<i>ck17</i> <i>e249</i>	
l(3)87Be⁷	EMS	<i>ck17</i> <i>e280</i>	
l(3)87Be⁸	EMS	<i>ck17</i> <i>e281</i>	
l(3)87Be⁹	EMS	<i>ck17</i> <i>nf8</i>	
l(3)87Be¹⁰	EMS	<i>ck17</i> <i>nf14</i>	
l(3)87Be¹¹	EMS	<i>ck17</i> <i>nf17</i>	
l(3)87Be¹²	EMS	<i>ck17</i> <i>nf25</i>	
l(3)87Be¹³	EMS	<i>ck17</i> <i>hs6</i>	
l(3)87Be¹⁴	EMS	<i>ck17</i> <i>hs31</i>	

l(3)87Bf

phenotype: Young hemizygous pupae undergo autolysis.

references: Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz, 1981, Genetics 98: 775-89.

allele	origin	synonym	comments
l(3)87Bf¹	EMS	<i>ck18</i> <i>e227</i>	
l(3)87Bf²	EMS	<i>ck18</i> <i>e290</i>	EP
l(3)87Bf³	EMS	<i>ck18</i> <i>e312</i>	EP
l(3)87Bf⁴	EMS	<i>ck18</i> <i>hs44</i>	
l(3)87Bf⁵	EMS	<i>ck18</i> <i>hs71</i>	temperature sensitive

l(3)87Bg

phenotype: Hemizygotes for *l(3)87Bg²* form fully-pigmented pharate adults. Viability reduced in certain *trans* heterozygotes with *Su(var)3-6¹*; several *l(3)87Bg⁺* genotypes suppress position-effect variegation (Reuter *et al.*).

references: Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz, 1981, Genetics 98: 775-89.

Reuter, Gausz, Gyurkovics, Friede, Bang, Spierer, Hall, and Spierer, 1987, Mol. Gen. Genet. 210: 429-36.

allele	origin	synonym	comments
l(3)87Bg¹	EMS	<i>ck19</i> <i>e78</i>	dominant suppressor of variegation
l(3)87Bg²	EMS	<i>ck19</i> <i>e168</i>	LP
l(3)87Bg³	EMS	<i>ck19</i> <i>e211</i>	L2-3
l(3)87Bg⁴	EMS	<i>ck19</i> <i>e241</i>	semilethal

allele	origin	synonym	comments
<i>l(3)87Bg</i> ⁵	EMS	<i>ck19</i> <i>hs16</i>	
<i>l(3)87Bg</i> ⁶	EMS	<i>ck19</i> <i>hs46</i>	
<i>l(3)87Bh</i> ¹	EMS	<i>ck20</i> <i>e9</i>	L1-2
<i>l(3)87Bh</i> ²	EMS	<i>ck20</i> <i>e214</i>	L1-2
<i>l(3)87B1</i> ²	EMS	<i>ck21</i> <i>hs48</i>	
<i>l(3)87Bj</i> ¹	EMS	<i>ck22</i> <i>e186</i>	L2-3
<i>l(3)87Bj</i> ²	EMS	<i>ck22</i> <i>e293</i>	L1-2
<i>l(3)87Bj</i> ³	EMS	<i>ck22</i> <i>nf4</i>	
<i>l(3)87Bj</i> ⁴	EMS	<i>ck22</i> <i>nf19</i>	
<i>l(3)87Bj</i> ⁵	EMS	<i>ck22</i> <i>nf30</i>	
<i>l(3)87Bj</i> ⁶	EMS	<i>ck22</i> <i>nf37</i>	
<i>l(3)87Bk</i> ¹	EMS	<i>ck23</i> <i>e67</i>	
<i>l(3)87Bk</i> ²	EMS	<i>ck23</i> <i>e115</i>	L1-2
<i>l(3)87Bk</i> ³	EMS	<i>ck23</i> <i>e158</i>	L1-3
<i>l(3)87Bk</i> ⁴	EMS	<i>ck23</i> <i>e162</i>	

l(3)87B1

phenotype: Young hemizygous pupae undergo autolysis.

references: Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz, 1981, *Genetics* 98: 775-89.

allele	origin	synonym	comments
<i>l(3)87B1</i> ¹	EMS	<i>ck24</i> <i>e33</i>	EP
<i>l(3)87B1</i> ²	EMS	<i>ck24</i> <i>e100</i>	EP
<i>l(3)87B1</i> ³	EMS	<i>ck24</i> <i>e149</i>	
<i>l(3)87B1</i> ⁴	EMS	<i>ck24</i> <i>nf23</i>	
<i>l(3)87B1</i> ⁵	EMS	<i>ck24</i> <i>hs23</i>	
<i>l(3)87B1</i> ⁶	EMS	<i>ck24</i> <i>hs45</i>	
<i>l(3)87Bm</i> ¹	EMS	<i>ck25</i> <i>e11</i>	
<i>l(3)87Bm</i> ²	EMS	<i>ck25</i> <i>e99</i>	L1-3
<i>l(3)87Bm</i> ³	EMS	<i>ck25</i> <i>e208</i>	
<i>l(3)87Bm</i> ⁴	EMS	<i>ck25</i> <i>e219</i>	L1-3
<i>l(3)87Bm</i> ⁵	EMS	<i>ck25</i> <i>e318</i>	
<i>l(3)87Bm</i> ⁶	EMS	<i>ck25</i> <i>hs13</i>	
<i>l(3)87Bm</i> ⁷	EMS	<i>ck25</i> <i>hs25</i>	
<i>l(3)87Bm</i> ⁸	EMS	<i>ck25</i> <i>hs37</i>	
<i>l(3)87Bm</i> ⁹	EMS	<i>ck25</i> <i>hs52</i>	

l(3)87C

locus	genetic		included in	excluded from	synonym
	location	cytological location			
<i>l(3)87Ca</i>	3-51.7	87C4-5	<i>Df(3R)kar-Sz29</i>	<i>Df(3R)kar-Sz3</i>	<i>l(3)SzA</i>
<i>l(3)87Cb</i>	3-[52]	87C6	<i>Df(3R)T10</i>	<i>Df(3R)kar-Sz31</i>	<i>l(3)SzB</i>
<i>l(3)87Cc</i>	3-[52]	87C8	<i>Df(3R)kar-Sz27</i>	<i>Df(3R)kar-Sz11</i>	<i>l(3)SzC</i>
<i>l(3)87Cd</i>	3-[52]	87C9	<i>Df(3R)kar3J</i>	<i>Df(3R)kar-Sz21</i>	<i>l(3)SzD</i>

l(3)87Ca

phenotype: Most extreme hemizygous phenotype is embryonic lethality, even though homozygous *Df(3R)kar3J*, which lacks the locus entirely, dies during the first larval instar (Ish-Horowicz, Holden, and Gehring, 1977, *Cell* 12: 643-52). First instar larvae have transparent Malpighian tubules; autonomous in transplants. Two alleles isolated on the basis of their dominant suppression of ectopic sex-comb teeth in *Pc*^{4/+} males (Kennison and Tamkun, 1988, *Proc. Nat. Acad. Sci. USA* 85: 8136-40).

references: Gausz, Bencze, Gyurkovics, Ashburner, Ish-Horowicz, and Holden, 1979, *Genetics* 93: 917-34.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(3)87Ca</i> ¹	EMS		<i>l(3)SzA</i> ¹	1	

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(3)87Ca</i> ²	EMS		<i>l(3)SzA</i> ²	1	L
<i>l(3)87Ca</i> ³	EMS		<i>l(3)SzA</i> ³	1	semilethal
<i>l(3)87Ca</i> ⁴	EMS		<i>l(3)SzA</i> ⁴	1	
<i>l(3)87Ca</i> ⁵	EMS		<i>l(3)SzA</i> ⁵	1	E-L
<i>l(3)87Ca</i> ⁶	EMS		<i>l(3)SzA</i> ⁶	1	
<i>l(3)87Ca</i> ⁷	EMS		<i>l(3)SzA</i> ⁷	1	E
<i>l(3)87Ca</i> ⁸	EMS		<i>l(3)SzA</i> ⁸	1	
<i>l(3)87Ca</i> ⁹	EMS		<i>l(3)SzA</i> ⁹	1	
<i>l(3)87Ca</i> ¹⁰	EMS		<i>l(3)SzA</i> ¹⁰	1	semilethal
<i>l(3)87Ca</i> ¹¹	EMS		<i>l(3)SzA</i> ¹¹	1	
<i>l(3)87Ca</i> ¹²	EMS		<i>l(3)SzA</i> ¹²	1	
<i>l(3)87Ca</i> ¹³	EMS		<i>l(3)SzA</i> ¹³	1	
<i>l(3)87Ca</i> ¹⁴		Gelbart	<i>l(3)ml07</i>	1	
<i>l(3)87Ca</i> ¹⁵	γ ray	Kennison	<i>l(3)87Ca</i> ^{E1}	2	
<i>l(3)87Ca</i> ¹⁶	EMS	Kennison	<i>l(3)87Ca</i> ^{E2}	2	

^α 1 = Gausz, Bencze, Gyurkovics, Ashburner, Ish-Horowicz, and Holden, 1979, *Genetics* 93: 917-34; 2 = Kennison and Tamkun, 1988, *Proc. Nat. Acad. Sci. USA* 85: 8136-40.

l(3)87Cb

phenotype: Hemizygotes die during prepupal stage; puparium formation normal, but head eversion never occurs.

references: Gausz, Bencze, Gyurkovics, Ashburner, Ish-Horowicz, and Holden, 1979, *Genetics* 93: 917-34.

allele	origin	discoverer	synonym	comments
<i>l(3)87Cb</i> ¹	EMS		<i>l(3)SzB</i> ¹	
<i>l(3)87Cb</i> ²	EMS		<i>l(3)SzB</i> ²	P
<i>l(3)87Cb</i> ³	EMS		<i>l(3)SzB</i> ³	
<i>l(3)87Cb</i> ⁴	EMS		<i>l(3)SzB</i> ⁴	
<i>l(3)87Cb</i> ⁵	EMS		<i>l(3)SzB</i> ⁵	P
<i>l(3)87Cb</i> ⁶	EMS		<i>l(3)SzB</i> ⁶	
<i>l(3)87Cc</i> ¹	EMS		<i>l(3)SzC</i> ¹	
<i>l(3)87Cc</i> ²	EMS		<i>l(3)SzC</i> ²	L
<i>l(3)87Cc</i> ³	EMS		<i>l(3)SzC</i> ³	L
<i>l(3)87Cc</i> ⁴	EMS		<i>l(3)SzC</i> ⁴	
<i>l(3)87Cc</i> ⁵	EMS		<i>l(3)SzC</i> ⁵	
<i>l(3)87Cc</i> ⁶		Gelbart	<i>l(3)jc4g</i>	
<i>l(3)87Cd</i> ¹	EMS		<i>l(3)SzD</i> ¹	
<i>l(3)87Cd</i> ²	EMS		<i>l(3)SzD</i> ²	
<i>l(3)87Cd</i> ³	EMS		<i>l(3)SzD</i> ³	
<i>l(3)87Cd</i> ⁴	EMS		<i>l(3)SzD</i> ⁴	
<i>l(3)87Cd</i> ⁵	EMS		<i>l(3)SzD</i> ⁵	
<i>l(3)87Cd</i> ⁶	EMS		<i>l(3)SzD</i> ⁶	L
<i>l(3)87Cd</i> ⁷	EMS		<i>l(3)SzD</i> ⁷	L
<i>l(3)87Cd</i> ⁸	EMS		<i>l(3)SzD</i> ⁸	L-P
<i>l(3)87Cd</i> ⁹	EMS		<i>l(3)SzD</i> ⁹	
<i>l(3)87Cd</i> ¹⁰	EMS		<i>l(3)SzD</i> ¹⁰	semilethal
<i>l(3)87Cd</i> ¹¹	EMS		<i>l(3)SzD</i> ¹¹	
<i>l(3)87Cd</i> ¹²	EMS		<i>l(3)SzD</i> ¹²	
<i>l(3)87Cd</i> ¹³	EMS		<i>l(3)SzD</i> ¹³	semilethal
<i>l(3)87Cd</i> ¹⁴	EMS		<i>l(3)SzD</i> ¹⁴	
<i>l(3)87Cd</i> ¹⁵	EMS		<i>l(3)SzD</i> ¹⁵	semilethal
<i>l(3)87Cd</i> ¹⁶		Gelbart	<i>l(3)ml14</i>	

l(3)87D

locus	genetic		included in	excluded from	synonym
	location	cytological location			
<i>l(3)87Da</i>	3-51.7	87D3-6	<i>Df(3R)kar-G27</i>	<i>Df(3R)ry1608</i>	<i>l(3)S3</i>
<i>l(3)87Db</i>	3-52.1	87D3-6	<i>Df(3R)kar-G27</i>	<i>Df(3R)ry1608</i>	<i>l(3)S5</i>
<i>l(3)87Dc</i>	3-[52]	87D3-6	<i>Df(3R)kar-G27</i>	<i>Df(3R)ry1608</i>	<i>l(3)B-103</i>
<i>l(3)87Dd</i>	3-[52]	87D4-9	<i>Df(3R)ry1608</i>	<i>Df(3R)ry36</i>	<i>l(3)ml4</i>
<i>l(3)87De</i>	3-[52]	87D7-12	<i>Df(3R)ry74</i>		<i>l(3)G9</i>
			<i>Df(3R)kar-G47</i>		
<i>l(3)87Df</i>	3-[52]	87D6-13	<i>Df(3R)ry36</i>	<i>Df(3R)kar-G47</i>	<i>l(3)S12</i>
<i>l(3)87Dg</i>	3-52	87D6-13	<i>Df(3R)ry36</i>	<i>Df(3R)ry506</i>	snk
<i>l(3)87Dh</i>	3-52.1	87D11-14	<i>Df(3R)ry75</i>	<i>Df(3R)ry36</i>	pic

genetic cytological
locus location location included in excluded from synonym

l(3)87Da

phenotype: Homozygous lethal. *l(3)87Da*² and *l(3)87Da*³ both partially complement *l(3)87Da*¹ (15% and 30% survival respectively) and each other (75% survival). Surviving heterozygotes with *l(3)87Da*¹ exhibit variation in dorsal central bristle number and length, and females greatly outnumber males; sex ratio and bristle morphology normal in *l(3)87Da*^{2/1} *l(3)87Da*³ survivors.

allele	origin	discoverer	synonym	ref ^α
<i>l(3)87Da</i> ¹	X ray	Schalet	<i>l(1)S3</i>	
<i>l(3)87Da</i> ²	EMS	Hilliker, Clark	<i>l(1)A34-1</i>	1, 2
<i>l(3)87Da</i> ³	EMS	Hilliker, Clark	<i>l(1)A46-1</i>	1, 2

^α 1 = Hilliker, Clark, and Chovnick, 1980, Genetics 95: 95-110; 2 = Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72.

l(3)87Db-Dd

allele	origin	discoverer	synonym	ref ^α
<i>l(3)87Db</i> ¹	X ray	Schalet	<i>l(3)S5</i>	
<i>l(3)87Db</i> ²	X ray	Chovnick	<i>l(3)C8a</i>	1, 2
<i>l(3)87Db</i> ³	X ray	Chovnick	<i>l(3)E4a</i>	1, 2
<i>l(3)87Db</i> ⁴	EMS	Gelbart	<i>l(3)G12</i>	1, 2
<i>l(3)87Db</i> ⁵	EMS	Hilliker, Clark	<i>l(3)9-13</i>	1, 2
<i>l(3)87Dc</i> ¹	EMS	Hilliker, Clark	<i>l(3)A6-1</i>	1, 2
<i>l(3)87Dc</i> ²	EMS	Hilliker, Clark	<i>l(3)B103</i>	1, 2
<i>l(3)87Dd</i> ¹	EMS	Deland	<i>l(3)ml4</i>	1, 2
<i>l(3)87Dd</i> ²	EMS	Hilliker, Clark	<i>l(3)10-194</i>	1, 2

^α 1 = Hilliker, Clark, and Chovnick, 1980, Genetics 95: 95-110; 2 = Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72.

l(3)87De

phenotype: Escapers resemble *MesB*, but without the abdomen effect. Interacts with *MesB* in *trans* heterozygotes (Skinner, Cole, and Chovnick).

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(3)87De</i> ¹	EMS	Hilliker, Clark	<i>l(3)2-228</i>	1, 2	
<i>l(3)87De</i> ²	EMS	Hilliker, Clark	<i>l(3)6-120</i>	1, 2	
<i>l(3)87De</i> ³	EMS	Hilliker, Clark	<i>l(3)11-147</i>	1, 2	
<i>l(3)87De</i> ⁴	EMS	Hilliker, Clark	<i>l(3)A39-2</i>	1, 2	
<i>l(3)87De</i> ⁵	EMS	Hilliker, Clark	<i>l(3)B10-1</i>	1, 2	
<i>l(3)87De</i> ⁶	EMS	Hilliker, Clark	<i>l(3)B13-2</i>	1, 2	
<i>l(3)87De</i> ⁷	EMS	Hilliker, Clark	<i>l(3)B13-3</i>	1, 2	
<i>l(3)87De</i> ⁸	EMS	Hilliker, Clark	<i>l(3)B23-1</i>	1, 2	
<i>l(3)87De</i> ⁹	EMS	Hilliker, Clark	<i>l(3)B25-1</i>	1, 2	
<i>l(3)87De</i> ¹⁰	EMS	Gelbart	<i>l(3)G9</i>	1, 2	
<i>l(3)87De</i> ¹¹	EMS	Gelbart	<i>l(3)G15</i>	1, 2	
<i>l(3)87De</i> ¹²	EMS	Gelbart	<i>l(3)G21</i>	1, 2	
<i>l(3)87De</i> ¹³	EMS	Hilliker, Clark	<i>l(3)H2</i>	1, 2	
<i>l(3)87De</i> ¹⁴	EMS	Hilliker, Clark	<i>l(3)H23</i>	1, 2	semilethal
<i>l(3)87De</i> ¹⁵	EMS	Hilliker, Clark	<i>l(3)H37</i>	1, 2	
<i>l(3)87De</i> ¹⁶	EMS	Hilliker, Clark	<i>l(3)H73</i>	1, 2	

^α 1 = Hilliker, Clark, and Chovnick, 1980, Genetics 95: 95-110; 2 = Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72.

l(3)87Df (A. Chovnick)

location: Maps immediately to the left of, and is contiguous with *ry* DNA.

references: Chovnick, Gelbart, McCarron, Osmond, Candido, and Baillie, 1976, Genetics 84: 233-55.

Hilliker, Clark, Chovnick, and Gelbart, 1980, Genetics 95: 95-110.

Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72.

Clark and Chovnick, 1986, Genetics 114: 819-40.

Dutton and Chovnick, 1990, submitted.

phenotype: *l(3)87Df*¹ and *l(3)87Df*² hemizygotes completely lethal. *l(3)87Df*³ cold-sensitive, conditional lethal. Surviving cold-sensitive mutants, and underexpression variants generated by *P*-element transformation, display a phenotypic syndrome that can include delayed development, abnormal bristle morphology and female sterility. Using these phenotypes, defects in "early" and "late" *l(3)87Df*¹ expression can be identified. *l(3)87Df*¹ function is required in embryos, early larvae, late pupae, and during oogenesis phenocritical periods, which coincide with peaks of *l(3)87Df*¹ transcript accumulation. Phenotypic and molecular analyses suggest that the *l(3)87Df*¹ gene probably encodes a ribosomal protein. Genetic and molecular mapping place *l(3)87Df*¹ immediately to the left of the *rosy* locus.

allele	origin	discoverer	synonym	ref ^α
<i>l(3)87Df</i> ¹	X ray	Schalet	<i>l(3)S12</i>	1
<i>l(3)87Df</i> ²	EMS	Hilliker, Clark	<i>l(3)B21-4</i>	2, 3
<i>l(3)87Df</i> ³	EMS	Gelbart	<i>l(3)G1</i>	2, 3

^α 1 = Chovnick, Gelbart, McCarron, Osmond, Candido, and Baillie, 1976, Genetics 84: 233-55; 2 = Hilliker, Clark, and Chovnick, 1980, Genetics 95: 95-110; 3 = Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72.

l(3)87E

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(3)87Ea</i>	3-52.2	87D14-E2		<i>Df(3R)ry75</i> <i>Df(3R)l26c</i>	<i>l(3)S8</i> , <i>slm</i> , <i>schm</i>
<i>l(3)87Eb</i>	3-{53}	87E1-2	<i>Df(3R)ry1608</i> <i>Df(3R)l26c</i>		<i>l(3)B16</i>
<i>l(3)87Ec</i>	3-{53}	87E1-2	<i>Df(3R)ry1301</i>	<i>Df(3R)ry1608</i>	<i>l(3)C9a</i>
<i>l(3)87Ed</i>	3-52.2	87E1-4	<i>Df(3R)ry1607</i>	<i>Df(3R)ry1301</i>	Ace
<i>l(3)87Ee</i>	3-{53}	87E2-6	<i>Df(3R)Ace-HD1</i>	<i>Df(3R)ry1607</i>	<i>l(3)G7</i>
<i>l(3)87Ef</i>	3-{53}	87E5	<i>Df(3R)l26d</i>	<i>Df(3R)Ace-HD1</i>	<i>l(3)m32</i>
<i>l(3)87Eg</i>	3-{53}	87E5-12	<i>Df(3R)lC4a</i>		<i>l(3)ml7</i>
<i>l(3)87Eh</i>	3-{53}	87E5-12	<i>Df(3R)lC4a</i>		<i>l(3)ml16</i>
<i>l(3)87Ei</i>	3-{53}	87E5-12	<i>Df(3R)lC4a</i>		<i>l(3)S9</i>
<i>l(3)87Ej</i>	3-{53}	87E5-12	<i>Df(3R)lC4a</i>		<i>l(3)G5</i>
<i>l(3)87Ek</i>	3-{53}	87E11-F1	<i>Df(3R)ry619</i>	<i>Df(3R)lC4a</i>	<i>l(3)ml12</i>
<i>l(3)87El</i>	3-{53}	87E4-6	<i>Df(3R)N78</i>	<i>Df(3R)ry1607</i>	Su(var)3-7

l(3)87Eb

phenotype: Lethal in all homoallelic and heteroallelic combinations except two: *l(3)87Eb*⁸ shows 4% and 15% survival in combination with *l(3)87Eb*² and *l(3)87Eb*⁵, respectively; phenotypes normal. *l(3)87Eb*⁶ allelism inferred from deficiency mapping; allele lost before others recovered.

allele	origin	discoverer	synonym	ref ^α
<i>l(3)87Eb</i> ¹	EMS	Hilliker, Clark	<i>l(3)B16-1</i>	1, 2
<i>l(3)87Eb</i> ²	EMS	Hilliker, Clark	<i>l(3)B16-4</i>	1, 2
<i>l(3)87Eb</i> ³	EMS	Hilliker, Clark	<i>l(3)B27-2</i>	1, 2
<i>l(3)87Eb</i> ⁴	EMS	Hilliker, Clark	<i>l(3)H13</i>	1, 2
<i>l(3)87Eb</i> ⁵	EMS	Hilliker, Clark	<i>l(3)H69</i>	1, 2
<i>l(3)87Eb</i> ⁶	X ray	Schalet	<i>l(3)S6</i>	3

^α 1 = Hilliker, Clark, and Chovnick, 1980, Genetics 95: 95-110; 2 = Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72; 3 = Schalet, Kernaghan, and Chovnick, 1964, Genetics 50: 1261-68.

l(3)87Ec-Ef

references: Hilliker, Clark, and Chovnick, 1980, Genetics 95: 95-110.

Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72.

phenotype: All alleles lethal in the hemizygous condition.

alleles: Limited complementation: *l(3)87Ec¹/l(3)87Ec²*, *l(3)87Ec¹/l(3)87Ec³*, and *l(3)87Ec²/l(3)87Ec³* give 2%, 8%, and 14% survival, respectively. Surviving progeny somewhat reduced in size relative to wild type.

allele	origin	discoverer	synonym
<i>l(3)87Ec¹</i>	EMS	Hilliker, Clark	<i>l(3)B2-6</i>
<i>l(3)87Ec²</i>	EMS	Hilliker, Clark	<i>l(3)B26-2</i>
<i>l(3)87Ec³</i>	X ray	Chovnick	<i>l(3)C9a</i>

allele	origin	discoverer	synonym	ref ^α
<i>l(3)87Ee¹</i>	EMS	Hilliker, Clark	<i>l(3)B1-3</i>	1, 2
<i>l(3)87Ee²</i>	EMS	Hilliker, Clark	<i>l(3)B9-1</i>	1, 2
<i>l(3)87Ee³</i>	EMS	Hilliker, Clark	<i>l(3)B13-1</i>	1, 2
<i>l(3)87Ee⁴</i>	EMS	Hilliker, Clark	<i>l(3)B30-2</i>	1, 2
<i>l(3)87Ee⁵</i>	EMS	Gelbart	<i>l(3)G7</i>	1, 2
<i>l(3)87Ee⁶</i>	γ ray	Hilliker, Clark	<i>l(3)H20</i>	1, 2
<i>l(3)87Ee⁷</i>	EMS	Hilliker, Clark	<i>l(3)H34</i>	1, 2
<i>l(3)87Ee⁸</i>	EMS	Hilliker, Clark	<i>l(3)H75</i>	1, 2
<i>l(3)87Ee⁹</i>	EMS	Hilliker, Clark	<i>l(3)H91</i>	1, 2
<i>l(3)87Ei¹</i>	EMS	Deland	<i>l(3)m32</i>	1, 2
<i>l(3)87Ei²</i>	EMS	J. Hall	<i>l(3)J38</i>	1, 2

^α 1 = Hilliker, Clark, and Chovnick, 1980, Genetics 95: 95-110; 2 = Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72.

l(3)87Eg

references: Hilliker, Clark, and Chovnick, 1980, Genetics 95: 95-110.

Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72.

alleles: Two complementation groups; *l(3)87Eg¹* is the single member of one; *l(3)87Eg²*, *l(3)87Eg⁴*, and *l(3)87Eg⁶* are members of the other; remaining alleles noncomplementing except for weak viability seen in *l(3)87Eg³/l(3)87Eg⁶* heterozygotes.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(3)87Eg¹</i>	EMS	Hilliker, Clark	<i>l(3)B1-1</i>	1	
<i>l(3)87Eg²</i>	EMS	Hilliker, Clark	<i>l(3)B11-1</i>	1	
<i>l(3)87Eg³</i>	EMS	Hilliker, Clark	<i>l(3)B16-3</i>	1	
<i>l(3)87Eg⁴</i>	EMS	Hilliker, Clark	<i>l(3)B26-3</i>	1	
<i>l(3)87Eg⁵</i>	EMS	Hilliker, Clark	<i>l(3)H45</i>	1	
<i>l(3)87Eg⁶</i>	EMS	Hilliker, Clark	<i>l(3)H77</i>	1	
<i>l(3)87Eg⁷</i>	EMS	Deland	<i>l(3)m17</i>	1	
<i>l(3)87Eg⁸</i>	EMS	Hilliker, Clark	<i>l(3)B9-2</i>	2	formally placed in <i>l(3)87Ek</i>

l(3)87Eh¹ EMS Deland *l(3)m116* 1

^α 1 = Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72; 2 = Manseau, Ganetzky, and Craig, 1988, Genetics 119: 407-20.

l(3)87Ei

alleles: *l(3)87Ei⁶* complements *l(3)87Ei²*, *l(3)87Ei³*, *l(3)87Ei⁵*, *l(3)87Ei¹⁰*, *l(3)87Ei¹⁴*, and *l(3)87Ei¹⁵*; partially complements *l(3)87Ei⁴*, *l(3)87Ei⁷*, and *l(3)87Ei⁸* (14%, 13%, and 22% relative viability, respectively); the remaining allelic combinations fail to complement.

allele	origin	discoverer	synonym	ref ^α
<i>l(3)87Ei¹</i>	X ray	Schalet	<i>l(3)S9</i>	3

allele	origin	discoverer	synonym	ref ^α
<i>l(3)87Ei²</i>	EMS	Hilliker, Clark	<i>l(3)B1-2</i>	1, 2
<i>l(3)87Ei³</i>	EMS	Hilliker, Clark	<i>l(3)B1-5</i>	1, 2
<i>l(3)87Ei⁴</i>	EMS	Hilliker, Clark	<i>l(3)B2-3</i>	1, 2
<i>l(3)87Ei⁵</i>	EMS	Hilliker, Clark	<i>l(3)B8-1</i>	1, 2
<i>l(3)87Ei⁶</i>	EMS	Hilliker, Clark	<i>l(3)B8-4</i>	1, 2
<i>l(3)87Ei⁷</i>	EMS	Hilliker, Clark	<i>l(3)B12-2</i>	1, 2
<i>l(3)87Ei⁸</i>	EMS	Hilliker, Clark	<i>l(3)B15-1</i>	1, 2
<i>l(3)87Ei⁹</i>	EMS	Hilliker, Clark	<i>l(3)B21-3</i>	1, 2
<i>l(3)87Ei¹⁰</i>	EMS	Hilliker, Clark	<i>l(3)B26-4</i>	1, 2
<i>l(3)87Ei¹¹</i>	EMS	Hilliker, Clark	<i>l(3)B28-1</i>	1, 2
<i>l(3)87Ei¹²</i>	EMS	Hilliker, Clark	<i>l(3)H9</i>	1, 2
<i>l(3)87Ei¹³</i>	EMS	Hilliker, Clark	<i>l(3)H30</i>	1, 2
<i>l(3)87Ei¹⁴</i>	EMS	Hilliker, Clark	<i>l(3)H32</i>	1, 2
<i>l(3)87Ei¹⁵</i>	EMS	Hilliker, Clark	<i>l(3)H57</i>	1, 2
<i>l(3)87Ei¹⁶</i>	EMS	Deland	<i>l(3)m102</i>	1, 2
<i>l(3)87Ej¹</i>	EMS	Hilliker, Clark	<i>l(3)B4-1</i>	1, 2
<i>l(3)87Ej²</i>	EMS	Gelbart	<i>l(3)G5</i>	1, 2

^α 1 = Hilliker, Clark, and Chovnick, 1980, Genetics 95: 95-110; 2 = Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72.

l(3)87Ek

references: Hilliker, Clark, and Chovnick, 1980, Genetics 95: 95-110.

Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72.

alleles: Three complementation groups, one with *l(3)87Ek²* and *l(3)87Ek⁴*, one with *l(3)87Ek¹*, and one with *l(3)87Ek⁵* and *l(3)87Ek⁶*. *l(3)87Ek³* fails to complement mutants in the first two but complements those in the third group. *l(3)87Ek⁷* partially complements *l(3)87Ek⁵* and *l(3)87Ek⁶* (40% and 24% relative survival, respectively) but fails to complement all other alleles; unlike other cases of complementation, these two combinations exhibit short, very thin bristles and irregularly arranged ommatidia.

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(3)87Ek²</i>	EMS	Hilliker, Clark	<i>l(3)B16-2</i>	1	
<i>l(3)87Ek³</i>	EMS	Hilliker, Clark	<i>l(3)B17-1</i>	1	
<i>l(3)87Ek⁴</i>	EMS	Hilliker, Clark	<i>l(3)H21</i>	1	partially complements <i>l(3)87Ek⁷</i>
<i>l(3)87Ek⁵</i>	γ ray	Hilliker, Clark	<i>l(3)H24</i>	1, 2	not an 87Ek allele?
<i>l(3)87Ek⁶</i>	γ ray	Hilliker, Clark	<i>l(3)H25</i>	1, 2	not an 87Ek allele?
<i>l(3)87Ek⁷</i>	EMS	Deland	<i>l(3)m112</i>	1, 2	partially complements <i>l(3)87Ek⁴</i>
<i>l(3)87Ek⁸</i>	EMS	Manseau	<i>l(3)E15</i>	2	0.9 kb deletion
<i>l(3)87Ek⁹</i>	EMS	Manseau	<i>l(3)E99</i>	2	0.9 kb deletion
<i>l(3)87Ek¹⁰</i>	EMS	Manseau	<i>l(3)E118</i>	2	
<i>l(3)87Ek¹¹</i>	EMS	Manseau	<i>l(3)E203</i>	2	

^α 1 = Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72; 2 = Manseau, Ganetzky, and Craig, 1988, Genetics 119: 407-20.

DEFICIENCY MAP OF REGION 87

side	breakpoint	variant	coordinates ^α
left	87A1-2	<i>Df(3R)kar-H5</i>	
	87A1-7	l(3)87Aa	
	87A1-7	l(3)87A	
right	87A5-7	<i>Df(3R)T63</i>	
right	87A5-7	<i>Df(3R)T55</i>	
left	87A6-7	<i>Df(3R)kar-1W</i>	
	87A6-7	Hsp70	
left	87A7	<i>Df(3R)kar-D1</i>	
	87A7-9	l(3)87Ac	
right	87A9	<i>Df(3R)T47</i>	
right	87A9	<i>Df(3R)T61</i>	
	87A9-B2	l(3)87Ad	
	87A9-B2	l(3)87Ae	

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side	breakpoint	variant	coordinates ^α
left	87B1	<i>Df(3R)kar-H1</i>	
right	87B1-2	<i>Df(3R)E229</i>	
left	87B1-2	<i>Df(3R)kar-Sz12</i>	
left	87B1-2	<i>Df(3R)kar-Sz15</i>	
	87B1-4	I(3)87Ba	
left	87B2-4	<i>Df(3R)E307</i>	
left	87B2-4	<i>Df(3R)kar3Q</i>	
	87B2-4	I(3)87Bb	
	87B2-4	I(3)87Bc	
left	87B4	<i>Df(3R)kar-H10</i>	
left	87B3-5	<i>Df(3R)kar-IG27</i>	
	87B4-6	I(3)87Bd	
left	87B5-6	<i>Df(3R)kar-H11</i>	
right	87B5-6	<i>Df(3R)T45</i>	
	87B5-9	I(3)87Be	
	87B5-9	I(3)87Bf	
	87B5-9	I(3)87Bg	
right	87B9	<i>Df(3R)E079</i>	
	87B9-13	I(3)87Bh	
	87B9-13	I(3)87Bi	
	87B9-13	I(3)87Bj	
left	87B11-13	<i>Df(3R)ry615</i>	
	87B11-C1	I(3)87Bk	
	87B11-C1	I(3)87Bl	
	87B11-C1	I(3)87Bm	
left	87B15-C1	<i>Df(3R)kar3J</i>	
left	87B15-C1	<i>Df(3R)ry1168</i>	
		Hap70	
right	87C1-2	<i>Df(3R)T41</i>	
left	87C1-3	<i>Df(3R)ry81</i>	
left	87C2-3	<i>Df(3R)kar31</i>	
left	87C3-4	<i>Df(3R)kar-Sz29</i>	
	87C4-5	I(3)87Ca	
left	87C5-6	<i>Df(3R)kar-Sz3</i>	
right	86C5-7	<i>Df(3R)T10</i>	
	87C6	I(3)87Cb	
left	87C6-7	<i>Df(3R)kar-Sz31</i>	
right	87C6-7	<i>Df(3R)T10</i>	
right	87C6-7	<i>Df(3R)T32</i>	
left	87C7	<i>Df(3R)kar-Sz21</i>	
left	87C7	<i>Df(3R)kar-Sz27</i>	
	87C7	I(3)87Cc	
left	87C7-8	<i>Df(3R)kar-Sz11</i>	
	87C8	kar	
right	87C8-9	<i>Df(3R)kar-Sz12</i>	
right	87C8-9	<i>Df(3R)kar-Sz21</i>	
	87C9	I(3)87Cd	
right	87C9-D1	<i>Df(3R)kar3J</i>	
right	87C9-D1	<i>Df(3R)kar-Sz29</i>	
right	87C9-D3	<i>Df(3R)kar-Sz31</i>	
	87D1-2	Men	
right	87D1-2	<i>Df(3R)E307</i>	
right	87D1-2	<i>Df(3R)kar3Q</i>	
left	87D1-2	<i>Df(3R)ry75</i>	
left	87D2-4	<i>Df(3R)ry614</i>	
left	87D2-4	<i>Df(3R)ry1301</i>	
left	87D2-4	<i>Df(3R)ry1402</i>	
left	87D3-4	<i>Df(3R)ry1607</i>	
right	87D3-4	<i>Df(3R)kar31</i>	
right	87D3-4	<i>Df(3R)kar-D1</i>	
right	87D3-4	<i>Df(3R)kar-D3</i>	
	87D3-6	I(3)87Da	
	87D3-6	I(3)87Db	
	87D3-6	I(3)87Dc	
left	87D4-6	<i>Df(3R)ry1608</i>	
	87D4-9	I(3)87Dd	
left	invisible	<i>Df(3R)ry36</i>	-213 to -201
	87D4-9	mes-A	
left		<i>Df(3R)ry74</i>	
	87D4-9	mes-B	
left	87D7-9	<i>Df(3R)ry619</i>	-213 to -201
	87D7-12	I(3)87De	
right	87D6-12	<i>Df(3R)kar-IG27</i>	-191 to -184
	87D6-13	I(3)87Df	
	87D6-13	ry	

side	breakpoint	variant	coordinates ^α
		snk	
right	invisible	<i>Df(3R)ry36</i>	--165
		Hsc70-2	
left	87D11-13	<i>Df(3R)l26d</i>	--160
	87D11-14	pic	
right	87D11-14	<i>Df(3R)ry614</i>	-139 to -125
right	87D12-E1	<i>Df(3R)kar-H1</i>	
right	87D13-14	<i>Df(3R)kar1W</i>	
right	87D14-E1	<i>Df(3R)ry75</i>	-139 to -125
	87D14-E2	slm	
right	87D14-E2	<i>Df(3R)ry81</i>	-112 to -100
left	87E1-2	<i>Df(3R)l26C</i>	
	87E1-2	I(3)87Eb	
right	87D14-E1	<i>Df(3R)kar-Sz37</i>	
right	87D14-E2	<i>Df(3R)ry1402</i>	-50 to -39
right	87E1-2	<i>Df(3R)ry1608</i>	-41 to -17
	87E1-2	I(3)87Ec	
right	87E1-2	<i>Df(3R)kar-Sz15</i>	
right	87E1-2	<i>Df(3R)ry1301</i>	+15 to +21.5
	3'	Ace	
right	87E2-3	<i>Df(3R)ry1607</i>	+26 to +32
left	87E3-4	<i>Df(3R)Ace-HD1</i>	+49 to +50.5
	5'	Ace	
left	87E4	<i>Df(3R)GE41</i>	+56.5 to +58.8
		Su(var)3-7	+59 to +63
		I(3)87Ee	
right	87E5-6	<i>Df(3R)Ace-HD1</i>	+72.5 to +77.8
right	87E5-6	<i>Df(3R)kar-Sz11</i>	+82 to +93
		I(3)87Ef	
right	87E4	<i>Df(3R)GE41</i>	
right	87E3-5	<i>Df(3R)l26d</i>	+79.5 to +90.5
left	87E5-7	<i>Df(3R)lC4a</i>	+73 to +85
	87E5-12	I(3)87Eg	
	87E5-12	I(3)87Eh	
	87E5-12	I(3)87Ei	
	87E5-12	I(3)87Ej	
right	87E11-13	<i>Df(3R)ry615</i>	
right	87E9-12	<i>Df(3R)ry1168</i>	
right	87E11-F1	<i>Df(3R)lC4a</i>	
		Act187E	
	87E11-F1	I(3)87Ek	
right	87E12-F1	<i>Df(3R)ry619</i>	
right	87F1	<i>Df(3R)kar-Sz27</i>	
right	87F11-12	<i>Df(3R)l26c</i>	

^α Molecular coordinates from Spierer, Bender, and Spierer (1983, J. Mol. Biol. 168: 35-50) and Gausz, Hall, Spierer, and Spierer, 1986, Genetics 112: 65-78). Origin 6.5 kb distal to left breakpoint of *ln(3R)Cbx^{rv1}* (Bender, Spierer, and Hogness, 1983, J. Mol. Biol. 168: 17-33).

I(3)88Aa

location: 3-50.3.

origin: Induced by N-methyl-N'-nitro-N-nitrosoguanidine.

synonym: *I(3)2004*.

references: Shearn, Rice, Garen, and Gehring, 1971, Proc. Nat. Acad. Sci. 68: 2594-98.

phenotype: Homozygous larvae die in third instar; discs reported as missing, probably rudimentary (see Szabad and Bryant, 1982, Dev. Biol. 93: 240-56). Mitotic chromosomes fuzzy and swollen; 27% of cells polyploid; chromosome breaks observed (Gatti and Baker, 1989, Genes Dev. 3: 438-53).

cytology: Placed in 87F12-88B1 on the basis of inclusion in *Df(3R)red31 = Df(3R)87F12-14;88B2-3* but not *Df(3R)red-P93 = Df(3R)88A10-B1;88C2-3*.

I(3)88Ab

location: 3-51.1.

origin: Induced by ethyl methanesulfonate.

synonym: *I(3)K43*.

references: Shearn, Rice, Garen, and Gehring, 1971, Proc.

Nat. Acad. Sci. USA 68: 2594-98.

Szabad and Bryant, 1982, Dev. Biol. 93: 240-56.

phenotype: Homozygous larvae contain rudimentary imaginal discs, but disc primordia do not grow during larval development; testes and ovaries smaller than normal, and cell number in central nervous system reduced. Mutant gonads do not survive metamorphosis when implanted into wild-type larvae. Homozygous cuticular clones appear to develop normally, but abdominal clones reduced in frequency and size compared to control clones. Mutant larvae support growth of implanted wild-type discs. Normal gene product postulated to be required for cell proliferation; survival of somatic epidermal clones attributed to perdurance. Larval ganglia exhibit extremely low mitotic index; chromosomes irregularly condensed and chromosome breakage observed (Gatti and Baker, 1989, Genes Dev. 3: 438-53); salivary chromosomes appear normal.

alleles:

allele	origin	discoverer	synonym	comments
<i>l(3)88Ab¹</i>	EMS		<i>l(3)K43</i>	
<i>l(3)88Ab²</i>	EMS	Nüsslein-Volhard	<i>fs(3)293-19</i>	hypomorph; viable α
<i>l(3)88Ab³</i>	γ ray	Spradling lab		
<i>l(3)88Ab⁴</i>	γ ray	Spradling lab		
<i>l(3)88Ab⁵</i>	γ ray	Spradling lab		
<i>l(3)88Ab⁶</i>	γ ray	Spradling lab		
<i>l(3)88Ab⁷</i>	EMS	Mortin	<i>ZM43</i>	
<i>l(3)88Ab⁸</i>	EMS	Mortin	<i>ZM47</i>	

α More detailed description below.

cytology: Placed in 88A12-B1 on the basis of its inclusion in *Df(3R)red-P52 = Df(3R)88A12-B1;88B4-5* but not *Df(3R)red-P93 = Df(3R)88A10-B1;88C2-3*.

molecular biology: Region of gene contained in a 400 kb cosmid walk; position narrowed to 50 kb between the proximal breakpoint of *Df(3R)red-P52* and the distal breakpoint of an overlapping more proximal inversion *Df(3R)293 = Df(3R)87E;88A*.

l(3)88Ab²

phenotype: Homozygous females weakly fertile; produce eggs with grossly defective chorions; thin, missing most appendages and permeable. All chorion proteins reduced in abundance; chorion gene amplification reduced to 10% wild-type level (Snyder, Galanopoulos, and Kafatos, 1986, Proc. Nat. Acad. Sci. USA 83: 3341-45). Heterozygotes with lethal alleles more extreme and sterile.

l(3)89A-B

Based on fine structure deficiency mapping of Hughes, Nelson, Yanuk, and Szauter (unpublished). The region of overlap between this study and that of Tiong, Bone, and Whittle in regions 89B-E have not been reconciled; they are treated separately here.

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(3)89Aa</i>	3-[58]	89A2-5	<i>Df(3R)c3G2</i>		
<i>l(3)89B1</i>	3-58	89A11-B4	<i>Df(3R)sbd105</i>	<i>Df(3R)Po4</i> <i>Df(3R)sbd45</i>	mor
<i>l(3)89B2</i>	3-58	89A11-B4	<i>Df(3R)sbd105</i>	<i>Df(3R)Po4</i> <i>Df(3R)sbd45</i>	srp
<i>l(3)89B3</i>	3-58.1	89B4-10	<i>Df(3R)sbd45</i>	<i>Df(3R)sbd26</i>	pnr

l(3)89Aa

cytology: Identified as a lethal found in some, and perhaps all, *TM3* balancer chromosomes based on the lethality of two different *TM3* chromosomes in combination with deficiencies for the region including *Df(3R)c3G2 = Df(3R)89A2-3;89A4-5*.

l(3)89B-E

Results of deficiency and complementation analysis of 62 lethal and eight visible mutations that fail to complement *Df(3R)bxd100 = Df(3R)89B5-6;89E2-3* plus 35 lethals and ten viable mutations that fail to complement *Df(3R)P15 = Df(3R)D9-E1;89E7-8* (Tiong, Bone, and Whittle, 1985, Mol. Gen. Genet. 200: 335-42) and 39 mutations that fail to complement *Df(3R)P115 = Df(3R)D9-E1;89E4-5* [Sánchez-Herrero, Vernós, Marco, and Morata, 1985, Nature (London) 313: 108-13].

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(3)89Ba</i>	3-[58]	89B5-8	<i>Df(3R)bxd100</i>	<i>Df(3R)P115</i>	
<i>l(3)89Bb</i>	3-[58]	89B7-C2	<i>Df(3R)P115</i>	<i>Df(3R)ss-a</i>	
<i>l(3)89Bc</i>	3-[58]	89B7-C2	<i>Df(3R)P115</i>	<i>Df(3R)ss-a</i>	
<i>l(3)89Bd</i>	3-[58]	89B7-C2	<i>Df(3R)ss-a</i>	<i>Df(3R)P10</i>	
<i>l(3)89Be</i>	3-[58]	89B7-C2	<i>Df(3R)ss-a</i>	<i>Df(3R)P10</i>	
<i>l(3)89Bf</i>	3-[58]	89B7-C2	<i>Df(3R)ss-a</i>	<i>Df(3R)P10</i>	
<i>l(3)89Bg</i>	3-[58]	89B7-C2	<i>Df(3R)ss-a</i>	<i>Df(3R)P10</i>	
<i>l(3)89Bh</i>	3-[58]	89B7-C2	<i>Df(3R)ss-a</i>	<i>Df(3R)P10</i>	
<i>l(3)89Bi</i>	3-[58]	89B7-C2	<i>Df(3R)ss-a</i>	<i>Df(3R)P10</i>	
<i>l(3)89Bj</i>	3-[58]	89B7-C2	<i>Df(3R)ss-a</i>	<i>Df(3R)P10</i>	
<i>l(3)89Bk</i>	3-[58]	89B7-C2	<i>Df(3R)ss-a</i>	<i>Df(3R)P10</i>	
<i>l(3)89Bl</i>	3-[58]	89B7-C2	<i>Df(3R)ss-a</i>	<i>Df(3R)P10</i>	
<i>l(3)89Ca</i>	3-[58]	89C	<i>Df(3R)P10</i> <i>Df(3R)sbd104</i>		
<i>l(3)89Cb</i>	3-[58]	89C	<i>Df(3R)P10</i> <i>Df(3R)sbd104</i>		
<i>l(3)89Cc</i>	3-[58]	89C	<i>Df(3R)P10</i> <i>Df(3R)sbd104</i>		
<i>l(3)89Cd</i>	3-[58]	89C	<i>Df(3R)ss-a</i>	<i>Df(3R)sbd104</i>	
<i>l(3)89Da</i>	3-[59]	89D9-E2	<i>Df(3R)P2</i>	<i>Df(3R)Ubx109</i>	
<i>l(3)89Db</i>	3-[59]	89D9-E2	<i>Df(3R)Ubx109</i>	<i>Df(3R)P9</i>	
<i>l(3)89Dc</i>	3-[59]	89D9-E2	<i>Df(3R)Ubx109</i>	<i>Df(3R)P9</i>	
<i>l(3)89Ea</i>	3-[59]	89E1-3	<i>Df(3R)P9</i> <i>Df(3R)bxd100</i>		<i>l(3)lb</i>
<i>l(3)89Eb</i>	3-58.8	89E1-3	<i>Df(3R)P9</i> <i>Df(3R)bxd100</i>		Ubx
<i>l(3)89Ec</i>	3-58.8	89E2-3	<i>Df(3R)Ubx109</i>	<i>Df(3R)bxd100</i>	abdA
<i>l(3)89Ed</i>	3-58.8	89E2-5	<i>Df(3R)P9</i>	<i>Df(3R)Ubx109</i>	AbdB
<i>l(3)89Ee</i>	3-[59]	89E2-5	<i>Df(3R)P9</i>	<i>Df(3R)Ubx109</i>	<i>l(3)rB</i>
<i>l(3)89Ef</i>	3-[59]	89E4-8	<i>Df(3R)P115</i>	<i>Df(3R)P9</i>	
<i>l(3)89Eg</i>	3-[59]	89E4-8	<i>Df(3R)P115</i>	<i>Df(3R)P9</i>	
<i>l(3)89Eh</i>	3-[59]	89E4-8	<i>Df(3R)P115</i>	<i>Df(3R)P9</i>	
<i>l(3)89Ei</i>	3-[59]	89E4-8	<i>Df(3R)P115</i>	<i>Df(3R)P9</i>	
<i>l(3)89Ej</i>	3-59.2	89E7-F1	<i>Df(3R)C4</i>	<i>Df(3R)P115</i>	

l(3)89Ba-Dc

allele	origin	discoverer	synonym	ref α
<i>l(3)89Ba¹</i>	EMS	Tiong	<i>l(3)S8</i>	1
<i>l(3)89Ba²</i>	EMS	Tiong	<i>l(3)S15</i>	1
<i>l(3)89Ba³</i>	EMS	Tiong		1
<i>l(3)89Bb¹</i>	EMS	Tiong	<i>l(3)S38</i>	1
<i>l(3)89Bb²</i>	EMS	Tiong	<i>l(3)S54</i>	1
<i>l(3)89Bc¹</i>	EMS	Tiong	<i>l(3)S78</i>	1
<i>l(3)89Bd¹</i>	EMS	Tiong	<i>l(3)S2</i>	1
<i>l(3)89Bd²</i>	EMS	Tiong	<i>l(3)S43</i>	1

allele	origin	discoverer	synonym	ref ^α
<i>l(3)89Be</i> ¹	EMS	Tiong	<i>l(3)S9</i>	1
<i>l(3)89Bf</i> ¹	EMS	Tiong	<i>l(3)S12</i>	1
<i>l(3)89Bf</i> ²	EMS	Tiong	<i>l(3)S26</i>	1
<i>l(3)89Bf</i> ³	EMS	Tiong		1
<i>l(3)89Bf</i> ⁴	EMS	Tiong		1
<i>l(3)89Bf</i> ⁵	EMS	Tiong		1
<i>l(3)89Bf</i> ⁶	EMS	Tiong		1
<i>l(3)89Bf</i> ⁷	EMS	Tiong		1
<i>l(3)89Bg</i> ¹	γ ray	Tiong	<i>l(3)S4</i>	1
<i>l(3)89Bg</i> ²	EMS	Tiong	<i>l(3)S36</i>	1
<i>l(3)89Bg</i> ³	EMS	Tiong		1
<i>l(3)89Bh</i> ¹	EMS	Tiong	<i>l(3)S37</i>	1
<i>l(3)89Bi</i> ¹	EMS	Tiong	<i>l(3)S74</i>	1
<i>l(3)89Bj</i> ¹	EMS	Tiong	<i>l(3)S77</i>	1
<i>l(3)89Bj</i> ²	EMS	Tiong	<i>l(3)S81</i>	1
<i>l(3)89Bk</i> ¹	EMS	Tiong	<i>l(3)S80</i>	1
<i>l(3)89Bl</i> ¹	EMS	Tiong	<i>l(3)S87</i>	1
<i>l(3)89Ca</i> ¹	EMS	Tiong	<i>l(3)S40</i>	1
<i>l(3)89Ca</i> ²	EMS	Tiong	<i>l(3)S89</i>	1
<i>l(3)89Ca</i> ³	EMS	Tiong		1
<i>l(3)89Cb</i> ¹	EMS	Tiong	<i>l(3)S53</i>	1
<i>l(3)89Cc</i> ¹	EMS	Tiong	<i>l(3)S73</i>	1
<i>l(3)89Cd</i> ¹	EMS	Tiong	<i>l(3)S59</i>	1
<i>l(3)89Cd</i> ²	EMS	Tiong	<i>l(3)S88</i>	1
<i>l(3)89Cd</i> ³	EMS	Tiong	<i>l(3)S93</i>	1
<i>l(3)89Da</i> ¹	EMS	Tiong	<i>l(3)S22</i>	1
<i>l(3)89Db</i> ¹	EMS	Tiong	<i>l(3)S10</i>	1
<i>l(3)89Db</i> ²	EMS	Tiong	<i>l(3)S28</i>	1
<i>l(3)89Db</i> ³	EMS	Tiong	<i>l(3)S</i>	1
<i>l(3)89Db</i> ⁴	EMS	Tiong	<i>l(3)S</i>	1
<i>l(3)89Db</i> ⁵	EMS	Tiong	<i>l(3)S</i>	1
<i>l(3)89Db</i> ⁶	EMS	Tiong	<i>l(3)S</i>	1
<i>l(3)89Db</i> ⁷	EMS	Tiong	<i>l(3)S</i>	1
<i>l(3)89Dc</i> ¹	EMS	Tiong	<i>l(3)S59</i>	1
<i>l(3)89Dc</i> ²	EMS	Tiong	<i>l(3)S88</i>	1
<i>l(3)89Dc</i> ³	EMS	Tiong	<i>l(3)S93</i>	1

^α 1 = Tiong, Bone, and Whittle, 1985, Mol. Gen. Genet. 200: 335-42.

l(3)89Ea

phenotype: Embryonic morphology normal.
alleles:

allele	origin	discoverer	synonym	ref ^α
<i>l(3)89Ea</i> ¹	EMS			1
<i>l(3)89Ea</i> ²	EMS			1
<i>l(3)89Ea</i> ³	EMS			1
<i>l(3)89Ea</i> ⁴	EMS			1
<i>l(3)89Ea</i> ⁵	EMS			1
<i>l(3)89Ea</i> ⁶	EMS			1
<i>l(3)89Ea</i> ⁷	EMS			1
<i>l(3)89Ea</i> ⁸	EMS			1
<i>l(3)89Ea</i> ⁹	EMS			1
<i>l(3)89Ea</i> ¹⁰	X ray			1
<i>l(3)89Ea</i> ¹¹	EMS	Tiong	<i>l(3)S3</i>	2
<i>l(3)89Ea</i> ¹²	EMS	Tiong	<i>l(3)S27</i>	2
<i>l(3)89Ea</i> ¹³	EMS	Tiong	<i>l(3)47</i>	2
<i>l(3)89Ea</i> ¹⁴	EMS	Tiong		2
<i>l(3)89Ea</i> ¹⁵	EMS	Tiong		2
<i>l(3)89Ea</i> ¹⁶	EMS	Tiong		2
<i>l(3)89Ea</i> ¹⁷	EMS	Tiong		2

allele	origin	discoverer	synonym	ref ^α
<i>l(3)89Ea</i> ¹⁸	EMS	Tiong		2
<i>l(3)89Ea</i> ¹⁹	EMS	Tiong		2
<i>l(3)89Ea</i> ²⁰	EMS	Tiong		2
<i>l(3)89Ea</i> ²¹	EMS	Tiong		2
<i>l(3)89Ea</i> ²²	EMS	Tiong		2
<i>l(3)89Ea</i> ²³	EMS	Tiong		2
<i>l(3)89Ea</i> ²⁴	EMS	Tiong		2
<i>l(3)89Ea</i> ²⁵	EMS	Tiong		2
<i>l(3)89Ea</i> ²⁶	EMS	Tiong		2
<i>l(3)89Ea</i> ²⁷	EMS	Tiong		2
<i>l(3)89Ea</i> ²⁸	EMS	Tiong		2
<i>l(3)89Ea</i> ²⁹	EMS	Tiong		2
<i>l(3)89Ea</i> ³⁰	EMS	Tiong		2
<i>l(3)89Ea</i> ³¹	EMS	Tiong		2
<i>l(3)89Ea</i> ³²	EMS	Tiong		2
<i>l(3)89Ea</i> ³³	EMS	Tiong		2
<i>l(3)89Ea</i> ³⁴	EMS	Tiong		2
<i>l(3)89Ea</i> ³⁵	EMS	Tiong		2
<i>l(3)89Ea</i> ³⁶	EMS	Tiong		2

^α 1 = Sánchez-Herrero, Vernós, Marco, and Morata, 1985, Nature (London) 313: 108-13; 2 = Tiong, Bone, and Whittle, 1985, Mol. Gen. Genet. 200: 335-42.

l(3)89Ee

phenotype: Hemizygous embryos normal in morphology.
alleles:

allele	origin	discoverer	synonym	ref ^α
<i>l(3)89Ee</i> ¹	EMS			1
<i>l(3)89Ee</i> ²	EMS			1
<i>l(3)89Ee</i> ³	X ray			1
<i>l(3)89Ee</i> ⁴	EMS	Tiong	<i>l(3)S56</i>	2
<i>l(3)89Ee</i> ⁵	EMS	Tiong	<i>l(3)S72</i>	2
<i>l(3)89Ee</i> ⁶	EMS	Tiong		2
<i>l(3)89Ef</i> ¹	EMS	Tiong	<i>l(3)S68</i>	2
<i>l(3)89Ef</i> ²	EMS	Tiong	<i>l(3)S85</i>	2
<i>l(3)89Eg</i> ¹	EMS	Tiong	<i>l(3)S79</i>	2
<i>l(3)89Eh</i> ¹	EMS	Tiong	<i>l(3)S92</i>	2
<i>l(3)89Eh</i> ²	EMS	Tiong	<i>l(3)S64</i>	2
<i>l(3)89Ei</i> ¹	γ ray	Tiong	<i>l(3)H4</i>	2
<i>l(3)89Ej</i> ¹	EMS	Ivy		

^α 1 = Sánchez-Herrero, Vernós, Marco, and Morata, 1985, Nature (London) 313: 108-13; 2 = Tiong, Bone, and Whittle, 1985, Mol. Gen. Genet. 200: 335-42.

l(3)89Ej

origin: Induced by ethyl methanesulfonate.
references: Ivy, 1981, Ph.D. Thesis, University of California, San Diego.

phenotype: Homozygotes die as embryos; cell lethal in cuticular clones. Heterozygous males display approximately 4% nondisjunction of both sex and fourth chromosomes; diplo-chromosome exceptions more frequent than nullo-chromosome exceptions. Slight evidence for autosomal nondisjunction and second-division sex-chromosome nondisjunction in males and sex- and fourth-chromosome nondisjunction in females.

DEFICIENCY MAP OF REGION 89

side	breakpoint	variant	DNA coordinates
left	88F7-89A1	<i>cv-c</i> <i>Tm2</i>	<i>Df(3R)Po4</i>

side	breakpoint	variant	DNA coordinates
		Act88F	
left	88F9-89A1	<i>Df(3R)sbd105</i>	
left	89A1-2	<i>Df(3R)Po2</i>	
left	89A1-2	<i>Df(3R)Po3</i>	
		Po	
		Aldox1	
left	89A2-3	<i>Df(3R)c3G2</i>	
		c(3)G	
		l(3)89Aa	
		rec1	
right	89A4-5	<i>Df(3R)c3G2</i>	
right	89A11-13	<i>Df(3R)Po2</i>	
right	89A11-13	<i>Df(3R)Po3</i>	
right	89A11-13	<i>Df(3R)Po4</i>	
		mor	
		srp	
left	89B4	<i>Df(3R)sbd45</i>	
		pnr	
left	89B9-10	<i>Df(3R)sbd26</i>	
right	89B9-10	<i>Df(3R)sbd105</i>	
		sbd	
		Sb	
right	89B10	<i>Df(3R)sbd45</i>	
right	89C7-D1	<i>Df(3R)sbd26</i>	
left	89B5-6	<i>Df(3R)bxd100</i>	
		l(3)89Ba	
left	89B7-8	<i>Df(3R)P115</i>	
		l(3)Bb	
		l(3)Bc	
left		<i>Df(3R)ss-a</i>	
		l(3)Bd	
		l(3)Be	
		l(3)Bf	
		l(3)Bg	
		l(3)Bh	
		l(3)Bi	
		l(3)Bj	
		l(3)Bk	
		l(3)Bl	
left	89C1-2	<i>Df(3R)P10</i>	
		l(3)Ca	
		l(3)Cb	
		l(3)Cc	
		ss	
right	89C	<i>Df(3R)sbd104</i>	
		l(3)Cd	
right		<i>Df(3R)ss-a</i>	
left	89D9-E1	<i>Df(3R)P2</i>	
		l(3)89Da	
left	89D1-2	<i>Df(3R)Ubx109</i>	
		Fas1	
		l(3)89Db	
		l(3)89Dc	
left	89D9-E1	<i>Df(3R)P9</i>	
		l(3)89Ea	
		Ubx	
left	89E2-3	<i>Df(3R)bxd110</i>	-27 kb
right	89E2-3	<i>Df(3R)bxd100</i>	-18 kb
left	89E3-4	<i>Df(3R)bxd111</i>	-3 kb
		abdA	
right	89E1-2	<i>Df(3R)P10</i>	+35 to +36 kb
right	89E2-3	<i>Df(3R)P2</i>	+78 to +81 kb
left	89E3-4	<i>Df(3R)Ubx109</i>	+86 to +93 kb
left	89E2-3	<i>Df(3R)C4</i>	+133.5 to +137 kb
		AbdB	
		l(3)89Ee	
right	89E4-5	<i>Df(3R)P9</i>	+225 to +230 kb
		l(3)89Ef	
		l(3)89Eg	
		l(3)89Eh	
		l(3)89Ei	
right	89E7-8	<i>Df(3R)P115</i>	
		l(3)89Ej	
right	89F2-4	<i>Df(3R)C4</i>	
right	90B	<i>Df(3R)bxd111</i>	

l(3)91C

A series of lethals that fail to complement a deficiency that includes *Cha*; investigated by Myers and Gelbart (unpublished).

locus	genetic		cytological		synonym
	location	location	included in	excluded from	
l(3)91Ca	3-{65}	91C2-D1	<i>Df(3R)Cha1</i>	<i>Df(3R)Cha6</i>	<i>l(3)CR1</i>
l(3)91Cb	3-{65}	91C2-D1	<i>Df(3R)Cha6</i>	<i>Df(3R)Cha9</i>	<i>l(3)CR2</i>
<i>l(3)91Cc</i>	3-64.6	91C7-D8	<i>Df(3R)Cha6</i>		Cha
			<i>Df(3R)Cha9</i>		
l(3)91Cd	3-{65}	91C7-D1	<i>Df(3R)5</i>	<i>Df(3R)Cha10</i>	<i>l(3)CR3</i>
allele	origin synonym				
l(3)91Ca¹	EMS	<i>l(3)CR1</i>	<i>M29</i>		
l(3)91Ca²	EMS	<i>l(3)CR1</i>	<i>M73</i>		
l(3)91Cb¹	EMS	<i>l(3)CR2</i>	<i>M19</i>		
l(3)91Cb²	EMS	<i>l(3)CR2</i>	<i>M24</i>		
l(3)91Cb³	EMS	<i>l(3)CR2</i>	<i>M33</i>		
l(3)91Cb⁴	EMS	<i>l(3)CR2</i>	<i>M44</i>		
l(3)91Cb⁵	EMS	<i>l(3)CR2</i>	<i>M76</i>		
l(3)91Cd¹	EMS	<i>l(3)CR3</i>	<i>M43</i>		
l(3)91Cd²	EMS	<i>l(3)CR3</i>	<i>M45</i>		
l(3)91Cd³	EMS	<i>l(3)CR3</i>	<i>M49</i>		

DEFICIENCY MAP OF REGION 91C-92A

side	breakpoint	variant
left	91B3	<i>Df(3R)Cha5</i>
left	91C2-3	<i>Df(3R)Cha1</i>
		l(3)91Ca
left		<i>Df(3R)Cha6</i>
		l(3)91Cb
left	91C7-D1	<i>Df(3R)Cha9</i>
		Cha
right		<i>Df(3R)Cha6</i>
		l(3)91Cd
right	91D1	<i>Df(3R)Cha5</i>
right	92A1	<i>Df(3R)Cha1</i>
right	92A2	<i>Df(3R)Cha9</i>

l(3)91F-92A

Two independent saturation studies carried out by groups at Köln and Bloomington, using different deficiencies for both selection and mapping of lethal mutations; the ambiguities resolved by Lehmann using complementation and deficiency mapping to a common set of deficiencies. The order *l(3)91Fa nos l(3)91Fb l(3)91Fe l(3)91Ff l(3)92Aa Dl* also determined by recombination.

locus	genetic		cytological		synonym
	location	location	included in	excluded from	
l(3)91Fa	3-{65}	91F11-13	<i>Df(3R)DI-X43</i>	<i>Df(3R)DI-A143</i>	<i>C2</i>
l(3)91Fb	3-{65}	91F13	<i>Df(3R)DI-A143</i>	<i>Df(3R)DI-HD28</i>	<i>C3</i>
l(3)91Fc	3-{65}	91F13	<i>Df(3R)DI-A143</i>	<i>Df(3R)DI-HD28</i>	<i>C5</i>
l(3)91Fe	3-{65}	91F13	<i>Df(3R)DI-A143</i>	<i>Df(3R)DI-HD28</i>	
l(3)91Ff	3-{65}	91F13-92A2	<i>Df(3R)DI-HD28</i>	<i>Df(3R)DI-KX12</i>	
l(3)92Aa	3-{66}	92A2	<i>Df(3R)Cha1a</i>		<i>C4, l(3)91Fd</i>
			<i>Df(3R)DI-KX12</i>		
<i>l(3)92Ab</i>	3-66.2				DI

allele	origin	synonym	ref ^α
<i>l(3)91Fa</i>¹	EMS	BE40	1
<i>l(3)91Fa</i>²	EMS	BE41	1
<i>l(3)91Fa</i>³	EMS	BE42	1
<i>l(3)91Fa</i>⁴	EMS	BE43	1
<i>l(3)91Fa</i>⁵	EMS	BE44	1
<i>l(3)91Fa</i>⁶	EMS	C2-8	2
<i>l(3)91Fa</i>⁷	EMS	C2-10	2
<i>l(3)91Fa</i>⁸	EMS	C2-13	2
<i>l(3)91Fa</i>⁹	EMS	C2-14	2
<i>l(3)91Fa</i>¹⁰	EMS	C2-15	2
<i>l(3)91Fa</i>¹¹	EMS	C2-22	2
<i>l(3)91Fa</i>¹²	EMS	C2-23	2
<i>l(3)91Fb</i>¹	EMS	BE50	1
<i>l(3)91Fb</i>²	EMS	BE51	1
<i>l(3)91Fb</i>³	EMS	BE52	1
<i>l(3)91Fb</i>⁴	EMS	C3-16	2
<i>l(3)91Fb</i>⁵	EMS	C3-17	2
<i>l(3)91Fb</i>⁶	EMS	C3-19	2
<i>l(3)91Fc</i>¹	EMS	BE60	1
<i>l(3)91Fc</i>²	EMS	BE61	1
<i>l(3)91Fc</i>³	EMS	BE62	1
<i>l(3)91Fe</i>¹	EMS	BE80	1
<i>l(3)91Fe</i>²	EMS	BE81	1
<i>l(3)91Fe</i>³	EMS	C4-4	2
<i>l(3)91Fe</i>⁴	EMS	C4-4	2
<i>l(3)91Fe</i>⁵	EMS	C4-7	2
<i>l(3)91Fe</i>⁶	EMS	C4-9	2
<i>l(3)91Fe</i>⁷	EMS	C4-18	2
<i>l(3)91Ff</i>¹	EMS	BE90	1
<i>l(3)91Ff</i>²	EMS	BE91	1
<i>l(3)92Aa</i>¹	EMS	BE70	1
<i>l(3)92Aa</i>²	EMS	BE71	1
<i>l(3)92Aa</i>³	EMS	BE72	1
<i>l(3)92Aa</i>⁴	EMS	C4-1	2
<i>l(3)92Aa</i>⁵	EMS	C4-2	2
<i>l(3)92Aa</i>⁶	EMS	C4-6	2
<i>l(3)92Aa</i>⁷	EMS	C4-20	2
<i>l(3)92Aa</i>⁸	EMS	C4-21	2

^α 1 = Alton, Fechtel, Terry, Meikle, and Muskavitch, 1988, Genetics 118: 235-45; 2 = Vässin and Campos-Ortega, 1987, Genetics 116: 433-45.

DEFICIENCY MAP OF REGION 91-92

side	breakpoint	variant
left	91F1-2	<i>Df(3R)DI-KX12</i>
left	91F11	<i>Df(3R)X43</i>
left	91F13	<i>l(3)91Fa</i> <i>Df(3R)DI-A143</i> <i>l(3)91Fb</i> <i>nos</i> <i>l(3)91Fe</i> <i>l(3)91Fc</i>
left	91F6-13	<i>Df(3R)DI-HD28</i> <i>l(3)91Ff</i>
left	92A2	<i>Df(3R)DI-KX12</i> <i>l(3)92Aa</i>
right	92A2	<i>Df(3R)ChaM1a</i> <i>DI</i>
right	92A2	<i>Df(3R)Bxd110</i>
right	92A2-3	<i>Df(3R)DI-A143</i>
right	92A2-3	<i>Df(3R)DI-HD28</i>
right	92A3-6	<i>Df(3R)DI-BX12</i>
right	92A4	<i>Df(3R)DI-KX12</i>
right	92A8-10	<i>Df(3R)DI-X43</i>

l(3)93

Eighteen loci that have mutated to 60 lethal alleles plus one locus inferred from the lethality of heterozygotes for a pair of overlapping deficiencies. Mutations were induced with a variety of mutagenic agents and selected on the basis of inviability in combination with either *Df(3R)e-Gp4 = Df(3R)93B11-13;93D7-9* or *Df(3R)e-H4 = Df(3R)93C3-6,93F6-8*, and therefore are confined to the region 93B11 to 93F8. The region also contains the locus of ebony and the 93D heat-shock locus; none of the mutants affected the heat-shock gene, but the locus inferred from the inviability of heterozygotes for two overlapping deficiencies, *l(3)93De*, appears to be responsible for the heat-shock puff (Mohler and Pardue, 1984, Genetics 106: 249-65).

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(3)93Ba</i>	3-{70}	93B11-C6	<i>Df(3R)e-F3</i>	<i>Df(3R)e-F4</i>	<i>l(3)er7</i>
<i>l(3)93Bb</i>	3-{70}	93B11-C6	<i>Df(3R)e-F3</i>	<i>Df(3R)e-F4</i>	<i>l(3)er4</i>

allele	origin	synonym	comments
<i>l(3)93Ba</i>¹	EMS	<i>l(3)er7</i> EC3	
<i>l(3)93Ba</i>²	EMS	<i>l(3)er7</i> EC4	
<i>l(3)93Ba</i>³	EMS	<i>l(3)er7</i> EC8	
<i>l(3)93Ba</i>⁴	EMS	<i>l(3)er7</i> EC9	
<i>l(3)93Ba</i>⁵	EMS	<i>l(3)er7</i> EC10	
<i>l(3)93Ba</i>⁶	EMS	<i>l(3)er7</i> EC30	
<i>l(3)93Bb</i>¹	EMS	<i>l(3)er4</i> EC2	
<i>l(3)93Bb</i>²	EMS	<i>l(3)er4</i> EC16	outspread wings
<i>l(3)93Bb</i>³	EMS	<i>l(3)er4</i> EC25	
<i>l(3)93Bb</i>⁴	EMS	<i>l(3)er4</i> EC27	
<i>l(3)93Bb</i>⁵	EMS	<i>l(3)er4</i> EC33	
<i>l(3)93Bb</i>⁶	γ ray	<i>l(3)er4</i> GC15	
<i>l(3)93Bb</i>⁷	γ ray	<i>l(3)er4</i> GC16	
<i>l(3)93Bb</i>⁸	γ ray	<i>l(3)er4</i> GC17	

locus	genetic location	cytological location	included in	excluded from	synonym
<i>l(3)93Ca</i>	3-{70}	93C3-6	<i>Df(3R)e-F4</i>	<i>Df(3R)e-D7</i>	<i>l(3)er6</i>
<i>l(3)93Cb</i>	3-{70}	93C3-6	<i>Df(3R)e-D7</i>	<i>Df(3R)e-H4</i>	<i>l(3)er5</i>
<i>l(3)93Cc</i>	3-70.5	93C3-D4	<i>Df(3R)e-H4</i>	<i>Df(3R)e-R1</i>	<i>l(3)er14</i>
<i>l(3)93Cd</i>	3-{70}	93C3-D4	<i>Df(3R)e-H4</i>		<i>l(3)er2</i>

allele	origin	synonym	comments
<i>l(3)93Ca</i>¹	EMS	<i>l(3)er6</i> EC7	
<i>l(3)93Ca</i>²	EMS	<i>l(3)er6</i> EC11	
<i>l(3)93Ca</i>³	γ ray	<i>l(3)er6</i> GC20	
<i>l(3)93Cb</i>¹	EMS	<i>l(3)er5</i> EC1	
<i>l(3)93Cb</i>²	EMS	<i>l(3)er5</i> EC18	
<i>l(3)93Cb</i>³	EMS	<i>l(3)er5</i> EC26	
<i>l(3)93Cb</i>⁴	γ ray	<i>l(3)er5</i> GC18	<i>In(3R)81F,93C</i>
<i>l(3)93Cb</i>⁵	γ ray	<i>l(3)er5</i> GC19	
<i>l(3)93Cb</i>⁶	γ ray	<i>l(3)er5</i> GC21	<i>In(3R)91A,93C</i>
<i>l(3)93Cc</i>¹	DEB	<i>l(3)er14</i> DC1	
<i>l(3)93Cd</i>¹	EMS	<i>l(3)er2</i> EC21	
<i>l(3)93Cd</i>²	EMS	<i>l(3)er2</i> EC24	temperature sensitive
<i>l(3)93Cd</i>³	γ ray	<i>l(3)er2</i> GC11	

locus	genetic		cytological		
	location	location	included in	excluded from	synonym
			<i>Df(3R)e-R1</i>		
I(3)93Da	3-{70}	93D2-6	<i>Df(3R)e-H5</i>	<i>Df(3R)e-R1</i>	<i>l(3)er1</i>
I(3)93Db	3-{70}	93D4-9	<i>Df(3R)e-Gp4</i>	<i>Df(3R)e-H5</i>	<i>l(3)er8</i>
I(3)93Dc	3-{70}	93D4-9	<i>Df(3R)e-Gp4</i>	<i>Df(3R)e-H5</i>	<i>l(3)er11</i>
I(3)93Dd	3-{70}	93D4-9	<i>Df(3R)e-Gp4</i>	<i>Df(3R)e-H5</i>	<i>l(3)er9</i>
I(3)93De	3-{70}	93D4-9	<i>Df(3R)e-Gp4</i>		<i>l(3)er3</i>
			<i>Df(3R)e-GC14</i>		
I(3)93Df	3-{70}	93D7-10	<i>Df(3R)e-F2</i>	<i>Df(3R)e-Gp4</i>	<i>l(3)er19</i>
I(3)93Dg	3-{70}	93D7-10	<i>Df(3R)e-F2</i>	<i>Df(3R)e-Gp4</i>	<i>l(3)er13</i>
I(3)93Dh	3-{70}	93D7-10	<i>Df(3R)e-F2</i>	<i>Df(3R)e-Gp4</i>	<i>l(3)er16</i>
I(3)93Di	3-{70}	93D7-10	<i>Df(3R)e-F1</i>	<i>Df(3R)e-GC9</i>	<i>l(3)er12</i>
I(3)93Dj	3-{70}	93D7-E8	<i>Df(3R)e-F3</i>	<i>Df(3R)e-F1</i>	<i>l(3)er10</i>

allele	origin	synonym	comments
I(3)93Da¹	EMS	<i>l(3)er1</i> EC19	10% survival
I(3)93Da²	EMS	<i>l(3)er1</i> EC20	
I(3)93Da³	DEB	<i>l(3)er1</i> DC3	
I(3)93Da⁴	γ ray	<i>l(3)er1</i> GC8	
I(3)93Da⁵	γ ray	<i>l(3)er1</i> GC31	
I(3)93Db¹	EMS	<i>l(3)er8</i> EC13	
I(3)93Db²	EMS	<i>l(3)er8</i> EC17	
I(3)93Db³	EMS	<i>l(3)er8</i> EC28	
I(3)93Db⁴	EMS	<i>l(3)er8</i> EC32	M-like with <i>ln(3)</i> GC25
I(3)93Db⁵	EMS	<i>l(3)er8</i> EC37	
I(3)93Dc¹	EMS	<i>l(3)er11</i> EC36	
I(3)93Dc²	EMS	<i>l(3)er11</i> EC38	warped opaque wings
I(3)93Dd¹	EMS	<i>l(3)er9</i> EC15	viable with <i>l(3)93Dd²</i>
I(3)93Dd²	γ ray	<i>l(3)er9</i> GC23	<i>ln(3)81F;93D</i> ; viable with roughened eyes
<i>l(3)93De</i>		Hsr93D	inferred from
		<i>l(3)er3</i>	deficiency analysis
I(3)93Df¹	γ ray	<i>l(3)er19</i> GC30	warped wings; cannot stand or walk; die soon
I(3)93Dg¹	EMS	<i>l(3)er13</i> EC40	
I(3)93Dg²	EMS	<i>l(3)er13</i> EC43	
I(3)93Dh¹	EMS	<i>l(3)er16</i> EC44	
I(3)93Dh²	γ ray	<i>l(3)er16</i> GC27	

I(3R)93Di

All alleles semilethal; survival of different alleles over deficiency varies.

allele	origin	synonym	comments
I(3)93Di¹	EMS	<i>l(3)er12</i> EC35	
I(3)93Di²	EMS	<i>l(3)er12</i> EC39	
I(3)93Di³	γ ray	<i>l(3)er12</i> GC32	
I(3)93Dj¹	EMS	<i>l(3)er10</i> EC34	
I(3)93Dj²	EMS	<i>l(3)er10</i> EC42	
I(3)93Dj³	γ ray	<i>l(3)er10</i> GC24	
I(3)93Dj⁴	γ ray	<i>l(3)er10</i> GC25	<i>ln(3R)93E;94A</i>
I(3)93Dj⁵	γ ray	<i>l(3)er10</i> GC29	
I(3)93Dj⁶	γ ray	<i>l(3)er10</i> GC33	

locus	genetic		cytological		
	location	location	included in	excluded from	synonym
I(3)93Fa	3-{73}	93E6-F8	<i>Df(3R)e-D7</i>	<i>Df(3R)e-F3</i>	<i>l(3)er17</i>
I(3)93Fb	3-{73}	93F6-8	<i>Df(3R)e-H4</i>	<i>Df(3R)e-D7</i>	<i>l(3)er15</i>
I(3)93Fc	3-{73}	93F6-8	<i>Df(3R)e-F2</i>	<i>Df(3R)e-Gp4</i>	<i>l(3)er18</i>

allele	origin	synonym	comments
I(3)93Fa¹	γ ray	<i>l(3)er17</i> GC28	
I(3)93Fa²	DEB	<i>l(3)er17</i> DC4	
I(3)93Fb¹	DEB	<i>l(3)er15</i> DC2	
I(3)93Fc¹	EMS	<i>l(3)er18</i> EC29	warped wings; female sterile
I(3)93Fc²	γ ray	<i>l(3)er18</i> GC26	5% survival

DEFICIENCY MAP OF REGION 93B-F

side	breakpoint	variant
left	93A6-B1	<i>Df(3R)e-F2</i>
left	93B3-5	<i>Df(3R)-R1</i>
left	93B8-13	<i>Df(3R)e-F1</i>
left	93B8-13	<i>Df(3R)e-F3</i>
left	93B11-13	<i>Df(3R)e-GC9</i>
left	93B11-13	<i>Df(3R)e-Gp4</i>
left	93B11-13	<i>Df(3R)e-H5</i>
	93B11-C6	I(3)93Ba
	93B11-C6	I(3)93Bb
left	93C3-6	<i>Df(3R)e-F4</i>
	93C3-6	I(3)93Ca
left	93C	<i>ln(3R)GC18^L GC23^R</i>
left	93C3-6	<i>Df(3R)e-D7</i>
left	93C3-6	<i>Df(3R)e-GC3</i>
left	93C3-6	<i>Df(3R)e-H6</i>
right	93D7-10	<i>Df(3R)e-F1</i>
	93C3-6	I(3)93Cb
left	93C3-6	<i>Df(3R)e-H4</i>
	93C3-D4	I(3)93Cc
	93C3-D4	I(3)93Cd
	93D2-6	e
right	93D2-4	<i>Df(3R)e-R1</i>
	93D2-6	I(3)93Da
right	93D4-6	<i>Df(3R)e-H5</i>
	93D4-9	I(3)93Db
	93D4-9	I(3)93Dc
	93D4-9	I(3)93Dd
left	invisible	<i>Df(3R)GC14</i>
right	93D	<i>ln(3R)GC18^L GC23^R</i>
	93D4-9	Hsr93D^α
right	93D7-9	<i>Df(3R)e-Gp4</i>
	93D7-10	I(3)93Df
	93D7-10	I(3)93Dg
	93D7-10	I(3)93Dh
right	invisible	<i>Df(3R)GC14</i>
right	93D7-10	<i>Df(3R)e-F2</i>
right	93D9-10	<i>Df(3R)e-GC9</i>
	93D9-10	I(3)93Di
right	93D7-10	<i>Df(3R)e-F1</i>
	93D7-E8	I(3)93Dj
right	93E6-11	<i>Dr(3R)e-F3</i>
	93E6-F8	I(3)93Fa
right	93F6-8	<i>Df(3R)e-D7</i>
	93F6-8	I(3)93Fb
	93F6-8	I(3)93Fc
right	93F6-8	<i>Df(3R)e-H4</i>
right	94F11-14	<i>Dr(3R)e-GC3</i>
right	94A	<i>Dr(3R)e-F4</i>
right	94A	<i>Dr(3R)e-H6</i>

^α Inferred from the lethality of *Df(3R)e-Gp4/Df(3R)GC14*; no mutant alleles recovered.

I(3)96F

Four complementation groups encountered in a screen for lethal mutations not complemented by *Df(3R)Esp14 = Df(3R)96F8-9;96F12-13* (Ziemer, Tietze, Knust, and

Campos-Ortega, 1988, Genetics 119: 63-74).

locus	genetic location	cytological location	included in	synonym
<i>I(3)96Fa</i>	3-{90}	96F8-13	<i>Df(3R)Esp14</i>	<i>I(3)C1</i>
<i>I(3)96Fb</i>	3-{90}	96F8-13	<i>Df(3R)Esp14</i>	<i>I(3)C2</i>
<i>I(3)96Fc</i>	3-{90}	96F8-13	<i>Df(3R)Esp14</i>	<i>I(3)C3</i>
<i>I(3)96Fd</i>	3-{90}	96F8-13	<i>Df(3R)Esp14</i>	<i>E(spl)</i>

allele origin synonym

***I(3)96Fa*¹** EMS *I(3)B103*

***I(3)96Fb*¹** EMS *I(3)A3*

***I(3)96Fb*²** EMS *I(3)B17*

***I(3)96Fc*¹** EMS *I(3)B105*

I(3)99D-E

Eleven lethal and one dominant flightless complementation group identified among 29 mutations uncovered by *Df(3R)ca124 = Df(3R)99A2-3;99E4-5* but distal to the breakpoint of *T(Y;3)B81 = T(Y;3)YL;99D3* (Warmke, Kreuz, and Falkenthal, 1989, Genetics 122: 139-51).

locus	genetic location	cytological location	included in α	excluded from α
<i>I(3)99Da</i>	3-101	99D3-9		<i>Dp(3;1)R10</i>
<i>I(3)99Db</i>	3-101	99D3-9		<i>Dp(3;1)R10</i>
<i>I(3)99Dc</i>	3-101	99D3-9		<i>Dp(3;1)R10</i>
<i>I(3)99Dd</i>	3-101	99D3-9		<i>Dp(3;1)R10</i>
<i>I(3)99De</i>	3-101	99D3-9		<i>Dp(3;1)R10</i>
<i>I(3)99Df</i>	3-101	99D3-9		<i>Dp(3;1)R10</i>
<i>I(3)99Dg</i>	3-101	99D3-9		<i>Dp(3;1)R10</i>
<i>I(3)99Dh</i>	3-101	99D3-9		<i>Dp(3;1)R10</i>
<i>I(3)99Di</i>	3-101	99D3-9	<i>Dp(3;1)R10</i>	<i>Dp(3;1)67A</i>
<i>I(3)99Dj</i>	3-101	99D3-9	<i>Dp(3;1)R10</i>	<i>Dp(3;1)67A</i>

I(3)99Ea 3-101 99D9-E3 *Dp(3;1)67A* *Dp(3;1)124P*

α Deficiency mapping based on the terminal deficiency, $3^P Y^D B81$, which is broken in 99D3, partially covered by terminal duplications of varying lengths.

allele origin synonym

***I(3)99Da*¹** EMS *I(3)S5*

***I(3)99Db*¹** EMS *I(3)J2*

***I(3)99Db*²** EMS *I(3)SF1*

***I(3)99Db*³** EMS *I(3)S*

***I(3)99Dc*¹** EMS *I(3)L1*

***I(3)99Dc*²** EMS *I(3)SF5*

***I(3)99Dd*¹** EMS *I(3)J4*

***I(3)99Dd*²** EMS *I(3)SF6*

***I(3)99De*¹** EMS *I(3)J6*

***I(3)99Df*¹** EMS *I(3)E38*

***I(3)99Dg*¹** EMS *I(3)L6*

***I(3)99Dg*²** EMS *I(3)L7*

***I(3)99Dg*³** EMS *I(3)SF3*

***I(3)99Dg*⁴** EMS *I(3)SF8*

***I(3)99Dh*¹** EMS *I(3)L5*

***I(3)99Di*¹** EMS *I(3)J5*

***I(3)99Di*²** EMS *I(3)L4*

***I(3)99Di*³** EMS *I(3)SF4*

***I(3)99Di*⁴** EMS *I(3)SF7*

***I(3)99Di*⁵** EMS *I(3)SF9*

allele origin synonym

***I(3)99Dj*¹** EMS *I(3)J7*

***I(3)99Dj*²** EMS *I(3)L3*

***I(3)99Dj*³** EMS *I(3)L7*

***I(3)99Dj*⁴** EMS *I(3)S1*

***I(3)99Dj*⁵** EMS *I(3)S3*

***I(3)99Ea*¹** EMS *I(3)J1*

***I(3)99Ea*²** EMS *I(3)J3*

***I(3)99Ea*³** EMS *I(3)L9*

***I(3)99Ea*⁴** EMS *I(3)S4*

DEFICIENCY MAP OF REGION 99D-E

side breakpoint variant

	99D3	<i>T(Y;3)B81</i> <i>I(3)99Da</i> <i>I(3)99Db</i> <i>I(3)99Dc</i> <i>I(3)99Dd</i> <i>I(3)99De</i> <i>I(3)99Df</i> <i>I(3)99Dg</i> <i>I(3)99Dh</i>
right	99D6-9	<i>Dp(3;1)ca-R10</i> <i>I(3)99Di</i> <i>I(3)99Dj</i>
right	99D9-E1	<i>Dp(3;1)ca67A</i> <i>I(3)99Ea</i> <i>Mic2</i>
right	99E2-3	<i>Dp(3;1)ca124P</i>

I(3)109

location: 3-36.5.

origin: Induced by ethyl methanesulfonate.

references: Ferrus and Kankel, 1981, Dev. Biol. 85: 485-504.

phenotype: Homozygous lethal; cell viable; homozygous cells 2-3 times normal size.

I(3)1215

location: 3-50.6.

origin: Induced by ethyl methanesulfonate.

discoverer: Shearn.

references: Bryant, Girton, and Martin, 1980, Insect Biology in the Future (M. Locke and D.S. Smith, eds.). Academic Press, New York, pp. 517-42 (fig.).

Held, Duarte, and Derakhshanian, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 145-57.

phenotype: Homozygotes die as pharate adults. Discs giving rise to segmented appendages develop abnormally; legs much shorter than normal; normal numbers of foreshortened segments with reduced numbers of bristles. Tibias of all legs show mirror-image duplication of distal elements with inverted polarity with indications of incipient joint development at the plane of symmetry; other segments not so affected although some pattern deficiencies and bristles with reversed polarity are observed. Tarsi unsegmented. Antennal joints either abnormal or absent; second segment with reduced number of bristles.

I(3)a

location: 3-81.6.

origin: Spontaneous in *In(3R)C*.

discoverer: Morgan, 111.

synonym: *I(3)l*.

references: Muller, 1918, Genetics 3: 422-99.

phenotype: Lethal homozygous. Reduced recovery of *M(3)95A*-bearing daughters from *In(3R)C*, *l(3)a*-*M(3)95A* females (Schultz). RK3.

other information: Results of Bridges interpreted to show allelism to *M(3)95A*³ (3-79.7); may have been related to maternal effect described by Schultz. Position based on crosses by Muller (1918), in which he used *l(3)a* separated from *In(3R)C*.

l(3)ac: lethal (3) accessory

location: 3- (midregion).

discoverer: Schultz, 25g.

phenotype: Enhances maternal effect of *In(3R)C*, *l(3)a* on recovery of *M(3)95A* daughters from *M(3)95A/In(3R)C*, *l(3)a* mothers (Schultz). RK3.

l(3)AFA

location: 3-72 [*l(3)AFA7*].

origin: Induced by ethyl methanesulfonate in *In(3R)AFA*.

references: Caggese, Caizzi, Morea, Scalenghe, and Ritossa, 1979, Proc. Nat. Acad. Sci. USA 76: 2385-89.

cytology: Placed in 93C3-E2 based on inclusion in *Df(3R)e-D7* and *Df(3R)e-F1*.

other information: Two lethals *l(3)AFA7* and *l(3)AFA17*; allelism not ascertained; also not tested against *l(3)93* lethals of Mohler and Pardue.

l(3)AM1

origin: Induced by ethyl methanesulfonate.

references: Seecof, 1977, Am. Zool. 17: 577.

phenotype: Nearly dominant lethal; only 4% of *l/l* plus *l/+* genotypes survive. Defective in myogenesis; embryo homogenates in culture display normal axon growth but reduced nucleus; axon ratio suggests myogenesis reduced.

****l(3)blo-l: lethal (3) bloated larvae***

location: 3- (to the left of *p*).

discoverer: Bridges, 25k7.

references: Chen, 1929, J. Morphol. 47: 135-99.

phenotype: Larvae become very large and transparent, die in the prepupal stage. Growth of imaginal disks irregular. RK2.

l(3)bt: lethal (3) brain tumor

references: Gateff, 1981, 7th European Drosophila Research Conference Abstracts, p. 42.

phenotype: Heat sensitive; at 29° third-instar larvae develop brain tumors and die either before or after pupation. Optic neuroblasts and their derivatives, the ganglion mother cells, proliferate. Brain enlarged and malformed; grow in malignant fashion when transplanted into normal hosts. TSP 0-12 hours of embryonic life.

l(3)ds: lethal (3) discs small

A series of late-larval, early-pupal lethals characterized by having small imaginal discs; isolated by Shearn and Garren (1974, Proc. Nat. Acad. Sci. USA 71: 1393-97). Originally designated *l(3)B*, but renamed *ds* to conform to the terminology for the same types of mutants recovered on the X chromosome.

locus	genetic location	origin	synonym	can differentiate	α	can support differentiation	β
<i>l(3)ds1</i>	68.0	EMS	<i>l(3)B1</i>			no	
<i>l(3)ds2</i> ¹	42.8	EMS	<i>l(3)B2</i>				
<i>l(3)ds2</i> ²		EMS	<i>l(3)B7</i>				

locus	genetic location	origin	synonym	can differentiate	α	can support differentiation	β
<i>l(3)ds3</i>	81.4	EMS	<i>l(3)B3</i>				
<i>l(3)ds4</i>	50.9	EMS	<i>l(3)B4</i>				
<i>l(3)ds5</i>	47.0	EMS	<i>l(3)B5</i>	yes		no	
<i>l(3)ds6</i>	49.3	EMS	<i>l(3)B6</i>				
<i>l(3)ds8</i>	53.6	NG	<i>l(3)B8</i>				
<i>l(3)ds9</i>	50.8	NG	<i>l(3)B9</i>				
<i>l(3)ds10</i>	35.7	NG	<i>l(3)B10</i>				
<i>l(3)ds11</i>	19.6	NG	<i>l(3)B11</i>				
<i>l(3)ds12</i>	30.9	NG	<i>l(3)B12</i>				
<i>l(3)ds13</i>	74.0	NG	<i>l(3)B13</i>				
<i>l(3)ds14</i>	48.7	ICR	<i>l(3)B14</i>				
<i>l(3)ds15</i> ¹	81.7	ICR	<i>l(3)B15</i>			yes	
<i>l(3)ds15</i> ² γ			<i>l(3)A11</i>				
<i>l(3)ds16</i>	49.0	ICR	<i>l(3)B16</i>				
<i>l(3)ds17</i>	44.1	ICR	<i>l(3)B17</i>				

α Test of the ability of suspensions of embryonic cells transplanted first into adult hosts for proliferation and then into larval hosts for metamorphosis to differentiate into adult cuticular structures.

β Test of ability of mutant larvae to act as hosts for the differentiation of wild-type imaginal discs. Wild-type discs transplanted into *l(3)ds5* hosts, but not into *l(3)ds1* hosts, can grow and differentiate when subsequently transplanted back into wild-type third-instar larvae.

γ Discless.

l(3)dsi: lethal (3) discless

A series of late-larval, early-pupal lethals characterized by the absence of imaginal discs; isolated by Shearn and Garren (1974, Proc. Nat. Acad. Sci. USA 71: 1393-97). Originally designated *l(3)A*, but renamed *dsi* to conform to the terminology for the same types of mutants recovered on the X chromosome.

locus	genetic location	origin	synonym	can differentiate	α	can support differentiation	β
<i>l(3)dsi1</i>	3-25.9	EMS	<i>l(3)A1</i>				
<i>l(3)dsi2</i>	3-82.7	EMS	<i>l(3)A2</i>				
<i>l(3)dsi3</i>	3-50.3	EMS	<i>l(3)A3</i>	no			
<i>l(3)dsi4</i>	3-0.0	EMS	<i>l(3)A4</i>				
<i>l(3)dsi5</i>	3-51.1	EMS	<i>l(3)A5</i>	no		yes	
<i>l(3)dsi6</i>	3-50.3	NG	<i>l(3)A6</i>				
<i>l(3)dsi7</i>	3-70.4	ICR170	<i>l(3)A7</i>				
<i>l(3)dsi8</i>	3-29.7	ICR170	<i>l(3)A8</i>				
<i>l(3)dsi9</i>	3-74.3	ICR170	<i>l(3)A9</i>				
<i>l(3)dsi10</i>	3-		<i>l(3)A10</i>	no		yes	

α Test of the ability of suspensions of embryonic cells transplanted first into adult hosts for proliferation and then into larval hosts for metamorphosis to differentiate into adult cuticular structures.

β Test of ability of mutant larvae to act as hosts for the differentiation of wild-type imaginal discs.

l(3)DTS: lethal (3) Dominant Temperature Sensitive

A group of ten ethyl-methanesulfonate-induced dominant-temperature-sensitive mutations on the third chromosome; heterozygotes are lethal when raised at 29°, but survive rearing at 22°. Only *l(3)DTS2* viable as homozygote at 22° (Holden and Suzuki, 1973, Genetics 73: 445-58). Only *l(3)DTS4* expressed dominant lethality in one dose in triploids.

locus	genetic location	phenotype	lethal phase at 29°
<i>l(3)DTS1</i>	3-78	Minute-like at 22°	LP-A
<i>l(3)DTS2</i>	3-33.4		EP
<i>l(3)DTS3</i> ^α	3-84.5		L3
<i>l(3)DTS4</i>	3-24.5		E-L1
<i>l(3)DTS5</i> ^β	3-44.3		P(<i>hfw</i> -like)
<i>l(3)DTS6</i>	<i>st-p</i> ^P	Minute-like at 22°	P

locus	genetic location	phenotype	lethal phase at 29°
<i>l(3)DTS7</i>	3-42.3		P
*<i>l(3)DTS8</i>	3-55.5		
*<i>l(3)DTS9</i>		dominant female sterile	
*<i>l(3)DTS10</i>		dominant female sterile	

α More detailed description follows.
 β See *l(3)73Ai*.

l(3)DTS3

Heterozygous larvae raised at 29° arrested at the third larval instar for up to two weeks; exhibit grossly hypertrophied ring glands due to the growth of the prothoracic gland cells. The nuclei possess polytene chromosomes which are as large as those of salivary gland nuclei; their banding pattern is similar but not identical to that of salivary gland polytene chromosomes, but the puffing patterns are quite different. Salivary gland chromosomes also hypertrophy and lack the normal ecdysone-induced puffs. Chromosomes in both tissues respond to administration of ecdysone by producing the characteristic set of puffs (Holden and Ashburner, 1978, Chromosoma 68: 205-27). At 29° imaginal discs unable to evert *in situ*; however evert normally if cultured in the presence of ecdysterone or in wild-type hosts. Implantation of a single wild-type ring gland rescues lethality (Holden, Walker, Maroy, Watson, White, and Gausz, 1986, Dev. Genet. 6: 153-62).

l(3)DTS-1165

origin: Induced by ethyl methanesulfonate in *TM2*.
discoverer: Suzuki.
references: Falke and Wright, 1972, DIS 48: 89-91.
phenotype: Among progeny of *TM2*, *l(3)DTS-1165/Sb* by Oregon R crosses raised at 30° no lethal-bearing progeny survive when lethal inherited from father, but survival of lethal heterozyotes is 4% when lethal inherited from mother.

****l(3)e: lethal (3) with ebony***

location: 3- (not located).
origin: Spontaneous in *In(3R)C, e*.
discoverer: Schultz.
phenotype: Dies as fully-developed, normal-appearing imago unable to eclose. RK3A.

l(3)ET: lethal (3) Elaine Tasaka

A series of ethyl-methanesulfonate-induced temperature-sensitive mutations induced by Tasaka and Suzuki (1973, Genetics 74: 509-20). Genetically mapped, but individual positions not given; most clustered between *st* and *Sb*. Heat-sensitive and cold-sensitive mutants as well as alleles sensitive to both high and low temperatures reported.

locus	genetic location	ref ^{α}	type of allele	phenotype
<i>l(3)ET2</i>	2		cold sensitive	
<i>l(3)ET4</i>	1		heat and cold sensitive	adult males die on shift to 28°
<i>l(3)ET5</i>	1, 2		heat and cold sensitive	males sterile at 22°
<i>l(3)ET6</i>	1		heat sensitive	males sterile at 28°
<i>l(3)ET7</i>	1		heat and cold sensitive	males sterile at 22°
<i>l(3)ET8</i>	1		heat sensitive	males sterile at 28°; motile sperm
<i>l(3)ET9</i>^{β}	3-47.9	2	heat sensitive	does not complement <i>l(3)ET23</i>

locus	genetic location	ref ^{α}	type of allele	phenotype
<i>l(3)ET10</i>	2		heat sensitive	tergites deranged
<i>l(3)ET13</i>	1		heat sensitive	males sterile at 28°; no motile sperm
<i>l(3)ET15</i>	1		heat sensitive	males sterile at 28°; motile sperm
<i>l(3)ET16</i>	3-71.5	2	heat sensitive	wings wrinkled longitudinally
<i>l(3)ET18</i>	2		heat sensitive	half-sized flies at 29°
<i>l(3)ET23</i>	2		heat sensitive	does not complement <i>l(3)ET9</i>
<i>l(3)ET26</i>	2		cold sensitive	

α 1 = Shellenbarger and Cross, 1979, Dev. Biol. 71: 308-22; 2 = Tasaka and Suzuki, 1973, Genetics 74: 509-20.
 β See *Spl^{ts}*.

l(3)F: lethal (3) Ferrus

A series of mutants selected by virtue of their abnormal phenotypes in homozygous clones; in all cases homozygous individuals turned out to be lethal.

origin: Induced with ethyl-methanesulfonate.

references: Ferrus, 1979, Dev. Biol. 68: 16-28 (fig.).

locus	location	lethal phase	phenotype in clones
<i>l(3)F12</i>	3-18.2	L2	whorls and bulges of cells in wing
<i>l(3)F13</i>	3-21	L2	large clones show late loss of cells
<i>l(3)F19</i>	3-4.6	L1-L2	large clones show late loss of cells
<i>l(3)F23</i>	3-22.5	L2	large clones show late loss of cells
<i>l(3)F24</i>	3-76.7	L3	swollen cells and long hairs in wing
<i>l(3)F31</i>^{α}	3-62	L3	in wing and abdomen invaginate producing no hairs or bristles
<i>l(3)F34</i>	3-10.6	L1	wing clones small and narrow
<i>l(3)F55</i>	3-14.7	L1	wing clones small and narrow
<i>l(3)F60</i>	3-35	L2-L3	wing and notum clones distorted

α Placed in 90C1-91A2 based on inclusion in *Df(3R)sr*.

l(3)gl: lethal (3) giant larvae

origin: Induced with ethyl-methanesulfonate.

references: Gateff, 1977, DIS 52: 4.

1978, Biol. Rev. 53: 123-42.

1978, Science 200: 1448-59.

phenotype: Late larval lethal; larvae bloated, enlarged; transparent owing to reduced fat body. Third larval instar may be prolonged for up to one week. Imaginal discs represent transplantable benign and lethal neoplasms; brain develops a malignant neuroblastoma. Penetrance incomplete.

****l(3)hd: lethal (3) head defect***

location: 3- (not located).

discoverer: Bridges, 1924.

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 230.

phenotype: Dies in pupal stage with black, tumor-like growth in head. RK3.

l(3)Lh: see *l(3)80F*

l(3)LVML: lethal (3) L. V. Morgan Left

l(3)LVMR: lethal (3) L. V. Morgan Right

Recessive lethal alleles associated with *In(3L)P* and *In(3R)P* respectively in the *LVM* balancer (Craymer, 1980, DIS 55: 197-204).

l(3)mbn: lethal (3) malignant blood neoplasm

origin: Induced by ethyl-methanesulfonate.

references: Gateff, 1977, DIS 52: 4.

1978, Biol. Rev. 53: 123-42.

1978, Science 200: 1448-59.

phenotype: Lethal at pupariation; larvae appear bloated

owing to having 150 times as many free hemocytes as normal; these can encapsulate and melanize larval tissue.

l(3)nc99Eb

location: 3-54.2.

references: Warmke, Krevz, and Falkenthal, 1989, *Genetics* 122: 139-51.

phenotype: Homozygous lethal; heterozygous flightless; interacts semilethally with heterozygous deficiency for *Mlc2*, i.e., *Df(3R)99D3-E3*.

l(3)PL: l(3) Payne Left

location: 3- (left arm).

origin: Spontaneous in *3L* carrying *In(3L)P*.

l(3)PR: l(3) Payne Right

location: 3-90.2.

origin: Spontaneous in *In(3R)P*.

phenotype: Homozygous lethal; lethal in combination with *M(3)96CF*. RK3.

l(3)QIII: see *M(3)80*

l(3)R

A series of late larval lethal mutations that complement one another in all pairwise combinations; not mapped; screened for antigens with 16 different antisera on double diffusion plates (Roberts and Evans-Roberts, 1976, *Mol. Gen. Genet.* 148: 57-64).

locus	comments
<i>l(3)R4</i>	L1, may live to 5 days; polyphasic
<i>l(3)R15</i>	P, monophasic
<i>l(3)R18</i>	L3, may live to 13 days; polyphasic
<i>l(3)R26</i>	L-P, polyphasic
<i>l(3)R33</i>	L-P, polyphasic
<i>l(3)R45</i>	L-P, polyphasic
<i>l(3)R52</i>	L-P, polyphasic
<i>l(3)R70</i>	L3, may live to 10 days; polyphasic
<i>l(3)R73</i>	L1-2, polyphasic
<i>l(3)R76</i>	see <i>l(3)68Am</i>
<i>l(3)R78</i>	L3, may live to 10 days; polyphasic
<i>l(3)R88</i>	
<i>l(3)R102</i>	L-P, polyphasic
<i>l(3)R112</i>	L-P, polyphasic
<i>l(3)R115</i>	L3, may live to 14 days; monophasic
<i>l(3)R138</i>	L3, may live to 30+ days; lack larval serum proteins, monophasic
<i>l(3)R142</i>	L-P, polyphasic
<i>l(3)R159</i>	L-P, polyphasic
<i>l(3)R200</i>	L3, may live to 12 days; monophasic
<i>l(3)R260</i>	L1, may live to 7 days; monophasic
<i>l(3)R340</i>	L1, may live to 5 days; monophasic
<i>l(3)R350</i>	L-P, polyphasic

l(3)R12

location: 3-[51].

origin: Induced by ethyl methanesulfonate.

references: Caggese, Caizzi, Morea, Scalenghe, and Ritossa, 1979, *Proc. Nat. Acad. Sci. USA* 76: 2385-89.

cytology: Placed in 87A6-D5 based on its failure to complement *Df(3R)kar-D2 = Df(3R)87A6-7;87D4-5*. Not checked for complementation with known *l(3)87* mutants.

l(3)Rh: see *l(3)81F*

l(3)S1: lethal (3) of Schalet

location: 3-51 (to the left of *kar*).

origin: X ray induced in a *kar*² chromosome.

discoverer: Schalet.

other information: Placed to the left of *l(3)S2* on the basis of its exclusion from *Df(3R)ry*⁷⁶, which is deficient for *l(3)S2* and loci to the right.

l(3)S1^a

origin: X ray induced in a *kar*² chromosome.

discoverer: Schalet.

other information: Allelism with *l(3)S1* tentative and based on similarity in interaction with *Df(3R)ry*⁷⁶.

l(3)S2

location: 3-51.5 [between *l(3)S1* and *kar*].

origin: X ray induced in a *kar*² chromosome.

discoverer: Schalet.

references: Schalet, Kernaghan, and Chovnick, 1964, *Genetics* 50: 1261-68.

other information: Placed between *l(3)S1* and *kar* on the basis of its inclusion in *Df(3R)ry*⁷⁶ but not *Df(3R)ry*²⁹, *Df(3R)ry*³³, or *Df(3R)ry*³⁶. None of these deficiencies includes *l(3)S1* and all include *kar* and genes to its right.

l(3)S3: see *l(3)87Da*

l(3)S4

location: 3-52.1 (to the right of *pic*).

origin: X ray induced in a *kar*² chromosome.

discoverer: Schalet.

references: Schalet, Kernaghan, and Chovnick, 1964, *Genetics* 50: 1261-68.

phenotype: Homozygous lethal, but there are a few, relatively normal-appearing survivors that are mostly females. RK3.

other information: Placed to the right of *ry* by recombination and to the right of *pic* on the basis of its survival in combination with *ry*³⁵, which behaves as though it were deficient for *ry* and *pic*. Placed to the left of *l(3)S5* by recombination.

l(3)S5: see *l(3)87Db*

l(3)S6: see *l(3)87Eb*

l(3)S7

location: 3-53 [to the right of *l(3)26*].

origin: X ray induced in a *kar*² chromosome.

discoverer: Schalet.

references: Schalet, Kernaghan, and Chovnick, 1964, *Genetics* 50: 1261-68.

alleles: Five alleles recovered; *l(3)S7*² - *l(3)S7*⁵ originally designated *l(3)S7*^a - *l(3)S7*^d. *l(3)S7*¹ but not *l(3)S7*² or *l(3)S7*³ complemented by an undescribed *red* deficiency.

other information: Placed to the right of *l(3)26* on the basis of its exclusion from *Df(3R)ry*⁶⁶, which includes *l(3)26* and loci to its left.

l(3)SG: lethal (3) of Shearn and Garen

Recessive lethal third-chromosome mutations in ninety-four loci isolated by Shearn and Garen (or occasionally by Shearn: *SG4*, *SG5*, *SG6*, *SG16*, *SG28*, *SG30*, *SG35*, *SG44*, *SG64*, *SG68*, *SG70*) mostly selected to be late larval or prepupal lethals. For simplicity they are designated numerically in their genetic map order; those that are unmapped continue the list at the end according to isolation number. As these loci become cytologically mapped or otherwise named, their *SG* designations will

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sink to synonymy.

locus	synonym	location	origin	ref ^α	phenotype	locus	synonym	location	origin	ref ^α	phenotype
I(3)SG1	<i>G60L</i>	3-0.0	EMS	9	L3-EP; discs missing	I(3)SG37	<i>P4</i>	3-49.3	EMS	9	L3; discs small
I(3)SG2	<i>13m237</i>	3-2.4	EMS	2, 6	L3; discs small; mitotic index normal	I(3)SG38	<i>m45</i>	3-49.5	EMS	2, 9	L3-EP; discs differentiate abnormally; mitosis normal
I(3)SG3	<i>M47</i>	3-4.3	EMS	2, 9	EP; eye-antenna and leg discs differentiate abnormally; wing and haltere discs normal; mitosis normal	<i>I(3)SG39</i>	I(1)85Ec	3-49.8	EMS	9	discs normal
I(3)SG4	<i>X11-10L</i>	3-10.7	ICR170	9	discs normal; mitosis normal	<i>I(3)SG40</i>	<i>WG89</i>	3-50.3	NNG	2, 9	L3; discs missing; fuzzy, swollen chromosomes; 27% polyploid cells (Gatti)
I(3)SG5	<i>MS332</i>	3-12.1	EMS		L2; discs normal	I(3)SG41	<i>507</i>	3-50.8	NNG	9	L3, discs small; swollen
I(3)SG6	<i>2813</i>	3-12.4	NNG	5, 9	P; discs normal	I(3)SG42	<i>N61</i>	3-50.9	EMS	9	L3; discs small
I(3)SG7	<i>J16</i>	3-15.6	EMS		L3-EP; eye-antennal disc and wing disc small; haltere and leg discs missing; mitosis normal	I(3)SG43	<i>GA55</i>	3-51.0	gamma		discs missing
I(3)SG8^β	<i>1602</i>	3-19.6	NNG	8, 9	L2; discs small	<i>I(3)SG44</i>	I(3)88Ab	3-51.1	EMS	2, 9, 10	EP; discs missing; extremely low mitotic index; irregular chromosome condensation; chromosome breaks
I(3)SG9	<i>17m28</i>	3-22.2	EMS	6	discs small	<i>I(3)SG45</i>	<i>7m68</i>	3-51.8	EMS	2, 6	L3; discs small; mitosis normal; low mitotic index
I(3)SG10	<i>m27</i>	3-23.3	EMS	2, 9	L3; discs normal; mitosis normal	I(3)SG46	<i>G21</i>	3-52.1	EMS	2, 9	L2-3; leg discs differentiate abnormally; other discs normal
I(3)SG11	<i>15m33</i>	3-24.0	EMS	2, 6	discs small; mitosis normal	I(3)SG47	<i>7m141</i>	3-53.2	EMS	6	discs small
I(3)SG12	<i>J51</i>	3-25.5	EMS	2, 9	EP-P; discs normal; mitosis normal	I(3)SG48	<i>7m70</i>	3-53.4	EMS	6	discs small
I(3)SG13^β	<i>E20</i>	3-25.9	EMS	2, 9	L3-EP; discs missing; abnormal mitoses	I(3)SG49	<i>427</i>	3-53.6	EMS	9	L3-EP; discs small
I(3)SG14	<i>13m281</i>	3-26.6	EMS	2, 6	discs small; highly polyploid, endoreduplicated cells; very low mitotic index	I(3)SG50	<i>7m75</i>	3-53.8	EMS	2, 6	discs small; weakly affects chromosome condensation; some chromosome breakage
I(3)SG15	<i>V11-3</i>	3-29.2	ICR170	9	EP-P; haltere disc normal; other discs differentiate abnormally	I(3)SG51	<i>13m112</i>	3-55.0	EMS	6	L3; discs small
I(3)SG16	<i>1509</i>	3-29.3	NNG	5, 9	L3-EP; discs normal	I(3)SG52	<i>15m25</i>	3-58	EMS	2, 6	discs missing; elongated, fuzzy, unevenly condensed chromosomes; chromosome breakage
<i>I(3)SG17</i>	E(2) , <i>pco</i> , <i>I(3)67Fa</i>	3-30.9	NNG	2, 7, 8, 9	L3-EP; discs small; low mitotic index	I(3)SG53	<i>1803L</i>	3-59.3	EMS	2, 9	E; discs normal; mitosis normal
I(3)SG18	<i>8m100</i>	3-31.9	EMS	6	L3; discs small	I(3)SG54	<i>1803X</i>	3-59.3	EMS		E; discs normal; heat sensitive
I(3)SG19	<i>605</i>	3-34.0	NNG7		L3-EP; eye-antenna disc develops abnormally; other discs normal; mitosis normal	I(3)SG55	<i>L36</i>	3-62.4	EMS	2, 9	EP-P; discs normal; mitosis normal
<i>I(3)SG20</i>	I(3)68Ad	3-35.7	NNG	2, 6	L3-EP; discs small; mitosis normal	I(3)SG56^β	<i>C21R</i>	3-62.5	EMS	1, 2, 4, 9	L3; discs small; mitosis normal
I(3)SG21	<i>7m62</i>	3-36.8	EMS	2, 6	discs small; 77% highly polyploid cells; no mitotic block	I(3)SG57	<i>7m147</i>	3-68.7	EMS	2, 6	discs missing; mitosis normal
I(3)SG22	<i>15m15</i>	3-37.8	EMS	2, 6	discs small; mitosis normal	I(3)SG58	<i>11B1</i>	3-70.4	ICR170	2, 9	L3; discs missing; low mitotic index; condensation irregular
I(3)SG23	<i>G49</i>	3-39.4	EMS	2, 9	L3-EP; discs differentiate abnormally; mitosis normal	I(3)SG59	<i>13m133</i>	3-71	EMS	6	discs missing
I(3)SG24	<i>V-1</i>	3-41	ICR170	9	L3; eye-antenna, wing, and third-leg discs differentiate abnormally; other discs normal	I(3)SG60	<i>2306</i>	3-74.0	NNG	2, 9	L3-EP; discs small; mitosis normal
I(3)SG25^β	<i>G30L</i>	3-41.4	EMS	9, 10	L3; discs missing	I(3)SG61	<i>X1-2</i>	3-76.6	ICR170	9	L3; discs missing
I(3)SG26	<i>G26</i>	3-42.9	EMS	6	L3; discs small	I(3)SG62	<i>X11-10R</i>	3-76.6	ICR170	2, 9	L3; discs small; weak effect on chromosome condensation-chromosome breakage; 4% polyploid cells
I(3)SG27	<i>IX-19</i>	3-44.1	ICR170	9	L2-3; discs small	I(3)SG63	<i>7M124</i>	3-78.2	EMS	6	discs missing
I(3)SG28	<i>8m12</i>	3-46.4	EMS	2, 6	discs missing; swollen fuzzy chromosomes; low mitotic index	I(3)SG64	<i>G30R</i>	3-79.8	EMS	2, 5, 9	discs normal; mitosis normal
I(3)SG29^β	<i>O52</i>	3-47.0	EMS	8, 9	EP; discs small	I(3)SG65	<i>703</i>	3-78.6	NNG	5	transforms arista to tarsus, haltere to wing, genital disc to leg
I(3)SG29a	<i>I(3)111-10</i>	3-46.6	ICR170	5		I(3)SG66	<i>G60A</i>	3-81.4	EMS	2, 6, 9	L3-EP; discs small; chromosomes elongated, unevenly condensed; some chromosome breakage
I(3)SG30	<i>Q13</i>	3-47.2	EMS	9	EP; eye-antenna disc differentiates abnormally; other discs normal	I(3)SG67^β	<i>IX-11</i>	3-81.7	ICR170	2, 8, 9	EP; discs small; extremely low mitotic index; thin, elongated chromosomes
I(3)SG31	<i>2612</i>	3-48.7	NNR	2, 9	L3; discs small; unevenly condensed, swollen chromosomes; 10% polyploid cells; low mitotic index	I(3)SG68	<i>F26</i>	3-82.7	EMS	9	L3; discs missing
I(3)SG32	<i>C21L</i>	3-48.7	EMS	2, 9	discs normal; mitoses normal	I(3)SG69	<i>L6B20</i>	3-84.5	EMS	9	discs normal, mitosis normal
<i>I(3)SG33</i>	I(3)85Ea	3-49.0	EMS	2, 3, 9	PP; eye-antennal disc small; wing and haltere discs large; leg discs abnormal; genital disc normal; heat sensitive; mitosis normal	I(3)SG70^β	<i>12m137</i>	3-106	EMS	2, 6	discs missing; weak effect on chromosome condensation
I(3)SG34	<i>IX-14</i>	3-49.0	ICR170	2, 9	L2-3; discs small; fuzzy, swollen chromosomes; chromosome breaks	I(3)SG71	<i>903</i>	3-110.9	NNG	9	L3; discs differentiate abnormally
<i>I(3)SG35</i>	I(1)85Eb	3-49.2	EMS	5, 9	EP; discs normal	I(3)SG72	<i>4m109</i>		EMS		E-L1; discs normal
I(3)SG36	<i>11m254</i>	3-49.3	EMS	2, 6	discs missing; weak effect on chromosome condensation; low mitotic index	I(3)SG73	<i>5m216</i>		EMS	6	discs normal
						I(3)SG74	<i>5m276</i>		EMS		E-L1; discs normal
						I(3)SG75	<i>5m323</i>		EMS		discs normal
						I(3)SG76	<i>7m177</i>		EMS		discs normal; heat sensitive
						I(3)SG77	<i>9m228</i>		EMS	6	L3; discs normal
						I(3)SG78	<i>12m86</i>		EMS	6	discs normal
						I(3)SG79	<i>609</i>		NNG		P-A; discs normal; heat sensitive
						I(3)SG80	<i>1117</i>		NNG		L3-EP; discs normal
						I(3)SG81	<i>1403</i>		NNG		P; discs normal

locus	synonym	location	origin	ref ^α	phenotype
I(3)SG82	1708		NNG		L3-EP; discs normal; heat sensitive
I(3)SG83	1711		NNG	9	L3-EP; eye-antenna and genital discs develop abnormally; other discs normal; mitosis normal
I(3)SG84	2614		NNG	2, 9	EP; eye-antenna disc develops abnormally; other discs normal; mitosis normal
I(3)SG85	C12		EMS		E; discs normal; heat sensitive; TSP = E-EP
I(3)SG86	C35		EMS		L3; discs normal; heat sensitive; TSP = L3
I(3)SG87	E26R		EMS	9	discs normal; mitosis normal
I(3)SG88	H2J3		EMS		E-L1; discs normal
I(3)SG89	H3J79		EMS		discs normal; heat sensitive
I(3)SG90	K60		EMS	2, 9	EP-P; leg discs differentiate abnormally; other discs normal; mitosis normal
I(3)SG91	K64		EMS		L1-2; discs normal; heat sensitive; TSP = E-L2
I(3)SG92	M38		EMS		discs normal
I(3)SG93	V-13		ICR170	2, 9	L3-EP; discs differentiate abnormally; mitosis normal
I(3)SG94	R12		EMS		EP-P; discs normal

^α 1 = Cheney, Miller, Lang, and Shearn, 1984, Proc. Nat. Acad. Sci. 81: 6422-26; 2 = Gatti and Baker, 1989, Genes Dev. 3: 438-53; 3 = Martin, Martin, and Shearn, 1977, Dev. Biol. 55: 213-32; 4 = Pentz and Shearn, 1979, Dev. Biol. 70: 149-70; 5 = Shearn, 1974, Genetics 77: 115-25; 6 = Shearn and Garen, 1974, Proc. Nat. Acad. Sci. USA 71: 1393-97; 7 = Shearn, Hersperger, and Hersperger, 1978, Genetics 89: 341-53; 8 = Shearn, Hersperger, Hersperger, Pentz, and Denker, 1978, Genetics 89: 355-70; 9 = Shearn, Rice, Garen, and Gehring, 1971, Proc. Nat. Acad. Sci. USA 68: 2594-98; 10 = Szabad and Bryant, 1982, Dev. Biol. 93: 240-56.

^β Additional information below.

allele	synonym
I(3)SG5¹	I(3)MS332
I(3)SG5²	I(3)NW1127
I(3)SG6¹	I(3)2813
I(3)SG6²	I(3)1307
I(3)SG7¹	I(3)J16
I(3)SG7²	I(3)RE620
I(3)SG7³	I(3)RG502
I(3)SG7⁴	I(3)RI506
I(3)SG7⁵	I(3)RJ022
I(3)SG7⁶	I(3)RT119
I(3)SG7⁷	I(3)SJ015
I(3)SG7⁸	I(3)SN423
I(3)SG7⁹	I(3)UM110
I(3)SG7¹⁰	I(3)UO321
I(3)SG7¹¹	I(3)VCC266
I(3)SG7¹²	I(3)VF287
I(3)SG7¹³	I(3)UK14
I(3)SG7¹⁴	I(3)VL476
I(3)SG7¹⁵	I(3)VQ350
I(3)SG7¹⁶	I(3)VQ441
I(3)SG7¹⁷	I(3)VR414
I(3)SG7¹⁸	I(3)VS300
I(3)SG7²⁰	I(3)VX266
I(3)SG7²¹	I(3)VZ292

I(3)SG8

phenotype: Homozygous larvae never form puparia; imaginal discs never exceed size seen in late second instar, neither do they grow when transplanted into normal adult female abdomens nor differentiate when transplanted back into wild-type larval hosts for metamorphosis (Shearn, Hersperger, Hersperger, Pentz, and Denker, 1978, Genetics 89: 355-70).

alleles: All alleles noncomplementing.

allele	synonym
I(3)SG8¹	I(3)GTK512
I(3)SG8²	I(3)MP120
I(3)SG8³	I(3)MS023
I(3)SG8⁴	I(3)MZ121
I(3)SG8⁵	I(3)NC502
I(3)SG8⁶	I(3)NF022
I(3)SG8⁷	I(3)NS040
I(3)SG8⁸	I(3)OG019
I(3)SG8⁹	I(3)OI233
I(3)SG8¹⁰	I(3)ON1235
I(3)SG8¹¹	I(3)OR1505
I(3)SG8¹²	I(3)OS1236
I(3)SG8¹³	I(3)OU1115
I(3)SG8¹⁴	I(3)PA037
I(3)SG8¹⁵	I(3)PB1131
I(3)SG8¹⁶	I(3)PC914
I(3)SG8¹⁷	I(3)PD1001
I(3)SG8¹⁸	I(3)PD1138
I(3)SG8¹⁹	I(3)PF834
I(3)SG8²⁰	I(3)PJ323
I(3)SG8²¹	I(3)PO432
I(3)SG8²²	I(3)PU927
I(3)SG8²³	I(3)JPW1029
I(3)SG8²⁴	I(3)PZ913
I(3)SG8²⁵	I(3)QG1026
I(3)SG8²⁶	I(3)QL125
I(3)SG8²⁷	I(3)QL1136
I(3)SG8²⁸	I(3)QM509
I(3)SG8²⁹	I(3)QM629
I(3)SG8³⁰	I(3)QO022
I(3)SG8³¹	I(3)RG509
I(3)SG8³²	I(3)RM329
I(3)SG8³³	I(3)RN330
I(3)SG8³⁴	I(3)VI307
I(3)SG8³⁵	I(3)VL469
I(3)SG8³⁶	I(3)VQ314
I(3)SG8³⁷	I(3)VX242

I(3)SG13

phenotype: Homozygous larvae contain rudimentary imaginal discs, but disc primordia do not grow during larval development; testes and ovaries smaller than normal, and cell number in central nervous system reduced. Mutant gonads do not survive metamorphosis when implanted into wild-type larvae. Homozygous cuticular clones appear to develop normally. Mutant larvae support growth of implanted wild-type discs. Normal gene product postulated to be required for cell proliferation; survival of somatic epidermal clones attributed to perdurance. Larval ganglia exhibit extremely low mitotic index; chromosomes irregularly condensed; extensive chromosome fragmentation observed frequently (Gatti and Baker, 1989, Genes Dev. 3: 438-53); salivary chromosomes appear normal.

allele	synonym
I(3)SG16¹	I(3)1509
I(3)SG16²	I(3)1905
I(3)SG23¹	I(3)G49
I(3)SG23²	I(3)m75

I(3)SG25

phenotype: Homozygous larvae contain rudimentary imaginal discs, but disc primordia do not grow during larval development; testes and ovaries smaller than normal, and cell number in central nervous system reduced; mitotic

abnormalities observed but salivary chromosomes appear normal. Mutant gonads do not survive metamorphosis when implanted into wild-type larvae. Homozygous cuticular clones appear to develop normally. Mutant larvae support growth of implanted wild-type discs. Normal gene product postulated to be required for cell proliferation; survival of somatic epidermal clones attributed to perdurance.

allele	synonym
<i>l(3)SG26</i>¹	<i>l(3)G26</i>
<i>l(3)SG26</i>²	<i>l(3)l3m115</i>
<i>l(3)SG26</i>³	<i>l(3)E26</i>

l(3)SG29

phenotype: Homozygous larvae do not form puparia; imaginal discs never attain size of those in young third-instar larvae. Mutant discs transplanted into wild-type female abdomens decrease in size and disappear during the first three days; however, in transplanted mixtures of wild-type and mutant embryonic cells allowed to pass through metamorphosis, mutant cells appear in cuticular derivatives from all discs. Wild-type discs transplanted into mutant larvae are capable of reduced growth and of differentiation upon retransplantation into wild-type larvae. Alleles with more extreme effects (*i.e.*, larval death in early second instar) and less severe effects (pharate adults that do not eclose) noted (Shearn, Hersperger, Hersperger, Pentz, and Denker, 1978, Genetics 89: 355-70).

alleles: Interallelic complementation not observed; heteroallelic combinations exhibit phenotypes intermediate between those of the two homozygotes.

allele	synonym
<i>l(3)SG29</i>¹	<i>l(3)O52</i>
<i>l(3)SG29</i>²	<i>l(3)MJ727</i>
<i>l(3)SG29</i>³	<i>l(3)MV1424</i>
<i>l(3)SG29</i>⁴	<i>l(3)MZA16</i>
<i>l(3)SG29</i>⁵	<i>l(3)NI428</i>
<i>l(3)SG29</i>⁶	<i>l(3)ON1212</i>
<i>l(3)SG29</i>⁷	<i>l(3)OS218</i>
<i>l(3)SG29</i>⁸	<i>l(3)OU902</i>
<i>l(3)SG29</i>⁹	<i>l(3)OY933</i>
<i>l(3)SG29</i>¹⁰	<i>l(3)OZ831</i>
<i>l(3)SG29</i>¹¹	<i>l(3)PE414</i>
<i>l(3)SG29</i>¹²	<i>l(3)PF123</i>
<i>l(3)SG29</i>¹³	<i>l(3)PL1117</i>
<i>l(3)SG29</i>¹⁴	<i>l(3)PO610</i>
<i>l(3)SG29</i>¹⁵	<i>l(3)PX837</i>
<i>l(3)SG29</i>¹⁶	<i>l(3)PX929</i>
<i>l(3)SG29</i>¹⁷	<i>l(3)PZ227</i>
<i>l(3)SG29</i>¹⁸	<i>l(3)QB212</i>
<i>l(3)SG29</i>¹⁹	<i>l(3)QE1103</i>
<i>l(3)SG29</i>²⁰	<i>l(3)QM709</i>
<i>l(3)SG29</i>²¹	<i>l(3)RO022</i>
<i>l(3)SG29</i>²²	<i>l(3)SE508</i>
<i>l(3)SG29</i>²³	<i>l(3)VL51</i>
<i>l(3)SG29</i>²⁴	<i>l(3)VL250</i>
<i>l(3)SG29</i>²⁵	<i>l(3)VP305</i>
<i>l(3)SG29</i>²⁶	<i>l(3)VP339</i>
<i>l(3)SG29</i>²⁷	<i>l(3)VX340</i>
<i>l(3)SG29</i>²⁸	<i>l(3)VY53</i>
<i>l(3)SG29</i>²⁹	<i>l(3)VZ412</i>
<i>l(3)SG29</i>³⁰	<i>l(3)VZ160</i>
<i>l(3)SG29</i>³¹	<i>l(3)WD545</i>
<i>l(3)SG29a</i>¹	<i>l(3)III-10</i>
<i>l(3)SG29a</i>²	<i>l(3)XVI-18</i>
<i>l(3)SG55</i>¹	<i>l(3)L23</i>

allele	synonym
<i>l(3)SG55</i>²	<i>l(3)L36</i>

l(3)SG56

references: Shearn, Hersperger, Hersperger, Pentz, and Denker, 1978, Genetics 89: 355-70.

Pentz and Shearn, 1979, Dev. Biol. 70: 149-70 (fig.).

Cheney, Miller, Lang, and Shearn, 1984, Proc. Nat. Acad. Sci. USA 81: 6422-26.

phenotype: 78% of mutant larvae reach third instar; development delayed, and larvae never pupariate; rather they remain alive on surface of food for up to 22 days. Larvae form melanotic masses. Imaginal disc growth retarded and development abnormal, with substantial cell death; growth rate of mutant discs, but not their ability to differentiate, partially restored when transplanted into normal larval host; mutant larvae defective in support of growth of implanted wild-type discs, but do not prevent their subsequent differentiation upon retransplantation into normal larvae. Homozygous mutant clones in a wild-type background were induced at control frequencies; however, sizes of mutant clones produced earlier, but not those produced later, were smaller than control clones; differentiation of cuticular elements normal in small clones, but in large thoracic clones produced in a *M* background cuticular development abnormal (Pentz and Shearn). Ganglion mitosis normal according to Gatti and Baker (1989, Genes Dev. 3: 438-53), disturbed according to Pentz. *l(3)SG56*²⁶ homozygotes lethal when raised at 27° but survive as fertile adults when reared at 20°; shifting such adult females to 27° demonstrates that *l(3)SG56* is a maternal-effect lethal and a female sterile; egg production decreases with time, and those eggs that are produced exhibit abnormal cellularization and fail to hatch; temperature effect reversible by shift down. Two-dimensional gel electrophoresis analysis of proteins from mutant tissue at restrictive vs. permissive temperatures shows reduction in levels of three proteins and a concomitant increase of three related proteins; two other proteins are absent from mutant tissue at restrictive temperatures; mutant postulated to be defective in post-translational modification of a small subset of proteins crucial to normal cell division, cell motility, and formation of adult hairs and bristles (Cheney, *et al.*).

alleles: No complementation between alleles observed. *l(3)SG56*⁶ hypomorphic; completes metamorphosis but dies as pharate adult without striking morphological defects. *l(3)SG56*²⁶ heat sensitive.

allele	synonym
<i>l(3)SG56</i>¹	<i>l(3)C21R</i>
<i>l(3)SG56</i>²	<i>l(3)CR12</i>
<i>l(3)SG56</i>³	<i>l(3)CR125</i>
<i>l(3)SG56</i>⁴	<i>l(3)MW416</i>
<i>l(3)SG56</i>⁵	<i>l(3)NB215</i>
<i>l(3)SG56</i>⁶	<i>l(3)NC806R2</i>
<i>l(3)SG56</i>⁷	<i>l(3)NZ806</i>
<i>l(3)SG56</i>⁸	<i>l(3)OI1015</i>
<i>l(3)SG56</i>⁹	<i>l(3)OL1112</i>
<i>l(3)SG56</i>¹⁰	<i>l(3)OL1116</i>
<i>l(3)SG56</i>¹¹	<i>l(3)OM901</i>
<i>l(3)SG56</i>¹²	<i>l(3)OX502</i>
<i>l(3)SG56</i>¹³	<i>l(3)PA734</i>
<i>l(3)SG56</i>¹⁴	<i>l(3)PB427</i>
<i>l(3)SG56</i>¹⁵	<i>l(3)PD432</i>

allele	synonym
<i>l(3)SG56</i> ¹⁶	<i>l(3)PY910</i>
<i>l(3)SG56</i> ¹⁷	<i>l(3)PZ007</i>
<i>l(3)SG56</i> ¹⁸	<i>l(3)QB1001</i>
<i>l(3)SG56</i> ¹⁹	<i>l(3)QE1321</i>
<i>l(3)SG56</i> ²⁰	<i>l(3)QE1332</i>
<i>l(3)SG56</i> ²¹	<i>l(3)QG1329</i>
<i>l(3)SG56</i> ²²	<i>l(3)QN407</i>
<i>l(3)SG56</i> ²³	<i>l(3)QR233</i>
<i>l(3)SG56</i> ²⁴	<i>l(3)QU1040</i>
<i>l(3)SG56</i> ²⁵	<i>l(3)RN323</i>
<i>l(3)SG56</i> ²⁶	<i>l(3)RW630</i>
<i>l(3)SG56</i> ²⁷	<i>l(3)UQ295</i>
<i>l(3)SG56</i> ²⁸	<i>l(3)UQ335</i>
<i>l(3)SG56</i> ²⁹	<i>l(3)VLA75</i>
<i>l(3)SG59</i> ¹	<i>l(3)13m133</i>
<i>l(3)SG59</i> ²	<i>l(3)7M63B</i>
<i>l(3)SG64</i> ¹	<i>l(3)e21R</i>
<i>l(3)SG64</i> ²	<i>l(3)g30R</i>
<i>l(3)SG64</i> ³	<i>l(3)g60R</i>
<i>l(3)SG64</i> ⁴	<i>l(3)g131</i>
<i>l(3)SG64</i> ⁵	<i>l(3)jn3</i>
<i>l(3)SG64</i> ⁶	<i>l(3)o51r</i>
<i>l(3)SG64</i> ⁷	<i>l(3)g6</i>
<i>l(3)SG65</i> ¹	<i>l(3)703</i>
<i>l(3)SG65</i> ²	<i>l(3)1803R</i>

l(3)SG67

phenotype: Homozygotes pupariate, but do not metamorphose; imaginal discs never exceed in size those of late second-instar larvae. Mutant eye-antennal and wing discs transplanted into adult wild-type female abdomens are never recovered after seven days in culture; they decrease in size and disappear during first three days. Mutant larvae permit reduced growth of implanted wild-type discs and do not interfere with their subsequent development in normal hosts (Shearn, Hersperger, Hersperger, Pentz, and Denker, 1978, *Genetics* 89: 355-70). Larval ganglion cells exhibit extremely low mitotic index and weak effects on chromosome condensation; chromosomes thin and elongated (Gatti and Baker, 1989, *Genes Dev.* 3: 438-53).

alleles: All alleles noncomplementing.

allele	synonym
<i>l(3)SG67</i> ¹	<i>l(3)jIX-11</i>
<i>l(3)SG67</i> ²	<i>l(3)GSS408</i>
<i>l(3)SG67</i> ³	<i>l(3)NH812</i>
<i>l(3)SG67</i> ⁴	<i>l(3)NLS22</i>
<i>l(3)SG67</i> ⁵	<i>l(3)NLS20</i>
<i>l(3)SG67</i> ⁶	<i>l(3)NR821</i>
<i>l(3)SG67</i> ⁷	<i>l(3)NV1101</i>
<i>l(3)SG67</i> ⁸	<i>l(3)OF840</i>
<i>l(3)SG67</i> ⁹	<i>l(3)OH633</i>
<i>l(3)SG67</i> ¹⁰	<i>l(3)ON1227</i>
<i>l(3)SG67</i> ¹¹	<i>l(3)OS334</i>
<i>l(3)SG67</i> ¹²	<i>l(3)OS335</i>
<i>l(3)SG67</i> ¹³	<i>l(3)PC914</i>
<i>l(3)SG67</i> ¹⁴	<i>l(3)PK829</i>
<i>l(3)SG67</i> ¹⁵	<i>l(3)QA1001</i>
<i>l(3)SG67</i> ¹⁶	<i>l(3)QB201</i>
<i>l(3)SG67</i> ¹⁷	<i>l(3)QB427</i>
<i>l(3)SG67</i> ¹⁸	<i>l(3)QL827</i>
<i>l(3)SG67</i> ¹⁹	<i>l(3)RS109</i>
<i>l(3)SG67</i> ²⁰	<i>l(3)SD715</i>
<i>l(3)SG67</i> ²¹	<i>l(3)VC180</i>
<i>l(3)SG67</i> ²²	<i>l(3)VI121</i>
<i>l(3)SG67</i> ²³	<i>l(3)VS384</i>
<i>l(3)SG67</i> ²⁴	<i>l(3)XVI-3</i>

allele	synonym
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l(3)SG70

phenotype: Homozygous larvae contain rudimentary imaginal discs, but disc primordia do not grow during larval development; testes and ovaries smaller than normal, and cell number in central nervous system reduced. Mutant gonads do not survive metamorphosis when implanted into wild-type larvae. Homozygous cuticular clones appear to develop normally, but with reduced frequency and size compared to control clones. Mutant larvae support growth of implanted wild-type discs. Normal gene product postulated to be required for cell proliferation; survival of somatic epidermal clones attributed to perdurance. Larval ganglion mitoses exhibit weak effect on chromosome condensation as well as chromosome breakage (Gatti and Baker, 1989, *Genes Dev.* 3: 438-53); salivary chromosomes appear normal.

l(3)Sp: lethal (3) of Spiess

A series of recessive lethal mutations extracted from a long term (170 generations) laboratory population.

references: Spiess, Helling, and Capenos, 1963, *Genetics* 48: 1377-88.

locus	genetic location
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<i>*l(3)Sp1</i>	3-33.8
<i>*l(3)Sp2</i>	3-79.3
<i>*l(3)Sp3</i>	3-41.0
<i>*l(3)Sp6</i>	3-40.4
<i>*l(3)Sp9</i>	3-101.1
<i>*l(3)Sp10</i>	3-41.7
<i>*l(3)Sp17</i>	3-38.4
<i>*l(3)Sp19</i>	3-100.9

l(3)tl: lethal (3) tumorous larvae

location: 3- to the left of *R* (3-1.4).

origin: Spontaneous.

references: Peterson and Gardner, 1965, *Genetics* 52: 465-66.

Kobel and Breugel, 1967, *Genetica* 38: 305-27.

Zhimulev and Lychev, 1972, *Genetika (Moscow)* 8(6): 51-55.

Zhimulev, Belyaeva, and Lychev, 1974, *Genetika (Moscow)* 10(10): 73-79 (fig.).

Zhimulev, Belyaeva, and Lychev, 1976, *Chromosoma* 55: 121-26.

phenotype: Homozygous larvae fail to pupate and live beyond normal time of pupation. Hemolymph contains an overabundance of blood cells as well as melanotic pseudotumors. Fat bodies and parts of gut disintegrate. Salivary nuclei contain nucleolus-like bodies six to seven times normal size; polytene chromosomes short, thick, and diffuse, especially the male X; bands appear to fuse. Mutant eye-antennal discs allowed to metamorphose in normal larvae produce some normal adult structures.

l(3)tr: lethal (3) translucida

location: 3-20 (18.1 to 22.0).

origin: Spontaneous.

discoverer: Hadorn, 40116.

references: 1947, *Expt. Biol. Symp.*, Vol. 2: 177-95, Cambridge Univ. Press.

1947, *DIS* 21: 68.

1956, *Cold Spring Harbor Symp. Quant. Biol.* 21: 363-

73 (fig.).

phenotype: Larvae become bloated and transparent from accumulation of abnormal amount of hemolymph. Concentration of amino acids in hemolymph higher than normal; concentration of proteins reduced. Pupation delayed one day (25°); dwarfed pupae formed in inflated puparia; death follows pupation or completion of imaginal differentiation of head and thorax; abdomen never metamorphoses. After transplantation into normal hosts, imaginal disks develop normally; ovaries also develop normally and are fully capable of producing viable eggs [Sobels, 1950, *Experientia* 6: 139-40 (fig.)]. In pure oxygen, frequency and extent of imaginal differentiation strongly increased [Sobels and Nijenhuis, 1953, *Z. Indukt. Abstamm. Vererbungslehre* 85: 579-92 (fig.)]. RK3.

cytology: Salivary chromosomes normal (Rosin).

l(3)W

location: 3- (right arm).

origin: Spontaneous in 3R carrying *In(3R)P*.

l(3)XaR

location: 3-91.8.

other information: Used to balance *T(2;3)ap^{Xa}*.

l(4)102

The following represents the results of extensive searches for mutations on chromosome 4, largely carried out by Hochman. Mutants can be examined for their ability to complement *Df(4)M4* and *Df(4)G*; those that complement both deficiencies are assumed to be located between them, although it is possible that some are located proximal to *Df(4)M4*.

references: Hochman, Gloor, and Green, 1964, *Genetica* 35: 109-26.

Hochman, 1971, *Genetics* 67: 235-52.

Hochman, 1973, *Cold Spring Harbor Symp. Quant. Biol.* 38: 581-89.

Hochman, 1976, *Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, and San Francisco, Vol. 1b, pp. 903-24.

locus	cytological location	included in	excluded from	synonym	lethal phase
<i>l(4)102ABa</i>	102A1-2	<i>Df(4)M101-63a</i>		M(4)101	L
l(4)102ABb	102B2-17	<i>Df(4)M101-1</i>	<i>Df(4)M101-63a</i>	<i>l(4)13</i>	
<i>l(4)102ABc</i>	102B2-17	<i>Df(4)M101-1</i>	<i>Df(4)M101-63a</i>	Ce , <i>l(4)17</i>	E/L
l(4)102ABd	102B2-17	<i>Df(4)M101-1</i>	<i>Df(4)M101-63a</i>	<i>l(4)1</i>	L
l(4)102ABe	102B2-17	<i>Df(4)M101-1</i>	<i>Df(4)M101-63a</i>	<i>l(4)18</i>	A
l(4)102ABf	102B2-17	<i>Df(4)M101-1</i>	<i>Df(4)M101-63a</i>	<i>l(4)20</i>	P
l(4)102ABg	102B2-17	<i>Df(4)M101-1</i>	<i>Df(4)M101-63a</i>	<i>l(4)24</i>	L-P
l(4)102ABh	102B2-17	<i>Df(4)M101-1</i>	<i>Df(4)M101-63a</i>	<i>l(4)25</i>	L
l(4)102ABI	102B2-17	<i>Df(4)M101-1</i>	<i>Df(4)M101-63a</i>	<i>l(4)26</i>	L
l(4)102CDa ^α	102B6-E10		<i>Df(4)M101-1</i>	<i>l(4)2</i>	E
			<i>Df(4)G</i>		
l(4)102Cdb	102B6-E10		<i>Df(4)M</i>	<i>l(4)23</i>	E
			<i>Df(4)G</i>		
l(4)102CDc	102B6-E10		<i>Df(4)M</i>	<i>l(4)4</i>	L
			<i>Df(4)G</i>		
l(4)102CDd	102B6-E10		<i>Df(4)M</i>	<i>l(4)5</i>	E-L
			<i>Df(4)G</i>		
l(4)102CDe	102B6-E10		<i>Df(4)M</i>	<i>l(4)6</i>	P
			<i>Df(4)G</i>		
l(4)102CDf	102B6-E10		<i>Df(4)M</i>	<i>l(4)7</i>	E
			<i>Df(4)G</i>		
l(4)102CDg	102B6-E10		<i>Df(4)M</i>	<i>l(4)8</i>	L-P
			<i>Df(4)G</i>		
l(4)102CDh	102B6-E10		<i>Df(4)M</i>	<i>l(4)10</i>	E
			<i>Df(4)G</i>		
l(4)102CDi	102B6-E10		<i>Df(4)M</i>	<i>l(4)14</i>	L

locus	cytological location	included in	excluded from	synonym	lethal phase
			<i>Df(4)G</i>		
l(4)102CDJ	102B6-E10		<i>Df(4)M</i>	<i>l(4)15</i>	P
			<i>Df(4)G</i>		
l(4)102CDk	102B6-E10		<i>Df(4)M</i>	<i>l(4)21</i>	P
			<i>Df(4)G</i>		
l(4)102CDI	102B6-E10		<i>Df(4)M</i>	<i>l(4)22</i>	P
			<i>Df(4)G</i>		
l(4)102CDm	102B6-E10		<i>Df(4)M</i>	<i>l(4)27</i>	L
			<i>Df(4)G</i>		
l(4)102CDn	102B6-E10		<i>Df(4)M</i>	<i>l(4)28</i>	L
			<i>Df(4)G</i>		
l(4)102CDo	102B6-E10		<i>Df(4)M</i>	<i>l(4)30</i>	L
			<i>Df(4)G</i>		
l(4)102CDp	102B6-E10		<i>Df(4)M</i>	<i>l(4)31</i>	P
			<i>Df(4)G</i>		
l(4)102CDq	102B6-E10		<i>Df(4)M</i>	<i>l(4)32</i>	P
			<i>Df(4)G</i>		
<i>l(4)102CDr</i> ^β	102B6-E10		<i>Df(4)M</i>	ey	P
			<i>Df(4)G</i>	<i>l(4)33</i>	
l(4)102CDs	102B6-E10		<i>Df(4)M</i>	<i>l(4)34</i>	A
			<i>Df(4)G</i>		
l(4)102CDt	102B6-E10		<i>Df(4)M</i>	<i>l(4)35</i>	L
			<i>Df(4)G</i>		
l(4)102CDu	102B6-E10		<i>Df(4)M</i>	<i>l(4)36</i>	P
			<i>Df(4)G</i>		
l(4)102CDv	102B6-E10		<i>Df(4)M</i>	<i>l(4)37</i>	L
			<i>Df(4)G</i>		

l(4)102EFa	102E2-F10		<i>Df(4)G</i>	<i>l(4)9</i>	P
l(4)102EFb	102E2-F10		<i>Df(4)G</i>	<i>l(4)16</i>	E-L
l(4)102EFc ^α	102E2-F10		<i>Df(4)G</i>	<i>l(4)29</i>	P
l(4)102EFd	102E2-F10		<i>Df(4)G</i>	<i>l(4)39</i>	E-L
<i>l(4)102EFe</i> ^β	102E2-F10		<i>Df(4)G</i>	sv	L
				<i>l(4)40</i>	
l(4)102EFf	102F2-end		<i>Df(4)G</i>	<i>l(4)19</i>	P

^α More detailed description below.

^β Genetic location as measured in diplo-4 triploids for *l(4)102CDr*: 4-2.0, *l(4)102EFc*: 4-3.0 (Sturtevant, 1951, *Proc. Nat. Acad. Sci. USA* 37: 405-07).

alleles:

locus	origin of alleles				
	spont	X ray	EMS	ICR-170	Melphalan
M(4)		1			
l(4)102ABb		1	1		
l(4)102ABc		2			
l(4)102ABd	2	6	3		
l(4)102ABe			1		
l(4)102ABf	1		3		
l(4)102ABg			10		
l(4)102ABh	1		3		1
l(4)102ABI			1	1	
l(4)102CDa	3	6	23	3	
l(4)102Cdb	1		6	1	
l(4)102CDc	2	3	9	1	
l(4)102CDd	1	1	9		
l(4)102CDe	1	3	3		
l(4)102CDf			1	3	
l(4)102CDg		3			
l(4)102CDh	1	1	1		
l(4)102CDi	1	2	4		
l(4)102CDj	3				
l(4)102CDk	1		4		
l(4)102CDl	1				
l(4)102CDm			1		
l(4)102CDn			3		
l(4)102CDo			1		
l(4)102CDp			1		
l(4)102CDq			1		
l(4)102CDs	1				
l(4)102CDt			1		

locus	origin of alleles				
	spont	X ray	EMS	ICR-170	Melphalan
<i>l(4)102CDu</i>			4		
<i>l(4)102CDv</i>			4		
<i>l(4)102EFa</i>	2		2		
<i>l(4)102EFb</i>	4		4	1	
<i>l(4)102EFc</i>	4				
<i>l(4)102EFd</i>			1		
<i>l(4)102EFf</i>			2	4	2

l(4)102CDa

alleles: A highly mutable locus. The 35 alleles designated *a* through *hh* by Hochman; alleles *b*, *k*, and *ii* die in the larval stage and are mutually partially complementing; a fourth complementation group comprises alleles *f*, *h*, and *t*, which are larval lethals. The remaining 29 alleles are embryonic lethals and are noncomplementing. Allele *c* fails to complement *l(4)102CDB* alleles as well.

***l(4)102EFc* (R. Denell)**

references: Hochman, Gloor, and Green, 1964, *Genetica* 35: 109-26.

Gehring, 1970 DIS 45: 103.

Hochman, 1971, *Genetics* 67: 235-52.

Duncan, 1982, *Genetics* 102: 49-70.

Sato, Russell, and Denell, 1983, *Genetics* 105: 357-70.

phenotype: Homozygotes from heterozygous mothers die as pupae (Hochman). Those that reach the pharate-adult stage show partial homeotic transformations of first and second antennal segments into proximal leg structures, meso- and metathoracic legs into prothoracic legs, as well as posteriorly directed transformations of features of the abdominal hypoderm and of parovaria into spermathecae in females. All three pairs of legs distorted, with tarsal segments swollen and sometimes fused with missing claws; some abnormalities of derivatives of the antennal disc and of the dorsal prothorax also present (Gehring; Duncan). Heterozygous adults normal, except that males also heterozygous for a deficiency or mutation of *E(Pc)* occasionally develop sex-comb teeth on meso- and metathoracic legs (Sato *et al.*). Heterozygous and homozygous larvae from heterozygous mothers normal; homozygotes from transplanted homozygous ovaries die as late embryos with a complex homeotic syndrome. Head involution incomplete, and portions of head develop abdominal-like denticles. All thoracic segments partially transformed to resemble the first abdominal segment, and more posterior segments may develop cuticular features characteristic of the eighth abdominal segment. Heterozygotes from homozygous ovaries sometimes die as late embryos displaying relatively mild homeotic effects of the head and mesothorax or slightly incomplete head involution or both, but often hatch and die during larval or pupal stages. Rarely, adults eclose, often with appendages that are crippled or are missing owing to failure of imaginal-disc evagination (Denell). Most embryos produced by oocytes hemizygous for *l(4)102EFc*³ and *l(4)102EFc*⁴, achieved by pole-cell transplantation, fail to form cuticle, even with a wild-type paternal complement; a few of the latter genotype produce enough cuticle to reveal extensive defects in segmentation; setal belts usually missing or transformed; head involution fails; anterior hole present in most embryos (Breen and Duncan, 1986, *Dev. Biol.*

118: 442-45).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>l(4)102EFc</i>¹	spont	Hochman, 62k	<i>l(4)BU-2</i>	1, 2, 3, 4	
			<i>l(4)29</i>		
<i>l(4)102EFc</i>²	spont	Kidwell, 62l	<i>l(4)OC-1</i>	4	
			<i>l(4)29^a</i>		
<i>l(4)102EFc</i>³	spont	Hochman, 63	<i>l(4)29^b</i>	1, 2, 4	
<i>l(4)102EFc</i>⁴	spont	Duncan	<i>l(4)29^c</i>	1	hypomorphic allele

^α 1 = Duncan, 1982, *Genetics* 102: 49-70; 2 = Gehring, 1970, DIS 45: 103; 3 = Hochman, 1971, *Genetics* 67: 235-52; 4 = Hochman, Gloor, and Green, 1964, *Genetica* 35: 109-26.

DEFICIENCY MAP OF REGION 102 CHROMOSOME 4

side	breakpoint	variant
left	101E-F	<i>Df(4)M101-1</i>
left	101E-F	<i>Df(4)M101-62e</i>
left	101E-F	<i>Df(4)M101-62f</i>
left	101F2-102A1	<i>Df(4)M101-63a</i>
	102A1-2	<i>M(4)101</i>
	102A1-2	<i>cl</i>
right	102A2-5	<i>Df(4)M101-63a</i>
right	102B2-5	<i>Df(4)M101-62f</i>
left		<i>ci^D?</i>
		<i>l(4)102ABb</i>
		<i>Ce</i>
right		<i>ci^D?</i>
		<i>gvl</i>
		<i>ar</i>
		<i>l(4)102ABd</i>
		<i>l(4)102ABe</i>
		<i>l(4)102ABf</i>
		<i>l(4)102ABg</i>
		<i>l(4)102ABh</i>
		<i>l(4)102ABI</i>
right	102B6-17	<i>Df(4)M101-1</i>
left		<i>Df(4)bt^D?</i>
left		<i>Df(4)2c?</i>
		<i>l(4)102CDa</i>
		<i>l(4)102CDB</i>
right		<i>Df(4)bt^D</i>
right		<i>Df(4)2c</i>
		<i>l(4)102CDc</i>
		<i>l(4)102CDd</i>
		<i>l(4)102CDe</i>
		<i>l(4)102CDf</i>
		<i>l(4)102CDg</i>
		<i>l(4)102CDh</i>
		<i>l(4)102CDI</i>
		<i>l(4)102CDJ</i>
		<i>l(4)102CDk</i>
		<i>l(4)102CDL</i>
		<i>l(4)102CDm</i>
		<i>l(4)102CDn</i>
		<i>l(4)102CDo</i>
		<i>l(4)102CDp</i>
		<i>l(4)102CDq</i>
		<i>ey</i>
		<i>l(4)102CDs</i>
		<i>l(4)102CDt</i>
		<i>l(4)102CDu</i>
		<i>l(4)102CDv</i>
right	102D13-E1	<i>Df(4)M101-62e</i>
left	102E2-10	<i>Df(4)11</i>
left	102E2-10	<i>Df(4)G</i>
left		<i>Df(4)3?</i>
left		<i>Df(4)38?</i>
left		<i>Df(4)Cat?</i>
		<i>l(4)102EFa</i>
		<i>l(4)102EFb</i>
		<i>l(4)102EFc</i>
right		<i>Df(4)38?</i>

side	breakpoint	variant
		<i>l(4)102EFd</i>
left		<i>Df(4)40 ?</i>
left		<i>l(4)14-310</i>
		<i>sv</i>
right		<i>Df(4)Cat ?</i>
right		<i>Df(4)3 ?</i>
right		<i>l(4)14-310</i>
		<i>spa</i>
right		<i>Df(4)40 ?</i>
right	102F2-10	<i>Df(4)11</i>
		<i>l(4)102EFf</i>

lab: see **ANTC**

labial: see **ANTC**

Labore: see *Fs(3)Sz18*

lac: **lacquered**

location: 1-7.3.

phenotype: Pale fly with chitin glistening as though polished. Bristles long and scraggly, frequently duplicated. Eyes smaller and slightly bright. Wings often longitudinally pleated. Slightly delayed eclosion; viability and fertility reduced in both sexes. RK2.

allele	origin ^α	discoverer	synonym	ref ^β	comments
<i>lac 1</i>	CB1506	Fahmy		2	
<i>lac 2</i>	EMS	Fahmy		2	
<i>lac 3</i>	EMS	Fahmy		2	
<i>lac 4</i>	EMS	Fahmy		2	
<i>lac 5</i>	EMS	Fahmy		2	
<i>lac 6</i>	EMS	Fahmy		2	
<i>lac 7</i>	EMS	Fahmy		2	
<i>lac 8</i>	EMS	Fahmy		2	
<i>lac 9</i>	MMS	Fahmy		2	
<i>lac 10</i>	MMS	Fahmy		2	
<i>lac 11</i>	CB2511	Fahmy		2	
<i>lac 12</i>	CB3025	Fahmy		2	
<i>lac 13</i>	CB3026	Fahmy		2	
<i>lac 14</i>	CB3034	Fahmy		2	
<i>lac 15</i>	CB3034	Fahmy		2	
<i>lac 16</i>	X ray	Fahmy		2	
<i>lac 17</i>	X ray	Fahmy		2	

^α CB = Chester Beatty mutagen number; CB1506 = 2-chloroethyl methanesulfonate; CB2511 = D-1:6-dimethanesulfonyl mannitol; CB3025 = L-*p*-N,N-di-(2-chloroethyl)aminophenylalanine; CB3026 = D-*p*-N,N-di-(2-chloroethyl)aminophenylalanine; CB3034 = *p*-N,N-di-(2-chloroethyl)aminophenylethylamine.

^β 1 = Banga, Bloomquist, Brodberg, Pye, Larrivee, Mason, Boyd, and Pak, 1986, Chromosoma 93: 341-46; 2 = Fahmy, 1959, DIS 33: 87.

cytology: Placed in 4C5-6 based on its inclusion in *Df(1)rb13 = Df(1)4C5-6;4D3-E1* but not *Df(1)GA56 = Df(1)4C5-6;4D1* and the failure of two translocations, *T(1;2)bi^{D2}* and *T(1;3)bi^{D1}*, with breakpoints in 4C5-6 to complement *lac* (Banga *et al.*).

lace

location: 2- {51.4}.

synonym: *l(2)br36; l(2)35Dc*.

phenotype: Strong alleles lethal; weak alleles viable with supernumerary fragments of wing veins.

alleles:

allele	origin	discoverer	synonym	comments
<i>lace 1</i>	spont	Ashburner	<i>AM1</i>	chromosome with <i>Su(H)²</i>
<i>lace 2</i>	EMS	Harrington	<i>HQ34</i>	
<i>lace 3</i>	X ray	Ashburner	<i>TB8</i>	with <i>T(Y;2)B8</i>
<i>lace 4</i>	EMS	Simpson	<i>P42</i>	

allele	origin	discoverer	synonym	comments
<i>lace 5</i>	EMS	El Messal	<i>VM1</i>	
<i>lace 6</i>	EMS	El Messal	<i>VM2</i>	
<i>lace 7</i>	EMS	Simpson	<i>VT1</i>	
<i>lace 8</i>	EMS	Simpson	<i>VT2</i>	
<i>lace 9</i>	EMS	Simpson	<i>VT3</i>	
<i>lace 10</i>	EMS	Simpson	<i>VT4</i>	
<i>lace 11</i>	EMS	Simpson	<i>VT5</i>	
<i>lace 12</i>	EMS	Simpson	<i>VT6</i>	
<i>lace 13</i>	EMS	Simpson	<i>VT7</i>	
<i>lace 14</i>	EMS	Simpson	<i>VT8</i>	
<i>lace 15</i>	EMS	Simpson	<i>VT9</i>	
<i>lace 16</i>	EMS	Carteret	<i>VT10</i>	viable
<i>lace 17</i>	EMS	Simpson	<i>VT11</i>	

cytology: Placed in 35D3-4 by Ashburner.

lacquered: see **lac**

Lam: **Laminin**

location: 2- {17}.

origin: Gene identified by screening a γ -gt11 cDNA expression library, constructed from early embryonic mRNA, with monoclonal antibodies against Drosophila lamin.

references: Gruenbaum, Landesman, Drees, Bare, Saumweber, Paddy, Sedat, Smith, Benton, and Fisher, 1988, J. Cell Biol. 106: 585-96.

phenotype: The structural gene for nuclear lamin. Translated as a prolamin of apparent molecular mass of 76-kd, which is quickly processed to a 74-kd species, presumably by proteolysis. The processed polypeptide is assembled into the nuclear envelope, where it becomes phosphorylated and again attains a mass of 76 kd (Smith, Gruenbaum, Berrios, and Fisher, 1987, J. Cell Biol. 105: 771-90).

molecular biology: Original cDNA clone identified two transcripts of 2.8 and 3.0 kb; although they encode identical polypeptides, they display differential expression profiles on developmental Northern blots. They are equally abundant during oögenesis, but in the early embryo the 2.8-kb species is four times more abundant than the 3.0-kb species; the latter increases in intensity until the two are again equally abundant at mid gastrulation; by 7.5 h, only the 3.0-kb molecule persists. Full length cDNA clones of both have been recovered and sequenced; their differences reside in the lengths of their untranslated 3' regions. The gene encodes a polypeptide of 621 amino acids and calculated molecular mass of 70,974. Amino-acid sequence shows approximately 35% identity with human lamin; shows secondary structural features characteristic of intermediate filament proteins; contains a segment with 49% sequence identity with hamster vimentin.

Lam-: Laminin

Three genes encode the three subunits of Drosophila laminin, which associate to form a cruciform molecule in which the carboxy-terminal ends are alpha helical and associate with one another and whose amino-terminal ends are free to form three short arms of the cross.

locus	genetic location	cytological location ^α	molecular weight
<i>Lam-A</i>	3- {20}	65A10-11	400 kd
<i>Lam-B1</i>	2- {33}	28D	220 kd
<i>Lam-B2</i>	3- {34}	67C	180 kd

α localized by *in situ* hybridization.

references: Fessler, Campbell, Duncan, and Fessler, 1987, *J. Cell Biol.* 105: 2383-91.

Montell and Goodman, 1988, *Cell* 53: 463-73.

phenotype: The three genes are coordinately expressed with little or no transcript detectable prior to germ-band elongation. At eight hr, expression detected in the mesoderm and to a lesser extent in a subset of the ectoderm, including the epidermis. In the developing CNS grains associated with certain glial elements; no transcript seen after 18 hr. Immunocytochemical observations in embryos show laminin to be localized to basement membranes surrounding internal organs and muscles, in the underlying hypodermal epithelium and in the nervous system; also in basement membranes of larvae and adults. In the developing CNS, high levels are seen in the mesectodermal strand, in a pair of mesodermal cells near the segment border, in and around axon pathways, and in glial cells. Neurons and glia of the PNS also show abundant laminin immunoreactivity [Montell and Goodman, 1989, *J. Cell Biol.* 109: 2441-53 (fig.)].

Lam-B1

molecular biology: All three genes cloned. *LamB1* sequenced; conceptual amino acid sequence of 1784 residues comprises six domains: starting from the C-terminus, domains I and II separated by a short stretch termed α comprise 67 kd are largely α helical, show 25% and 22% homology with mouse B1 subunit, respectively, and comprise the segment of the chain that associates with the A and B2 subunits. The α region has 27 amino acids including six cysteines and eight glycines; it shows 36% homology with the mouse α sequence; five of the six cysteines and five of the eight glycines are conserved. Domains III (44 kd) and V (30 kd) contain eight and five cysteine-rich repeats that exhibit homology with epidermal growth factor; they are 54% and 53% homologous with the mouse polypeptide, respectively. Domains IV (25 kd) and VI (28 kd) show 58% and 26% homology with mouse, respectively and have been postulated to be collagen-binding domains.

Lam-B2

molecular biology: Sequence determined (Chi and Hui, 1989, *J. Biol. Chem.* 264: 1543-50; Montell and Goodman, 1989, *J. Cell Biol.* 109: 2441-53). Conceptual amino acid sequence contains 1639 residues. Domain structure similar to that of LAM-B1, except that LAM-B2 lacks the 27 amino acid cysteine-rich α domain that separates domains I and II in LAM-B1. There is also a large deletion in domain V and seven rather than eight EGF repeats in domain III; five more EGF repeats reside in domain V. Shows higher homology to mouse LAM-B2 than to *Drosophila* LAM-B1.

lame: see *lme*

Lamin: see *Lam*

Laminin: see *Lam-*

lance B: see *lanceolate*

lance: see *nw*²

lance-b: see *ll*

lanceolate: see *ll*

lao: see *Aldox*

Lap-A: Leucine aminopeptidase A

location: 3-98 (near *Lap-D*; no recombination yet observed).

references: Beckman and Johnson, 1964, *Hereditas* 51: 221-30.

Walker and Williamson, 1980, *Insect Biochem.* 10: 535-41.

Walker, Williamson, and Church, 1981, *Biochem. Genet.* 19: 47-60.

phenotype: Structural gene for leucine aminopeptidase A [LAP-A(E.C.3.4.1.1)], one of six such enzymes revealed by starch-gel electrophoresis. More anodally migrating than LAP-D; molecular weight 280,000 daltons; pH optimum 6.7. Enzyme apparently monomeric; no hybrid molecules formed in heterozygotes for electrophoretic variants. Appears 12 hrs after oviposition; found in larvae and pupae but not adults. Localized in larval hemolymph; not induced by substrate feeding.

alleles: Naturally occurring alleles: *LapA^F*, *LapA^S*, *LapA⁰*; fast, slow, and null alleles respectively.

Lap-D: Leucine aminopeptidase D

location: 3-98.3 (Falke and MacIntyre).

references: Beckman and Johnson, 1964, *Hereditas* 51: 221-30 (fig.).

Falke and MacIntyre, 1966, *DIS* 41: 165-66.

Muh, 1973, *DIS* 50: 200.

Walker and Williamson, 1980, *Insect Biochem.* 10: 535-41.

Walker, Geer, and Williamson, 1980, *Insect Biochem.* 10: 543-46.

Walker, Williamson, and Church, 1981, *Biochem. Genet.* 19: 47-60.

phenotype: Structural gene for leucine aminopeptidase D [LAP-D(E.C.3.4.1.1)], one of six such activities revealed by starch-gel electrophoresis and a staining with L-leucil-B-naphthylamide and Fast Black K salt; enzyme monomeric; molecular weight = 280,000 daltons; pH optimum 7.6; may be Zn metalloenzyme. Found in larvae and pupae, but not adults; highest in young larvae. Localized to larval midgut where levels are induced by dietary substrate.

alleles: Three alleles mentioned in literature, *LapD^F* and *LapD^S* plus an unnamed, uncharacterized one alluded to by Sakai, Tung, and Scandalios (1968, *Genetics* 60: 219-20). *Lap-D^F/Lap-D^S* produce equal amounts of slowly and rapidly migrating LAP D and no enzyme of intermediate mobility.

Large: see *Lg*

Larval cuticle protein: see *Lcp*

Larval serum: see *Lsp*

Larval visceral protein: see *Lvp*

late hatching: see *lh*

Lcp: Larval cuticle protein

A series of genes encoding electrophoretically separable proteins extractable from the cuticles of third-instar larvae, but not other stages. Four immunologically related proteins are encoded by a cluster of genes on 2R

and another cluster of three unrelated to the preceding four, but related to each other, located on 3L. Designated numerically in order of increasing mobility on non-denaturing gels of encoded polypeptide, with the exception of *Lcp10*.

locus	genetic location	cytological location	ref ^α
<i>Lcp1</i>	2-59.4	44D	2, 3, 4
<i>Lcp1</i> ψ	2-59.4	44D	5
<i>Lcp2</i>	2-59.4	44D	2, 3, 4
<i>Lcp3</i>	2-59.4	44D	2, 3, 4, 5
<i>Lcp4</i>	2-59.4	44D	3, 4, 5
<i>Lcp5</i>	3-11		1, 2
<i>Lcp6</i>	3-11		1, 2
<i>Lcp7</i>			2
<i>Lcp8</i>	3-11		1, 2
<i>Lcp10</i>	3-11		1

^α 1 = Chihara and Kimbrell, 1986, Genetics 114: 393-404; 2 = Fristrom, Hill and Watt; 3 = Snyder, Hirsh, and Davidson, 1981, Cell 25: 165-77; 4 = Snyder, Hunkapiller, Yuen, Silvert, Fristrom, and Davidson, 1986, Cell 29: 1027-40; 5 = Snyder, Kimbrell, Hunkapiller, Hill, Fristrom, and Davidson, 1982, Proc. Nat. Acad. Sci. USA 79: 7430-34.

Lcp1-4

phenotype: Encode larval cuticle proteins CP1, CP2, CP3, and CP4 (alternatively L₃CP₁ to L₃CP₄); molecular weights 17.5, 17.5, 9 and 13 kd respectively. Each has a 15-residue signal peptide.

allele	origin	ref ^α	comments
<i>Lcp1^F</i>	population	1	
<i>Lcp1^S</i>	population	1	
<i>Lcp2^F</i>	population	1, 2	with <i>Lcp3^{nl}</i>
<i>Lcp2^S</i>	population	1, 2	
<i>Lcp3¹</i>	population	1, 2	
<i>Lcp3^{nl}</i>	population	1, 2	with <i>Lcp2^F</i>
	H.M.S. Beagle		
<i>Lcp4¹</i>	population	1	

^α 1 = Fristrom, Hill, and Watt; 2 = Snyder, Kimbrell, Hunkapiller, Hill, Fristrom, and Davidson, 1982, Proc. Nat. Acad. Sci. USA 79: 7430-34.

molecular biology: These four genes and a pseudogene are clustered within 7.9 kb of DNA located in region 44D. The genes are arranged in numerical order along the chromosome, but polarity with respect to the centromere is not known. Each gene is approximately 0.5 kb in length; *Lcp1* and *Lcp2* are separated by 2.9 kb, *Lcp2* and *Lcp3* by 0.87 kb, and *Lcp3* and *Lcp4* by 1.6 kb. All four genes contain a short intron between the third and fourth codon of the signal peptide. *Lcp1* and *Lcp2* show 91% amino acid homology, *Lcp3* and *Lcp4* show 85%, and between these two pairs there is approximately 60% homology (Snyder, Hirsh, and Davidson). A putative pseudogene, whose sequence suggests origin via unequal crossing over between *Lcp1* and *Lcp2*, found between these two genes, 500-600 bp from *Lcp2*. Transcription of *Lcp1*, *Lcp1psi* and *Lcp2*, takes place off of the opposite strand and divergently from that of *Lcp3* and *Lcp4* (Snyder, Hunkapiller, Yuen, Silvert, Fristrom, and Davidson). *Lcp2^F* gene product has been shown to differ from that of the more common *Lcp2^S* allele by two amino acid substitutions; *Lcp3^{nl}* contains a 7.3 kb DNA insertion, identified as H.M.S. Beagle, located within the TATA box (Snyder, Kimbrell, Hunkapiller, Hill, Fristrom and Davidson).

Lcp5

alleles: *Lcp⁺* is the commonly encountered allele; the other alleles are infrequent. Electrophoretic variants, when homozygous, may reveal two bands, one at the new position and one at the original position; these alleles are designated *r* for residual. Others leave no residual band and are designated *nr*.

allele	origin	ref ^α	comments
<i>Lcp5⁺</i>	population	1, 3	
<i>Lcp5^{FF}</i>	population	1, 2	very fast
<i>Lcp5^{Fnr}</i>	population	1, 3	fast, no residual
<i>Lcp5^{Fr}</i>	population	1	fast, residual
<i>Lcp5^{op}</i>	population	1, 2	over producer
<i>Lcp5^{Snr}</i>	population	1, 2	slow, no residual
<i>Lcp5^{Sr}</i>	population	1, 3	slow, residual

^α 1 = Chihara and Kimbrell, 1986, Genetics 114: 393-404; 2 = del Puerto, 1985, Thesis, University of San Francisco; 3 = Fristrom, Hill, and Watt, 1978, Biochemistry 17: 3917-24.

Lcp6

location: 3-11 (not separated from *Lcp5* among 487 F₁).

allele: *Lcp6⁺* is the common allele; the remainder rare or relatively so.

allele	origin	ref ^α	comments
<i>Lcp6⁺</i>	population	1, 3	
<i>Lcp6^f</i>	population	1, 2	faint, polymorphic
<i>Lcp6ⁿ</i>	population	1, 2	null allele
<i>Lcp6^S</i>	population	1, 2	slow

^α 1 = Chihara and Kimbrell, 1986, Genetics 114: 393-404; 2 = del Puerto, 1985, Thesis, University of San Francisco; 3 = Fristrom, Hill, and Watt, 1978, Biochemistry 17: 3917-24.

Lcp7

phenotype: Structural gene for CP7, which is antigenically related to CP5, CP6, and CP8.

alleles: One active allele plus a null allele, *Lcp7ⁿ* (Fristrom, Hill, and Watt, Biochemistry 17: 3817-24).

Lcp8

location: 3-11 (not separated from *Lcp5* in 563 F₁).

allele	origin	ref ^α	comments
<i>Lcp8⁺</i>	population	1, 2	
<i>Lcp8^S</i>	population	1	slow

^α 1 = Chihara and Kimbrell, 1986, Genetics 114: 393-404; 2 = Fristrom, Hill, and Watt, 1978, Biochemistry 17: 3917-24.

Lcp10

location: 3-11 (not separated from *Lcp5* in 524 F₁).

synonym: Rho.

references: Chihara and Kimbrell, 1986, Genetics 114: 393-404.

phenotype: No gene product detectable in wild type, only in the ethyl methanesulfonate-induced allele, *Lcp10^{rho}*; migrates between CP3 and CP4. Not clear that the mutant product is confined to third-instar larval cuticle.

alleles:

allele	origin	comments
<i>Lcp10⁺</i>	population	naturally repressed
<i>Lcp10^{rho}</i>	EMS	derepressed

ld: loboid

location: 3-102 [between *ca* and *bv* (Lewis, 1956, DIS 30: 130)].

origin: Spontaneous.

discoverer: Curry, 39a.

references: 1939, DIS 12: 45.

phenotype: Eyes resemble *L/+*. Malformation of eyes ranges from slight dorsoventral seam across middle of eye to a more extreme effect in which growth of anterior part is completely inhibited in most-extreme cases. Antenna-like outgrowth frequent where growth of eyes is suppressed. Tends to overlap wild type. In the presence of a sex-linked modifier, *oph* [*oph*; *ld* designated *ld^{oph}* by Kobel (1968, Genetica 39: 329-44)] *ld* flies show homeotic transformation of eye to wing tissue (Ouweneel, 1969, Wilhelm Roux's Arch. Entwicklungsmech. Org. 164: 1-114 and 14-36; 1970, 166: 76-88; 1970, Genetica 41: 1-20). RK3.

alleles	origin	discoverer	synonym	ref ^α	comments
<i>ld¹</i>	spont	Curry, 39a		1	
* <i>ld^{52a}</i>	spont	Edmonson, 52a		2	like <i>ld¹</i>
<i>ld^{eyr}</i>	spont		<i>eyr</i>	3, 4	details below

^α 1 = Curry, 1939, DIS 12: 45; 2 = Edmonson, 1952, DIS 26: 60; 3 = Edwards and Gardner, 1963, DIS 37: 47; 4 = Edwards and Gardner, 1966, Genetics 53: 785-98.

cytology: Tentative; placed in 99C-F based on its genetic localization.

****ld^{eyr}*: loboid-eyes reduced**

origin: Found among flies grown on food containing copper sulfate.

phenotype: Eyes vary from normal to absence of ommatidia. Shows some degree of dominance; many heterozygotes have some eye abnormality, usually a nick in anterior region of one or both eyes; an abnormal growth of wing tissue may be associated with the nick. *ld^{eyr}*; *ey⁴* flies have very small heads, usually without ommatidia. Viability greatly reduced. RK2.

lds: lodestar

location: 3-47.

synonym: *early*.

references: Tearl and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Maternal. In nuclear cycles 9-10, multiple complex anaphase spindles share several centrosomes. Multiple groups of chromosomes are often directed to same pole. Elongated polyploid nuclei develop. Mitotic abnormalities in larval brain cells (Gatti).

alleles: Four alleles, *lds¹*-*lds⁴*, isolated as 042, 072, 098, and 298.

cytology: Placed in 88D13-14 (Ashburner).

lea: leak

location: 2-3.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

phenotype: Embryonic lethal. Head involution incomplete. In combination with *Pc*-like mutants, abdominal transformations occur.

alleles: Two, *lea¹* and *lea²*, isolated as 25 and IIS.

cytology: Placed in 21F2-22B1 based on its inclusion in *Df(2L)S2 = Df(2L)21C6-D1;22A6-B1* but not *Df(2L)S3*

= Df(2L)21D2-3;21F2-22A1.

leg: see *run*

leg tumor: see *Igt*

****lem: lemon***

location: 1-17.5.

origin: Spontaneous.

discoverer: E. M. Wallace, 12h.

references: Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. No. 237: 48 (fig.).

phenotype: Body color pale yellow with dark trident and black bristles. Wings and veins pale yellow. Easily distinguished from wild type, viability about 70% wild type, and most flies sterile. RK3.

Les: Lesbian (J.C. Hall)

location: Complex genetic etiology.

origin: Isolated from a series of related, balancer-containing strains (used to maintain the *Fs(2)B* mutation).

references: Cook, 1975, Nature 254: 241-42.

phenotype: Females exhibit male-like courtship, including unilateral wing display, directed at other females; courtship bouts relatively short; not all females of the strain show the anomalous behavior; these behaviors have been observed in females of other strains (Cook, 1981, Z. Naturforsch. 36C: 475-83).

lethal (): see *l()*

Lethal hybrid rescue: see *Lhr*

Leucine aminopeptidase A: see *Lap-A*

Leucine aminopeptidase D: see *Lap-D*

Levente : see *Fs(3)Sz28*

lf: little fly

location: 1-68.1.

phenotype: Small fly with markedly narrow abdomen, frequently with small tumors. Ecdysis delayed; low viability and fertility in both sexes, especially females; some alleles lethal. RK3.

allele	origin ^α	discoverer	synonym	ref ^β	comments
<i>lf¹</i>	N Mustard	Fahmy, 1954		1	
<i>lf²</i>	CB1506	Fahmy, 1954		1	
<i>lf³</i>	EMS	Fahmy, 1954		1	
<i>lf⁴</i>	EMS	Fahmy, 1954		1	
<i>lf⁵</i>	CB1592	Fahmy, 1954		1	
<i>lf⁶</i>	CB3007	Fahmy, 1954		1	
<i>lf⁷</i>	CB3025	Fahmy, 1954		1	
<i>lf⁸</i>	CB3051	Fahmy, 1954		1	
<i>lf⁹</i>	X ray	Fahmy, 1954		1	
<i>lf¹⁰</i>	X ray	Lifschytz	<i>l(1)A58</i>	3, 4, 5	probably multilocus
<i>lf¹¹</i>	EMS	Lifschytz	<i>l(1)M122</i>	5	on <i>y⁺Ymal⁺</i>
<i>lf¹²</i>	X ray	Lefevre	<i>l(1)L40</i>	2	
<i>lf¹³</i>	X ray	Lefevre	<i>l(1)HC142</i>	2	

^α CB1506 = 2-chloroethyl methanesulfonate; CB1592 = ?; CB3007 = DL-*p*-N,N-di-(2-chloroethyl)aminophenylalanine; CB3025 = L-*p*-N,N-di-(2-chloroethyl)aminophenylalanine; CB3051 = ?.

^β 1 = Fahmy, 1959, DIS 33: 87; 2 = Lefevre, 1981, Genetics 99: 461-80; 3 = Lifschytz and Falk, 1968, Mut. Res. 6: 235-44; 4 = Lifschytz and Falk, 1969, Mut. Res. 8: 147-55; 5 = Lifschytz and Yakobovitz, 1978, Mol. Gen. Genet. 161: 275-84.

cytology: Placed in 19E5-6 based on its inclusion within *Df(1)T2-14A = Df(1)19E5-6;19E7-8* but not in *Df(1)Q539 = Df(1)19E6;19F6-20A1.*

***lfb*: little faint ball**

location: 3-26.
origin: Induced by ethyl methanesulfonate.
references: Jürgens, Kluding, Nüsslein-Volhard, and Wieschaus, 1983, DIS 59: 157-58.
phenotype: Embryonic lethal; embryo resembles faint little ball? Dorsal closure incomplete.
alleles: Five.

Lff11 : see *l(1)ff11*

***lfl*: Little fly like**

location: 1- {66}.
phenotype: Lethal under crowded conditions, but semilethal (20-30% survival) under optimal conditions. Survivors delayed three days in emergence. Adults small like *lf* but without the narrow abdomen. *lfl*²/*Df* not particularly small; lack thoracic hairs and bristles on head and thorax; also have rough eyes; *lfl*² males: survival of 1% expected; phenotype as in above females. *FM6/lfl*² females and *y*⁺*Ymal*¹⁰⁶/*lfl*² males frequently lack ocellar, postvertical and humeral bristles; posterior crossveins may be interrupted and vein L5 usually interrupted; some thoracic hairs absent. *FM6/lfl*¹ females normal.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>lfl</i> ¹	X ray	Lifschytz	<i>l(1)B56</i>	4, 5, 7	
<i>lfl</i> ²	X ray	Lifschytz	<i>l(1)B96</i>	4, 5, 7	
<i>lfl</i> ³		Lifschytz	<i>l(1)J22</i>	6	
<i>lfl</i> ⁴	X ray	Lefevre	<i>l(1)C94</i>	2	
<i>lfl</i> ⁵	X ray	Lefevre	<i>l(1)C231</i>	2	<i>T(1;3)19F1-2;80</i>
<i>lfl</i> ⁶	X ray	Lefevre	<i>l(1)JA128</i>	2	
<i>lfl</i> ⁷	X ray	Lefevre	<i>l(1)N109</i>	2	
<i>lfl</i> ⁸	EMS	Lefevre	<i>l(1)DF970</i>	3	
<i>lfl</i> ⁹	EMS	Lefevre	<i>l(1)EF446</i>	4	
<i>lfl</i> ¹⁰	EMS	Lefevre	<i>l(1)VA241</i>	5	
<i>lfl</i> ¹¹	HMS	Kramers	<i>l(1)HM46</i>	1	

^α 1 = Kramers, Schalet, and Huizer-Hoogteyling, 1983, Mut. Res. 107: 187-201; 2 = Lefevre, 1981, Genetics 99: 461-80; 3 = Lefevre, and Watkins, 1986, Genetics 113: 869-95; 4 = Lifschytz and Falk, 1968, Mut. Res. 6: 235-44; 5 = Lifschytz and Falk, 1969, Mut. Res. 8: 147-55; 6 = Lifschytz and Jakobovitz, 1978, Mol. Gen. Genet. 161: 275-84; 7 = Schalet and Lefevre, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902.

cytology: Placed in 19F1-3 based on its inclusion in *Df(1)DCB35b = Df(1)19F1-2;20E-F* but not in *Df(1)17-257 = Df(1)19F3;20A1-2*.

****Lg*: Large**

location: 1-27.
origin: Induced by P³².
references: Bateman, 1950, DIS 24: 55.
phenotype: Heterozygote large, late eclosing, with visibly smaller hairs; viability excellent. Tendency toward shortening of L4 and L5, missing postvertical bristles, and islands of vein tissue on either side of L2. Homozygous lethal. RK2.

****lgh*: long haired**

location: 1-20.7.
origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).
discoverer: Fahmy, 1956.
references: 1959, DIS 33: 87.
phenotype: Small fly; size reduction most noticeable in head and thorax. Wings short and slightly altered in

shape. Anterior thorax frequently dented in the mid-dorsal line. Hairs deranged; bristles long and scraggly. Abdomen nearly always abnormally pigmented, ranging from no melanization of tergites 5-7 to small, irregular, under-pigmented patches on these tergites. Male viability about 25% wild type. Males sterile. RK3.

lgl : see *l(2)gl*

lglG : see *ovo*^{Drv22}

****lgt*: leg tumor**

location: 2- (not located).
origin: Spontaneous.
discoverer: Spencer, 36c20.
references: 1937, DIS 7: 14.
phenotype: Black tumor growth inside thorax ventrally at bases of posterior legs. Sterile in both sexes; poor viability. RK3.

***lh*: late hatching**

location: 1-57.
origin: Spontaneous.
discoverer: Bridges, 31d6.
phenotype: Slow-developing semigiant. RK3.

LHP : see *Lsp2*

***Lhr*: Lethal hybrid rescue**

location: 2-95 (in *Drosophila simulans*).
origin: Spontaneous.
references: Watanabe, 1979, Jpn. J. Genet. 54: 325-31. Takamura and Watanabe, 1980, Jpn. J. Genet. 55: 405-08.
phenotype: When *Drosophila simulans* carries *Lhr*, classes of hybrids between *D. simulans* and *D. melanogaster* that do not ordinarily survive are recovered. Crosses of *D. melanogaster* females by *D. simulans* males, which ordinarily produce only daughters, produce sons and daughters in a 1:1 ratio. Crosses of *D. simulans* females to *D. melanogaster* males, which ordinarily produce only sons, produce 86% sons and 14% daughters. Crosses of *C(1)RM* - bearing *D. melanogaster* females to *D. simulans* males, which normally produce only sons, yield a few daughters when the *D. simulans* males carry *Lhr*.

light: see *lt*

lightoid: see *ltd*

limited: see *lm*

***lin*: lines (C. Nüsslein-Volhard)**

location: 2-59.
origin: Induced by ethyl methanesulfonate.
references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.
phenotype: Embryonic lethal; small anterior portion of each segment deleted; A8, spiracles, and anal plates absent. Head abnormal.
alleles: Three alleles, *lin*¹-*lin*³, isolated as 5E, IIF, and IIV.
cytology: Tentatively placed in 44F-46D.

little faint ball: see *lfb*

little fly: see *lf*

little fly like: see *lfl*

lix: little Isoxanthopterin**location:** 1-23.**origin:** Spontaneous.**discoverer:** Hessler, 1959.**references:** 1960, DIS 34: 50.

Hubby, 1962, Genetics 47: 109-14.

phenotype: Most likely the structured gene for dihydropterin oxidase. Flies indistinguishable from wild type; dissected testis sheath dark yellow-orange, but this character is not dependable for classification; causes striking changes in compounds that fluoresce in ultraviolet light on paper chromatograms of testes. Isoxanthopterin content of testis sheath greatly reduced. No detectable dihydropterin oxidase activity (Ordoño, Silva, and Ferré, 1988, DIS 67: 63). A blue fluorescent compound not otherwise detected in *D. melanogaster* (the *lix* substance) is present. Drosopterins present in the testis sheath, and quantities of sepiapteridine, biopterin, "Compound A," and a "riboflavinlike" compound are elevated. The colored pteridine gives testis sheath its darker color. Pteridine accumulation in testis sheath alone is affected. RK3.

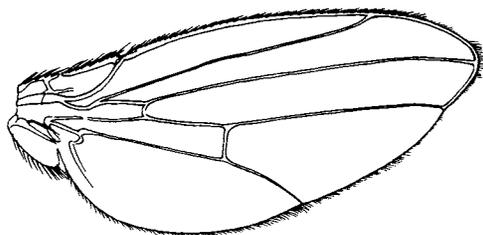
cytology: Placed in 7D10-F2 based on its inclusion in *Df(1)RA2 = Df(1)7D10;8A4-5* but not *Df(1)KA14 = Df(1)7F1-2;8A6* (Silva, Escriche, Ordoño, and Ferré).

liz: see *fs(1)A1621***ll: lanceolate****location:** 2-106.7.**origin:** Spontaneous.**discoverer:** Bridges, 23d3.**synonym:** lance-b.**references:** Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 227.

Bridges, 1937, Cytologia (Tokyo, Fujii Jub., Vol. 2: 745-55.

phenotype: Wings narrowed at tips and slightly divergent. Eyes slightly smaller than normal and bulging; head narrow. Waddington finds wing effect detectable in middle pupal stage. RK3.

cytology: Placed in region between 59E2 and 60B10 on the basis of its being to the right of *In(2R)bw^{VDel} = In(2R)41B2-C1;59E2-4* and to the left of *Df(2R)Px = Df(2R)60B8-10;60D1-2*.

**ll²: lanceolate-2**

Edith M. Wallace, unpublished.

ll²**origin:** Spontaneous.**discoverer:** Bridges, 23d25.

phenotype: Wings pointed and narrow. Eyes small and bulging. Head narrow. Wing shape first seen in early contraction stage of wing development (23-hr pupa at

25°) (Waddington, 1939, Proc. Nat. Acad. Sci. USA 25: 303). More extreme and more useful than *ll*. RK2.

llb: see *l(3)89Ea***lm: limited****location:** 2-50.**origin:** Spontaneous.**discoverer:** Bridges, 29125.

phenotype: Sternites small, rounded, or irregular; bristles sparse. Females sterile. RK3.

cytology: Not included in *Df(2L)64j = Df(2L)34E5-F1;35C3-D1* (E. H. Grell).

lme: lame*location:** 1-47.8.

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1956.**references:** 1959, DIS 33: 87.

phenotype: Legs weak, frequently deformed, and generally shortened as a result of reduction in length of tarsal segments. Wings atypically shaped and abnormally held. Flies so crippled they cannot move; they die soon after eclosion. RK3.

lme: see *l(2)me**Lms*: see *kl-***Lobe:** see **L****loboid:** see **ld****lobula-plateless:** see **lop****lon: longevity****location:** 1-21.7.**origin:** Induced by ethyl methanesulfonate.

references: King, Bahns, Horowitz, and Larramendi, 1978, Int. J. Insect Morphol. Embryol. 7: 359-75.

phenotype: Homozygotes and hemizygotes exhibit delay in eclosion of four days; become paralyzed at nine to ten days of age and die on day eleven or twelve.

cytology: Distal to the breakpoint of *T(1;Y)B138*, which is in 7F6-8.

long haired: see **lgh****lop: lobula-plateless (J.C. Hall)****location:** 2-ca. 70.**origin:** Induced by ethyl methanesulfonate.**synonym:** *N684*.**discoverers:** Heisenberg and Fischbach.

references: Fischbach and Heisenberg, 1984, J. Exp. Biol. 112: 65-93.

Bülthoff and Buchner, 1985, J. Comp. Physiol. 156: 25-34.

Helfrich, 1986, J. Neurogenet. 3: 321-43.

phenotype: Lobula plate (third-order optic ganglion) missing most of its usual neuropile (Fischbach and Heisenberg, 1984), including an absence of most "small-field" visual neurons; but the giant fibers of this lobe remain; certain of these (*i.e.* "VS"-cells) enter the neuropile of a more peripheral lobe (the medulla), as opposed to their orthodox (more central) targets; optomotor roll and pitch responses to movements in the visual field are weak or absent. Visually mediated stimulus-specific labelling (in 2-deoxyglucose experiments) does not occur in lobula

plate, though it is normal in the mutant's lamina (first order optic lobe) and medulla (Bülthoff and Buchner, 1985). Circadian rhythms of locomotor activity are quite robust and have normal *ca.* 24 hours free-running periodicities (Helfrich, 1986).

alleles: One mutant allele, *lop*¹ (= *lop*^{N684}).

lot (J.C. Hall)

location: 1-unlocalized.

synonym: *lot-235*.

origin: Induced by ethyl methanesulfonate.

references: Falk and Atidia, 1975, *Nature* 254: 325-26.

phenotype: Like *Lot* but non-allelic and recessive.

Lot (J.C. Hall)

location: 1-55.5 (to left of *f* between 55.5 - 56.0).

origin: Induced by ethyl methanesulfonate.

synonym: *Lot-94*.

references: Falk and Atidia, 1975, *Nature* 254: 325-26.

Atidia and Baker, 1974, *DIS* 51: 72.

Falk, 1979, *J. Insect Physiol.* 25: 87-91.

phenotype: Adults consume solutions of sodium chloride 10X more concentrated than will wild type; largely due to fact that *Lot/Lot* > *Lot/+* > *+/+* in liquid consumption; named *Lot* (Genesis 19 v 26) based on a perceived reduction in aversion to salt. Subsequently found that the amount of NaCl that had to be added to 0.1 M sucrose to switch preference from 0.1 M to 0.01 M sucrose same as wild type (Falk, 1979, *J. Insect Physiol.* 25: 87-91).

alleles: *Lot*¹, semidominant; *Lot*^r (= *Lot114*), completely recessive.

low aldehyde oxidase: see *Aldox*

low xanthine dehydrogenase: see *lxd*

lozenge: see *lz*

lozenge-like: see *rstl*

lozengelike: see *lzl*

lpo: see *Po*

lrb: see *l(3)89Ee*

Lsp1: Larval serum protein 1

Genes encoding three related polypeptides that combine in random hexamers to form LSP1, a heterogenous larval serum protein of 450,000 to 480,000 daltons (Wolfe, Akam, and Roberts, 1977, *Eur. J. Biochem.* 79: 47-53). LSP1 synthesized in fat body; appears early in third instar attaining peak levels at the white prepupal stage; level begins to decline at mid-pupal stage reaching base line about four days into adult life (Roberts, Wolfe, and Akam, 1977, *J. Insect Physiol.* 23: 871-78). Highly homologous to one another and all three share amino-acid homology with LSP2. Antigenically related as well but antisera to LSP1 polypeptides do not crossreact with LSP2 (Brock and Roberts, 1980, *Eur. J. Biochem.* 106: 129-35).

Lsp1 α : Larval serum protein-1 α chain
(D.B. Roberts)

location: 1-39.5 (27/47 of the distance from *v* to *g*).

discoverer: Evans-Roberts, 1977.

references: Roberts, Wolfe, and Akam, 1977, *J. Insect Physiol.* 23: 871-78.

Roberts and Evans-Roberts, 1979, *Genetics* 93: 663-79.

Smith, McClelland, White, Addison, and Glover, 1981, *Cell* 23: 441-49.

McClelland, Smith and Glover, 1981, *J. Mol. Biol.* 153: 257-72.

Roberts and Evans-Roberts, 1979, *Nature* 280: 691-92.

Ghosh, Chatterje, Bunick, Manning and Lucchesi, 1989, *EMBO J.* 8: 1191-96.

phenotype: Codes for the α polypeptide of LSP1; molecular weight 83,000 (Brock and Roberts, 1980, *Eur. J. Biochem.* 106: 129-35).

alleles: Electrophoretic variants isolated from natural populations: *Lsp1 α ^F*, *Lsp1 α ^{SI}*, and *Lsp1 α ^{S2}*.

cytology: Localized to 11A7-9 by *in situ* hybridization (Smith *et al.*). Included in *Df(1)HF368* = *Df(1)11A2;11B9* but not *Df(1)v65b* = *Df(1)9F13-10A1;11A7-8* (Roberts and Evans-Roberts).

molecular biology: The *Lsp1 α* gene has been cloned; the cloned DNA cross hybridizes to the *Lsp1 β* and γ genes; encodes a 2.85 kb polyadenylated mRNA (Smith *et al.*); clone DNA partially characterized and shows that the gene has a short intervening sequence of about 50 bp 400 nucleoties from the 5' end (McClelland *et al.*). 1.5 kb at 5' end of gene sufficient for normal control of tissue and stage specificity (Yedvobnick and Levine, 1982, *Nature* 297: 239-41). Transformants carrying the bacterial *Cat* gene linked to 377 base pairs of upstream sequence from *Lsp1 α* express CAT with the same developmental and tissue specificity as the endogenous *Lsp1 α* gene (Delaney, Sunkel, Genova-Seminova, Davies, and Glover, 1987, *EMBO J.* 6: 3849-54).

other information: Although X-linked *Lsp1 α* is not dosage compensated (Roberts and Evans-Roberts); however when relocated by transformation to an autosomal site or to an ectopic site on the X, the gene does show dosage compensation (Ghosh *et al.*, 1989).

Lsp1 β (D.B. Roberts)

location: 2-1.9 (4/28 of the distance from *al* to *dp*).

references: Roberts, Wolfe, and Akam, 1977, *J. Insect Physiol.* 23: 871-78.

Roberts and Evans-Roberts, 1979, *Genetics* 93: 663-79.

Smith, McClelland, White, Addison, and Glover, 1981, *Cell* 23: 441-49.

McClelland, Smith, and Glover, 1981, *J. Mol. Biol.* 153: 257-72.

Brock and Roberts, 1981, *Chromosoma* 83: 159-68.

phenotype: Codes for the polypeptide of LSP1 β ; a phosphorylated polypeptide of molecular weight 80,000 (Brock and Roberts, 1980, *Eur. J. Biochem.* 106: 124-35).

alleles: Naturally occurring electrophoretic variants: *Lsp1 β ⁺* (the predominant allele), *Lsp1 β ^F* and *Lsp1 β ⁿ*, a null allele.

cytology: Located to 21D3-5 by *in situ* hybridization (Smith *et al.*).

molecular biology: The *Lsp1 β* gene has been cloned; the cloned DNA cross hybridizes to the *Lsp1 α* and γ genes; encodes a 2.85 kb polyadenylated mRNA (Smith *et al.*); cloned DNA partially characterized; shows a short intervening sequence of about 50 bp 400 nucleotides from the 5' end of the gene (McClelland *et al.*). Transformants carrying the bacterial *Cat* gene linked to 471, but not 66, base pairs of upstream sequence from *Lsp1 β* express CAT with the same developmental and tissue specificity as the

endogenous *Lspβ* gene (Delaney, Sunkel, Genova-Seminova, Davies, and Glover, 1987, EMBO J. 6: 3849-54).

other information: *Dp(2;2;2;2)S* probably carries five copies of the *Lsp1β* gene (Brock and Roberts).

Lsp1γ (D.B. Roberts)

location: 3-1.4 (2/15 of the distance from *Dp(1;3)sc^{J4}* to *ve*).

references: Roberts, Wolfe, and Akam, 1977, J. Insect Physiol. 23: 871-78.

Roberts and Evans-Roberts, 1979, Genetics 93: 663-79.
Smith, McClelland, White, Addison, and Glover, 1981, Cell 23: 441-49.

McClelland, Smith, and Glover, 1981, J. Mol. Biol. 153: 257-72.

phenotype: Codes for the γ chain of LSP1; a phosphorylated polypeptide of molecular weight 77,000 (Brock and Roberts, 1980, Eur. J. Biochem. 106: 129-35).

alleles: In addition to *Lspγ⁺*, four null alleles recorded: *Lspγ⁰¹*, *Lspγ⁰²*, *Lspγ⁰³*, and *Lspγ⁰⁴*. *Lsp2^F* said to be homozygous lethal (Brock and Roberts, 1980, Eur. J. Biochem. 106: 129-35).

cytology: Localized to 61A1 by *in situ* hybridization (Smith *et al.*); 61A1-3 (Brock); uncovered by the deficiency generated by *T(Y;3)S50* (Roberts and Evans-Roberts).

molecular biology: The *Lsp1γ* gene has been cloned; cloned DNA cross hybridizes to the *Lsp1α* and β genes; encodes a 2.85 kb polyadenylated mRNA (Smith *et al.*); cloned DNA partially characterized; shows short intervening sequence of about 50 bp 400 nucleotides from the 5' end of the gene (McClelland *et al.*).

Lsp2: Larval serum protein-2 (D.B. Roberts)

location: 3-37 (3/17 of the distance from *vin* to *gv*).

discoverer: Akam, 1977.

synonym: *Pt-1, LHP*.

references: Hubby, 1963, Genetics 48: 871.

Roberts, Wolfe, and Akam, 1977, J. Insect Physiol. 23: 871-78.

Akam, 1977, D. Phil. Thesis, Oxford University.

Akam, Roberts, Richards, and Ashburner, 1978, Cell 13: 215-25.

Akam, Roberts, and Wolfe, 1978, Biochem. Genet. 16: 101-19;

Lepesant, Levine, Garen, Lepesant-Kejzlarova, Rat, and Somme-Martin, 1982, J. Mol. Appl. Genet. 1: 371-83.

Paco-Larson, Nakanishi, Levine, and Garen, 1986, Dev. Genet. 7: 197-203.

Paco-Larson, Nakanishi, Levine, and Garen, 1986, Dev. Genet. 7: 197-203.

phenotype: Codes for LSP2, a glycosylated polypeptide of 80,000 molecular weight (Brock and Roberts, 1980, Eur. J. Biochem. 106: 129-35) and one of the major serum proteins of third-instar larvae; a homohexamer of molecular weight 450,000 with subunit molecular weight of 78-83 X 10³ daltons. LSP2 appears during the third larval instar and levels increase until midway through the pupal stage; then levels decline reaching base line early in adult life. Transcription detectable only in third-instar fat body, ecdysone dependent; in presence of temperature-sensitive *ecd*, *Lsp2* transcription off at restrictive temperature; resumes following ecdysterone administration or shift to permissive temperature;

juvenile hormone blocks ecdysterone stimulation.

alleles: Naturally occurring alleles, *Lsp2^F* and *Lsp2^S*.

cytology: Localized to 68E3-4 by deficiency mapping (Akam *et al.* 1978); 68E by *in situ* hybridization (Lepesant *et al.*).

molecular biology: The *Lsp2* gene has been cloned (Lepesant *et al.*).

lt: light

location: 2-55.0.

references: Bridges, 1931, Eos 7: 229-48.

de Zulueta, 1931, Eos 7: 249-53.

phenotype: Eye color yellowish pink-lighter at high temperature, darker at low. Ocelli colorless; Drosoterins drastically reduced; no maternal effect on eye pigmentation (Nickla, 1972, Can. J. Genet. Cytol. 14: 105-11). When combined with *st*, eye color only slightly lighter than with *lt* alone; with *bw* it is clear yellow, pinkish in old flies (Schultz and Dobzhansky, 1934, Genetics 19: 344-64; Mainx, 1938, Z. Indukt. Abstamm. Vererbungslehre 75: 256-76). Eye color autonomous in mutant optic discs transplanted into wild-type hosts (Beadle and Ephrussi, 1936, Genetics 21: 230). Larval Malpighian tubes colorless in *lt* offspring of *lt* mothers; some color in tubes if mother is *lt/+*. The quantity of yellow pigment, composed primarily of riboflavin, in Malpighian tubes exhibits parallel maternal effects in larvae, pupae, and adults (Nickla, 1972, Can. J. Genet. Cytol. 14: 391-96); the amount of such pigment increases with maternal age for both *lt/+* and *lt/lt* parental females (Nickla, 1973, Can. J. Genet. Cytol. 15: 437-42). *lt stw* homozygotes completely inviable (Purdum); however, *lt stw³* homozygotes have good viability. *car lt* double mutants are also invariably lethal; the time of death varying from the third larval instar to late pupa, depending on the number of normal alleles of either gene carried by the mother (Nickla, 1977, Nature 268: 638-339). Lethal focus of the lethal interaction as measured in *car/+; lt/lt, car/0; lt/lt* gynandromorphs is in ventral nervous tissue (Nickla, Lilly, and Brown, 1980, Experientia 36: 402-03); larval brain histologically abnormal (McCarthy and Nickla, 1980, Experientia 36: 1361-62).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>lt¹</i>	spont	Bridges, 24d8		2, 3, 4	
<i>*lt²</i>	spont	Bridges, 30b14		2, 3	weak allele
<i>lt³</i>	spont	Beadle, 36e23		3	in <i>lt(2L+2R)Cy₁</i> al ² <i>Cy on 2L⁴ sp²</i>
<i>lt⁴</i>	UV	Meyer, 50d		3, 9	intermediate allele homozygotes short lived, sterile
<i>*lt⁵</i>	UV	Meyer, 51d		3, 8	lethal allele
<i>lt⁶</i>	EMS	Hilliker		5	
<i>lt⁷</i>	EMS	Hilliker		5	
<i>lt⁸</i>	EMS	Hilliker		5	
<i>lt⁹</i>	EMS	Hilliker		5	
<i>lt¹⁰</i>	EMS	Hilliker		5	
<i>lt¹¹</i>	EMS	Hilliker	<i>l(2)EMS40-12</i>	5	lethal
<i>lt¹²</i>	EMS	Hilliker	<i>l(2)EMS40-17</i>	5	lethal
<i>lt¹³</i>	EMS	Hilliker	<i>l(2)EMS56-03</i>	5	lethal
<i>lt¹⁴</i>	P	Wakimoto	<i>lt^{hd51}</i>		no P insert
<i>lt¹⁵</i>	P	Wakimoto	<i>lt^{hd52}</i>		P insert
<i>lt¹⁶</i>	EMS		<i>H</i>		
<i>lt¹⁷</i>	EMS		<i>lt^{X54}</i>		
<i>lt¹⁸</i>	X ray		<i>lt^{X69}</i>		
<i>lt¹⁹</i>	X ray		<i>lt^{VO17}</i>		
<i>lt²⁰</i>	spont		<i>lt^{hd1}</i>		
	P				P insert

allele	origin	discoverer	synonym	ref ^α	comments
<i>It</i> ²¹	P		<i>It</i> ^{hd2}		P insert
* <i>It</i> ^{56c}	spont	Meyer		3, 7	with <i>Alu</i> ^{56c} , strong allele
<i>It</i> ^{m100}	X ray	Spieler, 60a25		1	homozygous lethal
* <i>It</i> ^{pk}	spont	Lancefield, 18c18	<i>pinkoid</i> <i>pink wing</i>	2, 3, 6	intermediate allele, reduced viability

^α 1 = Baker and Rein, 1962, Genetics 47: 1399-1407; 2 = Bridges, 1931, Eos 7: 229-48; 3 = CP 627; 4 = de Zulueta, 1931, Eos 7: 249-53; 5 = Hilliker, 1976, Genetics 83: 765-82; 6 = Lancefield, 1918, Bio. Bull. 35: 207-10; 7 = Meyer, 1956, DIS 30: 77; 8 = Meyer and Edmondson, 1951, DIS 25: 73; 9 = Meyer, Edmondson, Byers, and Erickson, 1950, DIS 24: 60.

cytology: Located in 40F; proximal to the secondary construction on 2L (Hilliker and Holm, 1975, Genetics 81: 705-21). Placed in h35 of 2L heterochromatin (Pimpinelli).

molecular biology: Region cloned by transposon tagging; lesions of some mutations characterized (Wakimoto).

***It*^m: light mottled**

A series of X-ray induced light-variegated chromosome rearrangements (Hessler, 1958, Genetics 43: 395-403). Some have a mixture of light and wild-type ommatidia and are classified as pale; others have a mixture of wild-type and occasional darker ommatidia and are classified as dark.

allele ^α	phenotype	cytology
<i>It</i> ^{m1}	pale	<i>T(2,3)40B-F;63E-F</i>
<i>It</i> ^{m2}	dark	<i>ln(2L)22F-23A;40B-F</i>
<i>It</i> ^{m3}	dark	<i>ln(2LR)40B-F;60D</i>
<i>It</i> ^{m4}	dark	<i>T(2,3)40B-F;67E</i>
<i>It</i> ^{m5}	pale	<i>T(2,3)40B-F;98C</i>
<i>It</i> ^{m6}	pale	<i>T(2,3)26E-F;40B-F;96E</i>
<i>It</i> ^{m7}	pale	<i>T(2,3)40B-F;100F</i>
<i>It</i> ^{m8}	dark	<i>T(2,3)40B-F;92B</i>
<i>It</i> ^{m9}	dark	<i>ln(2LR)40B-F;56E</i>
<i>It</i> ^{m10}	dark	<i>T(2,3)40B-F;64E</i>
<i>It</i> ^{m11}	dark	<i>T(2,3)40B-F;96F</i>
<i>It</i> ^{m12}	dark	<i>ln(2LR)40B-F;60D</i>
<i>It</i> ^{m13}	dark	<i>T(2,3)40B-F;64F</i>
<i>It</i> ^{m14}	dark	<i>T(2,3)40B-F;95F</i>
<i>It</i> ^{m15}	pale	<i>T(2,3)40B-F;95F</i>
<i>It</i> ^{m16}	pale	<i>T(1,2)11A;12F;22D;40B-F</i>
<i>It</i> ^{m17}	pale	<i>T(2,3)40B-F;95C-D</i>
<i>It</i> ^{m18}	dark	<i>T(2,3)40B-F;98A</i>
<i>It</i> ^{m19}	dark	<i>T(2,3)40B-F;94B</i>
<i>It</i> ^{m20}	pale	<i>ln(2L)32C;40B-F</i>
<i>It</i> ^{m21}	dark	<i>T(2,3)40B-F;93D</i>
<i>It</i> ^{m22}	dark	<i>ln(2LR)40B-F;59D</i>
<i>It</i> ^{m23}	pale	<i>T(2,3)40B-F;62F</i>
<i>It</i> ^{m24}	pale	<i>T(2,3)40B-F;59F;75C</i>
<i>It</i> ^{m25}	pale	<i>ln(2LR)40B-F;57C-D</i>
<i>It</i> ^{m26}	pale	<i>ln(2L)27C;40B-F</i>
<i>It</i> ^{m27}	pale	<i>T(2,3)40B-F;88E-F</i>
<i>It</i> ^{m28}	pale	<i>T(2,3)40B-F;97E</i>
<i>It</i> ^{m29}	pale	<i>T(2,3)40B-F;99F</i>
<i>It</i> ^{m30}	dark	<i>T(2,3)40B-F;99C</i>
<i>It</i> ^{m31}	pale	<i>T(1,2)8F;28D;40B-F</i>
<i>It</i> ^{m32}	pale	<i>T(2,3)40B-F;97A</i>
<i>It</i> ^{m33}	pale	<i>ln(2LR)40B-F;58E</i>
<i>It</i> ^{m34}	pale	<i>T(2,3)40B-F;61B</i>
<i>It</i> ^{m35}	pale	<i>T(2,3)40B-F;64C</i>
<i>It</i> ^{m100β}		<i>T(2,3)40B-F;97F</i>

^α ^β All alleles except *It*^{m29} and *It*^{m100} lost (CP627). Also see data on previous *It* table.

***It*^v: light variegated**

origin: X ray induced as part of the construction of *SM5*.
references: Mislove and Lewis, 1955, DIS 29: 75.

***It*^d: lightoid**

location: 2-{59}.

origin: Spontaneous.

discoverer: Nichols-Skoog, 36d6.

phenotype: Eye color clear, light, translucent yellowish pink. Resembles *It* but is lighter, darkens with age. Ocelli colorless; larval Malpighian tubes colorless. Deficient in ommochrome synthesis. Lack detectable levels of 3OH kynurenine despite normal levels of kynurenine hydroxylase activity; administration of 3 hydroxykynurenine without effect (Phillips, Simmons, and Dowman, 1970, Biochem. Genet. 4: 481-87; Sullivan, Kitos, and Sullivan, 1973, Genetics 75: 651-61). Exhibits reduced levels of phenoazinone synthetase, an enzyme involved in the condensation of 3OH kynurenine molecules to xanthommatin, as do other mutants deficient in ommochrome synthesis (Phillips, Forrest, and Kulkarni, 1973, Genetics 73: 45-56). Uptake of kynurenine, which is produced in the fat body, by the Malpighian tubes, where it is converted to 3-hydroxykynurenine, is defective; similar defect seen in eye discs (Sullivan and Sullivan, 1975, Biochem. Genet. 13: 603-13). Sullivan and Sullivan postulate that *It*^d is a transport mutant that prevents the substrate kynurenine from reaching the sites of its conversion to ommochrome. Phenotypic interaction with other eye-color mutants examined by Rudy and Carvalier (1971, J. Hered. 62: 131-34) and by Silva and Ménsua, 1985, DIS 61: 156); produces nearly white eyes when combined with either *rb* or *g*.

cytology: Placed in 44E-46E based on inclusion in *Dp(2,3)eve*^{1.18} = *Dp(2,3)44B;46D-E*; *chrom3* (Hooper) but not in *Dp(2,3)P32* = *Dp(2,3)41A;42D-E;44C-D;-89D7-E1* (Lewis) or *Df(2R)44CE* = *Df(2R)44C;44E1-4* (Hooper).

***It*^{37b}**

origin: Spontaneous.

discoverer: Poulson, 37b.

references: Poulson and King. 1948, DIS 22: 55.

phenotype: Eye color of newly hatched adult bright red like *v*, darkens to a color like *pr* in old flies. Ocelli colorless; larval Malpighian tubes colorless. Viability excellent. RK1.

***luc*: luckenhaft (T. Schüpbach and E. Wieschaus)**

location: 2-65.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

phenotype: Maternal-effect female sterile: Embryos from homozygous females form cellular blastoderms with local defects; embryos form cuticle with big holes, variable head defects and segment fusions.

alleles: *luc*^{OG} = *luc*^I, *luc*^{QA}, *luc*^{RN}

***Lvp*: Larval visceral protein**

Three genes that are clustered in an 8 kb segment of DNA and lie 11 kb from the second-chromosome *Lcp* gene cluster (Snyder and Davidson, 1983, J. Mol. Biol. 166: 101-18). The order of the genes is *LvpH*--1.7kb--*LvpD*--1.1kb--*LvpL*, with *LvpH* being closest to the *Lcp* cluster. *LvpH* and *LvpL* are transcribed off of the same

strand as *Lcp1* and *Lcp2*, and *LcpD* off of the same strand as *Lcp3* and *Lcp4*. Three genes are completely sequenced and are 50-60% homologous in DNA sequence; they encode polypeptides of approximately 500 amino acids, which contain apparent signal peptide sequences but lack hydrophobic regions indicating that they are secreted rather than being membrane proteins; each has a sequence of 12 amino acids similar to known calcium binding domains of other proteins. The genes are coordinately transcribed in abundance in young larvae and adults, but not in late larvae when *Lcp* genes are transcribed; whereas *Lcp* genes are transcribed in the larval cuticle, *Lvp* transcripts are found in the larval viscera. The time and place of *Lvp* transcription suggests a possible digestive function of the gene products.

locus	genetic location	cytological location	comments
<i>LvpD</i>	2-59.4	44D	353 bp intron at 1322-1323
<i>LvpH</i>	2-59.4	44D	
<i>LvpL</i>	2-59.4	44D	62 bp intron at 145-146

lxd: low xanthine dehydrogenase

location: 3-34.5.

references: Keller and Glassman, 1964, *Genetics* 49: 663-68.

1964, *DIS* 39: 61.

Schott, Baldwin, and Finnerty, 1986, *Biochem. Genet.* 24: 509-27.

phenotype: Homozygotes exhibit little or no activity of four molybdenum hydroxylases, *i.e.*, 25% normal levels of xanthine dehydrogenase (XDH) activity (Keller and Glassman), 12% normal aldehyde oxidase (AO) activity (Courtright, 1967, *Genetics* 57: 25-39), no pyridoxal oxidase (PO) (Keller and Glassman) and 5-10% normal levels of sulfite oxidase (SO) (Bogaart and Bernini, 1981, *Biochem. Genet.* 19: 929-46) (2% according to Schott, Baldwin and Finnerty). Residual activity of AO and XDH thermolabile (Schott, Baldwin and Finnerty). These reduced activities not accompanied by comparable reductions in the levels of crossreacting material (CRM) indicating that the structural genes for the enzymes are still being expressed (Glassman, Shinoda, Duke, and Collins, 1968, *Ann. N.Y. Acad. Sci.* 151: 263-73; Browder, Wilkes, and Tucker, 1982, *Biochem. Genet.* 20: 111-24 and 125-32; Warner, Watts, and Finnerty, 1980, *Mol. Gen. Genet.* 180: 449-53). *lxd*⁺ postulated to be involved in production of an enzyme cofactor (Glassman *et al.*, 1968); dietary molybdenum shown to increase specific activity of AO and XDH in flies homozygous for some, but not all, *lxd* alleles (Duke, Rushing, and Glassman, 1975, *Biochem. Genet.* 13: 53-64). *lxd* homozygotes shown to have only 10% normal molybdenum cofactor activity (Schott, Baldwin, and Finnerty). Tungsten, a known antagonist of molybdenum cofactor, shown to mimic *lxd* in wild type (Warner and Finnerty, 1981, *Mol. Gen. Genet.* 184: 92-96) and to exacerbate the effects of *lxd* (Bentley, Williamson, and Oliver, 1981, *Can. J. Genet. Cytol.* 23: 597-609). The maternal effects of *cin* and *mal* are suppressed in *cin lxd* and *mal lxd* offspring of *cin*⁺ and *mal*⁺ mothers respectively; dietary molybdenum counteracts the suppression; *lxd* also suppresses complementation between various pairs of partially complementing *mal* alleles (Courtright, 1975,

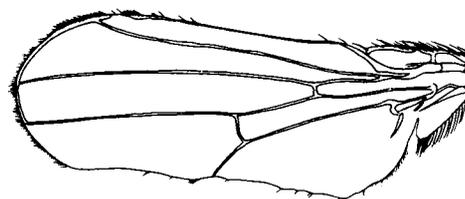
Mol. Gen. Genet. 142: 231-38). Dietary tungsten produces brown eye color in *lxd* flies, which normally have red eyes; used to select new *lxd* alleles (Bentley, Williamson, and Oliver); allopurinol used in the same way (Schott, Baldwin, and Finnerty).

alleles: All alleles non complementing.

allele	origin	ref ^α
<i>lxd</i> ¹	spont	3
<i>lxd</i> ^{2a}	EMS	1
<i>lxd</i> ^{3a}	EMS	1
<i>lxd</i> ^{3b}	X ray	4
<i>lxd</i> ⁴	X ray	4
<i>lxd</i> ⁵	EMS	4
<i>lxd</i> ⁷	X ray	4
<i>lxd</i> ¹⁰	EMS	4
* <i>lxd</i> ¹¹	EMS	4
<i>lxd</i> ¹²	EMS	4
<i>lxd</i> ¹³	EMS	4
* <i>lxd</i> ¹⁴	EMS	4
<i>lxd</i> ¹⁶	EMS	4
<i>lxd</i> ^c	spont	2
<i>lxd</i> ^{cb}	spont	4
<i>lxd</i> ^{ck}	X ray	4
<i>lxd</i> ^d	spont	2

^α 1 = Bentley, Williamson, and Oliver, 1981, *Can. J. Genet. Cytol.* 23: 597-609; 2 = Dukel, Rushing, and Glassman, 1975, *Biochem. Genet.* 13: 53-64; 3 = Keller and Glassman, 1964, *Genetics* 49: 663-68; 4 = Schott, Baldwin, and Finnerty, 1986, *Biochem. Genet.* 24: 509-27.

cytology: Localized to 68A4-9 based on its inclusion in *Df(3L)lxd9 = Df(3L)68A3-4;68B4-C1* but not *Df(3L)vin4 = Df(3L)68A8-9;78F3-6* (Schott, Baldwin, and Finnerty).



Ly: Lyra

From Bridges and Brehme, 1944, *Carnegie Inst. Washington Publ. No. 552*: 118.

Ly: Lyra

location: 3-40.5.

origin: X ray induced.

discoverer: Dubinin, 1929.

references: Coyne, 1935, *DIS* 4: 59.

Morgan, Bridges, and Schultz, 1937, *Year Book - Carnegie Inst. Washington* 36: 301.

phenotype: Lateral margins of wings excised, giving narrowed shape; angle between veins L2 and L5 reduced. Bristles shortened and stubby; postscutellars frequently missing. Eyes somewhat deformed with tufted vibrissae. Abdomen dark and narrow with rear edge of tergites raised. Homozygous lethal. *Ly/Df(3L)M69E* is lethal. Modification of wings first visible as marginal scalloping of prepupal wing buds; wing fold narrower (Waddington, 1939, *Proc. Nat. Acad. Sci. USA* 25: 304; 1940, *J. Genet.* 41: 75-139). RK1A.

alleles: *Ly*² like *Ly*¹ but with rougher eyes; spontaneous (Waddle, 1977, DIS 52: 3). Associated with same deficiency as *Ly*¹ (Zhimulev and Feldman, 1982, DIS 58: 152).

cytology: Placed in 70A3-5 on the basis of its association with *Df(3L)Ly = Df(3L)70A2-3;70A5-6* (Bridges).

lys: lysine

location: 2-22.9.

origin: Spontaneous.

discoverer: E. H. Grell, 1957.

references: 1960, DIS 34: 50. 1961, Genetics 46: 925-33.

phenotype: Larvae, pupae, and adults contain a higher concentration of lysine than wild type. Accumulation of lysine is postulated to result from block in its degradation. Flies homozygous for *lys* occasionally have faintly reddish fat cells, especially in thorax. This effect enhanced by starvation, by combining *lys* with *rc*, *rc*², or *cho*. RK3.

lz: lozenge

location: 1-27.7.

references: Gottschowski, 1936, Zool. Anz. Suppl. 9: 586-91.

Anderson, 1945, Genetics 30: 280-96.

Oliver, 1947, Univ. Texas Publ. 4720: 167-84.

Clayton, 1952, Univ. Texas Publ. 5204: 227-51.

Clayton, 1954, Univ. Texas Publ. 5422: 189-209.

Anders, 1955, Z. Indukt. Abstamm. Vererbungslehre 87: 113-86 (fig.).

Chovnick and Lefkowitz, 1956, Genetics 41: 79-92 (fig.).

Chovnick, Lefkowitz, and Fox, 1956, Genetics 41: 589-604.

Clayton, 1957, Genetics 42: 28-41 (fig.).

Clayton, 1958, Genetics 43: 261-73 (fig.).

Clayton, 1959, Genetics 44: 1041-52 (fig.).

phenotype: Many alleles with a wide range of phenotypes. Homozygous males have eyes size variably reduced and often ovoid in shape. Surface with fused facets producing a roughened glistening appearance (= glossy), or smooth with pigment either uniformly distributed or concentrated at periphery of the eye (= spectacle). Eye pigment variably reduced, and Malpighian tubes slightly lighter than normal (Brehme and Demerec, 1942, Growth 6: 351-56). Tarsal claws reduced to different extents by different alleles. Spermathecae and parovaria (= accessory glands) often missing in homozygotes with abnormal parovaria seen in some heterozygous females (Anderson, 1945). Females often sterile, but sterility appears to be primarily an ovarian defect, since some genotypes which lack parovaria and spermathecae are female fertile. Some alleles lack the class of hemocytes called crystal cells, or at least lack the crystalline inclusions of those cells; the inclusions can be shown to comprise prophenoloxidase, and flies lacking crystal cells are deficient in phenol oxidase activity and suppress the phenotype of *Bc*. Five of fifteen alleles tested (*lz*^{36f17}, *lz*⁴⁶, *lz*^D, *lz*^{rf8}, and *lz*^s) suppress *Bc* and lack crystal cells and phenol oxidase activity (Rizki and Rizki, 1981, Genetics 97: s90); postulated that *lz*⁺ crucial to differentiation of crystal cells, and is not a structural gene for any of the five phenol oxidase moieties. Peeples, Geisler, Whitcraft, and Oliver report defective phenol oxidase

activity in *lz*⁸ (1969, Biochem. Genet. 3: 563-69) *lz*^{64j}, *lz*^{66c}, *lz*^s, and *lz*^{y4}, but not in *lz*^{50e}, or *lz*^K (1969, Genetics 62: 161-70); Warner, Grell, and Jacobson (1974, Biochem. Genet. 11: 359-65) found no phenol oxidase activity in *lz*^{rf8}, but normal levels in *lz*¹ and *lz*⁸.

alleles:

allele	origin	discoverer	ref ^α	comments ^β
<i>lz</i> ¹		Bridges, 16b12	8, 14, 26, 27, 36	g/tsp ^γ
<i>lz</i> ²	gypsy	Bridges, 19g16	7	
<i>lz</i> ³	spont	Bridges, 22b14	8, 14, 27, 35	g/tspf ^γ
<i>lz</i> ³ⁿ	spont	Green	8, 14	s/tsp
<i>lz</i> ⁴	spont	Bridges, 23b6	7	
<i>lz</i> ⁵	spont	Bridges, 23k15	7	
<i>lz</i> ^{16l}	spont	Bridges	7	
<i>lz</i> ^{23b}	spont	Morgan	7	
<i>lz</i> ^{23c}	spont	Wallace	7	
<i>lz</i> ^{29a}	spont	Bridges	7	
<i>lz</i> ^{33e}		Ives, 33e18	7	
<i>lz</i> ³⁴	spont	Beadle, 34k22	2, 8, 14, 35	g/tspf ^γ
<i>lz</i> ³⁵	spont	Gottschewski, 1935	8, 12	?/f ^γ
<i>lz</i> ³⁶	spont	Spencer, 36c	8, 14, 35	s/spf
<i>lz</i> ^{36cD}		Dempster, 36c	8	g/F
<i>lz</i> ^{36f17}			39	
<i>lz</i> ³⁷	spont	Curry, 37h12	8, 14, 35, 44	g/tsp ^γ
<i>lz</i> ^{41e}	spont	Neel, 41e17	7	
<i>lz</i> ^{41h}	spont	Neel, 41h22	7	
<i>lz</i> ⁴⁶	X ray	Green	8, 13, 35	g/spf
<i>lz</i> ^{48c}	X ray		8, 14	s/tspf
<i>lz</i> ^{48f}	mustard	Lindsley, 48f	8, 14	s/tspf ^γ
<i>lz</i> ^{48l}	X ray		8, 14	s/tspf
<i>lz</i> ^{49g}	spont		8, 14	s/tspf
<i>lz</i> ^{49h}	X ray	W.K. Baker, 49h	8, 14	s/TSPF ^γ
<i>lz</i> ^{49KB}			35	
<i>lz</i> ⁵⁰			33	
<i>lz</i> ^{50d}	X ray	Ritterhoff, 50d	8, 10	s/tspf
<i>lz</i> ^{50e}	³² P	King	8, 14, 16, 36	s/TSPF ^γ
<i>lz</i> ^{50k}			35	
<i>lz</i> ^{50l}		Green, 50l30	22	
<i>lz</i> ^{51d}	spont	Mossige, 51d10	8, 28	s/f
<i>lz</i> ^{52c}	neutrons	King, 52c28	8, 19	s/t ^γ
<i>lz</i> ^{55d}	X ray	Clark, 55d	6, 8	s/f
<i>lz</i> ^{55l}	spont	Masterson, 55l	5, 8	s/tspf
<i>lz</i> ^{57j}	X ray	Mayo, 57j	8, 23	s/tspf
<i>lz</i> ^{58d}	spont	Schreckengost, 58d	5, 8	s/tspf
<i>lz</i> ⁵⁹	X ray	Polivanov, 1959	8, 37	s/t ^γ
<i>lz</i> ^{60f}			35	
<i>lz</i> ^{61f}	spont	Moynihan, 61f	4, 8, 20, 40	s/f ^γ
<i>lz</i> ^{62k}	X ray	Mickey, 62k11	8, 25	s/tspf
<i>lz</i> ⁶³	X ray	Halfer, 1963		s/F ^γ
<i>lz</i> ^{63f}	spont	Burdick, 63f17	41	s/tspf± ^γ
<i>lz</i> ⁶³ⁱ	spont	Polivanov, 63i	35, 38	
<i>lz</i> ^{64j}			35, 36	
<i>lz</i> ^{66c}	X ray	McCombs	35, 36	
<i>lz</i> ^{71a}	EMS	Snyder	42, 43	s/tspf ^γ
<i>lz</i> ^{71b}	EMS	Snyder	42	g/Tpf enhanced by <i>su(f)</i>
<i>lz</i> ^{75v}		Golubovsky	11	
<i>lz</i> ¹⁴⁴			16	<i>Tp(1;1)8E;NO</i>
<i>lz</i> ⁴⁹¹			16	<i>Tp(1;1)8E;NO</i>
<i>lz</i> ²⁶⁸⁻²⁹	X ray	Hoover		<i>T(1;3)8D8-9;81F</i>
<i>lz</i> ⁵²⁶			35	
<i>lz</i> ^A			33	unstable allele
<i>lz</i> ^{BS}	X ray		8, 14, 32	g/tspf
<i>lz</i> ^{c1}	Cu(SO) ₄	Hadorn, 45b27	1, 8, 15	s/tspf ^γ
<i>lz</i> ^{cs}	EMS	Wright	46	cold sensitive lethal
<i>lz</i> ^D	spont	Novitski, 47i	8, 20, 30, 35	s/tspf ^γ
<i>lz</i> ^{EF3}			35	
<i>lz</i> ^f	spont	Muller	8, 29	F±
<i>lz</i> ^g	X ray	Oliver, 31a7	8, 14, 32, 35, 36, 45	g/tspf± ^γ
<i>lz</i> ^{g1}	X ray	M. Bender, 53k	3, 8	g/ ^γ
<i>lz</i> ^{gM}	spont		8, 14	g/tspf

allele	origin	discoverer	ref ^α	comments ^β
<i>lz^K</i>	spont	Krivshenko, 55k9	8, 21, 35, 36	g/TSPF ^γ
<i>*lz^{Ki}</i>	spont	Kiil, 45k14	8, 18	F
<i>*lz^{M58}</i>	X ray	Meyer, 58k	8, 24	s/tspf± ^γ
<i>lz^{ntg}</i>	NNG	Kaufman	17	
<i>lz^{rfg}</i>		R.F. Grell	45	
<i>lz^s</i>	X ray	Patterson, 1928	8, 14, 34, 35, 36	s/tspf ^γ
<i>*lz^{sB}</i>	X ray		8, 16, 32, 35	s/spf
				<i>ln(1)8E;20F</i>
<i>lz^{s-ntg}</i>	NNG	Kaufman	17	
<i>lz^{sl}</i>			11	
<i>lz^{tear}</i>	spont		44	s/F
<i>lz^{tsl}</i>			9	
<i>lz^{y4}</i>	X ray		8, 14, 32, 35, 36	s/spf ^γ

^α 1 = Anders, 1955, Z. Indukt. Abstamm. Vererbungsl. 87: 113-86 (fig.); 2 = Beadle, 1935, DIS 4: 9; 3 = Bender, 1955, DIS 29: 69; 4 = Burdick 1963, DIS 37: 47; 5 = Clancy, 1960, DIS 34: 48; 6 = Clark, 1956, DIS 30: 71; 7 = CP552; 8 = CP627; 9 = Cross, 1977, Genetics 86: s13; 10 = Glass, 1951, DIS 25: 77; 11 = Golubovsky, 1978, DIS 53: 122; 12 = Gottschewski, 1937, DIS 8: 12; 13 = Green and Green, 1949, Proc. Nat. Acad. Sci. USA 35: 586-91; 14 = Green and Green, 1956, Z. Indukt. Abstamm. Vererbungsl. 87: 708-21; 15 = Hadorn and Anders, 1946, DIS 20: 65; 16 = Hannah-Alava, 1971, Mol. Gen. Genet. 113: 191-203; 17 = Kaufman, 1970, DIS 45: 34; 18 = Kiil, 1946, DIS 20: 66; 19 = King, 1951, DIS 26: 65; 20 = Klingele, 1968, DIS 43: 145; 21 = Krivshenko, 1956, DIS 30: 74; 22 = Lefevre and Moore, 1968, Genetics 58: 557-71; 23 = Mayo, 1958, DIS 32: 82; 24 = Meyer, 1959, DIS 33: 97; 25 = Mickey, 1963, DIS 38: 28; 26 = Modolell, Bender, and Meselson, 1983, Proc. Nat. Acad. Sci. USA 80: 1678-82; 27 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 230; 28 = Mossige, 1951, DIS 25: 69; 29 = Muller, 1946, DIS 20: 67; 30 = Novitski, 1949, DIS 23: 61; 31 = Oliver, 1935, DIS 4: 15; 32 = Oliver, 1947, Texas Univ. Publ. 4720: 167-84; 33 = Osipova, Korochkin, Golubovsky, Khelbodarova, and Kulutchkov, 1981, DIS 56: 104; 34 = Patterson and Muller, 1930, Genetics 15: 495-577; 35 = Peeples, Geisler, Whitcraft, and Oliver, 1969, Biochem. Genet. 3: 563-69; 36 = Peeples, Geisler, Whitcraft, and Oliver, 1969, Genetics 65: 161-70; 37 = Polivanov, 1963, DIS 38: 30-31; 38 = Polivanov, 1970, DIS 45: 37; 39 = Rizki and Rizki, 1981, Genetics 97: s90; 40 = Schwalm, Klingele, and Bender, 1970, DIS 45: 91; 41 = Seiger and Bender, 1963, DIS 38: 31; 42 = Snyder, 1973, DIS 50: 20; 43 = Snyder and Smith, 1976, Biochem. Genet. 14: 611-17; 44 = Thompson and Purnell, 1972, DIS 48: 16; 45 = Warner, Grell, and Jacobson, 1974, Biochem. Genet. 11: 359-65; 46 = Wright, 1973, Mol. Gen. Genet. 122: 101-18.

^β s/ = spectacle; g/ = glossy; sl/ = intermediate; t/ = tarsal structures reduced or absent; T/ = tarsal structures present; s = spermathecae absent; S = spermathecae present; p = parovaria absent; P = parovaria present; f = female sterile; F = female fertile.

^γ More detailed phenotype description below.

cytology: Placed in 8D8-9 (Lefevre, 1976, Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 31-66).

other information: The *lz* region has been subdivided into four recombinationally separable groups of alleles (Green and Green, 1949, Proc. Nat. Acad. Sci. USA. 35: 586-91; 1956, Z. Indukt. Abstamm. Vererbungsl. 87: 708-21); recombination is observed between but not within groups. Not all alleles have been mapped. The total genetic length of the region is 0.14 cM. Double mutants produced by recombination all have the extreme phenotype of *lz^s*. Mitotic exchange between *lz³⁶* and *lz^{y4}* reported by Tokunaga (1973, Mol. Gen. Genet. 125: 109-18).

lz¹

origin: Spontaneous; contains gypsy insert (Modolell, Bender, and Meselson, 1983, Proc. Nat. Acad. Sci. 80: 1678-82).

discoverer: Bridges, 16b12.

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 230.

Green and Green, 1956, Z. Induktive Abstammungs-Vererbungsl. 87: 708-21.

phenotype: Eye narrower than wild type and ovoid. Irregular facets in some areas cause rough patches; areas of fused facets appear as smooth patches. Eye color appears normal but, in combination with *st*, slight reduction in red pigment detectable. Tarsal claws reduced. Developmental study by Waddington and Pilkington (1942, DIS 16: 70) shows failure of middle cell layer of optic disk to penetrate between cells of outer layer; surface thus covered with primary pigment cells. Females sterile. Parovaria and spermathecae absent; some *lz/+* females have abnormal parovaria (Anderson, 1945, Genetics 30: 280-96). Suppressed by *su(f)⁶* (Schalet, 1970, Genen. Phaenen 14: 16-17), *su(Hw)²*, *e(w^e)^s*, and *su(pr)^{e3}*; enhanced by *su(s)³* and *su(w^a)* (Rutledge, Mortin, Schwarz, Thierry-Mieg, and Meselson, 1988, Genetics 119: 391-97). Phenol oxidase activity increased from 17% to 71% normal (Snyder and Smith, 1976, Biochem. Genet. 14: 611-17). RK1.

lz³

phenotype: Eye size sharply reduced; surface smooth. Optic disk of mature larva and prepupa two-thirds normal size (Chen, 1929, J. Morphol. 47: 135-99). Red pigment greatly reduced; color yellowish brown, cream colored in combination with *v*. Tarsal claws vestigial. Homozygous females lack parovaria and spermathecae and are sterile; *lz³/+* females lack parovaria and many have abnormal spermathecae [Anderson, 1945, Genetics 30: 280-96 (fig.)]. Unaffected by *su(f)⁶* (Schalet, Snyder, and Smith, 1976, Biochem. Genet. 14: 611-17). RK1.

lz³⁴

phenotype: Eye phenotype intermediate between *lz* and *lz³*. Surface of eye has large areas of fused facets with a few normal facets (Clayton, 1957, Genetics 42: 28-41); eye color dark red with small yellowish spots. Larval Malpighian tubes slightly lighter than normal; variable (Brehme and Demerec, 1942, Growth 6: 351-56). Tarsal claws reduced. Spermathecae and parovaria absent from homozygous females, which accumulate stage 14 oocyte and are quite infertile; some *lz³⁴/+* females have abnormal parovaria (Anderson, 1945, Genetics 30: 280-96). Eye effect, but not other aspects of phenotype, enhanced by *spa^{e(lz)}*; eye converted from a glossy to a spectacle phenotype (Beeson and Bender, 1975, J. Exp. Zool. 193: 177-90). In the presence of *su(lz³⁴)*, *lz³⁴* flies have virtually normal eyes. Beeson and Bender were unable to confirm previous observations of Bender and Green (1960, Genetics 45: 1563-66) that *su(lz³⁴)* increases the fecundity of *lz³⁴* females. Unaffected by *su(f)⁶* (Schalet) or *su(Hw)²*; however suppressed by *su(pr)* and enhanced by *su(s)* and *su(w^a)* (Rutledge, Mortin, Schwarz, Thierry-Mieg, and Meselson, 1988, Genetics 119: 391-97). RK1.

**lz³⁵*

phenotype: Eyes reduced and diamond shaped; color opaque brown. Homozygous females sterile. *lz³⁵/lz* females fertile. RK1.

lz³⁷

phenotype: Eye size reduced. Areas of irregular facets in posterior region of eye; eye color normal. Enhanced by *su(f)*⁶ (Schalet, 1970, Genen. Phaenen 14: 16-17); also enhanced by *su(Hw)*² and *su(s)* (Rutledge, Mortin, Schwarz, Thierry-Mieg, and Meselson, Genetics 119: 391-97). Phenol oxidase level decreased from 94% to 58% of normal (Snyder and Smith, 1976, Biochem. Genet. 14: 611-17). RK1.

lz^{48f}

origin: Induced by mustard gas.

discoverer: Lindsley, 48f.

references: Green and Green, 1956, Z. Indukt. Abstamm. Vererbungsl. 87: 708-21.

phenotype: Unaffected by *su(f)*⁶ (Schalet).

***lz^{49h}**

phenotype: Eye size sharply reduced; surface smooth; red pigment distributed over entire eye. Tarsal claws normal. Spermathecae and parovaria present and normal in females, which are fertile. Complements all *lz* alleles tested except *lz*^{50e}.

lz^{50e}

phenotype: Like *lz*^{49h}. Eyes reduced in size and almond shaped; no indication of facets; covered with indentations, giving a pock-marked appearance. Hairs on eye surface sparse or absent; eye surface glossy with many large black or brown flecks. Tarsal claws normal. Females fertile; spermathecae and parovaria present and normal. *lz*^{50e}/*lz* has normal eyes except for a few flecks. Complements most other *lz* alleles except *lz*^{49h}, *lz*^{52c}, and those associated with rearrangements or deficiencies. Unaffected by *su(f)*⁶ (Schalet). RK1.

***lz^{52c}**

origin: Recovered among progeny of male fed H₃BO₃ and exposed to thermal neutrons.

phenotype: Eyes mottled, yellowish brown, darker at rim; facets fused. Males semisterile with missing tarsal claws, although pulvilli and endopodia normal. Third antennal segment slightly reduced. *lz*^{52c}/*lz*^{50e} females resemble *lz*^{50e}. RK1.

***lz⁵⁹**

phenotype: Eyes reduced in size and ovoid; facets fused; surface slightly rough and almost or completely hairless; color light brown with darker, slightly reddish rim; almost colorless in combination with *v*. Tarsal claws practically absent as in *lz*^{cl}. Males sterile, (possibly associated with X-autosome translocation), transmit no motile sperm to females; therefore, homozygous females not observed. *lz*⁵⁹/*lz*³⁷ females intermediate between the two mutants in eye phenotype, have reduced tarsal claws, and are weakly fertile. RK2.

lz^{61f}

phenotype: Facets completely fused; eye color dark, but pigment unevenly distributed and concentrated at margin. Females found to be fertile by Burdicks, but were sterile when studied by Schwalm, Bender, and Klinge (1970, DIS 45: 91) who studied the ultrastructure of eggs produced by homozygous females. *lz*^{61f}/*lz* females more nearly normal than either mutant; facets disrupted and fused only in posterior third of eye; also fertile. RK1.

lz⁶³

phenotype: Eye shape oval; color brown, darkest at margin; surface smooth and glossy. Viability and fertility of both sexes good. RK1.

lz^{63f}

phenotype: Eye size moderately reduced; surface smooth; color brownish with darker margin. Tarsal claws and pulvilli strongly reduced. Spermathecae and parovaria absent; female reproductive capability strongly reduced. *lz*^{63f} complements *lz*^{50e} but not *lz*³⁴, *lz*^D, or *lz*^{61f} (Klingele). Spermathecal number of *lz*^{63f}/*lz*^K 0-3. RK1.

lz^{71a}

phenotype: Phenol oxidase activity severely reduced; further reduced in presence of *su(f)* (Snyder and Smith), 1976, Biochem. Genet. 14: 611-17.

***lz^{cl}: lozenge-clawless**

origin: Appeared as a male from an ovary treated *in vitro* with CuSO₄.

phenotype: Eyes narrow and small without facets; surface has rough spots; color amber, both pteridines and ommochromes affected, darker at rim. Tarsal claws absent. Third antennal segment reduced; sensilla on antennae abnormal. Phenotype similar in both sexes. Females infertile and lack spermathecae and parovaria. Autonomous in transplants. RK1.

lz^D: lozenge-Dominant

phenotype: Males and homozygous females resemble *lz*^S. Heterozygous females sometimes have roughened eyes. Apparent dominance shown by H. Bender to be caused by the presence of *spa*^{el(z)}; heterozygous expression additionally enhanced by presence of *In(2LR)bw*^{V1}.

lz^g: lozenge-glossy

phenotype: Eyes smaller than wild type; surface glossy from fused facets; a few normal facets also present; color dark blood red, bright red in combination with *st* or *v*. Larval Malpighian tubes slightly lighter than normal (Brehme and Demerec, 1942, Growth 6: 351-56). Tarsal claws reduced. Spermathecae and parovaria absent from homozygous females, which have reduced fertility; *lz*^g/*+* females tend to have abnormal parovaria [Anderson, 1945, Genetics 30: 280-96 (fig.)]. RK1.

other information: *lz*^g/*lz*^S provided probably the first recorded case of intra-allelic recombination (Oliver, 1940, Proc. Nat. Acad. Sci. USA 26: 452-54; 1940, DIS 13: 73).

***lz^{gl}: lozenge-glued**

phenotype: Eyes of male reduced and roughened like *Gl*; color dark; female eyes somewhat less extreme. *lz*^{gl}/*lz* intermediate between *lz*^{gl} and *lz* and sterile. Homozygous females fertile. RK1.

lz^K: lozenge of Krivshenko

synonym: *amx*⁵⁵: *almondex-55*; *lz*^k.

phenotype: Eyes narrow and moderately rough; facets irregular; eyes of homozygous females more nearly normal than those of males. Tarsal claws normal. Females fertile; spermathecae and parovaria present. Interactions of *lz*^K with other *lz* alleles described by Green [1961, Genetics 46: 1169-76 (fig.)]. Unaffected by *su(f)*⁶ or *su(Hw)*²; however suppressed by *su(pr)* and enhanced by *su(s)* and *su(w^a)* (Rutledge, Mortin, Schwarz, Thierry-

Mieg, and Meselson, 1988, *Genetics* 119: 391-97). RK1.

****lz*^{M58}: lozenge of Meyer**

phenotype: Eyes small and oval; surface glossy; color brownish. Tarsal claws missing. Homozygous females moderately fertile, although spermathecae absent; *lz*^{M58}/*lz*^s also fertile. RK1.

***lz*^s: lozenge-spectacled**

phenotype: Eye size reduced, narrower than normal; no true facets; whole eye has glossy surface; color yellow-brown with darker rim, creamy in combination with *v*. Tarsal claws vestigial. Homozygous females lack spermathecae and parovaria and are sterile. *lz*^s/+ females tend to have abnormal parovaria (Anderson, 1945, *Genetics* 30: 280-96). RK1.

other information: *lz*^s/*lz*^s provided probably the first recorded case of intra-allelic recombination (Oliver, 1940, *Proc. Nat. Acad. Sci. USA* 26: 452-54; 1940, *DIS* 13: 73).

***lz*^{Y4}: lozenge in yellow-4**

phenotype: Similar to *lz*^s but eye color redder. Homozy-

gous females lack spermathecae and parovaria and are sterile; *lz*^{Y4}/+ females have abnormal parovaria and tend to lack spermathecae and parovaria (Anderson, 1945, *Genetics* 30: 280-96). RK1.

lz-l: see *rstl*

****lzl*: lozengelike**

location: 1-11.

discoverer: Oliver, 29k24.

references: 1935, *DIS* 3: 28.

phenotype: Eyes rough. Both sexes fertile. RK3.

other information: Possibly an allele of *rg* (1-11.0).

Many spontaneous reversions of *ovo*^{DI} result in a phenotype similar to that originally reported for *lzl* (Busson, Gans, Komitopoulou and Masson, 1983, *Genetics* 105: 309-25; Oliver, Perrimon, and Mahowald, 1987, *Genes Dev.* 1: 913-23; Mével-Ninio, Mariol, and Gans, 1989, *EMBO J.* 8: 1549-58). The *lzl* alleles derived from the *ovo*^{DI} chromosome are characterized by rough or glazed eyes. All alleles are semidominant and cold sensitive. These mutations are likely to be new alleles of *lzl*. This cannot be tested as the original *lzl* allele was lost.



m: miniature

From Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. No. 237.

m: miniature

location: 1-36.1

phenotype: Wing size reduced, only slightly longer than abdomen and with normal proportions. Angle between L2 and L5 reduced. Wings dark gray and less transparent than normal. Wing cells smaller than normal (Dobzhansky, 1929, Arch. Entwicklungsmech. Organ. 115: 363-79). In poor cultures, wings may become divergent and stringy. Cell expansion inhibited in prepupae and pupae [Waddington, 1940, J. Genet. 41: 75-139 (fig.)]. Different *m* mutants complement slightly; *m/dy* is wild type.

RK1.

alleles:

allele	origin ^α	discoverer	ref ^β	comments
<i>m 1</i>	spont	Morgan, 10h	19	
<i>m 2</i>	X ray	Glass, 1929	9	<i>In(1)dl49</i>
<i>*m 17e</i>	spont	Bridges	4	
<i>*m 20f</i>	spont	Morgan	4	
<i>*m 20k</i>	spont	Bridges	4	
<i>*m 21e</i>	spont	Bridges	4	
<i>*m 22e</i>	spont	Bridges	4	
<i>*m 23e</i>	spont	Bridges	4	
<i>*m 23g</i>	spont	Plunkett	4	
<i>*m 25d</i>	spont	Morgan	4	
<i>*m 25g</i>	spont	Fogg	4	
<i>*m 27e</i>	spont	Bridges	4	
<i>*m 38d</i>	spont	Bridges	4	
<i>*m 20a</i>	spont	Mohr	4, 20	
<i>m 31a</i>	X ray	Oliver, 31a	4, 22	
<i>m 33c</i>	X ray	Oliver, 33c	4, 22	
<i>m 33d</i>	X ray	Oliver, 33d	4, 22	
<i>*m 34a</i>	spont	Tanaka	4	
<i>m 36f</i>	spont	Spencer 36f30	4, 25	
<i>m 37d</i>	spont	Nordensköld, 37d14	4, 21	
<i>m 37i</i>	spont	Braun	4	
<i>m 57</i>	X ray	Mayo, 57i	18	
<i>m 59</i>	spont	Karwinkel, 59a	3	
<i>m 60</i>	γ ray	Ives, 60l	12	
<i>m 61</i>	γ ray	Ives, 61e	13	
<i>m 66d</i>	X ray		26, 27	<i>T(1;Y;3)?</i> wings at 45° angle with body; post scutellars erect, male sterile female sterile; shows partial dominance
<i>m 68</i>	NNG	Kaufman	14	

allele	origin ^α	discoverer	ref ^β	comments
<i>m 71c</i>		Gelbart	8	
<i>m 73a</i>	spont		1	
<i>m 74f</i>	EMS		6	long-winged allele
<i>m 81</i>		Behnel	2	in <i>Basc</i> ; female sterile
<i>m 259-4</i>	X ray	Demerec, 33i	5	<i>Df(1)10C2-3;10E2-3</i>
<i>m D</i>	X ray	Slatis, 48k17	5, 23, 24	partial dominant ^β
<i>m how</i>	spont	Waddle	28	held out wings
<i>m I</i>	spont	Gassparian	7	
<i>m 127</i>	spont	Green	10	<i>m^μ</i> derivative ^β
<i>m K</i>	X ray	Krivshenko, 5513	5, 11, 17, 29	<i>In(1)10E;20B</i> ; v-type position effect
<i>m^μ</i>		Green	10	mutable allele ^β
<i>m PS</i>	γ ray		15, 16	homozygous females poorly fertile
<i>m s19</i>	spont	Green	10	<i>m^μ</i> derivative ^β

^α 1 = Anxolabèhère and Périquet, 1973, DIS 50: 21; 2 = Behnel, 1982, DIS 58: 183-84; 3 = Burdick, 1961, DIS 35: 45; 4 = CP552; 5 = CP627; 6 = Craymer, 1980, DIS 197; 7 = Gassparian, 1973, DIS 50: 22-23; 8 = Gelbart, 1974, Genetics 76: 51-63; 9 = Glass, 1935, DIS 4: 9; 10 = Green, 1975, Mutat. Res. 29: 77-84; 11 = Hartmann-Goldstein and Wargent, 1975, Chromosome, 52: 349-62; 12 = Ives, 1961, DIS 35: 46; 13 = Ives, 1962, DIS 36: 38; 14 = Kaufman, 1969, DIS 44: 44; 15 = Keller and Nash, 1960, DIS 34: 51; 16 = Keller and Nash, 1960, DIS 34: 47; 17 = Krivshenko, 1956, DIS 30: 75; 18 = Mayo, 1958, DIS 32: 82; 19 = Morgan and Bridges, 1916, Carnegie Inst. Wash. Publ. No. 237; 20 = Mohr, 1923, Z. Indukt. Abstamm. Vererbungslehre 32: 215; 21 = Nordensköld, 1939, DIS 12: 49; 22 = Oliver, 1937, DIS 7: 19; 23 = Slatis, 1949, DIS 23: 63; 24 = Slatis and Willmeret, 1954, Genetics 39: 45-58; 25 = Spencer, 1937, DIS 7: 14; 26 = Thompson and Braver, 1969, DIS 44: 43; 27 = Thompson and Braver, 1971, DIS 46: 40; 28 = Waddle, 1977, DIS 52: 3; 29 = Wargent, and Hartmann-Goldstein, 1974, Heredity 33: 317-26.

^β More complete description below.

cytology: Placed in 10E1-2 by Lefevre [Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 31-66].

other information: The miniature-dusky region has been subdivided into four recombinationally separable sites (Dorn and Burdick, 1962, Genetics 47: 503-18).

***m^D*: miniature-Dominant**

phenotype: Wings of homozygote smaller than *m/m*. *m^D/+* wings intermediate between homozygote and wild type. Viability 20-50% normal in males and 5% in homozygous females; most die in embryo. Fertility low in homozygous females. Wing size of *m^D/m* and *m^D/dy* intermediate between *m^D/+* and *m^D/m^D*. RK2.

***m^I*: miniature-intermediate**

origin: Stable spontaneous derivative produced repeatedly by *m^μ*.

phenotype: Wing length intermediate between that of *m^μ* and wild type. Resembles *dy⁷³* but complements *dy⁷³* and is mutant in combination with *m^μ*. Phenotype more extreme in combination with *Df(1)m-fw*.

***m^K*: miniature of Krivshenko**

phenotype: Variegated-type position effect; enhanced by *In(2LR)Rev^B = In(2LR)40;52C-E*; *In(1)m^K = In(1)10E;20B*; in turn enhances *Rev^B* (Wargent and Hartmann-Goldstein, 1974, Heredity 33: 317-26).

***m*^μ**

origin: A spontaneous derivative of *dy*⁷³, which was accompanied by reversion of *dy*⁷³.

phenotype: A strong allele that mutates at an inordinately high rate, both germinally and somatically, to two different states, *m*ⁱ and *m*^s.

***m*^s: miniature-subliminal**

origin: Stable spontaneous derivative produced repeatedly by *m*^μ.

phenotype: Hemizygotes, homozygotes, and heterozygotes with *dy*⁷³ are wild type. In heterozygous combination with either *m*^μ or *Df(1)m-fw* displays a *dy*-like phenotype.

M: Minute

Minutes are a class of genes, which, when the wild-type allele is present in hemizygous condition (not including X-linked Minutes in males), produce a characteristic phenotype consisting of short slender bristles and delayed development. That is, heterozygotes for deficiencies or loss of function alleles of these loci exhibit the Minute phenotype. Many of the early Minutes were mapped crudely, and those with similar map positions were seldom tested for complementation. As most of these Minutes have long since been lost, experimental establishment of allelism is not possible. Furthermore the results of deficiency analysis are also ambiguous; a Minute phenotype of a deficiency heterozygote indicates the presence of "at least one," not "only one" *M* gene. Given these uncertainties, tentative designations of allelism have been made, with the caveat that complementation tests have not or cannot be done. Furthermore inviability of *trans* heterozygotes does not invariably indicate allelism (Moscoso del Prado and Ripoll, 1983, Genet. Res. 42: 59-63). The existence of a Minute locus more frequently inferred from the phenotype of a deficiency heterozygote than a point mutant. Trans-acting suppressors turn out to be duplications of the locus on the *M*⁺ homologue (Schultz, Baker, 1972, DIS 49: 59; Broderick and Roberts, 1982, Genetics 102: 71-74). Phenotype of flies heterozygous for two different Minutes no more extreme than that of the more extreme mutant of the combination (Schultz, 1929, Genetics 14: 366-419); however, see *M(3)65F* and *M(3)69E*. Homozygotes and hemizygotes for mutant alleles are late embryonic or early larval lethals (reviewed by Wright, 1970, Adv. Genet. 15: 262-85). *M/+* triploids are normal in phenotype, whereas *M/M+* triploids are lethal (Schultz). Heterozygotes often display secondary effects such as small body size, large and somewhat rough eyes, missing arista, thin-textured wings with tendency to plexus venation, missing bristles (usually postverticals), and low fertility, especially in females. The developmental delay exhibited by *M/+* individuals is attributable to increased cell cycle times compared to that of wild type; this feature is autonomous such that *+/+* cells produced by mitotic exchange in an *M/+* fly proliferate more rapidly than the surrounding cells, leading to increased size of *M*⁺ clones, and facilitating ascertainment of mitotic recombination events (Morata and Ripoll, 1975, Dev. Biol. 42: 211-21); this observation was interpreted by Stern (1936, Genetics 21: 625-730) as increased incidence of somatic crossing over in the *M*-bearing chromosome arm of *M/+* flies. Overgrowth of *M*⁺ clones

used extensively in somatic genetic studies of development. Most Minutes enhance dominance of such venation characters as *px* and *net* or of such bristle characters as *sc*. Dominant lethal effects are frequent, in combination with *Dl*, *J*, and occasionally *D*. At least one Minute locus shown to encode a ribosomal protein (Kongsuwan, Yu, Vincent, Frisardi, Rosbash, Lengyel, and Merriam, 1985, Nature 317: 555-58), and presumably most if not all the others do as well. A consequent reduction in the rate of protein synthesis is postulated to lengthen the cell cycle and to limit the rate of bristle elaboration, which is postulated to require maximum rates of synthesis (Atwood).

Since many Minute loci have been named according to the lettered subdivision of the polytene map to which they have been localized, this revision attempts to standardize that mode of designation for all confirmed loci. Further restriction of designations to single lettered subdivisions should accompany revised localizations.

M^{zw}: see *M(G6PD)*

m-like: see *dy*^{31d}

M-pro: see *T(1;4)M-pro*

M(1)

The following table summarizes the Minute loci inferred to exist on the X chromosome on the basis of the phenotypes of females carrying mutations or deficiencies. The loci included are confirmed; others that have been claimed, but not confirmed are included among the entries that follow the table.

locus	genetic location	cytological location	included in	excluded from	synonym	ref ^α
M(1)1B	1-0.1	1B11-12	<i>Df(1)y74k24.1</i>	<i>Df(1)svr</i>	<i>M(1)Bld</i>	7
M(1)3E	1-[5]	3E3-4	<i>Df(1)N264-76</i>	<i>Df(1)N264-38</i>		1, 6
M(1)5A	1-[13]	5A6-13	<i>Y^PX^DW14</i>	<i>Y^PX^DB119</i>	<i>M(1)30?</i>	4, 6
M(1)5D6A	1-13.7	5D5-6A	<i>Df(1)5D:6A</i>	<i>Y^PX^DD10</i>	<i>M(1)30?</i>	3, 4, 6
M(1)7BC	1-[21]	7B-C	<i>Y^PX^DS27</i>	<i>Y^PX^DB123</i>		4
M(1)7C	1-[22]	7C-E3	<i>Y^PX^DW31</i>	<i>Y^PX^DB17</i>		4
M(1)8F	1-30.0	8F1-2		<i>Df(1)v⁺75d</i>	<i>M(1)14-171</i>	5
M(1)11F	1-[42]	11F1-4	<i>Df(1)wy2</i>	<i>Df(1)N12</i>		7
M(1)13A	1-[49]	12F6-13B6	<i>Y^PX^DS29</i>	<i>Y^PX^DB128</i>		4
M(1)14C	1-[54]	14B13-D1	<i>Df(1)r75c</i>	<i>Df(1)r-D1</i>	<i>M(1)19-153</i>	5
M(1)15D	1-56.6	15D			<i>M(1)o</i>	3
M(1)18C	1-62.7	18B9-D	<i>Y^PX^DV6</i>	<i>Y^PX^DB50</i>	<i>M(1)n</i>	4

^α 1 = Demerec, Kaufmann, Fano, Sutton, and Sansome, 1942, Carnegie Inst. Wash. Year Book 41: 191; 2 = Lefevre, Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 32-66; 3 = Lefevre, 1974, Cold Spring Harbor Symp. Quant. Biol. 38: 591-99; 4 = Merriam *et al.*; 5 = Schalet, 1986, Mutat. Res. 163: 115-44; 6 = Schultz, 1929, Genetics 14: 366-419; 7 = Scott, 1987, Ph.D. Thesis, University of California at San Deigo; 8 = Voelker.

M(1)1B

discoverer: Patterson.

synonym: *Vi: Viability*.

references: 1932, Z. Indukt. Abstamm. Vererbungsl. 60: 125-36.

phenotype: Heterozygous deficiency for locus is extreme Minute of low viability. In Patterson's work, the nonappearance of Minutes led him to postulate a factor for viability (*Vi*). Eclosion delayed 76 hr (Ferrus, 1975, Genetics 79: 589-99). RK3A.

alleles: Single ethyl-nitrosourea-induced mutant allele recovered by Voelker in a screen for lethals in the vicin-

ity of *su(s)*. Lethality occurs between the first larval instar and pupation (Eberl, Hilliker, and Voelker, 1988, DIS 67: 36).

molecular biology: Both the recessive lethality of *M(1)1B*^{B46} and the Minute phenotype of *Df(1)su(s)83* are rescued by a *P*-element transformant containing a 10 kb fragment of genomic DNA from immediately to the left of the *su(s)* gene. *M(1)1B* function is apparently encoded by a ~3.5 kb message (Voelker, Huang, Wisely, Sterling, Bainbridge, and Hiraizumi, 1989, Genetics 122: 625-42).

cytology: Associated with the *X*^P*2R*^D element of *T(1;2)Blid = T(1;2)1C3-4;60B12-13* when the *2R*^P*X*^D element is replaced by a normal second chromosome.

M(1)3E: Minute (1) in region 3E

location: 1-{5}.

discoverer: Demerec, 1938.

references: Demerec, Kaufman, Fano, Sutton, and Sansome, 1942, Year Book - Carnegie Inst. Washington 41: 191.

phenotype: Identified on basis of slight Minute phenotype, barely distinguishable from wild type, associated with deficiencies that include region 3E. RK3A.

cytology: Found and located in salivary chromosome bands 3E3-4 on the basis of slight *M* phenotype of females heterozygous for *Df(1)N264-76 = Df(1)3B4-C1;3E4-5* and non-*M* phenotype of females heterozygous for *Df(1)N264-38 = Df(1)2D3-4;3E2-3* and *Df(1)N264-117 = Df(1)3A6-7;3E2-3*. Also identified in a segmental deficiency scan of the *X* by Merriam and colleagues.

M(1)4BC: Minute (1) in region 4BC

location: 1-{6,8}.

discoverer: Demerec, 1938.

references: Demerec, Kaufmann, Fano, Sutton, and Sansome, 1942, Year Book - Carnegie Inst. Washington 41: 191.

phenotype: Strong Minute; easily distinguishable from wild type. RK2A.

cytology: Inferred to be in salivary gland chromosome region 4B5 through 4C6 on the basis of extreme *M* phenotype of females heterozygous for *Df(1)N*²⁶⁴⁻⁷³ = *Df(1)3C3-4;4C6-7* versus slight *M* phenotype [*M(1)3E*] of *Df(1)N*²⁶⁴⁻⁴² = *Df(1)3C4-5;4B4-6*. However, no minute phenotype observed in a segmental - deficiency scan of 3F through 4F by Merriam and Colleagues. As *M(1)4BC* was inferred only deficiencies including both 3E and 4BC, perhaps deficiency for 4BC acts as an enhancer of *M(1)3E*; however Merriam and colleagues fail to report such an enhancement.

M(1)5A

phenotype: Identified by extreme Minute phenotype and very low viability of segmental deficiencies for 5A6-13.

other information: Possibly the same as the lost *M(1)30*, a spontaneous deficiency, which included *cv* at 5B, recovered by Schultz (1929, Genetics 14: 366-419).

M(1)5D6A

other information: Considered the same as the lost spontaneous Minute, *M(1)30* (CP627; Lefevre, 1973, Cold Spring Harbor Symp. Quant. Biol. 38: 591-99; Merriam *et al.*); *M(1)30* was associated with *Df(1)M30*, which included *cv* at 5B.

M(1)8F

origin: Spontaneous.

phenotype: Readily classified Minute found between *lz* and *v* (Schalet, 1986, Mutat. Res. 163: 115-44).

cytology: Since the proximal breakpoint of *Df(1)lz5*, which is *M*⁺, is listed as 8F-9A (CP627), and the cytology of another *M*⁺ deficiency *Df(1)C52* is listed as *Df(1)8F3-9D6* (Mason, Green, Shaw, and Boyd, 1981, Mutat. Res. 81: 329-43), *M(1)8F* is probably located at 8F1-2 (Schalet, 1986).

M(1)11F

phenotype: Identified on the basis of the slightly finer bristles and protracted development of *Df(1)C246/+* females (Craymer and Roy, 1955, DIS 55: 204).

other information: Tentatively equated to *M(1)k* by Craymer and Roy; however, genetic map position of *M(1)k* discordant with cytological position of *Df(1)C146*; also *M(1)k* described as strong Minute.

M(1)14C

origin: Spontaneous.

phenotype: Moderate Minute.

cytology: Located in the 14B13 to 14D1-2 interval since allelic to *Df(1)r75c = Df(1)14B13;15A9* but not to *Df(1)r-D1 = Df(1)14D1-2;15D1-2* (Schalet, 1986, Mutat. Res. 163: 115-44).

M(1)15D

phenotype: Eclosion delayed 35 hr (Ferrus, 1975, Genetics 79: 589-99).

alleles:

allele	origin	discoverer	synonym	ref ^α
<i>M(1)15D</i> ¹	spont	Bridges, 24B24	<i>M(1)o</i>	1
<i>M(1)15D</i> ²	spont	Spencer, 32i28	<i>M(1)o</i> ^{Sp} <i>M(1)Sp</i>	1, 2, 3

^α 1 = CP627; 2 = Spencer, 1935, DIS 3: 28; 3 = Spencer, 1937, DIS 7: 14.

M(1)18C

phenotype: Heterozygous females have Minute bristles. Lethal in males. Viability and fertility low. Pupation delayed about 42 hr at 25° (Brehme). Wing cells smaller than normal (Brehme, 1941, J. Exp. Zool. 88: 135-60). RK2.

allele	origin	discoverer	synonym	ref ^α	comments
<i>M(1)18C</i> ¹	spont	Bridges, 1923	<i>M(1)n</i>	1, 2, 4	
* <i>M(1)18C</i> ^{2β}	spont	Curry, 36f10	<i>M(1)n</i> ³⁶ <i>M(1)36f</i>	2, 3	weak <i>M</i> in <i>C(1)RM</i>

^α 1 = Bridges, 1925, Proc. Nat. Acad. Sci. USA 11: 701-06; 2 = CP627; 3 = Curry, 1937, DIS 7: 14; 4 = Morgan, Sturtevant, and Bridges, 1924, Carnegie Inst. Wash. Year Book 23: 231-36.

^β Allelism inferred from locations of *M(1)18c*² at 62.

M(1)30: see *M(1)5D6A*

M(1)34i28: see *M(1)o*^{Sp}

M(1)36f: see *M(1)18C*

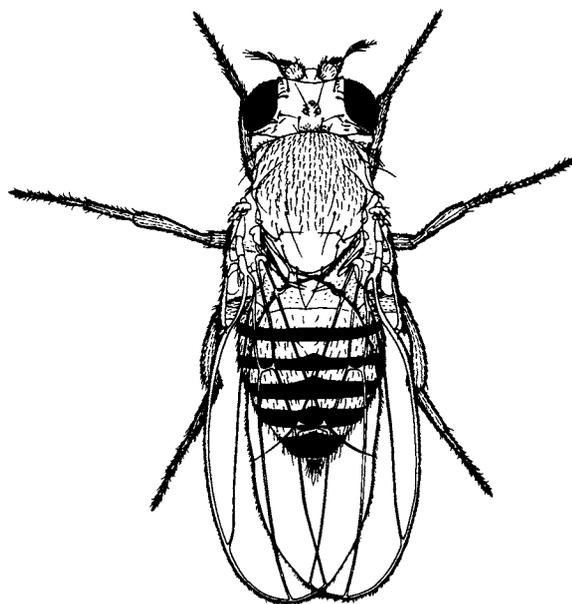
M(1)Blid: see *M(1)1B*

****M(1)k***

location: 1-36.3

discoverer: Bridges, 23d28.

phenotype: A strong Minute. RK2.



M(1)18C¹: Minute (1)18C¹

Edith M. Wallace, unpublished.

other information: Placed approximately 0.1 cm to the right of *m* in 10E1-2 in a cross of a type in which the *v* - *g* distance was normal. However, no Minute phenotype discovered by Merriam and colleagues in a segmental - deficiency scan of 9A-12F. The original mutant may have been a gain-of-function allele of a haplo-sufficient gene.

M(1)n: see *M(1)18C*

M(1)o: see *M(1)15D*

M(1)o^{Sp}: see *M(1)15D*

M(1)Sp: see *M(1)15D*

M(2)

Fifteen second-chromosome Minute loci considered to be well established and localized are tabulated below; additional information provided in entries that follow the table. Several described Minutes that are less well established are also included among the subsequent entries.

locus	genetic location	cytological location	included in	excluded from	synonym	ref ^α
M(2)21AB	2-{0}	21AB		<i>Df(2L)al</i>	<i>Su(2)5</i>	
M(2)21C	2-0.0	21C1-2	<i>Df(2L)al</i>	<i>Df(2L)S5</i>		4
M(2)24D	2-{12}	24D4-8	<i>Df(2L)sc19-1</i>	<i>Df(2L)dp-h28</i>	<i>M(2)LS2</i>	2, 5, 9, 10
M(2)24F	2-12.9	24F1-25A1	<i>Df(2L)sc19-6</i>	<i>Df(2L)ed-sz1</i>	<i>M(2)z</i>	2, 5, 9, 10
M(2)25C	2-15.0	25B9-C3	<i>Df(2L)sc19-7</i>	<i>Df(2L)sc19-10</i>	<i>M(2)S1</i>	2, 5, 9, 10
M(2)30A	2-{40.5}	30A	<i>Df(2L)J4</i>	<i>Df(2L)J2</i>	<i>M(2)e</i> , <i>M(2)LS3</i>	2, 8
M(2)36F	2-54	36F2-6	<i>Df(2L)TW202</i>	<i>Df(2L)hk18</i>	<i>M(2)m</i>	5, 2
M(2)39F	2-54.3	39E-40A	<i>Df(2L)TW161</i>	<i>Df(2L)TW65</i>	<i>M(2)H</i>	11
M(2)41A	2-55.1	41A			<i>M(2)S2</i>	2
M(2)44C	2-57	44C	<i>2^PY^DH136</i>	<i>2^PY^DB26</i>	<i>M(2)38b</i>	2, 12
				<i>Df(2R)cn9</i>		
M(2)53	2-77.5	52D-53E	<i>Y^P2^DR14</i>	<i>4^P2^DC4</i>	<i>M(2)S7</i>	5
M(2)56CD	2-87.5	56C-D	<i>Y^P2^DB184</i>	<i>Y^P2^DG100</i>	<i>M(2)b</i>	5, 7
M(2)56F	2-92.3	56F5-15	<i>Df(2R)017</i>		<i>M(2)173</i>	2, 3, 5
M(2)58F	2-101.2	58F	<i>Df(2R)M58F</i>		<i>M(2)l</i>	1, 6, 7
M(2)60E	2-108	60E	<i>Df(2R)M60E</i>		<i>M(2)c</i>	

locus	genetic location	cytological location	included in	excluded from	synonym	ref ^α
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^α 1 = Bridges, 1937, Cytologia, Fujii Jub., Vol. 2: 745-55; 2 = CP627; 3 = Duttagupta and Shellenbarger, 1980, Development and Neurobiology of Drosophila (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York and London, pp. 25-33; 4 = Lewis, 1945, Genetics 30: 137-66; 5 = Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84; 6 = Morgan, Bridges, and Schultz, 1937, Carnegie Inst. Wash. Year Book 36: 298-305; 7 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 231; 8 = Schultz, 1929, Genetics 14: 366-419; 9 = Semeshin and Szidonya, 1985, DIS 61: 148-54; 10 = Szidonya and Reuter, 1988, Genet. Res. 51: 197-208; 11 = Wright, Hodgetts, and Sherald, 1976, Genetics 84: 267-85; 12 = Yannopoulos, Stamatias, Zacharopoulou, and Pelecanos, 1981, Mutat. Res. 83: 383-93.

M(2)21AB

location: 2-0.0 (3.8 cm to the left of *shv*).

origin: Induced by ethyl methanesulfonate.

synonym: *Su(z)5*.

references: Persson, 1976, Hereditas 82: 57-62.

1977, DIS 52: 1.

phenotype: Body size and bristles reduced in heterozygotes; irregular facets in upper half of eye. Heterozygous females virtually sterile at temperatures above 18°; surviving offspring frequently have missing legs or halteres, somewhat narrowed wings, and reduced bristles. Homozygotes lethal. Selected on the basis of a dominant suppressor of *zeste* phenotype. Characterized by relatively frequent reversions of all features of the phenotype save homozygous lethality. One such revertant found to be deficient for the tip of *2L*, suggesting a gain of function rather than haplo-insufficiency as the nature of the mutation (Kennison).

cytology: Salivary-gland chromosomes appear normal. Placed in 21A-B on the basis of complementation by *M(2)21AB* of *Df(2L)al*, *Df(2L)S2*, and *Df(2L)S3*, the recovery of 0/196, 0/284, and 0/642 recombinants with *net*, *al*, and *ex*, respectively, and a position 3.8 cM from *shv*; complements *l(2)gl*.

M(2)21C

phenotype: Identified by virtue of the extreme Minute phenotype of *Df(2L)al/+*.

M(2)24F

phenotype: Medium Minute with good characteristics.

About two days delay in puparium formation (Dunn and Mossige, 1937, Hereditas 23: 70-90). RK2.

alleles: Three alleles are listed in the following table.

allele	origin	discoverer	synonym	ref ^α
M(2)24F¹	spont	Schultz	<i>M(2)z</i>	2
M(2)24F²	X ray	Green	<i>M(2)z G1</i>	1
M(2)24F³	X ray	Green	<i>M(2)z G2</i>	1

^α 1 = Persson, 1977, DIS 52: 1; 2 = Schultz, 1929, Genetics 14: 366-419.

*M(2)25C

origin: X ray induced.

discoverer: Schultz, 33a12.

phenotype: Small-bristled Minute with heavy body. Classification good. Viability and fertility fairly good. RK2.

M(2)29: see *M(2)56F*

M(2)30A

location: 2-40.5 [based on cytological position immediately to the left of *J* (2-41); originally mapped to 46±5].

phenotype: *M(2)30A*¹ is a medium Minute with delayed hatching. 50% of females and 10% of males show abnormal abdomen effect. Most females sterile and remainder produce few progeny. RK3(A).

alleles: Four presumptive alleles, but no complementation studies done. Allelism based on similarity of genetic positions of *M(2)30A*¹ = 46±5, *M(2)30A*² = 43, and *M(2)30A*³ = 46±5; also both *M(2)30A*^{1/+} and *M(2)30A*^{4/+} described as female sterile.

allele	origin	discoverer	synonym	ref ^α	comments
*M(2)30A ¹	spont	Bridges, 20b25	<i>M(2)e</i>	2	moderate allele
*M(2)30A ²	spont	Bridges, 24i16	<i>M(2)t</i> , <i>M(2)e</i> ¹	1	weak allele
M(2)30A ³	X ray	Schultz, 34k21	<i>M(2)S11</i> , <i>M(2)e</i> ^s	1	moderate allele
M(2)30A ⁴	X ray	Sandler	<i>M(2)fs</i>	3	

^α 1 = CP627; 2 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 231; 3 = Sandler, 1977, Genetics 86: 567-82.

cytology: Placed in 30A based on inclusion in *Df(2R)J4* = *Df(2R)31A-B;31F-32A*, but not in *Df(2R)J2* = *Df(2R)31B;32A*. Also distal to the breakpoint of *T(Y;2)LS2* = *T(Y;2)30F-31A*.

other information: Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero (1972, Genetics 71, 157-84) placed *M(2)e* in 28D-29F, where reexamination of the data suggests no *M* locus exists; their *M(2)LS3* presumably corresponds to *M(2)e* = *M(2)30A*.

M(2)33a: see *Df(2R)M60E*

M(2)36F

phenotype: Medium to strong Minute. Eclosion of heterozygous deficiency delayed 30 hr (Ferrus, 1975, Genetics 79: 589-99).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
*M(2)36F ¹	spont	Bridges, 23g12	<i>M(2)m</i>	1, 2	strong Minute
*M(2)36F ²	spont	Schultz, 24i28	<i>M(2)m</i> ^s , <i>M(2)s</i>	1, 4	medium Minute
*M(2)36F ³	X ray	Schultz, 33b3	<i>M(2)m</i> ^{S13} , <i>M(2)S13</i>	1	strong Minute
M(2)36F ⁴	X ray	Green	<i>M(2)m</i> ^G	3	
M(2)36F ⁵	X ray	Green	<i>M(2)H</i> ^G	3	

^α 1 = CP627; 2 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 231; 3 = Persson, 1977, DIS 52: 1; 4 = Schultz, 1929, Genetics 14: 366-419.

cytology: Originally assigned its present chromosomal position on the basis of its extreme phenotype and that of the combination *Df(2L)G/Dp(2;Y)H* plus its genetic map position estimated to be 2-54 by Bridges, even though the appropriate complementation tests not performed. Allelisms inferred from similarities in genetic map positions: *M(2)36F*² = 54.5; *M(2)36F*³ = 50; *M(2)36F*³ complemented by *Dp(1;Y)G*, but not by *Dp(1;Y)H*; *M(2)36F*⁴ fails to complement *Df(2L)M36-56* and *M(2)36F*⁵ fails to complement *Df(2L)M36F-S5*.

M(2)38b: see *M(2)44C*

M(2)39F

phenotype: Originally identified on the basis of the weak Minute phenotype of heterozygotes for the *Df(2L)H* segregant from *Tp(2;Y)H* = *Tp(2;Y)37B1-2;40B2-3*.

alleles:

allele	origin	discoverer	synonym	ref ^α
*M(2)39F ^{1β}	X ray	Schultz, 33a9	<i>M(2)H</i> ^{S5} , <i>M(2)SS</i>	1, 2
M(2)39F ^{2γ}	X ray	Schultz, 33b7	<i>M(2)H</i> ^{S12} , <i>M(2)S12</i>	1

^α 1 = CP627; 2 = Wright, Hodgetts, and Sherald, 1976, Genetics 84: 267-85.

^β Originally complemented *M(2)36F*; current stocks labelled *M(2)H*^{SS} do not; they are *Df(2L)M36F-S5* (Wright, Hodgetts, and Sherald, 1976, Genetics 84: 267-85).

^γ Allelism inferred from weak phenotype and inseparability from *pr*.

***M(2)40c**

location: 2-65±5.

origin: Spontaneous.

discoverer: Ives, 40c.

references: 1941, DIS 14: 39.

phenotype: Medium Minute with probable eye effect. RK2.

cytology: Crossing over normal. Genetic map position of 65±5 places *M(2)40c* in the vicinity of *vg*. Tentatively placed in the region deleted by the segmental deficiency formed from *T(Y;2)D19* and *T(Y;2)G53* [= *Df(2R)48E;50A*] [Pasztor, 1976, Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 185-206]; this region encroached upon from the right by *Df(2R)vgC* = *Df(2R)49B2-3;49E1-F1*, which has no Minute phenotype, thus restricting the tentative position of *M(2)40c* to 48E-49B.

alleles: Besides the original allele, a second allele, *M(2)40c*² (= *M(2)40c*^G), tentatively identified as an allele by genetic map position, X ray induced by Green (Persson, 1977, DIS 52: 1).

M(2)41A

phenotype: Medium Minute. Originally identified by virtue of Minute phenotype of *Df(2R)M41A10*; most recurrences shown to be deficiencies. Eclosion of heterozygous deficiency delayed 13 hr (Ferrus, 1975, Genetics 79: 589-99).

alleles: Formerly listed alleles of *M(2)41A* known to be deficiencies by virtue of failing to complement mutants in adjacent loci are listed with the deficiencies.

allele	origin	discoverer	synonym	comments
M(2)41A ¹	X ray	Schultz, 33a	<i>M(2)S2</i> ³ , <i>M(2)S3</i>	no <i>b-pr</i> recombination
M(2)41A ²	X ray	Schultz, 32k31	<i>M(2)S2</i> ⁹ , <i>M(2)S9</i>	

cytology: Placed in 41A, *i.e.*, in the proximal heterochromatin of 2R, based on the normal polytene configuration but reduced amount of heterochromatin at the base of 2R in ganglion prophase figures of *Df(2R)M41A10*.

M(2)44C

phenotype: Extreme Minute with small bristles and compact body. Viability varies with modifiers. *M(2)44C/stw* is non-*stw*; *M(2)44C/M(2)p* is viable. RK3.

cytology: Tentatively placed in 44C based on its genetic

map position, and the Minute phenotype of the segmental deficiency between *T(Y;2)B26* and *T(Y;2)H136* [= *Df(2R)43E-F;44C*], but not of *Df(2R)cn9* = *Df(2R)42E;44C*.

alleles: *M(2)44C*¹ is the original spontaneous allele isolated by Curry (33b18); a second allele, *M(2)44C*² X ray induced by Green (Persson, 1977, DIS 52: 1).

M(2)47: see *M(2)56F*

M(2)50J: see *Df(2R)M41A50J*

M(2)51: see *M(2)56F*

M(2)53

origin: X ray induced (occurred as a mosaic).

discoverer: Schultz, 33a2.

phenotype: Bristles very small; arista often reduced; venation plexus like. Ecloses 24 hr late (Ferrus, 1975, Genetics 79: 589-99). Viability about 70% of wild type and variable. Fertility good. RK2.

cytology: Salivary chromosomes apparently normal.

other information: May be the same as *RpA1*.

M(2)56CD

phenotype: Bristles extremely small. Abnormal abdomen effects in 90% of females and 40% of males. RK2.

other information: The first Minute found in chromosome 2.

M(2)56F

phenotype: Moderate Minute; expression enhanced when heterozygous to *l(2)56Fb*; enhances expression of *cp* (Schultz, 1929, Genetics 14: 366-419) and suppresses extra-sex-comb phenotype of *Msc* and *Pc* (Sinclair, Suzuki, and Grigliatti, 1981, Genetics 97: 581-606).

alleles: Identified through recovery of *M(2)56F*¹, which although cytologically normal fails to complement mutant alleles that complement each other, *l(2)56Fa*, *l(2)56Fb*, and *M(2)56F*; accordingly *M(2)56F*¹ considered a deficiency [*Df(2R)173*]. *M(2)56F*⁴ and *M(2)56F*⁵ lethal in combination with *Df(2R)017* and *Df(2R)173*, but survive as homozygotes exhibiting Minute-like bristles; enhance Minute phenotype when heterozygous to other *M(2)56F* alleles and to each other, except that *M(2)56F*⁴ and *M(2)56F*⁷ complement.

allele	origin	discoverer	synonym	ref ^α	comments
<i>M(2)56F</i> ¹		Csik	<i>M(2)173</i>	1, 3	multilocus; cytology normal
<i>M(2)56F</i> ²	EMS		<i>M(2)29</i>	2, 5	noncomplementing
<i>M(2)56F</i> ³	EMS		<i>M(2)47</i>	2, 5	noncomplementing
<i>M(2)56F</i> ⁴	EMS		<i>M(2)51</i>	2, 5	recessive; complementing
<i>M(2)56F</i> ⁵	EMS		<i>M(2)2362</i>	2, 5	recessive, noncomplementing
<i>M(2)56F</i> ⁶	EMS		<i>M(2)D741</i>	2, 5	noncomplementing
<i>M(2)56F</i> ⁷	EMS		<i>M(2)U</i>	2, 5	complementing
<i>M(2)56F</i> ⁸	X ray	Green	<i>M(2)173</i> ^G	4	

^α 1 = Csik, 1930, Magy. Biol. Kutatointez. Munkai 3: 438-53; 2 = Duttagupta and Shellenbarger, 1980, Development and Neurobiology of Drosophila (Siddiqi, Babu, Hall and Hall, eds.). Plenum press, New York and London, pp. 25-33, 1980; 3 = Gottschewski, 1935, DIS 4: 15; 4 = Persson, 1977, DIS 52: 1; 5 = Shellenbarger and Duttagupta, 1978, Mutat. Res. 52: 395-407.

M(2)58F

phenotype: Identified by the strong Minute phenotype of *Df(2R)M58F*. Heterozygosity for null allele as represented by the deficiency characterized by a two-day delay in eclosion (at 25°) owing to delay in puparium formation (Dunn and Mossige, 1937, Hereditas 23: 745-55); delay 19 hr according to Ferrus (1975, Genetics 79: 589-99). Eyes somewhat rough; veins often show plexus; abdominal sclerites often abnormal; ocelli often reduced. Viability 80-90% wild type and fertility low. Presumed mutant allele, *M(2)58F*¹, displays 13 hr delay in puparium formation at 25° (Brehme, 1939, Genetics 24: 131-61); slight delay in time of second larval molt. Viability, fertility, and classification excellent. Homozygote lethal in first larval instar.

alleles: *M(2)58F*¹ (formerly *M(2)l'* or *M(2)l*²), spontaneous with apparently normal polytene chromosomes, Schultz (26a7); *M(2)58F*², γ ray induced, with Minute phenotype suppressed by *SM5*, as if this Minute lies within the 58B-F segment duplicated in *SM5* (Kennison: *M* located between *Pu*² and *Pin*^B).

M(2)60E

phenotype: Fairly strong Minute; slow development; good viability and fertility [description based on *Df(2R)M60E* = *Df(2R)60E2-3;60E11-12*]. Eclosion of *Df(2R)M60E/+* delayed 49 hours (Ferrus, 1975, Genetics 79: 589-99).

alleles:

allele	origin	discoverer	synonym	ref ^α
* <i>M(2)60E</i> ¹	spont	Sturtevant, 20a7	<i>M(2)c</i>	1, 2, 3
<i>M(2)60E</i> ²	X ray	Green	<i>M(2)c</i> ^G	4

^α 1 = Bridges, 1937, Cytologia, Fujii Jub., Vol. 2, 745-55; 2 = CP627; 3 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 231; 4 = Persson, 1977, DIS 52: 1.

M(2)115: see *Df(2)M60E*

M(2)173: see *M(2)56F*

M(2)2362: see *M(2)56F*

M(2)a: see *M(2)60E*

M(2)b: see *M(2)56CD*

M(2)B: see *Df(2L)M24E-B*

M(2)c: see *M(2)60E*

M(2)C: see *Df(2L)M24E-C*

**M(2)d*

location: 2-72.

discoverer: Bridges, 20b25.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 231-34.

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 231.

phenotype: Heterozygote has no effect except when also heterozygous for *M(3)d*. The double heterozygote has Minute bristles in about 95% of flies. Probably lethal in homozygote. RK3.

M(2)D: see *Df(2R)M41A-D*

M(2)0741: see *M(2)56F*

M(2)e: see *M(2)30A*

M(2)fs: see *M(2)30A*

M(2)H: see *M(2)39F*

M(2)l: see *M(2)58F*

M(2)LS1: not a Minute (Spencer, Hoffmann, and Gelbart, 1982, Cell 28: 451-61).

M(2)LS2: see *M(2)24E*

M(2)LS3: see *M(2)30A*

M(2)m: see *M(2)36F*

***M(2)p**

location: 2- (to the right of *msf* at 2-55.2).

discoverer: Bridges, 24b6.

references: Curry, 1939, DIS 12: 46.

Morgan, Schultz, Bridges, and Curry, 1939, Year Book - Carnegie Inst. Washington 38: 273-77.

phenotype: Bristles small. Survives in combination with *M(2)44C*; not tested against Minutes further to the right. RK3(A).

other information: May also have a second Minute factor to left of *pr*. Crossing over possibly reduced.

M(2)p^D: see *Df(2R)M41A-D*

M(2)s: see *M(2)36F²*

M(2)S1: see *M(2)25C*

M(2)S2: see *Df(2R)M41A*

M(2)S3: see *M(2)41A*

M(2)S4: see *Df(2R)M41A4*

M(2)S5: see *M(2)39F*

M(2)S6: see *M(2)36F*

M(2)S7: see *M(2)53*

M(2)S8: see *Df(2R)M41A8*

M(2)S9: see *M(2)41A*

M(2)S10: see *Df(2R)M41A10*

M(2)S11: see *M(2)30A*

M(2)S12: see *M(2)39F*

M(2)S13: see *M(2)36F*

M(2)t: see *M(2)30A*

M(2)U: see *M(2)56F*

M(2)vg¹¹: see *Df(2R)M-vg11*

M(2)z: see *M(2)25A*

***M(2)**

Minute mutations in the second chromosome that are lost and were never characterized are tabulated below.

Mutant	origin	discoverer	ref ^α	comments
<i>*M(2)28</i>		Schultz	3	moderate, in <i>In(2R)Cy</i>
<i>*M(2)33d</i>	X ray	Oliver, 33d14	2	<i>In(2L+2R)Cy</i>
<i>*M(2)34b</i>	X ray	Oliver, 34b3	2	<i>In(2L+2R)Cy</i>
<i>*M(2)34d</i>	X ray	Oliver, 34d25	2	<i>In(2L+2R)Cy</i>
<i>*M(2)34k</i>	X ray	Oliver, 34k22	2	<i>In(2L+2R)Cy</i>
<i>*M(2)38k</i>	spont	Mosssige, 38k4	1	

^α 1 = CP627; 2 = Oliver, 1939, DIS 12: 48; 3 = Schultz, Genetics 14: 366-419.

M(3)1: see *M(3)99B*

M(3)6: see *M(3)69E*

M(3)33d: see *M(3)69E*

M(3)33j: see *M(3)69E*

M(3)36e: see *M(3)96C*

M(3)

18 third-chromosome Minute loci are tabulated; additional information provided in entries that follow the table.

locus	genetic location		cytological location		included in	excluded from	synonym	ref ^α
	locus	location	location	location				
<i>M(3)62A</i>	3-[0]	61F-62A	<i>Y^P₃^D_{D8}</i>	<i>Y^P₃^D_{A114}</i>			<i>M(3)LS1</i>	3
<i>M(3)62F</i>	3-[2]	62E-63A	<i>Y^P₃^D_{B21}</i>	<i>Y^P₃^D_{D8}</i>			<i>M(3)LS2</i>	3
<i>M(3)63B</i>	3-[5]	63B6-C1	<i>Df(3L)HR298</i>	<i>Df(3L)HR232</i>			<i>M(3)LS3</i>	3
<i>M(3)65F</i>	3-[23]	65F10-11	<i>Dp(3;3)MS3</i>	<i>Dp(3;3)MS2</i>			<i>M(3)S37</i> <i>M(3)h^{S37}</i> <i>M(3)i^{S5}</i>	6 6
<i>M(3)67C</i>	3-28.9	67C1-10	<i>Dp(3;3)MS6</i> <i>Dp(3;3)MS7</i>					
<i>M(3)69E</i>	3-40.2	69E2-69F	<i>Df(3L)VW3</i>	<i>3^P_{Y^D_{R7}}</i>			<i>M(3)h</i>	1, 6
<i>M(3)76A</i>	3-44.3	76A3-B2	<i>Df(3L)VW1</i>	<i>Y^P₃^D_{L131}</i>			<i>M(3)S34</i>	1, 7, 8
<i>M(3)80</i>	3-[47]	79E5-80F	<i>Y^P₃^D_{G72}</i>	<i>Y^P₃^D_{J162}</i>			<i>M(3)LS4</i>	3, 10
<i>M(3)82BC</i>	3-47	82A-82C	<i>Y^P₃^D_{J17}</i>	<i>Y^P₃^D_{A154}</i>			<i>M(3)S39</i>	3
<i>M(3)85E</i>	3-[50]	85E2-F1	<i>Df(3R)by10</i> <i>Df(3R)by62</i>				<i>M(3)LS5</i>	2, 3, 4
<i>M(3)86D</i>	3-50.0	86D1-4	<i>Df(3R)M86D</i>				<i>M(3)S31</i>	1, 3, 4
<i>M(3)95A</i>	3-79.7	94D-94E	<i>Dp(3;3)M95A</i>				<i>M(3)w</i> , <i>M(3)B</i> , <i>M(3)Fla</i>	9, 11
<i>M(3)96A</i>	3-[84]	95E6-96A5	<i>Y^P₃^D_{H173}</i>	<i>Y^P₃^D_{G73}</i>				3
<i>M(3)96C</i>	3-84.5	96C1-5	<i>Y^P₃^D_{B217}</i>	<i>Y^P₃^D_{H135}</i>			<i>M(3)be</i>	2
<i>M(3)96CF</i>	3-90.2	96C-97A	<i>Y^P₃^D_{H135}</i>	<i>Y^P₃^D_{R87}</i>			<i>M(3)j</i>	2
<i>M(3)99B</i>	3-101.2	99B5-9	<i>Dp(3;Y)L127</i>	<i>Dp(3;1)46A</i>			<i>M(3)l</i>	5
<i>M(3)99D</i>	3-[101]	99D1-9	<i>Dp(3;1)R14</i>	<i>Dp(3;1)R10</i>				5
<i>M(3)99E</i>	3-106.2	99E4-F1	<i>Dp(3;1)124P</i>	<i>Y^P₃^D_{G116}</i>			<i>M(3)g</i>	5
<i>M(3)100CF</i>	3-105	100C-tip	<i>Y^P₃^D_{L129}</i>				<i>M(3)j</i>	5

^α 1 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91; 2 = CP627; 3 = González, Molina, Casal, and Ripoll, 1989, Genetics 123: 371-77; 4 = Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84; 5 = Kempfues, Raff, and Kaufman, 1983, Genetics 105: 345-56; 6 = Kongswan, Dellavalle, and Merriam, 1986, Genetics, 112: 539-50; 7 = Moscoso del Prado and Ripoll, 1983, Genet. Res. 42: 59-63; 8 = Schalet, 1960, DIS 34: 55; 9 = Schultz, 1929, Genetics 14: 366-419 10 = Stern, 1927, Naturwissenschaften 15: 745 11 = Sinclair, Suzuki, and Grigliatti, 1981, Genetics 97: 581-606; 12 = Vässin, Vielmetter, and Campos-Ortega, 1985, J. Neurogenet. 2: 291-308.

M(3)65F

origin: X ray induced.

discoverer: Schultz, 33a12.

phenotype: Extreme Minute with fine bristles and small body. RK3.

other information: Considered to be allelic to *M(3)69E* [*M(3)h* in CP627] on the basis of lethality of the *trans* heterozygote; however, Moscoso del Prado and Ripoll (1983, Genet. Res. 42: 59-63) demonstrated that the two Minutes occupy different cytological positions, thus rendering fallible the rule that double Minute genotypes are no more severe in phenotype than the more severe member of the pair, and suggesting some caution in concluding allelism based on lethality of *trans* heterozygotes.

M(3)67C

phenotype: Moderate alleles have good heterozygous viability; extreme alleles very late eclosing [45 hr according to Ferrus (1975, Genetics 79: 589-99)] with poor viability, and females usually sterile. Viability further reduced in presence of *su(f)* alleles (Girton, Langer, Cejka, 1986, Roux's Arch. Dev. Biol. 195: 334-37).

alleles: Most assignments of allelism based on map positions in the vicinity of 3-30; *M(3)67C*⁴ through *M(3)67C*⁷ assigned on the basis of complementation results (Moscoso del Prado, 1983, Genet. Res. 42: 59-63; Persson, 1977, DIS 52: 1).

allele	origin	discoverer	synonym	ref ^α	comments
<i>*M(3)67C</i> ¹	spont	Bridges, 23d23	<i>M(3)j</i>	1	medium Minute
<i>*M(3)67C</i> ²	spont	Bridges, 24b28	<i>M(3)j</i> ^q , <i>M(3)q</i> ^{S33}	1	extreme Minute
<i>*M(3)67C</i> ³	spont	Schultz, 33a6	<i>M(3)j</i> ^{S33} , <i>M(3)S33</i>	1	extreme Minute
<i>M(3)67C</i> ⁴	EMS		<i>M(3)j</i> ^{S5}	2, 3	
<i>M(3)67C</i> ⁵	X ray	Green	<i>M(3)j</i> ^{G1}	4	
<i>M(3)67C</i> ⁶	X ray	Green	<i>M(3)j</i> ^{G2}	4	
<i>M(3)67C</i> ⁷	X ray	Green	<i>M(3)j</i> ^{G3}	4, 3	
<i>M(3)67C</i> ^{8β}	DEB	Leicht	<i>l(3)dt0A4</i>		non Minute?
<i>M(3)67C</i> ^{9β}	EMS	Leicht	<i>l(3)e80A6-1</i>		non Minute?

^α 1 = CP627; 2 = Morata and Ripoll, 1975, Dev. Biol. 42: 211-21; 3 = Moscoso del Prado, 1983, Genet. Res. 42: 59-63; 4 = Persson, 1977, DIS 52: 1.

^β Tentative assignments; these two recessive lethal alleles fail to complement each other, and *M(3)67C*⁹ fails to complement *M(3)67C*⁴.

M(3)69E

phenotype: Moderate alleles have good viability and fertility; puparium formation delayed two days (Dunn and Mossige, 1937, Hereditas 23: 70-90; Ferrus, 1975, Genetics 79: 589-99). Extreme alleles have low viability, flimsy or waxy wings with plexus effects along vein L2 and at posterior crossvein; eyes may be small and rough.

alleles: *M(3)69E*⁴ and *M(3)69E*⁵ both lethal in *trans* heterozygotes with *M(3)69E*¹, but see *M(3)66A*; allelism of *M(3)69E*² and *M(3)69E*³ inferred from genetic map positions. *Df(3L)M69E* considered an allele in CP627.

allele	origin	discoverer	synonym	ref ^α	comments
<i>*M(3)69E</i> ¹	spont	P. R. Sturtevant	<i>*M(3)h</i>	1, 2, 3, 5	medium Minute
<i>*M(3)69E</i> ²	heat	Ives, 33d30	<i>*M(3)h</i> ^{33d}	3, 6	extreme Minute
<i>M(3)69E</i> ³	X ray	Schultz, 33a12	<i>M(3)h</i> ^{S38}	3	extreme Minute
<i>M(3)69E</i> ⁴	spont	Bridges, 25dt8	<i>M(3)h</i> ^v , <i>M(3)v</i>	3	medium Minute
<i>M(3)69E</i> ⁵	spont	Sturtevant, 25g19	<i>M(3)h</i> ^v , <i>M(3)y</i>	3, 4, 5, 7	medium Minute

^α 1 = Bridges and Morgan, 1923, Carnegie Inst. Wash. Publ. No. 327: 244; 2 = Coyne, 1935, DIS 4: 59; 3 = CP627; 4 = Moscoso del Prado and Ripoll, 1983, Genet. Res. 42: 59-63; 5 = Mossige, 1938, Hereditas 23: 70-90; 6 = Plough and Ives, 1934, DIS 1: 33; 7 = Stern, 1927, Naturwissenschaften 15: 740-46.

M(3)76A

origin: X ray induced.

discoverer: Schultz, 33a6.

phenotype: Slight Minute; overlaps wild type; in existing lines bristles appear normal, but recessive lethal effect at 44.3 remains. RK3.

M(3)80

synonym: *M(3)Q-III*, *M(3)LS4 Q-III*.

phenotype: Originally identified by virtue of the Minute

phenotype of the segmental deficiency for 79E5 to the chromocenter. An EMS-induced temperature-sensitive allele recovered and described by Sinclair, Suzuki, and Grigliatti (1981, Genetics 97: 581-606). Heterozygotes raised at 22° normal in phenotype; those raised at 29° display small bristles, rough eyes, prolonged developmental time, reduced viability, and interact with several unrelated mutations; at 29° but not at 22°, in the presence of *M(3)80/+*, *vg/+* and *cp/+* exhibit reduced viability and incomplete wing margins; enhances *J^{34e}/+*, *Ly/+*, *Dfd/+*, and *DI^D/+* such that they are lethal or weakly viable; also suppresses extra-sex-comb phenotypes of *PC* and *Msc*. These effects are similar to those shown by other minutes at all temperatures. The temperature sensitive periods of these effects reflect the time of action of the modified genes (Sinclair, Grigliatti, and Kaufman, 1984, Genet. Res. 43:257-75). *M(3)80* homozygotes reared at 22° display moderate small bristle and rough eye phenes, as well as prolonged development; homozygotes lethal when reared at 29°. Homozygous females fertile, but switching to 29° blocks egg production; males at 22° are poorly fertile and become sterile upon switching to 29°. Temperature-pulse experiments yield a number of abnormal phenotypes which differ depending on the stage and duration of the pulse; described by Sinclair *et al.*

other information: As there is apparently but a single mutant allele, we designate it *M(3)80*¹, sinking to synonymy the specific designation *Q-III*, used by the authors. May be the same as *Rp21*.

M(3)82BC

origin: X ray induced.

discoverer: Schultz, 33a3.

phenotype: Extreme Minute with small body. Low viability and fertility. RK3.

M(3)85E

other information: Identified by virtue of the Minute phenotype of the segmental deficiency produced between *T(Y;3)G42* and *T(Y;2)L17* [*Df(3R)85E2-4;86A*] and equated to *M(3)S31* (Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84). *M(3)S31* subsequently shown to lie in 86D (Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91), and this Minute deficiency mapped to 85E2-F1 and renamed *M(3)LS5* (Kempthues, Raff, and Kaufman, 1983, Genetics 105: 345-56).

M(3)86D

phenotype: Fine-bristled Minute with medium viability. RK3.

other information: Identified by virtue of Minute phenotype of *Df(3R)M86D = Df(3R)86D1;86D4*; no mutant alleles identified. This Minute erroneously designated *M(3)S35* (Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84).

M(3)95A

phenotype: Strong Minute; eclosion delayed 40 hours (Ferrus, 1975, Genetics 79: 589-99).
alleles:

allele	discoverer	synonym	ref ^α	comments
M(3)95A¹	Schultz, 1925	<i>M(3)w</i>	7, 8	strong Minute
M(3)95A²	Csik	<i>M(3)w¹²⁴</i>	5, 6	strong Minute
M(3)95A³	Burkart	<i>M(3)124</i> <i>M(3)w^B</i>	3	moderate Minute
M(3)95A⁴	Bridges, 38c6	<i>M(3)B</i> <i>M(3)w^{B2}</i>	4	moderate Minute
M(3)95A⁵	Mossige, 35d	<i>M(3)B2</i> <i>M(3)w^{Fla}</i> <i>M(3)Fla</i>	1, 2	strong minute

^α 1 = Bryson, 1937, DIS 7: 18; 2 = Bryson, 1939, DIS 12: 50; 3 = Burkart, 1935, DIS 4: 15; 4 = CP627; 5 = Csik, 1930, Magy. Biol. Kutatointez. Munkai 3: 438-53; 6 = Gottschewski, 1935, DIS 4: 15; 7 = Schultz, 1929, Genetics 14: 366-419; 8 = Stern, 1927, Naturwissenschaften 15: 740-46.

cytology: Placed in 95A1 by Broderick and Roberts and in 94D-E by Vässin.

M(3)96A

phenotype: Heterozygotes for *3^PY^DG73 Y^P3^DH173* display slender bristles; viability and developmental time, however, normal.

M(3)96C

phenotype: Medium Minute of excellent viability. Eclosion delayed 31 hr (Ferrus, 1975, Genetics 79: 589-99). *M(3)96C²* reported to show plexus effect along vein L2 and at posterior crossvein. RK2.

alleles: Allelism based on similarity of genetic map positions which put *M(3)96C¹* at 87± and *M(3)96C²* at 84.5.

allele	origin	discoverer	synonym	ref ^α
*M(3)96C¹	spont	Stern, 26a20	<i>M(3)be</i>	1
M(3)96C²	spont	Bridges, 36e22	<i>M(3)be^{36e}</i> <i>M(3)36e</i>	

^α 1 = Stern, 1927, Naturwissenschaften 15: 740-46.

M(3)96CF

phenotype: Extreme Minute; very small bristles; late eclosing; females sterile or of very low fertility; males have fair viability and fertility. *M(3)96CF²* described as having broad wings with plexus of veins and somewhat abnormal abdominal bands.

alleles: Allelism of *M(3)96CF¹* and *M(3)96CF²* inferred from genetic map positions of 90.2 and 90±10, respectively.

allele	origin	discoverer	synonym	ref ^α	comments
M(3)96CF¹		Bridges, 23dt2	<i>M(3)j</i>		lethal with <i>l(3)PR</i>
M(3)96CF²	spont	Spencer, 36c21	<i>M(3)j^{Sp}</i> <i>M(3)Sp</i>	1	

^α 1 = Spencer, 1937, DIS 7: 14.

M(3)99B

origin: Spontaneous.

discoverer: Bridges, 19b8.

references: Bridges and Morgan, 1923, Carnegie Inst. Wash. Publ. No. 327: 206-7 (fig.).

phenotype: Bristles slender and shorter than wild type. Somewhat late hatching. Heterozygous deficiency for *M(3)99B* also displays rough eyes and slightly plexate

wings (Kongsuwan, Dellavalle, and Merriam, 1986, Genetics 112: 539-50). RK2.

other information: First Minute found.

M(3)99D

phenotype: Identified by the phenotype of heterozygotes for a synthetic deficiency; no mutant alleles identified. Strong Minute; small bristles, slightly abnormal wings; eclosion delayed two to three days (Kongsuwan, Dellavalle, and Merriam, 1986, Genetics 112: 539-50). *M(3)99D/+* females produce smaller than normal eggs; 10-15% of embryos fail to hatch often displaying fusion and partial deletion of segments posterior to A3 (Kongsuwan, Yu, Vincent, Frisardi, Rosbash, Lengyell, and Merriam, 1985, Nature 317: 555-58).

molecular biology: Encodes the large ribosomal subunit 49. Transformants carrying 2.1 kb of upstream and 0.6 kb of downstream sequence in addition to the entire coding sequence of *rp49* suppress the mutant phenotype, including the maternal effect, of heterozygous deficiencies for *M(3)99D* (Kongsuwan *et al.*, 1985).

M(3)99E

phenotype: Identified by virtue of moderate Minute phenotype of heterozygotes for a synthetic deficiency (Kongsuwan, Dellavalle, and Merriam, 1986, Genetics 112: 539-50).

other information: Designated *M(3)j* by Kongsuwan, Dellavalle, and Merriam, despite the fact that *M(3)j* was originally recorded as having a strong phenotype. Localized just proximal to the segmental deficiency for 99F-100B designated by Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero (1972, Genetics 71: 157-84) as deleting *M(3)j⁺*; Kongsuwan *et al.* found no Minute phenotype associated with deficiencies for that region. Most likely *M(3)j* corresponded to a mutant or deficiency for *M(3)99D* or *M(3)100CF*, both of which are recorded as having strong Minute phenotypes. The description of heterozygotes for deficiencies for *M(3)99E* correspond more closely to that for *M(3)g*.

M(3)100CF

phenotype: Identified by virtue of the strong Minute phenotype of heterozygotes for terminal deficiency for the region distal to 100C; very few escapers (Kongsuwan, Dellavalle, and Merriam, 1986, Genetics 112: 539-50). Reuter, Dorn, Wustmann, Friede, and Rauh (1986, Mol. Gen. Genet. 202: 481-87) describe *su(var)3-12*, which maps to 3-100.2, is associated with a Minute, and is deficient for 100F3-5.

other information: The distal segment of *3R* tentatively associated with a Minute by Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero (1972, Genetics 71: 157-84) on the basis of their failure to recover terminal deficiencies and the published genetic position of *M(3)g* at 3-106.2; Kongsuwan, Dellavalle, and Merriam (1986, Genetics 112: 539-50) followed suit, placing *M(3)g* distal to *M(3)j*; however, phenotypic considerations suggest the allelic correspondences presented in the table of third-chromosome Minutes.

Several Minute mutations in the third chromosome that

were never located and are no longer available are tabulated below:

mutant	origin	discoverer	ref ^α	comments
*M(3)32I	X ray	Oliver, 32I2	4	suppressed crossingover
*M(3)39b		Curry, 39b17	1	strong Minute
*M(3)54c	neutron	Mickey, 54c10	2	<i>In(3L)73A9-10;75D7-E1 + In(3LR)61C2-3;80C4-5;93B4-5;100B8-9</i>
*M(3)bb	spont	Mossige	3	medium Minute

^α 1 = Curry, 1939, DIS 12: 45; 2 = Mickey, 1963, DIS 38: 29; 3 = Mossige, 1946, DIS 20: 68; 4 = Oliver, 1939, DIS 12: 48.

M(3)124: see *M(3)95A*

M(3)B: see *M(3)95A*

M(3)be: see *M(3)96C*

***M(3)d**

location: 3-95.

discoverer: Bridges, 20b25.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 231.

phenotype: Part of digenic Minute. Produces no effect except when *M(2)d* is also heterozygous. Homozygote probably lethal. RK3.

M(3)f: see *M(3)100CF*

M(3)f: see *M(3)S35*

M(3)Fla: see *M(3)95A*

M(3)g: see *M(3)99E*

M(3)h: see *M(3)69E*

M(3)h^{S37}: see *M(3)66A*

M(3)i: see *M(3)67C*

M(3)j: see *M(3)96CF*

M(3)LS1: see *M(3)62A*

M(3)LS2: see *M(3)62F*

M(3)LS3: see *M(3)63B*

M(3)LS4: see *M(3)80*

M(3)LS5: see *M(3)85E*

M(3)q: see *M(3)67C*

M(3)Q-III: see *M(3)80*

M(3)S31: see *M(3)86D*

M(3)S32

location: 3- (not located).

origin: X-ray induced.

discoverer: Schultz, 33a5.

phenotype: Medium Minute. Most flies thickset. RK3.

M(3)S33: see *M(3)67C*

M(3)S34: see *M(3)76A*

***M(3)S35**

location: 3-64.

origin: X ray induced.

discoverer: Schultz, 33a11.

phenotype: Extreme Minute with small body. RK3.

alleles:

allele	origin	discoverer	comments
*M(3)S35¹	X ray	Schultz	3-64
*M(3)S35^{2α}		Moriwaki, 38f2	3-62.4

^α Synonym: *M(3)f*; *M(3)S35^f*.

References: Moriwaki, 1939, DIS 12: 50.

other information: Two mutants, both lost, mapped between 60 and 65, yet segmental deficiencies covering the region reveal no Minute phenotypes.

M(3)S36

location: 3- (not located).

origin: X ray induced.

discoverer: Schultz, 32k26.

phenotype: Variable phenotypes appear in stock; Minute and variegated for *ss*-like. Not studied. RK3.

M(3)S37: see *M(3)66A*

M(3)S38: see *M(3)69E*

M(3)S39: see *M(3)82BC*

M(3)Sp: see *M(3)96CF*

M(3)v: see *M(3)69E*

M(3>w: see *M(3)95A*

M(3)x: Minute (3) with C(3)x

location: 3- (on the left arm).

origin: Spontaneous in *In(3L)P*.

discoverer: Muller, 1929.

phenotype: Rather extreme Minute; expression reduced by *H*. RK3A.

M(3)y: see *M(3)69E*

M(4): see *Df(4)M101*

M(4)²: see *Df(4)M101-2*

M(4)³: see *Df(4)M101-3*

M(4)⁴: see *Df(4)M101-4*

M(4)101

location: 4-0.

references: Hochman, 1974, Cold Spring Harbor Symp. Quant. Biol. 38: 581-89.

phenotype: Medium Minute; viability good; development protracted; eclosion delayed 33 hr (Ferrus, 1975, Genetics 79: 589-99). *M(4)101/++* triplo-fours are non-Minute (Mohr, 1933, Hereditas 17: 317-32). Homozygous mutants die in embryonic stage (Farnsworth, 1951, Genetics 36: 550). RK2.

alleles: Formerly designated alleles nearly all deficiencies; only *M(4)101^{57g}* has normal polytenes and fails to uncover mutations in neighboring loci (Hochman, Gloor, and Green, 1964, Genetics 35: 109-26).

cytology: Placed in 101F2-102A5 on the basis of *Df(4)M101-63a = Df(4)101F2-101A1;101A2-5* (Hochman).

M(4)^{62e}: see *Df(4)M101-62e*

M(4)^{62f}: see *Df(4)M101-62f*

M(4)^{63a}: see *Df(4)M101-63a*

M(B): see *su(B)*

***m(Est6)*: modifier of Esterase 6**

location: 3-56.7.

origin: Spontaneous.

synonym: *m-est*, *M-est*.

references: Cochrane, 1976, Genetics 83: s16.

Cochrane and Richmond, 1979, Biochem. Genet. 17: 167.

phenotype: Two alleles; the dominant allele arbitrarily designated *m(Est6)*⁺. *m(Est6)* decreases the electrophoretic mobility of the products of some alleles, but not of others. Also modifies leucine amino peptidase, but not twelve other tested enzymes. Thought to change charge rather than conformation. Evidence that sialylation not involved.

M(g): see *e(g)*

***M(G6PD)*: Modifier of Glucose 6 Phosphate Dehydrogenase**

location: 1-65.

origin: Spontaneous.

synonym: *M*^{Zw}.

references: Komma, 1968, Biochem. Genet. 1: 229-37.

phenotype: A dominant modifier of the electrophoretic mobility of G6PD encoded by both *Zw*^A and *Zw*^B. Mobility faster in presence of *M(G6PD)* than in *M(G6PD)*⁺ homozygotes.

m(GPDH): see *r(αGPDH)*

M(Sd)*: Modifier of *Sd

location: 2-57 (right of *Rsp*; near *cn*).

origin: Spontaneous.

references: Hiraizumi, Martin, and Eckstrand, 1980, Genetics 95: 693-706.

phenotype: Enhances segregation distortion in genotypes heterozygous for *Sd*, *M(Sd)*, and *Rsp*^s; not the same as *E(Sd)*. Causes high levels of male infertility in genotypes that are heterozygous for *Sd* and *M(Sd)* and homozygous for *Rsp*^s; those males that are fertile produce very few offspring.

***ma*: maroon**

location: 3-49.7.

origin: Spontaneous.

discoverer: Bridges, 12c13.

references: 1918, Proc. Nat. Acad. Sci. USA 4: 316-18.

Bridges and Morgan, 1923, Carnegie Inst. Wash. Publ. No. 327: 53 (fig.).

phenotype: Eye color dull ruby, approaching wild type with age; classification slow. Larval Malpighian tubes pale yellow (Beadle, 1937, Genetics 22: 587-611). Eye color autonomous in transplant into wild-type host (Beadle and Ephrussi, 1936, Genetics 21: 230). Wild-type levels of aldehyde oxidase and xanthine dehydrogenase activity (Hickey and Singh, 1982, DIS 58: 74.)

alleles: *ma*⁴⁹, spontaneous (Oftedal, 1951, DIS 25: 69). RK2.

****Ma*: *Ma dominigene***

location: 1- (not located).

origin: Spontaneous.

discoverer: Goldschmidt, 1935.

references: Gardner, 1942, Univ. Calif. (Berkeley) Publ. Zool. 49: 95.

phenotype: In combination with *Ma*, *vg/+* is strongly scalloped. RK3.

***mab*: malformed abdomen**

location: 3- {47}.

phenotype: Tergites and sternites show defects in cuticle. Homozygous viable at 18° (wild-type) and 25° (mutant); lethal at 28° (Lewis, Kaufman, Denell, and Talerico, 1980, Genetics 95: 367-81).

allele	origin	discoverer	synonym	ref ^α
<i>mab</i> ¹	EMS	R. Lewis	<i>Ebmab</i> ¹	1, 2
<i>mab</i> ²	EMS	R. Lewis	<i>Ecmab</i> ²	1, 2

^α 1 = Cavener, Otteson, and Kaufman, 1986, Genetics 114: 111-23; 2 = Lewis, Kaufman, Denell, and Talerico, 1980, Genetics 95: 367-81.

cytology: Placed in 84B based on its inclusion in *Df(3R)Scr2 = Df(3R)84A4-5;84C1-4* but not *Df(3R)Win3 = Df(3R)84A4-5;84B1-2*.

macrofine: see *mf*

***mad*: many abnormal discs (A. Shearn)**

location: 3-78.6.

references: Shearn, Rice, Garen, and Gehring, 1971, Proc. Nat. Acad. Sci. USA 68: 2594-98.

Shearn, 1974, Genetics 77: 115-25.

phenotype: Homozygous lethal; labial and wing discs appear normal; eye-antenna, prothoracic, haltere, leg, and genital discs homeotically transformed. Disc and cell autonomous.

allele	synonym
<i>mad</i> ¹	<i>mad</i> ⁷⁰³
<i>mad</i> ²	<i>mad</i> ¹⁸⁰³
<i>mad</i> ³	<i>mad</i> ^{RO631}
<i>mad</i> ⁴	<i>mad</i> ^{SE420}
<i>mad</i> ⁵	<i>mad</i> ^{SH536}
<i>mad</i> ⁶	<i>mad</i> ^{VCS}
<i>mad</i> ⁷	<i>mad</i> ^{VD46}
<i>mad</i> ⁸	<i>mad</i> ^{VF284}
<i>mad</i> ⁹	<i>mad</i> ^{VF298}
<i>mad</i> ¹⁰	<i>mad</i> ^{VJ176}
<i>mad</i> ¹¹	<i>mad</i> ^{VK96}
<i>mad</i> ¹²	<i>mad</i> ^{VR9}
<i>mad</i> ¹³	<i>mad</i> ^{VU144}
<i>mad</i> ¹⁴	<i>mad</i> ^{VX8}
<i>mad</i> ¹⁵	<i>mad</i> ^{VZ410}
<i>mad</i> ¹⁶	<i>mad</i> ^{VZ415}

***mah*: mahogany**

location: 3-88.

discoverer: Beadle, 36b26.

references: Beadle and Ephrussi, 1937, Am. Nat. 71: 91-95.

phenotype: Eye color translucent brown in young flies, changing toward wild type and becoming dark brown with age. Eyes contain 77% normal red pigment and 102% normal brown pigment (Nolte, 1955, J. Genet. 53: 1-10). Larval Malpighian tubes wild type in color (Beadle, 1937, Genetics 22: 587-611). RK3.

***mal*: maroonlike**

location: 1-64.8 (Schalet, 1963, DIS 38: 82).

references: Chovnick, Finnerty, Schalet, and Duck, 1969, Genetics 62: 145-60.

Finnerty, Duck, and Chovnick, 1970, Proc. Nat. Acad. Sci. USA. 65: 939-46.

phenotype: Brownish eye color resulting from reduction in the red (drosoperin) pigments. Larval Malpighian tubes short, bloated, irregularly formed, and contain yellow to orange pteridine globules (Schwinck, 1960, DIS 34: 105). *mal* is nonautonomous for eye color in mosaics with wild-type tissue (Glassman, 1957, DIS 31: 121-22) and in transplants of *mal* eyes into wild-type hosts (Ursprung, 1961, Z. Vererbungsl. 93: 119-25). Activities of three molybdo-enzymes reduced or absent: aldehyde oxidase = AO (Courtright, 1967, Genetics 57: 25-39), pyridoxal oxidase = PO (Forrest, Hanley, and Lagowski, 1961, Genetics 46: 1455-63), and xanthine dehydrogenase = XDH (Forrest, Glassman, and Mitchell, 1956, Science 124: 725-26; Glassman and Mitchell, 1959, Genetics 44: 153-62). Measurements of cross reacting material (*e.g.*, Browder, Wilkes, and Tucker, 1982, Biochem. Genet. 20: 111-24, 125-32) show 75% and 50% normal levels of AO CRM in larval hemolymph and adult extracts respectively and 105% normal level of XDH CRM (see also Warner, Watts, and Finnerty, 1980, Mol. Gen. Genet. 180: 449-53). Activity of a fourth molybdo-enzyme, sulfite oxidase, is unaffected by *mal* (Bogart and Bernini, 1981, Biochem. Genet. 19: 929-46). Furthermore, unlike mutants in genes thought to be involved with the function of molybdenum cofactor, *e.g.* *cin* and *lxd*, the effects of *mal* not alleviated by administration of molybdenum; XDH cross reacting material (CRM) isolated from *mal* flies contains molybdenum (Andres, 1976, Eur. J. Biochem. 62: 591); *mal* flies contain high levels of molybdenum cofactor by Neurospora nitrate reductase activation assay (Warner and Finnerty, 1981, Mol. Gen. Genet. 184: 72-96). Accumulation of enzyme substrates (Forrest, Glassman, and Mitchell, 1956; Glassman and Mitchell, 1959; Glassman and McLean, 1962, Proc. Nat. Acad. Sci. USA 48: 1712-18) may account for the reported increase in uricase activity (Friedman, 1970, Genetics 68: s22). The absence of XDH activity renders *mal* flies sensitive to exogenously supplied purine (Glassman, 1965, Fed. Proc. 24: 1243), which has been used in selective schemes (Finnerty *et al.*, 1970); the cell autonomy of *mal* with respect to AO activity provides the basis of a staining procedure for differentiating *mal* from *mal*⁺ tissue in mosaics (Janning, 1972, Naturwissenschaften 59: 516-17). *mal* offspring of *mal*⁺ mothers appear normal in both eye color and Malpighian-tube morphology (Glassman and Mitchell, 1959; Schwinck, 1960); *mal*⁺ activity observed in germ line as AO activity (Marsh and Wieschaus, 1977, Dev. Biol. 60: 396-403) and maternally inherited XDH activity in *mal* offspring detectable until second day of pupal stage (Browder and Williamson, 1976, Dev. Biol. 53: 241-49). Maternal effect suppressed if offspring are also homozygous for *lxd* (Courtright, 1975, Mol. Gen. Genet. 142: 231-38). Interallelic complementation in females of constitution *mal*¹/*mal*^{F1}; eye color and Malpighian-tube morphology appear normal, but XDH activity about 10% normal (Glassman and Mitchell, 1959; Schwinck, 1960); complementation not seen in flies raised at 29° and reduced in flies that are also homozygous for *lxd* (Courtright, 1975, Mol. Gen. Genet. 142: 231-38); physical properties of XDH and AO altered in different heteroallelic combinations (Finnerty, McCarron, and Johnson, 1979, Mol. Gen. Genet. 172: 37-43; Finnerty and Johnson, 1979, Genetics

91: 696-722). *mal*¹, *mal*^{F1}, and *mal*^{F3} complement for eye color in all pairwise combinations; however, *mal*^{F1}/*mal*^{F3}/*mal*¹ is mutant. *mal* and *ry* extracts complement to produce XDH activity (Glassman, 1962, Proc. Nat. Acad. Sci. USA 48: 1491-97); they do not complement intercellularly *in vivo*, however, since reciprocal eye-disk or Malpighian-tube transplants reported to behave autonomously with respect to drosoperin formation (Schwinck, 1960; 1963, DIS 38: 87).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>mal</i> ¹	X ray	Oliver, 3011		2, 3, 9	IV
<i>mal</i> ²	X ray	Schalet, 1961		2, 10	II
<i>mal</i> ¹⁴	TEM			2, 12	I
<i>mal</i> ²⁰	TEM			2, 12	I
<i>mal</i> ²¹	EMS			2, 12	II
<i>mal</i> ²³	EMS			2, 12	I
<i>mal</i> ²⁴	TEM			2, 12	II
<i>mal</i> ²⁵	TEM			2, 12	I
<i>mal</i> ²⁶	TEM			2, 12	II
<i>mal</i> ²⁷	TEM			2, 12	II
<i>mal</i> ²⁸	TEM			2, 12	I
<i>mal</i> ²⁹	TEM			2, 12	
<i>mal</i> ³⁰	EMS			2, 12	II
<i>mal</i> ⁶⁰	DNA	Fahmy, 60j	<i>mal</i> ^{bz60j}	4	
<i>mal</i> ¹⁰⁶	X ray			2, 12	y+Y <i>mal</i> ¹⁰⁶ , I
<i>mal</i> ¹¹⁶	X ray			2, 12	y+Y <i>mal</i> ¹¹⁶ , III
<i>mal</i> ^{C1}	EMS			1	
<i>mal</i> ^{C2}	EMS			1	
<i>mal</i> ^{C3}	EMS			1	
<i>mal</i> ^{C4}	EMS			1	normal eye color, no XDH activity
<i>mal</i> ^{C5}	spont			1	leaky allele, I
<i>mal</i> ^{F1} γ	N mustard	Fahmy, 1954	<i>bz, mal</i> ^{bz}	2, 6, 7	III
<i>mal</i> ^{F2}	MMS	Fahmy	<i>mal</i> ^{bz} ^{56k}	2, 5	I
<i>mal</i> ^{F3}	ENU	Fahmy	<i>mal</i> ^{65c}	2, 5	V
<i>mal</i> ^{F4}	ENU	Fahmy	<i>bz</i> ^{65c}	2, 5	III
<i>mal</i> ^{F5}	CB3007 δ	Fahmy		6	
<i>mal</i> ^{F6}	CB3025 δ	Fahmy		6	
<i>mal</i> ^{F7}	CB3051 δ	Fahmy		6	
<i>mal</i> ^Q				8	I
<i>mal</i> ^S	spont	Schalet		11	

^α 1 = Bentley and Williamson, 1982, Can. J. Genet. Cytol. 24: 11-17; 2 = Chovnick, Finnerty, Schalet, and Duck, 1969, Genetics 62: 145-60; 3 = CP552; 4 = CP627; 5 = Fahmy and Fahmy; 6 = Fahmy and Fahmy, 1958, DIS 32: 68; 7 = Fahmy and Fahmy, 1958, Nature 184: 1927-29; 8 = Finnerty, McCarron, and Johnson, 1979, Mol. Gen. Genet. 172: 37-43; 9 = Oliver, 1935, DIS 3: 28; 10 = Schalet, 1961, DIS 38: 82; 11 = Schalet, 1986, Mutat. Res. 163: 115-44; 12 = Schalet and Finnerty, 1968, DIS 43: 65-66.

^β Complementation groups for eye color.

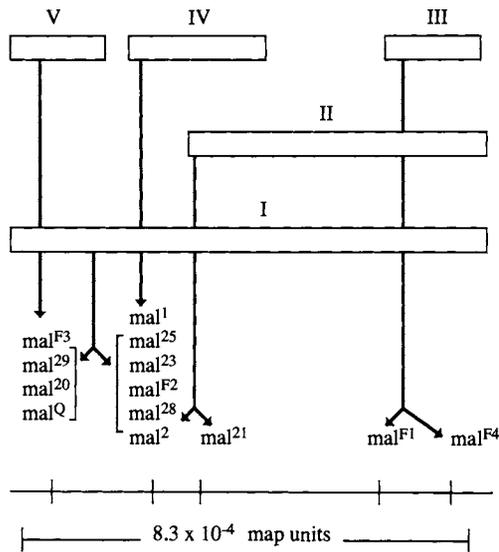
^γ *mal*^{F1} does not complement *mal*¹ when kept at 29°-30° throughout development; complementation occurs provided development proceeds at 25° during either of the two critical periods: the third quarter of the third larval instar or the middle of the pupal stage (Schalet, 1971, Mol. Gen. Genet. 110: 82-85).

^δ CB3007 = DL-p-N,N-di-(2-chloroethyl)aminophenylalanine.
CB3025 = L-p-N,N-di-(2-chloroethyl)aminophenylalanine.
CB3051 = ?

cytology: Placed in 19D2-3 based on its inclusion in the region of overlap of *Df(1)16-2-19 = Df(1)19A5;19D3* and *Df(1)16-3-35 = Df(1)19D2-3;19E34*.

other information: The alleles of *mal* have been mapped both by complementation for eye color and by recombination. Five complementation units have been defined (Chovnick, Finnerty, Schalet, and Duck, 1969, Genetics 62: 145-60), and *mal*^{C2} said to define a sixth (Bentley and Williamson, 1982, Can. J. Genet. Cytol. 24: 11-17). There is but a single lethal complementation group in

heteroallelic combinations raised on purine-enriched medium. Recombinational mapping utilized purine sensitivity to select for *mal*⁺ recombinants (Finnerty *et al.*, 1970). Maps co-linear.



Complementation map of *mal* alleles (groups I to V above) compared to genetic map; relative positions of mutants in each complementation group with respect to genetic map indicated (modified from Duck and Chovnick, 1974, *Genetics* 79: 459-66). Program by D. Conner.

Mal: Malformed

location: 2-44-50 (between *stw* and *cn*; Bridges inferred two loci, one near right end of 2R and one on chromosome 4).

origin: Spontaneous.

discoverer: Steinberg, 36k13.

references: 1937, *DIS* 7: 15, 20.

Baker and Tsai, 1977, *Dev. Biol.* 57: 221-25 (fig.).

phenotype: Heterozygote has either malformed pit in middle of eye or, more often, nick at front edge of eye and has bristle- or antenna-like outgrowth; outgrowth determined to be mirror image duplication of part of the orbital region including the orbital and fronto-orbital bristles; several sequential duplications sometimes observed. Penetrance low in heterozygote; supposedly enhanced by addition of extra brewer's yeast to medium. Homozygote shows larger nick and antennal outgrowth with up to 100% expression in *pr Mal* stock. RK3.

Malate dehydrogenase: see *Mdh*

male diplolethal: see *mdl*

Male Recombination: see *MR*

male-female-sterile: see *mfs*

male-specific lethal: see *msl*

male-sterile: see *ms*

Male-specific transcript: see *Mst*

maleless: see *mle*

Malformed: see *Mal*

malformed abdomen: see *mab*

Malic enzyme: see *Men*

mam: master mind (J.C. Hall)

location: 2-70.3.

synonym: *N-2G*.

references: Lehmann, Dietrich, Jiménez, and Campos-Ortega, 1981, *Wilhelm Roux's Arch. Dev. Biol.* 190: 226-29.

Lehmann, Jiménez, Dietrich, and Campos-Ortega, 1983, *Roux's Arch. Dev. Biol.* 192: 62-74.

Campos-Ortega, 1985, *Trends Neurosci.* 8: 245-50.

Smoller, Friedel, Schmid, Bettler, Lam and Yedvobnick, 1990, *Genes Dev.* 4: 1688-1700.

phenotype: Homozygous embryonic lethal; embryos display neural hyperplasia with compensatory epidermal hypoplasia; caused by failure of most ventral ectodermal cells to differentiate as epidermal cells rather than neuroblasts, as seen in *N*, *amx*, *bib*, *neu*, *Dl*, and *E(spl)*; *mam* tends to have less extreme neural hyperplasia than mutants at the other neurogenic loci. A similar diversion of cells from epidermigenic into neurogenic pathways seen to generate supernumerary peripheral nerve cells (Hartenstein and Campos-Ortega, 1986, *Roux's Arch. Dev. Biol.* 195: 210-21 (fig.)). When *mam* expressed in female germ cells and the ensuing embryos, neural hyperplasia is enhanced, but *mam*⁺ embryos from oocytes are normal [Jiménez and Campos Ortega, 1982, *Roux's Arch. Dev. Biol.* 191: 1901-201 (fig.)]. Homozygous clones in the eye display irregular ommatidial pattern characterized by lack of interommatidial bristles, enlarged facets with supernumerary retinular cells and reduced numbers of pigment cells; in the cuticle, clones homozygous for *mam*² are devoid of bristles [Dietrich and Campos-Ortega, 1984, *J. Neurogenet.* 1: 315-32 (fig.)]. *mam*¹ (formerly *N-2G*) heterozygotes occasionally exhibit apical wing nicking; not recorded for other alleles. Phenotype of homozygotes for null allele reduced by duplications for normal alleles of other neurogenic loci, *N*, *neu*, *Dl*, *E(spl)*, and *H*, but not *amx* or *bib* (de la Concha, Dietrich, Weigel, and Campos-Ortega, 1988, *Genetics* 118: 499-508).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>mam</i> ¹	spont	Ives. 41117	<i>N-2G</i>	3, 4	nicks; <i>In(2R)50C20;54B1</i> ; breakpoint near -27 kb
<i>mam</i> ²	EMS		<i>mam</i> ^{1B99}	1, 5, 6, 7	stronger allele
<i>mam</i> ³	EMS		<i>mam</i> ^{1F33}	7	
<i>mam</i> ⁴	EMS		<i>mam</i> ^{1IH57}	7	
<i>mam</i> ⁵	EMS		<i>mam</i> ^{1IJ14}	7	
<i>mam</i> ⁶	EMS		<i>mam</i> ^{1IL61}	7	
<i>mam</i> ⁷	EMS		<i>mam</i> ^{1IV06}	7	
<i>mam</i> ⁸	EMS		<i>mam</i> ^{1J113}	6, 7	stronger allele
<i>mam</i> ⁹	EMS		<i>mam</i> ^{1LA2}	7	
<i>mam</i> ¹⁰	EMS		<i>mam</i> ^{1L115}	7	
<i>mam</i> ¹¹	EMS	Wetter	<i>mam</i> ^{1Q19}		intermediate allele, deltas
<i>mam</i> ¹²	EMS	Wetter	<i>mam</i> ^{1Q78}		weak allele, deltas, nicks
<i>mam</i> ¹³	EMS	Wetter	<i>mam</i> ^{1μ12}		intermediate allele, deltas
<i>mam</i> ¹⁴	EMS	Wetter	<i>mam</i> ^{1μ97}		weak allele, deltas, nicks
<i>mam</i> ¹⁵	EMS	Wetter	<i>mam</i> ^{1μ112}		intermediate allele, deltas, nicks
<i>mam</i> ¹⁶	EMS	Wetter	<i>mam</i> ^{1μ127}		intermediate allele, deltas
<i>mam</i> ¹⁷	EMS	Wetter	<i>mam</i> ^{1μ129}		weak allele, deltas
<i>mam</i> ¹⁸	EMS	Wetter	<i>mam</i> ^{1μ168}		intermediate allele, deltas
<i>mam</i> ¹⁹	EMS	Wetter	<i>mam</i> ^{1μ196}		intermediate allele, deltas, nicks
<i>mam</i> ²⁰	EMS	Wetter	<i>mam</i> ^{1μ199}	2	weak allele

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>mam</i> ²¹	EMS	Wetter	<i>mam</i> ^{z3}		extreme allele, deltas
<i>mam</i> ²²	EMS	Wetter	<i>mam</i> ^{z9}		weak allele, deltas
<i>mam</i> ²³	EMS	Wetter	<i>mam</i> ^{z14}		strong allele
<i>mam</i> ²⁴	EMS	Wetter	<i>mam</i> ^{z33}		intermediate allele, deltas, nicks
<i>mam</i> ²⁵	EMS	Wetter	<i>mam</i> ^{z36}		weak allele, deltas, nicks
<i>mam</i> ²⁶	EMS	Wetter	<i>mam</i> ^{z47}		weak allele, deltas
<i>mam</i> ²⁷	EMS	Wetter	<i>mam</i> ^{z48}		weak allele, deltas
<i>mam</i> ²⁸	EMS	Wetter	<i>mam</i> ^{z62}		strong allele, deltas, nicks
<i>mam</i> ²⁹	X ray	Lehmann	<i>mam</i> ²¹¹¹		
<i>mam</i> ³⁰	X ray	Dietrich	<i>mam</i> ²¹⁷⁻⁴		
<i>mam</i> ³¹	X ray	Dietrich	<i>mam</i> ²²³⁻⁴		
<i>mam</i> ³²	X ray	Dietrich	<i>mam</i> ³¹⁵⁻⁵		
<i>mam</i> ³³	X ray	Dietrich	<i>mam</i> ⁴⁶⁶⁻¹		
<i>mam</i> ³⁴	X ray	Shrons	<i>mam</i> ¹³⁻²⁵		
<i>mam</i> ³⁵	X ray	Shrons	<i>mam</i> ¹²¹⁻¹⁰		<i>T(2;3)50C20-23;67</i>
<i>mam</i> ³⁶	X ray	Lehmann	<i>mam</i> ^{c2-4}	2	
<i>mam</i> ³⁷	X ray	Lehmann	<i>mam</i> ^{c7-9}		stronger allele
<i>mam</i> ³⁸	X ray	Lehmann	<i>mam</i> ^{c114-3}		stronger allele
<i>mam</i> ³⁹	X ray	Lehmann	<i>mam</i> ^{FX4}		stronger allele
<i>mam</i> ⁴⁰	P	Weigel	<i>mam</i> ^{KP1}		<i>P</i> element at 50C20-23
<i>mam</i> ⁴¹	HD ^γ		<i>HD2/3</i>	8, 9	11.5 to 23.5 kb (including exons 5-7) deleted
<i>mam</i> ⁴²	HD		<i>HD3/1</i>	8, 9	<i>In(2R)42C-D;50C-D</i> ; breakpoint near 0 ^δ
<i>mam</i> ⁴³	HD		<i>HD6/4</i>	8, 9	complex rearrangement;
<i>mam</i> ⁴⁴	HD		<i>HD10/6</i>	8, 9	<i>P</i> insert near -50 kb; at nucleotide 23 of exon 1
<i>mam</i> ⁴⁵	HD		<i>HD11/2</i>	8, 9	<i>In(2R)50C-D;57B</i> ; breakpoint at 0
<i>mam</i> ⁴⁶	HD		<i>HD13/6</i>	8, 9	<i>In(2R)42C-D;50C-D</i> ; breakpoint near 0
<i>mam</i> ⁴⁷	HD		<i>HD15/2</i>	8, 9	unstable allele, <i>P</i> excised
<i>mam</i> ⁴⁸	HD		<i>HD17/7</i>	8, 9	strong allele, <i>P</i> excised 146 bp deletion near -2.0 kb in exon 4 ^ε
<i>mam</i> ⁴⁹	<i>P-lacw</i>	Jan	<i>mam</i> ^{1E7}	8	<i>P-lacw</i> insert near -48 kb
<i>mam</i> ⁵⁰	<i>P-lacw</i>	Jan	<i>mam</i> ^{10E1}	8	<i>P-lacw</i> insert near -48 kb
<i>mam</i> ⁵¹	<i>P-lacw</i>	Jan	<i>mam</i> ^{2B11}	8	<i>P-lacw</i> insert near -15 kb

^α 1 = Dietrich and Campos-Ortega, 1984, J. Neurogenet. 1: 315-32; 2 = Hartenstein and Campos-Ortega, 1986, Roux's Arch. Dev. Biol. 195: 210-21; 3 = Ives, 1943, DIS 17: 50; 4 = Ives, 1957, DIS 31: 83; 5 = Jiménez and Campos-Ortega, 1982, Roux's Arch. Dev. Biol. 191: 191-201; 6 = Lehmann, Jiménez, Dietrich, and Campos-Ortega, 1983, Roux's Arch. Dev. Biol. 192: 162-74; 7 = Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Roux's Arch. Dev. Biol. 193: 267-82; 8 = Smoller, Friedel, Schmid, Bettler, Lam and Yedvobnick, 1990, Genes Dev. 4: 1688-1700; 9 = Yedvobnick, Smoller, Young, and Mills, 1988, Genetics 118: 483-97 (fig.).

^β "deltas" refers to the presence of delta-like widenings of the veins 2, 3, and 4 at the margins of the wings of heterozygotes. "nicks" refers to terminal notching of the wing margins of heterozygotes; penetrance 100% for *mam*¹, about 25% for other alleles.

^γ Eight alleles induced by hybrid dysgenesis in Harwich second chromosomes, which has *P*-elements at 42C-D, 50C-D, and 57B.

^δ 0 coordinate = the insertion point of the defective *P*-element at 50C-D in the Harwich strain; positive values to the right.

^ε Frameshift → product with first 377 residues normal followed by 69 amino acids from frameshifted sequence before termination after residue 446.

cytology: Maps at 50C20-23, by virtue of *P*-element insertion at this site, in *mam*⁴⁰; consistent with this is the fact that *mam*¹ is associated with *In(2R)G* = *In(2R)50C19;54B1*; and that *mam*³⁵ = *T(2;3)mam*³⁵ = *T(2;3)50C20-23;67* (Campos-Ortega).

molecular biology: Gene isolated by chromosome walking, physical mapping of breakpoints, and transposon tagging (Weigel, Knust, and Campos-Ortega, 1987, Mol. Gen. Genet. 207: 374-84; Yedvobnick, Smoller, Young, and Mills, 1988, Genetics 118: 483-97). Locus delimited to ca. 70 kb, within which *mam*-mutant lesions mapped over a 45 kb interval (cf. Weigel *et al.*; 1987

Smoller *et al.*, 1990). This region is rich in opa repeats (first noted by Weigel *et al.*, 1987), and regions encoding them are portions of *mam* exons; these are all contained within a 6.3 kb cDNA, cloned from an 8-12 hour library (Smoller *et al.*, 1990) probed with "unique-ORF"-containing (non-*opa*) genomic probes. Genomic sequence comprises seven exons (312, 127, 1010, 2073, 286, 1090, and 1435 bp) spanning 67 kb; transcription from left to right. Northern blots and *in situ* hybridizations (Smoller *et al.*, 1990) reveal complex patterns of *mam* mRNA expression: a maternal transcript; four embryonic ones (6.5-8.5 kb), two of which appear earlier than the other two, with all four waning by L1; transcripts return in late L3/early pupation; ubiquitous spatial expression in embryos seen during germ-band elongation, with neural-restricted signals observed later. Sequencing of the 6.3 kb cDNA revealed a 4788 bp ORF and predicts an 1596-amino-acid protein; there are 21 poly-Gln runs within the ORF, four poly-Gly, three poly-Asn, and one poly-Ala run; there are also 47 Gly-Gly doublets and three Gly-Val runs; the predicted protein contains relatively few charged residues. Antibodies generated using *mam*-fusion proteins detect a polypeptide of predicted size on Western blots and a nuclear signal in Schneider S2 cells and in whole mounts of early embryos (Smoller *et al.*, 1990).

other information: *N-2G* considered an allele based on its failure to complement other *mam* alleles and on the embryonic phenotype of such non-complementing *trans* heterozygotes. Interacts with *dx*, in that the great majority of *dx*^{ENU}/*Y*; *mam*¹⁰/*+* zygotes die as pupae (Xu and Artavanis-Tsakonas, 1990, Genetics 126: 665-77).

mam: mandarin

location: 3-50.5.

origin: Spontaneous.

references: Aparisi and Nájera, 1987, DIS 66: 13-14. 1988, DIS 67: 4-5, 5-6.

phenotype: Eye color bright orange.

***manikin*:** see *mn*

***many abdominal discs*:** see *mad*

***map*:** *midgut amylase pattern* (W.W. Doane)

Two closely linked genes that determine the pattern of alpha-amylase activity in three contiguous segments of the anterior portion of the adult midgut (gene *mapA*) and two segments of the posterior portion of the adult midgut (gene *mapP*). The short non-digestive middle midgut is without amylase activity in any genotype. *mapP* has three known alleles and *mapA* appears to have five since all 15 possible haplotypes have been reported (Doane, 1980, DIS 55: 36-39). In most cases, the pattern observed in the larval midgut is different from that seen in the adult of the same genotype. A pattern in which all three anterior segments and both posterior segments exhibit enzyme activity is designated 123-12 and one with no activity 000-00. The pattern observed is affected both by nutrition and adult age; for example, in some strains a 123-00 pattern may change to a 123-12 pattern at approximately 14 days of age in adults; in other strains, this change does not occur (Doane, Treat-Clemons, Gemmill, Levy, Hawley, Buchberg, and Paigen, 1983, Isozymes: Current Topics in Biological and Medical Research 9: 63-90). [Additional, as yet unnamed, genetic factors

control amylase patterns in the anterior midgut and posterior midgut of larvae (Treat-Clemons and Doane, 1983, Isozyme Bull. 16: 66; Klarenberg, Visser, Willemse, and Scharloo, 1986, Genetics 114: 1131-45); one is a *cis*-regulator of amylase activity in the anterior midgut and is located within 0.1 cM of *Amy* (Klarenberg *et al.*, 1986).

mapA: Anterior midgut amylase pattern

location: 2-82 (based on 7 recombinants between *Amy* and *mapA*).

synonym: *map-AMG*.

references: Doane, 1980, DIS 55: 36-39.

phenotype: The anterior adult midgut is divided into three segments which express discrete patterns of amylase activity.

alleles: Different *mapA* alleles exhibit enzyme activity in different combinations of the three segments of the anterior midgut. Presence of activity in the three regions is designated, from anterior to posterior, 1, 2, and 3; absence or near absence of activity in any region is designated 0; these notations appear as three-digit superscripts in *mapA* allelic designations: *mapA*⁰⁰⁰, *mapA*¹⁰⁰, *mapA*¹⁰³, *mapA*¹²⁰, and *mapA*¹²³.

mapP: Posterior midgut amylase pattern

location: 2-79 [based on 433 recombinants between *Amy* and *wt* (Klarenberg *et al.*, 1986); *mapP* also shown to lie to the left of *mapA* by crossovers between them (Doane, 1980) and to the left of *nw* (Doane, 1987, DIS 66: 49).

synonym: *map* (Abraham and Doane, 1978); *map-PMG* (Doane, 1980).

references: Abraham and Doane, 1978, Proc. Nat. Acad. Sci. USA 75: 4446-50.

Doane, 1980, DIS 55: 36-39.

Klarenberg, Visser, Willemse, and Scharloo, 1986, Genetics 114: 1131-45.

Doane, Treat-Clemons, Gemmill, Levy, Hawley, Buchberg, and Paigen, 1983, Isozymes: Current Topics in Biological and Medical Research 9: 63-90.

Treat-Clemons and Doane, 1983, Isozyme Bull. 16: 66.

Doane, 1987, DIS 66: 48.

Thompson and Doane, 1988, J. Cell Biol. 107: 331a.

Doane, Thompson, Norman, and Hawley, 1990, Isozymes: Structure, Function, and Use in Biology and Medicine, Prog. Clin. Biol. Res. 344: 19-48.

phenotype: Determines the level of amylase activity, amylase protein and amylase RNA in the posterior midgut. *Trans* regulation of amylase activity observed in adults (Abraham and Doane, 1978; Doane *et al.*, 1983, Klarenberg *et al.*, 1986) and in larvae of some strains (Treat-Clemons and Doane, 1983), but not others (Klarenberg *et al.*, 1986); nutritional control of *Amy* gene expression by dietary glucose repression, which is strain-specific in its magnitude (Hickey and Benkel, 1982, Biochem. Genet. 20: 1117-29), affects the level of amylase activity systemically in both the posterior midgut and the anterior midgut and can affect amylase activity patterns (Thompson and Doane, 1988; Doane *et al.*, 1990); similarly, dietary maltose, which is digested to glucose, can affect amylase activity patterns (Klarenberg *et al.*, 1986).

alleles: Two discrete regions of the larval and adult posterior midgut can express amylase activity on cornmeal-molasses-yeast food or a yeast diet with glucose containing sugars. Presence of activity in the anteriormost

region of the posterior midgut is designated 1 in the superscript, and activity in the region just behind the first region is designated 2; absence of activity in either region is designated 0. The following alleles have been recorded by Doane (1980): *mapP*⁰⁰, *mapP*¹⁰, and *mapP*¹² [synonyms *map*^A, *map*^B, and *map*^C, respectively (Abraham and Doane, 1978)].

Map205: Microtubule-associated protein 205 kd

location: 3- {105}.

references: Goldstein, Laymon, and McIntosh, 1986, J. Cell. Biol. 102: 2076-87.

phenotype: Encodes a 205 kd microtubule-associated protein, which by immunostaining can be localized to cytoplasmic microtubules and to the mitotic spindle.

cytology: Placed in 100E-F by *in situ* hybridization.

molecular biology: Genomic sequence isolated by screening an expression library with antibody. The genomic sequence hybridizes to a 6 kb mRNA on Northern blots.

mar: metaphase arrest

location: 3-{84}.

origin: X ray induced.

references: González, Molina, Casal, and Ripoll, 1989, Genetics 123: 371-77.

phenotype: Hemizygotes die as third-instar larvae; cells in larval brain have highly condensed X-shaped chromosomes, a mitotic index twice that of wild type, and a thirty-fold reduction in the ratio of anaphases to metaphases. Only about 1% of cells are tetraploid, the remainder remaining diploid. Complements *asp*, which is just to the left.

cytology: Placed in 96B based on its association with *T(Y;3)B97 = T(Y;3)h3;96B*, as indicated by the *mar* phenotype of *T(Y;3)B197Df(3R)LI6 = Df(3R)96A1-10;96E*.

Mar: Margin

location: 2-72.

origin: Induced by ethyl methanesulfonate.

references: Whittle, 1977, DIS 52: 2.

phenotype: Heterozygotes exhibit scalloping of wing margins; have gaps in triple row and double row of bristles and marginal hairs; wing outline sometimes ragged. Similar to *Bx*^G; penetrance incomplete. Homozygous lethal.

marionette: see *mrn*

maroon: see *ma*

maroonlike: see *mal*

Mas: see *dsx*^M

Masculinizer: see *dsx*^M

master mind: see *mam*

mat(2)cell: maternal lethal (2) cellularization defects (T. Schüpbach)

A series of ethyl methanesulfonate-induced maternal-effect-lethal female-sterile mutations on the second chromosome, which all result in the same phenotype. Embryos from homozygous females show variable defects in cellularization at the blastoderm stage; they form cuticles with holes, variable head defects, and segment fusions.

references: Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

locus	genetic location	cytological location	included in
<i>mat(2)cellHK35</i>	2-54		
<i>mat(2)cellPK42</i>	2-60		
<i>mat(2)cellPH65</i>	2-67		
<i>mat(2)cellPQ49</i>	2-12		
<i>mat(2)cellQA13</i>	2-64		
<i>mat(2)cellQC13</i>	2-35		
<i>mat(2)cellQE1</i>	2-54		
<i>mat(2)cellQL46</i>	2-67		
<i>mat(2)cellQQ55</i>	2-68		
<i>mat(2)cellRE43</i>	2-57	42C1-43F8	<i>Df(2R)pk78s</i>
<i>mat(2)cellRH36</i>	2-31		
<i>mat(2)cellRQ41</i>	2-86		

mat(2)dorsal: see *dl*

mat(2)ea: maternal lethal (2) early arrest
(T. Schüpbach)

A series of EMS induced maternal-effect-lethal, female-sterile mutations on the second chromosome, which all result in the same phenotype: Homozygous females lay eggs which show no signs of development when observed under transmitted light in a stereomicroscope.

references: Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

locus	genetic location	cytological location	included in	excluded form
<i>mat(2)eaPB28</i>	2-72	51A1-B4	<i>Df(2R)48</i>	
<i>mat(2)eaPGP44</i>	2-60			
<i>mat(2)eaQA26</i>	2-61			
<i>mat(2)eaQD68</i>	2-78			
<i>mat(2)eaQM47</i>	2-40	31B-32A	<i>Df(2L)J27</i>	
<i>mat(2)eaRL4</i>	2-41			
<i>mat(2)eaRS32</i>	2-17	25D7-E1	<i>Df(2L)cl¹</i>	<i>Df(2L)cl⁷</i>
<i>mat(2)eaRU28</i>	2-54			

mat(2)N: maternal lethal (2) Notchlike
(T. Schüpbach)

location: 2- {97}.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

phenotype: Maternal-effect lethal, female sterile.

Embryos from homozygous mothers form only a piece of dorsal cuticle, similar to the zygotic lethal Notch (*N*).

alleles:

allele	origin	synonym
<i>mat(2)N¹</i>	EMS	<i>mat(2)N^{RA}</i>
<i>mat(2)N²</i>	EMS	<i>mat(2)N^{QI}</i>
<i>mat(2)N³</i>	EMS	<i>mat(2)N^{RQ}</i>

cytology: Placed in either 57B5-14, or in 57D8-58B, since uncovered by *Df(2R)D17 = Df(2R)57B5;58B1-2*, but not by *Df(2R)PL3 = Df(2R)57B20;59D8-9* (cytology according to O'Donnell, Boswell, Reynolds, and MacKay, 1989, Genetics 121: 273-80).

other information: All three alleles are semilethal over *Df(2R)D17*.

mat(2)syn: maternal lethal (2) syncytial blastoderm arrest (T. Schüpbach)

A series of EMS-induced maternal-effect lethal mutations on the second chromosome, which all result in the same phenotype: Homozygous females lay eggs in which a narrow halo of clear cytoplasm appears around the time when nuclei would be expected to migrate to the egg periphery, but no cellularization takes place.

references: Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

locus	genetic location	cytological location	included in
<i>mat(2)synHB5</i>	2-49		
<i>mat(2)synHI10</i>	2-40		
<i>mat(2)synHK21</i>	2-65		
<i>mat(2)synPA75</i>	2-59		
<i>mat(2)synPJ50</i>	2-{40}	31B-32A	<i>Df(2L)J27</i>
<i>mat(2)synPL63</i>	2-72	51E3-52D1	<i>Df(2R)TES1D-18</i>
<i>mat(2)synQF75</i>	2-63		
<i>mat(2)synRE48</i>	2-24		
<i>mat(2)synSE10</i>	2-41		

mat(3)1

location: 3-54.8.

origin: Induced by ethyl methanesulfonate.

discoverer: Rice.

references: Rice and Garen, 1975, Dev. Biol. 43: 277-86 (fig.).

Regenass and Bernard, 1978, Mol. Gen. Genet. 164: 85-91.

1980, Wilhelm Roux's Arch. Dev. Biol. 188: 127-32.

phenotype: Maternal-effect lethal; embryos produced by homozygous females produce pole cells, but syncytial blastoderm fails to cellularize. Pole cells from defective *mat(3)1/+* embryos of *mat(3)1* mothers functional when transplanted into normal hosts (Regenass and Bernard).

mat(3)3

location: 3-45.3.

origin: Induced by ethyl methanesulfonate.

discoverer: Rice.

references: Rice and Garen, 1975, Dev. Biol. 43: 277-86 (fig.).

phenotype: Maternal-effect lethal; embryos produced by homozygous females produce pole cells, but syncytial blastoderm fails to cellularize completely; the posterior-dorsal 30% of the surface fails to form cells and is separated from the remainder of the blastoderm surface by a belt of incompletely cellularized nuclei. Mutant phenotype expressed in females kept at 29°; females kept at 25° are fertile and produce normal offspring; temperature-sensitive period during the last 12 hr of oogenesis.

mat(3)6: see *spg*

maternal haploid: see *mh*

maternal lethal: see *mat*

matt brown: see *mtb*

***mb: minus bar**

location: 3-43.4.

discoverer: Nordenskiöld, 33a30.

references: 1934, DIS 2: 7.

phenotype: Modifies Bar in such a way that *B/B* resembles

B/+, and *B/+* appears almost wild type; *B* male modified to resemble *B*¹. Homozygous female highly infertile. RK3.

***mbd: mushroom body-deranged* (J.C. Hall)**

location: 1-56.

origin: Induced by ethyl methanesulfonate.

synonym: *mbd*^{K565}.

references: Heisenberg, 1980, Development and Neurobiology of *Drosophila* (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York, pp. 373-90.

Technau and Heisenberg, 1982, Nature (London) 295: 405-07.

Borst and Heisenberg, 1982, J. Comp. Physiol. 147: 479-84.

phenotype: Most axons in peduncles of mushroom bodies in dorsal brain absent; no detectable lobes associated with these bodies (cf. alpha, beta, and gamma lobes of wild-type); posterior calyces of mushroom bodies enlarged. During post-larval mushroom body regeneration (on the first day of pupation), axons roll up at periphery of brain; other parts of mutant brains appear normal. Mushroom body abnormalities are variable; most behaviors of mutant adults are normal, including basic ability to discriminate between different odors; in tests using olfactory stimuli, learning in third larval instar is normal, but olfactory learning in mutant adults is aberrant (Heisenberg, Borst, Wagner and Byers, 1985, J. Neurogenet. 2: 1-30). Penetrance of the morphological phenotype diminishes rapidly under normal culturing, but does not wane when maintained in heterozygous condition.

cytology: Maps within 17A-18A2, based on uncovering by *Df(1)N19*.

***mbm: mushroom-body-miniature* (J.C. Hall)**

location: 2-{0} (inferred from cytology; also, maps to distal 2L meiotically).

origin: Induced by ethyl methanesulfonate.

discoverer: H. Heisenberg and K. Fishbach (from mutagenized second chromosome provided by C. Nüsslein-Volhard).

synonym: *mus: mushroom-bodies-small*, a preempted symbol.

references: Heisenberg, Borst, Wagner and Byers, 1985, J. Neurogenet. 2: 1-30.

phenotype: Abnormally small calyces associated with mushroom bodies in dorsal brain; peduncles and lobes are thin or missing, owing to degeneration of Kenyon cell fibers during third larval instar; these defects are essentially limited to mutant females. Mushroom bodies appear nearly normal in brains of *mbm* males. Osmotopotaxis (re odor discrimination) is essentially normal in females; mutant females (larvae or adults) are defective in learning tests involving odors, electric shock, or sugar, whereas males are normal or less defective.

cytology: Maps to 21B8-D1, because *mbm* is uncovered by *Df(2L)al = Df(2L)21B8-C1;21C8-D1*.

alleles: Three mutant alleles: *mbm*^{N337}, *mbm*^{K1}, *mbm*^{K7} (see "other information").

other information: All three *mbm* alleles, when heterozygous with *Df(2L)al*, lead to stronger mutant phenotypes than mutant homozygotes or heteroallelic combinations; *mbm*^{K1}/*mbm*^{K7} causes a very weak (i.e., near normal) mushroom body defect. *mbm*^{K1} and *mbm*^{K7} are from stocks originally termed *Df(2L)net*^{K1} and *Df(2L)net*^{K7};

each of these turns out not to be a deletion, i.e. they appear to be cytologically normal, but the former is mutant for *net*, *mbm* and a vital gene, as is the latter, though the lethal here complements that on the *K1* chromosome. In both of these stocks, the various abnormalities are separable by recombination.

***mbmB: mushroom-body-miniature-B* (J.C. Hall)**

location: 2-31.

origin: Induced by ethyl methanesulfonate.

synonym: *rem: reduced-mushroom-bodies*; *mbmB*^{N806}.

references: Heisenberg, Borst, Wagner and Byers, 1985, J. Neurogenet. 2: 1-20.

phenotype: Phenotype similar to that of *mbm*, but less extreme, and mushroom body defect occurs in both males and females; learning is weak in tests using olfactory stimuli.

***mbmC: mushroom-body-miniature-C* (J.C. Hall)**

location: 2-35.

origin: Induced by ethyl methanesulfonate.

synonym: *opa: olfactory pathway*, a preempted symbol; *mbmC*^{N28}.

references: Heisenberg, Borst, Wagner and Byers, 1985, J. Neurogenet. 2: 1-30.

phenotype: Phenotype similar to that of *mbm*; also, the pair of antennal lobes differ in volume in most *mbmC* adults (i.e. left smaller than right, or vice versa); learning very poor in tests of females, using olfactory stimuli.

****mbs: miniature blistered***

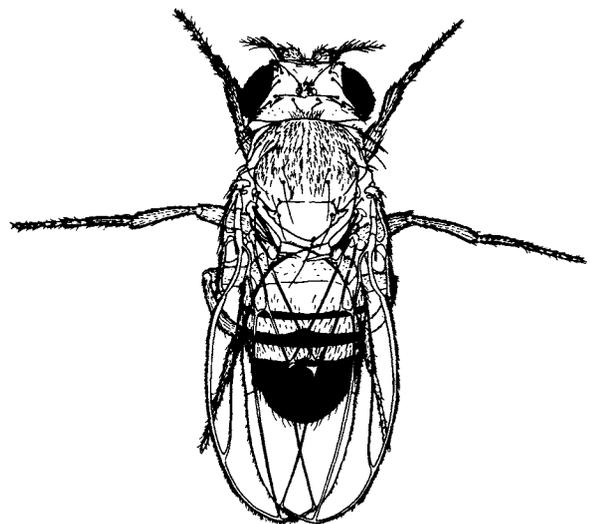
location: 2-56.

origin: Spontaneous.

discoverer: Neel, 41c13.

references: 1942, DIS 16: 51.

phenotype: Wings small, curled, blistered, and plexate. Bristle positions irregular and bristles often bent and twisted. Viability and fertility poor. RK3.



mc: microchaete

Edith M. Wallace, unpublished.

mc: microchaete

location: 1-54.0.

origin: X ray induced.

discoverer: Demerec, 28f20.

synonym: *tb-53*.

references: 1935, DIS 3: 13.

phenotype: Hairs on thorax fewer than wild type, more irregular, and frequently doubled. Bristles smaller, more sparse on scutellum, and occasionally on head. Eyes rough. Wings ovoid and short; marginal bristles disarranged. Abdominal sclerites ridged. RK1.

***mc²**

origin: Induced by D-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3026).

discoverer: Fahmy, 1955.

synonym: *mc-like*.

references: 1958, DIS 32: 71.

phenotype: Thoracic hairs irregularly distributed, occasionally reduced in number. Bristles small, sparse on scutellum. Eyes small and rough. Wings ovoid and short. Tergites in female sometimes disarranged. Viability and fertility good in both sexes. *mc²/mch* is wild type. RK2.

other information: Allelism inferred from location of *mc²* at 52.1 and from phenotype.

Mc: Microcephaluslocation: 3-59.0 (about 0.2 unit to the right of *bx*).

origin: Spontaneous.

references: Bateman, 1944, DIS 18: 40.

1945, DIS 19: 47.

phenotype: Eyes of heterozygote small or absent. Scutellars curve upward. Viability and fertility good. Homozygote usually more extreme than heterozygote but not reliably distinguishable. *ale Mc* homozygotes completely eyeless; fertile except when crossed to each other (Golubovsky and Zakarov, 1972, DIS 49: 112). Viability of homozygote varies from 100 down to 40%. RK1A.

cytology: Associated with a minute tandem repeat of one or more bands in 89E7-11; partially restores fertility to *Df(3R)P9* heterozygotes [Lewis, 1978, Nature (London) 276: 565-70]. Duplication appears to include at least a portion of the *AbdB* segment of *BXC* (Struhl and White, 1985, Cell 43: 507-19).

mc-like: see *mc²****mch: minute chaetae**

location: 1-52.0.

origin: Induced by methyl methanesulfonate (CB. 1540).

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 87.

phenotype: Extremely short, fine bristles. Hairs and body also small; delayed eclosion. Male viable and fertile. *mch/mc²* is wild type. RK2.

other information: One allele each induced by CB. 1246, CB. 1356, and CB. 3026.

Mcp: see *BXC****md: melanotic lesions**

location: 3-38.0.

origin: Found in experiments using benzopyrene.

discoverer: Gowen, 1933.

phenotype: Lesions occur in many places throughout head,

thorax, and abdomen. RK3.

***mdg: midgoid**

location: 1-64.7.

origin: Induced by D-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3026).

discoverer: Fahmy, 1955.

references: 1958, DIS 32: 71.

phenotype: Small in all dimensions; frequently underpigmented. Male infertile; viability about 20% wild type. RK3.

Mdh1: Malate dehydrogenase

location: 2-37.2.

synonym: *Mdh2* (Grell, 1969, DIS 44: 47); *MdhD* (Johnson and Schaeffer, 1973, Biochem. Genet. 10: 149-63); *sMdh* (Hay and Armstrong, 1976, Insect Biochem. 6: 367-76); *cMdh* (Voelker, Ohnishi, and Langley, 1979, Biochem. Genet. 17: 947-56).

references: Grell, 1969, DIS 44: 47.

O'Brien, 1969, DIS 44: 42, 113.

1973, Biochem. Genet. 10: 191-205.

Johnson and Schaeffer, 1973, Biochem. Genet. 10: 149-63.

Voelker, Ohnishi, and Langley, 1979, Biochem. Genet. 17: 947-56.

phenotype: The structural gene for the cytoplasmic, NAD-dependent malate dehydrogenase = (S)-Malate: NAD⁺ oxidoreductase [MDH1 (EC 1.1.1.37)]. Extracts from homozygotes produce two bands of activity plus a lighter, more electronegative band on gels; heterozygotes produce hybrid bands, indicating that the enzyme is a dimeric molecule. Subunit molecular weight estimated at 30,000 Daltons. Not necessary for survival, since *Mdh1ⁿ¹/Df(2L)J* is viable. Purification and biochemical characterization carried out by McReynolds and Kitto (1970, Biochim. Biophys. Acta 198: 165-75), Hay and Armstrong (1976, Insect Biochem. 6: 367-76), and Alhlotis (1979, Comp. Biochem. Physiol. 62B: 375-80).

alleles: For the electrophoretic variants, the Research Triangle Park conventions (1978, DIS 53: 117) are used; superscript 4 designates the most common allele; superscripts <4 migrate more slowly and >4 more rapidly than the most common allele. Null alleles all derivatives of *Mdh1⁴*.

allele	origin	synonym	ref ^α	comments ^β
<i>Mdh1²</i>	spont	<i>Mdh^A</i>	1, 2	rare allele
<i>Mdh1^{2a}</i>	spont		7	derivative of <i>Mdh1⁴</i>
<i>Mdh1⁴</i>	spont	<i>Mdh2^S</i>	4	common allele;
		<i>Mdh1^A</i>	8	<i>p</i> > 0.90
		<i>Mdh1^B</i>	2	
<i>Mdh1⁶</i>	spont	<i>Mdh2^V</i>	4	rare allele
		<i>Mdh1^B</i>	8	
		<i>sMdh²</i>	5	
		<i>Mdh^C</i>	2	
<i>Mdh1ⁿ¹</i>	γ ray	<i>cMdh^{n-*}10069</i>	9	in SM1; CRM-; dimer-
<i>Mdh1ⁿ²</i>	γ ray	<i>cMdh^{n-*}10081</i>	9	in SM1; CRM+; dimer-
<i>Mdh1^{nNC1}</i>	spont		3, 6	residual activity; dimer-
<i>Mdh1^{nNC2}</i>	spont		3, 6	residual activity; dimer-

^α 1 = Alhlotis, 1974, DIS 51: 88; 2 = Alhlotis, 1979, Comp. Biochem. Physiol. 62B: 375-80; 3 = Burkhart, Montgomery, Langley, and Voelker, 1984, Genetics 107: 295-306; 4 = Grell, 1969, DIS 44: 47; 5 = Hay and Armstrong, 1976, Insect Biochem. 6: 367-76;

6 = Langley, Voelker, Leigh Brown, Ohnishi, Dickson, and Montgomery, 1981, *Genetics* 99: 151-56; 7 = Mukai and Cockerham, 1977, *Proc. Nat. Acad. Sci. USA* 74: 2514-17; 8 = O'Brien, 1969, *DIS* 44: 42, 113; 9 = Racine, Langley, and Voelker, 1980, *Environ. Mutagen.* 2: 167-77.

β CRM = cross-reacting material; dimer- = no evidence of dimer formation in heterozygotes with *Mdh1*⁴ or *Mdh1*⁶.

cytology: Placed in 31B-E based on its being deleted by *Df(2L)J2 = Df(2L)31B;32A* and included in *Dp(1;Y)B31 = Dp(1;Y)27D;31E* (Voelker, Ohnishi, and Langley).

Mdh2

location: 3-62.6 [based on 21 recombinants between *sr* (3-62.0) and *gl* (3-63.1)].

synonym: *mMdh*.

references: Voelker, Ohnishi, and Langley, 1979, *Biochem. Genet.* 17: 947-56.

phenotype: Structural gene for the mitochondrial NAD-dependent malate dehydrogenase = (S)-Malate: NAD+ oxidoreductase [MDH2 (EC 1.1.1.37)].

alleles: *Mdh2*⁴ is the common allele; *Mdh2*⁶, which is more electronegative, was encountered only once.

cytology: Placed in 90C2-91A3 based on its inclusion in *Df(3R)P14 = Df(3R)90C2-D1;91A2-3*.

Mdh-NADP: see *Men*

mdl: male diplolethal

location: 1-{6}; between *cho* and *bi*.

references: Steinmann-Zwicky and Nöthiger, 1985, *Cell* 42: 877-87.

Steinmann-Zwicky, 1988, *EMBO Journal* 7: 3889-98.

phenotype: Locus inferred from the rescue of males hyperploid for the distal fourth of the X chromosome, which are normally inviable, by substitution of *Df(1)HC244 = Df(1)3E8;4F11-2* for the normal X. Lethality of the duplication observed in *Sxl*⁺ males but survival of such males carrying *Sxl*^{fl} reported.

cytology: Placed in 4A based on inclusion of the inferred gene in *Df(1)cho2 = Df(1)3E;4A* but not *Df(1)cho5 = Df(1)3D;4A*.

***me: focal melanosis**

location: 1-29.0.

origin: X ray induced.

discoverer: Gowen, 1928.

references: 1934, *Arch. Pathol.* 17: 638-47 (fig.).

1934, *Cold Spring Harbor Symp. Quant. Biol.* 2: 128-36 (fig.).

phenotype: Melanotic degeneration occurs at junction of tibia and femur. Lethal at end of pupal stage or shortly after eclosion. RK2.

Me: Moirè

location: 3-19.2 (to the left of *ju*; based on location of *Me*^{65d}).

synonym: *Mo*.

phenotype: Eye has watered-silk, shimmering, iridescent pattern owing to a ring of six flecks around normal fleck. Eye color brownish and translucent; 79% normal red pigment and 85% normal brown pigment (Nolte, 1955, *J. Genet.* 53: 1-10). Larval Malpighian tubes considerably lighter in color than normal but mutant classifiable with difficulty (Brehme and Demerec, 1942, *Growth* 6: 351-56). Contains a modifier of dominance of *dp* such that *dp/+*; *Me/+* has truncated wings. Classifiable in single

dose in triploids (Schultz, 1934, *DIS* 1: 55). Homozygous lethal. *Me/In(3L)P* is viable. RK1A.

allele:

allele	origin	discoverer	ref ^α	comments
<i>Me</i> ¹	X ray	Muller, 1929	3, 4, 5	
* <i>Me</i> ²	X ray	Moore, 1929	3	<i>T(2;3)Me</i>
<i>Me</i> ^{65d}	EMS	E.H. Grell 65d	2	light bristle tips
* <i>Me</i> ^{So}		Sytko	1	<i>T(2;3)Me</i> ^{So}

^α 1 = Agol, 1936, *DIS* 5: 7; 2 = CP627; 3 = Glass, 1933, *J. Genet.* 28: 69-112; 4 = Glass, 1934, *Am. Nat.* 68: 107-14; 5 = Muller, 1930, *J. Genet.* 22: 299-334 (fig.).

cytology: Placed in region 64C12-65E1 on the basis of its inclusion in *Df(3L)Vn = Df(3L)64C12-D1;65D2-E1* (Mohr, 1938, *Avh. Nor. Vidensk.-Akad. Oslo, Mat. Naturvidensk. Kl. No. 4: 1-7*). Associated with *In(3L)P = In(3L)63C;72E1-2* (Morgan, Bridges, and Schultz, 1937, *Year Book - Carnegie Inst. Washington* 36: 301).

***meg: megaoculus**

location: 1-61.9.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).

discoverer: Fahmy, 1954.

references: 1958, *DIS* 32: 71.

phenotype: Eyes large, abnormally shaped, and rough. Wings abnormally shaped and sometimes extremely small. Wing surface irregularly curved. Inner margin removed to various degrees and venation abnormal. Viability good; both sexes infertile. RK2.

other information: One allele induced by CB. 3025.

mei-1: meiotic mutant 1 (J. Valentin)

location: 3-45-55.

references: Valentin, 1973, *Hereditas* 75: 5-22.

Baker, Carpenter, Esposito, Esposito, and Sandler, 1976, *Ann. Rev. Genet.* 10: 53-134.

phenotype: Reduces exchange on the X chromosome in females by approximately 50% but has little or no effect on autosomal recombination. Recombination is decreased most severely in the central region of the X with more normal frequencies occurring proximally and distally. X chromosome nondisjunction is increased only slightly in *mei-1* females, while the fourth chromosomes disjoin quite regularly.

mei-9 (R.S. Hawley)

location: 1-6.5.

references: Baker and Carpenter, 1972, *Genetics* 71: 255-86.

Carpenter and Sandler, 1974, *Genetics* 76: 453-75.

Boyd, Golino, and Setlow, 1976, *Genetics* 84: 527-44.

Smith, 1976, *Mol. Gen. Genet.* 149: 73-85.

Nguyen and Boyd, 1977, *Mol. Gen. Genet.* 158: 141-47.

Baker, Carpenter, and Ripoll, 1978, *Genetics* 90: 531-78.

Baker and Smith, 1979, *Genetics* 92: 833-47.

Carpenter, 1979, *Chromosoma* 75: 259-92.

Gatti, 1979, *Proc. Nat. Acad. Sci. USA* 76: 1377-81.

Graf, Vogel, Biber, and Wurgler, 1979, *Mut. Res.* 59: 129-33.

Lutken and Baker, 1979, *Mut. Res.* 61: 221-27.

Baker, Gatti, Carpenter, Pimpinelli, and Smith, 1980. *DNA Repair in Eukaryotes* (Generoso, Shelby, and deSerres, eds.). Plenum Press, New York, London, pp. 189-208.

Lawlor, 1980, *DIS* 55: 81.

Smith, Snyder, and Dusenberry, 1980. *DNA Repair in Eukaryotes* (Generoso, Shelby, and deSerres eds.). Plenum Press, New York, London, pp. 175-88.

Carpenter, 1982, *Proc. Nat. Acad. Sci. USA* 79: 5961-65.

phenotype: *mei-9* alleles confer sensitivity to mutagens as a consequence of a defect in excision repair (Boyd *et al.*, 1976; Nguyen and Boyd, 1977). This defect in DNA repair is also manifested by a high frequency of mitotic chromosome breakage and instability (Baker *et al.*, 1978; Gatti, 1979). For example, larval neuroblasts of *mei-9/Y* males display a high frequency of spontaneous chromosome breaks in both the eu- and heterochromatin (Gatti, 1979). Females homozygous for *mei-9* show greatly reduced levels of meiotic exchange. However, the residual exchanges are distributed as in wild-type and chiasma interference is maintained. *mei-9* is thus considered to be defective in the exchange process itself, rather than in the establishment of the preconditions for exchange (Carpenter and Sandler, 1974). *mei-9^a* and *mei-9^b* have also been assayed with respect to their effects on gene conversion at the *rosy* locus (Carpenter, 1982). Although neither allele reduces the frequency of gene conversion events, both produce post-meiotic segregation events (*i.e.*, mosaic progeny) at high frequency. Thus, the recombinational phenotype of *mei-9* involves two components, namely a decrease in the frequency of heteroduplex repair and a decrease in the frequency of reciprocal exchange. At the ultrastructural level, both synaptonemal complex morphology and the number and distribution of recombination nodules are normal in *mei-9* females (Carpenter, 1979); but see Boyd *et al.* (1976). As a consequence of the decreased frequency of reciprocal exchange, *mei-9* females display greatly elevated frequencies of meiotic nondisjunction and chromosome loss (Baker and Carpenter, 1972). Meiotic chromosome behavior in males is not affected. Nor is there any effect of *mei-9* on spontaneous recombination in males (Lutken and Baker, 1979). Neither hypermutable to alkylation nor deficient in excision repair (Smith and Dusenberry, 1988, *Mechanisms and Consequences of DNA Damage Processing*, Alan R. Liss, Inc., pp. 251-55).

alleles: Baker and Smith (1979) demonstrated that the *mus-110^{AT1}*, *mus-110^{AT2}*, and *mus-110^{AT3}* mutations (Smith 1976) are alleles of *mei-9*. These mutations have been renamed as *mei-9^{A1}*, *mei-9^{A2}*, and *mei-9^{A3}*, respectively. *mei-9^{L1}*, also known as *mut¹⁵⁹*, was identified by Graf *et al.* (1979).

allele	reference	mutagen sensitive	reduces exchange	mitotic instability ^{2, 4, 6}
<i>mei-9^a</i>	1, 5	+	+	+
<i>mei-9^b</i>	1	+	+	+
<i>mei-9^{A1}</i> β	4, 9	+	+	+
<i>mei-9^{A2}</i>	9	+		+
<i>mei-9^{A3}</i>	9	+		+
<i>mei-9^{D1}</i>	8	+	+	
<i>mei-9^{D2}</i>	3, 8	+	+	
<i>mei-9^{D3}</i>	3, 8	+	+	
<i>mei-9^{D4}</i>	3, 8	+	+	
<i>mei-9^{L1}</i>	7	+	+	

^α 1 = Baker and Carpenter, 1972, *Genetics* 71: 255-86; 2 = Baker, Carpenter, and Ripoll, 1978, *Genetics* 90: 531-78; 3 = Baker, Gatti, Carpenter, Pimpinelli, and Smith, 1980, *DNA Repair in Eukaryotes* (Generoso, Shelby, and deSerres, eds.). Plenum Press, New York,

London, pp. 189-208; 4 = Baker and Smith, 1979, *Genetics* 92: 833-47; 5 = Boyd, Golino, and Setlow, 1976, *Genetics* 84: 527-44; 6 = Gatti, 1979, *Proc. Nat. Acad. Sci. USA* 76: 1377-81; 7 = Graf, Vogel, Biber, and Wurgler, 1979, *Mut. Res.* 59: 129-33; 8 = Mason, Green, Shaw, and Boyd, 1981, *Mut. Res.* 81: 329-43; 9 = Smith, 1976, *Mol. Gen. Genet.* 149: 73-85.
β Synonym: *mus110^{AT1}*.

cytology: Placed in 4B3-C1 based on its inclusion in *Df(1)bi-D1 = Df(1)4B3-4;4D1-2* but not in *Df(1)rb41 = Df(1)4B6-C1;4C7-8* (Banga, Bloomquist, Brodberg, Pye, Larrivee, Mason, Boyd, and Pak, 1986, *Chromosoma* 93: 341-46).

mei-41 (R.S. Hawley)

location: 1-54.2.

references: Baker and Carpenter, 1972, *Genetics* 71: 255-86.

Carpenter and Sandler, 1974, *Genetics* 76: 453-75.

Baker and Hall, 1976, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 352-434.

Boyd, Golino, Nguyen, and Green, 1976, *Genetics* 84: 485-506.

Boyd and Setlow, 1976, *Genetics* 84: 507-26.

Smith, 1976, *Mol. Gen. Genet.* 149: 73-85.

Mohler, 1977, *Genetics* 85: 259-72.

Baker, Carpenter, and Ripoll, 1978, *Genetics* 90: 531-78.

Baker and Smith, 1979, *Genetics* 92: 833-47.

Carpenter, 1979, *Chromosoma* 75: 259-92.

Gatti, 1979, *Proc. Nat. Acad. Sci. USA* 76: 1377-81.

Nguyen, Boyd, and Green, 1979, *Mut. Res.* 63: 67-77.

Mason, Green, Shaw, and Boyd, 1981, *Mut. Res.* 81: 329-43.

Boyd and Shaw, 1982, *Mol. Gen. Genet.* 186: 289-94.

Hawley and Tartof, 1983, *Genetics* 104: 63-80.

Hawley, Marcus, Cameron, Schwartz, and Zitron, 1985, *Proc. Nat. Acad. Sci. USA* 82: 8095-99.

Banga, Bloomquist, Brodberg, Pye, Larrivee, Mason, Boyd, and Pak, 1986, *Chromosoma* 93: 341-46.

Mason, Scobie, and Yamamoto, 1989, *Mol. Gen. Genet.* 215: 190-99.

phenotype: *mei-41* alleles confer sensitivity to mutagens as a consequence of a defect in a caffeine-sensitive post-replication repair pathway (Boyd and Setlow, 1976; Boyd *et al.*, 1976; Boyd and Shaw, 1982). This defect in DNA repair is also manifested by a high frequency of mitotic chromosome breakage and instability (Baker *et al.*, 1978; Gatti, 1979). *mei-41¹* not hypermutable to alkylation nor defective in excision repair; yet *mei-41^{D12}* displays hypermutability to alkylation and defective alkylation excision repair (Smith and Dusenberry, 1988, *Mechanisms and Consequences of DNA Damage Processing*, Alan R. Liss, Inc., pp. 251-55). Allelism based on lack of complementation (Mason, Scobie, and Yamamoto, 1989, *Mol. Gen. Genet.* 215: 190-99). Most alleles of *mei-41* also reduce female fertility and in some cases are female-sterile as homozygotes. Females homozygous for the more fertile alleles of *mei-41* exhibit reduced levels of meiotic exchange. The observed reductions in exchange are not uniform, but rather are most extreme in distal regions. Chiasma interference is also diminished (Carpenter and Sandler, 1974; Baker and Hall, 1976). These reduced levels of exchange allow for high frequencies of meiotic loss and nondisjunction (see Baker and

Hall, 1976). Ultrastructural analysis of pachytene in *mei-41* and *mei-41*² females demonstrates a reduced number of late recombination nodules which are distributed in a fashion that parallels residual exchange events (Carpenter, 1979). Over half of those nodules which are observed are morphologically abnormal and are associated with unusual regions of relatively uncondensed chromatin. Stocks in which *mei-41* is carried in the male are frequently observed to carry or acquire a *bb* mutation on the *mei-41*-bearing X chromosome (Hawley and Tartof, 1983). Several alleles of *mei-41* have also been shown to inhibit rDNA magnification in the male germline (Hawley and Tartof, 1983; Hawley *et al.*, 1985). Moreover, *mei-41* males undergoing magnification also produce a high frequency of X-Y exchanges which result from one break within the X-chromosome rDNA cluster and the other at any of a large number of sites on the Y chromosome (Hawley and Tartof, 1983).

alleles: More than fifty alleles of this locus have been recovered. This includes the *mus-103* and *mus-104* mutations which were shown by Mason *et al.* (1989) to be allelic to *mei-41*. The most commonly used alleles are listed in the table below. A fine structure map of the locus is presented in Mason *et al.* (1989).

allele	synonym	ref ^α	reduces exchange	female fertility	mitotic instability ^β
<i>mei-41</i>		1	++	reduced	+
<i>mei-41</i> ²		1	++	reduced	+
<i>mei-41 D1</i>	<i>mei-41</i> ¹⁹⁵	1	++	sterile	
<i>mei-41 D2</i>		2	+	reduced	
<i>mei-41 D3</i>		2		sterile	
<i>mei-41 D4</i>		2		sterile	
<i>mei-41 D5</i>		2-4	+	reduced	
<i>mei-41 D6</i>		3	+	reduced	
<i>mei-41 D7</i>		3	+	reduced	
<i>mei-41 D8</i>		3	+	reduced	
<i>mei-41 D9</i>		3	-	fertile	
<i>mei-41 D10</i>		3	-	reduced	
<i>mei-41 D11</i>		3		sterile	
<i>mei-41 D12</i>	<i>mus103 D1</i>	1a, 4	-	fertile	+
<i>mei-41 D13</i>	<i>mus103 D2</i>	1a, 4	-		+
<i>mei-41 D14</i>	<i>mus104 D1</i>	2, 4	-	fertile	+
<i>mei-41 D15</i>	<i>mus104 D2</i>	3, 4			
<i>mei-41 D16</i>	<i>mus104 D3</i>	2, 4			
<i>mei-41 D17</i>	<i>mei-41 A1-A17</i>	7		reduced	
<i>mei-41 D18</i>	<i>mei-41 12-1007</i>	3, 4		sterile	
<i>mei-41 D19</i>	<i>mei-41 AM</i>	3, 6		sterile	

^α 1 = Baker and Carpenter, 1972, Genetics 71: 255-86; 1a = Boyd, Golino, Nguyen, and Green, 1976, Genetics 84: 485-506; 2 = Boyd, Golino, and Setlow, 1976, Genetics 84: 527-44; 3 = Mason, Green, Shaw, and Boyd, 1981, Mut. Res. 81: 329-43; 4 = Mason, Scobie, and Yamamoto, 1989, Mol. Gen. Genet. 215: 190-99; 5 = Mohler, 1977, Genetics 85: 259-72; 6 = Nguyen, Boyd, and Green, 1979, Mut. Res. 63: 67-77; 7 = Smith, 1976, Mol. Gen. Genet. 149: 73-85.

^β Gatti, 1979, Proc. Nat. Acad. Sci. USA 76: 1377-81.

cytology: Placed in 14C4-6 by deficiency analysis (Banga and Boyd).

***mei-218* (R.S. Hawley)**

location: 1-56.2 (Whyte and Hawley, unpublished data).

references: Baker and Carpenter, 1972, Genetics 71: 255-86.

Carpenter and Sandler, 1974, Genetics 76: 453-75.

Baker and Hall, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp.

352-434.

Baker, Carpenter, and Ripoll, 1978, Genetics 90: 531-78.

Sandler and Szauter, 1978, Genetics 90: 699-712.

Carpenter, 1979, Chromosoma 75: 259-92.

Gatti, 1979, Proc. Nat. Acad. Sci. USA 76: 1377-81.

Lutken and Baker, 1979, Mut. Res. 61: 221-27.

Carpenter, 1982, Proc. Nat. Acad. Sci. USA 79: 5961-65.

Carpenter and Baker, 1982, Genetics 101: 81-89.

Carpenter, 1984, Cold Spring Harbor Symp. Quant. Biol. 49: 23-29.

Carpenter, 1989, Genome 31: 74-80.

phenotype: Females homozygous for *mei-218* exhibit reduced levels of meiotic exchange. The residual exchanges are distributed such that the probability of euchromatic exchange becomes more nearly proportional to the polytene-chromosome length (Baker and Carpenter, 1972; Carpenter and Sandler, 1974; Baker and Hall, 1976). This relaxation of the normal constraints on the distribution of euchromatic exchange is clearly demonstrated by the ability of *mei-218* females to allow exchange between the normally achiasmatic fourth chromosomes (Sandler and Szauter, 1978). *mei-218* does not, however, permit exchanges to occur in heterochromatic intervals (Carpenter and Baker, 1982). Ultrastructural analysis of pachytene in *mei-218* and *mei-218*⁶⁻⁷ females demonstrates a reduced number of late recombination nodules (to about 8% of normal), which are distributed in a fashion that parallels the residual exchange events (Carpenter, 1979, 1989). Many of the nodules which are observed are morphologically abnormal. There is also some evidence that early recombination nodules may be either fewer in number or more ephemeral in *mei-218* females (Carpenter, 1989). Although *mei-218* alleles reduce the frequency of reciprocal meiotic exchange, the absolute frequency of gene conversion at the *rosy* locus is two-fold elevated relative to wild-type controls (Carpenter, 1982, 1984). Moreover, co-conversion distances are shorter than those recovered from controls or from *mei-9* females (Carpenter, 1984). Thus the function of this locus is required for the generation of reciprocal exchanges and not for gene conversion (Carpenter, 1984). The reduced levels of reciprocal exchange which are characteristic of *mei-218* females allow for high frequencies of meiotic loss and nondisjunction (see Baker and Hall, 1976). Nondisjunction occurs at the first meiotic division and only nonexchange chromosomes nondisjoin (Carpenter and Sandler, 1974). All assays for an effect of *mei-218* on mitotic chromosome behavior in males or on somatic chromosome behavior are negative (Baker and Carpenter, 1972; Baker *et al.*, 1978; Lutken and Baker, 1979; Gatti, 1979).

alleles: There are two well characterized alleles of this locus: *mei-218* (Baker and Carpenter, 1972) and *mei-218*⁶⁻⁷ (Baker *et al.*, 1978).

mei-195: see *mei-41*

***mei-251* (R.S. Hawley)**

location: 1- unlocated.

references: Baker and Carpenter, 1972, Genetics 71: 255-86.

Baker, Carpenter, Esposito, Esposito, and Sandler, 1976 Annu. Rev. Genet. 10: 53-134.

Baker and Hall, 1976, The Genetics and Biology of *Dro-*

sophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 352-434.

Gatti, 1979, Proc. Nat. Acad. Sci. USA 76: 1377-81.

Carpenter and Baker, 1982, Genetics 101: 81-89.

phenotype: Females homozygous for *mei-251* exhibit reduced levels of meiotic exchange (to approximately 80% of control levels). The residual exchanges are distributed such that the probability of euchromatic exchange becomes more nearly proportional to polytene-chromosome length (Baker and Carpenter, 1972; Carpenter and Sandler, 1974; Baker and Hall, 1976). Both *X* and fourth chromosomal nondisjunction are slightly elevated, presumably as a consequence of decreased recombination. Cytological analysis of larval-neuroblast metaphases reveals no increase in spontaneous mitotic chromosome breakage in the presence of *mei-251* (Gatti, 1979).

mei-254: see *nod*^a

***mei-352* (R.S. Hawley)**

location: 1- unmapped.

origin: Induced by ethyl methanesulfonate.

references: Baker and Carpenter, 1972, Genetics 71: 255-86.

Baker, Carpenter, and Ripoll, 1978, Genetics 90: 531-78.

Baker and Hall, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 352-434.

Carpenter and Baker, 1982, Genetics 101: 81-89.

Gatti, 1979, Proc. Nat. Acad. Sci. USA 76: 1377-81.

Sandler and Szauter, 1978, Genetics 90: 699-712.

phenotype: *mei-352* females exhibit near normal levels of meiotic exchange but the distribution of crossover events is abnormal (*i.e.*, exchanges are more uniformly distributed per unit length than they are in wild-type). This relaxation of the normal constraints on the distribution of euchromatic exchange is clearly demonstrated by the ability of *mei-352* females to allow exchange between the normally achiasmate fourth chromosomes (Sandler and Szauter, 1978). *mei-352* does not, however, permit exchanges to occur in heterochromatic intervals (Carpenter and Baker, 1982). It remains to be demonstrated that the reduced fertility and the exchange phenotype are the consequence of the same mutational lesion. Although no increase in spontaneous chromosome aberrations was observed upon direct cytological examination of larval neuroblasts (Gatti, 1979), the *mei-352* mutation was shown to increase mitotic chromosome instability approximately three-fold when assayed genetically (Baker *et al.*, 1978). This suggests that the *mei-352*⁺ gene product is required both during meiosis and mitosis.

****mei-1029* (R.S. Hawley)**

location: 3- unlocated.

references: Szauter, 1984, Genetics 106: 45-71.

phenotype: Females homozygous for *mei-1029* show reduced levels of meiotic recombination and an abnormal distribution of residual recombination events. *X* and fourth chromosomal nondisjunction are elevated and occur at the first meiotic division. The observed *X* chromosome nondisjunction primarily results from the nondisjunction of nonexchange chromosomes.

****mei-1946* (R.S. Hawley)**

Entry identical to that of *mei-1029*.

****mei-1966* (R.S. Hawley)**

Entry identical to that of *mei-1029*.

****mei-2185* (R.S. Hawley)**

Entry identical to that of *mei-1029*.

****mei-2199* (R.S. Hawley)**

Entry identical to that of *mei-1029*.

****mei-2220* (R.S. Hawley)**

Entry identical to that of *mei-1029*.

****mei-2245* (R.S. Hawley)**

Entry identical to that of *mei-1029*.

****mei-2439* (R.S. Hawley)**

Entry identical to that of *mei-1029*.

****mei-2593* (R.S. Hawley)**

Entry identical to that of *mei-1029*.

****mei-2696* (R.S. Hawley)**

Entry identical to that of *mei-1029*.

****mei-B* (R.S. Hawley)**

location: Unlocated, probably 2.

references: Bridges, 1915, J. Exp. Zool. 19: 1-21.

Bridges, 1929, Carnegie Inst. Wash. Publ. 399: 63-83.

Baker and Hall, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 352-434.

phenotype: Females homozygous for *mei-B* exhibit reduced levels of meiotic exchange (to approximately 33% of control levels). The reduction in exchange is most severe in distal intervals (Baker and Hall, 1976). The effects of this mutation on disjunction were not examined. However its low fertility suggests that nondisjunction may be frequent.

mei-G17

location: 2- (distal third of 2R).

origin: Induced with ethyl methanesulfonate.

references: Gethmann, 1974, Genetics 78: 1127-42.

phenotype: A male-specific and chromosome specific meiotic mutant. Homozygous males, but not females, exhibit elevated frequencies of nondisjunction of second and sex but not third or fourth chromosomes. The incidence of *X-Y* nondisjunction varies from 4-6%, with nullo-sex-chromosome offspring greatly outnumbering *X-Y* bearing offspring; nondisjunction seems to occur in the first meiotic division. Among regular progeny, *X*-bearing genotypes are recovered in excess of *Y*-bearing genotypes. The frequency of second-chromosome exceptions varies, depending on the *Y* chromosome present, from 0.25 (*B^SY*) to 1.0 (normal *Y*) exceptions per parental male; this difference is attributable to variable recoveries of nullo-2 but not diplo-2 offspring. Generally the products of nullo-2 sperm are recovered more frequently than those of diplo-2 sperm. Sex-chromosome exceptions are more frequent in products of second-chromosome nondisjunction than in progeny that are regular for chromosome 2; furthermore, *XX;0* and *0;22* products are recovered much more frequently than *XX; 22* and *0;0* products.

mei-G87

location: 3- (approximately half way between *al* and *b*).

origin: Induced with ethyl methanesulfonate.

references: Gethmann, 1974, *Genetics* 78: 1127-42. 1984, *Genetics* 107: 65-77.

phenotype: A chromosome-specific but not sex-specific meiotic mutant; causes both reductional and equational nondisjunction of chromosome 2 in both males and females. In females, equational nondisjunction is independent of exchange, but reductional exceptions are primarily derived from non-exchange tetrads. In crosses of *mei-G87* males to *C(2)EN* females, in which half the exceptions are recoverable, 6-10 exceptions per thousand eggs were recovered; in the reciprocal cross, in which all the diplo-2 exceptions are theoretically recoverable, there were 1-2 exceptions per thousand eggs. In primary spermatocytes, a low incidence of cases in which the chromatids of one pair of autosomes have separated are observed.

mei-I1

location: 3-1.8 (1/15 the distance between *ru* and *h*).

origin: Induced with ethyl methanesulfonate.

references: Ivy, 1981, PhD thesis, University of California, San Diego.

phenotype: Homozygous males produce 43-49% gametes exceptional for sex chromosomes and 39-44% exceptional for chromosome 4. Sex and fourth chromosomes misassort independently. Consistently produce an excess of nullo-X-nullo-Y over XY-bearing gametes, but nullo-4 and diplo-4 gametes equally frequent. Nondisjunction confined to first meiotic division. Chromosomes 2 and 3 also give high frequencies of nondisjunction; appear to disjoin randomly based on chromosome analysis of secondary spermatocytes. Primary spermatocytes show mostly univalents; MII appears normal except for the non-haploid complements resulting from MI chromosome misbehavior. Spermatids often contain micronuclei, and nullo exceptions are more frequent than diplo exceptions, both indicative of chromosome loss.

cytology: Placed in 61A-62B1 based on the ability of $Y^{P_3^D}$ of $T(Y;3)D8 = T(Y;3)60A10-B1$ but not $Y^{P_3^D}$ of $T(Y;3)A114 = T(Y;3)61A$ to cover both the genetic and cytological effects of *mei-I1*.

mei-I3

location: 3- (unmapped).

origin: Induced with ethyl methanesulfonate.

references: Ivy, 1981, PhD thesis, University of California, San Diego.

phenotype: In males homozygous for this mutation, all chromosomes nondisjoin at meiosis I and reciprocal meiotic products are recovered with unequal frequencies. Furthermore, the X chromosomes nondisjoin at meiosis II. The meiotic behaviors of the sex and fourth chromosomes are positively correlated, *i.e.*, more double exceptions were recovered than expected; among double exceptions, however, nonhomologues behave independently. Homozygous females virtually sterile owing either to *mei-I3* or to a closely linked independent mutation.

***mei-O81**

location: 3- (unmapped).

origin: Natural population.

references: Sandler, Lindsley, Nicoletti, and Trippa, 1968, *Genetics* 60: 525-58.

phenotype: Male-specific recessive meiotic mutant. Apparently causes nondisjunction of all chromosomes; 5.0% for sex chromosomes, 7.8% for fourth chromosomes, and 0.78% double nondisjunction. Failure of homologous pairing seen in primary spermatocytes.

cytology: Polytene chromosomes normal.

***mei-S8**

location: 2-79.7.

origin: Natural population.

references: Sandler, Lindsley, Nicoletti, and Trippa, 1968, *Genetics* 60: 525-58.

phenotype: Male-specific recessive meiotic mutant. Causes high nondisjunction of chromosome 4, but has no effect on the sex chromosomes. Nullo-4 exceed diplo-4 gametes. Cytological examination of primary spermatocytes reveals nondisjunction of chromosome 4 in the first meiotic division.

cytology: Polytene chromosomes normal.

mei-S51 (R.S. Hawley)

location: Synthetic with at least one component on each large autosome.

references: Robbins, 1971, *Mol. Gen. Genet.* 110: 144-66. Sandler, Lindsley, Nicoletti, and Trippa, 1968, *Genetics* 60: 525-558.

phenotype: Females homozygous for *mei-S51* exhibit reduced exchange and high frequencies of nonhomologous disjunction, particularly with respect to the X and fourth chromosomes (XX <-> 44 segregations are frequent). In addition *mei-S51* decreases the frequency of secondary nondisjunction in structurally normal XXY females and increases the frequency of nondisjunction in X inversion heterozygotes. Robbins (1971) proposed that *mei-S51* disrupts a number of aspects of chromosome pairing and alignment prior to metaphase and therefore both reduces exchanges and prevents proper partner choice within the distributive system.

mei-S282 (R.S. Hawley)

location: 3-5.

origin: Spontaneous.

references: Sandler, Lindsley, Nicoletti, and Trippa, 1968, *Genetics* 60: 525-58.

Parry, 1973, *Genetics* 73: 465-86.

Sandler, Romans, and Fignshow, 1974, *Genetics* 77: 299-307.

Baker and Hall, 1976, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 352-434.

Baker, Carpenter, and Ripoll, 1978, *Genetics* 90: 531-78.

phenotype: Females homozygous for *mei-S282* exhibit reduced levels of meiotic exchange. The residual exchanges are distributed such that the probability of euchromatic exchange becomes more proportional to unit length (Parry, 1973; Baker and Hall, 1976). This relaxation of the normal constraints on the distribution of euchromatic exchange is clearly demonstrated by the ability of *mei-S282* females to allow exchange on the

normally achiasmatic fourth chromosome (Sandler and Szauter, 1978). The reduced levels of reciprocal exchange which are characteristic of *mei-S282* females allow for high frequencies of meiotic loss and nondisjunction (see Baker and Hall, 1976). Nondisjunction occurs at the first meiotic division and only nonexchange chromosomes nondisjoin (Parry, 1973). The *mei-S282* mutation also causes mitotic chromosome instability (Baker *et al.*, 1978) when assayed genetically. This suggests that the *mei-S282*⁺ gene product is required both during meiosis and mitosis.

***mei-S332* (R.S. Hawley)**

location: 2-95.

origin: Spontaneous.

references: Sandler, Lindsley, Nicolletti, and Trippa, 1968, *Genetics* 60: 525-58.

Baker and Hall, 1976, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 352-434.

Davis, 1971, *Mol. Gen. Genet.* 113: 251-72.

Sandler, Romans, and Figenshow, 1974, *Genetics* 77: 299-307.

Hardy, 1975, *Genetics* 79: 231-64.

Davis, 1977, *DIS* 52: 100.

Baker, Carpenter, and Ripoll, 1978, *Genetics* 90: 531-78.

Goldstein, 1980, *Chromosoma* 78: 79-111.

phenotype: *mei-S332* is a semidominant autosomal mutation that increases nondisjunction of all chromosomes to the same extent in both sexes. Although female recombination is normal, *mei-S332* causes high frequencies of equational nondisjunction in both sexes as well as chromosome loss (Davis, 1971; Goldstein, 1980). In the male germline sister chromatid associations are generally normal during prophase I and metaphase I. However, by telophase I sister chromatids have frequently undergone precocious separation (Goldstein, 1980). The equational exceptions produced by *mei-S332* males and females presumably result from precociously separated sister chromatids going to the same pole at anaphase II. Goldstein (1980) has also suggested that "in the case of *mei-S332*, chromosomes which lag in the second meiotic division are usually lost and most of the genetically observed loss in *mei-S332* occurs in the second meiotic division." The hypothesis that lagging chromatids are often lost at anaphase II is consistent with Hardy's observation that *mei-S332* has micronuclei present at the early spermatid stages (Hardy, 1975). Thus *mei-S332* presumably defines a function required for sister chromatid cohesion. Sandler *et al.* (1974) have shown that ring chromosomes are frequently converted into dominant lethals in *mei-S332* females, presumably resulting from an impaired ability to resolve sister ring chromosomes at anaphase II. Baker *et al.* (1978) have also shown that mitotic chromosome instability is elevated in the presence of *mei-S332*. They suggest that the function of the *mei-S332*⁺ locus is to delay the separation of sister chromatids at all divisions. Because the sole *mei-S332* allele is viable, they have further suggested that either this allele is leaky or that there are overlapping or redundant functions that can compensate for this defect.

cytology: Placed in 58A-E by Davis (1971, 1977); further confined to 58B by deficiency mapping (Kerrebrock and

Orr-Weaver).

mei-W5: see *pal*

mei-W22: see *c(3)G*

***mei-W68* (R.S. Hawley)**

location: 2-94.

references: Baker, Carpenter, Esposito, Esposito, and Sandler, 1976, *Ann. Rev. Genet.* 10: 53-134.

Baker, Carpenter, and Ripoll, 1978, *Genetics* 90: 531-78.

phenotype: Females homozygous for *mei-w68* show a complete absence of meiotic recombination (Baker, unpublished data). Ultrastructural studies of pachytene reveal an absence of chromosome condensation and little synaptonemal complex (Carpenter, cited in Baker *et al.*, 1976). A less severe allele (*mei-w68*^{LJ}-Lindsley) reduces exchange to approximately 60% of control levels and also alters the distribution of residual exchanges. Analysis of mitotic chromosome behavior (Baker *et al.*, 1978) suggests that the *mei-w68*⁺ gene product is also required in mitotic cells.

meiotic-: see *mei-*

***mel*: melanized**

location: 1-64.1.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).

discoverer: Fahmy, 1953.

references: 1958, *DIS* 32: 71.

phenotype: Body color darker than normal, especially in thorax; trident pronounced. Eye color dull red. Wing tips frequently curve upward. Classification rather difficult, best in young flies. Viability and fertility good in both sexes. RK3.

cytology: Placed in 19C2-3 by Schalet and Lefevre [1976, *Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1B, pp. 847-902].

other information: One allele induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

mel: see *mat*

mel(1)R1: see *pcx*

mel(3)5: see *ndl*

mel(3)9: see *Tl*^{8r}

melanized: see *mel*

melanizedlike: see *mell*

melanoscutellum: see *msc*

melanotic lesions: see *md*

melanotic tumor-A: see *tu-bw*

***mell*: melanizedlike**

location: 1-{64}.

references: Schalet and Lefevre, 1976, *Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, and San Francisco, Vol. 1B, pp. 847-902.

phenotype: Abdominal tergites have slight transverse wrinkles; perhaps thorax darker than normal; flies somewhat smaller with slightly broader wings; eyes of males slightly rough, wings of females variably wrinkled or

curled.

cytology: Placed in 19D2-E1 based on *mell* phenotype of heterozygotes between *Df(1)16-3-35 = Df(1)19D2-3;19E6-7* and *Df(1)mel8 = Df(1)18F4-5;19E1*.

Men: Malic enzyme

location: 3-51.73 [based on 91 recombinants between *kar* (3-51.7) and *ry* (3-52.0)].

synonym: *Mdh-NADP*.

references: Franklin and Rumball, 1971, DIS 47: 37.

Voelker, Ohnishi, Langley, Gausz, and Gyrukovics, 1981, *Biochem. Genet.* 19: 525-34.

phenotype: Structural gene for malic enzyme = (S)-Malate: NADP+ oxidoreductase [MEN (EC 1.1.1.40)], a tetramer with subunit molecular weight of 58,000 (Lee, Langley, and Burkhart, 1978, *Anal. Biochem.* 86: 697-296) or 67,250 (Geer, Krochko, Oliver, Walker, and Williamson, 1980, *Comp. Biochem. Physiol.* 65B: 25-34). Biochemical characterization by Geer, *et al.*, (1980). Enzyme known to provide NADP for lipogenesis; levels in larvae increased by dietary carbohydrate and decreased by dietary lipid. Levels normally high in early third-instar larvae, reducing to half in late third instar, rising again in ageing adults. Highest specific activity found in larval fat body and, among cellular fractions, in the cytosol (Geer, Krochko, and Williamson, 1979, *Biochem. Genet.* 17: 867-79). Histochemical staining of larval imaginal discs reveals enzyme activity localized to nervous elements, including ommatidial precursors, the morphogenetic furrow, and the optic nerve in the eye antennal disc; and the chordotonal organ and the nerve traversing the leg discs; wing, haltere, labial, and genital discs unstained; however genital disc derivatives, the ejaculatory duct and paragonia in males and oviducts of females are stained in adults (Finkbohner, Cunningham, and Kuhn, 1985, *Wilhelm Roux's Arch. Dev. Biol.* 194: 217-23). Staining of morphogenetic groove absent in *D. simulans* and in *simulans-melanogaster* hybrids except when such hybrids contain two copies of *3R* from *D. melanogaster* and none from *D. simulans* (Kuhn and Sprey, 1987, *Genetics* 115: 277-81). All null alleles tested survive and are fertile in combination with *Df(3R)ry3l*, which lacks *Men*⁺ (Voelker *et al.*). The NADPH/NADP ratio in such null genotypes is one-seventh that of wild type (Geer *et al.*, 1979). Presence of an extra dose of the region from 8D10-12 to 9A1-2 results in an approximately 25% increase in MEN activity (Williamson and Bentley, 1983, *Genetics* 103: 649-58).

alleles:

allele	origin	synonym	ref ^α	comments ^β
Men²	spont	<i>Men</i> ^{0.90}	2, 5	slow migration
Men⁴	spont	<i>Men</i> ^{1.0}	2, 5	common allele, p = 0.95 intermediate migration
Men⁶	spont	<i>Men</i> ^{1.1}	2, 5	fast migration
Men^{nNC1}	spont		1, 4, 6	3-5% normal activity ^γ ; dimer -
Men^{nNC2}	spont		1, 4	no activity; dimer -
Men^{nNC3}	spont		1, 4	no activity; dimer -
Men^{nNC4}	spont		1, 4	no activity; dimer -
Men^{nNC5}	spont		1, 4	residual activity; dimer -
Men^{nNC6}	spont		1, 4	residual activity; dimer -
Men^{nNC311}	spont		3	CRM -
Men^{nNC506}	spont		3	CRM -
Men^{nGB1}	spont		1	no activity; dimer -

^α 1 = Burkhart, Montgomery, Langley, and Voelker, 1984, *Genetics* 107: 295-306; 2 = Franklin and Rumball, 1971, DIS 47: 37; 3 = Lee and Bronson, 1979, *J. Biochem.* 254: 6374-81; 4 = Voelker, Langley, Leigh-Brown, Ohnishi, Dickson, Montgomery, and Smith, 1980, *Proc. Nat. Acad. Sci. USA* 77: 1091-95; 5 = Foelker, Ohnishi, Langley, Gausz, and Gyrukovics, 1981, *Biochem. Genet.* 19: 525-34; 6 = Williamson, 1982, *Can. J. Genet. Cytol.* 24: 409-16.

^β dimer- indicates inability of product to form heterodimer with electrophoretically variant polypeptides; CRM = immunologically cross-reacting material; quasi-null alleles derived from *Men*⁴.

^γ Enzyme extracted from *Men^{nNC1}* homozygotes kinetically indistinguishable from wild-type enzyme; Williamson accordingly proposes that *Men^{nNC1}* is a cis-acting regulatory mutant.

cytology: Placed in 87C9-D1 based on its exclusion from *Df(3R)kar3J = Df(3R)87B15-C1;87C9-D1* on the left and *Df(3R)ry27 = Df(3R)87D1-2;87F1-2* on the right.

merry-go-round: see *mgr*

mes: messy A

location: 1-51.9.

discoverer: Schalet.

references: Schalet, Kernaghan, and Chovnick, 1964, *Genetics* 50: 1261-68.

Hilliker, Clark, Chovnick, and Gelbart, 1980, *Genetics* 95: 95-110.

phenotype: Extra head and thoracic bristles, especially anterior scutellars; wings inflated, turned somewhat upward and outward, and shorter and broader than normal; posterior crossvein gapped or missing. Semilethal; male considerably less viable than female; sterile. RK3.

allele	origin	discoverer	synonym	ref ^α
mesA¹	X ray	Schalet	<i>mes</i> ¹	1, 4
mesA²	X ray	Schalet	<i>mes</i> ²	1, 4
mesA³	EMS	Hilliker, Clark	<i>l(3)2-34</i>	2, 3
mesA⁴	EMS	Hilliker, Clark	<i>l(3)4-22</i>	2, 3
mesA⁵	EMS	Hilliker, Clark	<i>l(3)8-9</i>	2, 3
mesA⁶	EMS	Hilliker, Clark	<i>l(3)10-140</i>	2, 3
mesA⁷	EMS	Hilliker, Clark	<i>l(3)13-62</i>	2, 3
mesA⁸	EMS	Hilliker, Clark	<i>l(3)A12-2</i>	2, 3
mesA⁹	EMS	Hilliker, Clark	<i>l(3)A13-1</i>	2, 3
mesA¹⁰	EMS	Hilliker, Clark	<i>l(3)A27-2</i>	2, 3
mesA¹¹	EMS	Hilliker, Clark	<i>l(3)B26-1</i>	2, 3
mesA¹²	EMS	Gelbart	<i>l(3)G2</i>	2, 3
mesA¹³	EMS	Gelbart	<i>l(3)G3</i>	2, 3
mesA¹⁴	EMS	Gelbart	<i>l(3)G8</i>	2, 3
mesA¹⁵	EMS	Gelbart	<i>l(3)G19</i>	2, 3

^α 1 = CP627; 2 = Hilliker, Clark, and Chovnick, 1981, DIS 56: 64-72; 3 = Hilliker, Clark, Chovnick, and Gelbart, 1980, *Genetics* 95: 95-110; 4 = Schalet, Kernaghan, and Chovnick, 1964, *Genetics* 50: 1261-68.

cytology: Placed in 87D4-8 based on its inclusion in *Df(3R)ry1608 = Df(3R)87D4-6;87E1-2* but not *Df(3R)ry74 = Df(3R)87D8;87D12*.

other information: All alleles complement *mesB*.

mesB

location: 3-51.9.

discoverer: Schalet.

references: Schalet, Kernaghan, and Chovnick, 1964, *Genetics* 50: 121-68.

Hilliker, Clark, Chovnick, and Gelbart, 1980, *Genetics* 95: 95-110.

phenotype: Wings outspread at about 45° from mid-line. Trident dark; occasional thoracic bristle duplication. Subtly abnormal abdomen. Semilethal. RK3.

alleles: Existence of thirteen alleles recorded by Skinner, Cole, and Chovnick. *mesB* alleles only partially comple-

ment alleles of *l(3)87De*.

allele	origin	discoverer	synonym	ref ^α
<i>mesB</i> ¹	X ray	Schalet	<i>mes</i> ³	1, 4
<i>mesB</i> ²	X ray	Schalet	<i>mes</i> ⁴	1, 4
<i>mesB</i> ³	X ray	Schalet	<i>mes</i> ^{5l}	1, 4
<i>mesB</i> ⁴	X ray	Schalet	<i>mes</i> ^{6l}	1, 4
<i>mesB</i> ⁵	EMS	Hilliker, Clark	<i>l(3)34-2</i>	2, 3
<i>mesB</i> ⁶	EMS	Hilliker, Clark	<i>l(3)B14-1</i>	2, 3

^α 1 = CP627; 2 = Hilliker, Clark, and Chovnick, 1981, DIS 56: 64-72; 3 = Hilliker, Clark, Chovnick, and Gelbart, 1980, Genetics 95: 95-110; 4 = Schalet, Kernaghan, and Chovnick, 1964, Genetics 50: 1261-68.

cytology: Placed in 87D8-12 based on its inclusion in the region of overlap of *Df(3R)ry74 = Df(3R)87D8;87D12* and *Df(3R)kar-IG27 = Df(3R)87B3-5;87D6-12*.

other information: Interacts in *trans* with *l(3)87De* (Skinner, Cole, and Chovnick).

messy: see *mes*

***Met: Metatarsi irregular**

location: 2- or 3- (rearrangement).

origin: X ray induced.

discoverer: Jonsson, 56a10.

references: Lüning, 1956, DIS 30: 73.

phenotype: First and second tarsal joints fused and swollen with extra hairs. Male sex combs enlarged. Fully penetrant when balanced with *Cy*; however, *Met/ss* is wild type or nearly so. RK2A.

cytology: Associated with *T(2;3)Met*.

Met: see *Rst(1)JH*

Metallothionein: see *Mtn*

metaphase arrest: see *mar*

Mex156: see *Sxl*

***mf: macrofine**

location: 1-5.5.

origin: Induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1955.

references: 1958, DIS 32: 71.

phenotype: Fly slightly smaller than normal with short, thin, bristles. Male viable and fertile. Female slightly delayed in eclosion and reduced in viability. RK3.

Mfcp: Myofibrillar contractile protein

location: 3 {50-51}.

references: Bernstein, Glenn, and Emerson, 1981, Genetics 97: s10.

phenotype: Structural genes for several contractile polypeptides of approximately 22,500 daltons molecular weight that initiate expression at the fusion stage of myogenesis (Bernstein and Donady, 1980, Dev. Biol. 79: 388-98).

cytology: Placed in 87B by *in situ* hybridization.

molecular biology: Genes cloned, presumably by differential screen of pre- and post-fusion stages of myocytes in culture.

mfd: myofibrillar-defective (J.C. Hall)

location: 1- {38}.

phenotype: Deletion of salivary segment 11A6-7 leads to inability to jump or fly, hemizygoty for this variant

allows viability, and the phenotypic defects induced by the deletion are recessive; myofibrils or thoracic muscles shortened; spots are missing in two dimensional gel analysis of proteins from fibrillar and tubular muscles; these differences from wild type apparently due to blocking of phosphorylation of proteins in these muscles; such proteins are very likely myosins that become phosphorylated during the first day after eclosion (in wild-type), in parallel with the gradual development of flying and jumping ability (Hiromi, Ohmura, Masaki, Hirose and Hotta).

cytology: Maps to 11A6-7; synthetic deletion, constructed using *T(1;Y)*'s, is the only *mfd* variant.

mfs(2)31: male-female-sterile (2) in region 31

location: 2-41.35 [based on 23 recombinants between *J* (2-41.0) and *msf(2)31* with *da* (2-41.3) as a middle marker].

origin: Induced by ethyl methanesulfonate.

synonym: *mfs48*.

references: Sandler, 1977, Genetics 86: 567-82.

Lindsley, Goldstein, and Sandler, 1980, DIS 55: 84-85.

phenotype: Homozygotes have short thin bristles; relative viability 25% at 25°; hemizygotes lethal. Both males and females sterile at 25° but some fertility observed in flies raised at 23°. Males raised at 28.5° have no motile sperm and some spermatids have micronuclei, and occasionally two basal bodies and axonemes are observed.

cytology: Placed in 31B-32A based on its lethality in combination with *Df(2L)527 = Df(2L)31B-D;31F-32A*

mfs(2)350

location: 2-50.7.

references: Fukunaga, 1980, J. Hered. 71: 349-52.

phenotype: Homozygous males and females sterile in certain cytoplasmic constitutions, fertile in others. Gonads rudimentary, although some males have normal testes without motile sperm.

mfs(2)7601: see *Dox-A2*^{*mfs1*}

mfs(3)73A

location: 3- {45}.

origin: Induced by ethyl methanesulfonate.

discoverer: Hoffmann.

cytology: Placed in 73A10-B1 between the right breakpoints of *Df(3L)st7P* and *Df(3L)st-g24*.

mfs(3)G: male-female-sterile of Gill

location: 3-59.

origin: X ray induced.

discoverer: Gill, 59a.

synonym: *fs(3)4*^{59a}.

references: 1960, Anat. Record 138: 351.

1961, Ph.D. Thesis, Yale Univ.

1962, DIS 36: 37.

1963, J. Exp. Zool. 152: 251-77 (fig.).

phenotype: Oogenesis incomplete; follicles usually cease development early in vitellogenesis (at or before stage 9); occasional breakthrough produces adult fly. Primary compound chambers in which two, occasionally three, incipient cysts are enclosed occur in about 10% of the cases. Male sterile. Adult fat body hypertrophied; body size reduced. Occasionally, metathoracic legs with tibiae more curved than normal and tarsi crooked. Viability low. RK3.

mgr: merry-go-round

location: 3-51.3.
origin: X-ray induced.
references: González, Casal, and Ripoll, 1988, J. Cell Sci. 89: 39-47.
phenotype: Most characteristic abnormality is appearance of mitotic and meiotic figures in which all the chromosomes are arranged in a circle. Other mutant traits include metaphase arrest, polyploid cells, postmeiotic cysts with 16 nuclei, and spermatids carrying four times the normal complement of chromosomes. The circular mitotic figures (CMF's) are caused by monopolar spindles. The wild-type allele of *mgr* apparently is necessary for correct centrosome behavior.

mgt: midget

location: 1-48.7.
origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).
discoverer: Fahmy, 1954.
references: 1958, DIS 32: 71.
phenotype: Small fly with delayed eclosion. Not easily classified. Male fertile and viability about 20% wild type. Expression more extreme in female and viability further reduced. RK3.
other information: One allele each induced by CB. 3025 and X rays; two alleles induced by CB. 1506.

mh: maternal haploid

location: 1-.
origin: Induced by ethyl methanesulfonate.
synonym: *fs(1)A1182*.
references: Gans, Audit, and Masson, 1975, Genetics 81: 683-704.
 Zalokar, Audit, and Erk, 1975, Dev. Biol. 47: 419-32.
 Santamaria and Gans, 1980, Nature (London) 287: 143-44.
 Santamaria, 1983, Dev. Biol. 96: 285-95.
phenotype: Homozygous *mh* females are fertile at 18° but at 25° they are sterile; irrespective of the males used in cross, nuclei of developing embryos appear to be X-bearing and haploid. 183/200 eggs developed to blastoderm; gastrulation was abnormal; 22 embryos gave evidence of segmentation and muscular movement. Nuclei from such embryos injected into normal early embryos are capable of developing into patches of tissue, some of which produce structures of normal number and size and are presumably diploid, some of which produce increased numbers of smaller-than-normal structures and are presumably haploid, and a few of which are mixed. Haploid tissue with female phenotype found in basis-tarsus, tergites, and terminalia. Nuclear division cycles of haploid embryos 2.1 min. longer than wild type; such embryos undergo an extra nucleus division in the syncytial blastoderm, possibly to achieve a proper nuclear: cytoplasmic ratio prior to cellularization (Edgar, Kichle, and Schuberger, 1986, Cell 44: 365-72). Haploid cell cultures established from 25° embryos (Debec, 1978, Nature 274: 255-56). Haploid cells and their diploidized derivatives lack centrioles (Debec, Szöllösi, and Söllöcs, 1982, Biology of the Cell 44: 133-38).

Mhc: Muscle myosin heavy chain

location: 2-52.
origin: A number of mutants have been induced by ethyl methanesulfonate.
references: Mogami and Hotta, 1981, Mol. Gen. Genet. 183: 409-17.
 Bernstein, Mogami, Donady, and Emerson, 1983, Nature 302: 393-97.
 Rozek and Davidson, 1983, Cell 32: 23-34.
 Mogami, O'Donnell, Bernstein, Wright, and Emerson, 1986, Proc. Nat. Acad. Sci. USA 83: 1393-97.
 Bernstein, Hansen, Becker, Wassenberg, Roche, Donady, and Emerson, 1986, Mol. Cell. Biol. 6: 2511-19.
 Rozek and Davidson, 1986, Proc. Nat. Acad. Sci. USA 83: 2128-32.
 Wassenberg, Kronert, O'Donnell, and Bernstein, 1987, J. Biol. Chem. 262: 10741-47.
 Homyk and Emerson, 1988 Genetics 119: 105-21.
 O'Donnell and Bernstein, 1988, J. Cell Biol. 107: 2601-12.
 Chun and Falkenthal, 1988, J. Cell Biol. 107: 2613-21.
 George, Ober, and Emerson, 1989, Mol. Cell Biol. 9: 2957-74.
 Hess, Kronert, and Bernstein, 1989, Cellular and Molecular Biology of Muscle Development (Kedes and Stockdale, eds.). A.R. Liss, New York, pp. 621-31.
 O'Donnell, Collier, Mogami, and Bernstein, 1989, Genes Dev. 3: 1233-44.
 Kazzaz and Rozek, 1989, Dev. Biol. 133: 550-61.
phenotype: Structural gene for the heavy chain of muscle myosin (MHC). Heterozygotes for dominant flightless mutant alleles are characterized by erect wings and disrupted myofibrils in the indirect flight muscles. Segmental-deficiency heterozygotes for the locus are also flightless with disrupted myofibrils. Flight can be rescued in heterozygotes for most alleles by addition of a second Mhc+ allele to the complement, or by making the fly simultaneously hemizygous for *Act88F* [Beall, Sepanski, and Fyrberg, 1989, Genes Dev. 3: 131-40 (fig.)].
alleles: Of five ethyl-methanesulfonate-induced alleles that are lethal as homozygotes, three have 9-10 kb inserts with some apparently similar restriction sites (Mogami and Hotta; Mogami *et al.*). Three other ethyl-methanesulfonate-induced mutants, which are homozygous viable, are judged to be allelic based on the failure to obtain recombinants between them and the lethal alleles; as yet no molecular characterization available.

allele	discoverer	synonym	ref ^α	comments ^β
Mhc¹	Mogami		5, 6, 7, 8	lethal; 0.1 kb deletion in fifth exon and preceding intron ^γ
Mhc²	Mogami		7	lethal; 10 kb insert in intron ~2 kb 3' to <i>Mhc¹</i>
Mhc³	Mogami		7	lethal; 10 kb insert in intron ~3 kb 3' to <i>Mhc¹</i>
Mhc⁴	Mogami		7	lethal; 9 kb insert in intron ~5 kb 3' to <i>Mhc¹</i>
Mhc⁵	Grell, 1969	<i>Bsh: Bashed</i>	4, 5	viable ^γ
Mhc⁶	Mogami	<i>Ifm(2)1</i>	5, 6	viable
Mhc⁷	Mogami	<i>Ifm(2)2</i>	1, 5, 6, 10	viable ^γ
Mhc⁸	Mogami	<i>Ifm(2)3</i>	5, 6, 7	lethal; apparent point mutation
Mhc⁹	Mogami		9, 10	viable ^γ
Mhc¹⁰	Mogami		9, 10	viable ^γ
Mhc¹¹	Mogami		9, 10	viable ^γ
Mhc¹²	Sparrow & Ball		9	viable ^γ

allele	discoverer	synonym	ref ^α	comments ^β
Mhc 13	Sparrow		3	viable; neomorphic γ
Mhc 14	Steward		11	lethal
Mhc 15	Collier & Finke	<i>Stp</i>	2, 5	viable
Mhc 16	Homyk	<i>Nup</i>	5	semilethal γ

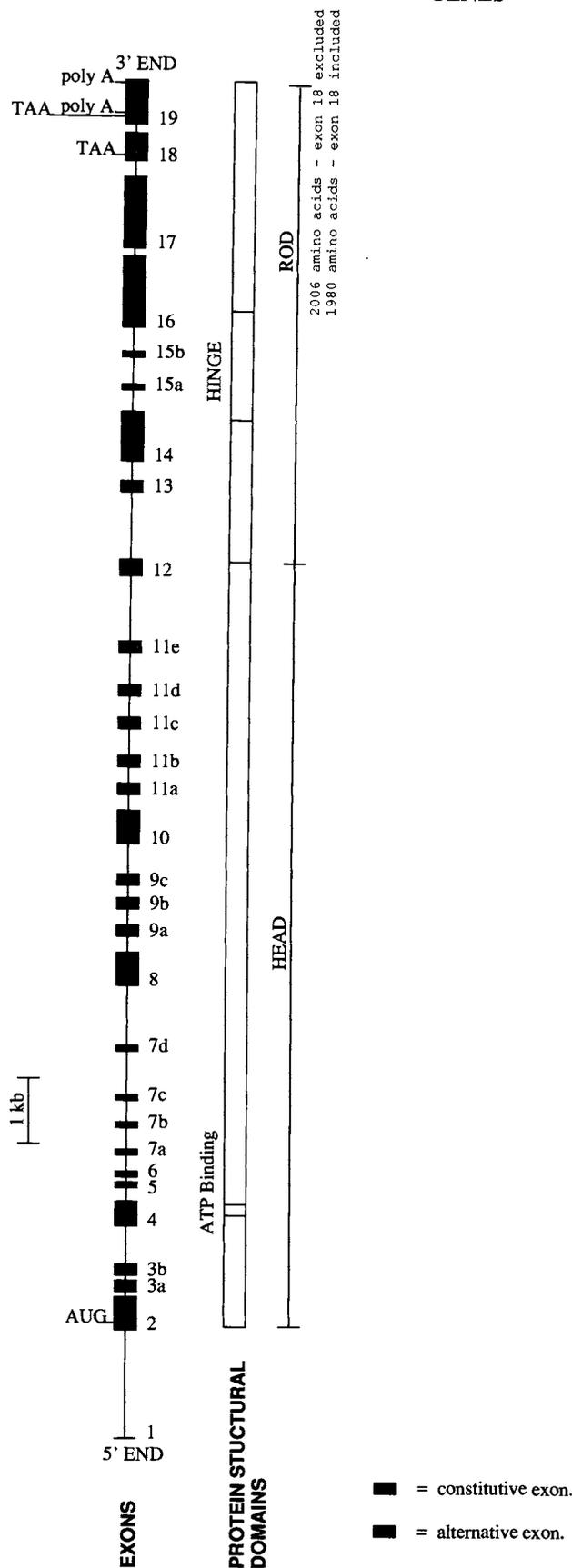
^α 1 = Chun and Falkenthal, 1988, J. Cell Biol. 107: 2613-21; 2 = Collier and Finke, 1984, J. Hered. 75: 477-79; 3 = Fieck, O'Donnell, Sparrow, and Bernstein, 1988, J. Cell Biol. 107: 257a; 4 = Grell, 1969, DIS 44: 46-47; 5 = Homyk and Emerson, 1985, Genetics 119: 105-21; 6 = Mogami and Hotta, 1981, Mol. Gen. Genet. 183: 409-17; 7 = Mogami, O'Donnell, Bernstein, Wright, and Emerson, 1986, Proc. Nat. Acad. Sci. USA 83: 1393-97; 8 = O'Donnell and Bernstein, 1988, J. Cell Biol. 107: 2601-12; 9 = O'Donnell, Mogami, and Bernstein, 1988, J. Cell Biochem. Suppl. 12C: 341; 10 = O'Donnell, Collier, Mogami, and Bernstein, 1989, Genes Dev. 3: 1233-46; 11 = Steward and Nüsslein-Volhard, 1986, Genetics 113: 665-78.

^β Viability notations refer to homozygotes.

γ Fuller description follows "molecular biology".

cytology: Placed in 36A7-C1 on the basis of its inclusion in *Df(2L)H20 = Df(2L)36A7-10;36E4-F1* but not in *Df(2L)H68 = Df(2L)36B2-C1;37A1-B1* (Steward and Nüsslein-Volhard, 1986, Genetics 113: 665-78); located in 36B by *in situ* hybridization to salivaries (Bernstein *et al.*, 1983; Rozek and Davidson, 1983).

molecular biology: *Mhc* is a single copy gene in *Drosophila melanogaster* (in contrast to mammals, chickens, and nematodes which have families of myosin heavy chain genes). The *Drosophila Mhc* gene has been cloned and the complete nucleotide sequence of the genomic DNA and the derived amino acid sequence of the MHC protein obtained (Bernstein *et al.*, 1983, 1986; Rozek and Davidson, 1983, 1986; George *et al.*, 1989). The gene has a complex exon structure that, by means of regulated alternative RNA splicing, enables it to produce a variety of larval and adult muscle isoforms. Its 21 kb transcription unit contains 19 exons, 14 single-copy exons and five other exons that are tandemly repeated [two copies of exon 3 (Wassenberg *et al.*, 1987), four of exon 7, three of exon 9, five of exon 11, and two copies of exon 15 (George *et al.*, 1989; Hess *et al.*, 1989)]. In the clones studied by George *et al.* (1989), the repeated exons are spliced in a mutually exclusive manner, so that only one form of each exon set is included. Exon 18 shows differential splicing by inclusion or exclusion of the exon in pre-mRNA; it is expressed mainly in the thorax of late pupae and adults. Coding sequences for the ATP binding domain have been located in exon 4 (Wassenberg *et al.*, 1987; George *et al.*, 1989); the hydrophobic region next to the ATP-binding domain is encoded by alternative exons 3a/3b as well as by exon 4. Northern blots show transcripts of 6.1 and 6.6 kb in larval stages and transcripts of 6.1, 6.6, and 7.1 kb in adult stages. Positions of the 5' introns are conserved in flies, vertebrates, and nematodes (Wassenberg *et al.*, 1987; George *et al.*, 1989). Fusion genes have been constructed using the 5' end of the *E. coli lac-z* gene introduced into the genome by *P*-element gene transfer, and the transformed flies stained for expression of the fusion protein to identify sequences involved in muscle-specific expression (Hess *et al.*, 1989). The results indicate that these elements are located 450 nucleotides upstream and 2095 nucleotides downstream of the transcription initiation site.



Mhc: Muscle myosin heavy chain map

From figure supplied by Bernstein and Kronert. Program by D. Conner.

Mhc¹

phenotype: Homozygous embryos show no movement; unable to hatch; ultrastructural observations show complete lack of thick filaments in muscles. Heterozygotes display nearly a 50% reduction in the numbers of thick filaments in indirect flight muscles and the tergal-depressor-of-the-trochanter muscle, resulting in disruption of the normal regular array of thick and thin filaments in these muscles. Other less regularly organized muscles, although having reduced numbers of thick filaments, appear to function adequately in *Mhc*^{1/+} flies (O'Donnell and Bernstein, 1988, *J. Cell Biol.* 107: 260-12).

molecular biology: 101 base pair deletion which removes most of exon 5 and the intron that precedes it. The splice-donor site of exon 4 rather than that of exon 5 appears to interact with the exon-6 splice-acceptor site (O'Donnell and Bernstein, 1988). Deletion generates a nonsense mutation, which likely results in production of an unstable truncated protein.

Mhc⁵

phenotype: 20% of heterozygotes display indented thorax and erect wings and are flightless; the remainder have normal phenotype but fly poorly. Homozygotes display erect wing phenotype. Judged to be antimorphic since not rescued by addition of *Dp(2;3)osp*³. *Mhc*⁵ interaction in double heterozygotes with other flightless mutants observed by Homyk and Emerson (1988, *Genetics* 119: 105-21). Heterozygous viability severely reduced in combination with hemizygous *hdp*², *int*³, *up*¹⁰¹; or *up*^x; rare escapers have gnarled legs, walk poorly, and die within two days of eclosion. Females doubly heterozygous for *Mhc*⁵ and *hdp*², *int*³, *up*¹⁰¹; or *up*^x have normal viability but are completely flightless and display abnormal wing posture.

Mhc⁶

phenotype: Heterozygotes fly moderately well and display normal wing posture; hemizygotes flightless and occasionally have abnormal wing posture. Double heterozygotes with *hdp*¹⁰¹, *hdp*¹⁰², *int*³, *up*¹⁰¹; or *up*^x; but not *hdp*², much more nearly flightless than *Mhc*^{6/+}; wing posture normal.

Mhc⁷

phenotype: Indirect flight muscles accumulate little or no MHC, have no thick filaments, and show no organized myofibrils. The four smaller cells of the tergal depressor of the trochanter muscle (TDT) display reduction in thick filament number and myofibril size; large TDT cells unaffected. Flies jump 33% as well as wild type. Leg muscle MHC found in normal amounts (O'Donnell *et al.*, 1989).

Mhc⁸

phenotype: Heterozygotes display indented thorax and erect wings. Judged to be antimorphic since not rescued by addition of *Dp(2;3)osp*³. *Mhc*⁸ interaction in double heterozygotes with other flightless mutants observed by Homyk and Emerson (1988, *Genetics* 119: 105-21). Heterozygous viability severely reduced in combination with either heterozygous or hemizygous *hdp*², *int*³, and *up*¹⁰¹; lethal in *up*^x/Y males. No interaction with the following: *hdp*³, *hdp*⁴, *hdp*⁵, *hdp*¹⁰¹, *hdp*¹⁰², *up*², *up*³, or *up*¹⁰².

Mhc⁹

phenotype: Indirect flight muscles accumulate little or no MHC, have no thick filaments, and show no organized myofibrils. The four smaller cells of the TDT display reduction in thick filament number and myofibril size; large TDT cells unaffected. Flies jump 59% as well as wild type. Leg muscle MHC found in normal amounts (O'Donnell *et al.*, 1989).

Mhc¹⁰

phenotype: Indirect flight muscles accumulate little or no MHC, have no thick filaments, and show no organized myofibrils. All 32 TDT cells lack thick filaments and lack myofibril organization. Flies cannot jump. Leg muscles accumulate 55% normal amounts of MHC (O'Donnell *et al.*, 1989).

molecular biology: Mutation within 3' splice acceptor of exon 15a that encodes the central region of the MHC hinge (Collier, Kronert, O'Donnell, Edwards, and Bernstein, 1990, *Genes Dev.* 4: 885-95).

Mhc¹¹

phenotype: Indirect flight muscles accumulate little or no MHC, have no thick filaments, and show no organized myofibrils. No apparent effect on TDT; unique among alleles in being capable of jumping. Leg muscle MHC found in normal amounts (O'Donnell *et al.*, 1989).

Mhc¹²

phenotype: Indirect flight muscles accumulate little or no MHC, have no thick filaments, and show no organized myofibrils. The four smaller cells of the TDT display reduction in thick filament number and myofibril size; large TDT cells unaffected.

Mhc¹³

phenotype: A dominant flightless mutation; in homozygotes but not heterozygotes, the myofibrils of dorsolateral indirect flight muscles, although displaying normal morphology at eclosion, degenerate with time so that each cell is composed of a narrow strip of material connected to a bulged-out region. Some areas of the cells contain over-contracted sarcomeres and others show arrays of thick and thin filaments splayed throughout the cytoplasm. Abnormal morphology is recessive. Unlike the situation with other *Mhc* mutations, the dominant flightlessness of *Mhc*¹³ not rescued by the addition of a second dose of *Mhc*⁺.

Mhc¹⁶

phenotype: Heterozygotes display indented thorax and erect wings; flightless; hemizygotes semilethal, very inactive, have weak mesothoracic legs which are generally folded beneath the thorax; die prematurely after eclosion. Judged to be antimorphic since not rescued by addition of *Dp(2;3)osp*³. *Mhc*¹⁶ interaction in double heterozygotes with other flightless mutants observed by Homyk and Emerson (1988, *Genetics* 119: 105-21). Heterozygous viability severely reduced in combination with hemizygous *hdp*², *int*³, *up*¹⁰¹; or *up*^x; rare escapers have gnarled legs, walk poorly, and die within two days of eclosion.

Mhc-c: Myosin heavy chain-cytoplasmic
(D.P. Kiehart)**location:** 2-{108}.**references:** Kiehart and Feghali, 1986, J. Cell Biol. 103: 1517-25.

Young, Pesacreta, Rose, and Kiehart, 1987, J. Cell Biol. 105: 172a.

Kiehart, Lutz, Chan, Ketchum, Laymon, Nguyen, and Goldstein, 1989, EMBO J. 8: 913-22.

Ketchum, Stewart, Stewart, and Kiehart, 1990, Proc. Nat. Acad. Sci. USA 87: 6316-20.

Kiehart, Ketchum, Young, Lutz, Alfenito, Chang, Awobuluyi, Pesacreta, Inové, Stewart, and Chen, 1990, Ann. N.Y. Acad. Sci. 582: 233-51.

Young, Pesacreta, Rose, and Kiehart, 1991, Development 111: 1-14.

phenotype: Encodes a 205 kilodalton myosin heavy chain found in Drosophila cell lines and all Drosophila developmental stages. Antibodies raised against this protein crossreact, but weakly with muscle myosin heavy chain. First appears in preblastoderm embryos; diffusely distributed until syncytial blastoderm at which time localization to cortex and pole cells observed; at cleavage furrow, canals at the time of cellularization; transiently present at points of invagination during gastrulation (Young *et al.*, 1987, 1991).**cytology:** Localized to 60E9 by *in situ* hybridization (Jones and Young).**molecular biology:** A 50 kb walk that includes the 20.5 kb transcription unit for the gene is cloned; *in vitro* transcription and translation yields a 205 kd protein that reacts specifically with anti-cytoplasmic-myosin serum. Hybridizes with at least 2 *ca.* 6.3 kilobase messages on Northern blots that result from a differential splice at the 5' end of the gene (Kiehart *et al.*, 1989; Ketchum *et al.*, 1990).**other information:** Appears to be allelic with *zipper*. An EMS-induced allele (*Mhc-c*¹) produces a truncated myosin heavy chain on Western blots and fails to complement *zip*¹ and *zip*². Western blots also indicate *zip*² fails to accumulate myosin heavy chain.**mi: minus****location:** 2-104.7.**discoverer:** Biddle, 281.**references:** Bridges, 1937, Cytologia (Tokyo), Fujii Jub., Vol. 2: 745-55.**phenotype:** Bristles almost as small as hairs; hairs reduced in number and size. Body size small. Eclosion delayed. Viability low and erratic. Female entirely sterile; male fertile. RK2.**cytology:** Locus is in 59D6-E4 of salivary gland chromosome (Schultz) on the basis of its being between the right breakpoints of *In(2R)bw*^{VDe1} = *In(2R)41B2-C1;59E2-4* and *In(2R)bw*^{VDe2} = *In(2R)41A-B;59D6-E1*.***mib: miniature bristles****location:** 1-8.7.**origin:** X-ray induced.**discoverer:** Fahmy, 1956.**references:** 1959, DIS 33: 88.**phenotype:** Short, thin bristles. Body slightly darker than normal, particularly thorax and posterior border of tergites. Wings occasionally upheld and inner margins frequently incised. Male viable and sterile. RK3.**micro-oculus:** see *mo***Microcephalus:** see *Mc***microchaete:** see *mc***microptera:** see *mp***Microtubule associated protein-205****kd::** see *Map205***microwing:** see *mwg***mid: midline****location:** 2-16.**references:** Nüsslein-Volhard, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.**phenotype:** Homozygous embryonic lethal; denticle bands defective in ventral midline.**alleles:**

allele	origin	synonym	ref ^α
<i>mid</i> ¹	EMS	<i>mid</i> ^{1K}	2
<i>mid</i> ²	EMS	<i>mid</i> ^{1HS}	2
<i>mid</i> ³	EMS	<i>mid</i> ^{1HSD}	2
<i>mid</i> ^{4β}	EMS	<i>l(2)gdh-1</i> ¹¹	1
<i>mid</i> ^{5β}	EMS	<i>l(2)gdh-1</i> ¹⁴	1
<i>mid</i> ^{6β}	EMS	<i>l(2)gdh-1</i> ³¹	1
<i>mid</i> ^{7β}	EMS	<i>l(2)gdh-1</i> ¹¹⁹⁰²	1
<i>mid</i> ⁸	X ray	<i>mid</i> ^{h2}	3

^α 1 = Kotarski, Pickert, and MacIntyre, 1983, Genetics 105: 371-86.
2 = Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82; 3 = Szidonya and Reuter, 1989, Genet. Res 51: 197-208.

^β Allelism inferred from deficiency mapping, not complementation mapping.

cytology: Placed in 25D7-F3 based on its inclusion within the region of overlap between *Df(2L)c11* = *Df(2L)25D7-E1;25E6-F3* and *Df(2L)GpdhA* = *Df(2L)25D7-E1;26A8-9*.**midget:** see *mgt***midgoid:** see *mdg***midgut amylase pattern:** see *map***midline:** see *mid***min: mini****location:** 2-90.**references:** Procnier and Tartof, 1975, Genetics 81: 515-23.

Procnier and Dunn, 1978, Cell 15: 1087-93.

phenotype: The locus encoding the genes for 5S RNA. Heterozygous deficiency for the region is normal in phenotype; however, *min* mutants, although normal in phenotype when heterozygous or homozygous, express a phenotype indistinguishable from *bb* when hemizygous. Ordinarily, the haploid complement carries approximately 165 copies of 5S genes; in *min*-bearing chromosomes this number is reduced to approximately 80; according to Procnier and Tartof, this number is increased by compensation to 265 in hemizygotes for the normal allele. Eclosion of *min* hemizygotes delayed about two days; also show reduced viability which is more severe at 29° than at 25°.**alleles:** *min*⁰ spontaneous (Procnier and Tartof). *min*¹ and *min*², induced by triethylenemelamine (Procnier and Dunn).

cytology: Placed in 56E-F by *in situ* hybridization in one of the earliest uses of that technique (Wimber and Stefensen, 1970, *Science* 170: 639-41); Szabo notes two bands of label, at 56F1-2 and 56F6-7 (1974, *J. Cell Biol.* 63: 341). Also located between the breakpoints of $T(Y;2)L139 = T(Y;2)56E$ and $T(Y;2)L141 = T(Y;2)56F$; $T(Y;2)L62 = T(Y;2)56E-F$ is broken within the tandem array of sequences with approximately 80 copies on either side.

molecular biology: Region contains a tandem array of 380-base-pair repeats, each comprising 120 base pairs of gene and 170 base pairs of spacer sequence; the spacer is 33% GC and the gene 57% GC (Hershey, Condad, Sodja, Yen, Cohen, Davidson, Ilgen, and Carbon, 1977, *Cell* 11: 585-98). Segments of tandem array cloned and restriction mapped (Artavanis-Tsakonas, Schedl, Tschudi, Pirrotta, Steward, and Gehring, 1977, *Cell* 12: 1057-67; Tschudi and Pirrotta, 1980, *Nucleic Acid Res.* 8: 441-51); variation in restriction maps attributable to variable numbers (4-7) of a tandemly repeated heptamer in the spacer region plus occasional other restriction site polymorphisms. In addition some strains appear to have a continuous array of repeats, whereas others appear to contain a segment with a number of restriction sites that separates the array into two subsegments of approximately equal size (Junakorvic, 1980, *Nucleic Acid Res.* 8: 3611-22). Nucleotide sequence and proposed secondary structure of 5S RNA provided by Behnamon and Jordan (1976, *FEBS Lett.* 62: 146-46) and Thompson, Wegnez, and Hearst (1981, *J. Mol. Biol.* 147: 417-36). Following heat shock, 5S RNA synthesis is reduced and an abnormal product with 55 extra nucleotides appended to the 3' end appears instead; may be a precursor that does not get processed (Rubin and Hogness, 1975, *Cell* 6: 207-13). Linker-scanning mutation studies reveal five regions important for normal transcription; one between -39 and -26, presumably involved in polymerase binding, and four internal sequences at 3-18, 37-44, 48-61, and 79-98 (Sharp and Garcia, 1988, *Mol. Cell. Biol.* 8: 1266-74).

min: see *mnb*

miniature: see *m*

miniature blistered: see *mbs*

miniature bristles: see *mib*

minibrain: see *mnb*

minus: see *mi*

minus bar: see *mb*

Minute-producer: see $T(1;4)M-pro$

minute chaetae: see *mch*

Minute (): see *M()*

minutelike: see *ml*

Mio: see *Dr^{Mio}*

Mir: Mirabile (M. Muskavitch)

location: 3- (rearrangement).

origin: X ray induced.

discoverer: Muskavitch.

phenotype: Mirror-image duplication of tergite structure.

Microchaetae are eliminated from the anterior portion of the tergite and replaced by a duplication consisting of an anteriorly oriented row of macrochaetae and the darkly pigmented cuticle normally found in the posterior portion of the tergite. Fat body and oenocytes underneath the tergite are also duplicated with mirror-image symmetry (Madhavan and Madhavan).

alleles: Original allele dominant and revertable by X ray mutagenesis suggesting that this allele constitutes a gain-of-function mutation. Animals heterozygous for any one of seven independent X- ray-induced revertant alleles, designated *Mir^{rv1}* through *Mir^{rv7}*, and a wild-type allele exhibit wild-type tergite structure.

cytology: Associated with $T(1;3)Mir$ with the new order 1 - 20F|81F - 64C|94A - 81F|94A-100F; 20F|64C - 61A (Gelbart). Analysis of segregation behavior of the phenotype in relation to the components of an X ray-induced detachment of $T(1;3)Mir$ indicates that the mutation maps to the third chromosome.

other information: Not allelic to disembodied, hedgehog, pointed, rhomboid or shrew as assessed on the basis of lethal complementation tests employing representative alleles of these five loci.

***mis: misproportioned**

location: 1-1.3.

origin: Induced by 1:4-dimethanesulfonylbut-2-yne (CB. 2058).

discoverer: Fahmy, 1951.

references: 1938, *DIS* 32: 71.

phenotype: Abdomen deformed: in male, large and broad; in female, tergites abnormal and hairs disarranged. Wings shortened in both sexes. Bristles thin. Body color rather pale. Eclosion slightly delayed. Male viability and fertility normal; female viability 50% wild type. RK3.

other information: One allele each induced by CB. 1540 and CB. 3034.

misformed: see *msf*

misheld wings: see *mwi*

misp: misstep (T. Schüpbach)

location: 2-59.

origin: Induced by ethyl methanesulfonate.

synonym: *misp^{RQ}*.

references: Schüpbach and Wieschaus.

phenotype: Maternal-effect lethal, female sterile. Embryos from homozygous mothers do not hatch. In cuticle preparations they show irregular segmentation and variable segment fusions.

misproportioned: see *mis*

missing: see *msg*

misstep: see *misp*

mit: mitotic loss inducer

location: 1-57 (between *f* and *Bx*).

origin: Spontaneous.

references: Gelbart, 1974, *Genetics* 76: 51-63.

phenotype: Mosaics produced by loss of maternal or paternal chromosomes in F_1 of homozygous *mit* females; progenies of *mit* males normal. Chromosome loss (*X* or *4*) usually occurs at third or fourth mitotic division of the

zygote, producing mosaic patches of intermediate size. Rod-X chromosomes lost as frequently as rings, but neither XY, nor Y chromosomes eliminated frequently by *mit*. Relative frequencies of observed loss of maternal and paternal X chromosomes depend on the markers carried rather than the parental origin of the X's. Double mosaic gynandromorphs indicative of independent loss of homologous chromosomes in different lineages. No increase in meiotic nondisjunction or chromosome loss observed. *mit* stocks show good viability and fertility, though modifiers reducing frequency of mosaics tend to accumulate. *mit*-induced gynandromorphs useful in constructing morphogenetic fate maps.

cytology: Placed in 16A6-F8 since *mit* is proximal to *f* (in 15F) and not included in *Df(1)B263-20 = Df(1)15F9-16A1;16A6-7* or in *Dp(1;1)Bxr = Dp(1;1)17A;17E-F*.

mit(1): mitotic (1)

A group of fourteen loci identified as temperature-sensitive lethal mutations (Baker) that exhibit, at semirestrictive temperatures, elevated frequencies of clones of homozygous *mwh* cells in the wings of surviving *mit(1);mwh/+* males and of γ clones and $\gamma//mwh$ twin spots in the abdomens of γ *mit(1);Dp(1;3)sc^{J4}*, γ^+ *mwh/+* males. The presence of large clones is consistent with origin via mitotic exchange, mitotic nondisjunction of both homologues, or mutation; twin spots are not expected to result from somatic mutation; only mitotic nondisjunction produces γ *Sb⁺* clones in *Dp(1;3)sc^{J4}*, γ^+ *Sb/+*. Preponderance of small clones suggests origin via chromosome breakage. Mitotic chromosome morphology examined in larval ganglion cells of *mit(1)* males. *inter se* allelism tests have not been performed.

locus	genetic location	phenotype
<i>mit(1)2</i>	1-15.3	small clones; few twin spots; 1.16% chromatid breaks
<i>mit(1)3</i>	1-46.1	small clones; twin spots; 1.63% chromatid and isochromatid breaks
<i>mit(1)4</i>	1-28.0	large clones; twin spots; chromosomes undercondensed
<i>mit(1)5</i>		large clones; twin spots; 2.46% chromatid and isochromatid breaks
<i>mit(1)6</i>	1-34.9	small clones; few twin spots; chromosomes normal
<i>mit(1)7</i>	1-15.7	large clones; few twin spots; 1.47% chromatid, isochromatid breaks, and exchanges
<i>mit(1)8</i>		large clones; few twin spots; 1.72% chromatid and isochromatid breaks
<i>mit(1)9</i>		small clones; few twin spots; 1.13% chromatid, isochromatid breaks, and exchanges
<i>mit(1)10</i>	1-1.4	large clones; few twin spots; chromosomes normal
<i>mit(1)11</i>	1-21.4	small clones; few twin spots; 0.9% chromatid and isochromatid breaks
<i>mit(1)12</i>		large clones; few twin spots; 0.99% chromatid and isochromatid breaks
<i>mit(1)13</i>	1-36.0	large clones; γ <i>Sb⁺</i> clones; 1.68% chromatid and isochromatid breaks
<i>mit(1)14</i>	1-49.0	small clones; γ <i>Sb⁺</i> clones; 7.20% chromatid and isochromatid breaks
<i>mit(1)15</i>	1-1.2	large clones; γ <i>Sb⁺</i> clones; mitotic nondisjunction

mit(1)15

synonym: *abe*; *l(1)zw10*.

phenotype: Semilethal as males or homozygous females; surviving flies have reduced roughened eyes, bristles on the head and thorax sometimes missing, abnormal wings with thin texture and thickened veins; both sexes sterile.

Up to 100 fold increase in the incidence of *mwh* clones per wing in *mit15;mwh/+* flies raised at sub-restrictive temperature. Half of all metaphases in larval ganglion cells are hyperploid for one or more chromosomes; hyperploidy for nearly all combinations of sex chromosomes and autosomes are observed. Chromosome breakage seen in less than 2% of cells. Cytological observations in *mit(1)15¹⁵*, *mit(1)15³* and *mit(1)15⁴* at either restrictive or permissive temperature.

alleles:

allele	origin α	discoverer	synonym	ref β	comments
<i>mit(1)15¹</i>	NNG	Reichert	<i>l(1)zw10^{1c}</i>	1	
<i>mit(1)15²</i>	EMS	Kaufman	<i>l(1)zw10⁸²</i>	1	
<i>mit(1)15³</i>	X ray + EI	Alexander	<i>l(1)zw10^{h10}</i>	1, 6	temperature sensitive
<i>mit(1)15⁴</i>	EI	Alexander	<i>l(1)zw10ⁱ²⁰</i>	1, 6	temperature sensitive
<i>mit(1)15⁵</i>	X ray + EI	Alexander	<i>l(1)zw10ⁱ²¹</i>	1	
<i>mit(1)15⁶</i>	EMS		<i>l(1)zw10^{e36}</i>	4	
<i>mit(1)15⁷</i>	EMS		<i>l(1)zw10^{e87}</i>	4	
<i>mit(1)15⁸</i>	EMS		<i>l(1)zw10^{e90}</i>	4	
<i>mit(1)15⁹</i>	TEM		<i>l(1)zw10²⁴</i>	4	
<i>mit(1)15¹⁰</i>	TEM		<i>l(1)zw10¹¹⁶</i>	4	
<i>mit(1)15¹¹</i>	MMS		<i>l(1)zw10^{m16}</i>	5	
<i>mit(1)15¹²</i>	MMS		<i>l(1)zw10^{m29}</i>	5	
<i>mit(1)15¹³</i>	MMS		<i>l(1)zw10^{m87}</i>	5	
<i>mit(1)15¹⁴</i>	EMS	Lefevre	<i>l(1)VE630</i>	3	
<i>mit(1)15¹⁵</i>	EMS	Baker	<i>l(1)zw10¹⁵</i>	6	temperature sensitive
<i>mit(1)15¹⁶</i>	spont	Schalet	<i>l(1)6-99</i> <i>l(1)zw10^{S1}</i>		
<i>mit(1)15¹⁷</i>	mei9 γ	Schalet	<i>l(1)zw10^{S2M}</i>		
<i>mit(1)15¹⁸</i>	HMS		<i>l(1)HM468</i>	2	

α EI = ethylenimine, HMS = hycanthon methanesulfonate, NNG = N'-nitro-N-nitrosoguanidine.

β 1 = Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56; 2 = Kramers, Schalet, Paradi, and Huizer-Hoogtyeyling, 1983, Mutation Res. 107: 187-201; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 4 = Lim and Snyder, 1974, Genet. Res. 24: 1-10; 5 = Liu and Lim, 1975, Genetics 79: 601-11; 6 = Smith, Baker, and Gatti, 1985, Genetics 110: 647-70.

γ Spontaneous in the paternal X chromosome of a cross between wild-type males and *mei9* females, such that the F_1 females were *mit(1)15/mei9*.

cytology: Located at 3A8 by Judd, Shen, and Kaufman; included in *Dp(1;2)w⁺70h31 = Dp(1;2)3A6-8;3C2-3* but not in *Df(1)64j4 = Df(1)3A8-9;3B1-2*.

mitotic: see *mit*

mk: murky

location: 1-0.8.

origin: Induced by triethylenemelamine (CB. 1246).

discoverer: Fahmy, 1950.

references: 1958, DIS 32: 71-72.

phenotype: Small fly with dull red eyes and extra body pigmentation; trident pattern especially marked. Delayed eclosion. Male fertile and viability 50% wild type; *mk/+* daughters, but not *mk* sons of *mk* mothers survive (Mohler, 1977, Genetics 85: 259-72). RK3.

alleles:

allele	origin	discoverer	synonym	ref α	comments
<i>mk¹</i>	CB1246	Fahmy, 1950		1	
<i>mk²</i>	CB1414	Fahmy, 1950		1	
<i>mk³</i>	CB1506	Fahmy, 1950		1	
<i>mk⁴</i>	CB1540	Fahmy, 1950		1	
<i>mk⁵</i>	CB3007	Fahmy, 1950		1	
<i>mk⁶</i>	CB3025	Fahmy, 1950		1	
<i>mk⁷</i>	CB3025	Fahmy, 1950		1	

allele	origin	discoverer	synonym	ref ^α	comments
<i>mk</i> ⁸	CB3034	Fahmy, 1950		1	
<i>mk</i> ^{Ml}	EMS	Mohler	<i>fs(1)M7</i>	2, 3	temperature sensitive

^α 1 = Fahmy, 1959, DIS 32: 71-72; 2 = Mohler, 1977, Genetics 85: 259-72; 3 = Mohler and Carroll, 1984, DIS 60: 236-41.

****ml*: minutelike**

location: 3-46.
discoverer: Mohr, 24c3.
synonym: *sb*: *short-bristle*.
references: 1924, Br. J. Exp. Biol. 2: 189-98 (fig.).
phenotype: Bristles small, as in Minute. Late hatching and poorly fertile. RK3.
alleles: **ml*² (Nichols-Skog, 36c); spontaneous; allelism inferred from phenotype and location on third chromosome.

***Mlc1*: Myosin light chain**

location: 3- {95}.
references: Falkenthal, Parker, Mattox, and Davidson, 1984, Mol. Cell. Biol. 4: 956-65.
 Falkenthal, Parker, and Davidson, 1985, Proc. Nat. Acad. Sci. USA 82: 449-53.
phenotype: Encodes the myosin alkali light chain (MLC-ALK), so called because it dissociates from MHC only at high pH. Expressed during periods of myogenesis: late embryo, larvae, late pupae, and adults; not expressed in early embryos or in early pupae during histolysis of larval muscle.

cytology: Placed in 98B on basis of *in situ* hybridization.
molecular biology: Genomic clones isolated by screening genomic library with cDNA from stages of active myogenesis. Sequence analysis of genomic and cDNA clones indicates the presence of six exons separated by introns varying in length from 59 to 986 nucleotides; five polyadenylation signals detected within and downstream from the sixth exon; postulated to account for the variation in transcript size. Differential splicing produces one transcript with all six exons, which has a stop codon in exon 5 and one that lacks exon 5 and has the stop codon in exon 6; these generate polypeptides that differ in fourteen C-terminal amino acids; the former is found in both larvae and pupae, whereas the second is found only in pupae. Open reading frames imply a polypeptide of 155 amino acids; the inferred amino-acid sequence reveals a region of especially high homology with one of the three Ca²⁺ binding domains of chicken MLC-ALK.

Mlc2

location: 3- {102}.
synonym: *Ifm(3)99Eb*.
references: Parker, Falkenthal, and Davidson, 1985, Mol. Cell. Biol. 5: 3058-68.
 Warmke, Krevz, and Falkenthal, 1989, Genetics 122: 139-51.
phenotype: Encodes a myosin light chain homologous to chicken MLC2. Expression abundant in late embryo, first and third larval instars, late pupae and adults; no expression in early embryos or early pupae. Phosphorylation of the polypeptide determined to be necessary for filament assembly (Hayashi and Hotta, 1982, Dev. Growth Differ. 24: 417).
alleles: One allele recovered as a dominant flightless

mutant in combination with a closely linked recessive lethal, *l(3)99Df¹* by Merriam (Warmke *et al.*). Heterozygous deficiency for the locus also flightless.

cytology: Placed in 99E1-3 by *in situ* hybridization.
molecular biology: Genomic clone independently isolated by screening genomic libraries with cDNA from stages of active myogenesis by Parker *et al.* and by Toffenetti, Mischke, and Pardue (1987, J. Cell Biol. 104: 19-28) and by screening with blastoderm cDNA by Roark, Mahony, Graham, and Lengyel (1985, Dev. Biol. 109: 476-88). Sequence determination from genomic and overlapping cDNA clones reveals alternative transcription start sites 67 and 55 nucleotides upstream from the first codon as well as two termination sites 109 and 259 nucleotides 3' to the stop codon; also there is no TATA box. Two introns, one of 699 nucleotides between amino-acid residues 1 and 2 and the other of 169 nucleotides between residues 82 and 83; these intron positions are conserved in rat MLC2. Northern blots reveal two abundant mRNA's of 1.1 and 1.4 kb plus a minor transcript of 2.7 kb; *in vitro* translation of hybrid-selected mRNA yields polypeptides of 17 and 26 kilodaltons; amino-acid sequence inferred from the nucleotide sequence suggests high Ca²⁺ binding affinity; also the serine that is phosphorylated in mammalian MLC2 is conserved in the *Drosophila* polypeptide, but is not known to be the site of phosphorylation. As in several vertebrate muscle proteins, *Drosophila* MLC2 has an acylated amino terminus (Toffenetti *et al.*).

other information: A series of alleles recovered on the basis of semilethality in combination with a deficiency for 99D3-E3 and behaving as dominant flightless mutations turn out to map to the same complementation group at 3-54.2 in region 88; the locus is designated *l(3)nc99Eb* by Warmke *et al.*

***Mlc-c*: Myosin alkali light chain cytoplasmic**
 (D.P. Kiehart)

location: 1- {14}.
references: Chang, Edwards, and Kiehart, in preparation.
phenotype: The structural gene for the alkali light chain of non-muscle myosin (whose other subunits are encoded by *Mhc-c* and *sqh*).
cytology: Localized to 5A6 by *in situ* hybridization of genomic clones.
molecular biology: Cloned using oligonucleotides corresponding to partial peptide sequence of purified non-muscle myosin light chain. A cDNA detects a 1.3 kb transcript at all developmental stages and encodes a 16 kd protein with 60-70% identity to vertebrate smooth-muscle-myosin alkali light chain.

***mle*: maleless**

location: 2-55.2 (just distal to *ap* based on deficiency mapping).
references: Fukunaga, Tanaka, and Oishi, 1975, Genetics: 81: 135-41.
 Tanaka, Fukunaga, and Oishi, 1978, Genetics 84: 257-66.
phenotype: Homozygous males die, but homozygous females survive. Males produced by homozygous females die during the third larval instar, whereas those produced by heterozygous females are late pupal lethals. Females transformed into phenotypic males (*tra*) or intersexes (*dsx*) unaffected by *mle*, i.e. *mle* acts only upon

single-X-bearing flies. No interaction with *msh-1* or *msh-2* (Belote, 1983, Genetics 96: 165-86). *mle*⁴ males surviving at 18° are sterile, small, and slow developing. Concluded to be defective in dosage compensation in males based on decreased levels of X-linked-enzyme activities (G6PD, 6GPD, FUM, β-HAD) but not autosomally encoded enzymes (ADH, AO, GPDH, IDH) in homozygous *mle*⁴ male larvae and escaping adults, e.g. β-HAD. The incorporation of labeled uridine by the polytene X chromosome relative to that of 2R is lower than normal in *mle*⁴ males (Belote and Lucchesi, 1980, Nature 285: 573-75); steady-state level of *Sgs4* mRNA incompletely compensated in *mle*⁴ and *mle*⁴*mle*⁶ male larvae (Breen and Lucchesi, 1986, Genetics 112: 483-91). Polytene X chromosome of *mle* males appears narrower and more densely stained than that of control males. Few homozygous *mle* gynandromorphs survive; XO patches small, with small bristles, and mostly confined to abdomen (Uenoyama, Uchida, Fukunaga, and Oishi, 1982, Genetics 102: 223-31). *mle* pole cell transplanted into wild-type hosts incapable of undergoing normal spermatogenesis (Bachiller and Sánchez, 1986, Dev. Biol. 118: 379-84). Homozygous (and to a lesser extent heterozygous) *mle* females that are heterozygous for *Sxl*^{F1} (Uenoyama, Fukunaga, and Oishi, 1982, Genetics 102: 233-43; Skripsky and Lucchesi, 1982, Dev. Biol. 94: 153-64) or are the surviving progeny raised at 17° of homozygous *da* mothers (Cline, 1982, Genetics 100: 641-63) develop as intersexes.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>mle</i> ¹	spont	Oshima, 1971		4, 10, 11	male lethal
<i>mle</i> ^{1a}	spont	Watanabe, 1978	<i>mle</i> ^{79K}	11	when homozygous independent isolate of <i>mle</i> ¹ ?
<i>mle</i> ^{1b}	spont	Watanabe, 1979	<i>mle</i> ^{K25}	11	independent isolate of <i>mle</i> ¹ ?
<i>mle</i> ²			<i>mle</i> ^{MAK β}	5	male lethal
<i>mle</i> ³	EMS		<i>mle</i> ¹³	11	when homozygous male lethal
<i>mle</i> ⁴	EMS	Belote, 1977	<i>mle</i> ^{ts}	1, 2, 3 11	when homozygous male lethal; heat sensitive lethality; 18° survivors sterile, small and slow to develop
<i>mle</i> ⁵	spont		<i>mll</i> , <i>mle</i> ^{Roma γ}	7	
<i>mle</i> ⁶	EMS		<i>mle</i> ¹⁸⁵	3, 10	
<i>mle</i> ⁷	γ ray		<i>mle</i> ^{Pml2}	8	
<i>mle</i> ⁸	γ ray		<i>mle</i> ^{Pml8}	8	
<i>mle</i> ⁹	γ ray		<i>mle</i> ^{γ38}	9	male lethal
<i>mle</i> ¹⁰	γ ray		<i>mle</i> ^{γ203}	9	when homozygous male lethal
<i>mle</i> ¹¹	γ ray		<i>mle</i> ^{γ22}	9	when homozygous male lethal
<i>mle</i> ¹²	γ ray		<i>mle</i> ^{γ235}	9	when homozygous male lethal
<i>mle</i> ¹³	γ ray		<i>mle</i> ^{γ28}	9	when homozygous male lethal
<i>mle</i> ¹⁴	γ ray		<i>mle</i> ^{γ293}	9	when homozygous male lethal
<i>mle</i> ¹⁵	EMS		<i>mle</i> ^{RK}	6	when homozygous male lethal

^α 1 = Belote and Lucchesi, 1980, Genetics 96: 165-86; 2 = Belote and Lucchesi, 1980, Nature 285: 573-75; 3 = Breen and Lucchesi, 1986,

Genetics 112: 483-91; 4 = Fukunaga, Tanaka, and Oishi, 1975, Genetics 81: 135-41; 5 = Golubovsky and Ivanov, 1972, DIS 49: 117; 6 = Kernan, unpublished; 7 = Loverre and Cicchetti, 1980, DIS 55: 88; 8 = Schüpbach, unpublished; 9 = Scott and Lucchesi, unpublished; 10 = Tanaka, Fukunaga, and Oishi, 1978, Genetics 84: 257-66; 11 = Uenoyama, Uchida, Fukunaga, and Oishi, 1982, Genetics 102: 223-31.

β Allelism confirmed by Ashburner.

γ Allelism inferred from map position at 2-55.2.

cytology: Placed in 42A2-8 as it is uncovered by *Df(2R)nap9 = Df(2R)42A (?)* (Kreber).

molecular biology: Locus cloned by Kuroda, Kernan, Kreber, Ganetzky, and Baker. Molecular analyses of *nap* and *mle* indicate that the same open reading frame encodes *mle*⁺, *nap*⁺ and *nap*^{ts} activities.

other information: *mle* alleles fail to complement the paralysis caused by *nap* alleles, indicating that *nap* and *mle* are allelic (Kernan and Ganetzky).

mle3

location: 3-25.8.

references: Uchida, Uenoyama, and Oishi, 1981, Jpn. J. Genet. 56: 523-27.

phenotype: Homozygous males exhibit delayed development; survive as larvae for ten days. Ten-day-old male larvae have small undeveloped imaginal discs, which, however, when transplanted into wild type larvae are capable of undergoing nearly complete (wing, leg) or partial (eye-antenna) differentiation; few homozygous *mle* gynandromorphs survive; XO patches small, with small bristles, and mostly confined to abdomen [Uenoyama, Uchida, Fukunaga, and Oishi, 1982, Genetics 102: 223-31(fig.)]. No interaction with *mle*. Females transformed into phenotypic males (*tra*, *tra2*) or intersexes (*dsx*) unaffected by *mle3*, i.e. *mle3* acts only upon single-X-bearing flies. Females heterozygous for *Sxl*^{F1} and homozygous (and to a lesser extent heterozygous) for *mle3* show signs of intersexual development; effect less severe when mothers homozygous for *mle3* [Uenoyama, Fukunaga, and Oishi, 1982, Genetics 102: 233-43 (fig.)]. Staining of polytene X chromosome of *mle3* males similar to that of *mle* males (Lucchesi, Skripsky, and Tax, 1982, Genetics 100: s42; Okuno, Satou, and Oishi, 1984, Jpn. J. Genet. 59: 237-47).

alleles:

allele	origin	synonym	ref ^α
<i>mle3</i> ¹	spont	<i>mle(3)132</i>	3, 4
<i>mle3</i> ²	EMS	<i>mst-3</i>	2
<i>mle3</i> ^{3 β}	EMS	<i>mst-3</i> ^b	1, 2

^α 1 = Bachiller and Sánchez, 1989, Roux's Arch. Dev. Biol. 198: 34-38; 2 = Lucchesi, Skripsky, and Tax, 1982, Genetics 100: s42; 3 = Okuno, Satou, and Oishi, 1984, Jpn. J. Genet. 59: 237-47;

β 4 = Uchida, Uenoyama, and Oishi, 1981, Jpn. J. Genet. 56: 523-27. Cell autonomous; clones homozygous for mutant are deleterious (Bachiller and Sánchez, 1989).

mll: see *mle*

mmy: mummy

location: 2-16.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82; Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26. (fig.).

phenotype: Embryonic lethal; mouth parts and denticles

poorly differentiated.

alleles: Five including one temperature-sensitive and one weak allele. *mmy*¹ and *mmy*² (isolated as *IK* and *IL*) retained.

mMdh: see *Mdh2*

mn: manikin

location: 1-38.4.

origin: Induced by D-p-N,N-di-(2-chloroethyl)amino-phenylalanine.

discoverer: Fahmy, 1954.

synonym: *dwX*^{mn}.

references: 1959, DIS 33: 88.

phenotype: Fly small with narrow abdomen. Reduction in size may be bilaterally asymmetrical and may affect abdomen and thorax independently. Male viability reduced; flies rarely survive more than 48 hr. Sterile, probably owing to reduced vigor. RK3.

alleles: One X-ray-induced allele.

mnb: minibrain (J.C. Hall)

location: 1-58.2 (apparently part of "Sh complex").

origin: Induced by ethyl methanesulfonate or from hybrid dysgenesis.

discoverer: M. Heisenberg (from a mutagenized X provided by J. Merriam).

synonym: *min*.

references: Fischbach and Heisenberg, 1984, J. Exp. Biol. 112: 65-93.

Heisenberg and Wolf, 1984, Vision in Drosophila: Genetics of Microbehavior, Springer-Verlag, Berlin, pp. 207, 209, 227.

Heisenberg, Borst, Wagner, and Byers, 1985, J. Neurogenet. 2: 1-30.

Helfrich, 1986, J. Neurogenet. 3: 321-43.

phenotype: Brain mass approximately half normal, including smaller than normal optic lobes and reduction in cell number (Fischbach and Heisenberg, 1984; Heisenberg and Wolf, 1984); exceptions: neuropile of the lamina optic ganglion appears normal in size and general morphology, and peduncle of mushroom body has apparently normal number of fibers; *mnb* flies take abnormally long to eclose (*i.e.*, emergence *per se* from the pupal case is a slow process); behaviorally, *mnb* leads to relatively subtle behavioral defects, such as mild leg shaking under ether (see "other information"), absence of learning in tests using olfactory stimuli (Heisenberg *et al.*, 1985), and aberrant visual fixation. In tests of locomotor activity rhythms (Helfrich, 1986), singly mutant *mnb* adults are basically normal, but a high proportion of *mnb so* double mutants show complex rhythmicities (dual circadian periodicities) with about 20% being arrhythmic (3 times greater than wild type). Other behaviors, such as basic optomotor responses (M. Heisenberg, unpublished) and male courtship song (Kulkarni and Hall, 1987, Genetics 115: 461-475), are normal; physiologically, *mnb*--when linked to *reduced optic lobes* and *small optic lobes* mutations--has been tested for effects on electroretinogram, which have diminished amplitudes of light-on and light-off transient spikes in the triple mutant (Coombe, 1986, J. Comp. Physiol. 159: 655-665).

alleles: Two alleles: *mnb*¹ (Heisenberg) induced by ethyl methanesulfonate and *mnb*² originally designated *mnb*^{UB913}, produced by hybrid dysgenesis and has a P-

element insertion in 16EF.

cytology: Located in 16E3-F3 based on coverage of *mnb*¹ by proximal X duplication in *Dp(1;3)JC153* = *Dp(1;3)16E3-4;17AB;99D*, but failure to be covered by duplication segregating from *T(1;Y)B55* = *T(1;Y)16F2-3* or *T(1;Y)W32* = *T(1;Y)16F3-4*; females carrying the deletion segregating from *Tp(1;3)JC153* plus the X^{PYD} element of *T(1;Y)W32*, with *mnb*¹ being on the homologous X, have the mutation's effects uncovered.

other information: Seems to be allelic to *Sh*, given map position and *mnb*-associated leg shaking (A. Ferrus and M. Heisenberg, unpublished); yet, *Sh* mutations do not lead to any grossly aberrant brain morphology, and *Sh*⁵ plus *Sh*¹⁴ complement *mnb*¹ with regard to anatomical defects caused by the latter (M. Heisenberg, unpublished); *mnb*¹ also complements lethal alleles of 3 genes mapping just distally to *Sh* alleles (see Tanouye, Lam, and Iverson, 1986, Ann. Rev. Neurosci. 9: 255-76), *i.e.*, *l(1)16Fa*, *l(1)16Fb* and *l(1)16Fc*; an X-linked enhancer of *mnb* has been identified, which causes lethality when expressed along with the latter (M. Heisenberg, unpublished); the former maps near *cv* and (when in combination with *mnb*¹) leads to death late in pupation (mutants can be "rescued" by opening the pupal case).

mo: micro-oculus

location: 1-6.7.

origin: Induced by DL-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).

discoverer: Fahmy, 1954.

references: 1958, DIS 32: 72.

phenotype: Eyes small. Wings narrow and frequently pleated longitudinally with irregular hairs, giving slight opacity. Body size slightly reduced. Not easily classified. Viability and fertility good in both sexes. RK3.

other information: Two alleles each induced by CB. 3007 and CB. 3026; four induced by CB. 1528; one each induced by CB. 1506, CB. 1540, CB. 1592, and CB. 3025.

mo: see *moo*

Mo: see *Me*

Mo^K: see *Mot-K*

mod: see *e(gs)*

mod: see *rsd*

modifier of Bar: see *Su(B)*

modifier of garnet: see *e(g)*

modifier of sexual dimorphism of glass: see *msd(gl)*

moira: see *mor*

Moiré: see *Me*

Monoplane: see *Mpl*

***moo: moorish**

location: 3-48.3.

origin: X ray induced.

discoverer: Thompson.

synonym: *mo* (preoccupied).

references: 1959, DIS 33: 99.

phenotype: Body color black. Homozygous lethal in male; female viability about 10% normal. RK3.

Moonrat: see *Mrt*

moorish: see *moo*

mor: *moira* (J.A. Kennison)

location: 3-58.1 (based on 18 recombinants between *jvl* and *sbd*²).

origin: Induced by ethyl methanesulfonate.

discoverer: Kennison, 1983.

references: Kennison and Tamkun, 1988, Proc. Nat. Acad. Sci. USA 85: 8136-40.

phenotype: Isolated as a dominant suppressor of the antenna-to-leg transformation in a *Pc*² *Antp*^{Ns} double heterozygote. Strongly suppresses the antennal transformation in an *Antp*^{Ns} heterozygote, but does not suppress the antennal transformation in a strain containing a heat-shock driven Antennapedia cDNA. Also behaves as a dominant suppressor of *Pc* and *Pcl* mutations. All alleles associated with a common recessive embryonic lethality. Mitotic clones induced during larval growth lead to transformation of haltere tissue to wing tissue. *Ubx*¹³⁰/*mor* has low frequency of haltere to wing transformation.

alleles:

alleles	origin
<i>mor</i> ¹	EMS
<i>mor</i> ²	EMS
<i>mor</i> ³	EMS
<i>mor</i> ⁴	EMS
<i>mor</i> ⁵	γ ray
<i>mor</i> ⁶	γ ray

cytology: Placed in 89A11-B4 based on its inclusion in *Df(3R)sbd105* = *Df(3R)88F9-89A1;89B9-10* but not in *Df(3R)Po4* = *Df(3R)88F7-89A1;89A11-13* nor *Df(3R)sbd45* = *Df(3R)89B4;89B10* (Hughes, Nelson, Yanuk, and Szauter).

morula: see *mr*

Mos

location: 3-.

origin: Spontaneous.

references: Golubowsky and Zakharov, 1979, Genetika 15: 1798-1808.

phenotype: Dominant factor on third chromosome that reduces dorsocentral, scutellar, and occasionally other thoracic bristles; penetrance 30-50%, higher in the presence of unstable *sn*⁺ alleles. Causes increased rates of mutation of unstable *sn*⁺ in both germ line and soma.

other information: Probably a manifestation of hybrid dysgenesis.

***mot-28: mottled**

location: 3-46.0.

origin: Found among progeny of males given supersonic treatment.

discoverer: Hersh, 28i19.

references: Hersh, Karrer, and Loomis, 1930, Am. Nat. 64: 552-59.

Hersh, 1934, DIS 1: 30.

Surrarer, 1935, Genetics 20: 357-62 (fig.).

1938, Genetics 23: 631-46 (fig.).

1940, DIS 13: 51.

phenotype: Eyes mottled with patches of dark brown or black on wild-type background. Sensitive to temperature. Always mottled at 18°, almost never above 25°. Temperature-effective period is 25-35 hr after beginning of pupation. Mottling more easily seen in presence of *v*, also manifested in *w* homozygotes (Schultz). RK1 at 18°; RK3 above 25°.

***mot-32l**

location: 1- (not located).

origin: X ray induced.

discoverer: Oliver, 32l28.

references: 1937, DIS 7: 19.

phenotype: Eye color mottled in female only. RK3.

***mot-36e**

location: 3- [left arm, with *In(3L)P*].

discoverer: Bridges, 36e11.

references: 1937, DIS 7: 12.

phenotype: Eyes mottled with translucent spots and roughness. Bristles twisted and stubby; hairs irregular. Wing venation plexoid around posterior crossvein. Female sterile. Enhances somatic crossing over in first, second, and third chromosomes. RK3.

Mot-K: Mottled of Krivshenko

location: 2- or 3- (rearrangement).

origin: X ray induced.

discoverer: Krivshenko, 54c25.

synonym: *Mo*^K.

references: 1954, DIS 28: 75.

1955, DIS 29: 76.

phenotype: Eyes liberally mottled with dark color on wild-type background; character barely noticeable in young flies but striking in older ones; number and size of spots variable. Homozygous lethal. Viability and fertility of heterozygotes good. RK2A.

cytology: Associated with *T(2;3)Mot-K* = *T(2;3)41;60D;80-81*.

mottled: see *mot*

mottler of white: see *mw*

mp: microptera

location: 3-0.0.

discoverer: Serebrovsky, 40g8.

references: 1941, DIS 15: 19.

phenotype: Wings small and spoonlike; veins irregular. Tarsi four jointed (rarely 3 or 5); joints 3 and 4 usually fused. Antennae shortened. Ecloses somewhat late. Viability and fertility low. RK2.

Mp20: Muscle protein 20 kd

location: 3-{68}.

references: Ayme-Southgate, Lasko, French, and Pardue, 1989, J. Cell Biol. 108: 521-31.

phenotype: Encodes a 20-kd protein, that is not detected in the asynchronous oscillatory flight muscles, but is found in most, if not all, other muscles (the synchronous muscles).

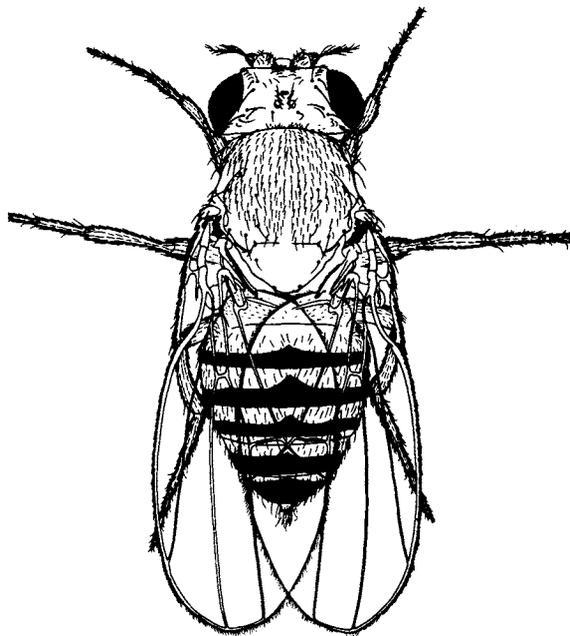
cytology: Placed in 49F9-13 based on *in situ* hybridization and deficiency mapping.

molecular biology: Identified in a cDNA library produced from pulsating cultured myotubes and prehybridized with mRNA from Schneider cells. The cDNA clones identify two myotube-specific transcripts of 0.9 and 1.0 kb, the

difference residing in the 3' untranslated region. Southern blots indicate that the gene is not a member of a multigene family. The coding sequence contains two introns, one of 140 nucleotides between codons 10 and 11, and the other of 67 nucleotides between codons 121 and 122. The open reading frame encodes a polypeptide of 184 amino acids with a molecular mass of 20,166 daltons. Two stretches of twelve amino acids match the proposed consensus sequence for the calcium binding site of calcium-binding proteins are found between residues 20-31 and 93-104. No other significant sequence homologies were found in the protein data base.

Mpl: Monoplane

location: 3-{50}.
origin: X ray induced.
discoverer: Shelton.
references: Hughes and Shelton, 1980, DIS 55: 204.
phenotype: Wings held out at 90° from body axis; tibial-tarsal joint of metathoracic legs swollen. Homozygotes exhibit reduced viability and sterility; heterozygous viability also reduced. Partially suppresses expression of *ey*.
cytology: Associated with 3R breakpoint of $T(2;3)Mpl = T(2;3)35B2-3;86C1-2$ (Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velisariou, and Walker, 1981, DIS 56: 186-91).



mr: morula

mr: morula

location: 2-106.7.
discoverer: Bridges, 13c8.
references: Bridges and Morgan, 1919, Carnegie Inst. Wash. Publ. No. 278: 230 (fig.).
 Bridges, 1937, Cytologia (Tokyo), Fujii Jub., Vol. 2: 745-55.
phenotype: Eyes rough. Bristles irregularly reduced in size and number. Abdominal sclerites often smaller. Developmental study by Lees and Waddington [1942, Proc. Roy. Soc. (London), Ser. B 131: 87-100] shows that effect on bristles results from general slowing of bristle growth. Female entirely sterile, has under-

developed ovaries. At 19°, bristles nearly normal and eyes nearly wild type. RK2 at 25° and above.

cytology: Placed in salivary chromosome region between 59E2 and 60B10 based on its being to the right of $In(2R)bw^{VDel} = In(2R)41B2-C1;59E2-4$ and to the left of $Df(2R)Px = Df(2R)60B8-10;60D1-2$ (Bridges, 1937).

mr²

origin: Spontaneous.
discoverer: Bridges, 25k24.
phenotype: Less extreme than *mr*. Nearly wild type at 19°. Female entirely sterile. Oogenesis normal through stage 4; then compound nurse cell chromosomes fall apart and degenerate. Karyosome of oocyte also disappears. Oogenesis does not proceed beyond sixth stage (King, 1964, Royal Entomol. Soc. London Symposium 2, Insect Reproduction, pp. 13-25). RK2 at 25° or above.

MR: Male Recombination

location: 2-54 [(between *Tft* and *pr*; 2/10 *Tft-pr* crossovers between *Tft* and *MR* (Slatko and Green, 1980, Biol. Zentralbl. 99: 149-55)].

origin: Spontaneous.
synonym: *Mister*; *MRF*.

phenotype: A series of second chromosomes isolated from natural populations in diverse regions of the globe that, in crosses of *MR*-bearing males to laboratory-strain females, but not in the reciprocal crosses, produce dysgenic progeny. Such progeny transmit the *MR* chromosome at reduced levels compared with the homologous second chromosome; abnormalities in spermiogenesis including failure of individualization observed in dysgenic males account for 70% of the deficiency in recovery of *MR* (Matthews, 1981, Genetics 97: 95-111). Increased levels of gonial recombination observed in both sons (Hiraizumi, 1971, Proc. Nat. Acad. Sci. USA 68: 268-700) and daughters [demonstrated in *c3G* females by Sinclair and Green (Mol. Gen. Genet. 170: 219-24)]; mitotic crossing over in wing disks unaffected (Thompson and Woodruff, 1980, Genetica 49: 77-80). Both sons and daughters exhibit increased rates of spontaneous mutations, both lethal (Slatko and Hiraizumi, 1973, Genetics 75: 643-49) and at some but not all specific loci (e.g., Green, 1977, Proc. Nat. Acad. Sci. USA 74: 3490-93); the latter types of mutants usually unstable, undergoing further mutation either to more extreme alleles or back to wild type; increased mutation rates observed when combined with *mei9* and *mei41* (1981, Mutat. Res. 83: 191-200). All classes of chromosome rearrangements produced by dysgenic progeny, but with preferential points of breakage (Yannopoulos, Stamatis, Zacharopoulou, and Pelecanos, 1983, Mutat. Res. 108: 185-202); also shown in some cases to promote gonadal aplasia, especially at higher temperatures (Stamatis, Yannopoulos, and Pelecanos, 1981, Genet. Res. 38: 125-35); metaphase I of meiosis normal, but bridges and fragments observed in anaphase I and anaphase II in dysgenic males (Henderson, Woodruff and Thompson, 1973, Genetics 88: 93-107; Yannopoulos, 1978, Genet. Res. 239-47). Gross deletion of X-chromosome material induced by *MR* shown to involve an array of breakpoints, which, by *in situ* hybridization, are free of P-element sequences; postulated to arise through illegitimate mitotic exchange (Green, Yamamoto, and Miklos, 1987, Proc. Nat. Acad. Sci. USA 84: 4533-37). The majority of the activity in

several of the isolates maps to a site between *Tft* and *pr* on the left arm of chromosome two, but when that site removed residual activity remains in the genome. Mappability of the major effect to the same site in independent isolates suggests the presence of an element that is able to promote transposition but that itself is unable to transpose. Circumstantial evidence indicates that the element is *P* and that in addition to a functional *P* element, *MR* second chromosomes also carry defective *P*'s. Males inheriting two doses of *MR* from *mei-S332* fathers crossed to *C(2)EN/0* females not discernably different from males with one dose; two doses of *MR* inherited from the mother are inactive (Green and Slatko, 1979, *Mutat. Res.* 62: 529-39).

isolate	geographical origin	ref ^α
MR-23.5	Patras, Greece	14, 15
MR-31.1	Patras, Greece	14
MR-gb39	Sonoma County, California	3
MR-h12	Haifa, Israel	2, 3, 4, 9
MR-n1	Napa County, California	3, 4, 5
MR-OK1	Oklahoma City, Oklahoma	7, 13
MR-S90 ^β	Northern California	1
MR-T007	Harlingen, Texas	6, 8, 10, 11
MR-WO	Ohio?	12

^α 1 = Benzec and Slatko, 1984, *Genet. Res.* 43: 149-58; 2 = Green, 1977, *Proc. Nat. Acad. Sci. USA* 74: 3490-94; 3 = Green, 1984, *Biol. Zentralbl.* 103: 1-8; 4 = Green and Shepherd, 1979, *Genetics* 92: 823-32; 5 = Green and Slatko, 1979, *Mutat. Res.* 62: 529-31; 6 = Hiraizumi, 1971, *Proc. Nat. Acad. Sci. USA* 68: 268-70; 7 = Henderson, Woodruff, and Thompson, 1978, *Genetics* 88: 93-107; 8 = Hiraizumi, Slatko, Langley, and Nill, 1973, *Genetics* 73: 439-44; 9 = Sinclair and Green, 1979, *Molec. Gen. Genet.* 170: 219-24; 10 = Slatko, 1978, *Genetics* 90: 105-24; 257-76; 11 = Slatko and Hiraizumi, 1973, *Genetics* 75: 643-49; 12 = Waddle and Oster, 1974, *J. Genet.* 61: 177-83; 13 = Woodruff and Thompson, 1977, *Heredity* 38: 291-307; 14 = Yannopoulos and Pelecanos, 1977, *Genet. Res.* 29: 231-38; 15 = Yannopoulos, Stamatidis, Zacharapoulou, and Pelecanos, 1983, *Mutat. Res.* 108: 185-202.

^β Also carries *Sd*.

mrn: marionette (Fuller)

location: 3-42.6 (between *Ly* and *th*).

synonym: *nc16*, *l(3)E35*.

references: Fuller, 1986, *Gametogenesis and the Early Embryo*, (G.J. Gall, ed.). *Proc. Soc. Dev. Biol.* 44, pp 19-41.

Robertson and Fuller, unpublished.

Irick and Cherbas, unpublished.

phenotype: Recessive larval lethal. Individuals heterozygous for certain combinations of alleles reach late third instar but do not pupate; they have small discs and small brains. The original allele *nc16*, is associated with a dominant enhancer of the *B2tⁿ*-tubulin mutation, suggesting a role for the gene in microtubule function. The dominant enhancer and the lethal mutation associated with *nc16* were not separated in 82 recombinants between *Ly* and *th*. None of the other alleles are dominant enhancers of *B2t^{null}* (Robertson and Fuller, unpublished).

alleles:

allele	synonym	origin	ref ^α	phenotype ^β
mrn 1	<i>mrn nc16 nc16</i>	EMS	1, 2	
mrn 2	<i>mrn E35</i>	EMS	2	b
mrn 3	<i>mrn E67</i>	EMS	2	c
mrn 4	<i>mrn E72</i>	EMS	2	c

allele	synonym	origin	ref ^α	phenotype ^β
mrn 5	<i>mrn E94</i>	EMS	2	b
mrn 6	<i>mrn E96</i>	EMS	2	b
mrn 7	<i>mrn 13B</i>	EMS	3	c
mrn 8	<i>mrn 10C</i>	EMS	3	c

^α 1 = Fuller (1986); 2 = Irick and Cherbas, unpublished; 3 = Robertson and Fuller, unpublished;

^β a = Larval lethal, isolated as dominant enhancer of tubulin mutants. b = Larval lethal, isolated as lethal under *Df(3L)BK10*. c = Lethal phase not yet established, isolated as lethal under *Df(3L)BK10*.

cytology: Placed in 71D-F, based on its being uncovered by *Df(3L)Brdr6* and *Df(3L)BK10*, but not *Df(3L)878.3*.

Mrt: Moonrat (J.A. Kennison)

location: 3-79.7 (based on 211 recombinants between *e^s* and *ca*).

origin: Spontaneous.

discoverer: Kennison.

phenotype: Heterozygote shows partial transformation of anterior wing to posterior (triple row bristles replaced by double row bristles in patches). A network of extra veins appears in the anterior compartment, beginning at the distal edge in the least affected flies, and covering the entire anterior compartment in the more extreme cases. Wing blade expanded anteriorly at the distal edge. Wing blade expansion and extra veins resemble phenotypes seen in *en¹* homozygotes in the presence of Minute mutations. Bubbles often form in the wing blade. More rarely, a mirror-image outgrowth from the anterior edge is present. Mirror-image duplications sometimes appear in halteres. Legs sometimes appear deformed (similar to phenotype of *en^{lethal}* clones induced in the larva). Dominant phenotypes strongly temperature-sensitive. Penetrance greater than 99% at 18° (with strong expressivity) but only 30-40% at 29° (with very weak expressivity). Shows paternal effect. Penetrance greater when mutant allele inherited from father than when inherited from mother. *Mrt/+* indistinguishable from *Mrt/+*.

ms: see *msc*

ms(1)1 to 16: male sterile (1)

A series of ethyl-methanesulfonate-induced mutations at the base of the X chromosome (Geer, Bowman, and Tyl, 1979, *J. Exp. Zool* 209: 387-93).

locus	cytological location	included in	excluded from	comments
ms(1)1	19F1-20B	y ⁺ <i>Ymal126</i>	y ⁺ <i>Ymal107</i>	
ms(1)3	19E8-20A1	y ⁺ <i>Ymal108</i>	y ⁺ <i>Ymal126</i>	
ms(1)4	18F-19E1	y ⁺ <i>Ymal</i> ⁺ 2	y ⁺ <i>Ymal102</i>	
ms(1)7	19E6-F1	y ⁺ <i>Ymal113</i>	y ⁺ <i>Ymal108</i>	
ms(1)10	19E6-F1	y ⁺ <i>Ymal113</i>	y ⁺ <i>Ymal108</i>	possible allele of <i>ms(1)7</i>
ms(1)12	19F1-20B	y ⁺ <i>Ymal126</i>	y ⁺ <i>Ymal107</i>	possible allele of <i>ms(1)1</i>
ms(1)14	19E8-20A1	y ⁺ <i>Ymal108</i>	y ⁺ <i>Ymal126</i>	possible allele of <i>ms(1)3</i>
ms(1)16	20B-F	B ^S Y		

ms(1)1

phenotype: Few motile sperm; none transferred; morphology normal until coiling stage; however, some axoneme degeneration evident; some failure of individualization. Axoneme stability defective (Dybas, Tyl, and Geer, 1981, *J. Exp. Zool.* 216: 299-310).

cytology: Placed in 19F1-20B based on its being covered by $y^+Ymal126$ (19F1-20A2 to the base) but not by $y^+Ymal107$ (20A1-B to the base) (Geer, Bowman, and Tyl, 1979, J. Exp. Zool. 209: 387-94).

other information: Possibly allelic to *ms(1)20A*. *ms(1)12* maps to same region; some males have motile sperm, but no transmission to female; may be allelic.

ms(1)4

phenotype: No sperm motility. Few cysts reach coiling stage; paracrystalline material of major mitochondrial derivative diffuse and differentiation of the derivative irregular; crystallization initiated late and within the matrix of the mitochondrion rather than at a point of contact with the endoplasmic reticulum (Dybas, Tyl, and Geer, 1981, J. Exp. Zool. 216: 299-310).

cytology: Placed in 18F-19E1; covered by *Ymal*⁺² (18F to 20F) but not $y^+Ymal102$ (19C3-E1 to 20F) (Geer, Bowman, and Tyl, 1979, J. Exp. Zool. 209: 387-94).

other information: Possibly allelic to *ms(1)19E*.

ms(1)6S

location: 1-.

origin: Induced by ethyl methanesulfonate.

discoverer: Shadholt.

references: Habliston, Stanley, and Bowman, 1977, J. Ultrastruct. Res. 60: 221-34 (fig.).

- phenotype:** Anatomy of the reproductive system of *ms(1)6S* males normal; however few or no motile sperm. Spermiogenesis defective; some centrioles fail to form basal bodies, reducing the numbers of axonemes formed; basal body fails to make normal attachment to nucleus; mitochondria fuse irregularly but do elongate and elaborate paracrystalline material; large cytoplasmic units with multiple mitochondrial derivatives but few if any axonemes seen in cross sections; isolated cytoplasmic elements with or without single axonemes also observed, defective acrosome formation; chromatin condensation, nuclear elongation, and formation of microtubules around elongating nucleus abnormal.

ms(1)7

phenotype: Males viable but sterile; motile sperm produced and transmitted to females in normal numbers. Sperm fertilize eggs, but development fails. Spermiogenesis ultrastructurally normal (Dybas, Tyl, and Geer, 1981, J. Exp. Zool. 216: 299-310).

cytology: Placed in 19E6-F1 based on its being covered by $y^+Ymal113$, which extends from the base of the X to 19E6-8 but not by $y^+Ymal108$, which extends to 19E8-F1 (Geer, Bowman, and Tyl, 1979, J. Exp. Zool. 209: 387-94).

other information: Possibly allelic to *ms(1)19Fa*. *ms(1)10*, mapped to same region, also had motile sperm transferred to female; possibly allelic.

ms(1)10A

location: 1-33.44.

phenotype: Spermiogenesis blocked at or before coiling stage. Abnormal associations of major mitochondrial derivatives with endoplasmic reticulum give rise to multiple foci of paracrystalline-material formation (Dybas, Harden, Machnicki, and Geer, 1983, J. Zool. 226: 293-302).

alleles:

allele	origin	synonym	ref ^α	comments
<i>ms(1)10A 1</i>	EMS	<i>ms(1)BP6</i>	2	
<i>ms(1)10A 2</i>	EMS	<i>ms(1)v3</i>	1	
<i>ms(1)10A 3</i>	EMS	<i>ms(1)v6</i>	1	
<i>ms(1)10A 4</i>	EMS	<i>ms(1)v13</i>	1	
<i>ms(1)10A 5</i>	EMS	<i>ms(1)v16</i>	1	
<i>ms(1)10A 6</i>	EMS	<i>ms(1)v101</i>	1	on y^+Yv^+
<i>ms(1)10A 7</i>	EMS	<i>ms(1)v102</i>	1	on y^+Yv^+
<i>ms(1)10A 8</i>	EMS	<i>ms(1)v105</i>	1	on y^+Yv^+
<i>ms(1)10A 9</i>	EMS	<i>ms(1)v111</i>	1	on y^+Yv^+
<i>ms(1)10A 10</i>	EMS	<i>ms(1)F3</i>	3, 4	
<i>ms(1)10A 11</i>	EMS	<i>ms(1)F5</i>	3, 4	

^α 1 = Geer, Lischwe, and Murphy, 1983, J. Exp. Zool. 225: 107-18; 2 = Zhimulev, Belyaeva, Pokholkova, Kochneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baricheva, Aizenzon, Kramers, and Eeken, 1982, DIS 58: 210-14. 3 = Zhimulev, Pokholkova, Bgatov, Umbetova, Solovjeva, and Belyaeva, 1987, Biol. Zentralbl. 106: 699-720. 4 = 1987, DIS 66: 194-97.

cytology: Placed in 10A1-5 based on its inclusion in *Df(1)v-L1 = Df(1)9F13;10A4-5* but not in *Df(1)v-L2 = Df(1)9F13;10A1* (Zhimulev *et al.*).

ms(1)14

phenotype: Spermiogenesis appears normal, but late degeneration seen. Some sperm enter seminal vesicles and are transferred to females.

cytology: Placed in 19E8-20A2; covered by $y^+Ymal108$ (19E8-F1 to 20F) but not by $y^+Ymal107$ (20A1-B to the base) (Geer, Bowman, and Tyl, 1979, J. Exp. Zool. 209: 387-94).

other information: Possibly allelic to *l(1)19F6*; *ms(1)3* maps to same region, has few motile sperm which are transferred to females; may be allelic.

ms(1)16

phenotype: Few spermiogenic cysts reach coiling stage; multiple associations of mitochondrial derivative with endoplasmic reticulum producing multiple foci of paracrystalline material elaboration; improper elongation of major mitochondrial derivative or failure of paracrystalline-body formation. Individualization of sperm defective; cellular degeneration obvious; axonemes defective with gaps in the circular arrays of outer microtubular doublets. Cysts with half or twice the normal number of spermatids observed (Dybas, Tyl, and Geer, 1981, J. Exp. Zool. 216: 299-310).

cytology: Placed in 20B-F based on its inclusion in *B^SY* (Geer, Bowman, and Tyl, 1979, J. Exp. Zool. 209: 387-94).

other information: Possibly allelic to *ms(1)20B*.

ms(1)19 and 20

origin: Induced by ethyl methanesulfonate.

references: Lifschytz and Yakobovitz, 1978, Mol. Gen. Genet. 161: 275-84.

locus	cytological location	included in	excluded from	synonym
<i>ms(1)19E</i>	19E1-5	<i>Df(1)B57</i>	<i>Df(1)A118</i>	<i>ms(1)E</i>
<i>ms(1)19Fa</i>	19F	<i>Df(1)su(f)7085</i>	<i>Df(1)su(f)9122</i>	<i>ms(1)D</i>
<i>ms(1)19Fb</i>	19F	<i>Df(1)su(f)7085</i>	<i>Df(1)su(f)9122</i>	<i>ms(1)C</i>
<i>ms(1)19Fc</i> ^α	19F	<i>Df(1)su(f)7085</i>	<i>Df(1)su(f)9122</i>	
<i>ms(1)19Fd</i> ^α	19F	<i>Df(1)su(f)7085</i>	<i>Df(1)su(f)9122</i>	
<i>ms(1)20A</i>	20A	<i>Df(1)su(f)795</i>	<i>Df(1)su(f)7009</i>	<i>ms(1)B</i>
<i>ms(1)20B</i>	20A	<i>Df(1)su(f)724</i>	<i>Df(1)su(f)733</i>	<i>ms(1)A</i>

α all alleles included on *Y mal*⁺.

alleles:

allele	synonym
<i>ms(1)19E</i> ¹	<i>ms(1)E</i>
<i>ms(1)19E</i> ²	<i>ms(1)496</i>
<i>ms(1)19Fa</i> ¹	<i>ms(1)D</i>
<i>ms(1)19Fa</i> ²	<i>ms(1)424</i>
<i>ms(1)19Fa</i> ³	<i>ms(1)515</i>
<i>ms(1)19Fa</i> ⁴	<i>ms(1)RA4</i>
<i>ms(1)19Fa</i> ⁵	<i>ms(1)RA10</i>
<i>ms(1)19Fb</i> ¹	<i>ms(1)C</i>
<i>ms(1)19Fb</i> ²	<i>ms(1)RB47</i>
<i>ms(1)19Fc</i> ¹	<i>ms(1)DD19</i>
<i>ms(1)19Fc</i> ²	<i>ms(1)DD23</i>
<i>ms(1)19Fc</i> ³	<i>ms(1)S5</i>
<i>ms(1)19Fc</i> ⁴	<i>ms(1)S27</i>
<i>ms(1)19Fd</i> ¹	<i>ms(1)DD5</i>
<i>ms(1)19Fd</i> ²	<i>ms(1)DD16</i>
<i>ms(1)19Fd</i> ³	<i>ms(1)DD17</i>
<i>ms(1)19Fd</i> ⁴	<i>ms(1)S35</i>

ms(1)202

origin: Induced by ethyl methanesulfonate.

references: Lifschytz and Meyer, 1977, *Chromosoma* 64: 371-92 (fig.).

phenotype: Meiosis I proceeds to metaphase and rarely anaphase; undivided co-oriented bivalents move irregularly toward the poles; more than 32 nuclei per cyst, frequent micronuclei; many cysts do not undergo meiosis. First division spindles tetrapolar, resemble two parallel bipolar spindles.

ms(1)244

origin: Induced by ethyl methanesulfonate.

references: Lifschytz and Meyer, 1977, *Chromosoma* 64: 371-92 (fig.).

phenotype: Shape of primary-spermatocyte nuclei highly irregular, with evaginated pockets, which sometimes contain chromosomes. Multipolar spindles observed; entire bivalents rather than dyads pass to pole at first meiotic anaphase; second division generally arrested, but some cysts with more than 32 nuclei observed. Nuclear size highly variable; micronuclei present. Mutant males exhibit shaking upon etherization.

ms(1)401

location: 1-58.

origin: Induced by ethyl methanesulfonate.

references: Lifschytz and Hareven, 1977, *Dev. Biol.* 276: 276-94 (fig.).

phenotype: Primary spermatocytes appear to accumulate; nuclei appear creased; nucleoli fragmented. Some elongating spermatids produced, but development arrested early. In *XO* males primary spermatocytes do not attain full size, and the intermediate-sized nucleolated spermatocytes accumulate; nuclear creases fail to form.

cytology: Claimed to be fertile in combination with y^2Y67g (probably 67g19.1, which carries 20A3-20F).

ms(1)413

location: 1-25.

origin: Induced by ethyl methanesulfonate.

references: Lifschytz and Hareven, 1977, *Dev. Biol.* 276: 276-94 (fig.).

Brick, Lifschytz, and Friedlander, 1979, *J. Ultrastruct. Res.* 66: 151-63 (fig.).

phenotype: Males carrying the mutation are sterile; exhibit

shaking behavior with intermittent proboscis extension. Meiosis appears to be arrested, but the cellular changes associated with spermiogenesis continue, including mitochondrial aggregation, nuclear condensation and elongation, cellular elongation, and coiling. Spermatocytes do not divide, but develop directly into spermatids with one nucleus and four axonemes; mitochondria cluster prematurely in the primary spermatocyte but do not form a nebenkern in the spermatid. No chromosome condensation or spindle formation in the spermatocytes. Irregular distribution of nuclear-envelope annuli, condensed chromatin, and perinuclear microtubules in the spermatids, basal bodies do not associate with nucleus.

alleles: Three mutations with similar location 4-5 units to the right of *ct* and shared somatic and germinal phenotypes presumed to be allelic: *ms(1)413*¹, *ms(1)413*² [synonym = *ms(1)682*], and *ms(1)413*³ [synonym = *ms(1)RD11*].

ms(1)516

location: 1-38.

origin: Induced by ethyl methanesulfonate.

references: Lifschytz and Hareven, 1977, *Dev. Biol.* 276: 276-94 (fig.).

Lifschytz and Meyer, 1977, *Chromosoma* 64: 371-92 (fig.).

phenotype: First meiotic divisions normal in both cell morphology and bivalent structure; second meiotic divisions appear monastral but bipolar; both centrioles pass to the same pole at the second meiotic division producing one daughter nucleus with two basal bodies associated and one with none. Dyads move to the poles as highly condensed bodies with sister centromeres remaining associated; spermatids display numerous micronuclei and nebenkerne of nonuniform size. Occasional apparently normal cysts encountered. Spermiogenesis proceeds albeit abnormally.

cytology: Claimed to be fertile in combination with *Dp(1;3)sn*^{12a1}, which does not include the region to which the mutant maps.

ms(1)RA40

origin: Induced by ethyl methanesulfonate.

references: Lifschytz and Meyer, 1977, *Chromosoma* 64: 371-92 (fig.).

phenotype: 90% of cysts in division in early anaphase I; apparently pause there as anaphase proceeds normally producing 32 diploid spermatids with nuclei and nebenkerne of uniform size and developing to a normal onion stage. In rare anaphase II divisions chromosomes appear to pass to the poles at random. Diploid spermatids have two centriolar bodies, usually both attached to the nuclear membrane; detached centriolar bodies also seen; centriolar bodies, whether attached or detached, undergo same morphological transformations as in normal males.

ms(1)RD7

origin: Induced by ethyl methanesulfonate.

references: Lifschytz and Hareven, 1977, *Dev. Biol.* 276-94 (fig.).

Lifschytz and Meyer, 1977, *Chromosoma* 64: 371-92 (fig.).

phenotype: Meiotic figures rare; those that are observed display abnormal chromosome behavior in both meiotic divisions. Bivalent rather than dyads pass to first ana-

phase poles. First meiotic divisions mostly abnormal with monastral, deformed diastral and multipolar spindles; second-division chromosome constitutions but not second-division spindles observed. Spermatid cysts contain fewer than 64 but more than 32 cells; two types of spermatid nuclei observed: some with 0-4 detached centrioles in cytoplasm of early spermatids in which centriolar bodies plus axonemes held together in vicinity of nucleus; others exhibit one centriole of a pair attached to nucleus and the other detached but close by.

ms(1)RD15

origin: Induced by ethyl methanesulfonate.
references: Lifschytz and Hareven, 1977, Dev. Biol. 276-94 (fig.)
phenotype: Meiosis appears to be held up at prometaphase of the first division; few metaphases or first anaphases encountered; twice as many cysts in division as normal with 90% in first prometaphase; tri- and sometimes tetrapolar spindles observed in M1. Second divisions not observed; cysts contain a maximum of 32 cells. Spermatid differentiation proceeds to an extent similar to that of *XO* males.

ms(1)RD33

location:
origin: Induced by ethyl methanesulfonate.
references: Lifschytz and Hareven, 1977, Dev. Biol. 276-94 (fig.)
phenotype: Spermatid development arrested at a stage slightly earlier than that observed in *XO* males; in *ms(1)RD33/0* males, however, development arrested before completion of primary-spermatocyte growth.

ms(Y)

Male-sterile mutations on the *Y* chromosome that have been mapped are designated according to the *Y*-fertility gene affected: *kl-1*, *kl-2*, *kl-3*, or *kl-5* on *YL* and *ks-1* or *ks-2* on *YS*.

ms(Y)hd: male sterile (Y) hybrid dysgenesis

Thirteen sterile *Y* chromosomes recovered from hybrid dysgenic crosses; Southern blots probed with P sequences reveal male specific bands not seen in the original P strain; complementation tests not carried out to determine locus affected (Schafer and Nahmias, 1985, Mol. Gen. Genet. 200: 182-84).

ms(2)1: male sterile (2) 1

location: 2-65.5.
origin: Ultraviolet induced.
discoverer: Meyer, 48c.
references: Meyer, Edmondson, Byers, and Erickson, 1950, DIS 24: 60.
phenotype: Male sterile; female fertile. Sperm present but not motile. RK3.

ms(2)2

location: 2-44.0 (Meyer).
origin: Spontaneous.
discoverer: Muller, 1951.
synonym: *ms*.
references: Meyer, 1959, DIS 33: 97.
phenotype: Homozygous male completely sterile; female fairly fertile. RK3.

ms(2)3R

location: 2-51.
origin: Induced by ethyl methanesulfonate.
references: Romrell, Stanley, and Bowman, 1972, J. Ultrastruct. Res. 38: 563-77.
 Laughran, Stanley, and Bowman, 1976, J. Ultrastruct. Res. 56: 21-30 (fig.).
phenotype: Meiosis in homozygous mutant males characterized by apparent failure of the normally occurring incomplete cytokinesis so that all four meiotic products are seen in one spherical cell. Incomplete cytokinesis and ring-canal formation occur normally during meiosis; however, furrow membranes subsequently fuse and disintegrate; double-membrane fragments and ring canals found freely floating in spermatid cytoplasm. All the mitochondria coalesce into a single nebenkern, which subsequently divides into two and may subdivide into as many as eight mitochondrial derivatives. Elongation appears to take place in separate cytoplasmic units of four spermatids each in which mitochondrial derivatives vary in size and may associate with the endoplasmic reticulum or more than one axoneme, forming like numbers of foci of crystallization. Later stages show massive degeneration.

ms(2)10R

location: 2-84.
origin: Induced by ethyl methanesulfonate.
references: Romrell, Stanley, and Bowman, 1972, J. Ultrastruct. Res. 38: 578-90.
phenotype: Spermatid elongation limited; the endoplasmic sheath surrounding the axoneme opens out producing linear rather than circular arrays of microtubular doublets in cross sections of elongating spermatids. Mitochondrial derivatives may be large and irregular in shape with multiple contact points with endoplasmic reticulum and correspondingly multiple foci of crystallization.

ms(2)73d

location: 2- (between *b* and *Sd*).
origin: Spontaneous in *In(2L)Cy*; separable.
references: Hartl, 1980, Genetics 96: 685-96.
phenotype: Homozygous males have immature, unelongated spermatid bundles.

ms(2)E

A series of complementing male-sterile mutations induced by ultraviolet light (Edmondson, 1951, DIS 26: 61).

locus	genetic location	synonym
<i>ms(2)E3</i>	2-28	<i>ms2.3</i>
<i>ms(2)E4</i>	2-47.9	<i>ms2.4</i>
<i>ms(2)E5</i>	2-54.8	<i>ms2.5</i>
<i>ms(2)E6</i>	2-54.8	<i>ms2.6</i>
<i>ms(2)E7</i>	2-54.8	<i>ms2.7</i>
<i>ms(2)E8</i>	2-55.6	<i>ms2.8</i>
<i>ms(2)E9</i>	2-57	<i>ms2.9</i>
<i>ms(2)E10</i>	2-66.5	<i>ms2.10</i>
<i>ms(2)E11</i>	2-68	<i>ms2.11</i>
<i>ms(2)E12</i>	2-68.2	<i>ms2.12</i>

Ms(2)M: Male sterile (2) of Meyer

location: 2- (just to the right of *cn*).
origin: X ray induced.
references: Meyer, 1966, DIS 41: 167.

phenotype: Heterozygous males sterile; heterozygous females fertile.

ms(3)10R

location: 3-.

origin: Induced by ethyl methanesulfonate.

references: Wilkinson, Stanley, and Bowman, 1974, *J. Ultrastruct. Res.* 48: 242-58.

phenotype: Spermiogenesis of homozygous mutant males defective; nuclear elongation variable owing to variably reduced numbers of perinuclear microtubules; absence of perinuclear microtubules associated with irregular condensation of chromatin beneath the nuclear envelope. Evidence for folding in axonemes leading to more than 64 profiles in transverse sections of spermatid bundles. Cross sections of elongating spermatid bundles revealed deranged axonemes with linear rather than circular arrays of the nine pairs of microtubules, with the doublets either still associated with the endoplasmic reticulum or free; mitochondrial derivatives multiply associated with endoplasmic reticulum with up to four foci of deposition of paracrystalline material instead of the normal one.

***ms(3)HO5A: male sterile (3)* of Hardy and Orevi**

location: 3-45.9.

origin: Induced by ethyl methanesulfonate simultaneously with *fs(3)HO5A*, *fs(3)HO5B*, and *ms(3)HO5B*.

synonym: *ms(3)ml*^{5A}.

references: Nishida, 1980, *Jpn. J. Genet.* 55: 427-39 (fig.).

phenotype: Testes rudimentary; germ-cell development arrested in immature primary-spermatocyte stage; few cysts escape to produce spermatids. A more advanced stage of development attained, including production of motile sperm, in the presence of a recessive suppressor on chromosome 2.

other information: Possibly *fs(3)HO5A* and *ms(3)HO5A* are the same mutant and should be designated *ms(3)HO5A*.

ms(3)HO5B

location: 3-.

origin: Induced by ethyl methanesulfonate simultaneously with *fs(3)HO5A*, *fs(3)HO5B*, and *ms(3)HO5A*.

synonym: *ms(3)ms2*^{5A}.

references: Nishida, 1980, *Jpn. J. Genet.* 55: 427-39 (fig.).

phenotype: Spermatogenesis proceeds to coiling stage, but no motile sperm produced.

ms(3)K81

location: 3-91.3.

origin: Spontaneous.

synonym: *ph: paternal haploid*.

references: Fuyama, 1984, *Jpn. J. Genet.* 59: 91-96. 1986, *Genetics* 112: 237-48.

1986, *Genetics* 114: 495-509.

phenotype: Homozygous males produce motile sperm capable of fertilizing eggs, but defective in pronuclear function. Development of fertilized eggs initiated; arrested after several nuclear divisions in three-fourths of the embryos, the remainder developing beyond blastoderm and proving to be haploid upon cytological examination. Nuclear division cycle protracted compared to wild type, and the syncytial blastoderm nuclei undergo an extra division to produce twice the normal density prior to cellularization (Edgar, Kiehle, and Schuberg, 1986,

Cell 44: 365-72). A small fraction of the eggs diploidize and yield viable daughters, mostly by fusion of products of the first meiotic division (85%); fusion of products of second meiotic division rare or nonexistent. Incidence of gynogenetic diploids becomes appreciable in crosses to a stock designated G9 by Fuyama (1986, *Genetics* 114: 495-509).

cytology: Placed in 97D2-6 on the basis of the sterility of *ms(3)K81/Df(3R)ro-XB3* males [=Df(3R)97D2-3;97D5-6].

ms(3)KK^D: see *βTub85D*

ms(3)m1

location: 3-45.9.

origin: Induced by ethyl methanesulfonate.

references: Nishida, 1980, *Jpn. J. Genet.* 55: 427-39.

phenotype: Homozygous males have rudimentary testes exhibiting degenerating cysts of spermatocytes and cysts arrested early in development. Homozygous females also have rudimentary gonads, but Nishida attributes this to an independent but closely linked mutation; only the male sterility is suppressed by *su(msm1)*.

ms(3)m2

location: 3- (near centromere).

origin: Induced by ethyl methanesulfonate.

references: Nishida, 1980, *Jpn. J. Genet.* 55: 427-39.

phenotype: Homozygous males sterile; small coiled testes with elongated spermatid cysts.

ms(3)nc2: see *hay*

ms(3)nc3

location: 3-47.0.

origin: Induced by ethyl methanesulfonate.

synonym: *nc3*.

references: Fuller, 1986, *Proc. Soc. Dev. Biol.* 74: 19-41.

phenotype: Fails to complement certain *βTub* mutant alleles for male fertility although mapping to another site. Male fertility of homozygotes not determined. *ms(3)nc3/+* males fertile.

cytology: Located at 77E-F.

ms(3)nc4: see *wrl*

ms(3)nc32

location: 3-82.9.

origin: Induced by ethyl methanesulfonate.

synonym: *nc32*.

references: Fuller, 1986, *Proc. Soc. Dev. Biol.* 74: 19-41.

phenotype: Fails to complement certain *βTub85D* mutant alleles for male fertility although mapping to another site; *βTub85D* synthesis, meiosis, and spermatid differentiation are normal in these sterile trans-heterozygotes. *ms(3)nc32/ms(3)nc32* males fail to enter meiosis and show no spermatid differentiation. Testes from these males contain gonial cells, primary spermatocyte cysts, and cysts with abnormal cells. Adult male homozygotes show no *βTub85D* synthesis. *ms(3)nc32/+* males fertile.

cytology: Located in 95F-96A.

ms(3)nc33: see *αTub84D*

ms(3)neol: see *fwd*

ms(3)sa: male sterile (3) spermatocyte arrest**location:** 3-44 (Kemphues).**origin:** Spontaneous.**synonym:** *ms(3)VO45*.**phenotype:** Primary spermatocytes arrested premeiotically but apparently after attaining full size; testes become filled with mature primary spermatocytes.**cytology:** Located at 78A3;79E1-2 (Fuller).*ms2*: see *ms(2)E*****msc: melanoscutellum*****location:** 1-52.6.**origin:** Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).**discoverer:** Fahmy, 1954.**synonym:** *ms* (preoccupied).**references:** 1958, DIS 32: 72.**phenotype:** Extra pigmentation confined to scutellum. One or more thoracic bristles duplicated. Eyes slightly more oval than normal. Wings slightly abnormal in shape and position. Characters not always penetrant. Viability and fertility good in both sexes. RK3.**other information:** One allele each induced by CB. 1506 and CB. 3025; two induced by CB. 3007.*Msc*: see *Scr*^{*Msc*} under *ANTC****msd(gl): modifier of sexual dimorphism of glass*****location:** 1-0.96.**origin:** Spontaneous.**references:** Birchler, 1984, Genet. Res. 44: 125-32.**phenotype:** Two alleles; homozygous *gl* males carrying the allele that we designate *msd(gl)*^{*d*} (dimorphic) have brick-red eyes, whereas *msd(gl)*^{*d*}; *gl* females' eyes are lemon yellow. In the presence of *msd(gl)*^{*m*} (monomorphic), however, both *gl* males and females have the lighter eye color. Thus allelic differences are discernable only in males; females, either normal or homozygous for *tra* do not respond to *msd(gl)* genotype. Males carrying duplications for the *msd(gl)* locus show that *msd(gl)*^{*d*} is dominant to *msd(gl)*^{*m*} and that the eye colors of males with one or two doses of the dimorphic allele are indistinguishable. In *gl*⁺ flies, the *msd(gl)* constitution is without phenotypic consequence. Not an allele of *zeste*.**cytology:** Located in 3A8-C2 based on the presence of *msd(gl)*^{*d*} in *Dp(1;2)w*⁺70h = *Dp(1;2)3A7-8;3C2-3*.***msf: misformed*****location:** 2-55.2 (originally located at 55.6 but arbitrarily placed at 55.2 to be consistent with cytological indication that it is to the left of *pk*).**discoverer:** Bridges, 30b8.**references:** Curry, 1939, DIS 12: 46.**phenotype:** Eyes misshapen. Wings short and crumpled; legs shortened. Characteristics variable and overlap wild type. RK3.**cytology:** Placed between 41A and 42A3 on the basis of its inclusion in *Df(2R)bw*^{*VDe2LCy*}^{*R*} = *Df(2R)41A-B;42A2-3* (Schultz). Certainly included in *Df(2R)M41A-vg11* = *Df(2R)40F-41A1;42A19-B1* (Morgan, Schultz, Bridges, and Curry, 1939, Carnegie Inst. Wash. Year Book 38: 275).****msg: missing*****location:** 2- (not located).**origin:** Spontaneous.**discoverer:** Mossige, 50b4.**references:** 1951, DIS 25: 69.**phenotype:** Bristles greatly reduced or missing. In extreme cases, almost like *sv*. Female sterile; male sparingly fertile. RK2.***msl-1: male-specific lethal*****location:** 2-53.3.**references:** Belote and Lucchesi, 1980, Genetics 96: 165-86.**phenotype:** Homozygous male embryos hatch but die as much as twelve days later in larval or prepupal stages; females and heterozygous males survive; phenotype slightly more severe in sons of homozygous than of heterozygous mothers. Viability of two-X individuals that develop as phenotypic males (*tra2*) or intersexes (*dsx*) is unaffected by *msl-1*, indicating that the one-X condition is required for *msl-1* lethality. No interaction with *mle* or *msl-2* (Belote, 1983, Genetics 105: 881-96). Females heterozygous for *Sxl*^{*fl*} and homozygous for *msl-1* show signs of intersexual development [Skripsky and Lucchesi, 1982, Dev. Biol. 94: 153-64 (fig.)]. Pole cells from *msl-1* male embryos capable of undergoing normal spermatogenesis when transplanted into wild-type hosts (Bachiller and Sánchez, 1986, Dev. Biol. 118: 379-84). Concluded to be defective in dosage compensation in males based on decreased levels of X-linked-enzyme activities (G6PD, 6GPD, FUM) but not autosomally encoded enzymes (ADH, AO, GPDH, IDH) in homozygous *msl-1*^{*1*} and *msl-1*^{*2*} male larvae when compared with non-*msl-1* controls (Belote and Lucchesi, 1980, Nature 285: 573-75).**alleles:**

allele	origin	discoverer	synonym	ref ^α	comments
<i>msl-1</i> ^{<i>1</i>}	EMS	Belote		1, 2	amorph
<i>msl-1</i> ^{<i>2</i>}	EMS	Belote	<i>msl-1</i> ^{<i>b</i>}	1, 2	less severe allele

^α 1 = Belote and Lucchesi, 1980, Genetics 96: 165-86. 2 = Uenoyama, Uchida, Fukunaga, and Oishi, 1982, Genetics 102: 223-31.**cytology:** Placed in 36F7-37B8 based on its inclusion in *Df(2L)TW3* = *Df(2L)36F7-37A1;37B2-8*.***msl-2*****location:** 2-9.0.**references:** Belote and Lucchesi, 1980, Genetics 96: 165-86.**phenotype:** Homozygous male embryos hatch but die as much as fourteen days later in larval or prepupal stages; females and heterozygous males survive; no maternal effect. Viability of two-X individuals that develop as phenotypic males (*tra2*) or intersexes (*dsx*) is unaffected by *msl-1*, indicating that the one-X condition is required for *msl-1* lethality. No interaction with *mle* or *msl-1* (Belote, 1983, Genetics 105: 881-96). Few homozygous *msl-2*^{*2*} gynandromorphs survive; *XO* patches small, with small bristles, and mostly confined to abdomen (Uenoyama, Uchida, Fukunaga, and Oishi, 1982, Genetics 102: 223-31). Pole cells from *msl-2* male embryos capable of undergoing normal spermatogenesis when transplanted into wild-type hosts (Bachiller and Sánchez, 1986, Dev. Biol. 118: 379-84). Females heterozygous

for *Sxl^{fl}* and homozygous (and to a lesser extent heterozygous) for *mst-2* show signs of intersexual development; effects greater when mother homozygous for *mst-2* [Uenoyama, Fukunaga, and Oishi, 1982, Genetics 102: 233-43 (fig.); Skripsky and Lucchesi, 1982, Dev. Biol. 94: 153-64 (fig.)]. Concluded to be defective in dosage compensation in males based on decreased levels of X-linked-enzyme activities (G6PD, 6GPD, FUM) but not autosomally encoded enzymes (ADH, AO, GPDH, IDH) in homozygous *mst-2¹* male larvae when compared with non-*mst* controls (Belote and Lucchesi, 1980, Nature 285: 573-75).

alleles:

allele	origin	discoverer	synonym	ref ^α
<i>mst-2¹</i>	EMS	Belote		1
<i>mst-2²</i>	EMS	Oishi, 1976	<i>mst-2²⁷</i>	2

^α 1 = Belote and Lucchesi, 1980, Genetics 96: 165-86; 2 = Uenoyama, Uchida, Fukunaga, and Oishi, 1982, Genetics 102: 223-31.

mst-3: see *mle3²*

mst-3^b: see *mle3³*

Msp: Muscle specific proteins

location: 3- {50}.

references: Bernstein, Glenn, Emerson, and Donady, 1981, Genetics 97: s10.

cytology: Placed at two sites in 87B by *in situ* hybridization.

molecular biology: Isolated as clones homologous to mRNA's coding for several developmentally regulated, sequence related polypeptides of 22,500 daltons molecular weight. Genes activated at fusion stage of myogenesis. Restriction maps and partial sequencing as yet uninformative.

Mst: Male-specific transcript

Refers to cloned sequences isolated from genomic libraries differentially screened with male and female cDNA. Cytological localizations all determined by *in situ* hybridization.

locus	genetic location	cytological location	ref ^α	synonym	comments ^β
<i>Mst25F</i>	2-{17}	25F	2	<i>mst323</i>	germ line
<i>Mst26Aa</i>	2-{20}	26A	1, 4	<i>mst355a</i>	accessory gland
<i>Mst26Ab</i>	2-{20}	26A	1, 4	<i>mst355b</i>	accessory gland
<i>Mst47A</i>	2-{59}	47A	2	<i>mst325</i>	germ line
<i>Mst51F</i>	2-{73}	51F	5	<i>mst(2)ag-35</i>	accessory gland
<i>Mst57D</i>	2-{99}	57D	5, 6, 3	<i>mst(2)ag-1</i>	accessory gland
<i>Mst66D</i>	3-{26}	66D	2	<i>mst349</i>	germ line
<i>Mst75C</i>	3-{45}	75C	5	<i>mst(3)ag-2</i>	accessory gland
<i>Mst87F</i>	3-{54}	87F	3, 5	<i>mst(3)gl-9</i>	germ line
<i>Mst95E</i>	3-{81}	95E	1, 2	<i>mst316</i>	accessory gland
<i>Mst95EF</i>	3-{81}	95E-F	2	<i>mst345</i>	germ line
<i>Mst95F</i>	3-{81}	95F	5	<i>mst(3)ag-3</i>	somatic
<i>Mst98CE</i>	3-{95}	98C-E	2	<i>mst336</i>	germ line

^α 1 = Chapman and Wolfner, 1988, Dev. Biol. 126: 195-202; 2 = DiBenedetto, Lakich, Kruger, Belote, Baker, and Wolfner, 1987, Dev. Biol. 119: 242-51; 3 = Kuhn, Schäfer, and Schäfer, 1988, EMBO J. 7: 447-54; 4 = Monsma and Wolfner, 1989, Genes Dev. 2: 1063-73; 5 = Schäfer, 1986, Mol. Gen. Genet. 202: 219-25; 6 = Schäfer, 1986, EMBO J. 5: 3579-82.

^β Tissue-specific expression.

Mst26Aa

phenotype: Expressed in males, but not in females; also expressed in the germ-cell-free sons of *tud* females as well as in XX individuals that are homozygous for *dsx*, *ix*, *tra2*, or *tra*. 1986, Presence of *tra2⁺* activity in XX flies during a part of the third larval instar is sufficient to repress expression of *Mst26Aa* and prevent accessory-gland development; absence of *tra2⁺* activity during that period leads to accessory-gland formation and the production of *Mst26Aa* transcript.

molecular biology: Transcribed from the same strand as *Mst26Ab*, the transcription terminating 20 base pairs 5' to the initiation of *Mst26Ab* transcription. 0.9 kilobase transcript has a 56-base-pair intron between the first and second nucleotides of codon 12. Open reading frame encodes a 265-amino-acid polypeptide. Conceptual amino-acid sequence reveals a region in which 11 of 17 amino acids are identical to the egg-laying hormone of the sea hare *Aplysia californica*.

Mst26Ab

phenotype: Expressed in males, but not in females; also expressed in the germ-cell-free sons of *tud* females as well as in XX individuals that are homozygous for *dsx*, *ix*, *tra2*, or *tra*. Presence of *tra2⁺* activity in XX flies during a part of the third larval instar is sufficient to repress expression of *Mst26Ab* and prevent accessory-gland formation and the production of *Mst26Ab* transcript.

molecular biology: Transcription begins 20 base pairs downstream from the termination of transcription and on the same strand as *Mst26Aa*. 0.5 kilobase transcript sequence reveals intron of 61 base pairs between the first and second nucleotides of codon 11. Open reading frame encodes a 90-amino-acid polypeptide.

Mst51F

phenotype: Expressed in normal and X0 males, as well as in the XX pseudomales or intersexes homozygous for *dsx*, *ix*, *tra2*, or *tra* and in the germ-cell-free sons of *tud* females.

Mst87F

phenotype: Expressed in normal and X0 males, but not in the XX pseudomales homozygous for *dsx*, *ix*, *tra2*, or *tra* nor in the germ-cell-free sons of *tud* females.

molecular biology: Sequence analysis shows that the gene comprises two exons of 79 and 338 base pairs separated by a 91-base-pair intron. A 165 base pair open reading frame is in the second and larger exon. Conceptual amino acid sequence reveals repeated motifs of Cys Cys Gly Pro or Cys Gly Pro such that Cys, Gly, and Pro comprise 38, 29, and 20% of the amino acids present. 102 base pairs of upstream sequences suffice to specify the gene's specific expression characteristics, *i.e.*, premeiotic transcription and postmeiotic translation separated by three days of development (Kuhn, Schäfer, and Schäfer, 1988, EMBO J. 7: 447-54).

Mst95E

phenotype: Expressed in males, but not in females; also expressed in the germ-cell-free sons of *tud* females as well as in XX individuals that are homozygous for *dsx*, *ix*, *tra2*, or *tra*. Presence of *tra2⁺* activity in XX flies during a part of the third larval instar is sufficient to repress expression of *Mst95E* and prevent accessory-gland development; absence of *tra2⁺* activity during that

period leads to accessory-gland formation and the production of *Mst95E* transcript.

mt^A: see *tu-bw*

mt: see *mul*

***mtb: matt brown**

location: 1-3.6.

origin: Induced by ethyl methanesulfonate (CB. 1528).

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 88.

phenotype: Eye color flat and browner than normal with greatly reduced reflection spots. Wing position varies from slightly to completely outspread, sometimes upheld. Male sterile; viability about 30% wild type. RK2.

mTml: see *Tm2*

mTmlI: see *Tml*

Mtn: Metallothionein (G. Maroni)

location: 3-48.8.

references: Lastowski-Perry, Otto, and Maroni, 1985, J. Biol. Chem. 260: 1527-1530.

Maroni, Otto and Lastowski-Perry, 1986, Genetics 112: 493-504.

phenotype: Codes for metallothionein, a metal binding protein. Inducible by ingestion of Cu or Cd (but not Zn) ions. In larvae, it is expressed mainly in the midgut. No point mutations are available, but *Dp(3;3)Mtn⁺H²²* shows increased tolerance to Cd.

cytology: 85E10-15 based on *in situ* hybridization and its inclusion in *Df(3R)by10* but not in *Df(3R)γB104* (Maroni *et al.*).

molecular biology: Restriction maps and sequences of genomic and cDNA clones are available. The gene size is 631 bp; this includes 120 bp of coding sequence and a 365 bp intron. It shows similarity to the mammalian metallothioneins both at the amino-acid and nucleotide sequence level. Metal-inducible promoter useful in molecular constructs (Bunch, Grinblat, and Goldstein).

***mu: mussed**

location: 3-50.

origin: Spontaneous.

discoverer: Mohr, 37121.

references: Mossige, 1939, DIS 12: 47.

phenotype: Wings thin textured. Dorsal surface of thorax arched. RK1.

mu1: mutator

location: 3-57.

origin: Spontaneous.

synonym: *mt*; *mu* (preoccupied).

references: Green, 1970, Mutat. Res. 10: 353-63.

Green and Lefevre, 1973, Mutat. Res. 16: 59-64.

Gold and Green, 1974: Mol. Gen. Genet. 135: 245-55.

phenotype: Homozygous and hemizygous females exhibit increased rates of sex-linked visible and lethal mutations (*e.g.*, $y^2 \rightarrow y^+$ = 1/2500; more rarely mutations to intermediate or more extreme alleles; also f^{3N} reversions and $w^a \rightarrow w$); heterozygous females display slightly elevated rates of mutation; about half of mutants occur as clusters indicating premeiotic origin. Lethal mutations frequently associated with deficiencies of one to several bands. Mutation rates not elevated in males. y - *sn* recombina-

tion reduced 30% in *mul* homozygous and 25% in *mul/+* heterozygous females; also females but not males display elevated frequencies of nondisjunction. Effects of 100r on the induction of sex-linked lethals significant in *mul* but not + females (Gold and Green, 1975, Genetics 80: s35). The incidence of y and f^{36} clones is five times control values in *mul/mul* and ten times control in *mul/Df(3R)sbd105* females (Martensen and Green, 1976, Genetics 83: s47).

cytology: Placed in 88F9-89B10 based on its inclusion in *Df(3R)sbd105 = Df(3R)88F9-88A1;89B9-10*.

mu2

location: 3-[1.5].

origin: Spontaneous.

discoverer: Green, 1977.

references: Mason, Strobel, and Green, 1984, Proc. Nat. Acad. Sci. USA 81: 6090-94.

phenotype: Irradiation of females homozygous for *mu2* with low doses of X rays produces substantial numbers of terminal deficiencies for all chromosome arms, [see for example *Df(1)yT*]; irradiation of heterozygous females produces frequencies of deficiencies intermediate between those obtained from homozygous *mu2* and wild-type females. Extents of deficiencies limited only by the ability to recover them in viable offspring. Incidence of terminal losses linear with dose; such deficiencies visible cytologically and can be shown by *in situ* hybridization to lack sequences characteristically present at the termini of chromosome arms. Irradiation of *mu2* females does not increase the incidence of either interstitial deficiencies or sex-linked lethal mutations. Irradiation of *mu2* males does not increase the frequency of deficiencies.

cytology: Placed in polytene section 62, (Biessmann).

molecular biology: Newly formed chromosome ends unstable; lose approximately 75 base pairs per generation (Biessmann and Mason, 1988, EMBO J. 7: 1081-86).

Mu(f^{3N}): Mutator forked-3N

location: 1-56.7-59.4 (between *f* and *Bx*).

references: Woodruff, 1975, Genet. Res. 25: 163-77.

phenotype: A dominant cis-acting enhancer of the reversion rate of f^{3N} ; thought responsible for reported instability of f^{3N} . Ineffective in reverting other alleles and in increasing the rate of sex-linked lethal mutations.

***mu-F: mutability factor from Florida**

location: 2- (not located).

origin: Spontaneous in Florida wild stock.

discoverer: Demerec, 1936.

references: 1937, Genetics 22: 469-78.

phenotype: Homozygote shows increase in lethal and visible mutation rate. Factor acts during development of germ cells in both male and female. Description resembles that of *MR*. RK3.

mud: mushroom body defect (J.C. Hall)

location: 1-50 [(Heisenberg, 1980), but cytology implies 1-[47]].

references: Heisenberg, 1980, Development and Neurobiology of Drosophila, (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York, pp. 373-90.

Technau and Heisenberg, 1982, Nature 295: 405-07.

phenotype: Neuropile of mushroom bodies (usually in dorsal brain) is missing (Heisenberg, 1980); penetrance for

this anatomical phenotype (on which criterion the mutant was isolated) *ca.* 90%. Calyces that should innervate pedunculi and lobes of mushroom bodies are enlarged, as are antennal lobes in anterior brain. At sites of calyces, large numbers of thin axons form distinct lobes outside main neuropile of brain. Viability of mutant females poor, and they are sterile as well. Development of mushroom bodies apparently normal until pupation, when "reconstruction" of this bilaterally paired brain entity (this process being a normal feature of wild-type metamorphosis) is aberrant (Technau and Heisenberg, 1982). Supernumerary neuroblasts observed in the larval brain, owing to anomalous proliferation of such cells post-embryonically.

alleles:

allele	origin	discoverer	synonym	comments
<i>mud</i> ¹	EMS	Heisenberg	<i>mud</i> ^{KS63}	
<i>mud</i> ²	P	Fischbach	<i>mud</i> ^{UB686}	no P insert
<i>mud</i> ³	EMS	Heisenberg		
<i>mud</i> ⁴	EMS	Heisenberg		

cytology: maps to 12E9-11 (Fischbach), based on its inclusion in *Df(1)KA9 = Df(1)12E2-3;12F5-13A1* but in neither *Df(1)g-1 = Df(1)12A;12E9-11* nor *Df(1)RK5 = Df(1)12E9-11;13A9-B1*.

mud: see *mudl*

Mud: Muddled

location: 1- (rearranged).

discoverer: E.H. Grell.

phenotype: Heterozygote has rough eyes with fused facets; eye color brownish. Hemizygous males have low viability but are fertile.

cytology: Associated with *In(1)Mud = In(1)3C3-4;5A6-B1*.

other information: Not allelic to *Sc*².

mudl: mudlike

location: 3-50.5.

origin: Spontaneous.

synonym: *mud*.

references: Aparisi and Nájera, 1988, DIS 67: 4-5 and 5-6.

phenotype: Eye color grayish brown.

mul: multiple

location: 1-0.0 (between *l(1)Ca* and *tw*).

phenotype: Eyes rough and oval. Wings weak and held out. Bristles occasionally missing or disarranged. Body may show abnormal protuberances covered with hairs. Female sterile. After a few generations in stock, *mul*¹ showed only the eye abnormality. RK2.

alleles:

allele	origin ^α	discoverer	synonym	ref ^β	comments
<i>mul</i> ¹	spont	Neel, 41c13		4	
<i>mul</i> ²	X ray	Lefevre	<i>l(1)JF1</i>	3	
<i>mul</i> ³	Dysgenesis	Eeken	<i>l(1)D1</i>	2	P element insert
<i>mul</i> ⁴	Dysgenesis	Eeken	<i>l(1)D52</i>	2	P element insert
<i>mul</i> ⁵	ENU	Voelker	<i>l(1)A89</i>	1	
<i>mul</i> ⁶	ENU	Voelker	<i>l(1)A110</i>		
<i>mul</i> ⁷	ENU	Voelker	<i>l(1)B23</i>		
<i>mul</i> ⁸	ENU	Voelker	<i>l(1)B37</i>		
<i>mul</i> ⁹	ENU	Voelker	<i>l(1)B41</i>		
<i>mul</i> ¹⁰	ENU	Voelker	<i>l(1)B42</i>		
<i>mul</i> ¹¹	EMS	Voelker	<i>l(1)C7</i>		

allele	origin ^α	discoverer	synonym	ref ^β	comments
<i>mul</i> ¹²	EMS	Voelker	<i>l(1)C8</i>		

^α 1 = Eberl, Hilliker, and Voelker, 1988, DIS 67: 36; 2 = Eeken, Sobels, Hyland, and Schalet, 1985, Mutat. Res. 150: 261-75; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 4 = Neel, 1942, DIS 16: 51.

cytology: Located in 1C by deficiency analysis (Voelker).

multimorph: see *bcd*

multiple: see *mul*

multiple wing hairs: see *mwh*

Multiple sex comb: see *Msc*

mum: see *bcd*

mummy: see *mmy*

***mur: murrey**

location: 1-14.3.

origin: Spontaneous as one mosaic male.

discoverer: E. H. Grell, 57c.

references: 1957, DIS 31: 81.

phenotype: At 25°, eye color reddish purple, bristles very small, and body size reduced. At 17°, eye color and body size normal but bristles rather small. Original mosaic male transmitted only an X containing *mur*. He was mated to his daughters to produce homozygous *mur* females. *mur/mur* female and *mur* male are sterile. RK3.

murky: see *mk*

murrey: see *mur*

mus: see *mbm*

mus: mutagen sensitive

A series of mutants selected on the basis of their enhanced sensitivity to various mutagenic treatments. The nomenclatural conventions employed depart somewhat from those of other mutants that share a particular phenotype. *mus* followed by a number between 101 and 199 indicates a mutation on the X chromosome, 201-299 on the second chromosome, and 301-399 on the third chromosome; thus *mus101* [formerly redundantly designated *mus(1)101*] represents the first locus identified on the X. Some allelic designations begin with an upper-case letter indicating the laboratory in which the allele was selected; D = University of California, Davis, A = Emory University in Atlanta, B = University of British Columbia.

mus101 (J.B. Boyd)

location: 1-44.2 [0.2 cM to the left of *g* (Schalet)].

references: Boyd, Golino, Nguyen, and Green, 1976, Genetics 84: 485-506.

Boyd and Setlow, 1976, Genetics 84: 507-26.

phenotype: Survival of homozygous and hemizygous larvae hypersensitive to exposure to methyl methanesulfonate, nitrogen mustard, 2-acetylaminofluorene, and gamma rays. Partially deficient in post-replication repair (Boyd and Setlow, 1976; Brown, and Boyd, 1981, Mol. Gen. Genet. 183: 356-62); nitrogen-mustard mutagenesis abolished; *mus101*⁺ implicated in recovery from DNA crosslinking (Graf, Green, and Würzler, 1979, Mutat. Res. 63: 101-12). Displays hypermutability to alkylating

agents; defective in alkylation repair pathway (Smith and Dusenberg, 1988, Mechanisms and Consequences of DNA Damage Processing, Alan R. Liss, Inc., pp. 251-55). Increases X-ray-induced post-meiotic chromosome loss (Cooper and Zimmering, 1981, Mutat. Res. 81: 345-56). Mutants inhibit premeiotic rDNA magnification in males (Hawley, Marcus, Cameron, Schwartz, and Zitron, 1985, Proc. Nat. Acad. Sci. USA 82: 8095-99). *mus101* function required for chorion-gene amplification (Snyder, Galanopoulos, and Kafatos, 1986, Proc. Nat. Acad. Sci. USA 83: 3341-45). Homozygotes for female-fertile allele exhibit elevated meiotic nondisjunction (Boyd *et al.*, 1976). Causes mitotic chromosome instability (*mus101*^{D1}, Baker and Smith, 1979, Genetics 92: 833-47), and defective mitotic condensation of heterochromatin (*mus*^{ts1}, Gatti, Smith, and Baker, 1983, Science 221: 83-85).

alleles:

allele	origin	discoverer	ref ^α	comments
<i>mus101</i> ^{D1}	EMS	Boyd	1	female fertile
<i>mus101</i> ^{D2}	EMS	Boyd	1	female sterile
<i>mus101</i> SM	<i>mei-9</i> ^β	Schalet		lethal
<i>mus101</i> ^{ts1}	EMS	Baker	2	

^α 1 = Boyd, Golino, Nguyen, and Green, 1976, Genetics 84: 485-506; 2 = Gatti, Smith, and Baker, 1983, Science 221: 83-85.

^β Spontaneous in the paternal X chromosome in a cross of wild-type males to *mei-9* females such that the F₁ female was *mus101*SM/*mei9*.

cytology: Located in 12A6-D3 (Mason, Green, Shaw, and Boyd, 1981, Mutat. Res. 81: 329-43).

***mus102* (J.B. Boyd)**

location: 1-0.5 (Smith, 1976).

references: Boyd, Golino, Nguyen, and Green, 1976, Genetics 84: 485-506.

Smith, 1976, Mol. Gen. Genet. 149: 73-85.

phenotype: Survival of homozygous and hemizygous larvae hypersensitive to exposure to methyl methanesulfonate, formaldehyde (Alexandrov, Alexandrova, and Ankina, 1982, DIS 58: 12-13), and gamma rays. No evidence of hypermutability to alkylation nor defects in excision repair functions (Smith and Dusenberg, 1988, Mechanisms and Consequences of DNA Damage Processing, Alan R. Liss, Inc., pp. 251-55). Homozygous females fertile; exhibit elevated frequencies of nondisjunction. Increases frequency of *mwh* clones in *mwh*⁺ wings; also increases incidence of isochromatid breaks in the heterochromatin and euchromatin of mitotic cells, with breaks occurring 1.4-1.6 times more frequently in females than males (Gatti, 1979, Proc. Nat. Acad. Sci. USA 76: 1377-81).

alleles:

allele	origin	discoverer	ref ^α
<i>mus102</i> ^{A1}	EMS	Smith	3
<i>mus102</i> ^{A2}	EMS	Smith	3
<i>mus102</i> ^{A3}	EMS	Smith	3
<i>mus102</i> ^{A4}	EMS	Smith	3
<i>mus102</i> ^{A5}	EMS	Smith	3
<i>mus102</i> ^{A6}	EMS	Smith	3
<i>mus102</i> ^{A7}	EMS	Smith	3
<i>mus102</i> ^{A8}	EMS	Smith	3
<i>mus102</i> ^{D1}	EMS	Boyd	1
<i>mus102</i> ^{D2}	EMS	Boyd	1
<i>mus102</i> ^{D3}	EMS	Mason	2

^α 1 = Boyd, Golino, Nguyen, and Green, 1976, Genetics 84: 485-506; 2 = Mason, Green, Shaw, and Boyd, 1981, Mutat. Res. 81: 329-43; 3 = Smith, 1976, Mol. Gen. Genet. 149: 73-85.

cytology: Placed between 1B14 and 2B17-18 (Baker and Smith, 1979, Genetics 92: 833-47).

mus103: see *mei41*

mus104: see *mei41*

***mus105* (J.B. Boyd)**

location: 1- {14}.

synonym: *l(1)d.deg-1*.

references: Boyd, Golino, Nguyen, and Green, 1976, Genetics 84: 485-506.

Smith, 1976, Mol. Gen. Genet. 149: 73-85.

phenotype: Strong alleles originally recovered as late larval lethals with degenerate imaginal discs (Stewart, Murphy, and Fristrom, 1972, Dev. Biol. 27: 71-83; Baker, Smith, and Gatti, 1982, Proc. Nat. Acad. Sci. USA 79: 1205-09). Weaker alleles viable and female fertile; hypersensitive to methyl methanesulfonate; interact synergistically with *mus109*^{D1}; increase the incidence of *mwh* clones in *mwh*⁺ wings. All alleles tested display increased frequencies of breaks and exchanges in euchromatin of mitotic cells, with breaks occurring 1.5 times more frequently in females than males (Baker, Smith, and Gatti, 1982, Proc. Nat. Acad. Sci. USA 79: 1205-09; Gatti, 1979, Proc. Nat. Acad. Sci. USA 76: 1377-81).

alleles:

allele	origin	discoverer	synonym	ref ^α
<i>mus105</i> ^{A1}	EMS	Smith		3
<i>mus105</i> ^{D1}	EMS	Boyd		2
<i>mus105</i> ^{1a}	EMS		<i>l(1)d.deg-1</i> ^a	1, 4
<i>mus105</i> ^{1b}	EMS		<i>l(1)d.deg-1</i> ^b	4
<i>mus105</i> ^{1c}	EMS		<i>l(1)d.deg-1</i> ^c	1, 4
<i>mus105</i> ^{1d}	EMS		<i>l(1)d.deg-1</i> ^d	1, 4

^α 1 = Baker, Smith, and Gatti, 1982, Proc. Nat. Acad. Sci. USA 79: 1205-09; 2 = Boyd, Golino, Nguyen, and Green, 1976, Genetics 84: 485-506; 3 = Smith, 1976, Mol. Gen. Genet. 149: 73-85; 4 = Stewart, Murphy, and Fristrom, 1972, Dev. Biol. 27: 71-83.

cytology: Placed in 5A8-C3 on the basis of its inclusion in *Df(1)C149 = Df(1)5A8-9;5C5-6*, but not in *Df(1)N73 = Df(1)5C2-3;5D5-6* (Baker and Smith, 1979, Genetics 92: 833-47).

mus106

location: 1- {*m - g* [Smith *et al.*, 1980, DNA Repair and Mutagenesis in Eukaryotes (Generoso, Shelby, and de Serres, eds.). Plenum, New York, pp. 175-88]}.

references: Boyd, Golino, Nguyen, and Green, 1976, Genetics 84: 485-506.

phenotype: Survival of homozygous and hemizygous larvae hypersensitive to exposure to methyl methanesulfonate and gamma rays. Hypermutable to alkylating agents; defective in alkylation repair pathway (Smith and Dusenberg, 1988, Mechanisms and Consequences of DNA Damage Processing, Alan R. Liss, Inc., pp. 251-55). Homozygous females sterile.

alleles: A single ethyl-methanesulfonate-induced allele, *mus106*^{D1}.

mus107^{D1}: see *mus109*^{D1}

mus108**location:** 1-11.**references:** Smith, Snyder, and Dusenbery, 1980, DNA Repair and Mutagenesis in Eukaryotes (Generoso, Shelby and de Serres, eds.). Plenum Press, New York, pp. 175-88.**phenotype:** Survival of homozygous and hemizygous larvae hypersensitive to exposure to methyl methanesulfonate (Smith *et al.*) and gamma rays (Oliveri, Harris, and Boyd, 1990, *Mutat. Res.* 235: 25-31). Moderately deficient in post-replication repair (Boyd and Shaw, 1982, *Mol. Gen. Genet.* 186: 289-94). Both premeiotic and meiotic magnification of rRNA sequences inhibited in males (Hawley, Marcus, Cameron, Schwartz, and Zitron, 1985, *Proc. Nat. Acad. Sci. USA* 82: 8095-99).**mus109****location:** 1-30.2.**references:** Smith, 1976, *Mol. Gen. Genet.* 149: 73-85. Mason, Green, Shaw, and Boyd, 1981, *Mutat. Res.* 81: 329-43.**phenotype:** Strong alleles are recessive lethal; for weaker alleles, survival of homozygous and hemizygous larvae hypersensitive to exposure to methyl methanesulfonate (Mason *et al.*; Smith *et al.*) and gamma rays (Oliveri, Harris, and Boyd, 1990, *Mutat. Res.* 235: 25-31). Exhibits synergistic interaction with *mus105^{D1}* (Baker, Smith, and Gatti, 1982, *Proc. Nat. Acad. Sci. USA* 79: 1205-09). Females homozygous for viable alleles are sterile. Causes increased incidence of *mwh* clones in *mwh/+* wings (Baker and Smith, 1979, *Genetics* 92: 833-47) and chromosome breakage at euchromatic-heterochromatic junctions in larval ganglion cells (Gatti, 1979, *Proc. Nat. Acad. Sci. USA* 76: 1377-81; Baker, Smith, and Gatti, 1982, *Proc. Nat. Acad. Sci. USA* 79: 1205-09).**alleles:**

allele	origin	discoverer	synonym	ref ^α
<i>mus109^{A1}</i>	EMS	Smith		4
<i>mus109^{D1}</i>	EMS	Boyd	<i>mus107^{D1}</i>	3
<i>mus109^{D2}</i>	EMS	Mason		2
<i>mus109^{IS}</i>	spont	Schalet		1

^α 1 = Baker, Smith, and Gatti, 1982, *Proc. Nat. Acad. Sci. USA* 79: 1205-09; 2 = Mason, Green, Shaw, and Boyd, 1981, *Mutat. Res.* 81: 329-43; 3 = Nguyen, Green, and Boyd, 1978, *Mutat. Res.* 49: 139-43; 4 = Smith, 1976, *Mol. Gen. Genet.* 149: 73-85.

cytology: Placed in 8E-9B2 based on the female sterility of *mus109* alleles in combination with *Df(1)C52 = Df(1)8E,9C-D* but not *Df(1)v-L15 = Df(1)9B1-2;10A1-2* (Baker and Smith, 1979, *Genetics* 92: 833-47). Restricted to 8F3-9A2 by Mason based on refined cytology of the above deficiencies.*mus110^{A1}*: see *mei-9^{A1}***mus111 (J.B. Boyd)****location:** 1-27.**origin:** Induced by ethyl methanesulfonate.**references:** Mason, Green, Shaw, and Boyd, 1981, *Mutat. Res.* 81: 329-43.**phenotype:** Survival of homozygous and hemizygous larvae hypersensitive to exposure to methyl methanesulfonate, nitrogen mustard, gamma rays and X rays (Oliveri, Harris, and Boyd, 1990, *Mutat. Res.* 235: 25-

31). Homozygous females sterile.

mus201**location:** 2-23 (based on 14 recombinants between *dp* and *b*).**references:** Boyd, Snyder, Harris, Presley, Boyd, and Smith, 1982, *Genetics* 100: 239-57.**phenotype:** Larval survival hypersensitive to exposure to methyl methanesulfonate, nitrogen mustard, and ultraviolet light; weakly hypersensitive to X rays. Female fertility unimpaired, and meiotic disjunction regular. Homozygotes devoid of detectable excision repair (Boyd *et al.*; Dusenbery, McCormick, and Smith, 1983, *Mutat. Res.* 112: 215-30); postreplication repair and repair of single-strand breaks induced by X rays normal. Hypermutable to alkylating agents (Smith and Dusenbery, 1988, *Mechanisms and Consequences of DNA Damage Processing*, Alan R. Liss, Inc., pp. 251-55). Mutant defect rescued by transformation with the endonuclease V gene (*denV*) of bacteriophage T4 (Banga, Boyd, Valerie, Harris, Kurz, and de Riel, 1989, *Proc. Nat. Acad. Sci. USA* 86: 3227-31).**alleles:** Two alleles: *mus201^{A1}*, recovered by Smith among chromosomes treated with ethyl-methanesulfonate by Hardy and Orevi; and *mus201^{D1}* (Boyd, Golino, Shaw, Osgood and Green, 1981, *Genetics* 97: 607-23).**mus202****location:** 2- (not located).**origin:** Induced by ethyl methanesulfonate.**references:** Snyder and Smith, 1982, *Mol. Gen. Genet.* 188: 249-55.**phenotype:** Larval survival hypersensitive to exposure to methyl methanesulfonate, nitrogen mustard, formaldehyde, ultraviolet light, and X rays. Fecundity of homozygous females reduced, although partial rescue effected by fertilization with sperm carrying *mus202⁺*. X-chromosome disjunction regular; slight increase in the number of fourth-chromosome exceptions noted.**alleles:** One allele, *mus202^{A1}*.**mus203****location:** 2- (not located).**origin:** Induced by ethyl methanesulfonate.**references:** Snyder and Smith, 1982, *Mol. Gen. Genet.* 188: 249-55.**phenotype:** Larval survival hypersensitive to exposure to methyl methanesulfonate, nitrogen mustard, formaldehyde, ultraviolet light, and X rays. Fecundity of homozygous females reduced, although partial rescue effected by fertilization with sperm carrying *mus203⁺*. Small but significant increase in X- and fourth-chromosome nondisjunction in the first but not the second meiotic division; X-chromosome recombination reduced to 80% normal levels.**alleles:** One allele, *mus203^{A1}*.**mus204****location:** 2- (not located).**origin:** Induced by ethyl methanesulfonate.**references:** Snyder and Smith, 1982, *Mol. Gen. Genet.* 188: 249-55.**phenotype:** Larval survival hypersensitive to exposure to methyl methanesulfonate, formaldehyde, and ultraviolet light, weakly sensitive to nitrogen mustard, but insensitive to X rays. Hypermutability to alkylating agents;

defective both in alkylation and UV excision repair pathways (Smith and Dusenberg, 1988, Mechanisms and Consequences of DNA Damage Processing, Alan R. Liss, Inc., pp. 251-55). Fecundity of homozygous females reduced, although partial rescue effected by fertilization with sperm carrying *mus204*⁺. Small but significant increase in X- and fourth-chromosome nondisjunction in the first but not the second meiotic division; X-chromosome recombination reduced to 80% normal levels.

alleles: One allele, *mus204*^{A1}.

mus205

location: 2-54.9

origin: Induced by ethyl methanesulfonate.

references: Snyder and Smith, 1982, Mol. Gen. Genet. 188: 249-55.
Henderson, Bailey, Sinclair, and Grigliatti, 1987, Mutat. Res. 177: 83-93.

phenotype: Larval survival hypersensitive to exposure to methyl methanesulfonate and ultraviolet light, moderately sensitive to benzo[a]pyrene, but not to formaldehyde, nitrogen mustard or ionizing radiation. Partially deficient in excision repair (Boyd and Harris, 1981, Chromosoma 97: 607-23) and postreplication repair (Boyd and Shaw, 1982, Mol. Gen. Genet. 186: 289-94; Brown, and Boyd, 1981, Mol. Gen. Genet. 183: 356-62). Displays hypermutability to alkylating agents; defective both in alkylation and UV excision repair pathways (Smith and Dusenberg, 1988, Mechanisms and Consequences of DNA Damage Processing, Alan R. Liss, Inc., pp. 251-55). Fecundity of homozygous females reduced, although partial rescue effected by fertilization with sperm carrying *mus205*⁺. X-chromosome disjunction regular; slight increase in the number of fourth-chromosome exceptions noted.

alleles: Two alleles, *mus205*^{A1} and *mus205*^{B1} (Henderson *et al.*).

mus206

location: 2- (not located).

origin: Induced by ethyl methanesulfonate.

references: Snyder and Smith, 1982, Mol. Gen. Genet. 188: 249-55.

phenotype: Larval survival hypersensitive to exposure to methyl methanesulfonate, nitrogen mustard, and ultraviolet light, but not to formaldehyde or X rays. Partially deficient in postreplication repair (Boyd and Shaw, 1982, Mol. Gen. Genet. 186: 289-94). Hypermutability to alkylating agents; defective in alkylation repair pathway (Smith and Dusenberg, 1988, Mechanisms and Consequences of DNA Damage Processing, Alan R. Liss, Inc., pp. 251-55). Fecundity of homozygous females normal.

alleles: One allele, *mus206*^{A1}.

mus207

location: 2- (not located).

origin: Induced by ethyl methanesulfonate.

references: Snyder and Smith, 1982, Mol. Gen. Genet. 188: 249-55.

phenotype: Larval survival hypersensitive to exposure to methyl methanesulfonate, nitrogen mustard, and ultraviolet light; weakly sensitive to formaldehyde and X rays. Hypermutability to alkylating agents; defective in alkylation repair pathway (Smith and Dusenberg, 1988,

Mechanisms and Consequences of DNA Damage Processing, Alan R. Liss, Inc., pp. 251-55). Fecundity of homozygous females normal.

alleles: One allele, *mus207*^{A1}.

mus208

location: 2-89.

origin: Induced by ethyl methanesulfonate.

references: Henderson, Bailey, Sinclair, and Grigliatti, 1987, Mutat. Res. 177: 83-93.

phenotype: Larval survival hypersensitive to exposure to methyl methanesulfonate, *N*-acetyl-2-aminofluorene (*mus208*^{B2} only weakly sensitive), and benzo[a]pyrene; slight allele-specific sensitivity to nitrogen mustard, and gamma rays.

alleles: Two alleles, *mus208*^{B1} and *mus208*^{B2}.

mus209

location: 2-92.8.

origin: Induced by ethyl methanesulfonate.

references: Henderson, Bailey, Sinclair, and Grigliatti, 1987, Mutat. Res. 177: 83-93.

phenotype: Larval survival hypersensitive to exposure to methyl methanesulfonate and gamma rays, but not to *N*-acetyl-2-aminofluorene, benzo[a]pyrene, or nitrogen mustard.

alleles: One allele, *mus209*^{B1}.

mus210

location: 2-69.1.

origin: Induced by ethyl methanesulfonate.

synonym: *mus(2)201*^{G1} (*mus201* preoccupied); *mus212*.

references: Khromykh and Zakharov, 1981, Genetika (Moscow) 17: 658-66.

Luchkina, Khromykh, and Sharigin, 1982, Genetika (Moscow) 18: 625-33.

Levina and Sharigin, 1984, Genetika (Moscow) 20: 416-24.

Henderson, Bailey, Sinclair, and Grigliatti, 1987, Mutat. Res. 177: 83-93.

phenotype: Larval survival hypersensitive to exposure to methyl methanesulfonate, *N*-acetyl-2-aminofluorene, and benzo[a]pyrene, moderately sensitive to nitrogen mustard, and insensitive to gamma rays. Excision of pyrimidine dimers impaired; 4-5 fold decrease in activity of ultra-violet-specific endonuclease observed in primary cell cultures.

alleles: Two alleles, *mus210*^{B1} and *mus210*^{G1}.

mus211

location: 2-47 (alleles mapped to 50.4 and 44.7).

origin: Induced by ethyl methanesulfonate.

references: Henderson, Bailey, Sinclair, and Grigliatti, 1987, Mutat. Res. 177: 83-93.

phenotype: Larval survival hypersensitive to exposure to methyl methanesulfonate, nitrogen mustard, and gamma rays; insensitive to *N*-acetyl-2-aminofluorene and benzo[a]pyrene.

alleles: Two alleles, *mus211*^{B1} and *mus211*^{B2}.

mus212: see *mus210*

mus301 (J.B. Boyd)

location: 3-23 (between *ru* and *h*).

references: Boyd, Golino, Shaw, Osgood and Green, 1981, Genetics 97: 607-23.

phenotype: Larval survival hypersensitive to exposure to X rays (Oliveri, Harris, and Boyd, 1990, *Mutat. Res.* 235: 25-31), methyl methanesulfonate, and nitrogen mustard. When fertile, homozygous females exhibit elevated frequencies of nondisjunction.

alleles:

allele	origin	discoverer	comments
<i>mus301</i> ^{D1}	EMS	Boyd	fertile
<i>mus301</i> ^{D2}	EMS	Boyd	male sterile
<i>mus301</i> ^{D3}	EMS	Boyd	male sterile
<i>mus301</i> ^{D4}	EMS	Boyd	female sterile
<i>mus301</i> ^{D5}	EMS	Boyd	fertile

mus302 (J.B. Boyd)

location: 3-45 (between *st* and *cu*).

references: Boyd, Golino, Shaw, Osgood and Green, 1981, *Genetics* 97: 607-23.

phenotype: Larval survival hypersensitive to exposure to X rays (Oliveri, Harris, and Boyd, 1990, *Mutat. Res.* 235: 25-31), methyl methanesulfonate, and nitrogen mustard. Postreplication repair deficient (Boyd and Shaw, 1982, *Mol. Gen. Genet.* 186: 289-94); partially excision deficient (Boyd and Harris, 1981, *Chromosoma* 97: 607-23). Homozygotes exhibit increased X-ray-induced chromosome loss in postmeiotic cells (Cooper and Zimmering, 1981, *Mutat. Res.* 81: 345-56), increased sex-chromosome loss following treatment of males with procarbazine and diethylnitrosamine (Zimmering, 1982, *Mutat. Res.* 103: 141-44), and decreased incorporation of DNA precursors following ultra-violet irradiation (Brown, and Boyd, 1981, *Mol. Gen. Genet.* 183: 356-62).

alleles:

allele	origin	discoverer	comments
<i>mus302</i> ^{D1}	EMS	Boyd	
<i>mus302</i> ^{D2}	EMS	Boyd	homozygous sterile
<i>mus302</i> ^{D3}	EMS	Boyd	
<i>mus302</i> ^{D4}	EMS	Boyd	
<i>mus302</i> ^{D5}	EMS	Boyd	
<i>mus302</i> ^{D6}	EMS	Boyd	

mus304 (J.B. Boyd)

location: 3-46 (between *st* and *cu*).

references: Boyd, Golino, Shaw, Osgood and Green, 1981, *Genetics* 97: 607-23.

phenotype: Larval survival hypersensitive to exposure to methyl methanesulfonate and nitrogen mustard. Neither hypermutable to alkylating agents nor defective in alkylation repair pathway; however, defective in UV-damage repair pathway (Smith and Dusenberg, 1988, *Mechanisms and Consequences of DNA Damage Processing*, Alan R. Liss, Inc., pp. 251-55). When fertile, homozygous females exhibit uniformly decreased recombination along the X chromosome (Green, 1982, *Biol. Zentralbl.* 101: 223-26). Partially deficient in post-replication repair (Boyd and Shaw, 1982, *Mol. Gen. Genet.* 186: 289-94) and excision (Boyd and Harris, 1981, *Chromosoma* 97: 607-23); DNA synthesis modified (Boyd and Shaw).

alleles:

allele	origin	discoverer	comments
<i>mus304</i> ^{D1}	EMS	Boyd	female fertile
<i>mus304</i> ^{D2}	EMS	Boyd	female sterile

allele	origin	discoverer	comments
<i>mus304</i> ^{D3}	EMS	Boyd	female fertile; sensitive only to MMS

mus305 (J.B. Boyd)

location: 3-44 (between *st* and *cu*).

references: Boyd, Golino, Shaw, Osgood and Green, 1981, *Genetics* 97: 607-23.

phenotype: Larval survival hypersensitive to exposure to X rays (Oliveri, Harris, and Boyd, 1990, *Mutat. Res.* 235: 25-31), methyl methanesulfonate, and nitrogen mustard. Homozygous females display elevated rates of nondisjunction.

alleles:

allele	origin	discoverer	comments
<i>mus305</i> ^{D1}	EMS	Boyd	
<i>mus305</i> ^{D2}	EMS	Boyd	male sterile
<i>mus305</i> ^{D3}	EMS	Boyd	

mus306 (J.B. Boyd)

location: 3-56 (between *cu* and *sr*).

origin: Induced by ethyl methanesulfonate.

references: Boyd, Golino, Shaw, Osgood and Green, 1981, *Genetics* 97: 607-23.

phenotype: Larval survival sensitive to exposure to methyl methanesulfonate. Displays hypermutability to alkylating agents; defective both in alkylation and UV excision repair pathways (Smith and Dusenberg, 1988, *Mechanisms and Consequences of DNA Damage Processing*, Alan R. Liss, Inc., pp. 251-55). Homozygotes fertile. Partially excision deficient (Boyd and Harris, 1981, *Chromosoma* 97: 607-23).

alleles: One allele, *mus306*^{D1}.

mus307 (J.B. Boyd)

location: 3-59 (between *cu* and *sr*).

references: Boyd, Golino, Shaw, Osgood and Green, 1981, *Genetics* 97: 607-23.

phenotype: Larval survival hypersensitive to exposure to X rays (Oliveri, Harris, and Boyd, 1990, *Mutat. Res.* 235: 25-31), methyl methanesulfonate, and nitrogen mustard. Homozygotes fertile. Modified DNA synthesis (Boyd and Shaw, 1982, *Mol. Gen. Genet.* 186: 289-94).

alleles: One allele, *mus307*^{D1}.

mus308 (J.B. Boyd)

location: 3-55 (between *cu* and *sr*).

references: Boyd, Golino, Shaw, Osgood and Green, 1981, *Genetics* 97: 607-23.

phenotype: Larval survival hypersensitive to exposure to X rays (Oliveri, Harris, and Boyd, 1990, *Mutat. Res.* 235: 25-31), and nitrogen mustard. Partially deficient in post-replication repair (Boyd and Shaw, 1982, *Mol. Gen. Genet.* 186: 289-94) and excision (Boyd and Harris, 1981, *Chromosoma* 97: 607-23); DNA synthesis modified (Boyd and Shaw). Displays hypermutability to alkylating agents; defective both in alkylation and UV excision repair pathways (Smith and Dusenberg, 1988, *Mechanisms and Consequences of DNA Damage Processing*, Alan R. Liss, Inc., pp. 251-55). Defective in levels of nuclease 3, which is apparently active in the repair of DNA cross links (Boyd, Sakaguchi, and Harris, 1990, *Genetics* 125: 813-19).

alleles:

allele	origin	discoverer	comments
mus308 D1	EMS	Boyd	homozygous fertile
mus308 D2	EMS	Boyd	
mus308 D3	EMS	Boyd	male sterile
mus308 D4	EMS	Boyd	male sterile
mus308 D5	EMS	Boyd	male sterile
mus308 D6	EMS	Boyd	male sterile

mus309 (J.B. Boyd)

references: Boyd, Golino, Shaw, Osgood and Green, 1981, Genetics 97: 607-23.

phenotype: Larval survival hypersensitive to exposure to methyl methanesulfonate and nitrogen mustard.

alleles:

allele	origin	discoverer	comments
mus309 D1	EMS	Boyd	homozygous sterile
mus309 D2	EMS	Boyd	female sterile
mus309 D3	EMS	Boyd	

mus310 (J.B. Boyd)

location: 3-47 (between *st* and *cu*).

references: Boyd, Golino, Shaw, Osgood and Green, 1981, Genetics 97: 607-23.

phenotype: Larval survival hypersensitive to exposure to methyl methanesulfonate. Post-replication repair deficient with modified DNA synthesis (Boyd and Shaw, 1982, Mol. Gen. Genet. 186: 289-94); reduced incorporation of DNA precursors following ultra-violet treatment (Brown, and Boyd, 1981, Mol. Gen. Genet. 183: 356-62). Displays hypermutability to alkylating agents; defective both in alkylation and UV excision repair pathways (Smith and Dusenberg, 1988, Mechanisms and Consequences of DNA Damage Processing, Alan R. Liss, Inc., pp. 251-55).

alleles: One allele, *mus310 D1*.

mus311 (J.B. Boyd)

location: 3-47 (between *st* and *cu*).

references: Boyd, Golino, Shaw, Osgood and Green, 1981, Genetics 97: 607-23.

phenotype: Larval survival hypersensitive to exposure to methyl methanesulfonate. Post-replication repair deficient with modified DNA synthesis (Boyd and Shaw, 1982, Mol. Gen. Genet. 186: 289-94). When fertile, females produce elevated rates of nondisjunction.

alleles:

allele	origin	discoverer	comments
mus311 D1	EMS	Boyd	female sterile
mus311 D2	EMS	Boyd	female fertile
mus311 D3	EMS	Boyd	female fertile

mus312 (J.B. Boyd)

location: 3-18 (between *ru* and *h*).

references: Boyd, Golino, Shaw, Osgood and Green, 1981, Genetics 97: 607-23.

phenotype: Larval survival hypersensitive to exposure to X rays (Oliveri, Harris, and Boyd, 1990, Mutat. Res. 235: 25-31), methyl methanesulfonate, and nitrogen mustard. Reduces meiotic recombination (Green, 1981, Chromosoma 82: 259-66).

alleles:

allele	origin	discoverer	comments
mus312 D1	EMS	Boyd	homozygous fertile
mus312 D2	EMS	Boyd	male sterile

Muscle myosin heavy chain: see *Mhc*

Muscle protein: see *Mp*

Muscle specific proteins: see *Msp*

mushroom-body-defect: see *mud*

mushroom-body-deranged: see *mbd*

mushroom-body-miniature: see *mbm*

mussed: see *mu*

mutability factor from Florida: see *mu-F*

mutagen sensitive: see *mus*

mutator: see *mu*

mw: mottler of white

location: 1-30.9.

references: 1946, DIS 20: 88-89.

Gelbart, 1971, Genetics 68: s22.

Birchler, 1986, Genetics 113: s47.

Birchler, Hiebert, and Rabinow, 1989, Genes Dev. 3: 73-84.

phenotype: Normal by itself. A specific dilutor of *w^a* and other intermediate alleles at the *w* locus. Eyes assume a lighter mottled appearance. Affects *w^a* and seven other intermediate alleles (Gelbart), including *w^{aa}*, *w^{bf}*, *w^h*, *w^{sp55}*, and *z w^{zm}* (Birchler); without effect on other intermediate alleles. Sensitive alleles are insertion alleles as determined by Zachar and Bingham (1982, Cell 30: 529-41); at least six different transposable elements involved. Insensitive *w* alleles are not insertion mutants. Expression not affected by dosage of *Y* chromosome (Oster, 1957, DIS 31: 150). RK1.

alleles:

allele	origin	discoverer	ref ^α
mw 1	spont	Muller, 1946	4
mw 2	spont	Gelbart	3
mw 2a β	dysgenesis	Birchler	2
mw 3P	dysgenesis	Birchler	1

^α 1 = Birchler, 1986, Genetics 113: s47; 2 = Birchler, Hiebert, and Rabinow, 1989, Gene Dev. 3: 73-84; 3 = Gelbart, 1972, Genetics 68: s22; 4 = Muller, 1946, DIS 20: 88-89.

^β *w^a mw^{2a}/Y* males not mottled like other *w^a mw/Y* males (Birchler *et al.*, 1989).

cytology: Located between 9A2 and 9B1 on the basis of being covered by *Dp(1;2)v⁺75d = Dp(1;2)9A2; 10C2; 40-41* and not being deleted by *Df(1)v-LI5 = Df(1)9B1;10A1*. (Birchler *et al.*, 1989).

mwg: microwing

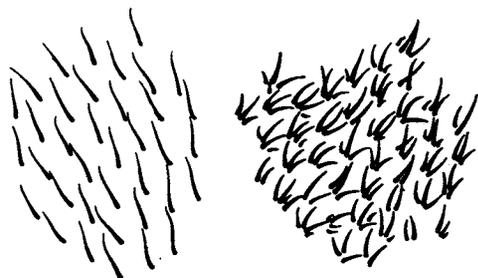
location: 2- (left arm).

origin: Spontaneous.

references: Moran and Neeley, 1971, DIS 46: 43.

phenotype: Wings reduced in size, extending slightly beyond scutellum; only proximal regions of veins obvious; wings usually curled or curved at posterior margins, often filled with fluid. Halteres slightly shorter than nor-

mal. Homozygous females fully fertile.



mwh: multiple wing hairs

Wing hairs. Left: wild type. Right: *mwh*.

A. Di Pasquale, unpublished.

***mwh*: multiple wing hairs**

location: 3-0.3 [between *fap* and *ru* (Robertson and Rivera, 1972, DIS 48: 21; Roberts and Evans-Roberts, 1979, Genetics 93: 663-79). See also Strommer and Falk (1980, DIS 56: 196)].

origin: Spontaneous.

discoverer: di Pasquale, 50l.

references: 1951, DIS 25: 70.

1952, Rend. Ist. Lombardo Sci. Lettere, Ser. B 85: 1-8.

Peyer and Hadorn, 1965, Arch. Julius Klaus-Stift. Vererbungsforsch. Sozialanthropol. Rassenhyg. 40: 19-26 (fig.).

phenotype: Affects the trichomes (hairs) of all body regions in the same general way: An increase in the number of elements is correlated with a reduction in length and a disturbance of orientation. Arista and bracts are included in this pattern; bristles and other sensilla are not. Wing cells contain groups of 2-7 hairs instead of one hair per cell as in wild type; causes supernumerary trichomes over entire integument, but tufts of trichomes only in wing blade (Ouweneel, 1970, Genetica 41: 1-20); may be supernumerary hairs and sensilla on halteres (Ouweneel and van der Meer, 1972, Wilhelm Roux's Arch. Entwicklunsgmech. Org. 172: 149-61). Also causes disruption of polarity in legs, wings, and halteres; may disrupt orientation of hairs on leg without affecting their numbers (Bryant and Schneiderman, 1969, Dev. Biol. 20: 263-90); trichomes on wings tend to diverge from vein L3 rather than parallel it as in wild type (Gubb and García-Bellido, 1982, J. Embryol. Exp. Morphol. 68: 37-57). *mwh/+* develop *mwh* phenotype following heat shock at or just prior to the time of cell-hair-extrusion (Mitchell and Petersen, 1984, Genetics 107: s74). Transplants of mutant wing disks to wild-type hosts develop autonomously (Ursprung and Hadorn, 1962, Dev. Biol. 4: 40-60). Widely used as a cell marker in the analysis of cuticular clones. The frequency of *mwh* spots after somatic recombination in *mwh/+* flies increases with increase in the temperature at which the larvae and pupae are raised (Graf, 1986, DIS 63: 65). RK1.

cytology: Placed in 61E2-62A3 (Roberts and Evans-Roberts).

****mwh^{semi}*: multiple wing hairs-semi**

origin: Spontaneous derivative of *mwh*.

discoverer: di Pasquale, 51e.

phenotype: Like *mwh* except that the groups of wing hairs are restricted to wing margins. Cells of wing surface between second and fifth longitudinal veins have single hair with only an occasional group. *mwh^{semi}/mwh* is like *mwh^{semi}/mwh^{semi}*. RK1.

****mwi*: misheld wings**

location: 1-0.4.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).

discoverer: Fahmy, 1954.

references: 1958, DIS 32: 72.

phenotype: Wings diverge upward and outward at various angles. Eye shape oval. Viability and fertility good in male and reduced in female. RK2.

***Myb*: Myb proto-oncogene sequence**

location: 1- {50}.

origin: Isolated from genomic library using the c-myb sequence as probe.

references: Katzen, Kornberg, and Bishop, 1985, Cell 41: 449-56.

phenotype: Considered to be the *Drosophila* homologue of avian c-myb. Open reading frame identified to date encodes a polypeptide exhibiting 73% homology with chicken c-myb protein.

molecular biology: Cloned region of homology (probably < half the total) sequenced; 67% identity (253/375 residue) with chicken c-myb nucleotide sequence. *Drosophila C-myb* appears to lack two upstream exons present in chicken; the portion cloned comprises a single open reading frame, lacking two introns present in the homologous genomic sequence of the chicken. Homologous transcripts of 3.8 (predominantly) and 3.0 kb found in *Drosophila* embryos.

cytology: Probe hybridizes to the 13E-F boundary.

***Myc*: Myc proto-oncogene sequence**

location: 2- {58}.

origin: Isolated from a genomic library using N-myc probe from human neuroblastoma.

references: Voss, Strand, Anderson, Nystrom, Spiver, and McDonald, 1986, Genetics 113: s37.

cytology: Located to 44A by *in situ* hybridization.

molecular biology: Homologous to a major 7 kb transcript as well as six less abundant transcripts ranging in size from 2-5 kb.

***Myofibrillar contractile protein*:** see *Mfcp*

***myofibrillar defective*:** see *mfd*

***Myosin heavy chain*:** see *Mhc*

***Myosin heavy chain-cytoplasmic*:** see *Mhc-c*

***Myosin light chain*:** see *Mlc*

***mys*: myspheroid**

location: 1-21.7.

references: Rizki, 1956, J. Exp. Zool. 131: 203-22 (fig.).

Wright, 1958, Proc. Intern. Congr. Genet., 10th., Vol. 2: 323.

1960, J. Exp. Zool. 143: 77-99 (fig.).

Leptin, Bogaert, Lehman, and Wilcox, 1989, Cell

56: 401-08.

phenotype: Structural gene for the β subunit of position-specific integrins 1 and 2, PS1 and PS2. Twenty-hour embryos (25°) show middorsal herniation of brain and midgut, or both; abnormal somatic, visceral, and pharyngeal muscles; and incomplete morphogenesis of yolk-filled midgut. Development of embryo normal up to 13 hr, even in embryos produced from homozygous germ-line clones (Wieschaus and Noell, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 63-73). Between 13 and 14.5 hr the first muscular contractions occur, while basement membrane is incomplete. This results in dorsal rupture of hypoderm and retraction of myogenic elements of somatic and pharyngeal muscles into spheroidal masses. Continuation of myogenesis produces spheroidal muscles with a cortex of disoriented fibrillae surrounded by a medulla of nucleated sarcoplasm. Homozygous clones of *mys*¹¹ on either surface of the wing lead to separation of the two surfaces of the membrane and the formation of blisters in the vicinity of the clone [Brower and Jaffe, 1989, Nature (London) 342: 285-87]. Western blots with antibodies specific to the β subunit of *Drosophila* PS integrins detects no β integrin in *mys*¹⁰, *mys*¹¹, and *Df(1)C128*; in addition, PS1 α is not cleaved properly in these genotypes, nor do α chains become localized in mutant embryos. PS β expression in wild type is diffuse in early embryos, becoming localized between the mesodermal and ectodermal layers at the extended-germ-band stage; also seen at interfaces between epidermal cells and deep in the intersegmental grooves where intersegmental muscles attach. In late embryos antibody staining is seen at basal surface of entire gut epithelium and is also concentrated at muscle attachment sites.

alleles:

allele	origin	discoverer	synonym	ref ^{α}	comments
<i>mys</i> ¹	32 P	Poulson 48j	<i>l(1)48j</i>	4, 7, 8	
<i>mys</i> ²	EMS		<i>l(1)40</i>	2	
<i>mys</i> ³	EMS	Lefevre	<i>l(1)DA548</i>	3	
<i>mys</i> ⁴	EMS	Lefevre	<i>l(1)DA573</i>	3	
<i>mys</i> ⁵	EMS	Lefevre	<i>l(1)EF484</i>	3	
<i>mys</i> ⁶	EMS	Lefevre	<i>l(1)VA333</i>	3	
<i>mys</i> ⁷	EMS	Lefevre	<i>l(1)VE609</i>	3	
<i>mys</i> ⁸	EMS		<i>nj42</i>	5	adults survive as nonjumpers

allele	origin	discoverer	synonym	ref ^{α}	comments
<i>mys</i> ⁹	P		<i>l(1)L74</i>	1	roo insert at 21.7 to 23.4 kb
<i>mys</i> ¹⁰	EMS		<i>l(1)mys</i> ^{xB87}	6	
<i>mys</i> ¹¹	EMS		<i>l(1)mys</i> ^{xG43}	6	
<i>mys</i> ¹²	EMS			6	
<i>mys</i> ¹³	EMS			6	
<i>mys</i> ^{ts1}	EMS	Wright		9	E, L
<i>mys</i> ^{ts2}	EMS	Wright		9	E, L
<i>mys</i> ^{ts3}	EMS	Wright		9	E

^{α} 1 = Digan, Haynes, Mozer, Dawid, Forquignon, and Gans, 1986, Dev. Biol. 114: 161-69; 2 = Gans, Audit, and Masson, 1975, Genetics 81: 683-704; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 4 = Rizki, 1956, J. Exp. Zool. 131: 203-22 (fig.); 5 = Thomas, 1982, Neurosci. Abstr. 6: 742; 6 = Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Roux's Arch. Dev. Biol. 193: 296-307; 7 = Wright, 1958, Proc. Int. Congr. Genet., 10th, Vol. 2: 323; 8 = Wright, 1960, J. Exp. Zool. 143: 77-99 (fig.); 9 = Wright, 1968, Proc. Int. Congr. Genet., 12th, Vol. 1: 141.

cytology: Placed in 7D1-6 based on its inclusion in *Df(1)C128 = Df(1)7D1;7D5-6*.

molecular biology: Gene localized in an eighty-kilobase walk by virtue of the *roo* insert associated with *mys*⁹; the position of the insert is in a 1.7 kb restriction fragment between 21.7 and 23.4 kb distal to the first *Sma*I site proximal to *Df(1)C128*, which is designated as the 0 coordinate. *mys*², *mys*⁵ and *mys*⁸ have no gross molecular lesion (Digan *et al.*). Sequences from this region identify a 4.4-kb mRNA on Northern blots; cDNA clones provide a sequence accounting for a message of such length; the conceptual amino-acid sequence contains a 23-amino acid N-terminal signal sequence and a single 23-amino-acid hydrophobic membrane-spanning domain, and specifies an 846-amino-acid polypeptide of 90 kd following removal of the signal sequence; there are six consensus sites for glycosylation of asparagine residues. The molecule contains 56 cystein residues arranged into cystein-rich motifs characteristic of the vertebrate family of β integrins. Overall it displays 45% identity with chicken β integrin with substantial segments in both the intracellular and extracellular domains showing up to 95% identity (MacKrell, Blumberg, Haynes, and Fessler, 1988, Proc. Nat. Acad. Sci. USA 85: 2633-37).

N: Notch (W.J. Welshons)

The Notch locus is involved in the differentiation of the ectoderm and is found in band 3C7. It includes phenotypically distinct regions *Ax*, *Co*, *fa*, *l(1)N*, *N*, *nd*, and *spl*. The wild-type allele of Notch is essential for the proper development of the neurogenic region, appearing to direct some ectodermal elements into the epidermal pathway of development. The expression of the Notch phenotype depends on the dosage of N^+ , two doses in females and one in males being essential to produce wild-type flies. The mutant expression of *N*, characterized by notched wings as well as vein and microchaetal abnormalities (Mohr, 1919, *Genetics* 4: 285-82), is found in N/N^+ females. *l(1)N* mutants, on the other hand, do not show a Notch phenotype over N^+ and are lethal with *N* (Poulson, 1968, *Proc. Int. Congr. Genet.*, 12th, Vol. 1: 143). *Ax* mutants show dominant wing and bristle phenotypes distinct from *N* (Foster, 1975, *Genetics* 81: 99-120; Portin, 1975, *Genetics* 81: 121-33). *Co* is a duplication that affects wing veins when expressed in homo- and hemizygotes. The recessive visible mutants *fa* and *spl* affect the surface of the eye; in heterozygotes with *N*, the eyes are rough. Another recessive visible mutant *nd* (which includes $fa^{no} = nd^3$) shows notched-wings, thickened veins, and interaction with *N* (Bauer, 1943, *Z. Indukt. Abstamm. Vererbungsl.* 81: 374-90; Welshons and Von Halle, 1962, *Genetics* 47: 743-59; Welshons, 1965, *Science* 150: 112-29).

The expression of N^+ during neurogenesis has been examined by *in situ* hybridization (Hartley, Xu, and Artavanis-Tsakonas, 1987, *EMBO J.* 6: 3407-17) and by antibody staining (Kidd, Baylies, Gasic, and Young, 1989, *Genes Dev.* 3: 1113-29; Johansen, Fehon, and Artavanis-Tsakonas, 1989, *J. Cell Biol.* 109: 2428-40). The protein is expressed in cells destined for neural and epidermal lineages and also in mesodermal cells. Later Notch expression is restricted to the neuroblasts and their derivatives, the neurons and nerve processes.

The use of temperature-sensitive mutants, especially *l(1)N^{ts}*, has identified the TSP's for numerous defects at Notch (Shellenbarger and Mohler, 1975, *Genetics* 81: 143-62; 1978, *Dev. Biol.* 62: 432-46). Complementation between alleles can be understood as due to the spatial or temporal separation of defects in the course of development. The defects occur throughout developmental stages from embryo to late pupa.

Notch mutants, recessive lethals, and visibles alter activities of four enzymes of the mitochondrial respiratory chain (Thörig, Heinstra and Scharloo, 1981, *Mol. Gen. Genet.* 182: 31-38; 1981, *Genetics* 99: 65-74; Thörig and Scharloo, 1982, *Genetics* 57: 219-23).

Construction of germ line mosaics homozygous for an *N* mutant reveal the existence of a maternal component of Notch expression (Jiménez and Campos-Ortega, 1982, *Wilhelm Roux's Arch. Dev. Biol.* 191: 191-201), and Notch transcripts can be detected in unfertilized eggs (Artavanis-Tsakonis, Muskavitch, and Yedvobnick, 1983, *Proc. Nat. Acad. Sci. USA* 80: 1977-81). Furthermore, *N* expression is generalized and not confined to tissues affected by mutant alleles; phenotypic effects, therefore, seem to be context-specific (Hartley *et al.*, 1987).

The Notch locus in *D. hydei* shares many similarities with the locus in *D. melanogaster* (Van Breugel, 1971, *Genetica* 42: 25-41; Van Breugel and Van Zyll

Langhout, 1983, *Genetics* 103: 197-217).

The components of Notch are described in separate entries on the following pages.

cytology: Placed in 3C7 since heterozygotes for a mutation or a deficiency involving this band show the Notch phenotype (Slizynska, 1938, *Genetics* 23: 29-99; Demerec, 1939, *Proc. Int. Congr. Genet.*, 7th, pp. 99-103; Demerec, Kaufman, Fano, Sutton, and Sansome, 1942, *Year Book - Carnegie Inst. Washington* 41: 191). High resolution *in situ* hybridization and computer-aided optical microscope data collection and image analysis indicate that coding portions and introns of the Notch gene are contained in 3C7. DNA 5' to the transcription start site lies in the interband distal to 3C7 (Rykowski, Parmelee, Agard, and Sedat, 1988, *Cell* 54: 461-72). Relation to *Co* illustrated by three *T(1;Y)*'s with breakpoints in 3C: *T(1;Y)D7*, *T(1;Y)R38*, and *T(1;Y)W17*. The segmental duplication formed from *T(1;Y)D7* and *T(1;Y)R38* is *Confluens* in phenotype; the corresponding deficiency is normal. The segmental deficiency formed from *T(1;Y)R38* and *T(1;Y)W17* is Notch in phenotype and the corresponding duplication is normal. In addition to *T(1;Y)R38*, two other translocations separate *Co* and *N*; *T(1;Y)R30* and *T(1;Y)J100* (Merriam and colleagues). Many *N* mutants have normal cytology. Translocations and inversions with a breakpoint at or near 3C7 also yield *N* mutants, some of which variegate for *N* and loci linked to it. Notch alleles identified as point mutations, inversions, translocations and transpositions are described in the mutant section as *N* and *l(1)N*, while *N* deficiencies are described in the deficiency section as *Df(1)N*.

Deficiencies and inversions with a breakpoint in Notch can also recombine with mutant sites such that a systematic recombinational analysis will identify the lesion at Notch on the genetic map (Welshons, 1974, *Genetics* 76: 775-94; Welshons and Keppy, 1981, *Mol. Gen. Genet.* 181: 319-24).

molecular biology: Molecular cloning techniques have yielded a physical map of Notch in which a 37-40 kb transcription unit contains sequences homologous to a 10.5-11.7 kb poly(A)⁺ RNA; the DNA sequence of this transcription unit has been determined (Kidd, Lockett, and Young, 1983, *Cell* 34: 421-33; Wharton, Johansen, Xu, and Artavanis-Tsakonas, 1985, *Cell* 40: 567-81; Yedvobnick, Muskavitch, Wharton, Halpern, Paul, Grimwade, and Artavanis-Tsakonas, 1985, *Cold Spring Harbor Symp. Quant. Biol.* 50: 841-54; Kidd, Kelley, and Young, 1986, *Mol. Cell. Biol.* 6: 3094-3108; Kidd and Young, 1986, *Nature* 323: 89-91; Kelley, Kidd, Berg, and Young, 1987, *Mol. Cell Biol.* 7: 1545-48; Kelley, Kidd, Deutsch, and Young, 1987, *Cell* 51: 539-48; Ramos, Grimwade, Wharton, Scottgale, and Artavanis-Tsakonas, 1989, *Genetics* 123: 337-48). Except for portions of the two largest intervening sequences, the 5' and 3' ends of the gene have been mapped, and the location of nine exons have been determined. The major Notch transcript encodes a stable 350 kd transmembrane glycoprotein (2,703 amino acids) which is expressed throughout development. The protein includes extracellular epidermal growth factor-like repeats with cysteines that probably form intramolecular disulfide bonds; different X chromosomes display frequent silent nucleotide substitutions in the EGF-like repeat region (Hartley *et al.*, 1987; Kelley *et al.*, 1987; Artavanis-Tsakonas, 1988, *TIG*

4: 97-100; Kidd *et al.*, 1989; Johansen *et al.*, 1989).

P-element-mediated rescue of the homozygous lethal and heterozygous visible phenotypes of the mutants N^{5419} and N^{60g11} was accomplished with about 40 kb of genomic DNA carried in a Notch cosmid vector (Ramos *et al.*, 1989). A 15-kb "minigene" of cDNA lacking most introns but carrying all the 10.2 kb exons was able to rescue *nd* and partially rescue *fa^{swb}* and *spl*, although it could not rescue *fa^g* and *fa^{no}*.

The zero coordinate used in locating the molecular lesions in the various Notch alleles is the first *EcoRI* site in Canton-S DNA proximal to the 3C7 breakpoint of *In(1)N^{76b8}* (Kidd, Kelley, and Young, 1986, Mol. Cell Biol. 6: 3094-108; Kidd, Lockett, and Young, 1983, Cell 34: 421-33); it is 1.1 kb to the right of the zero coordinate used by Artavanis-Tsakonis *et al.*, 1983.

other information: Combined genetic and physical information is presented in the map of Notch.

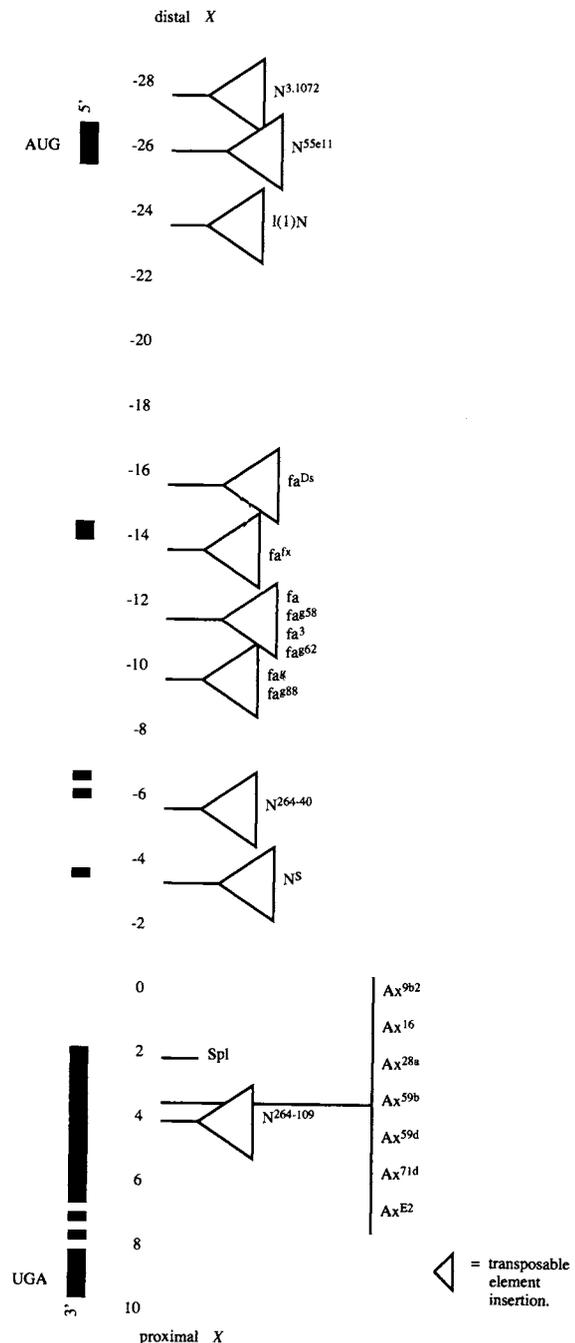
Ax: Abruptex (W.J. Welshons)

location: 1-3.0.

phenotype: Homozygous females and males show shortened L5 vein, usually also L4, L2, and sometimes L3. Wings shortened, arched, and thin. Costal bristles clumped and frayed; costal veins thickened. Thorax shows midfurrow with rearranged hair directions; hairs on thorax and head fewer, with clear patches and streaks. Male genitalia often rotated. *Ax/+* females show short L5 in half of the flies and sparse hair pattern on thorax. Lower temperature (19°) markedly decreases expression, and higher temperature enhances it. Some *Ax* alleles enhance *N* expression in *Ax/N* heterozygotes, but others suppress the dominant *N* phenotype. For example, *Ax/N^g* approaches wild type in all characteristics. No wing-vein interruption in *Ax/+* at 18° and 26°, and enhancement by *H* occurs so that *Ax/Y;H/+* and *Ax/Ax;H/+* are nearly lethal at 26° (House, 1959, Anat. Record 134: 581-82). *Ax/Ax;ci^D/+* and *Ax/Y;ci^D/+* are lethal or nearly so at 26°. At 22°, males survive and show enhanced wing-vein interruption and more missing bristles. At 26°, wing-vein interruption approaches 100% in *Ax/+;ci^D/+* (House and Lutes, 1975, Genetics 80: s42-43).

Wing nicking is suppressed in *Ax/N^{55e11}* at 25°, and *Ax* venation is weakly expressed; *Ax/Ax;Dp(1;2)51b/+* shows weak *Ax* venation (Portin, 1975, Genetics 81: 121-33). Nearly lethal when reared at 29°; temperature-sensitive period early pupa (Portin and Sirén, 1976, Hereditas 84: 109-16). In heterozygotes of *Ax* with the recessives at Notch at 18° and 25°, there is neither expression of the recessive nor *Ax*-type venation. At 29°, only *Ax/fa^{no}* shows some weak expression of the recessive, and all heterozygotes except *Ax/nd²* show some *Ax* venation (Portin, 1977, Hereditas 87: 77-84). *Ax* interacts with alleles *Ax⁹*, *Ax^{59b}*, *Ax^{71d}*, *Ax¹⁶*, and *Ax^{E2}* (see appropriate entry). RK2 in males.

alleles: Three classes of *Ax* mutations can be distinguished: Some alleles (*Ax¹⁶*, *Ax^{71d}* and *Ax^{E2}*) in heterozygous combination with *N* enhance the wing-incision phenotype; others (*Ax¹* and *Ax⁹*) suppress wing incision of *N* and in turn display suppression wing-vein gapping by *N*; yet another class (*Ax^{59b}* and *Ax^{59d}*) are homozygous lethal. Notch-enhancing and Notch-suppressing alleles are homozygous viable, but lethal in heterozygous



Notch transcription and molecular map
Data modified from maps of M. Young. Program by D. Conner.

combination with each other. Phenotypic, cytological, and fine-structure mapping descriptions of these alleles appear below.

allele	origin	discoverer	synonym	ref ^α	comments ^β
Ax¹γ	spont	Nazarenko, 28a	Ax ²⁸	3, 6, 7, 15	Asn ⁹⁸⁶ → Ile
Ax⁹γ	EMS	Lefevre	Ax ^{9B2-2b}	2, 3, 8, 9, 10	Asp ⁹⁴⁸ → Val
Ax¹⁶γ	EMS	Lewis & Bacher	Ax ¹⁶¹⁷²	1, 2, 3, 8, 10	Gly ¹¹⁷⁴ → Ala
Ax^{59b}γδ	X ray	Green		3, 8, 10, 11, 12, 13, 16	Cys ⁹⁷² → Gly
Ax^{59d}γδ	X ray	Green		3, 8, 11, 13, 16	Cys ⁹⁷² → Tyr Ser GT replaced by ATATA Ser ¹⁰⁸⁸ → Ile
Ax^{71d}γ	X ray	Portin & Ruohonen		3, 8, 10	
Ax^{75c}	EMS	Welter	Ax ^{75c24}	8, 13	Canton S
Ax^{E1}	EMS			2	Oregon R
Ax^{E2}γ	EMS	Foster		2, 3, 8, 9, 10, 14	Oregon R His ¹¹⁶⁷ → Tyr
Ax^{J14}	X ray			5	
Ax^S	spont	Schalet	<i>l(1)16-55</i>	4	
Ax^{tsl}	EMS	Shellenbarger	<i>l(1)Ax^{tsl}</i>	15	Oregon R

^α 1 = Foster, 1973, Dev. Biol. 32: 282-96; 2 = Foster, 1975, Genetics 81: 99-120; 3 = Kelley, Kidd, Deutsch, and Young, 1987, Cell 51: 539-48; 4 = Kidd, Lockett, and Young, 1983, Cell 34: 421-33; 5 = Lefevre, 1974, DIS 51: 22; 6 = Mohr, 1932, Prox. Intern. Congr. Genet., 6th, Vol. 1: 190-212 (fig.); 7 = Nazarenko, 1930, Biol. Zentralbl. 50: 385-92 (fig.); 8 = Portin, 1975, Genetics 81: 121-33; 9 = Portin, 1977, Genetics 86: 309-19; 10 = Portin, 1977, Hereditas 87: 77-84; 11 = Portin, 1980, Hereditas 92: 303-07; 12 = Portin, 1981, Hereditas 94: 93-98; 13 = Portin, 1981, Hereditas 95: 247-51; 14 = Portin and Sirén, 1976, Hereditas 84: 109-16; 15 = Shellenbarger and Mohler, 1975, Genetics 81: 143-62; 16 = Welshons, 1971, Genetics 68: 259-68;

^β Background on which mutation induced.

^γ Mutants partially sequenced; each correlated with single amino acid substitutions within six adjacent EGF-homologous elements of the *N* protein; all have Gly at residue 2057 characteristic of Oregon R rather than Ser of Canton S (Kelley *et al.*, 1987).

^δ Both alleles show changes in cysteine (cysteine to glycine in Ax^{59b}, cysteine to tyrosine-serine in Ax^{59d}). Developmental studies of Young have shown that Ax^{59d} is a larval lethal and Ax^{59b} is a pupal lethal (Kelley *et al.*, 1987).

Ax¹

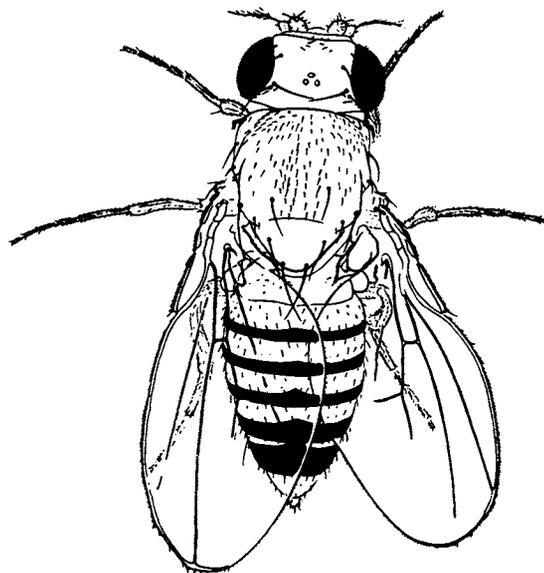
phenotype: Temperature-sensitive lethal; male viable at 25° but nearly lethal at 29°. Ax¹/Ax^{E2} semilethal at 25° and lethal at 29°. Temperature-sensitive period for lethality of Ax¹ at beginning of pupal stage; of Ax¹/Ax^{E2} at end of third instar and into early pupal stage.

cytology: A single-band duplication, presumed to be for 3C7 by Schultz (Morgan, Schultz, and Curry, 1941, Year Book - Carnegie Inst. Washington 40: 283). Lefevre, Ratty, and Hanks (1953, Genetics 38: 345-59), on the other hand, argue against a duplication for 3C7 on the basis of equal X-ray-induced mutability to *N* in Ax and +. Molecular information also incompatible with the presence of a duplication.

molecular biology: Asparagine replaced by isoleucine at residue 986 in EGF repeat #25; AAT → ATT (Kelley *et al.*, 1987).

Ax⁹

phenotype: Viable in both sexes but poorly fertile or sterile. Bristle loss and vein interruptions are more extreme at 29°. Heterozygotes of Ax⁹ with Ax¹ and Ax^{E1} are viable, but Ax⁹ is inviable with Ax^{71d}, Ax¹⁶,



Ax: Abruptex

From Mohr, 1932, Proc. Intern. Congr. Genet., 6th, Vol. 1: 190-212.

and Ax^{E2} (negative complementation). The lethality associated with negative complementation is suppressed by 23 lethal Notch alleles as well as by alleles of *Dl* and *mam* (Xu, Rebay, Fleming, Scottgale, and Artavanis-Tsakonas). When heterozygous with *N* mutants, phenotypes of Ax and *N* tend toward normal, but there is temperature sensitivity for suppression of wing nicks (Foster, 1975; Portin, 1975). Ax⁹ complements every recessive visible on the Notch map at 18° to 29° (Portin, 1977, Hereditas, 87: 77-84); with Ax^{59b} and Ax^{59d}, it is semilethal. Negative complementation is eliminated by *Dp(1;2)51b* and results in a strong Ax phenotype (Portin, 1975). The fate map for negatively complementing heteroallelic Ax⁹/Ax^{E2} suggests a focus of lethality in tissue close to hypodermal sites of central thoracic structures; in surviving gynandromorphs, negative complementation for morphological defects is autonomous (Portin, 1977, Genetics, 86: 309-19).

molecular biology: Mutation results in an amino acid change from aspartic acid to valine at residue 948 in EGF repeat #24; GAC → GTC (Kelley *et al.*, 1987).

Ax¹⁶

phenotype: Homozygotes resemble Ax¹. Ax¹⁶ is less fertile than alleles Ax^{E2}, Ax^{71d}, and Ax⁹ (Portin, 1975), and temperature sensitive for the bristle and wing effects of Ax (Foster, 1975). In heterozygotes with Notch, Ax is expressed and the Notch wing effect is enhanced (Foster, 1975; Portin, 1975). At 29°, heterozygotes with *N* are lethal. In Ax¹⁶/*N*²⁶⁴⁻⁴⁰ heterozygotes, the TSP for lethality is in the second instar, and for Ax-morphological effects, it is in the third instar (Foster, 1973, 1975). In heterozygotes with recessive visibles at Notch, all are complementary at 18° and 25°; at 29°, there are mild indications of noncomplementarity with *nd* and *nd*² (Portin, 1977). Heteroalleles Ax¹⁶/Ax^{E2} and Ax¹⁶/Ax^{71d} are viable (Foster, 1975; Portin, 1975); Ax¹⁶/Ax^{E1} is inviable (negative heterosis) and heterozygotes with Ax⁹ and Ax¹ are lethal (negative complementation) (Foster, 1975; Portin, 1975), but *Dp(1;2)51b* restores viability (Portin, 1977). Heterozygotes with the

lethal alleles Ax^{59b} and Ax^{59d} are lethal and mostly inviable upon the addition of $Dp(1;2)51b$ (Portin, 1975, 1977).

cytology: Salivary chromosomes are normal (Foster, 1975).

molecular biology: Mutation involves a change from glycine to alanine in residue 1174 in EGF-like repeat #29; GGA → AGA (Kelley *et al.*, 1987).

other information: On the genetic map of Notch, probably between N^{264-40} and N^{Co} based on the failure to obtain recombinants between Ax^{16} and Ax^9 (Foster, 1975).

Ax^{59b}

phenotype: Homozygotes and hemizygotes semilethal at 22°; lethality approximates 100% at 25°. Lethal in heterozygotes with N mutants but viable and fertile with recessive visibles at Notch. $Ax^{59b}/Ax^{59b}; Dp(1;2)51b/+$ are poorly viable and infertile, and mutant phenotype is enhanced. $Ax^{59b}/+; Dp(1;2)51b/+$ females have diminished mutant expression compared to $Ax^{59b}/+$ females which in turn are similar to males $Ax/Y; Dp(1;2)51b/+$. In heterozygotes with spl at 25°, the eye is reduced in size but is larger than in spl/spl , and eye roughness varies from very mild to undetectable. The report by Welshons that Ax^{59b}/spl did not express the split phenotype was an error caused by uncontrolled temperature variation. In *cis* heterozygotes, $spl Ax^{59b}/++$, expression of split is enhanced compared to Ax^{59b}/spl ; the eyes are rough and reduced in size. No such enhancement is seen when fa^g is coupled to Ax^{59b} , and in Ax^{59b}/fa^g , the expression of the recessive is very mild and frequently nonpenetrant (Welshons, 1971). Ax^{59b} is semilethal with Ax^9 and lethal with alleles Ax^{16} , Ax^{E2} , Ax^{71d} , Ax^1 , and the addition of $Dp(1;2)51b$ to heterozygotes of Ax^{59b} with Ax^9 and Ax^1 restores viability (Portin, 1975). The temperature sensitivity of the Ax^{59b} phenotype is strongest at 25°; mutant expression decreases at both 18° and 29°, with the least mutant expression at 29° (Portin, 1981, *Hereditas* 94: 93-98). At 18°, there is complementarity with all recessive visibles at Notch and strong Ax expression in every case except when heterozygous with fa^g . At 29°, all heterozygotes are noncomplementary with the exception of nd ; Ax expression is diminished. At 29°, homozygotes or hemizygotes with $Dp(1;2)51b$ are more viable than at 18° or 25° (Portin, 1977). At 25°, wing-vein interruption and bristle loss increases with an increased dose of the mutant gene (Portin, 1981, *Hereditas* 95: 247-51). Somatic crossing over yields twin spots on cuticular surface of flies, indicating that Ax^{59b} is not a primary cell lethal (Portin, 1980).

cytology: Salivary chromosomes normal (Welshons).

molecular biology: Cysteine replaced by glycine at residue 972 in EGF repeat #24; TGC → GGC (Kelley *et al.*, 1987). Some Ax^{59b} strains have acquired an eleven-base-pair deletion leading to chain termination downstream of the substitution at residue 972, which is independent of the Ax^{59b} lesion. Such double mutants show a typical N phenotype (Kelley *et al.*, 1987).

other information: Ax^{59b} placed on the map of the Notch locus between spl and nd^2 ; Ax^{59d} placed between spl and N^{Co} (Welshons).

Ax^{71d}

phenotype: Homozygous, viable, phenotype like Ax^1 . Viable with alleles Ax^{E2} and Ax^{16} , and lethal with Ax^1 and Ax^9 (negative complementation), but viability restored by $Dp(1;2)51b$. There is no obvious effect on the Notch phenotype in heterozygotes with N^8 or N^{55el1} , but Ax phenotype is expressed. In heterozygotes with Ax^{E2} , the mutant phenotype is weakly expressed; heterozygotes with lethal alleles Ax^{59b} and Ax^{59d} are lethal. Ax^{71d} is complementary with recessive alleles at Notch at 18°, 25°, and 29°. The mutant expression of Ax tends to increase with increasing temperature except that Ax^{71d}/nd^2 at 29° has no Ax expression.

molecular biology: Mutation involves a change from serine to isoleucine at residue 1088 in EGF repeat #27; AGT → ATT (Kelley *et al.*, 1987).

Ax^{75c}

phenotype: Recessive lethal like alleles Ax^{59b} and Ax^{59d} . $Ax^{75c}/+$ is temperature sensitive for pleiotropic effects; the variation in mutant expression with temperatures of 18°, 25°, and 29° resembles that of Ax^{59b} and Ax^{59d} with some variation in detail (Portin, 1981, *Hereditas* 94: 93-98). The Ax mutant phenotype increases with increasing dose of the allele (Portin, 1981, *Hereditas* 95: 247-51).

Ax^{E1}

phenotype: $Ax^{E1}/+$ females at 20.5° have gaps in wing veins and a reduction in number of ocellar and postvertical bristles. Semilethal as hemizygote or homozygote. Heterozygotes Ax^{E1}/Ax^9 are viable and phenotypically intermediate: Ax^{E1}/Ax^{E2} and Ax^{E1}/Ax^{16} are inviable (negative heterosis). Ax^{E1} is inviable with most N mutants, but heterozygotes with $N^{264-103}$ (a temperature-sensitive mutant) survive at 22° but not at 29°.

cytology: Salivary chromosomes are normal.

other information: On the map of Notch to the right of fa^{no} and probably close to spl based on failure to obtain recombinants between Ax^{E1} and spl .

Ax^{E2}

phenotype: Homozygous viable, phenotype like Ax^1 . Temperature sensitive for morphological phenotypes (Foster, 1975) but stable for viability (Portin and Sirén, 1976). Viable in heterozygotes with N ; Notch-wing phenotype is enhanced. At 18° and 25°, complementary in heterozygotes with recessive alleles at Notch; at 29°, spl and nd^2 are weakly expressed (Portin, 1977, *Hereditas* 87: 77-94). Heterozygotes $fa^{no} spl Ax^{E2}/+++$ are like spl/spl with suppression of wing-vein gaps; $fa^{no}++/+ spl Ax^{E2}$ and $+spl Ax^{E2}/+++$ show mild expression of spl (Foster, 1975). Ax^{E2} is viable with alleles Ax^{71d} and Ax^{16} , lethal with lethal alleles Ax^{59b} and Ax^{59d} and with Ax^{E1} , Ax^9 , and Ax^1 (Foster, 1975; Portin, 1975), and the lethality with Ax^1 is more pronounced at 29° (Portin and Sirén). In Ax^{E2}/Ax^1 , the TSP for lethality is monophasic from the end of the third instar to early pupa (Portin and Sirén, 1976). In Ax^{E2}/Ax^9 , the focus of lethality is close to hypodermal sites of ventral thoracic structures, and in surviving gynandromorphs, the negative interaction between alleles is autonomous (Portin, 1977, *Genetics* 86: 309-19).

cytology: Salivary chromosomes are normal (Foster, 1975).

molecular biology: Histidine replaced by tyrosine at residue 1167 in EGF-like repeat #29; CAT → TAT (Hartley, Xu, and Artavanis-Tsakonas, 1987, EMBO J. 6: 3407-17; Kelley *et al.*, 1987).

other information: Ax^{E2} is placed on the genetic map of Notch close to and to the right of *spl* (Foster).

Ax^{J14}

phenotype: Male lethal, mutant phenotype similar to Ax^1 . Lethality is covered by $Dp(1;2)51b$ and $Dp(1;2)w^{64d}$, and males with the duplication show the Ax phenotype. When lethality is covered by w^+Y , males have normal wing venation but lack ocellar bristles. In heterozygotes with fa^{12} , females survive exhibiting a strong Ax phenotype and rough eyes.

cytology: Salivary chromosomes normal.

Ax^S (A. Schalet)

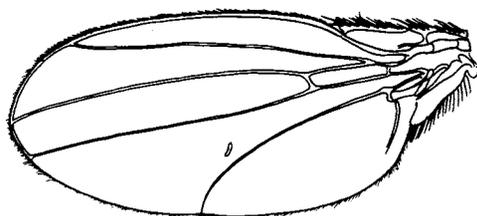
phenotype: Male and female homozygotes lethal. $Ax^S/+$ males and females show sparse thoracic hairs. Ax^S/Ax similar to Ax/Ax , viability strongly reduced. Ax^S/N^S is lethal. Not suppressed by $su(Hw)^2$.

molecular biology: No detectable change in restriction map (Kidd *et al.*, 1983).

Ax^{tsl}

phenotype: Ax phenotype 100% penetrant in heterozygotes at 18° and 29°; homozygous lethal at 29° and semilethal at 18°. Surviving homozygotes have a stronger Ax expression than in heterozygotes. Lethal with N mutants at 29° and semilethal with Ax^{59d} . Ax in phenotype and complementary with recessive visibles at Notch at 18° and noncomplementary at 29°.

other information: On the genetic map of the Notch locus to the right of *spl*.



Co: Confluens

Edith M. Wallace, unpublished.

Co: Confluens (W.J. Welshons)

location: 1-3.0 [distal to N ; not separated by recombination, but separated by $T(X;Y)$ breakpoints (Merriam and colleagues)].

origin: Recovered among progeny of cold-treated fly.

discoverer: Gottschewski 34c.

references: 1935, DIS 4: 7, 14, 16.

1937, Z. Indukt. Abstamm. Vererbungsl. 73: 131-42.

1937, DIS 8: 12.

phenotype: Veins irregularly thickened, especially toward tips, which are usually deltas and fused broadly to marginal vein. Stronger expression in males than in females. Co/N^8 wild type except for slightly thicker L3 vein. Co/Ax like $Ax/+$. RK1A.

cytology: Associated with a tandem duplication, $Dp(1;1)Co = Dp(1;1)3C4-5;3D6-E1$ (Schultz, 1941, DIS 14: 54-55). Result of duplication of 3C7, deficiency for

which gives Notch (Morgan, Schultz, and Curry, 1941, Year Book - Carnegie Inst. Washington 40: 283). Relation to N illustrated by three $T(1;Y)$'s with breakpoints in 3C: $T(1;Y)D7$, $T(1;Y)R38$, and $T(1;Y)W17$. The segmental duplication formed from $T(1;Y)D7$ and $T(1;Y)R38$ is Confluens in phenotype; the corresponding deficiency is normal. The segmental deficiency formed from $T(1;Y)R38$ and $T(1;Y)W17$ is Notch in phenotype and the corresponding duplication is normal. In addition to $T(1;Y)R38$, two other translocations appear to separate Co and N : $T(1;Y)R30$ and $T(1;Y)J100$ (Merriam and colleagues).

other information: Reversion to wild type occurs in Co/Co by unequal crossing over.

fa: facet (W.J. Welshons)

location: 1-3.0.

phenotype: Facet mutants affect the texture of the eye and in some cases cause slight to moderate wing nicks. Until now some recessive mutations with wing nicking but with normal eye texture have been designated as alleles of *fa* based upon their not being complemented by N mutants; in this treatment their designations have been changed to *nd*: notchoid, since they fail to complement *nd* mutants and like *nd* alleles, they complement *fa* alleles. All *fa* alleles complement *spl*, another eye-texture mutant in the N locus.

alleles: Two general phenotypes: the facet phenotype has rough eyes owing to irregularities in size, shape and arrangement of ommatidia; eye color is uniform and wild type. The glossy phenotype also displays irregular facets, but the eye surface is smooth and pigment distribution may be uneven. Most of the alleles tabulated below are described in separate entries to follow.

allele	origin	discoverer	synonym	ref ^α	comments ^β
fa^1	spont	Bridges, 14b		7, 10, 13, 14	<i>opus</i> at -11.9 to -11.2; weak allele; temperature sensitive; nicked wings; not dosage compensated
fa^3	spont ^γ	Welshons		7, 10, 13	<i>springer</i> at -11.9 to -11.2; intermediate allele; not temperature sensitive; dosage compensated
* fa^{do-vg}	spont			4, 6	wild type as hemi- and homozygote; <i>fa</i> with <i>fa hopper</i> at -15.0 to -14.2
$fa^{Ds\delta}$	spont	Green		10	<i>flea</i> at -13.8 to -13.0; strong allele; not dosage compensated
fa^{fx}	3 HTdR	Kaplan, Hays	<i>fx</i>	2, 7, 8, 10, 13	<i>flea</i> at -10.3 to -9.4; strong allele; temperature sensitive; dosage compen-
fa^g	spont	Pratt, 1962		7, 10, 13, 17, 18	<i>flea</i> at -10.3 to -9.4; strong allele; temperature sensitive; dosage compen-

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allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>fa</i> ⁹⁵⁸	N-mustard	Fahmy	<i>swb fa</i> ^{sw}	3, 7, 10, 12, 13	sated "flea" at -11.9 to -11.2; strong allele; wings nicked; dosage compensated
<i>fa</i> ⁹⁶²	X ray	Ives	<i>fa</i> ⁸² <i>swb</i> ^{62b}	7, 10, 12, 13	<i>flea</i> at -11.9 to -11.2; strong allele; wings nicked; dosage compensated
<i>fa</i> ^{988ε}	spont	Kidd			
<i>*fa</i> ¹		Muller, 19h		15, 16	
<i>fa</i> ¹²	X ray	Lefevre	<i>fa</i> ^{IN118}	11	
<i>fa</i> ⁿ			<i>nd</i> ⁰	5	see <i>nd</i>
<i>fa</i> ^{no}			<i>nd</i> ³	1, 9	see <i>nd</i>
<i>fa</i> ^{no69}			<i>nd</i> ⁴		see <i>nd</i>
<i>fa</i> ^{swb}	X ray	Lefevre	<i>swb</i> ^{71b}	9, 12, 19	0.8 kb deletion in -28.4 to -27.1
<i>fa</i> ^{swbBGζ}				9, 10	<i>flea</i> at -11.9 to -11.2; wings nicked

^α 1 = Bauer, 1943, Z. Indukt. Abstamm. Vererbungsl. 81: 374-90; 2 = Craymer, 1980, DIS 55: 200; 3 = Fahmy, 1958, DIS 34: 49; 4 = Gardner, 1942, Univ. Calif. Publ. Zool. 49: 85-102; 5 = Glass, 1933, J. Genet. 27: 233-41; 6 = Goldschmidt, 1935, Biol. Zentralbl. 55: 535-54; 7 = Grimwade, Muskavitch, Welshons, Yedvobnick, and Artavanis-Tsakonas, 1985, Dev. Biol. 107: 503-19; 8 = Kaplan and Hayes, 1957, DIS 42: 38; 9 = Keppy and Welshons, 1977, Genetics 85: 497-506; 10 = Kidd and Young, 1986, Nature 323: 89-91; 11 = Lefevre, 1974, DIS 51: 22; 12 = Lefevre and Kelley, 1972, DIS 48: 146-47; 13 = Markopoulou, Welshons, and Artavanis-Tsakonas, 1989, Genetics 122: 417-28; 14 = Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. 237: 76; 15 = Muller, 1935, DIS 3: 30; 16 = Muller and Altenberg, 1921, Anat. Rec. 20: 213; 17 = Welshons, 1965, Science 150: 1122-29; 18 = Welshons, 1974, Genetics 76: 775-94; 19 = Welshons and Keppy, 1975, Genetics 80: 143-55.

^β Insertion of copia-like elements designated *flea*, *hopper*, *springer*, and *opus* into restriction fragments placed with respect to a zero point defined as the first *EcoRI* site proximal to the left breakpoint of *In(1)N*^{76b8}. "flea" represents a derivative of *flea* in which 3 kb are replaced with 1.5 kb of foreign sequence (Kidd and Young, 1986).

^γ Occurred in *nd*-bearing X.
^δ *Ds* = *Drosophila simulans*.

^ε Phenotype like *fa*⁸. Insertion in *N* intervening sequence.
^ζ *fa*^{swbBG} is not *fa*^{swb}; it does not carry the *fa*^{swb} deletion; it is possibly a mislabeled *fa*⁸⁶², since it contains the same insert at exactly the same nucleotide.

molecular biology: With the exception of *fa*^{swb}, the *fa* alleles that have been studied have *copia*-like inserts in a 0.1 kb region of the second intron of the Notch gene (in *fa* of *Drosophila simulans* the insert is in the 3' end of the first intron). Five glossy-like alleles (*fa*^{fx}, *fa*⁸, *fa*⁸⁵⁸, *fa*⁸⁶², *fa*^{swbBG}) have the same element inserted, whereas each of three facet-like alleles (*fa*¹, *fa*³, *fa*^{Ds}) has a different inserted element (see allele table). In every case the orientation of the inserted element is such that they are transcribed in the opposite direction to that of Notch.

***fa*¹**

phenotype: Eyes of all males moderately rough owing to irregularity in size, shape, and arrangement of facets. Not dosage compensated; eyes of females less rough than those of males with about 10% overlap of wild type. Eye roughness of the females varies from nearly normal at 18° to marked at 29°; pupal stage temperature sensitive

(Shellenbarger and Mohler, 1975, Genetics 81: 143-62). Eye abnormality caused by overgrowth of secondary pigment cells, which compresses cones and causes overlying corneal facets to bulge (Waddington and Pilkington, 1942, DIS 16: 70). Wings have apical nicks in 0.25% of males and 0-5% of females. *N/fa*¹ has rough eyes of *fa*¹ as well as a Notch phenotype

***fa*³**

phenotype: Eyes equally rough in both sexes; wings not notched. Eyes rougher than in *fa* males but not glossy as in *fa*⁸; heterozygotes *fa*^{3/fa}⁸ are rough, not glossy (Welshons).

****fa*^{do-vg}: facet-dominigene for vestigial**

phenotype: By itself, it is wild type. *fa*^{do-vg/fa}¹ shows rough eye character of *fa*¹. *fa*^{do-vg/fa}^{do-vg}; *vg/+* produces some wing notching. Presumed by Goldschmidt to enhance dominance of *vg* and thus termed a "dominigene". RK3.

***fa*^{fx}: facet-frostex**

phenotype: Strong echinus-like eyes, darkening with age with glistening frosted appearance. Homozygous females sterile, but sterility may be separable from *fa*^{fx} (Kaplan and Hayes, 1967, DIS 42: 38).

***fa*^g: facet-glossy**

phenotype: Eyes have facets more irregular than *fa*, but surface is smoothed, giving a glossy effect. Equal mutant expression in both sexes. Pigment distribution may be uneven, contributing to an impression of altered eye color. No wing effect. Eyes of *fa*^{8/fa}¹ intermediate between the two homozygotes. Complementary with *spl*, *fa*^{no}, *nd*, and *nd*² (Welshons, 1965, Science 150: 1122-29). RK1.

***fa*⁹⁵⁸**

phenotype: Large, rough eye with semiglazed surface and irregular pigment distribution causing a patchy red color. About 2/3 flies have incisions of the inner wing margin. Viable and fertile as a male, reduced fertility in females (Fahmy, 1958, DIS 34: 49).

***fa*⁹⁶²**

phenotype: Like *fa*⁸ and cannot be distinguished from it. In heterozygotes with *N* mutants and in *fa*^{862/fa}⁸, the *fa*⁸ phenotype is exhibited; *fa*^{862/fa}¹ has a *fa*¹ phenotype, and *fa*^{862/spl} is wild type.

****fa*^l: facet-lethal**

phenotype: *fa*^{l/fa}¹ resembles *fa*^{1/fa}¹; not notched. Homozygous lethal. RK2.

***fa*^{l2}**

phenotype: A male-lethal allele of Notch. Females nearly wild type but show occasional slight traces of Notch. Full complementation with *spl* but interacts with *fa*⁸ showing rough irregular eyes.

***fa*^{swb}: facet-strawberry**

phenotype: In males, eyes are rough with a variable tendency to be glossy; with *fa*⁸ and *fa*⁸⁶², eyes are very rough, but mutant condition is not as extreme as that found in homozygous glossy-eyed mutants. In heterozygotes with *fa*¹, eyes are slightly rough, overlapping wild type; with *spl*, the eyes are wild type.

The *fa*^{swb} allele, like *fa*¹, is not dosage compensated,

and the mutant condition is poorly expressed in females. fa^{swb}/fa^{no} has slight deltas at junction of longitudinal veins with marginal veins; fa^{swb}

complements nd and nd^2 ; and in heterozygotes of fa^{swb} , N^{55el1} and N^{264-40} , the eyes are glossy and the Notch phenotype is enhanced, resulting in reduced viability and fertility; with the temperature-sensitive N^{60g11} , heterozygotes are less mutant, viable, and fertile. In double mutants, $fa^{swb}fa^g$, the males have fa^g -like eyes; and wing veins are thickened and delta like at tips; they resemble fa^{no} males except that wings are seldom notched. The wing-venation effect is less extreme in homozygous females (Welshons and Keppy, 1975, Genetics 80: 143-55; Keppy and Welshons, 1977, Genetics 85: 497-506).

cytology: Deficiency for interband between 3C6 and 7, causing these bands to fuse (Keppy and Welshons, 1977).

molecular biology: Caused by a deficiency of 800 base pairs in the restriction fragment —26.2 to —25.0 (Grimwade, Muskavitch, Welshons, Yedvobnick, and Artavanis-Tsakonas, 1985, Dev. Biol. 107: 503-19; this is 67 base pairs upstream from the start of transcription of Notch. Deletes the 3' end of the locus immediately to the left of N (Kidd, Kelley, and Young, 1986, Mol. Cell. Biol. 6: 3094-3108).

other information: Removal of the region of 3C2-5 from the vicinity of fa^{swb} , either by deletion or inversion results in suppression of fu^{swb} ; also affected by spontaneously arising, cis-acting enhancer [$e(fa^{swb})$] and suppressor [$su(fa^{swb})$] mutations, both of which map to the 3C2-5 region. (Welshons and Welshons, 1986).

l(1)N: lethal (1) Notch (W.J. Welshons)

location: 1-3.0.

phenotype: There are four phenotypic varieties of $l(1)N$ alleles: (1) Those that are lethal with N and wild type with the recessive visibles [see $l(1)N^1$]; (2) Those that are lethal with N but not wild type with the recessive visibles [see $l(1)N^2$, $l(1)N^3$]; (3) Alleles whose heterozygotes with N^+ have a phenotype not recognized as Notch [see $l(1)N^B$], or (4) Alleles that are temperature sensitive for lethality and do not express a Notch phenotype in heterozygotes with N^+ [see $l(1)N^{ts}$]. The embryological defects in $l(1)N^1$ are related to those in N ; the development in $l(1)N^B$ is sufficiently normal to escape embryonic lethality (Poulson, 1967, 1968).

alleles:

allele	origin	discoverer	synonym	ref ^α
$l(1)N^1$	spont	Welshons	l^N	3, 5, 6, 8, 10
$l(1)N^2$	γ ray	Abrahamson	l^{N2}	3, 5, 6, 8, 10
$l(1)N^3$	γ ray	Abrahamson	l^{N3}	3, 5, 6, 8, 10
$l(1)N^{27-3}$	HD	Gergen		2
$l(1)N^{27-3rv}β$	HD	Kelley		2
$l(1)N^{32/25/B}β$	HD	Shalet, Eeken		2
$l(1)N^{37-10}β$	HD	Gergen		2
$l(1)N^{42-2}β$	HD	Gergen		2
$l(1)N^{57-4}β$	HD	Gergen		2
$l(1)N^{69e}$	EMS	Shellenbarger		7, 8
$l(1)N^B$	X ray	Abrahamson		1, 3, 5, 8, 10
$l(1)N^{ts1}γ$	EMS	Shellenbarger		4, 7-9
$l(1)N^{ts2}$	EMS	Shellenbarger		4, 7, 8

^α 1 = Arnheim, 1967, Genetics 56: 253-63; 2 = Kelley, Kidd, Berg, and Young, 1987, Mol. Cell Biol. 7: 1545-48; 3 = Kidd, Lockett, and Young, 1983, Cell 34: 421-33; 4 = Lehmann, Jiménez, Dietrich, and Campos-Ortega, 1983, Wilhelm Roux's Arch. Dev. Biol. 192: 62-74; 5 = Poulson, 1967, DIS 42: 81; 6 = Poulson, 1968,

Proc. Int. Congr. Genet., 12th, 1: 143; 7 = Shellenbarger, 1972, DIS 48: 55; 8 = Shellenbarger and Mohler, 1975, Genetics 81: 143-62; 9 = Shellenbarger and Mohler, 1978, Dev. Biol. 62: 432-46; 10 = Welshons, 1965, Science 150: 1122-29.

β P-element insertion at 5' end of Notch transcription unit within 0.1 kb of transcription start site. $l(1)N/+$ = wild type; $l(1)N/N$ and $l(1)N/Y$ = embryonic lethal. Most heterozygous combinations with recessive visibles not tested.

γ Temperature shifts of mature larvae result in most ommatidial cells becoming photoreceptors (Cagan and Ready, 1989, Genes Dev. 3: 1099-1112).

l(1)N¹

phenotype: $l(1)N^1/+$ females are wild type; $l(1)N^1/N$ females and $l(1)N^1/Y$ males are lethal; $l(1)N^1/Y;Dp(1;2)51b$ males are *Co*-like. Heterozygotes with recessive visibles at Notch are wild type. Developmental defects in $l(1)N^1/Y$ males are more limited than in N/Y males and the defects are confined to the anterior ectoderm (Poulson, 1967; 1968). Like N mutants, $l(1)N^1$ mutants are defective as embryos (Shellenbarger and Mohler, 1975, 1978).

molecular biology: $l(1)N^1$ is associated with a 8.5 kb insertion between coordinates -24.2 and -23.0 about 2.3 kb to the right of the insertion associated with N^{55el1} (Kidd *et al.*, 1983).

l(1)N²

phenotype: $l(1)N^2/+$ females are wild type; $l(1)N^2/N$ females and $l(1)N^2/Y$ males are lethal. Heterozygotes with fa and fa^g are *fa*-like; with nd , they have *nd*-like wings and small eyes; with nd^3 , they are viable, fertile and *nd^3*-like. Developmental defects in $l(1)N^2/Y$ males and time of lethal effect same as in $l(1)N^1/Y$. Some $l(1)N^2/l(1)N^{ts1}$ females survive to late pupal stage (Shellenbarger and Mohler, 1975).

l(1)N³

phenotype: Same as $l(1)N^2$.

l(1)N^B

phenotype: $l(1)N^B$ females have small eyes, fewer mesonotal bristles, and, sometimes, bald areas on the thorax (Welshons, 1965). The dominant bristle effect is more extreme in $l(1)N^B/Y;Dp(1;2)51b$ males than in $l(1)N^B/+$ females. Heterozygotes with fa have *fa*-like eyes and, frequently, nicked wings; with nd^3 , they show notched wings and thickened veins; with nd^3 , they are viable, fertile, and *fa*-like. $l(1)N^B/Y$ males die during early larval life (Poulson, 1967). $l(1)N^B/l(1)N^{ts1}$ females die before pupation (Shellenbarger and Mohler, 1975). Bristle effect autonomous in $l(1)N^B$ cells; homozygous mutant cells survive in mosaics (Arnheim, 1967).

l(1)N^{69e}

phenotype: $l(1)N^{69e}/l(1)N^{69e}$ and $l(1)N^{69e}/Df(1)N-8$ females are lethal at 18° and 29°; $l(1)N^{69e}/+$ heterozygotes are almost always wild type. $l(1)N^{69e}$ homozygotes die before pupation, but $l(1)N^{69e}/l(1)N^{ts1}$ heterozygotes survive until the pupal stage.

l(1)N^{ts1}

phenotype: $l(1)N^{ts1}/+$ females are wild type at 18° and 29°, while $l(1)N^{ts1}/Df(1)N-8$ females are lethal at 29°, but a few escapers are found at 18°. $l(1)N^{ts1}$ homozygotes are viable at 18°, but lethal at 29°. If homo- and hemizygotes kept at 18°C until eclosion are transferred to 29°C and kept at this temperature for six days, they gra-

dually become flightless and show gross histological changes in the flight muscles (Vikki and Portin, 1987, William Roux's Arch. Dev. Biol. 196: 12-15). Heterozygotes show recessive visible defects at 18°, but not at 29°. $l(1)N^{ts1}/l(1)N^2$ and $l(1)N^{ts1}/l(1)N^3$ females survive until the late pupal stage at 29°. When heat pulses are given to pupae prior to sensillum-precursor-cell-determination, extra sensilla are produced; when given after sensillum-precursor-cell determination, the precursor cells form neurons only, not accessory cells (Hartenstein and Posakony, 1990, Dev. Biol. 142: 13-30).

l(1)N^{ts2}

phenotype: Similar to $l(1)N^{ts1}$ except for occasional survival of homozygotes to the pupal stage at 29° and weaker expression of recessive visible defects in heterozygotes at this temperature.

N: Notch (W.J. Welshons)

location: 1-3.0.

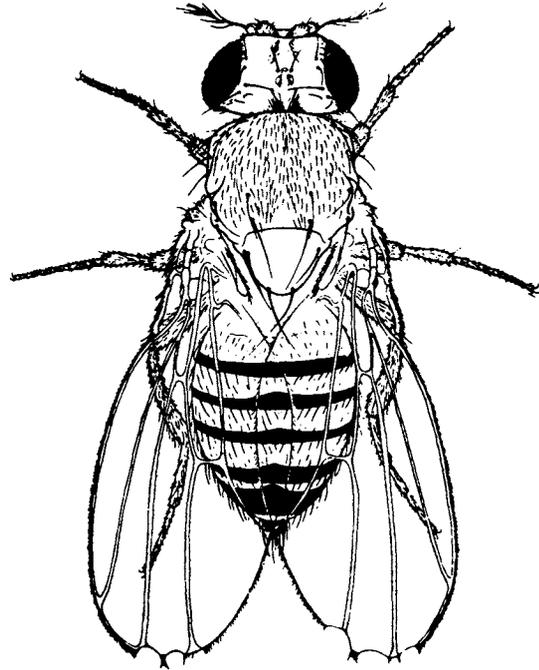
phenotype: Mutant alleles are characterized by the following types of expression: Wings of heterozygotes incised at tips and often along edges; veins L3 and L5 thickened; thoracic microchaetae crowded and irregularly distributed (Mohr, 1919, Genetics 4: 275-82; 1923, Z. Indukt. Abstamm. Vererbungsl. 32: 108-232). Males and homozygous females are lethal. In some *N* mutants, the phenotype is mild and varies in one or more of its typical features, but such *N*'s can usually be identified by phenotypes expressed when heterozygous with recessive visible eye and wing mutants that also occur at Notch.

Females N/N^+ are Notch; females $N/N^+;Dp(1;2)51b$ (representing a duplication for the Notch locus) are wild type. In the hemizygous male, N/Y is lethal, whereas $N/Y;Dp(1;2)51b$ is viable and phenotypically normal; the wild phenotype is dependent upon the presence of the normal dosage of 3C6-7 for each sex. An extra dose of 3C6-7 [as in $Dp(1;2)51b$ or $Dp(1;1)Co$] causes the expression of the dominant phenotype Confluens (*Co*); thus $N^+/N^+;Dp(1;2)51b$ females and $N^+/Y;Dp(1;2)51b$ males are *Co*-like (Welshons, 1965). Deficiency mapping places *Co* to the left of *N* (Merriam).

Homozygotes and hemizygotes for all *N* mutants suffer the same embryological defects. In developing embryos, the pattern of differentiation of anterior and ventral embryonic ectoderm is aberrant; both presumptive hypoderm and presumptive neuroblasts develop as neuroblasts, resulting in embryos with a hypertrophied central nervous system lacking ventral and ventral-lateral hypoderm [Poulson, 1939, DIS 12: 64-65; 1940, J. Exp. Zool. 83: 271-325; 1950, Biology of Drosophila (M. Demerec, ed.). Wiley, New York, pp.168-274; 1967, DIS 42: 81; Wright, 1970, Adv. Genet. 15: 305-15]. Sensillum differentiation in peripheral nervous system of embryos also abnormal (Hartenstein and Campos-Ortega, 1986, Roux's Arch. Dev. Biol. 195: 210-21). In mosaic embryos (N/N^+ and $N/0$ cells), the $N/0$ cells never give rise to hypoderm within the neurogenic region (Hoppe and Greenspan, 1986, Cell 46: 773-83). However, single $N/0$ cells transplanted to N^+ recipient embryos can give rise to hypoderm (Technau and Campos-Ortega, 1987, Proc. Nat. Acad. Sci. USA 84: 4500-04).

alleles: Notch mutants and rearrangements (other than deficiencies) are described in the following tables, the first listing the extant alleles and the second the lost alleles.

(Alleles that are also given a separate description are superscripted "#"). Notch deficiencies are listed only as chromosome rearrangements.



N⁸

Mohr, 1924, Z. Induktive Abstammungs-Verebungslehre 32: 118.

N^{55e11}

phenotype: A weak Notch. Deltas on wing veins are most reliable character for classification. Lethal when heterozygous with nd^3 , N^{60g11} , and N^{Co} . In homozygotes and hemizygotes hyperplasia of central nervous system extreme; embryonic peripheral nervous system abnormal with sensilla undifferentiated (Hartenstein and Campos-Ortega, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 210-21).

molecular biology: Lesion in Notch caused by a 3.5 kb insertion in the vicinity of coordinate -26, "+" values to the right, "-" values to the left (Kidd *et al.*, 1983).

N^{60g11}

phenotype: Wings seldom notched; veins thickened; deltas at tips. $N^{60g11}/+$ heterozygotes have normal eyes at 29° and a disrupted facet arrangement at 21°. With increasing temperature, rough eye phenotype diminishes and Notch mutant characteristics are expressed. TSP for disrupted facets is in the third instar. $N^{60g11}/N^{60g11}Dp(1;2)51b7$ females are viable at 29°; survival sharply decreased at 20-23°; TSP for lethality in middle of embryonic stage. N^{60g11}/fa flies have eyes like *fa*. Semilethal with nd^3 . Viability poor with *nd*.

N^{64d6}

phenotype: Typical Notch. $N^{64d6}spl$ flies cannot be distinguished from $N^{64d6}spl^+$ flies. When *spl* is coupled to N^{64d6} , *spl* is not enhanced by *E(spl)*.

Table I

allele	origin	discoverer	ref ^α	comments
N²			18, 20	
N^{3b22}	EMS	Wieschaus	29	
N²²				like <i>N^{j24}</i>
N^{24/46A/1 β}	HD	Schalet, Ecken	23	
N^{30-4 β}	HD	Gergen		
N^{30-4rv β}	HD	Kelley		wild type revertant
N^{50k11}	X ray	Lefevre, 50k	26, 27	<i>T(1;3)IE3-4;3C6-7;3C8-9;89A</i> 3C7,3C8 missing
N^{55e11 #}	spont	Mohler, 55e	2, 3, 18, 20, 25, 29, 34, 37, 44, 48, 50	
N^{60f10}	γ ray	Ives	52	
N^{60g11 #}	γ ray	Ives	13, 14, 44, 53	
N^{60h21 γ}	γ ray	Ives	52	
N^{60j14 γ}	γ ray	Ives	52	
N^{61f19 γ}	γ ray	Ives	53	
N^{61h10 γ}	γ ray	Ives	53	
N^{62b10 γ}	γ ray	Ives	52	
N⁶²ⁱ	radio waves	Mickey, 62i	33	
N^{64d6 #}	X ray	Judd	49	
N^{66h26 #}	spont	Green	3, 16, 24, 50, 51	<i>In(1)3C1-2;3C7-8</i>
N^{67k28}	X ray		28	
N^{68j #}	EMS	Maddern, 68j	19	
N^{68j1 #}	EMS	Shellenbarger	40, 41	
N^{68j2}	EMS	Shellenbarger	40, 41	
N^{68l}	EMS	Shellenbarger	40, 41	
N⁶⁹	NTG	Kaufman	22	
N^{69c #}	EMS	Shellenbarger	40, 41	
N^{69dt}	EMS	Shellenbarger	40, 41	
N^{69d2}	EMS	Shellenbarger	40, 41	
N^{69d3}	EMS	Shellenbarger	40, 41	
N^{69d4}	EMS	Shellenbarger	40, 41	
N^{69d5}	EMS	Shellenbarger	40, 41	
N^{69e1}	EMS	Shellenbarger	40, 41	
N^{69e2}	EMS	Shellenbarger	40, 41	
N^{69e3}	EMS	Shellenbarger	40, 41	
N^{69f1}	EMS	Shellenbarger	40, 41	
N^{76b8 #}	γ ray	Keppy	2, 3, 24, 51	<i>In(1)3C7-9;3C9-10</i>
N^{77c17 #}	X ray	Keppy	2, 3	<i>In(1)1D2-E1;3C7-9</i>
N^{80j9 #}	unstable revertant		17	<i>In(1)3C6-7;3C7-9</i>
N^{81k2}	X ray	Muskavitch	17	
N^{81k3 #}	X ray	Muskavitch	17	
N^{81k4}	X ray	Muskavitch	17	
N^{81k5}	X ray	Muskavitch	17	
N^{81k6 #}	X ray	Muskavitch	3, 17	<i>In(1)3C6-9;13A12-B2</i>
N^{81k8 #}	X ray	Muskavitch	2, 17	
N^{81k9 #}	X ray	Muskavitch	17	<i>T(1;3)3C6-9;3L</i>
N^{81k10 #}	X ray	Muskavitch	17	<i>In(1)3C5-9;20A3-F</i>
N^{81l1 #}	X ray	Muskavitch	17	<i>In(1)3C5-9;20A3-F</i>
N^{81l2}	X ray	Muskavitch	17	
N^{81l3 #}	X ray	Muskavitch	2, 17	
N^{81l5 #}	X ray	Muskavitch	2, 3, 17	<i>In(1)3C5-9;20A3-F</i>
N^{81l7}	X ray	Muskavitch	17	complex
N^{81l8}	X ray	Muskavitch	17	
N^{81l9 #}	X ray	Muskavitch	2, 17	<i>In(1)3C3-D3;20A3-F</i>
N^{264-6 δ}	X ray	Demerec, 33k	4	<i>Tp(3;1)N²⁶⁴⁻⁶</i>
N^{264-10 δ}	X ray	Demerec, 33l	8	<i>T(1;2)</i>
N²⁶⁴⁻¹²	X ray	Demerec, 34a	10, 21	<i>T(1;4)3C6-7;101F</i>
N^{264-40 #}	X ray	Demerec, 37d	2, 25, 29, 36, 37, 44	
N^{264-47 #}	X ray	Demerec, 37f	29, 36, 47	
N^{264-58 ε}	X ray	Demerec, 38d	9	<i>Tp(1;3)3B2-3;3D6-7;80D-F</i>
N^{264-66 #}	X ray	Demerec, 38e	8	<i>T(1;2)3C6-7;41 +</i> <i>T(1;2)7C9-D1;53F</i>
N^{264-84 ε}	X ray	Demerec, 39c	10	<i>In(1)3C6-7;20 A-B</i>
N^{264-103 #}	X ray	Demerec, 40a	13, 52	
N^{264-107 #}	X ray	Demerec, 40a	47, 48	
N^{264-109 #}	X ray	Demerec, 40a	25, 53	
N^{ag}				like <i>N²⁶⁴⁻¹²</i>
N^{BH9 β}	HD	Gergen	23	
N^{Co #}	spont	Welshons	37, 38, 45, 46, 52	
N^{Co69}	NTG	Kaufman	22	
N^{D13 β}	HD	Schalet, Eeken	23	
N^{D16 β}	HD	Schalet, Eeken	23	

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	ref ^α	comments
<i>N^{D30β}</i>	HD	Schalet, Eeken	23	
<i>N^{DTVβ}</i>	HD	Gergen	23	
<i>N^{FE1}</i>	X ray	Campos-Ortega	29	
<i>N^{FE2}</i>	X ray	Campos-Ortega	29	
<i>N^{FX1}</i>	X ray	Campos-Ortega	29	
<i>N^{FX2}</i>	X ray	Campos-Ortega	29	
<i>N^{FX3}</i>	X ray	Campos-Ortega	29	
<i>N^{FX4}</i>	X ray	Campos-Ortega	29	
<i>N^{FX5}</i>	X ray	Campos-Ortega	29	
<i>N^{FX6}</i>	X ray	Campos-Ortega	29	
<i>N^{FX7}</i>	X ray	Campos-Ortega	29	
<i>N^{FX8}</i>	X ray	Campos-Ortega	29	
<i>N^{FX9}</i>	X ray	Campos-Ortega	29	
<i>N^{hdA171 #}</i>	HD	Engels	12, 17	
<i>N^{hdC8 #}</i>	HD	Engels	12, 17	
<i>N^J</i>	NTG	Kaufman	22	
<i>N^{J24 #}</i>	spont	Welshons	46, 52	
<i>N^{M #}</i>	spont	Mischaikow, 56l	7	
<i>N^{Nic #}</i>	X ray	Nicoletti	44, 52	
<i>N^{ntg}</i>	NTG	Kaufman	22	
<i>N^P</i>	P ³²	Bateman, 1950	8	
<i>N^{Plβ}</i>	HD	Gergen	23	
<i>N^{S #}</i>	spont	Schalet	25	inserted DNA element
<i>N^T</i>	NTG	Kaufman	22	
<i>N^W</i>	spont	Williams, 56j	7	
<i>N^{X114}</i>	EMS	Wieschaus	29	
<i>N^{XL86}</i>	EMS	Wieschaus	29	
<i>N^{Y166}</i>	EMS	Wieschaus	29	
<i>tsN^{69c}</i>				see <i>N^{69c}</i>

^α 1 = Aronson, 1958, DIS 32: 67; 2 = Artavanis-Tsakonas, Grimwade, Harrison, Markopoulou, Muskavitch, Schlesinger-Bryant, Wharton, and Yedvobnick, 1984, Dev. Genet. 4: 233-54; 3 = Artavanis-Tsakonas, Muskavitch, and Yedvobnick, 1983, Proc. Nat. Acad. Sci. USA 80: 1977-81; 4 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91; 5 = Barigozzi, 1940, DIS 13: 69; 6 = Barigozzi, 1942, Rev. Biol. (Perugia) 34: 59-72; 7 = Cicak and Oster, 1957, DIS 31: 80; 8 = CP627; 9 = Demerec, 1940, Genetics 25: 618-27; 10 = Demerec, 1941, Proc. Int. Congr. Genet., 7th, pp. 99-103; 11 = Demerec and Sutton, 1940, Proc. Nat. Acad. Sci. USA 26: 532-36; 12 = Engels, 1979, Proc. Nat. Acad. Sci. USA 76: 4011-15; 13 = Foster, 1973, Dev. Biol. 32: 282-96; 14 = Foster and Suzuki, 1970, Proc. Nat. Acad. Sci. USA 67: 738-45; 15 = Gottschewski, 1935, DIS 4: 15, 16; 16 = Green, 1967, Genetics 56: 467-82; 17 = Grimwade, Muskavitch, Welshons, Yedvobnick, and Artavanis-Tsakonas, 1985, Dev. Biol. 107: 503-19; 18 = Hartenstein and Campos-Ortega, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 210-21; 19 = Hayman and Maddern, 1969, DIS 44: 50; 20 = Jiménez and Campos-Ortega, 1982, Wilhelm Roux's Arch. Dev. Biol. 191: 191-201; 21 = Judd, 1955, DIS 29: 126-27; 22 = Kaufman, 1970, DIS 45: 34; 23 = Kelley, Kidd, Berg, and Young, 1987, Mol. Cell Biol. 7: 1545-48; 24 = Keppy and Welshons, 1980, Chromosoma 76: 191-200; 25 = Kidd, Lockett, and Young, 1983, Cell 34: 421-33; 26 = Lefevre, 1951, DIS 25: 71; 27 = Lefevre, 1952, DIS 26: 66; 28 = Lefevre and Green, 1972, Chromosoma 36: 391-412; 29 = Lehmann, Jiménez, Dietrich, and Campos-Ortega, 1983, Wilhelm Roux's Arch. Dev. Biol. 192: 62-74; 30 = Mather, 1942, DIS 16: 49; 31 = Meyer, 1952, DIS 26: 67; 32 = Meyer and Edmondson, 1951, DIS 25: 73; 33 = Mickey, 1963, DIS 38: 29; 34 = Mohler, 1956, DIS 30: 78; 35 = Oliver, 1937, DIS 7: 19; 36 = Poulson, 1939, DIS 12: 64-65; 37 = Poulson, 1967, DIS 42: 81; 38 = Poulson, 1968, Proc. Int. Congr. Genet., 12th, 1: 143; 39 = Ratty, 1954, Genetics 39: 513-28; 40 = Shellenbarger, 1972, DIS 48: 55; 41 = Shellenbarger and Mohler, 1975, Genetics 81: 143-62; 42 = Sliizynska, 1938, Genetics 23: 291-99; 43 = Sutton, 1940, Genetics 25: 534-40; 44 = Thörig, Heinstra, and Scharloo, 1981, Genetics 99: 65-74; 45 = Welshons, 1956, DIS 30: 79; 46 = Welshons, 1958, Cold Spring Harbor Symp. Quant. Biol. 23: 171-76; 47 = Welshons, 1958, Proc. Nat. Acad. Sci. USA 44: 254-58; 48 = Welshons, 1965, Science 150: 1122-29; 49 = Welshons, 1971, Genetics 68: 259-68; 50 = Welshons, 1974, Genetics 76: 775-94; 51 = Welshons and Keppy, 1981, Mol. Gen. Genet. 181: 319-24; 52 = Welshons and Von Halle, 1962, Genetics 47: 743-59; 53 = Welshons, Von Halle, and Scandlyn, 1963, Proc. Int. Congr. Genet. 11th, 1: 1-2.

^β Shows standard *N* phenotype in homo- and heterozygotes; no complementation of *fa* and *fa*^β. All except *N^{D30}* carry *P*-element insertions within 0.1 kb of *N* transcription start site (*N^{D30}* maps 0.3 kb downstream of start site).

^γ *N^{fa}* lethal.
^δ *XYY* males viable, sterile.
^ε *N^{fa}* variegates for *fa*.

Table II

allele	origin	discoverer	ref ^α	phen. ^β	cytology
<i>*N²⁷</i>	spont	Mohr,30l	8		
<i>*N³⁰</i>	spont	Mohr,38b	8		
<i>*N^{34b}</i>	X ray	Oliver,34b	35		<i>T(1;3)N^{34b} (?)</i>
<i>*N^{40j}</i>	spont	Sismanidis,40j	30		
<i>*N⁴⁷ⁱ</i>	UV	Meyer,47i	31		
<i>*N^{51d}</i>	UV	Beyer,51d	31, 32	1	
<i>*N²¹⁸</i>	X ray	Barigozzi	5, 6	1, 2	
<i>*N²⁶⁴⁻⁷</i>	X ray	Demerec,33k	8		<i>In(1)3C6-7;3C8-9;8C5-7</i> <i>3C7-8 missing</i>
<i>*N²⁶⁴⁻⁸</i>	X ray	Demerec,33k	36, 42	3	
<i>*N²⁶⁴⁻⁹</i>	X ray	Demerec,33l	8	4	<i>T(1;2)3C;41</i>
<i>*N²⁶⁴⁻²⁰</i>	X ray	Demerec,34g	8		<i>T(1;4)3C4-5;3C7-8;101F</i> <i>3C5-7 missing</i>
<i>*N²⁶⁴⁻²³</i>	X ray	Demerec,35h	8	5	<i>T(1;2)3C8-9;41A</i>
<i>*N²⁶⁴⁻²⁴</i>	X ray	Demerec,35h	10		<i>T(1;2)3C8-9;40F</i>
<i>*N²⁶⁴⁻²⁹</i>	X ray	Demerec,36d	10	5	<i>T(1;3)3D4-5;80</i>

allele	origin	discoverer	ref ^α	phen. ^β	cytology
*N ²⁶⁴⁻³⁴	X ray	Demerec,37a	10	3	T(1;3)3C3-5;70C2-3
*N ²⁶⁴⁻⁴⁸	X ray	Demerec,37f	10		In(1)1B6-7;1B10-11;3C7-8 1B7-9 missing
*N ²⁶⁴⁻⁵⁰	X ray	Demerec,37k	10	1	Tp(1;2)3C7-9;20C1-F;22A2-3
*N ²⁶⁴⁻⁵²	X ray	Demerec,38a	10	1	In(1)3C3-5;20B2-C1
*N ²⁶⁴⁻⁵³	X ray	Demerec,38d	10		T(1;2)3C6-7;34C7-D1
*N ²⁶⁴⁻⁵⁵	X ray	Demerec,38b	10	1	T(1;3)3D4-5;80F9-81F1
*N ²⁶⁴⁻⁵⁶	X ray	Demerec,38c	8		T(1;3)3D4-5;80
*N ²⁶⁴⁻⁵⁷	X ray	Demerec,38d	10		In(1)3C9-11;20D2-E1
*N ²⁶⁴⁻⁵⁹	X ray	Demerec,38d	10	2	T(1;2)3C8-9;40F
*N ²⁶⁴⁻⁶⁰	X ray	Demerec,38d	8		
*N ²⁶⁴⁻⁶²	X ray	Demerec,38e	10		T(1;2)3C7-8;41A-B
*N ²⁶⁴⁻⁶³	X ray	Demerec,38e	10, 43		Tp(1;1)3C7-9;13C7-8;19F
*N ²⁶⁴⁻⁶⁴	X ray	Demerec,38e	10		T(1;3)3E5-6;80C-F
*N ²⁶⁴⁻⁶⁵	X ray	Demerec,38e	8	1	T(1;3)2B10-16;96C4-5 + T(1;3)3D4-5;81E
*N ²⁶⁴⁻⁶⁹	X ray	Demerec,38k	10		T(1;2)3C7-8;44C4-5
*N ²⁶⁴⁻⁷⁰	X ray	Demerec,38k	43	1, 6	T(1;3)3C4-5;80D-F + T(1;3)6F2-7A1;100B2-3
*N ²⁶⁴⁻⁷¹	X ray	Demerec,38k	10		In(1)3C6-7;20D-F
*N ²⁶⁴⁻⁷⁴	X ray	Demerec,38k	43	1	T(1;2;3)3C10-11;20D-E;40C-D; 92E6-8
*N ²⁶⁴⁻⁸⁰	X ray	Demerec,39d	10		Tp(2;1)3C6-7;36;40 + In(1)11;20
*N ²⁶⁴⁻⁸²	X ray	Demerec,39d	8	1	T(1;2)3C3-4;41A + T(1;2)20A;57
*N ²⁶⁴⁻⁸³	X ray	Demerec,39d	10		T(1;3)3C6-7;12F2-4;79E2-3 + In(3R)81;88
*N ²⁶⁴⁻⁸⁵	X ray	Demerec,39d	9	1	T(1;2;4)3B4-C1;6A2-B1; 60A4-5;101F-102A
*N ²⁶⁴⁻⁸⁶	X ray	Demerec,39i	9, 11, 43	1	T(1;4)3C6-7;3C7-8;3E5-6; 101F
*N ²⁶⁴⁻⁸⁷	X ray	Demerec,39j	43		T(1;2;3)3C7-9;10A2-B1; 45F-46A;59F-60A;97C-D; 100E-F
*N ²⁶⁴⁻⁸⁸	X ray	Demerec,39j	8		
*N ²⁶⁴⁻⁹¹	X ray	Demerec,39g	8		
*N ²⁶⁴⁻⁹⁴	X ray	Demerec,39k	8		
*N ²⁶⁴⁻⁹⁵	X ray	Demerec,39k	8		
*N ²⁶⁴⁻⁹⁷	X ray	Demerec,39k	8		
*N ²⁶⁴⁻¹⁰⁰	X ray	Demerec,39l	9, 43	1	Tp(1;3)3B4-C1;4B4-5;80
*N ²⁶⁴⁻¹⁰²	X ray	Demerec,39l	8		Tp(2;1)3C6-7;50E;56C
*N ²⁶⁴⁻¹⁰⁴	X ray	Demerec,39j	8		T(1;3)3C7-9;87D1-E1 + In(1)1B4-5;18-19
*N ²⁶⁴⁻¹⁰⁸	X ray	Demerec,40a	8		In(1)3C3-5;3E7-8;20A4-5 3C5-3E7 missing
*N ²⁶⁴⁻¹¹²	X ray	Demerec,40b	8		In(1)3C6-7;3F5-6
*N ²⁶⁴⁻¹¹³	X ray	Demerec,40c	43		T(1;4)3C10-D1;101
*N ²⁶⁴⁻¹¹⁶	X ray	Sutton,40e	8		In(1)2C8-10;3C7-9
*N ²⁶⁴⁻¹¹⁹	X ray	Demerec,40i	8		
*N ²⁶⁴⁻¹²¹	X ray	Demerec,40j	8		T(1;3)3C7-9;81F;86B6-C1
*N ²⁶⁴⁻¹²²	X ray	Demerec,40j	8		
*N ²⁶⁴⁻¹²³	X ray	Demerec,40k	8		
*N ²⁶⁴⁻¹²⁴	X ray	Demerec,41a	8		
*N ²⁶⁴⁻¹²⁹	X ray	Demerec,41c	8		
*N ²⁶⁴⁻¹³¹	X ray	Demerec,41c	8		
*N ^A	spont	Aronson	1		several bands to right of 3C4 deranged
*N ^G	heat	Goldschmidt	15		

^α See references following Table I.

^β 1 = Variegation for *fa* in *N/fa*; 2 = Variegation for *spl* in *N/spl*; 3 = Developmental abnormalities of male same as in *Df(1)N-8*; 4 = *XYY* male viable but sterile; *XY* male lethal; 5 = A few males, normal in phenotype, survive; 6 = Male viable and mottled for *w* and *rsr*.

N^{66h26}

phenotype: The Notch inversion *N^{66h26}* (synonym: *In(1)w^{8x}N^{66h26}*), with breakpoints in *w* and *N*, was derived from *Df(1)N-8* and is unstable in crosses involving a *w^afa⁸rb* stock, giving rise to *N⁺* revertants such as *w^{8x1}N⁺* and *w^{8x2}N⁺* (Welshons and Keppy, 1981; Grimwade *et al.*, 1985). Recombination between *w* and *N*, which does not occur in *N^{66h26}*, does take place in these reversions, indicating that reversion to *N⁺* is

accompanied by reinversion of *In(1)N^{66h26}*. The *w^{8x1}N⁺* derivative of *N^{66h26}* is also unstable in crosses involving the *w^afa⁸rb* stock, generating (stepwise) various mutant and wild-type Notch alleles (Grimwade *et al.*, 1985). *Df(1)w79*, another derivative of *N^{66h26}*, is deficient for both *N* and *w* (Welshons and Keppy, 1981).
molecular biology: The lesion associated with the 3C7 breakpoint of *N^{66h26}* lies between -24.6 and -19.4 kb.

- N^{68j}**
phenotype: Typical Notch. *N^{68j/+}* females have wings excised at the tips; *N^{68j/spl}* females are *spl*. *N^{64j}* flies carrying *Dp(1;1)Co* are almost wild type. Mutant males with *w⁺Y* are viable (Hayman and Maddern, 1969).
- N^{68j1}**
phenotype: Typical Notch. *N^{68j1/Df(1)N-8}* females are lethal at 29° and 18°; Notch-wing phenotype shows little or no response to temperature (Shellenbarger and Mohler, 1975).
- N^{69c}**
phenotype: Shows variable Notch-wing expression depending on temperature. Lethal in homozygotes and in heterozygotes with *Df(1)N-8* at 29° and 18°. *N^{69c/+}* heterozygotes show greater expression of Notch-wing at 18° than at 29° (Shellenbarger and Mohler, 1975).
- N^{76b8}**
phenotype: Typical Notch.
molecular biology: The lesion associated with the 3C7-9 breakpoint lies between -2.2 and -1.3 kb on the physical map of Notch (Kidd *et al.*, 1983).
other information: Recombinational analysis indicates that the Notch locus distal to *spl* is in normal sequence.
- N^{77c17}**
phenotype: Typical Notch.
molecular biology: The lesion associated with the 3C7-9 breakpoint lies between -0.1 and 7.0 kb.
other information: *N^{77c17}* does not recombine with *N^{60g11}* (Welshons).
- N^{80j9}**
phenotype: Typical Notch. Genetically unstable, giving rise spontaneously to six independent *N⁺* revertants. The *N⁺10* revertant is rather unstable (Grimwade *et al.*, 1985).
molecular biology: The lesion associated with the 3C7-9 breakpoint of the inversion lies between -24.6 and -19.4 kb.
- N^{81k3}**
phenotype: Typical Notch.
molecular biology: This allele lies between -26.9 and -25.3 kb and seems to involve a deletion of 500 bp (Grimwade *et al.*, 1985).
- N^{81k6}**
phenotype: Typical Notch.
molecular biology: The lesion associated with the 3C6-9 breakpoint of the inversion lies between +5.5 and +7.0 kb.
- N^{81k8}**
phenotype: Typical Notch.
molecular biology: This allele affects the restriction pattern of two regions at the same time (Grimwade *et al.*, 1985), the first mapping between -10.6 and -9.5 kb and the second between -26.9 and -23.7 kb. Sequences between the fragments are unchanged as if *N^{81k8}* is an intralocus inversion.
- N^{81k9}**
phenotype: Typical Notch.
molecular biology: The lesion associated with the 3C6-9 breakpoint of the translocation lies between -25.3 and -23.7 kb.
- N^{81k10}**
phenotype: Typical Notch.
molecular biology: Mutant phenotype thought to be caused by position effect from the juxtaposition of the Notch DNA sequence and heterochromatin at 20A3-F (Grimwade *et al.*, 1985).
- N^{81l1}**
phenotype: Typical Notch.
molecular biology: The lesion associated with the 3C5-9 breakpoint of the inversion lies between -19.4 and -9.5 kb.
- N^{81l3}**
phenotype: Typical Notch.
molecular biology: This allele lies between +0.9 and +3.7 kb and seems to involve a small deletion of 2200 bp (Grimwade *et al.*, 1985).
- N^{81l5}**
phenotype: Typical Notch.
molecular biology: The lesion associated with the 3C5-9 breakpoint lies between 0 and +7.0 kb.
- N^{81l9}**
phenotype: Typical Notch.
molecular biology: The lesion associated with the 3C3-3D3 breakpoint lies between -16.7 and -9.5 kb.
- N²⁶⁴⁻⁴⁰**
phenotype: Typical Notch. Male embryos show developmental abnormalities like those of *Df(1)N-8* (Poulson, 1939). Lethal with *nd³*.
molecular biology: Lesion associated with a 0.4 kb insertion between -6.2 and -5.6 kb on the physical map of Notch (Kidd *et al.*, 1983).
- N²⁶⁴⁻⁴⁷**
phenotype: Typical Notch. Male embryos show developmental abnormalities like those of *Df(1)N-8* (Poulson, 1939). Lethal with *nd³*.
other information: *N²⁶⁴⁻⁴⁷/spl* heterozygotes produce nonrecombinant *N⁺* chromosomes with relatively high frequency (Welshons, 1958, Proc. Nat. Acad. Sci. USA 44: 254-58).
- N²⁶⁴⁻⁶⁶**
phenotype: Wing-notching weak and rarely visible. *N²⁶⁴⁻⁶⁶/fa* heterozygotes variegates for *fa*. Some *N²⁶⁴⁻⁶⁶Y* males are viable and have cream-colored eyes with spots of normal red pigment.
- N²⁶⁴⁻¹⁰³**
phenotype: Temperature-sensitive Notch allele. *N²⁶⁴⁻¹⁰³/nd³* females are viable at 22° and lethal at 29°, with a long, possibly polyphasic, TSP beginning in the embryonic stage. *N²⁶⁴⁻¹⁰³/spl* females show eye-facet disarray, notching, bristle-number variation, and tarsal-segment fusion, the TSP being in the third instar (Foster, 1973). *N²⁶⁴⁻¹⁰³/spl* variegates for *spl*; *N²⁶⁴⁻¹⁰³/fa* is *fa*.
- N²⁶⁴⁻¹⁰⁷**
phenotype: Typical Notch.
other information: The *N²⁶⁴⁻¹⁰⁷* chromosome carries another Notch mutant site, *l(1)N*, at the distal end near *N^{55e11}* (Welshons, 1965).

N²⁶⁴⁻¹⁰⁹

phenotype: Typical Notch except for semilethality with *nd*³.

molecular biology: Lesion associated with a 14.8 kb insertion between +3.7 and +4.4 kb on the physical map of Notch (Kidd *et al.*, 1983).

N^{Co}

phenotype: Wing tips seldom notched; veins thickened, with deltas. Acrostichal rows irregular. *N^{Co}nd*³ heterozygotes lethal; rare survivors sterile and weak. *N^{Co}/+* females show thickened wing veins (a Confluens-like phenotype) more frequently than nicked or notched wings. Also, *N^{Co}* heterozygous females with an extra dose of 3C6-7 [*Dp(1;1)Co* or *Dp(1;2)51b*] or hemizygous males with *Dp(1;2)51b* have an enhanced Confluens-like wing phenotype.

N^{hdA171}

phenotype: Typical Notch.

molecular biology: Lesion associated with a 6.3 kb insertion between -28.4 and -27.1 kb on the physical map of Notch. *N^{hdA171}* is the most distal of the dominant Notch alleles and is thought to be caused by a defective *P*-element (Grimwade *et al.*, 1985).

N^{hdC8}

phenotype: Typical Notch.

molecular biology: Lesion associated with a 5.2 kb insertion between +2.7 and +3.7 kb, the insertion carrying moderately repetitive DNA sequences (Grimwade *et al.*, 1985).

N^{J24}

phenotype: Typical Notch. Lethal with *nd*³.

N^M: Notch Mischiakow

phenotype: Wings notched at tips and occasionally at sides; veins thickened, with deltas. Eyes slightly smaller than normal; occasionally one eye extremely small.

molecular biology: The Notch locus in the reverted chromosome *N^{+M}* contains an insertion homologous to the FB4 foldback element (Grimwade *et al.*).

N^{Nic}: Notch Nicoletti

phenotype: Typical Notch. Lethal with *nd*³. Cell lethal in tergites and dorsal mesothorax (Ripoll and Garcia-Bellido, 1979, *Genetics* 91: 443-53).

N^S: Notch Schalet

synonym: *l(1)16-178*.

phenotype: Weak Notch. *N^S/Ax^S* is lethal.

molecular biology: Lesion associated with a five kb insertion between +3.5 and +2.90 kb on the physical map of Notch (Kidd *et al.*, 1983).

***nd*: notchoid (W.J. Welshons)**

location: 1-3.0.

phenotype: Wings notched and veins thickened. The notching is found mostly on anterior and posterior margins and is the result of cell death (Thompson and Spivey, 1984, *Genet. Res.* 44: 201-69). Homozygotes are viable and fertile in both sexes. *N/nd*¹ heterozygotes are partially viable and relatively infertile (Portin, 1977) and show notched and straplike wings and small eyes. About 10% of *fa/nd*¹ flies have small notches in one or both wings. *nd*³/*nd*¹ heterozygotes have slightly thick-

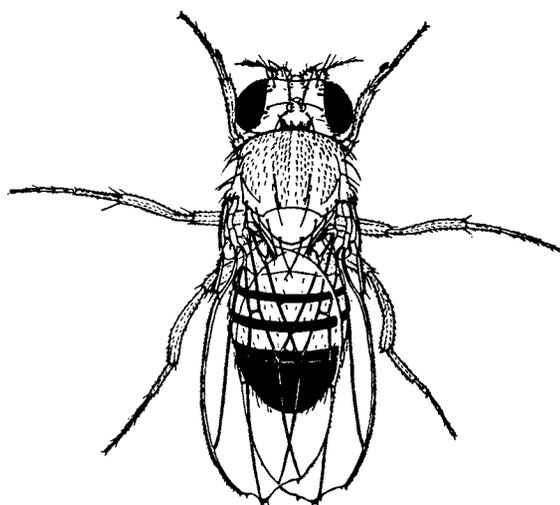
ened wing veins with deltas; *spl/nd*¹ heterozygotes lack a few bristles (like *spl/+*) and their eyes are sometimes smaller than normal and roughened. *spl nd*¹ males have rough eyes, *nd*-like wings, and irregular, bushy sex combs.

alleles:

allele	origin	discoverer	synonym	ref ^α
<i>nd</i> ⁰	X ray	Glass, 1929	<i>fa</i> ⁿ	4
<i>nd</i> ¹	TEM	Fahmy, 1951	<i>n¹fa^h</i>	2, 3, 6-9, 11
<i>nd</i> ²		R.M. Valencia,		6, 8, 10, 11
<i>nd</i> ³	X ray	Bauer	<i>fa</i> ^{no}	1, 8, 9, 11, 12
<i>nd</i> ^{3.1072}	spont	R. Berg		5
<i>nd</i> ^{3.1072rv}		R. Berg		5
<i>nd</i> ⁴	EMS	Shellenbarger and Mohler	<i>fa no69db</i>	8
<i>nd^{ts69d}</i>	EMS	Shellenbarger		8
<i>nd^{ts69f}</i>	EMS	Shellenbarger		8
<i>nd^{ts69j}</i>	EMS	Shellenbarger	<i>nd^{ts69j3}</i>	8
<i>nd^{ts70j}</i>	EMS	Shellenbarger		8

^α 1 = Bauer, 1943, *Z. Indukt. Abstamm. Vererbungsl.* 81: 374-90. 2 = Fahmy, 1958, *DIS* 32: 72; 3 = Foster, 1973, *Genetics* 73: 435-38; 4 = Glass, 1933, *J. Genet.* 27: 233-41; 5 = Kelley, Kidd, Berg, and Young, 1987, *Mol. Cell Biol.* 7: 1545-48; 6 = Kidd, Lockett, and Young, 1983, *Cell* 34: 421-33; 7 = Portin, 1977, *Hereditas* 87: 77-84; 8 = Shellenbarger and Mohler, 1975, *Genetics* 81: 143-62; 9 = Welshons, 1965, *Science* 50: 1122-29; 10 = Welshons, 1971, *Genetics* 68: 259-68; 11 = Welshons, 1974, *Genetics* 76: 775-94; 12 = Welshons and Von Halle, 1962, *Genetics* 76: 775-94.

cytology: Salivary chromosomes are normal (Fahmy).



nd⁰ (= **fa**ⁿ)

From Glass, 1933, *J. Genet.* 27: 233-41.

***nd*⁰**

synonym: *fa*ⁿ: facet-notched.

phenotype: Wings have apical nicks or notches in 90-100% of males, but only 8% of homozygous females. Eyes not rough. *fa/nd*⁰ is wild type. Viability and fertility excellent. RK2 in male.

cytology: *nd*⁰ is on an *In(1)dl-49* chromosome, and has not been separated from the inversion.

nd¹

phenotype: *nd¹* is temperature-sensitive. In homozygotes at 29°, the eyes are rough and reduced in size, there is extreme wing notching, and wing veins are thick; at 25°, the abnormalities are less severe, and at 18°, the eyes are normal and the wings are nicked. At 29°, heterozygotes with *fa*, *fa⁸*, and *spl* are complementary (Foster, 1973; Shellenbarger and Mohler, 1975). *nd¹/Y;E(spl)^{r19}/+* males have severely reduced and crumpled wings (Xu, Rebay, Fleming, Scottgale, and Artavanis-Tsakonas). Wing development also affected in *nd¹/Y;mam¹⁰/+* males. *nd¹* and *mam¹⁰* double heterozygotes are wild type. Wing notching is suppressed in *nd¹/Y* males by *Dll+*.

molecular biology: *nd¹* maps at the 3' end of the Notch locus and carries a three bp insertion in the opa repeat producing an extra glutamine and a missense mutation resulting in a threonine to isoleucine change (Xu *et al.*).

nd²

phenotype: *nd²/nd²* and *nd²/nd¹* flies resemble *nd¹* homozygotes; *nd²/nd³* heterozygotes are noncomplementary (Welshons). The *nd²* allele is temperature sensitive; in homozygotes at 29°, the eyes are small and rough (*spl*-like), wings have extreme notches, wing veins are thickened, tarsi are shortened, and the mutants are semilethal as late pupae; at 25°, the abnormalities are much less severe; at 18°, the eyes are slightly *spl*-like, wings are nicked, wing veins are incomplete, some bristles are missing, and the mutants are semilethal as late pupae. At 29°, *fa/nd²* heterozygotes have nicked wings, *spl/nd²* heterozygotes are *spl*-like, and *nd⁴/nd²* heterozygotes resemble *N/+* (Shellenbarger and Mohler, 1975). Similar wing abnormalities in *nd¹/Y;E(spl)^{r19}/+* and *nd²/Y;E(spl)^{r19}/+* males. The temperature-sensitive rough-eye phenotype of *nd²* is enhanced by *E(spl)/+* (Xu *et al.*).

molecular biology: *nd²* maps proximal to *nd¹* at the 3' end of Notch and shows a deletion of one bp.

nd³

synonym: *fa^{no}*: facet-notchoid.

phenotype: Wings of both sexes notched at ends of L3 and L4 veins; wing veins enlarged and delta-like at tips. Mild mutant expression often limited to wing-vein effect. Mutant expression diminished at high temperature (Shellenbarger and Mohler, 1975, Genetics 81: 143-62). Heterozygotes show extremely weak dominance. *nd³/N* almost completely lethal; survivors are sterile and have an exaggerated Notch phenotype. *nd³/fa* closely resembles wild type. Heterozygotes with *fa⁸* and *spl* are complementary; with *nd* and *nd²*, heterozygotes are noncomplementary with a mild mutant expression of *nd³*-like wings. Up to 5% of *nd³* males from aged cultures show hyper- and hypodeveloped external genitalia (Kroeger, 1960, J. Morphol. 107: 227-32).

nd^{3.1072}

phenotype: Viable when homo- or hemizygous; shows adult wing nicking. Lethal when heterozygous with *N* deficiencies. Phenotype similar to *nd*.

molecular biology: Associated with 1.2 kb *P*-element insertion within 0.1 kb of *N* transcription start site (Kelley *et al.*, 1987). Unlike *nd* and *nd²*, occupies left end of genetic map.

nd^{3.1072rv}

phenotype: Wild-type revertant of *nd^{3.1072}*. Viable in combination with *N* deficiencies.

molecular biology: Retains 200 bp of the original 1.2 kb *P*-element insertion (Kelley *et al.*, 1987).

nd⁴

synonym: *fa^{no69}*.

phenotype: Temperature sensitive and semilethal in homozygotes; wing phenotype more extreme and survival greater at 18° than at 29°. *nd⁴/+* lethal at both temperatures.

nd^{ts69d}

phenotype: Like *nd^{ts69j}* (see below).

nd^{ts69f}

phenotype: Like *nd^{ts69j}* (see below).

nd^{ts69j}

phenotype: Temperature-sensitive semilethal. Homozygotes express weak notches, mild deltas, and extra bristles at both 18° and 29°; both homozygotes and hemizygotes show significantly better survival at 18° than at 29°. *nd^{ts69j}/+* heterozygotes have normal wings at 18° and 29°. *Df(1)N-8/nd^{ts69j}* heterozygotes have significantly better survival at 18° than at 29°.

nd^{ts70j}

phenotype: In general, *nd^{ts70j}* homozygotes and heterozygotes resemble the other *N^{ts}* mutants. *nd^{ts70j}* homozygotes, however, are wild type at 18° and some *nd^{ts70j}/+* heterozygotes have notched wings at 29°.

spl (W.J. Welshons)

location: 1-3.0.

origin: X ray induced.

discoverer: Dubinin.

synonym: *shd; fa³*.

references: Serebrovsky and Dubinin, 1930, J. Heredity 21: 259-65.

Agol, 1931, Genetics 16: 262.

Dubinin, 1934, DIS 1: 10.

Welshons and Von Halle, 1962, Genetics 21: 743-69.

Welshons, 1971, Genetics 68: 259-68.

Foster, 1973, Dev. Biol. 32: 282-96.

Shellenbarger and Mohler, 1975, Genetics 81: 143-62.

Hartley, Xu, and Artavanis-Tsakonas, 1987, EMBO J. 6: 3407-17.

Kelley, Kidd, Deutsch, and Young, 1987, Cell 51: 539-48.

Cagan and Ready, 1989, Genes Dev. 3: 1099-1112.

Shephard, Broverman, and Muskavitch, 1989, Genetics 122: 429-38.

phenotype: In homozygotes, eyes are rough and small, bristles are often doubled or split (sometimes missing). Hemizygotes show a more extreme reduction in eye size as well as an increase in facet and bristle abnormalities (Shephard *et al.*, 1989). Both eye and bristle abnormalities occur at all temperatures from 18° to 29°, an exception being a *spl* stock from Novosibirsk, Russia, that shows temperature sensitivity (Mglinetz, 1980, DIS 55: 107-08). The bristle phenotype is caused by an extra division of an initial bristle-forming cell (Lees and Waddington, 1943, Proc. R. Soc. London, B 131: 87-110; Van Breugel and Van der Aart, 1979, Dev. Biol.

186: 267-71). A few bristles (sockets remaining) are usually removed from the posterior border of tergites in *spl/+* heterozygotes (Welshons). The eye abnormalities are the result of abnormal differentiation of photoreceptors at the morphogenetic furrow (Cagan and Ready, 1989). Heterozygotes with the other recessive visibles at Notch are almost normal except for *spl/nd²* flies; the latter are *spl*-like at 29° (Shellenbarger and Mohler, 1975). Another temperature-sensitive effect is shown by *N²⁶⁴⁻¹⁰³/spl* flies, which have abnormal eye facets at 28-29° but are almost wild type at 20-22° (Foster, 1973, Dev. Biol. 32: 282-96). The *spl* phenotype can be enhanced by *E(spl)/+* or *E(spl)/E(spl)*. *spl/+;E(spl)/+* flies resemble *spl/spl* flies; *spl/spl*; *E(spl)/+* and *spl/Y*; *E(spl)/+* flies show a very extreme mutant phenotype (Shephard *et al.*, 1989). *spl/Y;E(spl)^{RI9}/+* males and *spl/+;E(spl)^{RI9}/+* females show *spl* and *Ax*-like phenotypes (Xu *et al.*). The *spl* phenotype is reduced in *mam* heterozygotes. When, however, *spl* is coupled to a *N* point mutant, as in *N^{64d6} spl/+ +;E(spl)/+*, the phenotype is not *spl* (Welshons, 1971) split behaves autonomously in mosaics in regard to both eye and bristle phenotypes (Stern and Tokunaga, 1968, Proc. Nat. Acad. Sci. USA 60: 1252-59). The *spl* phenotype becomes dominant if *spl* is coupled, in *cis*, to lethal *Ax* alleles. Thus *Ax spl/+* is *spl*, while *+spl/+* is wild type (Welshons, 1971; Kelley *et al.*, 1987).

alleles: Three alleles have been reported: *spl*, **spl²*, *spl^{66c29}*.

cytology: Salivary chromosomes normal. Placed in 3C7 on the basis of the interaction of split with Notch.

molecular biology: *spl* cloned and sequenced. Located on the molecular map of *N* to the right of *fa⁸* at about +2 kb. It is a missense mutation in the fourteenth EGF-like repeat in the extracellular domain of the putative *N* protein and involves a change from thymine to cytosine causing an isoleucine-to-threonine substitution at residue 578 (Hartley *et al.*, 1987; Kelley *et al.*, 1987).

***spl²**

origin: Spontaneous.

discoverer: Gottschewski, 1935.

phenotype: Resembles *spl* except for smaller eyes.

N-2G: see *mam*

***N-b: Notch-b**

location: 2- (not located).

origin: Spontaneous.

discoverer: Mann, 1921.

synonym: *Notch 2*.

references: 1923, Genetics 8: 27-36.

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 232.

phenotype: Resembles Notch. Wing nicked in about 10% of heterozygous flies. Homozygote probably lethal. RK3.

other information: Possibly a *vg* or *mam* allele.

na: narrow abdomen

location: 1-45.2.

origin: X ray induced.

discoverer: H. M. Miller, 34c.

references: 1934, DIS 2: 9.

1935, DIS 4: 9.

Hardy, Lindsley, Livak, Lewis, Sivertsen, Joslyn,

Edwards, and Bonaccorsi, 1984, Genetics 107: 591-610.

phenotype: Abdomen long and cylindrical in both sexes. Viability low; female fertility low. Ovaries in juvenile condition (Brehme). RK2.

cytology: Placed in 12E1-13A5 on the basis of its inclusion in *Df(1)KA9 = Df(1)I2E1;13A5*.

***na²**

origin: Ultraviolet induced.

discoverer: Edmondson, 51g.

references: 1952, DIS 26: 60.

phenotype: Like *na*. RK2.

Na-CP: Na-channel-protein

location: 2-{107}.

references: Ashburner.

phenotype: Structural gene for sodium-channel protein.

cytology: Placed in 60E.

nac: neuronally-altered-carbohydrate (F. Katz)

location: 3-{48}.

origin: Induced by ethyl methanesulfonate.

references: Katz, Moats, and Jan, 1988, EMBO J. 7: 3471-77.

phenotype: Mutants show alteration or loss of a normally neuron-specific glycoconjugate; staining by anti-HRP antibodies in imaginal and adult neural tissue is eliminated. At 25° the mutant flies are viable and fertile; *nac/nac* females, however are sterile at 18°. Under heat stress (37° for five minutes) they show abnormal jittery behavior. Developmental abnormalities, including defects in the assembly of the ommatidia and in the formation of the wing, appear at 18° in the homozygous offspring of heterozygous parents. Maternal effect embryos show loss of the anti-HRP glycan determinant and are lethal.

cytology: Located in 84F4-84F12.

naked cuticle: see *nkd*

nanos: see *nos*

nap^{ts}: no action potential (J.C. Hall; M. Kernan)

location: 2-56.2.

origin: Induced by ethyl methanesulfonate.

references: Wu, Ganetzky, Jan, Jan, and Benzer, 1978, Proc. Nat. Acad. Sci. USA 75: 4047-51.

Wu and Ganetzky, 1980, Nature (London) 286: 814-16.

Kauvar, 1982, Mol. Gen. Genet. 187: 172-73.

Jackson, Wilson, Strichartz and Hall, 1984, Nature 308: 189-91.

Ganetzky, 1984, Genetics 108: 897-911.

Kyriacou and Hall, 1985, Nature (London) 314: 171-73.

Burg and Wu, 1986, J. Neurosci. 6: 2968-76.

O'Dowd and Aldrich, 1988, J. Neurosci. 8: 3633-43.

Stern, Kreber, and Ganetzky, 1990, Genetics 124: 133-43.

Elkins and Ganetzky, 1990, J. Neurogenet. 6: 207-19.

Nelson and Wyman, 1990, J. Neurobiol. 21: 453-69.

Budnick, Zhong, and Wu, 1990, J. Neurosci. 10: 3754-68.

phenotype: Larvae or adults become rapidly paralyzed when exposed to 37° and rapidly recover on return to lower temperatures. Rearing stocks chronically at room temperature or above causes *nap^{ts}* to "adapt" such that

higher temperatures (> 40°) are required for paralysis (Kyriacou and Hall, 1985). Experiments involving one-time rearing at low temperature caused *nap^{ts}* to paralyze at relatively low temperatures (Nelson and Wyman, 1990). Axonal conduction (but not synaptic transmission) fails in larvae at high temperatures (Wu *et al.*, 1978; Wu and Ganetzky, 1980), but action potentials in the giant fiber (GF) pathway of adults are not blocked at temperatures up to 43° (Elkins and Ganetzky, 1990; Nelson and Wyman, 1990), though the latency from brain stimulation to response of thoracic muscles are aberrantly long, even at low temperatures (Nelson and Wyman, 1990), and this long-latency disappears as the temperature is raised to 35° (Elkins and Ganetzky, 1990). "Following frequency" of *nap^{ts}* thoracic muscle responses (*re.* GF pathway stimulation) reduced at elevated temperatures, an effect which can be reversed by injection of 4-aminopyridine (Nelson and Wyman, 1990). At permissive temperatures, refractory period for elicitation of a series of action potentials is abnormally long (Ganetzky and Wu, 1980); at these low temperatures, *nap^{ts}* suppresses effects of "hyperexcitability" mutations such as *Sh*, *bas*, *bss*, *eas*, *Hk*, *kdn*, and *tko* (Ganetzky and Wu, 1982, Genetics 100: 597-614). *nap^{ts}* is unconditionally lethal (Ganetzky and Wu, 1980) in a double mutant with *para^{ts1}* (death occurring during 1st larval instar) and the viability of other *para*; *nap^{ts}* combinations is poor (Ganetzky, 1984); two doses of *para⁺* (in males) suppresses high-temperature paralysis of *nap^{ts}* (Stern *et al.*, 1990). In mosaic experiments, cuticular clones of *para^{ts1}* in a *nap^{ts}* background (after low-temperature development) have non-functioning sensory cells, probably due to lack of nerve conduction which, however, did not cause any anatomical abnormalities involving the central projections of these sensory neurons (Burg and Wu, 1986). Another developmental study, examining larval nerve terminal innervating body-wall muscles (Budnik *et al.*, 1990), showed slight reduction in the extent of branching caused by *nap^{ts}* at permissive temperature; the increase in branching (and higher than normal number of varicosities on motor-neurites) induced by an *eag Sh* double mutant was suppressed by *nap^{ts}* (*re.* low-temperature rearing). *nap^{ts}* in combination with *tipE* leads to poor viability at permissive temperature for both mutants (Ganetzky, 1986, J. Neurogenet. 3: 19-31; Jackson, Wilson, and Hall, 1986, J. Neurogenet. 3: 1-17). *nap^{ts}* is, at permissive temperature, hypersensitive to blocking effects of tetrodotoxin (TTX) on action potentials (Ganetzky and Wu, 1980); brain membrane extracts of *nap^{ts}*, assayed at low or high temperatures, have subnormal levels of tetrodotoxin (Kauvar, 1982) or saxitoxin (Jackson *et al.*, 1984) binding activity; the latter study reports that there are no qualitative alterations of this binding activity (kd is normal). Cultured neurons from *nap^{ts}* larvae are 4 to 5-fold more resistant than wild-type cells to killing effects of veratridine, irrespective of temperature (22° vs 35°) (Suzuki and Wu 1984, J. Neurogenet. 1: 225-38), but TTX has no effects on these mutant cells, whose general growth characteristics are also normal (Wu, Suzuki and Poo, 1983, J. Neurosci. 3: 1888-99). The mutation does not seem to modify the expression of sodium currents in embryonic neurons (O'Dowd and Aldrich, 1988, J. Neurosci. 8: 3633-43). Exposure of *nap^{ts}* males to high temperature causes

arrest of oscillator underlying rhythmic component of courtship song (Kyriacou and Hall, 1985); in experiments on conditioned courtship, *nap^{ts}* males learn normally but have shortened memory spans, and *nap^{ts}* suppresses *Sh*-induced decrements in courtship learning (Cowan and Siegel, 1984, J. Neurogenet. 1: 333-44; 1986, J. Neurogenet. 3: 187-201).

alleles:

allele	origin	synonym	discov/ref ^α	comments
<i>nap^{ts1}β</i>	EMS		2	temperature-sensitive paralysis when homo- or hemizygous; male viable when homozygous; lethal with <i>para^{ts1}</i>
<i>nap^{ts2}</i>	HD	<i>nap^{hd9}</i>	1	fails to complement <i>nap^{ts1}</i> paralysis; no P element insertion male viable when homozygous
<i>nap^{ts3}</i>	EMS	<i>nap¹⁻¹</i>	1	fails to complement <i>nap^{ts1}</i> paralysis; male viable when homozygous
<i>nap^{ts4}</i>	EMS	<i>nap¹⁰⁻¹⁻¹</i>	1	fails to complement lethality of <i>nap^{ts1}</i> in <i>para^{ts1}</i> background; male viable when homozygous
<i>nap^{ts5}</i>	EMS	<i>nap²⁰⁻⁴⁻¹</i>	1	fails to complement lethality of <i>nap^{ts1}</i> in <i>para^{ts1}</i> background; male viable when homozygous
<i>nap⁶</i>	EMS	<i>nap¹⁷⁻¹</i>	1	temperature-sensitive paralysis with <i>nap^{ts1}</i> ; male viable when homozygous
<i>nap^{7γ}</i>	γ ray			fails to complement <i>nap^{ts1}</i> paralysis; male lethal when homozygous or with <i>mle</i> ; homozygous females not temperature sensitive
<i>nap^{rv1}</i>	γ ray	<i>nap^{tsrev1}</i>	1	male lethal in homozygotes or with <i>mle</i> ; homozygous females not temperature sensitive; reduction in temperature sensitivity with <i>nap^{ts1}</i> and <i>para^{ts1}</i>

α 1 = Kernan and Ganetzky; 2 = Wu, Ganetzky, Jan, Jan, and Benzer, 1978, Proc. Nat. Acad. Sci. USA 75: 4047-51.
 β Homozygous *nap¹* flies paralyzed at lower temperature than *nap¹ Df(2R)nap* flies (Kernan and Ganetzky). Paralysis of homozygotes suppressed by *para⁺* duplications (Stern and Ganetzky).
 γ *T(2;3)nap⁷ = T(2;3)42A2-7;80-81.*

cytology: Located in 42A2-8, since uncovered by *Df(2R)nap9* (R. Kreber).

molecular biology: Locus cloned by Kuroda, Kernan, Kreber, Ganetzky, and Baker. Molecular analyses of *nap* and *mle* indicate that the same open reading frame encodes *mle⁺*, *nap⁺* and *nap^{ts}* activities.

other information: The paralysis of *nap* is not complemented by *mle* alleles, thus *nap* appears to be allelic to *mle* (Kuroda, Kernan, Kreber, Ganetzky, and Baker). Germline transformation and analysis of mutations also show that *nap^{ts}* is a gain-of-function mutation of *mle*.

narrow: see *nw*

narrow abdomen: see *na*

narrow-Blade: see *nw^B*

narrow-Dominant: see *nw*^D

narrow eye: see *ney*

narrow scoop: see *nrs*

nbA: night blind A (J.C. Hall)

location: 1-36.6.

synonym: *nonB*; *omp18*; *P18*.

references: Heisenberg and Gotz, 1975, *J. Comp. Physiol.* 98: 217-241.

Heisenberg and Buchner, 1977, *J. Comp. Physiol.* 117: 127-62.

Heisenberg, 1979, *Handbook of Sensory Physiology* (H. Autrum, ed.). Springer-Verlag, Berlin, Vol. 7, pp. 665-79.

Bülthoff, 1982, *Biol. Cybernet.* 45: 63-70.

1982, *DIS* 58: 31.

Kulkarni and Hall, 1987, *Genetics* 115: 461-75.

Homyk and Pye, 1988, *J. Neurogenet.* 5: 37-48.

phenotype: Higher than normal light-intensity thresholds needed for optomotor or phototactic responses (Heisenberg and Götz, 1975). More specifically, high-acuity optomotor responses (ability to respond to relatively narrow moving stripes) relatively normal, but high-sensitivity flies (ability to respond to moving stimuli in dim light conditions) are impaired, hence, a "night-blind" phenotype (Heisenberg). In "slow phototaxis/Y-tube" tests (using ordinary white fluorescent light), mutant adults were extremely subnormal and in fact preferred the dark-arm of the Y (Kulkarni and Hall, 1987). Light-on and light-off transient spikes of electroretinogram reduced in amplitude, possibly absent (Heisenberg, 1979; Homyk and Pye, 1989). Weak orientation to spots in Y-maze test (Bülthoff, 1982). *nbA* mutation may define an essential gene, because *nbA*³/*l(1)11Aa*¹ heterozygotes lack ERG transients (Homyk and Pye) and are severely defective in phototaxis (Kulkarni and Hall, 1987).

alleles:

allele	origin	discoverer	synonym
<i>nbA</i> ¹			
<i>nbA</i> ²	EMS	Heisenberg	<i>nbA</i> ^{H18} ; <i>omp18</i>
<i>nbA</i> ³	EMS	Heisenberg	<i>nbA</i> ^{H47} ; <i>omp47</i>
		Eichenberger	<i>nbA</i> ^{EE171} ; <i>ldg</i>

cytology: Placed in 11A2. Consistent with the non-complementation of *l(1)11Aa*¹ is that *nbA*³ has its ERG (Homyk and Pye, 1989) and phototaxis (Kulkarni and Hall, 1987) defects uncovered by *In(1)A78*, *In(1)A97*, or *Tp(1;1)A101*, which have common breakpoints in 11A2 and fail to complement *l(1)11Aa* lethals (Homyk and Pye; Lefevre, 1981). Hence, *nbA* maps to 11A2. Included in *Df(1)RC29*, a cytologically invisible deletion that removes *l(1)11Aa*, *gd*, *tsg*, and *fw* (see Kulkarni and Hall, 1987, and Homyk and Pye, 1989 for further details).

other information: The *ldg* (lamina-degeneration) designation for one of the alleles of this gene (*nbA*³) turned out to be a misnomer. *nbA* was also once called *nonB* (no-on-transient-B) [Pak, 1975, *Handbook of Genetics* (King, ed.). Plenum Press, New York, Vol. 3, pp. 703-33], but this designation was subsequently dropped. Possible complexity of the genes in the 11A2 region is suggested by the failure of not only *nbA*, but also the very closely linked *cac* courtship song mutation to complement *l(1)11Aa* and the (lethal) breakpoints in 11A2 (Kul-

karni and Hall, 1987; also see **cytology**). *cac* seems otherwise unrelated to *nbA* (Kulkarni and Hall, 1987), in that this song mutant has normal vision (behaviorally and physiologically), *nbA* males sing normally, and the two kinds of mutation complement each other for both phenotypes (tested in a homozygous *tra* background, with regard to courtship song).

nc2: see *hay*^{nc2}

nc3: see *ms(3)nc3*

nc4: see *wrl*

nc16: see *mrn*

nc32: see *ms(3)nc32*

ncd: non-claret disjunctional

location: 3-100.7 (to the left of *ca*).

origin: Induced by ethyl methanesulfonate.

references: Davis, 1969, *Genetics* 61: 577-24.

Sequeira, Nelson, and Szauter, 1989, *Genetics* 123: 511-24.

Yamamoto, Komma, Shaffer, Pirrotta, and Endow, 1989, *EMBO J.* 8: 3543-52.

Endow, Henikoff, and Niedziela, 1990, *Nature (London)* 345: 81-83.

McDonald and Goldstein, 1990, *Cell* 61: 991-1000.

phenotype: Disjunction in homozygous females abnormal; incidence of nondisjunction of all chromosome pairs in the first meiotic division and of meiotic and early-cleavage mitotic loss of maternally inherited chromosomes is high. X-chromosome recombination normal among both regular and exceptional progeny. Behavior of nonhomologues correlated; doubly disomic and doubly nullosomic ova more frequent than expected (Davis). Similar in action to *ca* of *D. simulans* (Sturtevant, 1929, *Z. Wiss. Biol. Abt. A* 135: 323-56). Two thirds of mitotic loss of chromosomes in progeny of *ncd* mothers takes place in the first zygotic division; one third takes place subsequently. The majority of X-chromosome loss (85-95%) is of the maternally inherited X but there is also appreciable loss of the paternally inherited X as well (Sequeira *et al.*). Somatic loss of X and 4 correlated (Portin, 1978, *Heredity* 41: 193-203). Cytological description of meiotic behavior in *D. simulans ca* females (Wald, 1936, *Genetics* 21: 264-81) includes abnormal first meiotic spindle, second meiotic arrest, and dispersal of chromosomes into multiple micronuclei; micronuclei also observed in *ncd* females (Roberts). Spindles frequently multipolar in first meiotic division (Puro) and in early-embryo nuclear divisions, and nuclei remain close together in center of egg (Kimble and Sandstedt, 1981, *Genetics* 978: s97). Kimble and Church (1981, *J. Cell Sci.* 62: 301-18) observed four classes of metaphase I configurations: (1) two or more spindles (2) abnormally wide spindles with widely separated bivalents (3) unipolar spindles, and (4) normal spindles; the first three comprise 80% of configurations. Approximately 20% of eggs of *ncd*¹ females asymmetrical or with more than two appendages (Kimble and Church). Hinton and McEarchen (1963, *DIS* 37: 90) reported a haploid-diploid mosaic. *ncd* ovaries transplanted into normal host behave autonomously (Roberts, 1962, *DIS* 36: 120). Chromosome segregation normal in *ncd* males.

alleles: Most *ncd* mutations recovered simultaneously with *ca* mutations; the double mutants are designated *cand* in the claret entry; they are caused by a single lesion affecting both transcription units, and are inseparable.

allele	origin	synonym	ref ^α	comments
<i>ncd</i> ¹	X ray		2	recovered as <i>cand</i> ; 2.6 kb deletion
<i>ncd</i> ²	EMS		3	recovered as <i>ca⁺</i> ; no 2.6 kb deletion
<i>ncd</i> ³	γ ray	<i>ca^{nd2}</i>	4	recovered as <i>ca^{nd3}</i>
<i>ncd</i> ⁴	γ ray	<i>ca^{nd3}</i>	4	recovered as <i>ca^{nd4}</i>
<i>ncd</i> ⁵	HD	<i>ca³⁻¹</i>	5	recovered as <i>ca^{nd5}</i> ; 2.6 kb deletion
<i>ncd</i> ⁶	HD	<i>ca²²⁻⁸</i>	5	recovered as <i>ca^{nd6}</i> ; 2.6 kb deletion
<i>ncd</i> ⁷	spont	<i>ca^{Cm}</i>	5	recovered as <i>ca^{nd7}</i> ; 2.6 kb deletion
<i>ncd</i> ⁸	HD	<i>ca^{P3}</i>	5	recovered as <i>ca^{nd8}</i> ; 2.6 kb deletion
<i>ncd</i> ^{Dβ}	EMS	<i>NCD</i>	1	slight dominant effect

^α 1 = Kennison; 2 = Lewis and Gencarella, 1952, Genetics 37: 600-01; 3 = O'Tousa and Szauter, 1980, DIS 55: 119; 4 = Sequeira, Nelson, and Szauter, 1989, Genetics 123: 511-24; 5 = Yamamoto, Komma, Shaffer, Pirrotta, and Endow, 1989, EMBO J. 8: 3543-52.
^β Discoverer: Steiner.

cytology: Using *in situ* hybridization, *ncd* was placed in 99B8-10 by Yamamoto *et al.* (1989) and in 99B-C by McDonald and Goldstein (1990).

molecular biology: Region cloned in a 160-kb chromosome walk from 99C6-8 to 99B8-10 (Yamamoto *et al.*, 1989); the presumed *ncd* gene was later cloned and identified by PCR (McDonald and Goldstein, 1990). Transformation with a 0.9-kb fragment that contains the site of insertion of the *P* element in *ca³⁴* rescues the *ncd* phenotype. This fragment hybridizes to a 2.2-kb mRNA that is abundant in adult ovaries but not in the ovary-minus carcasses nor in males. The transcript is absent in *ncd*¹ and *ncd*⁷, but is present in *ncd*², *ca*¹, and *ca*³⁴. The 2.2-kb mRNA is transcribed from right to left and its 5' end is very close to that of a transcription unit on the opposite strand that generates a 7.4-kb mRNA thought to encode the *ca* product. All five *ncd* *ca* chromosomes tested have the same or similar 2.6-kb deletions for the 5' region shared by the two transcription units: *ncd*², which is *ca*⁺, fails to show the deficiency, and is presumed to be a point mutation. Nucleotide and putative nucleic acid transcripts have been obtained by Endow *et al.* (1990) and McDonald and Goldstein (1990). The cDNA is 2294 nucleotides in length, has a single open reading frame, and is predicted to encode a 75,795-dalton protein 685 amino acids in length (McDonald and Goldstein, 1990). The amino acid sequence shows significant similarity in its carboxy-terminal domain to the motor amino-terminal domain of kinesin heavy chain (Endow *et al.*, 1990; McDonald and Goldstein, 1990), but the non-motor domains of the proteins encoded by *ncd* and *Kin* are not similar. Biochemical properties of the presumed *ncd* protein were tested and found to be similar to those of kinesin, suggesting that *ncd* encodes a kinesin-like protein.

***ncn*: nurse cell number**

location: 3-68.
origin: Induced by ethyl methanesulfonate.
discoverer: Nüsslein-Volhard.

synonym: *ncn-1*.

references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-69.

phenotype: Female sterile; no eggs laid. Follicles have increased numbers of nurse cells and are often tumorous.

alleles: *ncn*⁰¹⁸ and *ncn*⁰⁷⁷.

***nd*:** see *N*

***ndl*:** *nudel*

location: 3-17.

synonym: *mat(3)2*; *mel(3)5*.

references: Rice, Ph.D. Thesis, Yale University.

Anderson and Nüsslein-Volhard, 1984, Nature 311: 223-27.

phenotype: Maternal effect mutation producing totally dorsalized embryos. Spacing of transverse stripes of *ftz* protein altered in mutant embryos (Carroll, Winslow, Twombly, and Scott, 1987, Development 99: 327-32).

alleles: *ndl*¹ through *ndl*⁷ recovered as *ndl*⁰⁴⁶, *ndl*⁰⁹³, *ndl*¹¹¹, *ndl*¹³³, *ndl*¹⁶⁹, *ndl*²⁶⁰, and *ndl*^{rm2}.

***Ndw*:** *Nicked wing*

location: 3-87.2.

origin: Treatment with DNA of nuclear polyhedrosis virus from *Galleria melonella*.

references: Shandala, 1985, Tsitol. Genet. 19: 179-83.

phenotype: Homozygous lethal. Wings nicked in heterozygotes. Maximum penetrance of wing phenotype at 16°, minimum at 28°; TSP in larval period.

****ne*:** *nicked eye*

location: 2- (not located).

references: Kiil, 1946, DIS 20: 66.

phenotype: Eye margin nicked. Overlaps wild type. RK3.

other information: Probably an allele of *L*.

ne: see *fs(1)ne*

****Ne*:** *Nelson's mutant*

location: 3-31.

references: Gowan and Nelson, 1942, Science 96: 558-59.

Gowan, 1961, Sex and Internal Secretions (Young and Corner, eds.). The Williams and Wilkins Co., Baltimore, Vol. 1, pp. 27-28.

Belote and Lucchesi, 1980, Genetics 96: 165-86.

phenotype: Female progeny from *Ne* mothers are embryonic lethals.

***neb*:** *nebbish (M. Fuller)*

location: 2-

discoverer: Wolf, 1988.

origin: Recovered in a single P-element screen by Berg, McKearn and Spradling, but has not yet been shown to be associated with the insert.

synonym: *sl(2)ry3*.

references: Wolf and Fuller, unpublished.

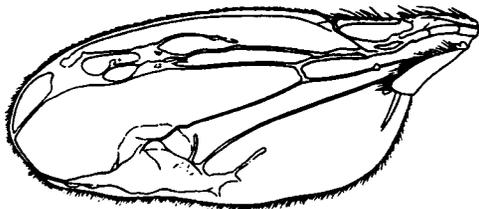
phenotype: Recessive late lethal. Some homozygotes survive until the pupal period, but very few eclose. Squash preparations of pupal testes reveal that the mitochondrial derivative (*nebenkern*) fails to elongate during spermatid differentiation. Flagellar axonemes are assembled. Onion stage early spermatids appear normal.

nec: necrotic

location: 2-{56}.

references: Ashburner.

cytology: Placed in 43A1-3.

neither inactivation nor afterpotential: see nina**Nelson's mutant: see *Ne****neshier: see nr****net: net**

Edith M. Wallace, unpublished.

net: net

location: 2-0.0.

phenotype: Wing veins form plexus-like net; first posterior cell between L3 and L4 widens toward tip; branch missing from posterior crossvein; all veins fused at base of wing, like *bi*. According to Waddington [1940, J. Genet. 41: 75-139 (fig.)], spaces form between epithelial layers owing to inadequate contraction during pupal period; spaces later fuse and form extra veins (Diaz-Benjumea, González-Gaitán, and García-Bellido, 1989, Genome 31: 612-19).

RK1.

alleles: Six net alleles [not including *Df(2L)net62*] are described in the following table.

allele	discoverer	origin	ref ^α	cytology
net¹	Bridges, 31c10	spont	1, 2, 5, 7	<i>Df(2L)21A1;21B4-5</i>
*net²	Braun, 1937	spont	1	
*net³	Williams, 56f	spont	1, 8	
*net⁴	Meyer, 56c	spont	1, 6	
net¹⁸ β		X ray	4	<i>In(2LR)21B3;42C-D1 + terminal df?</i>
net^{38j}	Ives	spont	3	

^α 1 = CP627; 2 = Golubovsky, Kulakov, and Korochkina, 1978, Genetika 14: 294-305; 3 = Ives, 1968, DIS 43: 64; 4 = Korochkina and Golubovsky, 1978, DIS 53: 197-200; 5 = Lewis, 1945, Genetics 30: 137-66; 6 = Meyer, 1956, DIS 30: 77; 7 = Waddington, 1940, J. Genet. 41: 75-139; 8 = Williams, 1956, DIS 30: 79-80.

^β Terminal deficiency tentative.

cytology: Placed in region *21A1-B5* on the basis of its association with *Df(2L)net62 = Df(2L)21A1;21B4-5*.

***neu: neuter**

location: Autosomal.

origin: Spontaneous.

discoverer: Travers, 1955.

references: Clarke, 1957, DIS 31: 80.

phenotype: Homozygous female intersex; homozygous male normal. RK3.

other information: Not an allele of *ix* (Maynard Smith).

neu: neuralised (J.C. Hall)

location: 3-50.

origin: Induced by ethyl methanesulfonate.

references: Lehmann, Dietrich, Jiménez, and Campos-Ortega, 1981, Wilhelm Roux's Arch. Dev. Biol. 190: 226-29.

Lehmann, Jiménez, Dietrich, Campos-Ortega, 1983, Wilhelm Roux's Arch. Dev. Biol. 192: 62-74.

Jürgens, Wieschaus, Nüsslein-Volhard, and Klüding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

Campos-Ortega, 1985, Trends Neurosci. 8: 245-50.

Hartenstein and Campos-Ortega, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 210-21.

phenotype: Homozygous lethal, with hyperplasia of neural components at the expense of epidermal components as seen in other neurogenic lethal mutations (*N*, *bib*, *mam*, etc.); *neu* mutations cause especially strong neural hypertrophy; also aberrant imaginal disc development when expressed in mosaic clones (Dietrich and Campos-Ortega, 1984, J. Neurogenet. 1: 315-32). *N*⁺ duplications reduce the *neu* neural hypertrophy, while *neu*⁺ duplications have no effect on *N* defects (De la Concha, Dietrich, Weigel, and Campos-Ortega, 1968, Genetics 118: 499-508).

alleles:

allele	origin	discoverer	synonym
neu¹	EMS	Nüsslein-Volhard, Wieschaus	<i>neu^{9L119}</i>
neu²	EMS	Nüsslein-Volhard, Wieschaus	<i>neu^{11B116}</i>
neu³	EMS	Nüsslein-Volhard, Wieschaus	<i>neu^{12H56}</i>
neu⁴	X ray		<i>neu^{21.1 α}</i>
neu⁵	EMS	Bremer	<i>neu^{aL119}</i>
neu⁶	EMS	De la Concha	<i>neu^{EK1}</i>
neu⁷	EMS	De la Concha	<i>neu^{EK2}</i>
neu⁸	EMS	De la Concha	<i>neu^{EK3}</i>
neu⁹	EMS	De la Concha	<i>neu^{EK4}</i>
neu¹⁰	EMS	De la Concha	<i>neu^{EK5}</i>
neu¹¹	EMS	Nüsslein-Volhard, Wieschaus	<i>neu^{IF65}</i>
neu¹²	EMS	Nüsslein-Volhard, Wieschaus	<i>neu^{IN94}</i>
neu¹³	EMS	Nüsslein-Volhard, Wieschaus	<i>neu^{III A83}</i>
neu¹⁴	EMS		<i>neu^{KE2}</i>
neu¹⁵	X ray	De la Concha	<i>neu^{KK1}</i>
neu¹⁶	X ray	Knust	<i>neu^{KK2}</i>

^α Recovered as revertant of the enhancing effect of *E(spl)* on *spl*. Does not complement other *neu* alleles.

cytology: Placed between 86C1-2 and 86D8 (Lehmann *et al.*, 1983) since it is uncovered by *Df(3R)cu = Df(3R)86C1-2;86D8* (Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91); also placed in 85C based on cytology of *In(3R)neu^{KK2} = In(3R)85C;87D5-14;90E-F* (Campos-Ortega, 1985).

neural disrupted: see nrd**neuralized: see neu**

Neurogenic-element-binding transcription factor: see Ntf

Neuroglian: see Nrg**Neuron-specific: see Nsp****neuronally-altered-carbohydrate: see nac****neuter: see *neu**

***ney: narrow eye**

location: 1- (rearrangement).
origin: X ray induced.
discoverer: Becker, 1950.
references: 1952, DIS 26: 69.
phenotype: Homozygote has narrow eyes halfway between *B* and wild type. Heterozygote usually normal. RK1A.
cytology: Associated with *In(1)ney = In(1)10A;16D*.

***ni: nicked**

location: 3-40 (35 to 45).
origin: Spontaneous.
discoverer: Neel, 41c26.
references: 1942, DIS 16: 51.
phenotype: Small notches or nicks in wing tips of 60-90% of homozygous males and 80-100% of homozygous females. RK3.

***ni2: nicked on chromosome 2**

location: 2- (not located).
origin: Spontaneous.
discoverer: Travers, 1955.
references: Clarke, 1957, DIS 31: 80.
phenotype: Wing tips deeply emarginated between L2 and L4 and occasionally between L4 and L5. Penetrance and viability good. RK3.

nicked: see *ni*

nicked on chromosome 2: see *ni2*

nicked eye: see *ne*

Nicked wing: see *Ndw*

night blind A: see *nba*

ninaA: neither inactivation nor afterpotential A
(J.C. Hall; B.H. Shieh)

location: 2-1.4.
references: Pak, Conrad, Kremer, Larrivee, Schinz, and Wong, 1980, *Development and Neurobiology of Drosophila* (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York, pp. 331-46.
 Larrivee, Conrad, Stephenson, and Pak, 1989, *J. Gen. Physiol.* 78: 521-45.
 Homyk, Pye, and Pak, 1981, *Genetics* 100: s30.
 Stephenson, O'Tousa, Scavarda, Randall, and Pak, 1983, *The Biology of Photoreceptors* (Cosens and Vince-Prue, eds.). Cambridge University Press, England, pp. 471-95.
 Zuker, Mismar, Hardy, and Rubin, 1988, *Cell* 53: 475-85.
 Shieh, Stamnes, Seavello, Harris, and Zuker, 1989, *Nature* (London) 338: 67-70.
 Schneuwly, Shortridge, Larrivee, Ono, Ozaki, and Pak, 1989, *Proc. Nat. Acad. Sci. USA* 86: 5390-94.
phenotype: Strong blue light leads to anomalously small degree of photoreceptor inactivation, and the prolonged depolarizing afterpotential PDA (seen after such treatment of wild-type photoreceptor cells) degrades rapidly following blue-light exposure reaching baseline levels within a few seconds; only the six outer photoreceptors (R1-R6) in each eye facet are aberrant physiologically (Larrivee *et al.*, 1981). Rhodopsin levels are much lower than normal in these outer cells; altered gene dosage of

ninaA⁺ does not effect changes in rhodopsin levels; *ninaA*'s mutant phenotypes are not suppressed by feeding on retinoid (*e.g.* vitamin A-enriched) media.
alleles: With respect to defective PDA, *ninaA*² > *ninaA*¹ > *ninaA*³.

allele	origin	discoverer	synonym	molecular biology
<i>ninaA</i> ¹	EMS	Pak	<i>ninaA</i> ^{P228}	Trp 208 → stop α β
<i>ninaA</i> ²	EMS	Pak	<i>ninaA</i> ^{P263}	Gln 87 → stop α
<i>ninaA</i> ³	EMS	Pak	<i>ninaA</i> ^{P269}	His 227 → Leu α
<i>ninaA</i> ⁴	EMS	Ondek		Asn 58 → Lys
<i>ninaA</i> ⁵	EMS	Ondek		Arg 60 → Pro
<i>ninaA</i> ⁶	EMS	Ondek		Arg 76 → Stop
<i>ninaA</i> ⁷	EMS	Ondek		Gly 88 → Asp
<i>ninaA</i> ⁸	EMS	Ondek		Gly 89 → Asp
<i>ninaA</i> ⁹	EMS	Ondek		Gly 89 → Ser
<i>ninaA</i> ¹⁰	EMS	Ondek		Gly 96 → Glu
<i>ninaA</i> ¹¹	EMS	Ondek		Gly 98 → Ser
<i>ninaA</i> ¹²	EMS	Ondek		Glu 110 → Lys
<i>ninaA</i> ¹³	EMS	Ondek		Lys 112 → stop
<i>ninaA</i> ¹⁴	EMS	Ondek		Gly 125 → Asp
<i>ninaA</i> ¹⁵	EMS	Ondek		Met 126 → Ile
<i>ninaA</i> ¹⁶	EMS	Ondek		Asn 128 → Tyr
<i>ninaA</i> ¹⁷	EMS	Ondek		Asn 134 → His
<i>ninaA</i> ¹⁸	EMS	Ondek		Gln 137 → His
<i>ninaA</i> ¹⁹	EMS	Ondek		Gln 137 → Leu
<i>ninaA</i> ²⁰	EMS	Ondek		Gln 137 → stop
<i>ninaA</i> ²¹	EMS	Ondek		Trp 147 → stop
<i>ninaA</i> ²²	EMS	Ondek		Gly 156 → Asp
<i>ninaA</i> ²³	EMS	Ondek		intron
<i>ninaA</i> ²⁴	EMS	Ondek		intron
<i>ninaA</i> ²⁵	EMS	Ondek		Asp 174 → stop
<i>ninaA</i> ²⁶	EMS	Ondek		Pro 179 → Leu
<i>ninaA</i> ²⁷	EMS	Ondek		Cys 188 → Tyr
<i>ninaA</i> ²⁸	EMS	Ondek		Pro 200 → Leu
<i>ninaA</i> ²⁹	EMS	Ondek		stop → Cys ?

α Schneuwly, Shortridge, Larrivee, Ono, Ozaki, and Pak, 1989, *Proc. Nat. Acad. Sci. USA* 86: 5390-94.
 β Shieh, Stamnes, Seavello, Harris, and Zuker, 1989, *Nature* 338: 67-70.

cytology: Placed in 21D4-E2, by *in situ* hybridization (Shieh *et al.*, 1989). Included in *Df(2L)ast4 = Df(2L)21D1-2;21E1-2* but not *Df(2L)ast6 = Df(2L)21E1-2;21E2-3*.

molecular biology: *ninaA* has been cloned, the nucleotides sequenced, and deduced amino acid sequences determined (Shieh *et al.*, 1989; Schneuwly *et al.*, 1989). It encodes a 237-amino acid protein with 40% sequence identity to the vertebrate cyclosporin A-binding protein, cyclophilin, that is involved in the activation of T-lymphocytes. Cyclophilin shown to be a peptidylprolyl *cis-trans*-isomerase that catalyzes rate-limiting steps in the folding of a number of proteins *in vitro*.

ninaB (J.C. Hall)

location: 3-53.5.
references: Pak, 1979, *Neurogenetics: Genetic Approaches to the Nervous System* (Breakfield, ed.). Elsevier/North-Holland, New York, pp. 67-99.
 Stephenson, O'Tousa, Scavarda, Randall, and Pak, 1982, *The Biology of Photoreceptors* (Cosens and Vince-Prue, ed.). Cambridge University Press, England, pp. 471-95.
phenotype: Superficially like *ninaA* in aberrant visual physiology, but all eight photoreceptors in each eye facet affected by *ninaB*; can be restored to wild-type phenotype by dietary supplement of retinal but not by other retinoids.

alleles:

allele	origin	synonym
ninaB¹	EMS	<i>ninaB</i> ^{P315}
ninaB²	EMS	<i>ninaB</i> ^{P319}
ninaB³	EMS	<i>ninaB</i> ^{P360}

cytology: Located in 87D14-F12 based on inclusion in *Df(3R)l26c = Df(3R)87D14-E1;87F11-12* (Kremer, Wong, Pak).

ninaC (J.C. Hall)

location: 2-22.

origin: Induced by ethyl methanesulfonate.

references: Pak, 1979, Neurogenetics; Genetic Approaches to the Nervous System (Breakfield, ed.). Elsevier/North-Holland, New York, pp. 67-99.

Stephenson, O'Tousa, Scavarda, Randall, and Pak, 1982, The Biology of Photoreceptors (Cosens and Vince-Prue, ed.). Cambridge University Press, England, pp. 471-95.

Matsumoto, Pye, Isono, and Pak, 1983, Neurosci. Abstr. 9: 325.

Matsumoto, Isono, Pye, and Pak, 1987, Proc. Nat. Acad. Sci. USA 84: 985-89.

Montell and Rubin, 1988, Cell 52: 757-72.

Stowe and Davis, 1990, Cell Tissue Res. 260: 431-34.

phenotype: Basic physiological and rhodopsin-depleted phenotypes like those of *ninaB* or *ninaD* mutants, except that amplitude of prolonged depolarizing afterpotential and rhodopsin levels are not so severely depressed, and these phenotypes cannot be rescued by vitamin A (or other retinoid) feeding. Decreased rhodopsin content is due to reduction in diameter of the rhabdomeres; the microvilli are shorter than normal and have reduced cytoskeletal electron-dense regions (Matsumoto *et al.*, 1983). In particular, the central axial filament of the microvillus is absent (*ninaC*⁵) or greatly reduced (*ninaC*², *ninaC*³). Actin immunoreactivity retained in the villi of flies homozygous for these alleles (Stowe and Davis). The mutant phenotype can be rescued using P-element mediated germ line transformation.

alleles: At least ten ethyl-methanesulfonate-induced alleles.

allele	synonym	comments
ninaC¹	<i>ninaC</i> ^{P216}	
ninaC²	<i>ninaC</i> ^{P221}	decrease in concentration and mobility of the 4.8 kb RNA
ninaC³	<i>ninaC</i> ^{P225}	decrease in concentration of the 4.8 kb RNA
ninaC⁴	<i>ninaC</i> ^{P230}	
ninaC⁵^α	<i>ninaC</i> ^{P235}	decrease in concentration of the 3.6 and the 4.8 kb RNA
ninaC⁶	<i>ninaC</i> ^{P238}	
ninaC⁷	<i>ninaC</i> ^{P239}	
ninaC⁸	<i>ninaC</i> ^{P240}	
ninaC⁹	<i>ninaC</i> ^{P262}	
ninaC¹⁰	<i>ninaC</i> ^{P266}	

^α Appears to be null allele.

cytology: Located in 28A1-3; included in synthetic deletion constructed using *T(Y;2)R147 = T(Y;2)B^SXhj;27E* and *T(Y;2)R50 = T(Y;2)h1-2;28B*.

molecular biology: The *ninaC* gene was cloned, sequenced and the eye-specific amino-acid sequences deduced (Montell and Rubin, 1988). The locus was expressed as two extensively overlapping mRNAs of 3.6

and 4.8 kb found in late pupae and adults; the RNAs code for a 132 kd protein (made up of 1135 amino acids) and a 174 kd protein (made up of 1501 amino acids) found in the rhabdomeres of the photoreceptor cells. Near the N terminus of both proteins is a putative protein kinase domain joined to a domain homologous to the globular head of the myosin heavy chain. The upstream DNA region contains an eleven-base-pair region in common with *ninaE* and *Rh2* (Mismer, Michael, Laverty, and Rubin, 1988, Genetics 120: 173-80).

other information: *ninaC* appears to be inseparable from *Sp* (2-22.0), though no *ninaC* mutations cause Sternopleural-like defects.

ninaD (J.C. Hall)

location: 2-57.

origin: Induced by ethyl methanesulfonate.

references: Pak, 1979, Neurogenetics: Genetic Approaches to the Nervous System (Breakfield, ed.). Elsevier/North-Holland, New York, pp. 67-99.

Stephenson, O'Tousa, Scavarda, Randall, and Pak, 1982, The Biology of Photoreceptors (Cosens and Vince-Prue, ed.). Cambridge University Press, England, pp. 471-95.

Johnson and Pak, 1986, J. Gen. Physiol. 88: 651-73.

phenotype: Basic physiological and rhodopsin-depleted phenotypes like those of *ninaB* mutants (*e.g.* rhodopsin levels reduced in all three classes of photoreceptors); *ninaD* mutants can be rescued by dietary supplement of vitamin A and several other retinoids. Measurements of light-induced "quantum bumps" (the basic "units" of the photoreceptor potential) in *ninaD*² (rhodopsin content, 10⁻² X wild-type) showed these responses to be basically normal (implying that, since extensive inter-rhodopsin molecular interactions are likely to be extremely rare, such interactions are not necessary for generation and adaptation of the bumps); yet, bump amplitudes approximately 4X normal (Johnson and Pak, 1986).

alleles: Five ethyl-methanesulfonate-induced alleles.

allele	synonym
ninaD¹	<i>ninaD</i> ^{P245}
ninaD²	<i>ninaD</i> ^{P246}
ninaD³	<i>ninaD</i> ^{P258}
ninaD⁴	<i>ninaD</i> ^{P261}
ninaD⁵^α	<i>ninaD</i> ^{TH382}

^α Smith, Shieh, and Zuker, 1990, Proc. Nat. Acad. Sci. USA 87: 1003-07.

cytology: Placed in 36D1-E4 on the basis of its inclusion in *Df(2L)H20 = Df(2L)36A8-9;36E3-4* and *Df(2L)M-HS5 = Df(2L)36D1-E1;36F1-37A1*.

other information: Not the same gene as the very closely linked *Arr1* (which produces a "phototransduction protein"); this was shown by sequencing coding exons of genomic DNA from *ninaD*², *ninaD*³, and *ninaD*⁵, all of which exhibited wild-type arrestin sequences in these ORFs (Smith *et al.*, 1990).

ninaE (J.C. Hall)

location: 3-66.4.

synonym: *Rh1*.

references: Pak, 1979, Neurogenetics: Genetic Approaches to the Nervous System (Breakfield, ed.). Elsevier/North Holland, New York, pp. 67-99.

Scavarda, O'Tousa, and Pak, 1983, Proc. Nat. Acad. Sci. USA 80: 4441-45.

O'Tousa, Baehr, Martin, Hirsh, Pak, and Applebury, 1985, Cell 40: 839-50.
 Zuker, Cowman, and Rubin, 1985, Cell 40: 851-58.
 Pollack and Benzer, 1988, Nature (London) 333: 779-82.
 Johnson and Pak, 1986, J. Gen. Physiol. 88: 651-73.
 Stark and Sapp, 1987, J. Neurogenet. 4: 227-40.
 Zuker, Mismar, Hardy, and Rubin, 1988, Cell 55: 475-82.
 O'Tousa, Leonard, and Pak, 1989, J. Neurogenet. 6: 41-52.
 Washburn and O'Tousa, 1989, J. Biol. Chem. 264: 15464-66.

phenotype: *ninaE*⁺ encodes the opsin moiety of the major rhodopsin, RH1, which occupies the rhabdomeres of the outer six photoreceptor cells R1-R6 in each ommatidium of the adult fly. This rhodopsin is also expressed in the larval light sensitive organs (Zucker *et al.*, 1985; Pollack and Benzer, 1988). RH1 is a 39 kd basic protein (Pak and Nichols, 1985, J. Biol. Chem. 260: 12670-74). Homozygous *ninaE* mutants display severe depletion of rhodopsin from the outer photoreceptors, shown microspectrophotometrically and physiologically (Scavarda *et al.*, 1983; Johnson and Pak, 1986; also see below), as well as by absence of R1-6 staining with an anti-(*Drosophila*)rhodopsin MAb (de Couet and Tanimura, 1987, Eur. J. Cell Biol. 44: 50-56). Electroretinograms demonstrate that the prolonged depolarizing afterpotential (PDA) is absent; also, the sustained corneal-negative light-coincident response is reduced in some alleles and nearly wild type in amplitude in others. Physiological measurements of light-induced "quantum bumps" in three *ninaE* mutants (whose RH1 decrements range from 10⁻² to 10⁻⁶ of wild-type) indicate that these responses—at the level of a given bump—are basically normal (implying that interactions among rhodopsin molecules are not likely to be critical for generation and adaptation of these "basic units" of photoreceptor potential (Johnson and Pak, 1986); bump amplitudes were higher than normal (more so in the more severe of the three mutants). Increased and decreased dosages of *ninaE*⁺ cause higher than normal and lower than normal rhodopsin levels (Scavarda *et al.*, 1983). Some mutants, when heterozygous to wild type, show less than 50% of the normal rhodopsin level (e.g., *ninaE*^{7/+} yields 35% of the normal level); in heterozygotes of *ninaE*⁵, *ninaE*⁶, and *ninaE*⁷, the basic photoreceptor potential, as seen in electroretinograms, may be reduced. In mutant homozygotes, the cross-sectional area of rhabdomeres 1-6 is smaller than normal; in some mutants (*ninaE*¹, *ninaE*³, *ninaE*⁷, and *ninaE*⁸), an age dependent, light-independent degeneration of R1-6 rhabdomeres (but not cell bodies) is observed; in the case of severe alleles like *ninaE*¹ (see other information) or *ninaE*¹⁷, the rhabdomeres are present at eclosion, but degenerate rapidly thereafter (e.g., Stark and Sapp, 1987, O'Tousa *et al.*, 1989); degeneration is cell autonomous in mosaics (Stark, Srygley, and Greenberg, 1981, DIS 56: 132-33).

allele	origin	discoverer	synonym	ref ^α	comments
<i>ninaE</i> ¹	EMS	Koenig	<i>ninaE</i> ^{ora}	2, 4, 8	induced with <i>ort</i> ¹ as <i>ora</i> ^{JK84} ; ca 10 ⁻⁶ normal RH1 level; Gin ²⁵¹ → stop
<i>ninaE</i> ²	spont	Engles	<i>ninaE</i> ^{ENG}	3, 5	4.2 kb insert; <1% normal transcript level
<i>ninaE</i> ³	EMS	Pak	<i>ninaE</i> ^{P223}	9	

allele	origin	discoverer	synonym	ref ^α	comments
<i>ninaE</i> ⁴	EMS	Pak	<i>ninaE</i> ^{P228}	9	
<i>ninaE</i> ⁵	EMS	Pak	<i>ninaE</i> ^{P318}	6, 8	3-13% normal RH1 level; normal transcript level; Pro ¹²⁰ → Leu
<i>ninaE</i> ⁶	EMS	Pak	<i>ninaE</i> ^{P322}	7	RH1 undetectable
<i>ninaE</i> ⁷	EMS	Pak	<i>ninaE</i> ^{P332}	3, 6, 7, 8	<2% normal RH1 level; normal transcript level; Gly ¹²⁸ → Arg
<i>ninaE</i> ⁸	EMS	Pak	<i>ninaE</i> ^{P334}	3, 7, 8	<1% normal RH1 level; normal transcript level; Thr ²⁸³ → Met; Trp ²⁸⁹ → Arg; Cys ²⁹⁷ → Ser
<i>ninaE</i> ^{9β}	EMS	Pak	<i>ninaE</i> ^{P352}	1, 8	ca 10 ⁻⁶ normal RH1 level; Gin ²⁵¹ → stop
<i>ninaE</i> ¹⁰	EMS	Pak	<i>ninaE</i> ^{P353}		
<i>ninaE</i> ¹¹	EMS	Pak	<i>ninaE</i> ^{P354}		
<i>ninaE</i> ¹²	EMS	Pak	<i>ninaE</i> ^{P361}		
<i>ninaE</i> ¹³	EMS	Pak	<i>ninaE</i> ^{P362}		
<i>ninaE</i> ¹⁴	EMS	Hardy, Orevi	<i>ninaE</i> ^{RH7}		
<i>ninaE</i> ¹⁵	EMS	Hardy, Orevi	<i>ninaE</i> ^{RH88}		
<i>ninaE</i> ¹⁶	EMS	Hardy, Orevi	<i>ninaE</i> ^{US62}		
<i>ninaE</i> ¹⁷	γ ray	O'Tousa	<i>ninaE</i> ^{O117}	4	intragenic deletion

^α 1 = Johnson and Pak, 1986, J. Gen. Physiol. 88: 651-73; 2 = Koenig and Merriam, 1977, DIS 52: 50-51; 3 = Leonard and Pak, 1984, Neurosci. Abstr. 10: 1032; 4 = O'Tousa, Leonard, and Pak, 1989, J. Neurogenet. 6: 41-52; 5 = O'Tousa, Baehr, Martin, Hirsh, Pak, and Applebury, 1985, Cell 40: 839-50; 6 = Scavarda, O'Tousa, and Pak, 1981, Neurosci. Abstr. 7: 61; 7 = Scavarda, O'Tousa, and Pak, 1983, Proc. Nat. Acad. Sci. USA 80: 4441-45; 8 = Washburn and O'Tousa, 1989, J. Biol. Chem. 264: 15464-66; 9 = Zuker, Mismar, Hardy, and Rubin, 1988, Cell 55: 475-82.

^β Same nonsense mutation and polymorphisms as *ninaE*¹ (Washburn and O'Tousa, 1989).

cytology: Placed in 92B6-7 based on its inclusion in *Df(3R)jo11 = Df(3R)92B5-6;92B7-8* (O'Tousa *et al.*, 1985).

molecular biology: Genomic clone isolated by cross homology to a bovine opsin-encoding cDNA (O'Tousa *et al.*, 1985; Zuker *et al.*, 1985). Opsin-encoding subclone identified by transformation of *ninaE* mutant homozygotes. Nucleotide sequence contains an open reading frame interrupted by four introns with consensus splicing sequences; the conceptual amino acid sequence, which comprises 373 amino acids, indicates the presence of seven putative membrane-spanning domains and several glycosylation sites. Probing of northern blots reveals the presence of an 1.5-1.7 kb eye-specific transcript, which is abundant throughout the depth of the retina (Pollock and Benzer, 1988). Three separable *cis*-acting control regions in the *ninaE* promoter extending from -120 to +61 have been identified by Mismar and Rubin (1987, Genetics 116: 565-78), the first two appearing to be quantitative regulatory elements, the third affecting tissue-specificity of the promoter. There is an eleven-base-pair sequence in common with *ninaC* and *Rh2* (Mismar, Michael, Laverty, and Rubin, 1988, Genetics 120: 173-80). The first two regions have the properties of enhancers required for normal promoter expression (Mismar and Rubin, 1989, Genetics 121: 77-87), although they do produce a *ninaE* expression pattern in a truncated *Hsp70* promoter. As is the case for other opsins, putative phosphorylation sites are located near the C terminus of *Drosophila* RH1 protein; however, removal of the relevant Ser and Thr residues by site-specific mutagenesis such that a premature termination codon is placed at amino-acid position 356, and transformation of

that construct into a *ninaE*⁻ host (Ozaki, Zuker, Pak, and Rubin, 1986, Neurosci. Abstr. 12: 639) led to light-induced physiological response of R1-6 indistinguishable from those of fully rescued control transformants (Zuker *et al.*, 1988). The amount of rhodopsin phosphorylation in the engineered mutant was approximately half normal.

other information: In one clean use of a *ninaE* variant to eliminate responses of R1-6 photoreceptors, turn-on of *per* gene expression in nuclei of such cells (which requires exposures of the flies to light-dark transitions) was normal in *ninaE*¹⁷ (Zerr, Hall, Rosbash, and Siwicki, 1990, J. Neurosci. 10: 2749-62). A number of studies of this general sort have been carried out on the double mutant, *ort ninaE* (recovered as *ora*^{JK84}); *ort* by itself is known to cause deficits in ERG light-on and light-off transient spikes (O'Tousa *et al.*, 1989). The application of "*ora*" have usually been aimed at using it as a R1-6-removing tool for behavioral (*e.g.*, Coombe, 1984, J. Comp. Physiol. 155: 661-72) or physiological (*e.g.*, Stark, Schilly, Christianson, Bone, and Landrum, 1990, J. Comp. Physiol. 166: 429-36) experiments. Many of the abnormalities, such as assessments of visual pigment content (most classically, Harris, Stark, and Walker, 1976, J. Physiol. 256: 415-39) are probably attributable to the *ninaE* component only; this includes an explicit demonstration that rhabdomere degeneration (Stark and Sapp, 1987, J. Neurogenet. 4: 227-40) is caused by *ninaE* (O'Tousa *et al.*, 1989), similar to that caused by any other severe *ninaE* mutation. But certain effects of "*ora*" on visually mediated behaviors, such as decrements in male courtship (Markow and Manning, 1980, Behav. Neurobiol. 29: 276-80), the absence of blue-light influenced phototaxis (Willmund and Fischbach, 1977, J. Comp. Physiol. 118: 261-71), or the absence of R1-6-dependent optomotor responses (Heisenberg and Buchner, 1977, J. Comp. Physiol. 117: 127-62) could be affected by both factors.

nj42: see *mys*⁸

nj156: see *shakB*

nj522: see *gfa*

NK1 (M. Nirenberg)

location: 3-{72}.

references: Kim and Nirenberg, 1989, Proc. Nat. Acad. Sci. USA 86: 7716-20.

phenotype: Homeobox gene expressed in embryos in several types of striated muscle cells and in some cells in the ventral nervous system. Fewer transcripts were detected in the central nervous systems (CNS) of larvae, pupae, and adults. No transcripts were detected in 0-3 hour embryos.

cytology: Located at 93E3-5 close to *NK3* and *NK4*.

molecular biology: Sequence analysis of cloned cDNA and genomic DNA (7,609 nonoverlapping bp) revealed four exons. One major species of poly A⁺ RNA from embryos 2.9 kb in length was detected. NK1 protein contains 661 amino acid residues and it is rich in S, A, P, and H. The amino acid sequence of the homeobox is closely related to the H-40 homeobox of the honeybee, *Apis mellifera* (Walldorf, Fleig, and Gehring, 1989, Proc. Nat. Acad. Sci. USA 86: 9971-75) and chicken CHox3 homeobox (Rangini *et al.*, 1989, Gene 76: 61-74). NK1 protein contains alternating repetitive, H/P residues (a

paired repeat similar to those of *prd* and *bcd* proteins), and contains regions rich in H/Q, S/P, S/T, repetitive A, and repetitive G residues. NK1 also contains an acidic domain before the homeobox. Intron 3 is located within the homeobox between codons for homeobox amino acid residues 44 and 45. Homeobox genes cloned from monkey and rat DNA contain homeobox amino acid sequences identical to that of NK1 (52 amino acid residues compared).

NK2 (M. Nirenberg)

location: 1-{0.0}.

references: Kim and Nirenberg, 1989, Proc. Nat. Acad. Sci. USA 86: 7716-20.

Nakayama, Nakayama, Kim, and Nirenberg, unpublished.

phenotype: *NK2* is a homeobox gene whose expression starts at the blastoderm stage during cell membrane formation (2.5-3.25-hour embryos) in the region that gives rise to the ventral nervous system. *NK2* transcripts detected in the ventral nervous system at later stages of embryonic development. *NK2* transcripts also found in the midgut starting at 12 hours of embryonic development and thereafter during embryonic development. *NK2* mRNA also found in larvae and pupae but the abundance of the mRNA is lower than that of embryos. *NK2* mRNA is expressed in the central nervous system of the adult.

cytology: Located at 1C.

molecular biology: Sequence analysis of *NK2* cDNA and part of the cloned genomic DNA (3033 nonoverlapping bp) showed that *NK2* is a homeobox gene with two exons. *NK2* is a basic protein with 723 amino acid residues and relatively high levels of A, S, H, G, and P with localized regions of alternating acidic and basic amino acid residues, and with regions of abundant S/T and P, or H, Q, and P, with repetitive A residues, pest sequences, an acidic domain, and an alternating H/A repeat. Northern analysis revealed one species of poly A⁺ RNA 3.5 kb in length.

NK3 (M. Nirenberg)

location: 3-{72}.

references: Kim and Nirenberg, 1989, Proc. Nat. Acad. Sci. USA 86: 7716-20.

Webber, Kim, Guo, and Nirenberg, unpublished.

phenotype: Homeobox gene expressed transiently during embryonic development in some mesodermal cells (foregut and hindgut visceral muscle cells). *NK3* poly A⁺ RNA was not detected in 0-3-hour embryos, but was found in low abundance in RNA from 3-6-hour embryos. The abundance was maximum in RNA from 6-12-hour embryos and then declined during further embryonic development.

cytology: Located at 93E1-3 close to homeobox genes *NK4* and *NK1*.

molecular biology: Genomic DNA fragments were cloned that contain both *NK3* and *NK4* homeobox genes. *NK3* and *NK4* homeobox regions are separated by approximately 7.5 kb in genomic DNA. Sequence analysis of cloned *NK3* cDNA and genomic DNA revealed two exons separated by a short intron. *NK3* protein consists of 374 amino acid residues and contains S/T rich regions and several regions rich in acidic and basic amino acid residues. Northern analysis revealed one species of *NK3* poly A⁺ RNA, approximately 1.5 kb in length.

NK4 (M. Nirenberg)

location: 3-{72}.

references: Kim and Nirenberg, 1989, Proc. Nat. Acad. Sci. USA 86: 7716-20.

Rajni, Kim, Guo, and Nirenberg, unpublished.

phenotype: The *NK4* homeobox gene is first expressed in 3-hour embryos; no *NK4* mRNA is detected in embryos at earlier stages of development. *NK4* mRNA is detected only in mesodermal cells. The transcripts are most abundant in 3-9-hour embryos; the mRNA decreases in abundance thereafter.

cytology: Located at 93E1-3 close to homeobox genes *NK3* and *NK1*.

molecular biology: The homeobox sequences of *NK4* and *NK3* are separated in genomic DNA by approximately 7.5 kb. Sequence analysis of cloned *NK4* cDNA and genomic DNA revealed three exons. *NK4* is a basic protein (41,192 M_r) with 371 amino acid residues, and has relatively high levels of S, Q, A, and P. *NK4* protein contains a homeobox that is not closely related to any known homeobox protein, contains pest sequences, Q repeats, and regions with abundant H and Q, or S, T, and P. One major species of poly A⁺ RNA was detected, 1.7 kb in length.

nkd: naked cuticle

location: 3-47.3.

origin: Induced by ethyl methanesulfonate.

references: Jürgens, Kluding, Nüsslein-Volhard, and Wieschaus, 1983, DIS 59: 157-58.

Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

Arias, Baker, and Ingham, 1988, Development 103: 157-70.

phenotype: Denticle bands partially deleted. Embryonic lethal. Terminal phenotype partly due to cell death after germ band shortening (Martinez-Arias).

alleles: Six ethyl-methanesulfonate-induced alleles.

allele	synonym	ref ^α	comments
<i>nkd</i> ¹	<i>nkd</i> ^{6J}	2	weak allele
<i>nkd</i> ²	<i>nkd</i> ^{7E}	2	strong allele
<i>nkd</i> ³	<i>nkd</i> ^{7H}	2	strong allele
<i>nkd</i> ⁴	<i>nkd</i> ^{9G}	2	
<i>nkd</i> ⁵	<i>nkd</i> ^{9H}	2	
<i>nkd</i> ⁶	<i>nkd</i> ^{YE88}	1	

^α 1 = Carrol and Scott, 1986, Cell 45: 113-26; 2 = Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

cytology: Located in 75D-76B; uncovered by *T(Y;3)LI31^DLI4^P* = *T(Y;3)75D1;76B5-10* (Nüsslein-Volhard).

nmd: no mitochondrial derivative (M. Fuller)

location: 2-.

discoverer: Wolf, 1988.

origin: Recovered in a single P-element screen by Berg, McKearn and Spradling. The male sterile mutation has not yet been shown to be associated with the insert.

synonym: *ms(2)ry4*.

references: Wolf, Madder, Bonneville and Fuller, unpublished.

phenotype: Recessive male sterile. Onion stage early spermatids in homozygous males either lack or have only tiny mitochondrial derivatives. Flagellar axonemes

elongate without mitochondrial derivatives. Mitochondria present in polar primary spermatocytes, but degenerate prior to meiosis in mature primary spermatocytes. Females are fertile.

cytology: The transposable element is inserted at 31.

no action potential: see *nap*^{ts}

no bridge: see *nob*

no distributive disjunction: see *nod*

no mitochondrial derivative: see *nmd*

no object fixation: see *nof*

no ocelli: see *noc*

no ocelli; narrow eyes: see *none*

no on-transient: see *non*

no receptor potential A: see *norpA*

no wings: see *ap*³

nob: no bridge (J.C. Hall)

location: 1-12.

origin: Induced by ethyl methanesulfonate.

discoverer: Heisenberg.

references: Heisenberg, Borst, Wagner, and Byers, 1985, J. Neurogenet. 2: 1-20.

Bouhouche and Vaysse, 1991, J. Neurogenet.

phenotype: Protocerebral bridge between left and right sides of adult brain seems disintegrated into two or more glomeruli; fiber number in interhemispheric commissure of white pupa reduced; learning is poor in "arena paradigm" tests of adults, using sugar and odorants (Heisenberg *et al.*, 1985); larval learning test using electric shocks and odorants gave approximately half the normal score (Heisenberg, unpublished). Locomotor activity of mutant adults is abnormal (spontaneous walking speed, regarding forward strides of legs, is slow), and in flight, there is reduced frequency of flight starts, altered "object response" and reduced occurrences of "body saccades" (Heisenberg). Adults exhibit aberrant habituation of proboscis-extension reflex (Bouhouche and Vaysse, 1991).

alleles: One mutant allele, called *nob*^{KS49}.

cytology: Placed in 4F5-12 (Heisenberg).

noc: no ocelli

location: 2-{50}.

discoverer: Ashburner.

references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

Ashburner, Aaron, and Tsubota, 1982, Genetics 102: 421-35.

Ashburner, Tsubota, and Woodruff, 1982, Genetics 102: 401-20.

Ashburner and Harrington, 1984, Chromosoma 89: 329-37.

Chia, Carp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706.

Gubb, Roote, Harrington, McGill, Durrant, Shelton, and Ashburner, 1985, Chromosoma 92: 116-23.

McGill, Chia, Karp, and Ashburner, 1988, Genetics 119: 647-61.

phenotype: The genetic complexity of *noc* is indicated by phenotypic as well as molecular data (McGill *et al.*, 1988). Mutants are characterized by partial or complete absence of ocelli and their associated bristles. Strong *noc* alleles lack all three ocelli and have fewer interocellar microchaetae; ocellar and interocellar bristles are absent; anterior postalar and notopleural bristles may not be present; postvertical bristles are crooked, an adventitious pair often occurring between a normal pair. Weak alleles may overlap wild type in phenotype, the flies having smaller ocelli and an aberrant pattern of interocellar microchaetae. Male and female homozygotes are viable and fertile. Penetrance and expressivity is stronger in males and in flies that emerge later. While a homozygous deletion of the entire *noc* region is lethal, distal or proximal regions can be homozygously deleted without causing lethality. Some *noc* alleles and deficiencies are lethal with *In(2L)Sco^{rv1}*, whose 2L breakpoint lies within *noc*, while other alleles are viable with the *Sco* inversion.

alleles: Mutants and rearrangements are listed in the table. *noc* deficiencies are described in the rearrangement section.

allele	origin	discoverer	ref ^α	phenotype	cytology	mol. biol ^β
<i>noc</i> ²	EMS	Tsubota	1, 2, 5-8	weak	<i>In(2L)35B1-2</i> ; 36D3	-62 to -68
<i>noc</i> ³	γ ray	Tsubota	2, 5-7	very weak		
<i>noc</i> ⁴ γ	γ ray	Harrington	1, 5-8	strong	<i>In(2LR)35B1-2</i> ; 41	-109 to -117
<i>noc</i> ⁶ γ	γ ray	Harrington	7	strong		
<i>noc</i> ⁷ γδ	γ ray	Harrington	1, 6-8	weak	<i>In(2LR)35A1-4</i> ; 40	-77 to -80
<i>noc</i> ¹⁴ γ	γ ray	Harrington	7	weak		
<i>noc</i> ¹⁵	γ ray	Harrington	7	weak		
<i>noc</i> ¹⁸	EMS	Spoerel	5, 7	weak		
<i>noc</i> ¹⁹ γ	EMS	Spoerel	5, 7	weak		
<i>noc</i> ²¹	γ ray	McGill		strong		
<i>noc</i> ^{56.1}	EMS					
<i>noc</i> ^{56.3}	EMS					
<i>noc</i> ^{TE35A}	spont	Ising	2-4, 5-8	very strong	<i>TE35A</i> insertion ^ε	-108
[<i>Sco</i>]	X ray	Krivshenko	3, 8	antimorph of <i>noc</i> ? ^ζ	<i>Tp(2;2)</i>	-24.6 to -107.8

^α 1 = Ashburner, Aaron, and Tsubota, 1982, *Genetics* 102: 421-35; 2 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, *Dis* 56: 186-91; 3 = Ashburner, Detwiler, and Woodruff, 1983, *Genetics* 104: 405-31; 4 = Ashburner and Harrington, 1984, *Chromosoma* 89: 329-37; 5 = Ashburner, Tsubota, and Woodruff, 1982, *Genetics* 102: 401-20; 6 = Chia, Carp, McGill, and Ashburner, 1985, *J. Mol. Biol.* 186: 689-706; 7 = Gubb, Roote, Harrington, McGill, Durrant, Shelton, and Ashburner, 1985, *Chromosoma* 92: 116-23; 8 = McGill, Chia, Karp, and Ashburner, 1988, *Genetics* 119: 647-61.

^β From the molecular map of the wild-type *noc-Adh* region (McGill *et al.*, 1988); coordinate 0 an *EcoRI* restriction site 1321 bp to the left of the start of transcription of the larval *Adh* transcript; "+" values to the right, "-" values to the left.

^γ Lethal or semilethal with *In(2LR)Sco^{rv}*.

^δ Inversion in *T(Y;2)60D7-F*.

^ε Synonym: *TE146*.

^ζ Phenotype of *Sco* enhanced by deletion and suppressed by duplication of the *noc* gene (McGill *et al.*, 1988).

cytology: Placed in 35B1-2 on the basis of its inclusion in *Df(2L)A178 = Df(2L)35B1-2;35B2-3* but not in *Df(2L)A48 = Df(2L)35B2-3; 35D5-7*.

molecular biology: The DNA region associated with *noc* can be defined by the mapping of breakpoints of *noc* mutants which locate *noc* approximately between -117 and -62 kb on the molecular map of the wild-type *noc* -

Adh region (McGill *et al.*, 1988). If, however, *Sco* (a rearrangement in which the *noc* gene has been transposed from 35B to 35D) is considered to be an antimorphic allele of *noc* and is included in the locus, the *noc* region will be enlarged and will be located approximately between -117 and -24.6 kb. This region, almost 100 kb in estimated total length, is not thought to be continuous, but to be functionally divided into at least three components, the two distal ones (*nocA* and *nocB*) resulting in a mutant phenotype when deleted and the proximal one (*nocC*) resulting in a *noc*⁺ phenotype when deleted (McGill *et al.*, 1988). Breakpoints of *Sco* revertants are only found in *nocB* and *nocC*. *Tp(2;3)Mpe*, which shows a *noc*⁺ rather than a *noc* phenotype over deletions for the entire *noc* locus, has its 2L breakpoint between *nocA* and *nocB* (McGill *et al.*, 1988).

nod: no distributive disjunction (R.S. Hawley)

location: 1-36.

references: Baker and Carpenter, 1972, *Genetics* 71: 255-86.

Carpenter, 1973, *Genetics* 73: 393-428.

Wright, 1973, *Mol. Gen. Genet.* 122: 101-18.

Baker and Hall, 1976, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 352-434.

Baker, Carpenter, and Ripoll, 1978, *Genetics* 90: 531-78.

Zhang and Hawley, 1990, *Genetics* (submitted).

phenotype: Females homozygous for *nod* alleles exhibit high frequencies of meiotic chromosome loss and nondisjunction at meiosis I. Most *nod*-induced nondisjunctional events involve nonexchange chromosomes. For example, in *nod/nod* females nondisjunction frequencies for the always nonexchange fourth chromosomes approaches 90% (the vast majority of gametes are nullo-4 ova), whereas nonexchange X chromosomes apparently disjoin at random. Both the frequency of exchange and the disjunction of exchange bivalents was shown to be normal in *nod/nod* females. Thus, with respect to its role in meiosis, the *nod*⁺ function appears to be limited to the distributive segregation system. Based on an analysis of secondary nondisjunction in *nod^a/nod^a* females, Carpenter concluded that the *nod* defect does not impair the process of partner choice within the distributive system, but rather specifically impairs the disjunctional process. Nonexchange chromosomes derived from *nod^a/nod^a* mothers also undergo nondisjunction, and presumably loss, at meiosis II. In addition, chromosomes derived from *nod^a/nod^a* mothers are mitotically unstable. *nod*-induced mitotic chromosome loss is restricted to maternal nonexchange chromosomes and does not exert any discernible effect on meiosis in males or on mitotic chromosome stability (Baker *et al.*, 1978). Although none of the existing *nod* alleles is lethal or female sterile, the dosage-sensitive antimorphic mutation *l(1)TW6* (Wright, 1973) is argued to be allelic to *nod* on the basis of three lines of evidence. First, *l(1)TW6/+* females display a meiotic phenotype that is virtually identical to that exhibited by *nod^a/nod^a* females. Second, the two loci map to the same position on the X chromosome (Wright, 1973; Baker). Third, a γ-ray induced revertant of *l(1)TW6* was shown to be a recessive *nod* allele (New and Hawley).

alleles:

allele	origin	synonym	ref ^α	comments
nod^a	EMS	<i>mei254</i>	1, 2	
nod¹	γ rays	<i>nod^{b1}</i>	4	induced in <i>FM7a</i>
nod²	γ rays	<i>nod^{b9}</i>	4	induced in <i>FM7a</i>
nod³	γ rays	<i>nod^{b17}</i>	4	induced in <i>FM7a</i>
nod⁴	γ rays	<i>nod^{b27}</i>	4	induced in <i>FM7a</i>
nod⁵	γ rays	<i>nod^{b29}</i>	4	induced in <i>FM7a</i>
nod⁶	γ rays	<i>nod^{b34}</i>	4	induced in <i>FM7a</i>
nod⁷	γ rays	<i>nod^{bd}</i>	4	induced in <i>FM7a</i>
				Associated with <i>In(1)10C;15D-E</i>
nod⁸	EMS	<i>l(1)TW6</i>	3	

^α 1 = Baker and Carpenter, 1972, *Genetics* 71: 255-86; 2 = Carpenter, 1973, *Genetics* 73: 393-428; 3 = Wright, 1973, *Mol. Gen. Genet.* 122: 101-18. 4 = Zhang and Hawley, 1990, *Genetics* (submitted);

cytology: Placed in 10C2-3 by deficiency mapping.

molecular biology: *nod* cloned and sequenced (Zhang, Knowles, Goldstein, and Hawley, 1990, *Cell* 62: 1053-62). 2.4 kb transcript found in meiotically-active ovaries; not found in males. N-terminal domain of predicted protein shows amino acid similarity to the mechanochemical domain of kinesin heavy chain.

nofA: no object fixation A (J.C. Hall)

location: 1- (not localized).

origin: Induced by ethyl methanesulfonate.

synonym: *S100*.

references: Götz, 1980, *Development and Neurobiology of Drosophila* (Siddiqi, Babu, Hall, and Hall, ed.). Plenum Press, New York, pp. 391-407.

Bülthoff, 1982, *DIS* 58: 31.

1982, *Biol. Cybernet.* 45: 63-70.

1982, *Biol. Cybernet.* 45: 71-77.

phenotype: Poor orientation in tests involving fixation on objects or movements to and from in a "choice" experiment between two identical objects (*i.e.*, flies in an arena). Normal response to spots in a Y-maze test. Mutants make rapid continuous changes in their visual gaze instead of sporadic changes as in wild type.

alleles: One mutant allele, called *nofA^{S100}* (Heisenberg and Wolf, 1979, *J. Comp. Physiol.* 130: 113-30).

nofC: see *norpA*

nofD (J.C. Hall)

location: 1- (not localized).

origin: Induced by ethyl methanesulfonate.

references: Bülthoff, 1982, *DIS* 58: 31.

1982, *DIS* 58: 32-33.

1982, *Biol. Cybernet.* 45: 63-70.

1982, *Biol. Cybernet.* 45: 71-77.

phenotype: Poor orientation to objects, including spots in Y-maze test; electroretinogram normal.

alleles: One mutant allele, *nofD^{B11}*.

nofE: see *sol¹⁶*

nofF (J.C. Hall)

location: 1- (not localized).

origin: Induced by ethyl methanesulfonate.

references: Bülthoff, 1982, *DIS* 58: 31.

1982, *Biol. Cybernet.* 45: 63-70.

phenotype: Same phenotype as *nofD* or *nofE*.

alleles: One mutant allele, *nofF^{S71}*.

nofG (J.C. Hall)

location: 1- (not localized).

origin: Induced by ethyl methanesulfonate.

references: Bülthoff, 1982, *DIS* 58: 31.

phenotype: Same basic phenotype as *nofA* in relation to visual objects. Electroretinogram normal.

alleles: One mutant allele, *nofG^{S13}*.

nofI (J.C. Hall)

location: 1- (not localized).

origin: Induced by ethyl methanesulfonate.

references: Bülthoff, 1982, *DIS* 58: 32-33.

1982, *Biol. Cybernet.* 45: 63-70.

phenotype: Poor orientation to spots in Y-maze test. Eye color brownish; screening pigment in photoreceptors disrupted. Irregularities in pattern of rhabdomere endings. Electroretinogram normal.

alleles: One mutant allele, *nofI^{B3}*.

nofK (J.D. Hall)

location: 1- (not localized).

origin: Induced by ethyl methanesulfonate.

references: Bülthoff, 1982, *DIS* 58: 32-33.

1982, *Biol. Cybernet.* 45: 63-70.

phenotype: Same phenotype as *nofI*.

alleles: One mutant allele, *nofK^{B6}*.

nofL (J.C. Hall)

location: 1- (not localized).

origin: Induced by ethyl methanesulfonate.

references: Bülthoff, 1982, *DIS* 58: 32-33.

1982, *Biol. Cybernet.* 45: 63-70.

phenotype: Same phenotype as *nofI*.

alleles: One mutant allele, *nofL^{B7}*.

non-claret disjunctional: see *ncd*

nonA: no on or off transient-A (J.C. Hall)

location: 1-52.3 (Pak); 1-56 (Heisenberg) (the former location more consistent with Kulkarni *et al.*, 1988, and with cytology).

origin: Induced by ethyl methanesulfonate.

synonym: *Positive spike II* group (Benzer); *x14* (Pak), *opm2* (Heisenberg).

references: Hotta and Benzer, 1970, *Proc. Nat. Acad. Sci. USA* 67: 1156-63.

Pak, Grossfield, and Arnold, 1970, *Nature* 227: 518-20.

Heisenberg, 1971, *DIS* 46: 68.

1972, *J. Comp. Physiol.* 80: 119-36.

Heisenberg and Götz, 1975, *J. Comp. Physiol.* 98: 217-41.

Heisenberg and Buchner, 1977, *J. Comp. Physiol.* 117: 127-62.

Bülthoff, 1982, *Biol. Cybernet.* 45: 63-70, 71-77.

Kulkarni, Steinlauf, and Hall, 1988, *Genetics* 118: 267-85.

Wheeler, Kulkarni, Gailey, and Hall, 1989, *Behav. Genet.* 19: 503-28.

Jones and Rubin, 1990, *Neuron* 4: 711-23.

Besser, Schnabel, Wieland, Fritz, Stanewsky, and Saumweber, 1990, *Chromosoma*, in press.

phenotype: Defective optomotor responses and phototaxis, with the former being especially defective (Heisenberg, 1972; Heisenberg and Buchner, 1977; Kulkarni *et al.*, 1988). Poor orientation behavior in Y-maze (Bülthoff, 1982a, b). *nonA⁵* flies exhibit specific lack of responses

to front-to-back moving visual stimuli, whereas reaction to back-to-front motion is intact (Heisenberg, 1972). Physiologically, there are reduced or absent light-on and light-off transient spikes in electroretinogram, whereas photoreceptor potential is normal (Hotta and Benzer, 1970; Pak *et al.*, 1970; Heisenberg, 1971; Kulkarni *et al.*, 1988; Jones and Rubin, 1990). Larval visual response (*re* negative phototaxis) normal [Hotta and Keng, 1984, Animal Behavior: Neurophysiological and Ethological Approaches (Aoki *et al.*, eds.). Springer-Verlag, Berlin, pp. 49-60]. Courtship song is abnormal, as influenced by one, possibly two, mutant alleles; *nonA*⁹ (originally *diss*) males produce abnormal song, regarding pulses relatively late in trains of such song sounds (each one resulting from a bout of wing vibration); the abnormalities are polycyclicity (Kulkarni *et al.*, 1988) and anomalous intra-pulse frequency components (Wheeler *et al.*, 1989); pulses early in trains, or throughout short ones, are nearly normal; courtship hum sounds manifest irregular sine waves, though their fundamental frequencies are normal (Wheeler *et al.*, 1989). *nonA*⁹ by itself is also an optomotor-defective/ERG-abnormal mutant (Kulkarni *et al.*, 1988) and fails to complement other mutant alleles with regard to these visual phenotypes (Rendahl, Kulkarni, and Hall, unpublished); but these heterozygotes, in a homozygous *tra* genetic background, sing normally (with the possible exception of *nonA*²/*nonA*⁹, Rendahl *et al.*, unpublished), as do males hemizygous for alleles isolated as visual mutants.

alleles:

allele	discoverer	synonym	comments
<i>*nonA</i> ¹	Benzer	<i>nonA</i> ^{BS18}	
<i>nonA</i> ²	Pak	<i>nonA</i> ^{P14}	does not complement <i>nonA</i> ⁹ song defects
<i>nonA</i> ³	Pak	<i>nonA</i> ^{P49}	complements <i>nonA</i> ⁹ song defects
<i>nonA</i> ⁴	Pak	<i>nonA</i> ^{P60}	complements <i>nonA</i> ⁹ song defects
<i>nonA</i> ⁵	Heisenberg	<i>nonA</i> ^{H2}	
<i>*nonA</i> ⁶	Heisenberg	<i>nonA</i> ^{H17}	<i>opm2</i>
<i>*nonA</i> ⁷	Heisenberg	<i>nonA</i> ^{H48}	
<i>*nonA</i> ⁸	Heisenberg	<i>nonA</i> ⁵³⁰	
<i>nonA</i> ⁹	Steinlauf	<i>diss</i>	isolated as song mutant

cytology: Placed in 14C1-2, by *in situ* hybridization, using a cDNA probe (von Besser *et al.*, 1990). Covered by *Dp(1;2)r⁺75c = Dp(1;2)14B13;15A9;35D-E*; not included in *Df(1)81k21e = Df(1)14C1-2;15A5* (Jones and Rubin, 1990, Neuron. 4: 711-23).

molecular biology: Cloned from two very different starting points. Genetic one: beginning with a probe hybridizing to proximal 14B, and by walking in the proximal direction, the distal breakpoints of *Dp(1;2)r⁺75c* and *Df(1)81k21e* were localized at approximately +64 kb and +84 kb, respectively, on the coordinates of Steller (Jones and Rubin, 1990). Two overlapping genomic fragments from a portion of this interval rescued *nonA*⁵- and *nonA*⁹-associated ERG defects (Jones and Rubin, 1990) and the song defects of the latter as well (Rendahl *et al.*, unpublished). Genomic probes from within the "rescuing region" detected 4-, 3-, and 2.8-kb transcripts (Jones and Rubin, 1990), which are relatively constant in abundance through the life cycle (though the last of these was at relatively lower concentrations in embryos; also see von

Besser *et al.* for embryo results). Same kind of probes led to cDNAs (from adult head library), one of which when sequenced predicts a *ca.* 700-amino-acid-basic protein (Jones and Rubin, 1990) that contains "RNP" RNA binding motifs (von Besser *et al.*, 1990). No homologs found in a data-base search. The gene was also cloned serendipitously by a completely independent method. A particular monoclonal antibody raised against embryonic chromatin (Frasch and Saumweber, 1989, Chromosoma 97: 272-81), which appears to react specifically with a protein in polytene chromosome puffs, was used to isolate clones from an expression library (von Besser *et al.*, 1990); the sequence of one such clone predicts the same protein as that described by Jones and Rubin. *In situ* hybridization to embryos with a cDNA probe detects fairly ubiquitous spatial expression of the transcript(s) (von Besser *et al.*, 1990). This kind of expression is also seen using an anti-NONA monoclonal antibody to stain sections throughout the life cycle (Rendahl, Jones, and Hall, unpublished), with the additional fact that the nervous system is included in the many tissues so expressing and that the subcellular localization appears to be nuclear. The gene comprises five exons and is transcribed from left to right. Analysis of the mRNAs expressed from *nonA* indicates the transcript complexity includes alternative splicings and protein products that differ in approximately 35 C-terminal amino acids (Jones and Rubin, 1990; von Besser *et al.*, 1990). Truncation of both the predicted protein forms (I and II) by oligonucleotide directed mutagenesis followed by transformation, leads to no rescue of *nonA* ERG defects; whereas a stop codon that prematurely terminates only form II does so rescue (Jones and Rubin, 1990).

other information: Mosaic analysis (Hotta and Benzer, 1970) suggested that ERG defects map to photoreceptors; but too few gynandromorphs were analyzed to rule out an optic-lobe "focus", which is more likely to be the focus for visual-movement-response abnormalities (especially those reported by Heisenberg, 1972). Two genomic clones, whose distal endpoints are just distal to *nonA*-rescuing DNA fragments, rescue each of two non-allelic lethals [*l(1)14Cc*, and *l(1)14Ca*] that map to the same cytogenetic interval as does *nonA*; transformation with overlapping genomic clones indicates an order from left to right of *l(1)14Cc l(1)14Ca nonA* (Jones and Rubin, 1990). Both lethals complement the ERG defects of *nonA*⁵ (Jones and Rubin, 1990) and complement the song abnormality of *nonA*⁹ (Kulkarni and Hall, unpublished).

nonB: see *nba*

nonC (J.C. Hall)

location: 1- (not localized).

origin: Induced by ethyl methanesulfonate.

references: Heisenberg, 1979, Handbook of Sensory Physiology (Antrum, ed.). Springer-Verlag, Berlin, Vol. VII/6A, pp. 665-79.

Bülthoff, 1982, DIS 58: 31.

1982, Biol. Cybernet. 45: 63-70.

phenotype: Defective phototaxis and optomotor responses; no light-on or light-off transients in electroretinogram, probably due to defect in photoreceptor cells *per se*; abnormal orientation to spots in Y-maze test.

alleles: Two mutant alleles, *nonC*¹ (=X37), *nonC*²

(=X72).

none: no ocelli; narrow eyes

location: 3-

discoverer: Shearn.

references: Stark, Srivastava, Carlson, and Garment, 1984, DIS 60: 191-93 (fig.).

phenotype: No ERG or deep pseudopupal in *none/none* flies. In the compound eye, corneal facets are fused and corneal hairs displaced, but corneal nipples are present. There is an all-glial cell mass in the peripheral retina and there are no rhabdomeres or optic cartridges as in the wild-type fly. In the ocellar area, only remnants of the ocellar lenslets can be observed.

other information: *none* mutants resemble *Gl* mutants in regard to morphology of the compound eye (Harte and Kankel, 1982, Genetics 101: 477-501) and absence of an ERG.

nonpupariating 3: see *npr3*

***norpA*: no receptor potential A (J.C. Hall)**

location: 1-6.5.

origin: Induced by ethyl methanesulfonate.

discoverer: Pak, Grossfield, and Arnold; Hotta and Benzer; Heisenberg (all independently).

synonym: *nofC*; *x12*, *x13*, *x16*, *x24*.

references: Hotta and Benzer, 1970, Proc. Nat. Acad. Sci. USA 67: 1156-63.

Pak, Grossfield and Arnold, 1970, Nature (London) 227: 518-20.

Deland and Pak, 1973, Nature (London), New Biol. 244: 184-86.

Pak, Ostroy, Deland, and Wu, 1976, Science 194: 956-59.

Harris and Stark, 1977, J. Gen. Physiol. 69: 261-91.

Ostroy, 1978, J. Gen. Physiol. 72: 717-32.

Hotta, 1979, Mechanisms of Cell Change (Ebert and Okada, eds.). John Wiley, New York, pp. 169-82.

Minke and Armon, 1980, Photochem. & Photobiol. 32: 553-62.

Lo and Pak, 1981, J. Gen. Physiol. 77: 155-75.

Matsumoto, O'Tousa, and Pak, 1982, Science 217: 839-41.

Yoshioka, Inoue, and Hotta, 1983, Biochem. Biophys. Res. Comm. 111: 567-73.

Inoue, Yoshioka, and Hotta, 1985, Biochem. Biophys. Res. Comm. 132: 513-19.

Matsumoto and Pak, 1985, Neurobiology (Gilles and Balthazart, eds.). Springer-Verlag, Berlin, pp. 398-412.

Banga, Bloomquist, Brodberg, Pye, Larrivee, Mason, Boyd, and Pak, 1986, Chromosoma 93: 341-46.

Wilson and Ostroy, 1987, J. Comp. Physiol. 161: 785-91; 793-98.

Bloomquist, Shortridge, Schneuwly, Perdew, Montell, Steller, Rubin, and Pak, 1988, Cell 54: 723-33.

Inoue, Yoshioka, and Hotta, 1988, J. Biochem. 103: 91-94.

Zinkl, Maier, Studer, Sapp, Chen, and Stark, 1990, Vis. Neurosci. 5: 429-39.

phenotype: Structural gene for phospholipase-C (PLC; specifically, phosphatidyl inositol 4.5 biphosphate phosphodiesterase). *norpA* mutants are blind and have no (or reduced) light-elicited photoreceptor potentials (*re*: electroretinograms) in the compound eyes and ocelli. Adults

homozygous or hemizygous for severe alleles are completely blind, whereas those carrying weaker alleles have amplitude-subnormal ERGs induced by light (Ostroy and Pak, 1974, BBRC 59: 960-66; Wilson and Ostroy, 1987a). Light-induced behavior (negative phototaxis) of larvae also absent under influence of severe alleles [Markow, 1981, Behav. Neur. Biol. 31: 348-53; Hotta and Keng, 1984, Animal Behavior: Neurophysiological and Ethological Approaches (Aoki *et al.*, eds.). Springer-Verlag, Berlin, pp. 49-60]. A decrease in the amount of rhodopsin occurs under influence of severe alleles (Ostroy, 1978), though this was largely blocked when the mutant flies were reared and kept in constant darkness (Zinkl *et al.*, 1990), and precedes an age-dependent degeneration of adult photoreceptors (Wilson and Ostroy, 1987b), which is accentuated at high temperatures (Zinkl *et al.*, 1990). Severe mutants show no pigment granule migration with light adaptation (Lo and Pak, 1981). Electrophysiological as well as behavioral phenotypes are present in the youngest flies tested, long before the degenerative changes become apparent. There are zipper-like membrane specializations on plasmalemma of *norpA* retinula cells (Alawi, Jennings, Grossfield, and Pak, 1972, Adv. Exp. Med. Biol. 24: 1-21; Stark, Sapp, and Carlson, 1988, J. Neurogenet. 5: 49-59). Microvillar membranes of the photoreceptor-cell rhabdomeres are severely depleted in six-day-old *norpA*⁷ adults [Hiro-sawa and Hotta, 1982, The Structure of the Eye (Hollifield, ed.). Elsevier, New York, pp. 45-53]. *norpA* mutants show polypeptide differences with respect to eye proteins on 1-d or 2-d gels [Ostroy and Pak, 1973, Nature (London) 243: 120-21; Hotta, 1979]. The mutation blocks light-induced phosphorylation of three eye-specific proteins (Matsumoto *et al.*, 1982); one of these proteins has been identified as R1-6 opsin (Nichols and Pak); blockage of this phosphorylation is most complete in severely blind *norpA* alleles, less so in *norpA* alleles with measurable ERGs. Phospholipid kinase (diglyceride kinase) activity is nearly absent in *norpA* mutants, as is phosphorylation of the photoreceptor phospholipid, phosphatidic acid (Yoshioka *et al.*, 1983). Hydrolysis of phosphatidyl-inositol 4.5-biphosphate, liberation of the inositol triphosphate product, and activity of PLC are only 2-3% normal (Inoue *et al.*, 1985, 1988), under the influence of a severe allele (*norpA*⁷) and are about 10% of normal in *norpA*⁹, decreasing another five-fold (as does heat-sensitive blindness) after shift to 28° (Inoue *et al.*, 1985, 1988). *so* (sine oculis) removes about 90% of PLC activity, whereas a *norpA* mutation can remove substantially more (Inoue *et al.*, 1985; Yoshioka, Inoue, and Hotta, 1985, J. Biochem. 97: 1251-54); this would seem to jibe with the provisional demonstration, by *in situ* hybridization, of a low level of *norpA* expression (however, this was not shown to be specifically a gene product) in the optic lobes and central brain (Bloomquist *et al.*, 1988).

alleles:

allele	discoverer	synonym	ref ^α	comments ^β
<i>norpA</i>¹	Bülthoff	<i>norpA</i> ^{B1}	3, 4	γ
<i>norpA</i>²	Bülthoff	<i>norpA</i> ^{B2}	3, 4	γ
<i>norpA</i>³	Bülthoff	<i>norpA</i> ^{B5}	3, 4	γ
<i>norpA</i>⁴	Bülthoff	<i>norpA</i> ^{B8}	3, 4	Group II γ
<i>norpA</i>⁵	Bülthoff	<i>norpA</i> ^{B9}	3, 4	Group II γ
<i>norpA</i>⁶	Bülthoff	<i>norpA</i> ^{B10}	3, 4	Group II γ

allele	discoverer	synonym	ref ^α	comments ^β
<i>norPA</i> 7	Benzer	<i>norPA</i> ^{EE5}	2, 7	Group II
<i>norPA</i> 8	Benzer	<i>norPA</i> ^{JM11}		
<i>norPA</i> 9	Benzer	<i>norPA</i> ^{KO50}	8	Ser [?] → Phe; Gly [?] → Ser temperature-sensitive allele
<i>norPA</i> 10	Benzer	<i>norPA</i> ^{KS85}		
<i>norPA</i> 11	Benzer	<i>norPA</i> ^{KS115}		
<i>norPA</i> 12	Benzer	<i>norPA</i> ^{SB37}		
<i>norPA</i> 13	Heisenberg	<i>norPA</i> ^{H3}		Group III
<i>norPA</i> 14	Heisenberg	<i>norPA</i> ^{H4}		Group II
<i>norPA</i> 15	Heisenberg	<i>norPA</i> ^{H5}		Group IV
<i>norPA</i> 16	Heisenberg	<i>norPA</i> ^{H9}		Group I
<i>norPA</i> 17	Heisenberg	<i>norPA</i> ^{H10}		Group II
<i>norPA</i> 18	Heisenberg	<i>norPA</i> ^{H14}		Group III
<i>norPA</i> 19	Heisenberg	<i>norPA</i> ^{H19}		Group II
<i>norPA</i> 20	Heisenberg	<i>norPA</i> ^{H21}		Group III
<i>norPA</i> 21	Heisenberg	<i>norPA</i> ^{H23}		Group IV
<i>norPA</i> 22	Heisenberg	<i>norPA</i> ^{H26}		Group III
<i>norPA</i> 23	Heisenberg	<i>norPA</i> ^{H27}		Group IV
<i>norPA</i> 24	Heisenberg	<i>norPA</i> ^{H28}		Group II
<i>norPA</i> 25	Heisenberg	<i>norPA</i> ^{H30}		Group II
<i>norPA</i> 26	Heisenberg	<i>norPA</i> ^{H33}		Group III
<i>norPA</i> 27	Heisenberg	<i>norPA</i> ^{H35}		Group I
<i>norPA</i> 28	Heisenberg	<i>norPA</i> ^{H39}		Group II
<i>norPA</i> 29	Heisenberg	<i>norPA</i> ^{H43}		Group III
<i>norPA</i> 30	Heisenberg	<i>norPA</i> ^{H44}		Group II
<i>norPA</i> 31	Heisenberg	<i>norPA</i> ^{H52}	2, 5, 8	Ser [?] → Tyr temperature-sensitive allele
		<i>opm52</i>		
<i>norPA</i> 32	Heisenberg	<i>norPA</i> ^{H54}		Group III
<i>norPA</i> 33	Pak	<i>norPA</i> ^{P12}		Group III
<i>norPA</i> 34	Pak	<i>norPA</i> ^{P13} ; x12	2, 9-11	Group II
<i>norPA</i> 35	Pak	<i>norPA</i> ^{P16} ; x13	2, 10, 11	Group III
<i>norPA</i> 36	Pak	<i>norPA</i> ^{P24} ; x16	2, 11	Group I
<i>norPA</i> 37	Pak	<i>norPA</i> ^{P39}	1, 2, 10	Group I
<i>norPA</i> 38	Pak	<i>norPA</i> ^{P40}		Group II
<i>norPA</i> 39	Pak	<i>norPA</i> ^{P41}		Group I
<i>norPA</i> 40	Pak	<i>norPA</i> ^{P42}		Group III
<i>norPA</i> 41	Pak	<i>norPA</i> ^{P45}		Group III
<i>norPA</i> 42	Pak	<i>norPA</i> ^{P46}		Group II
<i>norPA</i> 43	Pak	<i>norPA</i> ^{P47}		Group IV
<i>norPA</i> 44	Pak	<i>norPA</i> ^{P51}		Group III
<i>norPA</i> 45	Pak	<i>norPA</i> ^{P54}		Group III
<i>norPA</i> 46	Pak	<i>norPA</i> ^{P55}		Group I
<i>norPA</i> 47	Pak	<i>norPA</i> ^{P57}		Group IV
<i>norPA</i> 48	Pak	<i>norPA</i> ^{P64}		Group III
<i>norPA</i> 49	Pak	<i>norPA</i> ^{P70}		Group II
<i>norPA</i> 50	Pak	<i>norPA</i> ^{P71}		Group II
<i>norPA</i> 51	Pak	<i>norPA</i> ^{P76}		Group III
<i>norPA</i> 52	Pak	<i>norPA</i> ^{P78}		Group III
<i>norPA</i> 53	Pak	<i>norPA</i> ^{P79}		Group III
<i>norPA</i> 54	Pak	<i>norPA</i> ^{P81h}	2	
<i>norPA</i> 55 δ		<i>norPA</i> ^{sul}	6	Group II
<i>norPA</i> 56 δ		<i>norPA</i> ^{sull}	6, 12	Group IV
<i>norPA</i> 57 δ		<i>norPA</i> ^{sulll}	6	Group II

^α 1 = Banga, Bloomquist, Brodberg, Pye, Larrivee, Mason, Boyd, and Pak, 1986, Chromosoma 93: 341-46; 2 = Bloomquist, Shortridge, Schneuwly, Perdew, Montell, Steller, Rubin, and Pak, 1988, Cell 54: 723-33; 3 = Bülthoff, 1982, DIS 58: 31; 4 = Bülthoff, 1982, DIS 58: 32; 5 = Deland and Pak, 1973, Nature (London) New Biol. 244: 184-86; 6 = Harris and Stark, 1977, J. Gen. Physiol. 69: 261-91; 7 = Hirotsawa and Hotta, 1982, The Structure of the Eye (Hollifield, ed.) Elsevier, New York, pp. 45-53; 8 = Hotta and Masai, 1991, J. Neurogenet., in press; 9 = Ostroy and Pak, 1973, Nature (London), New Biology 243: 120-21; 10 = Ostroy and Pak, 1974, Biochim. Biophys. Acta 368: 259-68; 11 = Pak, Grossfield, and Arnold, 1970, Nature (London) 227: 518-20; 12 = Stark, Chen, Johnson, and Frayer, 1983, J. Insect Physiol. 29: 123-31.

^β Alleles classified arbitrarily into four groups based on the electroretinogram amplitudes:

I = no ERG; II = very small (<3mV) ERG, even with high intensity stimuli; III = intermediate ERG; IV = ERG amplitudes approaching

that of wild type ERGs when intense stimuli are used.

^γ Fixation defective; isolated on basis of poor orientation to spots in a Y-maze test and originally designated *nofC* mutations (Bülthoff, 1982). Exhibit defective visual response in a freely walking test. ERG's light sensitivity severely reduced and there are no light-on or light-off transient spikes.

^δ Recovered as allele specific suppressors of *rdgB*⁹ (Harris and Stark, 1977, see also Stark, Chen, Johnson, and Frayer, 1983, J. Insect Physiol. 29: 123-31).

cytology: Located at 4B6-C1 by *in situ* hybridization (Bloomquist *et al.*, 1988); lies between the cytologically indistinguishable distal breakpoints of *Df(1)rb*⁴¹ and *Df(1)bi*^{D2} at 4B6-C1.

molecular biology: Gene cloned and sequenced (Bloomquist *et al.*, 1988). Encodes a 7.5 kb RNA expressed in the head. Putative *norPA* protein of 1095 amino acid residues shows extensive sequence similarity to a phospholipase C (PLC) amino acid sequence from bovine brain (Bloomquist *et al.*, 1988). Report of a decrement in an eye-specific diacylglyceride kinase activity (diacylglyceride being produced as a result of PLC action) in *norPA* (Yoshioka, Inoue, and Hotta, 1984, Biochem. Biophys. Res. Comm. 119: 389-95) is not readily explainable by other enzymatic studies of the mutant, which are consistent with the clone and sequence data. Note that the PLC decrements are correlated with physiological severities of various *norPA* mutations, whereas DGK reductions are not (Inoue *et al.*, 1988).

other information: *norPA* mutations have been used to assess the role of "basic vision" in complex behaviors such as courtship and circadian rhythms. Initiation of courtship and beginning of mating are prolonged in mutant males (Markow and Manning, 1980, Behav. Neur. Biol. 29: 276-80; Tompkins, Gross, Hall, Gailey, and Siegel, 1982, Behav. Genet. 12: 295-307), but *norPA* females appear to mate more readily than normal females (Tompkins *et al.*, 1982; Markow and Manning, 1982, DIS 58: 104-05). Blind *norPA* adults can respond to light changes, with regard to their cyclically changing locomotor activity in 12h:12h light:dark (LD) cycles, and in terms of being entrained to exhibit free-running circadian rhythms of activity after transfer from LD to constant darkness (Konopka, 1980, Neurosci. Abstr. 6: 706; Dushay, Rosbash, and Hall, 1989, J. Biol. Rhythms 4: 1-27); yet, the free running periodicities were about 1 hour shorter than control values (Dushay *et al.*, 1989). Experiments involving turning on *per*-gene expression in adult photoreceptors, which requires exposure (of wild-type) to light-dark transition, is normal in two severe *norPA*'s (Zerr, Hall, Rosbash, and Siwicki, 1990, J. Neurosci. 10: 2749-62). Pressure injections of PLC into eye during ERG recordings does not ameliorate defective *norPA* physiology (Zinkl *et al.*, 1990). Rhabdomere turnover rhodopsin cycling rhythms (Stark, Sapp, and Schilly, 1988, J. Neurocytol. 17: 499-509) are damped or absent in *norPA* (Zinkl *et al.*, 1990).

nos: nanos

location: 3-66.2.

origin: Induced by ethyl methanesulfonate.

discoverer: Lehmann.

references: Nüsslein-Volhard, Frohnhöfer, and Lehmann, 1987, Science 238: 1675-81.

Lehmann, 1988, Development 104 (Suppl.): 17-27.

Hülskamp, Schröder, Pfeifle, Jäckle, and Tautz, 1989,

Nature (London) 338: 629-32.

Irish, Lehmann, and Akam, 1989, Nature (London) 338: 646-48.

Struhl, 1989, Nature (London) 338: 741-44.

phenotype: Maternal-effect lethal. Mutant embryos lack abdominal segments, but have normal pole cells and pole plasm; no posterior activity in pole plasm. Transport or diffusion of the *nos* gene product from the posterior of the embryo seems to be essential for development of the wild-type abdominal pattern. Presence of the *nos* protein represses the activity of the gene product encoded by the *hb* maternal transcript in the posterior half of the embryo (Hülkamp *et al.*, 1989; Irish *et al.*, 1989; Struhl, 1989). Eggs deficient for both *hb* and *nos*, when fertilized by *hb*⁺ sperm, develop into normal embryos and subsequently into viable flies.

alleles: Four alleles, three strong and one weak (Wang and Lehmann, 1989).

cytology: Located in 91F-92A.

molecular biology: *nos* has been cloned (Lehmann) and a maternal transcript identified (Wang and Lehmann, 1989).

Not upheld: see *Mhc*¹⁶

Notch: see *N*

Notch 2: see *N-b*

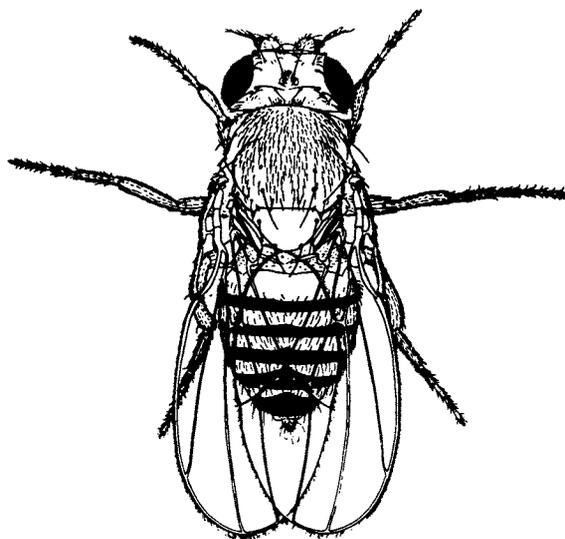
Notch b: see *N-b*

Notch Xasta: see *NX*

notchoid: see *nd* under *N*

notchy: see *ny*

Notopleural: see *Np*



Np: Notopleural

From Bridges, Skoog, and Li, 1936, Genetics 21: 788-95.

***Np: Notopleural**

location: 2-58.7 to 60.2 (between *cn* and *en*; inseparable from *blo*).

origin: Spontaneous.

discoverer: Nichols-Skoog, 33b20.

references: Bridges, Skoog, and Li, 1936, Genetics 21: 788-95 (fig.).

Li, 1936, Peking Nat. Hist. Bull. 11: 39-48.

phenotype: Notopleural, humeral, presutural, and pretarsal bristles shorter and blunter than normal. Wings short and broad. Female produces few or no progeny. Viability fair. Development retarded. More extreme at 19° than at 25°, also more extreme in female. Lethal over *T(2;3)dp*. Homozygous lethal. RK2A.

cytology: Locus lies between 44F1 and 45E2 on the basis of its association with *Df(2R)Np = Df(2R)44F1-2;45E1-2* (Bridges).

*npr*¹: see *BRC*

*npr*²: see *BRC*

npr3: nonpupariating 3

location: 1-65.8.

origin: Induced by ethyl methanesulfonate.

synonym: *l(1)npr-3; l(1)d.deg.12*.

references: Kiss, Bencze, Fodor, Szabad, and Fristrom, 1976, Nature (London) 262: 136-38.

Kiss, Szabad, and Major, 1978, Mol. Gen. Genet. 164: 77-83.

Kiss, Szabad, Belyaeva, Zhimulev, and Major, 1980, Development and Neurobiology of Drosophila (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York and London, pp. 163-81.

phenotype: Male lethal; continues development through larval stage, but does not pupariate. Imaginal discs are very small and structureless; ring glands are normal in size on eighth to tenth day of larval life. Puparium formation can be induced by implantation of wild-type ring glands.

nr: nesh

location: 2-28.3 (26.5 cM to left of Bristle).

origin: Isolated from isofemale lines carrying eagle that were derived from natural populations in California.

references: Prout and Green, 1986, DIS 63: 169.

phenotype: Identical to *eg*; *nr* and *eg* combined are *eg* in phenotype.

nrd: neural disrupted

location: 2- {7}.

origin: Differential screen of genomic clones using cDNA probes prepared from neural and non-neural cells.

references: Neumann and Mahowald.

phenotype: Embryonic lethal typified by an enlarged brain, irregular ventral nerve cord formation, and disorganized peripheral ganglia; these defects first appear in 10-12 hour embryos and include a delay in germband contraction and a failure of head involution. The CNS of mutant embryos fails to complete the normal condensation that leads to compact paired cerebral hemispheres and a ventral ganglion. In late mutant embryos, the brain, which has protruded dorsally through a hole in the cuticle, remains extended and flattened against the vitelline membrane and the ventral neuromeres are extended posteriorly; discontinuities or thinnings of the ventral nerve cord can frequently be observed. Normal development of the mouth parts and cephalopharyngeal apparatus is disrupted.

cytology: Located in 23A1-3 by *in situ* hybridization of cloned DNA.

molecular biology: Gene (selectively expressed in neural cells) cloned; 2.3 kb transcript obtained both maternally and in 10-18 hour old embryos. Transcript remains in larval, pupal, and adult stages. Non-neural specific 1.3 and 0.9 kb RNAs are transcribed off the opposite strand. The 2.3 kb transcript is expressed in neural cultures as well as *in vivo*.

Nrg: Neuroglian

location: 1-23.6.

synonym: *l(1)7Fa*.

references: Bieber, Snow, Hortsch, Patel, Jacobs, Traquina, Schilling, and Goodman, 1989, Cell 59: 447-60. Hortsch, Bieber, Patel, and Goodman, 1990, Neuron 4: 697-709.

phenotype: Encodes a protein that is likely to play a role in neural and glial cell adhesion in the developing *Drosophila* embryo. Widely expressed in the embryo, particularly on the surfaces of a large subset of neurons and glia that interact with and adhere to one another along the dorsal surface of the developing CNS and within the peripheral nerve roots; the protein can be detected along neuronal axons, but not cell bodies. Three polypeptides of 155, 167, and 180 kd are detected on Western blots; deglycosylation experiments reduce these to a single 155 kd band; removal of N-linked high-mannose oligosaccharides causes a shift to about 162 kd. It is also expressed in a number of non-neuronal tissues such as trachea, hindgut, salivary gland and muscle. Recessive alleles are embryonic lethals and show little or no evidence of Neuroglian expression; the overall structure of the CNS and PNS, and in particular the peripheral nerve roots and CNS axon pathways develop in a relatively normal way in lethal embryos.

alleles: Neuroglian has been identified with a previously isolated lethal mutation of which ten alleles have been described.

allele	origin	discoverer	synonym	ref ^α	comments
<i>Nrg</i> ¹¹	X ray	Lefevre	<i>l(1)HC280</i>	3	<i>T(1;3)4A;7E;80-81?</i>
<i>Nrg</i> ¹²	X ray	Lefevre	<i>l(1)HC293</i>	3	<i>In(1)6D-E1;7F3-4</i>
<i>Nrg</i> ¹³	X ray	Lefevre	<i>l(1)HF336</i>	3	
<i>Nrg</i> ¹⁴	X ray	Lefevre	<i>l(1)RA35</i>	1, 3	amorphic allele; <i>In(1)6E;7F1</i>
<i>Nrg</i> ¹⁵	EMS	Lefevre	<i>l(1)EC255</i>	4	
<i>Nrg</i> ¹⁶	EMS	Lefevre	<i>l(1)EF435</i>	4	
<i>Nrg</i> ¹⁷	EMS	Lefevre	<i>l(1)VA142</i>	1, 4	hypomorphic allele
<i>Nrg</i> ¹⁸	EMS	Lefevre	<i>l(1)VA321</i>	4	
<i>Nrg</i> ¹⁹	spont	Schalet	<i>l(1)17-145</i>		
<i>Nrg</i> ¹¹⁰	EMS	Digan	<i>l(1)B4</i>	2	

^α 1 = Bieber, Snow, Hortsch, Patel, Jacobs, Traquina, Schilling and Goodman, 1989, Cell 59: 447-60; 2 = King, Mohler, Riley, Storto, and Nicolazzo, 1986, Dev. Genet. 7: 1-20; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Lefevre and Watkins, 1986, Genetics 113: 869-95.

cytology: Placed in 7F by *in situ* hybridization; by deficiency analysis placed in the region common to *Df(1)KA14 = Df(1)7F1-2;8C5* and *Df(1)RA2 = Df(1)7D10;8A4-5*.

molecular biology: A Neuroglian-specific antibody was used to screen an embryonic cDNA expression library; cDNA's corresponding to a 5.1 kb transcript were recovered and localized to 7F on polytene chromosomes. Encodes two different proteins by tissue-specific alternative splicing, which differ in their cytoplasmic domains; the longer form is restricted to the surface of neurons in the CNS and some support cells in the PNS. The shorter

form is more widely expressed in other cells and tissues. Northern blots probed with sequences specific to each of the polypeptides reveal transcripts of 5.5 kb for the short form and 5.7 and 7.6 kb for the long form. Sequence analysis reveals that Neuroglian is an integral membrane glycoprotein that is a member of the immunoglobulin superfamily. The extracellular portion of the protein consists of six immunoglobulin C2-type domains followed by five fibronectin type III domains. It is closely related to vertebrate neural adhesion molecules, most extensively to mouse L1.

nrs: narrow scoop

location: 1-54.2.

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 88.

phenotype: Wings narrow and slightly shorter than normal; frequently scooped. Slightly thinner bristles. Eyes large and dull red. Eye and body colors darken with age. Viability and fertility good in male; fertility low in female. RK2.

Nrt: Neurotactin (J.C. Hall)

location: 3-{45}.

origin: Molecular cloning, starting with monoclonal-antibody screening of expression library.

references: de la Escalera, Bockamp, Moya, Piovant, and Jiménez, 1990, EMBO J. 9: 3593-3601.

Barthalay, Hipeau-Jacquotte, de la Escalera, Jiménez, and Piovant, 1990, EMBO J. 9: 3603-09.

Hortsch, Patel, Bieber, Traquina, and Goodman, 1990, Development 110: 1327-40.

phenotype: Three monoclonal antibodies selected because of binding to presumptive imaginal neurons within the larval central nervous system (CNS) (de la Escalera *et al.*, 1990) and an additional one found by virtue of binding to neuronal surfaces in embryos (Hortsch *et al.*, 1990) detected the same *ca.* 135 kd protein on Western blots of homogenates from embryos, late larva, and pupae. The membrane-bound material is called neurotactin (NRT) because of its expression at points of interneuronal cell contact. Antibody staining (de la Escalera *et al.*, 1990, Hortsch *et al.*, 1990) shows concentration of the protein in dorsal and ventral portions of embryonic blastoderm (where staining appears cell surface-limited), all over gastrulating embryos (although Hortsch *et al.*, 1990, imply a somewhat more restricted expression pattern), in the "proliferating" CNS (including neuroblasts and their progeny) of stage 10-11 embryos, and in regions of contact between neuroblasts. In visceral mesoderm (stage 13), non-neuronal expression diminishes, although it is seen on fat body cells and the "dorsal vessel"; intense staining continues in embryonic CNS (but is relatively weak in axons of motor neurons); PNS expression is evident as well (seemingly restricted to sensory cells that send out multiple dendritic projections, and, in fact, PNS cell-body signals are weaker than on dendrites); cell-surface expression apparent in the various expressing tissues during mid-embryogenesis; in early L1, NRT signals decay but reappear in CNS (in optic formation centers and in neuron clusters and associated axons in ventral cord); the protein's expression persists in imaginal neurons through mid-pupal stage, wanes as such cells com-

plete maturation, and is undetectable in adults. L3 imaginal discs are NRT positive (e.g. on developing chordotonal neurons of leg discs and in developing photoreceptor cells plus their axons, posterior to the morphogenetic furrow). Cell culture studies, including electron-microscope observations (Barthalay *et al.*, 1990), suggest further that NRT is a "contact molecule" between neurons or epithelial cells; there is uniform expression along intercellular contact areas; non-adhesive Schneider-2 cells, transfected with *Nrt* cDNA, do not become self adhesive, but these cells bind to a subpopulation of embryonic cells.

cytology: Placed in 73C1-2 by *in situ* hybridization (de la Escalera *et al.*, 1990; Hortsch *et al.*, 1990). Embryos deficient for this region do not stain with anti-NRT monoclonal antibodies (de la Escalera *et al.*, 1990; Hortsch *et al.*, 1990).

molecular biology: cDNAs cloned from expression libraries (de la Escalera *et al.*, 1990; Hortsch *et al.*, 1990). Such clones, when used in Northern blots, detected three or more transcripts, whose expression patterns are different during the course of embryogenesis (Hortsch *et al.*, 1990), and which exhibit a later "peak" in early pupae (de la Escalera *et al.*, 1990); the larger RNA species appear to be so because of more 3' untranslated nucleotides (de la Escalera *et al.*, 1990). *In situ* hybridizations (Hortsch *et al.*, 1990) revealed temporal/spatial expression patterns similar to those seen immunohistochemically. Sequencing of a complete cDNA (de la Escalera *et al.*, 1990; Hortsch *et al.*, 1990) predicts an 846-amino-acid polypeptide [which would, as a core protein, be ca. 93 kd, though actual measurements of its size indicate 120 kd (Barthalay *et al.*, 1990)]. There is one predicted transmembrane region, and details of which monoclonal antibodies can stain simple whole-mounted embryos vs. permeabilized ones (de la Escalera *et al.*, 1990), as well as biochemical analyses (epitope mapping), using antibodies raised against *Nrt*-fusion protein constructs (Hortsch *et al.*, 1990), are consistent with the protein's being inserted into membranes, with the N-terminus cytoplasmic and the C-terminus extracellular. Data-base comparisons (de la Escalera *et al.*, 1990; Hortsch *et al.*, 1990) indicate similarity of portion of NRT's extracellular domain to cholinesterases, including product of *Drosophila Ace* locus; this NRT region is also similar to *Drosophila glutactin* and to rat thyroglobulin. NRT apparently lacks the "landmark" serine residue that would indicate it to have actual esterase activity (Hortsch *et al.*, 1990).

other information: Embryos homozygous for either of the two *Nrt*⁻ deletions used by de la Escalera *et al.* (1990) [*Df(3L)st-k10* = *Df(3L)73A3-4;73D1-2* and *Df(3L)73A11-B1;73D1-2*] do not exhibit any "extra" gross abnormalities of the CNS. Deletion of regions just distal to *Nrt* [i.e. *Df(3L)st100.62* = *Df(3L)72F3-7;73B3* or *Df(3L)st-e5* = *Df(3L)?;73A9-10*] leads to defects in axonal patternings (because of the absence of the *abl* and *dab* genes in 73B, cf. Gertler, Bennet, Clark, and Hoffman, 1989, Cell 58: 103-113), but the further removal of *Nrt*⁺ does not make this phenotype worse (de la Escalera *et al.*, 1990).

Ns: see *Antp*^{Ns}

Ntf: Neurogenic element binding transcription factor

location: 2-{83}.

synonym: *Elf-1: Element I binding activity.*

references: Bray, Burke, Brown and Hirsh, 1989, Genes Dev. 3: 1130-45.

Dynlacht, Attardi, Admon, Freeman, and Tjian, 1989, Genes Dev. 3: 1677-88.

phenotype: Gene encodes at least three isoforms of a protein that binds to upstream sequences of *Ddc*, *en*, *ftz*, and *Ubx*. The proteins share epitopes as recognized by two monoclonal antibodies. Expression first detected in 4- to 8-hour embryos, peaking from 8 to 12 hours and declining from 12 to 16 hours; NTF protein detected in nuclei of ectodermal derivatives; seen in all epidermal cells and changing subsets of neurons in the developing central nervous system.

cytology: Placed in 54F1-2 by *in situ* hybridization.

molecular biology: Protein purified by DNA-affinity chromatography using promoter sequences derived from *Ddc*, *Ubx*, or both. Three prominent polypeptides recovered (140, 120, and 83 kd); the purified proteins shown to bind to specific neurogenic control regions upstream from *Ddc*, *ftz*, and *Ubx* by DNA footprinting and to stimulate transcription of these genes *in vitro*. Northern blots of embryonic RNA detect mRNA's of 7.4 and 10.6 kb. cDNA clones isolated by screening an expression library either with monoclonal antibody (Bray *et al.*) or with synthetic oligonucleotides (Dynlacht *et al.*). Three different cDNA's identified by Bray *et al.* Presumed to arise as a result of alternative splicing; one of these encodes a polypeptide of 1063 residues and approximately 116 kd. The conceptual amino acid sequence contains several polyglutamine stretches (OPA repeats) in the N-terminal half; it shows no evidence of homeobox or zinc-finger domains; there is weak homology, however, to the helix-loop-helix motifs of *myoD* and myogenin.

***Nu: Nude**

location: 2- or 3- (rearrangement).

origin: X ray induced.

discoverer: Sutton, 41a27.

phenotype: Many bristles missing from head and thorax; postscutellars, notopleurals, verticals, and postverticals usually present. Homozygous lethal. RK2A.

cytology: Associated with $T(2;3)Nu = T(2;3)24;36-37;-39-40;73-74;75-76;77-78;81-82;85-86;89-90$.

nub: nubbin

location: 2-47.0.

origin: Spontaneous.

discoverer: Mickey, 48e10.

references: 1949, DIS 23: 61.

phenotype: Wings very small, opaque, curved spoonlike up or down; inflated at eclosion. Wing margins interrupted. Only one vein (L2 or L3) present. Halteres somewhat reduced. Viability excellent. RK1.

cytology: Placed in 33F1-5. Not included in *Df(2L)64j* = *Df(2L)34E5-F1;35C3-D1* (E. H. Grell).

nub²

origin: Probably X ray induced.

discoverer: R. F. Grell, 56f1.

references: 1956, DIS 30: 71.

phenotype: Wings small and spoonlike but less extreme than *nub*. Patches of dried blood on wings. Veins L1 to L4 almost indiscernible; L5 and alula frequently absent. Viability and fertility excellent. RK1.

nub^{62d}

origin: X ray induced.

discoverer: Seiger, 62d.

references: 1963, DIS 37: 53.

Abbadessa and Burdick, 1963, DIS 37: 54.

phenotype: Wings very small and spoonlike. RK1.

Nuc: Nuclease

location: 3-38.2.

references: Angelosanto and Boyd, 1976, Genetics 83: s2.

phenotype: Structural gene for a desoxyribonuclease active at alkaline pH and correlated with the appearance of a puff in the salivary glands of third instar larvae.

alleles: Two electrophoretic variants, *Nuc*¹, and *Nuc*², were found in laboratory stocks.

cytology: Located in 68D-70A (Y-autosome translocations); puffing activity correlated positively with level of DNase activity in salivaries during last 24 hours of larval development.

nuc1: nucleoside auxotroph 1

location: 2-104.

synonym: *pyr2*.

references: Naguib and Nash, 1976, Mol. Gen. Genet. 147: 13-21.

phenotype: Nucleoside auxotroph; responds to dietary ribonucleosides.

alleles: *nuc1*¹ (= *pyr2-1*); *nuc1*² (= *pyr2-2*).

Nuclease: see *Nuc*

nucleoside auxotroph 1: see *nuc1*

Nude: see *Nu*

nudel: see *ndl*

numb: numb

location: 2- {35}.

references: Uemura, Shepherd, Ackerman, Jan, and Jan, 1989, Cell 58: 349-60.

phenotype: The *numb* gene must be able to function in the *Drosophila* embryo in order for the peripheral sensory neurons to acquire their correct identity. In the mutants, the precursors of neurons and glial cells in the external sensory (es) organs are, for the most part, transformed into nonneural support cells; some of the es organs are duplicated. Transformation of neuron precursors into nonneural cells also occurs in the chordotonal (ch) organs. Precursors of the multiple dendrite (md) neurons undergo similar changes. Muscle development is abnormal in *numb* mutant alleles; some muscles are fewer in number than in wild type and show pattern changes (Uemura *et al.*).

alleles: Three mutant alleles have been identified, all apparently nulls since the neuronal phenotype of homozygotes is indistinguishable from that of hemizygotes.

allele	origin	molecular biology
<i>numb</i> ¹	HD	P-element at 30B
<i>numb</i> ²	DEB	
<i>numb</i> ³	DEB	

cytology: Located in 30A-C since *numb*¹ is not complemented by a deletion in this region; excision of the P-element at 30B leads to a reversion of the mutant phenotype.

molecular biology: 50 kb of genomic DNA flanking the insertion of a transposon, pUCHsneo, cloned by the plasmid rescue method and a chromosome walk. Two transcripts of 3.1 and 3.5 kb result from alternative splicing of *numb*, the first mRNA being maternal in origin and the second zygotic. cDNA clones from the 5' end of the maternal transcript and from the entire zygotic transcript have been sequenced. The zygotic DNA shows a single open reading frame. From these cDNA sequences, a 56 kd maternal protein and 61 kd zygotic protein have been predicted. Both maternal and zygotic products have putative zinc finger domains and are very basic. A 60 kd protein can be detected by anti-*numb* antibodies in very young (0-1 hr and 3-5 hr) *numb*⁺ embryos; a larger protein (65 kd) can be detected by this antibody in older *numb*⁺ embryos (11-13 hr and 15-17 hr). Homozygous *numb*¹ embryos fail to show either protein. Uemura *et al.* suggest that these proteins may be involved in nucleic acid binding.

Nup: see *Mhc*¹⁶

nurse cell number: see *ncn*

**nw: narrow*

location: 2-79.6 (Rizki *et al.*, 1980; see *nw*^D); 2-79.3 (Doane and Clark, 1984; see *nw*²).

phenotype: Wings long, narrow, and somewhat pointed. Low viability and fertility in both sexes. At 25°C, may overlap wild type; at 19°, nearly all flies approach wild type but have longer wings. RK2.

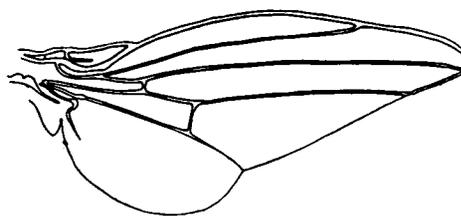
alleles:

allele	origin	discoverer	ref ^α
* <i>nw</i>	spont	Bridges, 16b7	4
<i>nw</i> ^{2β}	spont	Payne, 1615	2, 4, 5
<i>nw</i> ^B	X ray (?)	P.H. Lewis, 1947	1
<i>nw</i> ^D	X ray	E.H. Grell, 59f	3, 6

^α 1 = Craymer, 1980, DIS 55: 197-200; 2 = Doane and Clark, 1984, DIS 60: 234; 3 = Grell, 1962, DIS 36: 37; 4 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 227; 5 = Payne, 1924, Genetics 9: 327-42; 6 = Rizki, Rizki, and Grell, 1980, Wilhelm Roux's Arch. Dev. Biol. 188: 91-99.

^β Synonym: *lance*.

cytology: Placed in 54A-55A.



***nw*²: narrow 2**

From Payne, 1924, Genetics 9: 327-42.

***nw*²**

phenotype: Wings like *nw*, long, narrow, and somewhat pointed. Classification easier in females. Slight notching or tufting of marginal hairs on tip of wings. Both sexes nearly sterile. Ovaries tumorous at eclosion [King, Burnett, and Staley, 1957, *Growth* 21: 239-61 (fig.); King, 1964, *Roy. Entomol. Soc. (London) Symp. Insect Reproduction*, pp. 13-25]. Oogonia proliferate asynchronously within ovariole; follicle development inhibited [Beatty, 1949, *Proc. R. Soc. Edinburgh*, B 63: 249-70 (fig.)]. RK2.

***nw*^B: narrow-Blade**

phenotype: Wings of *nw*^B/+ long and narrow; variable but does not overlap wild type. Female fertility reduced. Homozygote and *nw*^B/*nw*² heterozygote lethal. RK2.

***nw*^D: narrow-Dominant**

phenotype: Wings of heterozygote longer and narrower than normal. Expression variable and sometimes approaches wild type. Viability of *nw*^D/+ low. Homozygous lethal, as is *nw*^D/*nw*². RK2.

***NX*: Notch Xasta**

location: 3- (between *st* and *Dfd*; 44.0-47.5).

origin: X ray induced.

discoverer: Ohnishi, 49116.

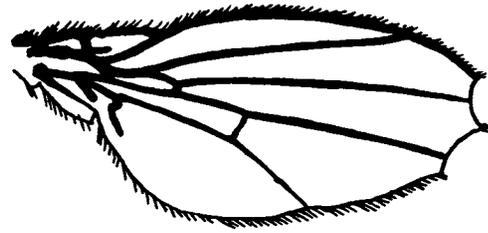
references: 1950, *DIS* 24: 61.

1951, *DIS* 25: 79.

Schalet, 1960, *DIS* 34: 55.

phenotype: Resembles Notch but more extreme. Homozygote resembles *ap*^{Xa}. Viability of heterozygote fair;

homozygote semilethal. Enhanced by *D1* and suppressed by *H*. Combination of *NX* and *ap*^{Xa} produces small wings, like *vg*, and lower viability. RK2 as heterozygote.



***ny*: notchy**

From Grüneberg, 1929, *Biol. Zentralbl.* 49: 680-94.

***ny*: notchy**

location: 1-32.

origin: X ray induced.

discoverer: Grüneberg, 28j29.

references: 1929, *Biol. Zentralbl.* 49: 680-94 (fig.).

1934, *DIS* 2: 8.

phenotype: Wing tips slightly nicked. Expression variable; overlaps wild type in some females and most males. Viability about 70% wild type. RK3.

cytology: Placed in 10B5-18 based on its inclusion in *Df(1)N71 = Df(1)10B5;10D4* but not *Df(1)DA662 = Df(1)10B8;10D2*.

ob: oblique*location:** 1-37.2.**origin:** Spontaneous.**discoverer:** Neel, 41f30.**references:** 1942, Genetics 27: 532.

1942, DIS 16: 51.

phenotype: Wings obliquely truncated from inner margin outward. Venation disturbed. Viability about 20% wild type. RK3.**obl: oblique wings****location:** 1-60.1.**origin:** Induced by triethylenemelamine (CB. 1246).**discoverer:** Fahmy, 1953.**references:** 1958, DIS 32: 72.**phenotype:** Wings slightly upheld and outspread; small blister occasionally present. Body color slightly darker. Male viability and fertility good; female viability about 40% wild type and fertility reduced. RK2.**other information:** One allele induced by CB. 1506.**oblique:** see **ob****oblique wings:** see **obl****obt: obtuse****location:** 3-77.5.**discoverer:** E. M. Wallace, 35g1.**phenotype:** Wings shorter and blunter but overlap wild type slightly. Thorax somewhat humpy; body chunky; eyes slightly bulging. RK3.**cytology:** Not in 94A-E (Jones, 1971, DIS 47: 90).**oc: ocelliless**

"Edith M. Wallace, unpublished."

oc: ocelliless**location:** 1-23.1.**synonym:** *otd: orthodenticle*.**references:** Wieschaus and Noell, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 63-73.

Finkelstein, Smouse, Capaci, Spradling, and Perrimon, 1990, Genes Dev. 4: 1516-27.

phenotype: Ocelli completely absent. Bristles in ocellar area and on top of head irregular and more numerous; postverticals usually absent. Eyes somewhat reduced and body size dwarfed. Phototaxis normal (Benzer, 1967, Proc. Nat. Acad. Sci. USA 58: 1112-19). Viability about 90% wild type. *oc* and deficiencies for *oc* show partial dominance to *oc*⁺; ocelli placed somewhat far back on head and slight indentation apparent between postvertical bristles (Craymer and Roy, 1980, DIS 55: 204). Eggs from *oc*¹ homozygotes have defective chorions and abnormal beta yolk spheres (Johnson and King, 1974, Int. J. Insect. Morphol. Embryol. 3: 385-95). Sterility is due to the presence of *In(1)oc*, one breakpoint of which

disrupts *oc*, and the other of which interferes with amplification of the chorion-protein genes *Cp36* and *Cp38* (Spradling and Mahowald, 1981, Cell 27: 203-09). *oc*¹ in heterozygous combination with non-complementing lethal alleles survives and is female fertile. *Ubl*/+ enhances dominance of *oc*; *Df(1)RA2*/+ exhibit delayed hatch and an *oc* phenotype; *oc*²/+ display strong *oc* phenotype, and *oc*¹/*oc*¹ are lethal in combination with *Ubl*/+ (Mortin and Lefevre, 1981, Chromosoma 82: 237-47). Lethal alleles (recovered as *otd: orthodenticle*) die as embryos; all denticles in anterior abdominal segments point posteriorly; defects at ventral midline; head defects. Also cause embryonic neural defects (Finkelstein *et al.*, 1990): In developing ventral cord, commissures within each segment appear fused. At the cellular level, certain ventral unpaired medial (VUM) neurons do not seem to migrate ventrally (as do the normal VUMs at approximately 14 hours of embryogenesis) and are absent from most segments. Other "midline-associated" neurons are missing as well in *otd*-type embryos. Homozygous germ-line clones survive in females; homozygosity for lethal alleles in germ-line clones without effect on survival of heterozygous offspring or on phenotype of hemizygotes (Wieschaus and Noell). Putative *oc* transcript abundant in embryos, peaking between 4 and 13 hours, before and coincident with neural-developmental defects observed in central nervous system of embryos hemizygous for lethal alleles; there is also putative maternally derived *oc* mRNA (which could be alternately spliced form of the "major" transcript). Northern signals weaken in L1 and L2, are still detectable in early pupae, but are absent in late pupae and adults. *In situ* hybridization reveals earliest embryonic expression at cellular blastoderm; later, signals seen in a longitudinal strip along ventral midline, then in "head region" and in developing ventral cord.

alleles: Except for *oc*¹, alleles are lethal; alleles *oc*² through *oc*⁵ non complementing; *oc*⁶ and *oc*⁷ complement *oc*¹ but not one another.

allele	origin	discoverer	synonym	ref ^α	comments
oc ¹	X ray	Bedichek, 30c15		1	<i>In(1)7F1-2;8A1-2</i>
oc ²	X ray	Lefevre	<i>l(1)JA101</i>	2	
oc ³	X ray	Lefevre	<i>l(1)KA17</i>	2	
oc ⁴	X ray	Lefevre	<i>l(1)RC42</i>	2	
oc ⁵	EMS	Lefevre	<i>l(1)EF504</i>	3	
oc ⁶	EMS	Lefevre	<i>l(1)VA111</i>	3	complements <i>oc</i> ¹
oc ⁷	EMS	Lefevre	<i>l(1)VA126</i>	3	complements <i>oc</i> ¹
oc ⁸	EMS		<i>otd</i> ^{C8}	4, 5	embryonic lethal
oc ⁹	EMS		<i>otd</i> ^{H1}	4, 5	embryonic lethal
oc ¹⁰	EMS			5	embryonic lethal
oc ^{db}	spont	Mohler			β

^α 1 = Bedichek, 1934, DIS 2: 9; 2 = Lefevre, 1981, Genetics 99: 461-80; 3 = Lefevre; 4 = Wieschaus and Noell, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 63-73; 5 = Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307.

β More detailed description follows.

cytology: Placed in 8A1-2 on the basis of its assignment to the proximal breakpoint of *In(1)oc* = *In(1)7F1-2;8A1-2*.

molecular biology: DNA in region 8A1-2 amplifies in *oc*¹ under the influence of *In(1)oc* (Spradling and Mahowald). Identified by Finkelstein *et al.*, (1990), within approximately 80 kb of cloned material from the region of the distal breakpoint of *In(1)oc*. A 4 kb *EcoRI* fragment detects a 4.7 kb transcript; this was designated

as *oc* mRNA because of its embryonic expression pattern. Sequence of a cDNA clone predicts a 670-amino-acid protein with various repeats of single amino acids (e.g. Gly, Ser, Gln), pairs of amino acids (e.g. alternating Gly-Val), and a tandemly-duplicated 19-mer. There is also a "paired-group"-like homeodomain relatively near the N-terminus.

other information: Relationship of *oc* to *otd* indicated by co-mapping (cf. Spradling and Mahowald, 1981; Wieschaus *et al.*, 1984) and the failure of *otd*-type alleles to complement *oc* (Finkelstein *et al.*, 1990). Moreover, *otd*⁺ females have altered adult bristle pattern in ocellar region, similar to that exhibited by homozygotes for the weak allele *oc*^{db} (Finkelstein *et al.*, 1990).

***oc*^{db}: ocelliless-disturbed bristles**

phenotype: Ocelliless; ocellar, interocellar, and postvertical bristles variably extra, missing, or misplaced with about 80% penetrance; acts as a non complementing *oc* allele.

o.c.c: see *BRC*

***Ocd*: Out cold (J.C. Hall)**

location: 1-55.

origin: Induced by ethyl methanesulfonate.

references: Søndergaard, 1975, *Hereditas* 81: 199-210.

1979, *Hereditas* 90: 93-101.

1986, *Hereditas* 104: 313-16.

phenotype: Dominant cold-sensitive, reversible paralytic; heterozygous mutant females begin uncoordinated behavior (progressively: abnormal leg movements, leg stretching, wing fluttering) on shift from 25° to 18-20°; paralysis eventually occurs, and recovery is gradual on shift back to higher temperatures; critical temperature to include the debilitations is ca. 2° lower from *Ocd*⁷ than for other alleles; also males hemizygous for *Ocd*⁷ have better viability than those expressing the other alleles, with *Ocd*⁴ being the most severely affected *i.e.*, nearly lethal (*Ocd*⁴ also causes near lethality when heterozygous with any of the other alleles); at 25°, mutant males walk in reeling manner and fall over frequently; none can fly, and attempts to coax jumps (to initiate flight) cause the males merely to fall over when touched; these phenotypes also seen in homozygotes and heteroallelic combinations; certain of the homozygous mutant females (e.g. *Ocd*² and *Ocd*³) hold their wings in drooped position; also seen in *Ocd*¹ males; this phenotype also observed in *Ocd*²/⁺ and *Ocd*³/⁺ females, which also walk in unsteady manner; other alleles, when heterozygous, allow for seemingly normal behavior at 25°, except that their legs shake under etherization (with *Ocd*⁴ causing the strongest aberrant shaking). Abrupt, anomalous changes in Arrhenius activation energy of the mitochondrial enzyme, succinate cytochrome c reductase, are seen at temperatures close to those which induce paralysis (Søndergaard, FEBS Lett. 51: 126). Two-dimension gels of mitochondria isolated from *Ocd*¹ contain an additional polypeptide not seen in extracts of wild-type (Søndergaard, 1986).

cytology: Placed in 13F10-16A2 because the severe effects of any mutant allele are ameliorated in males hyperploid for *Dp(1;4)r*⁺ (13F10-16A2 appended to chromosome 4).

alleles: Seven mutants; concluded, not from problematical

complementation tests, but by failure of wild-type recombinants to be recovered from heteroallelic *Ocd* combinations. *Ocd*¹ (Søndergaard, Nelson, and Smillic, 1975, FEBS Lett. 51: 126-29) through *Ocd*⁷ (Søndergaard, 1979).

***Oce*: Ocellarless**

location: 1-5.7.

origin: Induced by triethylenemelamine (CB. 1246).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 72.

phenotype: One or both ocellar bristles, frequently postverticals, missing; other bristles, especially the scutellars, sometimes absent. *Oce/Oce* females lack 90% of postvertical and ocellar macrochaetae and 10-40% of anterior dorsocentrals; *Oce*/⁺ females lack 60-80% of ocellars and 90-95% of postverticals but show minimal loss of dorsocentral bristles; in *Hw*^{49c}/*Oce* females *Oce* appears to be epistatic to *Hw*^{49c} in the thoracic region (10% missing dorsocentrals) and additive in the head with 95% missing postverticals and 100% missing ocellars (Stoddard, 1972, DIS 48: 137-38). Wings frequently positioned abnormally, have incised margins; effect more marked in homozygous females. Bristle effect dominant. Good viability and fertility in both sexes. RK1.

other information: One allele induced by each of the following: CB. 3025, CB. 1592, CB. 1540, and CB. 1528.

ocelliless*: see *oc

****ocr*: ochracea**

location: 2-0.

discoverer: Serebrovsky, 40g25.

references: 1941, DIS 15: 19.

phenotype: Eye color lighter at eclosion, darkening with age. RK1.

Octanol dehydrogenase*: see *Odh

***Ocr*: Octopamine receptor (J.C. Hall)**

location: 3-{100}.

origin: Molecular cloning (using a putatively homologous clone from human and by synthesis of appropriate oligonucleotide probes).

synonym: *Tyr-dro*.

references: Arakawa, Gocayne, McCombie, Urquhart, Hall, Fraser, and Venter, 1990, *Neuron* 4: 343-54.

Saudou, Amlaiky, Plassat, Borrelli, and Hen, 1990, *EMBO J.* 9: 3611-17.

phenotype: Independent cDNAs were cloned into expression vectors and transfected into CHO-1 (Arakawa *et al.*, 1990) or Cos-7 (Saudou *et al.*, 1990) cells. Membranes from such cells had high-affinity binding activity to the adrenergic receptor agonist yohimbine; other agonists or antagonists used to compete with this substance showed the octopamine analog, synephrine, to have the highest agonist affinity, higher than octopamine itself; these results along with others which showed greater affinity of the cloned/transduced protein for yohimbine and chlorpromazine than for metoclopramide, suggested that this *Drosophila* receptor is an "octopamine-1" subtype (Arakawa *et al.*, 1990). Membranes preparations from the Cos-cell transfectants also had high affinity for yohimbine, and tyramine (e.g. greater than octopamine), suggesting to Saudou *et al.* (1990) that this *Drosophila*

gene encodes a tyramine receptor. Stably transfected mouse cell lines were also made (Saudou *et al.*, 1990); additions of monoamines to them (*e.g.* tyramine, octopamine) do not by themselves lead to changes in cAMP levels, but forskolin-elicited cAMP increases were reduced by added tyramine (which was more effective than octopamine). Rough anatomical localization revealed a head-enriched mRNA (3.6 kb: Arakawa *et al.*, 1990; 3.5 kb: Saudou *et al.*, 1990).

cytology: Mapped to 99A10-B1 by *in situ* hybridization (Arakawa *et al.*, 1990).

molecular biology: A cDNA encoding human brain β_2 -adrenergic receptor used in low stringency hybridizations to clone a *Drosophila* genomic fragment, partial sequence analysis of which revealed homology to adrenergic receptors (Arakawa *et al.*, 1990). A similar approach, starting with degenerate oligonucleotides corresponding to sequences found in vertebrate receptors, led to an independent genomic clone (Saudou *et al.*, 1990). Complete sequencing of head cDNAs, hybridizing to these genomic clones (Arakawa *et al.*, 1990; Saudou *et al.*, 1990), reveals a 601-amino-acid "G-protein-coupled" type of receptor protein, with highest homology to α_2 -adrenergic receptor subtypes, containing seven putative membrane-spanning regions and a N-terminal signal sequence.

other information: Saudou *et al.* (1990) call their form of the gene *Tyr-dro* (based on their different results from, and interpretation of, pharmacological experiments; see above).

od: see *os*^o

odd: odd skipped

location: 2-8.

references: Nüsslein-Volhard and Wieschaus, 1980, *Nature* 287: 795-801 (fig.).

Nüsslein-Volhard, Kluding, and Jürgens, 1985, *Cold Spring Harbor Symp. Quant. Biol.* 50: 145-54.

phenotype: Embryonic lethal; posterior part of the denticle band and adjacent naked cuticle replaced by mirror-image duplication of the anterior part of the denticle band and adjacent naked cuticle in T2, A1, A3, A5, and A7. Pole-cell-transplantation experiments indicate that *odd*⁺ not maternally required. Positional expression of *ftz* unaffected (Carroll and Scott, 1986, *Cell* 54: 113-26). However, stripes of *en* expression are broader than normal in even-numbered, but of normal width in odd-numbered parasegments; *Ubx* expression is consequently low in broader-than-normal stripes in even-numbered parasegments (Martinez-Arias and White, 1988, *Development* 102: 325-38).

alleles:

allele	origin	synonym	ref ^α	comments
odd¹	EMS	<i>odd^{7L}</i>	2, 3	amorph
odd²	EMS	<i>odd^{9P}</i>	2, 3	hypomorph
odd³	X ray	<i>odd^{1.36}</i>	1, 3	
odd⁴	EMS	<i>odd^{III C}</i>	3	
odd⁵	EMS	<i>odd^{IIID}</i>	3	

^α 1 = Nüsslein-Volhard, Kluding, and Jürgens, 1985, *Cold Spring Harbor Symp. Quant. Biol.* 50: 145-54; 2 = Nüsslein-Volhard and Wieschaus, 1980, *Nature* 287: 795-801 (fig.); 3 = Tearle and Nüsslein-Volhard, 1987, *DIS* 66: 209-69.

cytology: Located in 23E-24B; to the left of the breakpoint

of *T(1;2)1.10* = *T(1;2)4A3-4;24B* and within the transposed segment of *Tp(2;?)5.1* = *Tp(2;?)23E;24E* (Nüsslein-Volhard, Wieschaus, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 267-82).

odd paired: see *opa*

odd skipped: see *odd*

Odh: Octanol dehydrogenase

location: 3-49.2.

discoverer: Ursprung.

references: Ursprung and Leone, 1965, *J. Exp. Zool.* 160: 147-54.

Courtright, 1966, *DIS* 41: 59.

Courtright, Imberski, and Ursprung, 1966, *Genetics* 54: 1251-60.

phenotype: The structural gene for octanol dehydrogenase [ODH (EC. 1.1.1.73)], a multimer of 109,000 molecular weight (Sieber, Fox, and Ursprung, 1972, *FEBS Lett.* 26: 274-76); evidence for tetrameric structure in other *Drosophila* species (Pipkin, 1969, *Genetics* 63: 405-18); immunologically unrelated to ADH (Courtright, 1968, *DIS* 43: 144). Preferred substrates are long-chain primary alcohols, with lesser activity on short-chain and branched-chain primary alcohols; inactive on secondary alcohols (Bremner, Douglas, and Ogonji, 1971, *DIS* 47: 93-94). Enzyme activity higher in adults than in larvae or pupae (Debec, 1974, *Wilhelm Roux's Arch. Dev. Biol.* 174: 1-9); mobility also increases from larvae to pupae to adults (Hewitt, 1974, *Genetics* 77: s30-31). *Odh*⁺ not a vital gene; homozygotes for null allele, *Odh*^{nNC1}, viable and fertile (Voelker, Langley, Leigh-Brown, Ohnishi, Dickson, Montgomery, and Smith, 1980, *Proc. Nat. Acad. Sci. USA* 77: 1091-95).

alleles: Existing alleles recovered from natural populations; designated according to their migration rate toward the cathode in agar gel electrophoresis. *Odh*^F the predominant allele; the incidence of *Odh*^S in populations decreases with distance from the equator (Oakeshott, Gibson, Wilcocks, and Chambers, 1983, *Theoret. Appl. Genet.* 65: 191-96).

allele	synonym ^α	ref ^β	comments
Odh^F	<i>Odh^M</i>	1, 2	fast allele
Odh^{nNC1}		3	null allele
Odh^R		1	rare allele
Odh^S	<i>Odh^L</i>	1, 2	slow allele

^α Because Costa *et al.* used a different gel system in which migration was anodal, the designations *F* and *S* were not descriptive; accordingly they use *M* = most common allele, *L* = less common allele, and *R* = rare allele, which encodes a polypeptide whose anodal migration is slower than that produced by *Odh*^F.

^β 1 = Costa, Danieli, and Rodino, 1977, *DIS* 52: 92; 2 = Courtright, Imberski, and Ursprung, 1966, *Genetics* 54: 1251-60; 3 = Voelker, Langley, Leigh-Brown, Ohnishi, Dickson, Montgomery, and Smith, 1980, *Proc. Nat. Acad. Sci. USA* 77: 1091-95.

cytology: Localized to 86D1-4 based on its inclusion in *Df(3R)M86D* = *Df(3R)86D1;86D4* (Clark, 1983, *Biochem. Genet.* 21: 375-90).

odsy: see *os*

Of: see *Dl^{Of}*

***Off: Off**

location: 2-82.

origin: Spontaneous.

discoverer: Bridges, 23e14.

references: Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 232.

phenotype: Some bristles missing in heterozygotes, especially from side of abdomen; basal rings remain as in *H.* Homozygote lacks more bristles. Eyes large, creased, and roughened. RK2.

other information: Agrees with *abr* in locus and description; may have been an allele.

ogre: optic ganglia-reduced (J.C. Hall)

location: 1-18.8.

references: Niklas and Cline, 1983, *Genetics* 103: 617-631.

Lipshitz and Kankel, 1985, *Dev. Biol.* 108: 50-77.

Watanabe and Kankel, 1990, *Genetics* 126: 1033-44.

phenotype: Original allele (*ogre*¹) recovered on the basis of simple lethality (Niklas and Cline, 1983); viable mutant (*ogre*²) isolated with respect to inability of adults to orient to vertical line of black-white contrast (Lipshitz and Kankel, 1985); optic lobes generally disorganized, as seen in adults expressing the viable allele, or in late pupae/pharate adults expressing a lethal allele; such lethals (all except *ogre*²) cause the behavioral and anatomical abnormalities just noted when heterozygous with viable allele; viability associated with one of these heteroallelic types, *i.e.*, *ogre*¹/*ogre*² is poor, especially when reared at 18°C (*vs* 29°C); temperature shift experiments using this combination implies gene action in late larval stage; hemizyosity for lethal alleles causes development to cease in late larval-pupal stages, when parts of CNS appear abnormal (*e.g.*, holes in sections of brain and thoracic ganglia, with severe defects appearing in the optic lobe formation centers); holes in CNS are also seen in "escapers", *e.g.*, rare adults hemizygous for alleles other than *ogre*², or heterozygous for *ogre*¹ and *ogre*²; mosaic analysis suggests that CNS defects are due to action of this gene in those developing tissues (Lipshitz and Kankel, 1985).

alleles: Five mutant alleles, four of which are embryonic lethal.

allele	origin	discoverer	synonym	ref ^α	comments
<i>ogre</i> ¹	EMS	Niklas	<i>l(1)jnL3</i>	3	
<i>ogre</i> ²	EMS	Kankel	<i>ogre^{vcb8}β</i>	1, 2	viable allele
<i>ogre</i> ³	ENU	Lipshitz	<i>l(1)ogre^{ij555}</i>	2	
<i>ogre</i> ⁴	ENU	Lipshitz	<i>l(1)ogre^{ll523}</i>	2	
<i>ogre</i> ⁵	spont	Schalet	<i>l(1)l18-183</i>	4, 5	10 kb deficiency

^α 1 = Kankel and Lipshitz, 1981, *Proc. 7th Int. Symp. Div. Biophys. The Taniguchi Foundation* (Y. Hotta, ed.), pp. 215-38; 2 = Lipshitz and Kankel, 1985, *Dev. Biol.* 108: 56-77; 3 = Niklas and Cline, 1983, *Genetics* 103: 617-31; 4 = Schalet, 1986, *Mutat. Res.* 163: 115-44; 5 = Watanabe and Kankel, 1990, *Genetics* 126: 1033-44.

^β *vcb* = viable contrast blind.

cytology: Placed in 6E2-4 based on its inclusion in *Df(1)Sxl-bt = Df(1)6E2;7A6* but not *Df(1)HA32 = Df(1)6E4-5;7A6*.

molecular biology: Isolated (Watanabe and Kankel, 1990) by walking distally from clone mapping to 6E4-5. Walk crosses distal breakpoints of *Df(1)HA32 (= ogre⁺)* and of *Df(1)Sxl-bt (= ogre⁻)*, which are 70 kb apart. Five tran-

scripts from this interval were detected on Northern blots of RNA from embryos and larvae; one mRNA of 2.9 kb was reduced in abundance in larvae hemizygous for *ogre*¹; precise mapping of the *ogre*⁵ deletion indicated it would be missing at least part of this 2.9 kb transcript (and two other RNAs, including an 1.35 kb species). Transformation with a 12.5 kb genomic fragment, which should include the source of the 2.9 kb mRNA, rescues *ogre*-associated lethality and the optic lobe abnormalities that can be seen in pharate adults expressing *ogre*¹; these transformants, in an *ogre*⁵ genetic background, exhibit restoration of the entire 2.9 kb, but only part of the 1.35 kb mRNA. cDNAs were cloned from embryonic libraries, and sequencing of the largest insert (2.45 kb) indicated that it can encode the entire protein, a 362-amino-acid polypeptide; features of the conceptual translation product include a putative membrane spanning region and a potential glycosylation site; there were no significant similarities, in data bases, to the *ogre* sequence.

okr: okra (T. Schüpbach)

location: 2-1.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus.

phenotype: Female sterile; homozygous females lay eggs which are of variable shapes; in the most extreme cases, the eggs are longer than normal, more pointed at the posterior end, and lack dorsal appendages, resembling eggs produced by the dominant female-sterile mutation *Fs(2)D*.

alleles: Two, *okr^{RU} = okr¹*, *okr^{WS} = okr²*.

ojos castaños: see *cast*

ol-2: see *sp*

olfactory: see *olf*

olfactory pathway: see *mbmC*

olfactory-trap-abnormal: see *ota*

olfA: olfactory-A (J.C. Hall)

location: 1-21.0 (from cytology; maps between *ct* and *sn* meiotically).

origin: Induced by ethyl methanesulfonate.

references: Rodrigues and Siddiqi, 1978, *Proc. Indian Acad. Sci. B* 87: 140-60.

Rodrigues, 1980, *Development and Neurobiology of Drosophila* (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York, pp. 361-69.

Venard and Pichon, 1984, *J. Insect Physiol.* 30: 1-5.

Venard, Antony, and Jallon, 1989, *Neurobiology of Sensory Systems* (Singh and Strausfeld, eds.). Plenum Press, New York, pp. 377-85.

Ayyub, Paranjape, and Siddiqi, 1990, *J. Neurogenet.* 6: 243-62.

phenotype: Larvae and adults are relatively poorly repelled by aldehydes (Rodrigues, 1980; benzaldehyde usually employed in the relevant Y-tube/olfactometric tests); strongest allele, in this regard is *olfA^{x1}*, and yet it still detects these odorants (Ayyub *et al.*, 1990). Responses to ethyl acetate, acetone, acetic acid, and ethanol are normal (first of these tested in adults and larvae, the latter three only in larvae). In odor-induced jump assay, mutants show about half-normal frequency

of such responses (Ayyub *et al.*, 1990). Electroantennogram recordings from adults indicate somewhat reduced response to benzaldehyde, but normal ones to butanol and butyl acetate (Venard and Pichon, 1984). The physiological defect seen, with benzaldehyde as the stimulus, involves a biphasic dose-response curve (Venard *et al.*, 1989), as if there are two kinds of antennal receptors for this substance, only one of them being affected by *olfA*.

alleles:

allele	discoverer	ref ^α
<i>olfA</i> ^{x1}	Rodrigues	1
<i>olfA</i> ^{x6}	Rodrigues	1
<i>olfA</i> ^{x8}	Rodrigues	1
<i>olfA</i> ^{x11}	Rodrigues	1
<i>olfA</i> ^{x24}	Ayyub	2
<i>olfA</i> ^{x25}	Ayyub	3

^α 1 = Rodrigues, 1980, Development and Neurobiology of *Drosophila* (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York, pp. 361-69. 2 = Ayyub, Paranjape, and Siddiqi, 1990, J. Neurogenet. 6: 243-62.

cytology: Maps to 7B8-D1 (Ayyub *et al.*, 1990), based on its inclusion in the region of overlap of $Dp(1;2)sn^{+72d} = Dp(1;2)6C;7C9-D1$ and $Dp(1;3)sn^{13al} = Dp(1;3)7A8;-8A5$ and its complementation by $Df(1)ct268-42 = Df(1)7A5-6;7B8-C1$.

other information: Original group of these mutations reported initially by Rodrigues and Siddiqi (1978), but not then noted as "*olfA*" (see Rodrigues, 1980, and subsequent references listed above). *olfA* alleles complement the closely linked *olfC* and *olfE* mutations.

***olfB*: olfactory-B (J.C. Hall)**

location: 1-{0.5}.

origin: Induced by ethyl methanesulfonate.

discoverer: Rodrigues.

references: Rodrigues and Siddiqi, 1978, Proc. Indian Acad. Sci. B 87: 147-60.

Rodrigues, 1980, Development and Neurobiology of *Drosophila* (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York, pp. 361-69.

Ayyub, Paranjape, Rodrigues, and Siddiqi, 1990, J. Neurogenet. 6: 243-62.

phenotype: Similar phenotype to *olfA* (Rodrigues, 1980), but subnormal responses of larvae or adults to aldehydes [benzaldehyde and formaldehyde plus salicylaldehyde as well (Ayyub *et al.*, 1990)] seen only after rearing at 28° (permissive temperature in these tests, first performed by Rodrigues, 1980, extended by Ayyub *et al.*, 1990, was 22°). Larvae and adults grown at non-permissive temperature normal in responses to ethyl acetate, acetic acid, ethanol and acetone (Ayyub *et al.*, 1990). Physiological responses of antennae to aldehydes briefly noted to be normal by Siddiqi [1984, Genetics, New Frontiers (Chopra, Sharma, Joshi, and Bansal, eds.). Oxford and IBH Publishing Co., New Delhi, Vol. III, pp. 243-61].

alleles: One mutant allele, *olfB*^{x4}.

cytology: Maps to 2B17-C2 (Ayyub *et al.*, 1990), based on complementation by $Df(1)w-vco$ (2C1;3C5) but not by $Dp(1;3)w^{vco}$ (2B17-C1;3C5-6;77D3-5;81).

other information: Complements *olfA*, *olfE*, and *olfF*. The one extant mutant allele, now known as *olfB*^{x4}, originally reported by Rodrigues and Siddiqi (1978), but not then called *olfB* (see Rodrigues, 1980).

***olfC*: olfactory-C (J.C. Hall)**

location: 1-21.0. (from cytology, meiotically maps between *cv* and *sn*, closer to the latter).

origin: Induced by ethyl methanesulfonate.

discoverer: Rodrigues.

references: Rodrigues and Siddiqi, 1978, Proc. Indian Acad. Sci. B 87: 147-60.

Rodrigues, 1980, Development and Neurobiology of *Drosophila* (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York, pp. 361-69.

Tompkins and Hall, 1981, Z. Naturforsch 36c: 694-96.

Mane, Tompkins, and Richmond, 1983, Science 222: 419-21.

Venard and Pichon, 1984, J. Insect Physiol. 30: 1-5.

Curcillo and Tompkins, 1987, Behav. Genet. 17: 81-86.

Venard, Antony, and Jallon, 1989, Neurobiology of Sensory Systems (Singh and Strausfeld, eds.). Plenum Press, New York, pp. 377-85.

Ayyub, Paranjape, Rodrigues, and Siddiqi, 1990, J. Neurogenet. 6: 243-62.

phenotype: Adults show poor responses to acetates and acetone, and have diminished responses to some alcohols, in tests involving Y-tube olfactometer (Rodrigues, 1980). Odor-induced jump assays on *olfC*^{x3} and *olfC*^{x17} adults (Ayyub *et al.*, 1990) gave results paralleling those using olfactometer (whereby these mutants are in one of two different "acetate defective" categories; see alleles). Larvae respond abnormally to acetates and normally to aldehydes (Rodrigues, 1980). Electroantennograms (EAGs) recorded from adults show *olfC* to exhibit diminished responses of olfactory receptors to acetates (Venard and Pichon, 1984) and to 2-butanone as well (Venard *et al.*, 1989). Similar physiological [Siddiqi, 1984, Genetics: New Frontiers (Chopra, Sharma, Joshi, and Bansal, eds.). Oxford and IBH Publishing Co., New Delhi, Vol. III, pp. 243-61], and also behavioral (Rodrigues, 1980), experiments involving pairs of odorants, *e.g.* ethyl and iso-amyl acetate, suggested more than one independent "channel" for the reception of these substances. In SEM observations (Venard *et al.*, 1989) mutant antenna seems to have normal number and distribution of the three kinds of sensilla on the anterior face of the funiculus (from where EAGs recorded). Electrophoresis of triton extracts of antennae generates an extra band of esterase activity in *olfC*, which is found in neither wild-type nor in mutant thoraces and abdomens (Venard *et al.*, 1989). *olfC* males fail to have their courtship of wild-type males inhibited by high concentrations of volatile compounds (unlike normal males, which are inhibited). Mutant males court other males with abnormally high vigor yet court females with subnormal intensity (Tompkins *et al.*, 1981). Further studies showed that the mutant courts immature males vigorously (as do wild-type males), and this wanes as the courtee ages, but not to the same extent as in normal pairings; the inappropriately high levels of *olfC* courtships directed at maturing males includes all sex behaviors except attempted copulation (Curcillo and Tompkins, 1987). An inability of mutant males to discriminate between recently mated and virgin females was reported by Mane *et al.* (1983), who also showed that *olfC* males can detect, 6 hours post insemination, a difference between females mated to *Est-6*⁰ males *vs.* males carrying a non-null allele of this gene (the latter kind of mated females were said to be relatively inhibi-

tory to male courtship); these experiments suggested that the enzyme encoded by *Est-6* turns a "pre-anti-aphrodisiac" compound, cis-vaccenyl acetate, which is transferred from males to females during copulation into a further, or the actual aphrodisiac, cis-vaccenyl alcohol; some elements of such results and inferences (Mane *et al.*, 1983) have been called into question (Vander Meer, Obin, Zawistowski, Sheehan, and Richmond, 1986, *J. Insect Physiol.* 32: 681-86; Scott and Richmond, 1987, *J. Insect Physiol.* 33: 363-69) but argued by others [Ferveur, Cobb, and Jallon, 1989, *Neurobiology of Sensory Systems* (Singh and Strausfeld, eds.). Plenum Press, New York, pp. 377-85, pp. 397-409] to still have force, at least insofar as an anti-aphrodisiac role for cis-vaccenyl acetate goes.

alleles:

allele	discoverer	comments
<i>olfC^{x2}</i>	Rodrigues	reduced attraction to both ethyl and iso-amyl acetate
<i>olfC^{x3}</i>	Rodrigues	reduced attraction to both ethyl and iso-amyl acetate
<i>olfC^{x5}</i>	Rodrigues	normal response to ethyl acetate; reduced to iso-amyl acetate
<i>olfC^{x10}</i>	Rodrigues	like <i>olfC^{x2}</i> and <i>olfC^{x3}</i>
<i>olfC^{x13}</i>	Rodrigues	not mentioned anymore by Ayyub <i>et al.</i> (1990)
<i>olfC^{x14}</i>	Ayyub	like <i>olfC^{x5}</i>
<i>olfC^{x17}</i>	Ayyub	like <i>olfC^{x5}</i>

cytology: Maps to 7D1-6 (Ayyub *et al.*, 1990); *olfC^{x3}* and *olfC^{x17}* uncovered by *Df(1)C128 = Df(1)7D1;7D5-6*.

other information: Originally isolated (Rodrigues and Siddiqi, 1978) in the same "then-unnamed" manner as *olfA*, *olfB* and *olfD* (the latter = *sbl*). *olfC^{x3}* over *olfC* deletion behaves with same poor responses to acetates as does homozygous mutant; however, *olfC^{x17}* female hemizygotes defective in responses to both ethyl and iso-amyl acetates, whereas, this allele in homozygous females allows for normal responses to ethyl acetate. Mutagenesis of *olfC^{x17}* led to some strains with "stronger" acetate-based olfactory deficit than the starting mutant. In one of these called *olfC^{x17-1a}*, responses to iso-amyl acetate worse than in *olfC^{x17}*, and the new strain also shows reduction in responses to benzaldehyde; both of these abnormalities covered by *Dp(1;2)sn⁺72d = Dp(1;2)6C;7C9-D1* and uncovered by *Df(1)ct-J4 = Df(1)7A2-3;7C1*, apparently placing this novel genetic defect in 7A2-C1, a region separate from *olfC*.

olfD: see *sbl*

olfE: olfactory-E (J.C. Hall)

location: 1-{21.0}.

origin: Induced by ethyl methanesulfonate.

discoverer: Ayyub.

references: Hasan, 1989, *J. Genet.* 68: 139-46.

Ayyub, Paranjape, Rodrigues, and Siddiqi, 1990, *J. Neurogenet.* 6: 243-62.

Hasan, 1990, *Proc. Nat. Acad. Sci. USA* 87: 9037-41.

phenotype: Larvae and adults show reduction in responses to benzaldehyde in petri-plate-based and Y-tube olfactometric assays, respectively. In odor-induced-jump assay (using benzaldehyde), mutants jump less than half as often as normal flies.

allele: There is one extant allele, *olfE^{x26}*.

cytology: Best estimate of location is 7C9;7D1 (Ayyub *et al.*, 1990; Hasan, 1989, 1990). The gene is covered by *Dp(1;2)sn⁺72d = Dp(1;2)7B1;8A5*. It is also covered by *Dp(1;3)sn^{13a1} = Dp(1;3)6C5;7C9;79E* and uncovered by *Df(1)C128 = Df(1)7D1;7D5-6*, rearrangements that overlap according to molecular data.

molecular biology: Cloned by Hasan (1989, 1990) using chromosomal walking beginning with *sn*-region probes. Southern-blot mapping of proximal breakpoint of *Dp(1;3)sn^{13a1}* and the distal breakpoint of *Df(1)C128* defines a 25 kb interval that includes *olfE* (see cytology). A 5.4 kb transcript detected with a probe relatively near (but distal to) the 7D9 breakpoint of *Dp(1;3)sn^{13a1}*, is believed to represent *olfE* mRNA (Hasan, 1990). This transcript is found throughout the life cycle; it is in the head and bodies of adults. Another mRNA of 1.7 kb hybridizes to probes that detect the 5.4 kb transcript. A 14 kb genomic fragment that includes the 5.4 kb transcript partially rescued olfactory defects. In jump assays, more normal phenotypes were obtained, when the DNA insert was homozygous; in Y-tube tests, homozygosity was required for any appreciable rescue.

other information: *olfE* complements *olfA*, *olfB*, and *olfF*. The closely-linked mutants *olfA* and *olfE* are both impaired in their responses to benzaldehyde.

olfF: olfactory-F (J.C. Hall)

location: 1-{1}.

origin: Induced by ethyl methanesulfonate.

discoverer: Ayyub.

references: Ayyub, Paranjape, Rodrigues, and Siddiqi, 1990, *J. Neurogenet.* 6: 243-62.

phenotype: Compared to wild-type flies, *olfF* adults require a higher concentration of benzaldehyde for detection in Y-tube olfactometer (*e.g.* no response at a dilution of 10⁻⁵, which readily repels wild-type flies). Mutant shows normal responses to acetates, propionic acid and butanol. *olfF/Df(1)w-vco* females are fertile.

alleles: There is one extant allele, *olfF^{x267}*.

cytology: Located in 2E1-3C2 since uncovered by *Df(1)64c18 = Df(1)2E1-2;3C2*.

other information: *olfF* complements *olfA*, *olfB*, and *olfE*.

olive-2: see *sp*

olv^D: see *dp^D*

***om: ommatidia**

location: 1-0.1 (to the right of *sc*).

origin: X ray induced in (or with) *ac³*.

discoverer: Muller.

references: Muller, Prokofyeva, and Raffel, 1935, *Nature* 135: 253-55.

Muller, 1935, *DIS* 3: 30.

phenotype: Ommatidia disarranged, giving a slight eye roughness difficult to classify. RK3.

cytology: Thought by Muller to be in or very close to 1C1.

omb: optomotor-blind (J.C. Hall)

location: 1- {7.5}.

origin: Induced by ethyl methanesulfonate.

discoverer: Heisenberg.

synonym: *omb^{H31}*; *opm31*.

references: Heisenberg, Wonneberger, and Wolf, 1978, *J. Comp. Physiol.* 124: 287-96.

Blondeau and Heisenberg, 1982, *J. Comp. Physiol.*

145: 321-29.

Bausenwein, Wolf, and Heisenberg, 1986, *J. Neurogenet.* 3: 87-109.

Pflugfelder, Schwartz, Roth, Poeck, Sigl, Kerscher, Jonschker, Pak, and Heisenberg, 1990, *Genetics* 126: 91-104.

phenotype: Adults expressing *omb*¹, the only allele studied in phenotypic detail, are impaired in optomotor turning responses in tests involving tethered or freely moving flies (Heisenberg *et al.*, 1978; Blondeau and Heisenberg, 1982; Kulkarni, Steinlauf, and Hall, 1988, *Genetics* 118: 267-285; Dushay, Rosbash, and Hall, 1989, *J. Biol. Rhythms* 4: 1-27). The mutant is also aberrant in orientation to vertical stripe (Heisenberg *et al.*, 1978). More detailed examination of "yaw torque" optomotor responses show that *omb*¹ is restricted in responses to stimulation of "frontal visual field", with mutant behavior summarized as retaining "object responses" but missing "large field responses" (Bausenwein *et al.*, 1986). The mutant is relatively normal in "lift/thrust" response to vertical pattern movement and in regard to landing response elicited by front-to-back horizontal motion. Slow phototaxis, using Y-tube, was markedly subnormal (Dushay *et al.*, 1989). Giant arborizing fibers in lobula plate. Since isolation and the original histological examinations, anatomical abnormalities have become more pronounced, *e.g.*, the optic lobes remain in "pre-imaginal" orientation in some *omb* adults, *viz.*, long axis of medulla optic ganglia oriented more frontally than in wild-type (Blondeau and Heisenberg, 1982); other behavioral abnormalities: *omb* shows anomalous avoidance reaction, *i.e.*, "antifixation" to objects in Y-maze test (Bulthoff, 1982, *Biol. Cybernet.* 45: 63-70); courting males exhibit diminished tracking responses of and turning responses to moving females (Cook, 1980, *Biol. Cybernet.* 37: 41-51; Tompkins, Gross, Hall, Gailley, and Siegel, 1982, *Behav. Genet.* 12: 295-307). Visual stimulus-induced metabolic activity in the optic lobes (monitored by distribution of radioactive 2-deoxyglucose) is normal, suggesting that basic structure and function of ganglia distal to lobula plate is normal (Bulthoff and Buchner, 1985, *J. Comp. Physiol.* 156: 25-34). Without effect on locomotor activity rhythm (Helfrich, 1986, *J. Neurogenet.* 3: 321-43).

alleles: Only one viable allele, *omb*¹ (isolated as *omb*^{H31}). A number of lethal rearrangements with breakpoints in 4C5-6 uncover *omb* along with other closely linked mutations.

cytology: Placed in 4C5-6 based on its inclusion in *Df(1)GA56 = Df(1)4C5-6;4D1* and *Df(1)rb13 = Df(1)4C5-6;4D3-E1* but not *Df(1)ovo6 = Df(1)4C5-6;4E2-3* or *Df(1)ovo7 = Df(1)4C5-6;4E2-3* (Pflugfelder *et al.*, 1990). Associated with *In(1)omb = In(1)4C4-7;12D2-E1*.

molecular biology: Located in an 340-kb walk initiated from polytene bands microdissected from 4B-C (Pflugfelder *et al.*, 1990). Seven *omb*⁻ breakpoints identified in a 80-kb subsegment; in distal-to-proximal order, they are *T(1;3)bi*^{D1}, *Tp(1;1)bi*^{D1} (which contains a 30 kb insert within the *omb* region), *T(1;2)bi*^{D2}, *In(1)omb*, *Df(1)GA56*, *Df(1)rb13*, and *Df(1)rb5*; these are spread over a distance of 80 kb. The distal breakpoint of *Df(1)ovo7* (which is *omb*⁺) is 15 kb proximal to that of *Df(1)rb5* (which is *omb*⁻ and was mapped within 4C5-6

by Southern blots, though no cytological breakpoints for it are given). Northern blot probes (Pflugfelder *et al.*, 1990), encompassing all breakpoints that cause an *omb* phenotype, detected two transcripts that could be associated with the *omb* function. T3 (6 kb) and T7 (an approximately 2-kb smear); the genomic sources of these RNAs are 80 kb apart; they are most prominent in mid-embryos, late L3, early pupae, and adults; another fainter transcript (T7'), with a similar developmental profile, comes from a more proximal region to the right of the *In(1)omb* breakpoint. There are also several additional low-abundance embryonic/pupal transcripts that map between two relatively distal *omb*⁻ breakpoints. Various cDNAs have been cloned, including those representing T3 and T7; the modest amount of sequencing done, as of Pflugfelder *et al.* (1990), revealed no significant matches.

other information: Two EMS-induced lethals with no physically detectable lesions within the relevant portion of the walk noted above fail to complement *bi*, *Qd*, *lac*, *omb*, and *l(1)bi*, the designation applied to the lethality of several of the rearrangements in the region; these complementation groups overlap the distal 20 kb of the *omb* region. The lethals are designated *l(1)omb*²⁸² and *l(1)omb*³¹⁹⁸ by Pflugfelder *et al.* (1990), but the law of parsimony suggests that they be designated *l(1)bi*¹ and *l(1)bi*², respectively. Other observations suggesting that the *omb* region is genetically complex (Pflugfelder *et al.*, 1990) are exemplified by the fact that *l(1)bi*¹/*Qd* females have lacquered wings.

ome: omega

location: 3-36.

origin: Induced by ethyl methanesulfonate.

references: Chihara and Kimbrell, 1986, *Genetics* 114: 393-404.

phenotype: In the presence of homozygous *omega*, larval cuticle protein 5 migrates more slowly, irrespective of the *Lcp5* alleles present; migration of the residual *Lcp5* bands also altered. *omega*⁺ product postulated to play a role in modifying the *Lcp5* gene product.

oml: ommatidiless

location: 1-31.

origin: X ray induced.

discoverer: Ritterhoff.

references: Biggin, 1969, *DIS* 44: 49.

phenotype: Expression varies from normally shaped eyes with displaced facets to complete absence of eyes; variable penetrance; ventral part of eye most often affected. 10% of flies show palps in epidermis that replaces missing eye tissue; 5% show reduced head, fused ocelli, and abnormal antennae.

omm: ommatoreductum

location: 1-12.8.

origin: Induced by triethylenemelamine (CB. 1246).

discoverer: Fahmy, 1953.

references: 1958, *DIS* 32: 72.

phenotype: Some peripheral ommatidia absent, frequently in an irregular manner, giving a rough eye and a notched border. Shape of head abnormal; head bristles deranged or absent. Palps absent or deformed. Thoracic bristles deranged. Wings often unexpanded. Good viability and fertility in both sexes. RK2.

other information: One allele each induced by CB. 1246,

CB. 1522, CB. 1592, CB. 1528; two alleles induced by CB. 3026.

ommatidia: see *om*

ommatidiless: see *oml*

ommatoeductum: see *omm*

***On: Open**

location: 3-26.

origin: X ray induced.

discoverer: Tanaka, 36c26.

references: 1937, DIS 7: 21.
1937, DIS 8: 11.

phenotype: Wings spread. Homozygous viable. RK2.

***op: opaque**

location: 1-50.

origin: X ray induced.

discoverer: H. M. Miller, 33k.

references: 1934, DIS 2: 9.
1935, DIS 3: 14.
1935, DIS 4: 10.

phenotype: Wings opaque and whitish, usually divergent and slightly convex. Viability and fertility good in male, poorer in female. RK3.

opa: odd paired

location: 3-48.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

Nüsslein-Volhard, Kluding, and Jürgens, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 145-54.

phenotype: Deletes alternate metasegments, as defined by a line separating the anterior from the posterior parts of the anterior compartments; portions of denticle bands of T2, A1, A3, A5, and A7 and naked cuticle of T3, A2, A4 and A6 missing. Pattern of *Ubx* protein distribution in the double-sized units reveals them to be composite, the anterior half being derived from an odd-numbered parasegment and the posterior half from an even-numbered parasegment (read metasegment) (Ingham and Martinez-Arias, Nature 324: 592-97). Without discernable effect on *ftz* expression (Carroll and Scott, 1980, Cell 45: 113-26). However, exhibits loss of *en* product with a consequent increase in *Ubx* expression in even-numbered parasegments (Martinez-Arias and White, 1988, Development 102: 325-38).

alleles: Nine ethyl methanesulfonate-induced alleles, *opa*¹-*opa*⁹, isolated as *5H*, *7N*, *9C*, *9O*, *13D*, *E8*, *IIC*, *IIP*, and *T* (Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-69).

cytology: Placed in 82A-E; between autosomal breakpoints of *T(Y;3)J17 = T(Y;3)Xhy⁺;82A* and *T(Y;3)D107 = T(Y;3)h7;82E*.

opaque: see *op*

***opb: opaque broad**

location: 1-28.3.

origin: Induced by L-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1955.

references: 1959, DIS 33: 88.

phenotype: Short, broad, and opaque wings with slightly

convex or concave membranes. Slightly brownish eye color. Legs short with long segments frequently bowed. Abdomen slightly abnormal in shape; genitalia deformed. Males fertile; viability about 10% wild type. Females sterile. RK3.

Open: see *On*

***oph: ophthalmopedia**

location: 2-45.

origin: Spontaneous.

discoverer: Gordon, 1934.

references: 1936, J. Genet. 33: 25-60.
1941, DIS 14: 39.

phenotype: In extreme form, an appendage grows from eye; in less extreme form, eye is kidney shaped. Expression sensitive to genetic and environmental modification. Effect caused by enlargement and abnormal folding of eye-forming portion of optic disk in late larvae [Waddington and Pilkington, 1943, J. Genet. 45: 44-50 (fig.)]. Disc development autonomous in reciprocal transplants with wild type (Abaturova and Ginter, 1968, Genetika 4(11): 58-64); partial *oph* disks display reduced mutant development when transplanted into wild-type hosts (Ginter and Abaturova, 1969, Genetika 5(10): 38-43). RK3.

oph: ophthalmoptera

location: 1-5.

origin: Spontaneous.

references: Ouweneel, 1970, Genetika 41: 1-20.

phenotype: A semidominant enhancer of *ld* and *Dfd*^r and perhaps other eye-shape mutants. In the presence of *oph*, flies bearing the above mutants produce wing-like outgrowths in the eye. Expression more extreme at 17° than at 29°; temperature sensitive period in second- and third-instar larvae (Postelthwaite, 1974, Dev. Biol. 36: 212-17).

Ophthalmoptera: see *Opt*

opm2: see *nonA*

opm3: see *rdgA*¹²

opm4: see *rdgA*¹³

opm5: see *rdgA*¹⁴

opm8: see *t*^{H8}

opm9: see *rdgA*¹⁵

opm10: see *rdgA*¹⁶

opm14: see *rdgA*¹⁷

opm18: see *nonB* or *nbA*

opm24: see *t*^{H24}

opm37: see *elf*

opm47: see *nbA*

opm52: see *norpA*

Opt: Ophthalmoptera

location: 2-68 (based on progeny testing of 32 *Bl-c* recombinants from *Opt^B/Bl c* females).

phenotype: A homeotic modifier of *ey*² and possibly other small-eye mutants (e.g., *ey^{D/+}*). In the presence of *Opt*,

*ey*² flies exhibit protrusions of wing tissue from the region of the eye; development is normal at 17°, but homeotic transformation is observed at 29°; the temperature-sensitive period is during the second larval instar [Postlethwait, 1974, *Dev. Biol.* 36: 212-17 (fig.)]. Protrusions from the anterior portion of the eye have the triple row of bristles characteristic of the anterior wing margin; the fronto-orbital region of the head produces bristles with bracts characteristic of the costa in the proximal wing (Postlethwait).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
Opt^B	EMS	Bacher, 1966		3	
Opt^G	spont		<i>ey^{opt}</i>	1, 2	

^α 1 = García-Bellido, 1969, *DIS* 44: 52 2 = E. Goldschmidt and Lederman-Klein, 1958, *J. Hered.* 49: 262-66; 3 = Lewis and Bacher, 1969, *DIS* 44: 48.

other information: Allelism not tested; assumed from similarity of phenotype and location on the second chromosome.

optic ganglia-reduced: see *ogre*

optomotor: see *opm*

optomotor-blind: see *omb*

or: orange

location: 2-106.7 [0.3 unit to the right of *sp* based on 11 recombinants among 3317 flies (Ives, 1967, *DIS* 43: 400)].

phenotype: Eye color bright orange. *or/ptd* wild type (Von Halle). 5% normal complement of drosopterin; implantation of phenylalanine crystals in pupal abdomen one day before eclosion results in bright red eye with a four-fold increase in drosopterin content (Schwinck, 1969, *Genetics* 61: s53). RK1.

alleles: *or^{45a}* and *or^{49h}* weaker alleles than *or¹* and *or^{66k}* (Schwinck, 1969, *DIS* 4: 46).

allele	origin	discoverer	ref ^α
or¹	spont	Mossige, 1942	2
or^{45a}	spont	Ives, 45a	1
or^{49h}	spont	Ives, 49h31	1
or^{66k}	spont	Schwinck, 66k20	3

^α 1 = Ives, 1951, *DIS* 25: 70; 2 = Mossige, 1950, *DIS* 24: 61; 3 = Schwinck, 1964, *DIS* 44: 46.

ora: see *ort*

Double mutant *ort¹ ninaE¹* described as *ora^{JK84}*; components separable by recombination.

ora transientless: see *ort*

ord: orientation disruptor (R.S. Hawley)

location: 2-103.5.

origin: Induced by ethyl methanesulfonate.

references: Baker and Hall, 1976, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 352-434.

Mason, 1976, *Genetics* 84: 545-72.

Baker, Carpenter, and Ripoll, 1978, *Genetics* 90: 531-78.

Goldstein, 1980, *Chromosoma* 78: 79-111.

phenotype: In females homozygous for *ord*, exchange is strongly reduced. In both males and females, *ord* causes

a dramatic increase in both reductional and equational nondisjunction as assayed genetically. In male meiosis, Goldstein observed abnormal sister-chromatid associations during prophase as well as precocious sister-chromatid separation, followed by random disjunction during anaphase I. "The bulk of the first division misbehavior consists of sister chromatids' disjoining from one another, a process which normally occurs only during the second meiotic division" (Goldstein). *ord* also elevates the frequency of mitotic chromosome misbehavior (Baker *et al.*, 1978). "A substantial proportion of this mitotic instability can be accounted for by a hypothesis in which *ord* causes precocious sister-chromatid separation, followed by random disjunction, in somatic as well as germline cells" (Goldstein). Thus the *ord* locus likely specifies a function required for sister-chromatid cohesion during most or all cell divisions.

cytology: Placed in 59B-D by Mason (1976). Further confined to 59D4-10 by deficiency analysis (Miyazaki and Orr-Weaver).

ort: ora transientless (J.C. Hall)

location: 2-66.4.

origin: Induced by ethyl methanesulfonate.

references: Koenig and Merriam, 1977, *DIS* 52: 50-51.

O'Tousa, Leonard, and Pak, 1989, *J. Neurogenet.* 6: 41-52.

phenotype: Electroretinogram lacks on-transient, and shows either no off-transient or a delayed one, occurring 100-150 milliseconds after the light coincident photoreceptor potential is elicited. Rhodopsin levels, prolonged depolarizing afterpotentials, and rhabdomere structure are normal in R1-6 photoreceptors of *ort* eyes, unlike the case of *ninaE* single or double mutants (see allele tables of *ort* and *ninaE*).

alleles:

allele	discoverer	synonym	ref ^α	comments
ort¹	Koenig	<i>ora</i>	1, 2	recovered as double mutant with <i>ninaE¹</i> called <i>ninaE^{JK84}</i>
ort²	Hardy, Orevi and Merriam	<i>ort US1471</i>	2	
ort³	Hardy, Orevi and Merriam	<i>ort US1606</i>	2	
ort⁴	Hardy, Orevi and Merriam	<i>ort US2525</i>	2	
ort⁵	Hardy, Orevi and Merriam	<i>ort US6096</i>	2	
ort⁶	Pak	<i>ort P306</i>	2	

^α 1 = Koenig and Merriam, 1977, *DIS* 52: 50-51; 2 = O'Tousa, Leonard, and Pak, 1989, *J. Neurogenet.* 6: 41-52.

cytology: Located at 92A4-B1 (O'Tousa *et al.*, 1989).

Uncovered by *Df(3R)I2 = Df(3R)92A4-11;92D2-8*; complemented by *Df(3R)B16 = Df(3R)92A11-B1;92E10-F2*.

other information: *ort¹* has been placed to the left of *ninaE¹* by recombinational separation of the two genes (O'Tousa *et al.*, 1989) as well as by cytological location. (For further discussion of these genes, see *ninaE*).

orthodenticle: see *otd*

os: outstretched small eye

location: 1-59.2.

origin: X ray induced.

discoverer: Abrahamson, 1953.

synonym: *odsy*.

references: Verderosa and Muller, 1954, Genetics 39: 999.

phenotype: Wings held virtually at right angles to body. Eyes small and rounded. *os/os^o* has wing effect but eyes normal. *os/os^s* has eye effect but wings normal. RK1.

cytology: Localized to 17A5-6 (Laughnan).

other information: Originally considered two separate loci, *od*: outstretched and *sy*: small eye, Muller proposed a single gene based on the recovery of a mutant with both phenotypes, which was not resolvable into two components by recombination as well as on the failure to recover recombinants between *od* and *sy* in *trans* heterozygotes (Verderosa and Muller).

***os^{bdw}*: outstretched small eye-bending wings**

origin: X ray induced.

discoverer: Halfer, 1960.

synonym: *bdw*.

phenotype: Wings divergent and drooping, size and shape normal. Males sterile. RK2A.

cytology: Associated with $T(1;3)os^{bdw} = T(1;3)16E;80C$.



***os^o*: outstretched small eye-outstretched**

Edith M. Wallace, unpublished.

***os^o*: outstretched small eye-outstretched**

origin: X ray induced.

discoverer: Muller, 1930.

synonym: *od*.

references: 1930, J. Genet. 22: 303 (fig.). 1935, DIS 3: 30.

Verderosa and Muller, 1954, Genetics 39: 999.

phenotype: Wings extremely divergent, often at right angles to body. *os^o/os^s* is wild type. RK1.

***os^s*: outstretched small eye-small eye**

origin: Spontaneous.

discoverer: Bridges, 19g3.

synonym: *sy*.

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 236.

phenotype: Eyes small and rounded, high on the head but not bulging. RK1.

***osa*: osa (J.A. Kennison)**

location: 3-60.0.

origin: Induced by ethyl methanesulfonate.

discoverer: Kennison, 1983.

references: Kennison and Tamkun, 1988, Proc. Nat. Acad. Sci. USA 85: 8136-40.

phenotype: Isolated as a dominant suppressor of the antenna to leg transformation in $Pc^2 Antp^{Ns}$ double heterozygotes. Also acts as a dominant suppressor of *Antp^{Ns}*, *Pc* and *Pcl* alleles. Recessive embryonic lethality associated with all alleles. Also fails to complement $In(3R)Antp^{rv1}$ for viability.

alleles: Four from ethyl methanesulfonate and three from P-M hybrid dysgenesis.

cytology: 90B1-90D1 based on its inclusion within the transposed segment of $Tp(3;3)S462 = Tp(3;3)64C-E;89D1-2;90D1$ but not within the transposed segment of $Tp(3;1)bxdl11 = Tp(3;1)AD;89E;90B2$ and its failure to complement $In(3R)Antp^{rv1} = In(3R)81F;90B-C$.

****osh*: outshifted**

location: 1-33.0 (no crossover with *v* in 997 chromosomes).

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1955.

references: 1958, DIS 32: 73.

phenotype: Wings shortened and often slightly divergent. Body and wings pale in color. Eyes somewhat smaller and browner than normal. Viability and fertility good in both sexes. RK2.

***osk*: oskar**

location: 3-48 (distal to *p* and *hb*).

references: Lehmann and Nüsslein-Volhard, 1986, Cell 47: 141-52.

phenotype: Homozygous females normally viable and fecund; homozygous males fertile. Embryos produced by homozygous females lack pole plasm and fail to produce pole cells; abdominal region remains unsegmented and eventually dies. Temperature-sensitive period for the germ-line effect is the last six hours of oogenesis and for abdominal development the last twelve to fourteen hours (*osk^s*). Homozygous *osk* germ cells autonomous in pole cell transplants. Polar cytoplasm from unfertilized eggs or normal embryos capable of rescuing pole cell formation if injected at the posterior extremity of the early embryo and abdominal segmentation, to the extent of producing viable but sterile adults, if injected into the posterior half of preblastoderm embryos. Embryos produced by homozygous *BicD*; *osk* females are indistinguishable from those produced by *osk* alone suggesting that *osk⁺* product is required for the formation of bicaudal embryos; also required for the early anterior-posterior gradient of *hb* expression (Tautz, 1988, Nature 332: 181-84).

alleles: Nine ethyl-methanesulfonate-induced alleles.

allele	synonym	comments
<i>osk¹</i>	<i>osk⁵⁴</i>	
<i>osk²</i>	<i>osk⁸⁴</i>	strong allele
<i>osk³</i>	<i>osk⁸⁸</i>	
<i>osk⁴</i>	<i>osk¹²³</i>	
<i>osk⁵</i>	<i>osk¹⁵⁰</i>	
<i>osk⁶</i>	<i>osk¹⁶⁶</i>	strong allele
<i>osk⁷</i>	<i>osk²⁵⁵</i>	
<i>osk⁸</i>	<i>osk³⁰¹</i>	hypomorphic allele
		hypomorphic allele
		temperature sensitive
<i>osk⁹</i>	<i>osk³⁴⁶</i>	strong allele

cytology: Placed in 85B based on its inclusion in *Df(3R)p-XT26 = Df(3R)85A3;85C1-2* and *Df(3R)p-XT103 = Df(3R)85A2;85C1-2* but not in *Df(3R)p-XT118 = Df(3R)84F;85B1* (Lehmann and Nüsslein-Volhard, 1987, Dev. Biol. 119: 402-17).

osp: outspread (M. Ashburner)

location: 2-50.1 [0.01 cM to the left of *Adh* (Detwiler)].

references: Woodruff and Ashburner, 1977, Genetics 92: 117-132.

Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

phenotype: Wings held out at 45° to body axis. Strong alleles, and homozygous deficiencies show, in addition, a "tenting" of the wings so that they resemble those of *arc*. *Df(2L)osp* homozygous viable and fertile, though viability reduced in crowded cultures.

alleles:

allele	origin	discoverer	ref ^α	cytology
<i>osp</i> ¹	EMS?	E. H. Grell		
<i>osp</i> ³	γ ray	Detwiler		<i>Tp(2;3)35B3-4;36C11;98E2-3</i>
<i>osp</i> ⁴	EMS	Harrington	1	
<i>osp</i> ⁶	EMS	Harrington	1	
<i>osp</i> ⁷	EMS	Harrington	1	
<i>osp</i> ⁸	EMS	Harrington		
<i>osp</i> ¹⁶	EMS	Detwiler		
<i>osp</i> ¹⁹	EMS	Detwiler		
<i>osp</i> ²²	EMS	Detwiler		
<i>osp</i> ²⁴	EMS	Detwiler		<i>In(2L)35B3;35D3-5</i>
<i>osp</i> ²⁵	EMS	Detwiler		
<i>osp</i> ²⁷	EMS	Detwiler		
<i>osp</i> ³⁵	γ ray	Detwiler		
<i>osp</i> ⁴⁰	γ ray	Detwiler		
<i>osp</i> ⁵⁹	γ ray	Detwiler		<i>In(2L)35B3;35B3-6</i>
<i>osp</i> ^{76e}	EMS	Littlewood	1, 2	
<i>osp</i> ^{77e}	EMS	Littlewood	1, 2	
<i>osp</i> ^{90.1D}	DEO ^β	Detwiler	2	
<i>osp</i> ⁸³	γ ray	Detwiler		
<i>osp</i> ⁹⁰	γ ray	Detwiler		<i>T(2;3)35B2-3;89B9-11</i>
<i>osp</i> ¹⁰⁴	γ ray	Detwiler		
<i>osp</i> ²⁰¹	γ ray	Detwiler		
<i>osp</i> ^{CH62}	EMS	O'Donnell		
<i>osp</i> ^{CH63}	EMS	O'Donnell		
<i>osp</i> ^{pb3}	X ray	Kaufman		<i>T(2;3)35B2-3;82E2-8;89A9-10</i>
<i>osp</i> ^{Mpl}	X ray	Shelton		

^α 1 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186; 2 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.

^β diepoxyoctane.

cytology: Placed in 35B2-3.

other information: Probably the gene immediately to the left of *Adh*.

Osw: Outspread wing

location: 3- (between *Tb* and *ca*).

references: Sequeira, Nelson, and Szauter, 1989, Genetics 123: 511-24.

phenotype: Wings held out (Ganetzky).

ot: outheld

location: 1-65.7.

references: 1958, DIS 32: 73.

phenotype: Wings held horizontally; inner margin slightly cut away in many males. Ocellar bristles usually absent or reduced; effect variable. Hairs sparse, especially in posterior midthoracic region. Males sterile; viability

about 20% wild type. RK3.

cytology: Placed in 19A5 on the basis of its inclusion in *Df(1)16-2-19 = Df(1)19A5;19D3* but not in *Df(1)mal10 = Df(1)19A5-6;19E1*. (see also Schalet, Lefevre, and Singer, 1970, DIS 45: 165).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>ot</i> ¹	TEM	Fahmy, 1952		1	viable
<i>ot</i> ²	EMS	Lefevre	<i>l(1)DC711</i>	2	lethal
<i>ot</i> ³	EMS	Lefevre	<i>l(1)VA132</i>	2	lethal

^α 1 = Fahmy, DIS 32: 73; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95.

ota1: olfactory-trap-abnormal-1 (J.C. Hall)

location: 1-43.

origin: Induced by ethyl methanesulfonate.

references: Woodard, Huang, Sun, Helfand, and Carlson, 1989, Genetics 123: 315-26.

phenotype: Poor odor responses in tests involving an olfactory trap; entry into such traps was subnormal when odorant was food medium or ethyl acetate. Phenotype also subnormal in odor-induced jump responses (using ethyl acetate, propionic acid, or benzaldehyde as stimulant). Electroretinogram abnormal; very weak light induced depolarization and no light-on or light-off transients.

cytology: Maps to 12A6-13A5, owing to coverage of both olfactory and ERG defects by *Dp(1;f)LJ9*.

ota2: olfactory-trap-abnormal-2 (J.C. Hall)

location: 1-between *y* and *cv* (nearer to former).

origin: Induced by X-rays or ethyl methanesulfonate.

references: Woodard, Huang, Sun, Helfand, and Carlson, 1989, Genetics 123: 315-26.

phenotype: Abnormal trap-entry kinetics (*cf. ota1*), with food medium or ethyl acetate as stimulant (*ota2*¹ isolated using former, *ota2*² using latter). In odor induced jump tests *ota2*¹ responded normally to ethyl acetate, propionic acid, and benzaldehyde; but *ota2*² gave subnormal responses to the first two of these stimulants. ERG normal (both alleles).

alleles: Two alleles, *ota2*¹ (X-rays) and *ota2*² (formerly *ota4*). Allelism determined only by trap-entry behavior of the double heterozygote, *i.e.* no mapping data on *ota2*².

cytology: Proximal to 1B14, because *ota2* not covered by *y*²*Y611* (duplicated from X distal tip to the site just noted).

Ota3: Olfactory-trap-abnormal-3 (J.C. Hall)

location: 1-(not localizable; see **other information**).

origin: Induced by ethyl methanesulfonate.

references: Woodard, Huang, Sun, Helfand, and Carlson, 1989, Genetics 123: 315-26.

phenotype: Abnormal trap-entry kinetics with ethyl acetate as stimulant (*cf. ota1*). In odor-elicited jump tests, subnormal responses occurred to ethyl acetate and propionic acid but not to benzaldehyde. ERG normal. Uppercase-ness of this gene's symbol stems from at least partial dominance associated with *Ota3*/+ females' trap-entry behavior.

other information: A map position for *Ota3* was not indicated by testing segregants from *y cv v flOta3* heterozygotes. Genetic origin of *Ota3* may be more complicated;

Ota3 phenotype may not be the result of a single X-linked mutation.

ota5: olfactory-trap-abnormal-5 (J.C. Hall)

location: 1-unlocalized.

origin: Induced by ethyl methanesulfonate.

references: Woodard, Huang, Sun, Helfand, and Carlson, 1989, *Genetics* 123: 315-26.

phenotype: Males exhibit abnormal trap-entry kinetics with ethyl acetate as stimulant; not as subnormal (in these behavioral tests) as are the other *ota* genes. In odor-elicited jump tests with ethyl acetic or propionic acid as stimuli, however, *ota5* shows the same degree of impairment as *ota2*², and like *ota2*², shows normal jump responses to benzaldehyde. ERG normal. Homozygous *ota5* females behaved normally in the behavioral tests described.

ota7: olfactory-trap-abnormal-7 (J.C. Hall)

location: 1-between *y* and *cv* (nearer to former).

origin: Induced by ethyl methanesulfonate.

references: Woodard, Huang, Sun, Helfand, and Carlson, 1989, *Genetics* 123: 315-26.

phenotype: Abnormal trap-entry kinetics (*cf. ota1*) with ethyl acetate as stimulant. This phenotype is semi-dominant, as indicated from similar testing of *ota7/+* females. Odor-induced jump responses normal. ERG abnormal. The photoreceptor potential begins anomalously to decay to baseline soon after normal light-on transient appears; then renewed (cornea-negative) depolarization is observed (all of these dynamics observed during 0.5 second stimulus). Finally, a normal light-on transient is observed. The abnormal ERG phenotype, which co-segregates with subnormal trap-entry kinetics, is recessive.

cytology: Located proximal to 1B14, because the recessive ERG abnormality is not covered by *y*²*Y611*.

otd: see *oc*

otu: ovarian tumor (R. King)

location: 1-23.2.

references: King, Bahns, Horowitz, and Larramendi, 1978, *Int. J. Insect Morphol. Embryol.* 7: 359-75.

King and Riley, 1982, *Dev. Genet.* 3: 69-89.

King, Mohler, Riley, Storto, and Nicolazzo, 1986, *Dev. Genet.* 7: 1-20.

King and Storto, 1988, *BioEssays* 8: 18-24.

Mulligan, Mohler, and Kalfayan, 1988, *Mol. Cell. Biol.* 8: 1481-88.

phenotype: Homozygous females defective in proliferation, differentiation, or maturation of the germ line, depending on the level of activity of the particular allele. So-called quiescent alleles (QUI) produce ovarioles lacking in germ cells; oncogenic alleles (ONC) produce cystocytes that continue dividing and form tumors; differentiated alleles (DIF) produce chambers containing only "pseudonurse" cells (PNCs) or nurse cell/oocyte (NC/O) syncytia. In these, transport of nurse cell cytoplasm to the oocyte is inhibited and chambers are arrested at a pseudo-12 stage [Bishop and King, 1984, *J. Cell Sci.* 67: 87-119 (fig.)]. Mutant nurse cells that fail to pump their cytoplasm into the oocytes are also unable to form a system of actin microfilament bundles in their cortical cytoplasm during stage 10B (Storto and King, 1988, *Dev. Genet.* 9: 91-120). The proportions of ovarioles with the

different phenotypes appear to reflect the level of function of the particular allele; homozygotes are less severely affected than hemizygotes (80% of ovarioles of females carrying *otu*¹, *otu*⁴, *otu*⁵, or *otu*⁷ in combination with an *otu* deficiency lack germ cells, whereas 5% of the ovarioles of homozygotes lack germ cells); similarly, the levels of function of certain alleles decline as the developmental temperature is raised. Thus *otu*¹ behaves like a DIF allele at 18°, an ONC allele at 23°, and a QUI allele at 28°.

The ovarian tumors which give the mutant gene its name are made up of large numbers of single cystocytes and small numbers of clones of 2-4 interconnected cells [King, 1979, *Int. J. Insect Morphol. Embryol.* 8: 297-309 (fig.)]. Most cystocytes undergo complete cytokinesis, and there are defects in the construction and functioning of the polyfusomal system during the cycles of cystocyte divisions [Storto and King, 1989, *Dev. Genet.* 10: 70-86 (fig.)]. *Drosophila* nurse cells normally undergo nine or ten cycles of DNA replication (Mulligan and Rasch, 1985, *Histochemistry* 82: 233-47), and the chromatids dissociate so that each nucleus is filled with a jumbled mass of oligotene threads. In *otu* PNCs, the chromatids remain in register, generating banded polytene chromosomes [Dabbs and King, 1980, *Int. J. Insect Morphol. Embryol.* 9: 215-29 (fig.)]. Homologues pair and rearrangement configurations can be discerned [King, Riley, Cassidy, White, and Paik, 1981, *Science* 212: 441-43 (fig.)]. The largest polytenes have undergone 12 cycles of endonuclear replication (Rasch, King, and Rasch, 1984, *Histochemistry* 81: 105-10). The banding pattern of PNC polytenes is similar to that of the polytenes from larval salivary gland cells (Sinha, Mishra, and Lakhota, 1987, *Chromosoma* 95: 108-16; Heino, 1989, *Chromosoma* 97: 363-73).

At 25°, *otu*¹¹ behaves as an ONC allele, the cells dividing to form tumors, but at 18°, homozygous females produce oocytes that reach a pseudo-14 stage, contain beta yolk spheres and can undergo early embryogenesis. In the case of DIF alleles such as *otu*⁴, females generate pseudo-12 eggs which lack beta yolk spheres and never initiate development. When *otu*¹¹ is combined with alleles from the QUI class such as *otu*², the heteroallelic females are sterile. Heteroalleles between *otu*¹¹ and certain DIF alleles show various degrees of fertility [Storto and King, 1987, *Roux's Arch. Dev. Biol.* 196: 210-21 (fig.)]. *otu*¹¹/*otu*¹⁴ females are fully fertile although the nurse cells, unlike those of wild-type females, contain banded chromosomes (Storto and King, 1988).

Oocyte differentiation is destabilized in certain *otu* alleles; for example, the presumptive oocytes in about 20% of *otu*⁷ homozygotes resemble nurse cells in their polytenization, although they lag behind the remaining nurse cells by at least one replication cycle [King, Rasch, Riley, O'Grady, and Storto, 1985, *Histochemistry* 82: 131-34 (fig.)]. Germ line autonomy has been demonstrated for *otu*³, *otu*⁴, and *otu*⁷ (Wieschaus, Audit, and Masson, 1981, *Dev. Biol.* 88: 92-103; unpublished work cited in King *et al.*, 1986).

alleles: 21 alleles are listed in the following table. Heteroallelic combinations usually produce intermediate

phenotypes, but some show partial complementation.

allele	origin	discoverer	synonym	ref ^α	comments
<i>otu</i> ¹	EMS	Gans	<i>fs(1)A231</i>	1, 2, 3	oncogenic
<i>otu</i> ²	EMS	Mohler	<i>fs(1)14-97</i>	2, 3, 4	quiescent
<i>otu</i> ³	EMS	Gans	<i>fs(1)A116</i>	1, 2, 3	oncogenic
<i>otu</i> ⁴	EMS	Mohler	<i>fs(1)13-1994</i>	2, 3, 4	differentiated
<i>otu</i> ⁵	EMS	Mohler	<i>fs(1)11-1037</i>	2, 3, 4	differentiated
<i>otu</i> ⁶	EMS	Engstrom	<i>fs(1)209</i>	2	quiescent
<i>otu</i> ⁷	EMS	Engstrom	<i>fs(1)1304b</i>	2	differentiated
<i>otu</i> ⁸	EMS	Engstrom	<i>fs(1)1396</i>	2	quiescent
<i>otu</i> ⁹	EMS	Mohler	<i>fs(1)12-3266</i>	2, 4	differentiated
<i>otu</i> ¹⁰	EMS	Mohler	<i>fs(1)12-4474</i>	2, 4	quiescent
<i>otu</i> ¹¹	EMS	Mohler	<i>fs(1)14-334</i>	2, 4	oncogenic
<i>otu</i> ¹²	EMS	Mohler	<i>fs(1)12C-129</i>	2, 4	quiescent
<i>otu</i> ¹³	EMS	Mohler	<i>fs(1)13F-3</i>	2, 4	oncogenic
<i>otu</i> ¹⁴	EMS	Vyse	<i>fs(1)4077</i>	2	differentiated
<i>otu</i> ¹⁵	EMS	Digan	<i>fs(1)A3</i>	2	quiescent
<i>otu</i> ¹⁶	EMS	Digan	<i>fs(1)1001</i>	2	quiescent
<i>otu</i> ¹⁷	EMS	Digan	<i>fs(1)1401</i>	2	quiescent; 2 kb deletion
<i>otu</i> ¹⁸ β	P insert	Mohler	<i>otu</i> ^{P1}	5	oncogenic
<i>otu</i> ¹⁹	P insert	Mohler	<i>otu</i> ^{P2}	5	oncogenic
<i>otu</i> ²⁰ β	P insert	Mohler	<i>otu</i> ^{P3}	5	normal
<i>otu</i> ²¹ β	P insert	Mohler	<i>otu</i> ^{P4}	5	oncogenic

^α 1 = Gans, Audit, and Masson, 1975, *Genetics* 81: 683-704; 2 = King, Mohler, Riley, Storto, and Nicolazzo, 1986, *Dev. Genet.* 7: 1-20; 3 = King and Riley, 1982, *Dev. Genet.* 3: 69-89; 4 = Mohler and Carroll, 1984, *DIS* 60: 236-41; 5 = Mulligan, Mohler, and Kalfayan, 1988, *Mol. Cell. Biol.* 8: 1481-88.

^β P-elements in *otu*¹⁸, *otu*²⁰, and *otu*²¹ are of molecular sizes 2.9, 0.6, and 0.5 kb, respectively (Kalfayan, Walsh, Ousley, and Nishiharu, unpubl.).

cytology: Placed in 7F1 based on its inclusion in *Df(1)KA14 = Df(1)7F1-2;8C6* and its mapping genetically 0.4 cM to the left of *l(1)7Fa*¹⁰ which is also placed in 7F1 (King *et al.*, 1986).

molecular biology: Identification of *otu* sequences based on the insertion of transposable sequences in the same *EcoRI* restriction fragment of a lambda clone from 7F1 into each of four hybrid-dysgenesis-induced *otu* alleles and the correlation between excision of these sequences and restoration of fertility in revertants of *otu18* and *otu19*. *otu17* contains a deletion of 2 kb in the restriction fragment distal to the fragment altered in the insertional alleles (Mulligan *et al.*, 1988). The *otu* gene is at least 4.6 kb long and is subdivided into seven introns and eight exons. The first and sixth introns are large (534 and 583 b, respectively); the others range in size from 53 to 68 b. The first exon specifies an untranslated leader which contains the ribosomal binding sites. The *otu* gene encodes at least two ovary-specific and four testis-specific mRNAs. The major ovarian message is 3.2 kb, while a minor message is 4.0 kb. The 3.2 kb mRNA, which is transcribed from exons 2 through 8, specifies a proline-rich protein, 811 amino acids long. Exons 2-6 encode segments of 76, 33, 51, and 46 amino acids, respectively. The largest exon (number 7) encodes 415 amino acids, while the eighth exon specifies 58 amino acids at the carboxyl end of the protein and a trailer which contains the binding site for the polyadenylating enzyme (Mulligan *et al.*, 1988; Steinhauer, Walsh, and Kalfayan, 1989, *Mol. Cell. Biol.*, 9: 5726-32).

Out cold: see *Ocd*

outer rhabdomeres absent: see *ort*¹ *ninaE*¹

outheld: see *ot*

outshifted: see *osh*

outsread: see *osp*

Outspread wing: see *Osw*

outstretched small eye: see *os*

***ov:** *oval*

location: 1-17.5.

discoverer: Steinberg, 37h15.

phenotype: Eyes somewhat oval and quite rough. RK1.

ovaleless: see *ovl*

ove: *overetherized*

location: 2- (not located).

origin: Spontaneous.

discoverer: Plaine and Aubele, 64b.

references: 1965, *DIS* 40: 36.

phenotype: Wings held vertically within 1 hr after eclosion, vibrate feebly but are incapable of supporting flight. Movements of first two pairs of legs uncoordinated. Viable and fertile although *ove* male often unsuccessful in mating with *ove*⁺ female. RK2.

Overflow: see *Dl*^{Of}

overlapping complementation complex: see *BRC*

***ovi:** *ovioculus*

location: 1-0.9.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).

discoverer: Fahmy, 1953.

references: 1958, *DIS* 32: 73.

phenotype: Eyes small, egg shaped, and rough. Wings spread or elevated to varying degrees; edges incised, especially inner margin. Eclosion slightly delayed. Males sterile. Viability 20-60% wild type. RK2.

***ovl:** *ovaleless*

location: 2- (not located).

origin: Spontaneous.

discoverer: Bridges, 21a3.

references: Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 232.

phenotype: Rough eyes. Males fertile; females entirely sterile. Small groups of cells in place of ovaries; ducts and genitalia normal. Abdomen of female grayish and translucent. RK3.

ovo: *ovo* (*B. Oliver*)

location: 1-10.2.

references: Mohler, 1977, *Genetics* 85: 259-72.

Busson, Gans, Komitopoulou, and Masson, 1983, *Genetics* 105: 309-25.

Komitopoulou, Gans, Margaritis, Kafatos, and Masson, 1983, *Genetics* 105: 897-920.

Perrimon and Gans, 1983, *Dev. Biol.* 100: 365-73.

Perrimon, 1984, *Genetics* 108: 927-39.

Mohler and Carrol, 1984, *DIS* 60: 236-41.

Oliver, Perrimon, and Mahowald, 1987, *Genes Dev.* 1: 913-23 (Fig.).

Mével-Ninio, Mariol, and Gans, 1989, *EMBO J.* 8: 1549-58.

phenotype: All *ovo* alleles fully penetrant for female sterility; no male function for *ovo*⁺ known. Lack of zygotic activity results in complete absence of germ-line cells in

adult female; reduction in numbers of germ cells first evident during early gastrulation. Homozygotes for weaker alleles produce germ cells, but oogenesis defective; egg chambers may degenerate prior to vitellogenesis or proceed through oogenesis and be oviposited, depending on allele; laid eggs are permeable to neutral red and never develop. Heterozygotes for two of the dominant alleles phenotypically similar to homozygotes for weak recessive alleles. Ovarian tumors formed in females carrying *ovo*^{D3} in heterozygous combination with the hypomorphic alleles, e.g., *ovo*^{D1rv20}, or with *fs(1)A1621*. The *ovo*^{D2} mutation is partially suppressed by many *Sxl* alleles. *ovo*^{D1/+} females produce no eggs; extensively utilized in the selection of *ovo*⁺ germ-line clones.

alleles: Recessive alleles, mostly recovered as revertants of dominant alleles, usually but not always, simultaneously mutant for *svb* or *lzl*; some *svb* alleles also simultaneously mutant for *ovo*; *ovo* proximal to *svb* and distal to *rg* by deficiency analysis. Many revertants are homozygous lethal owing to their being simultaneously mutant for *svb*. The *lzl* eye phenotype is cold sensitive and semi-dominant. Most revertants are amorphic in that, when tested in heterozygous combination with *ovo*^{D1rv22} or *ovo*^{D1rv23}, they have atrophic ovaries, with no egg chambers observable.

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>ovo</i> ^{D1}	EMS	Komitopoulou	<i>fs(1)K1237</i>	2	antimorph; germ-cell arrest
<i>ovo</i> ^{D2}	EMS	Komitopoulou	<i>fs(1)K1103</i>	2	antimorph; permeable eggs
<i>ovo</i> ^{D3}	EMS	Komitopoulou	<i>fs(1)K155</i>	2	antimorph; permeable eggs
<i>ovo</i> ^{D1rv1}	EMS	Oliver	<i>ovo</i> ^{D1rE1}	4	<i>svb</i>
<i>ovo</i> ^{D1rv2}	EMS	Oliver	<i>ovo</i> ^{D1rE2}	4	<i>svb</i>
<i>ovo</i> ^{D1rv3}	EMS	Oliver	<i>ovo</i> ^{D1rE3}	4	<i>svb</i>
<i>ovo</i> ^{D1rv4}	EMS	Oliver	<i>ovo</i> ^{D1rE4}	4	<i>svb</i>
<i>ovo</i> ^{D1rv5}	EMS	Oliver	<i>ovo</i> ^{D1rE5}	4	
<i>ovo</i> ^{D1rv6}	EMS	Oliver	<i>ovo</i> ^{D1rE6}	4	<i>svb</i>
<i>ovo</i> ^{D1rv7}	γ ray	Perrimon	<i>ovo</i> ^{D1rG2}	4	<i>svb</i>
<i>ovo</i> ^{D1rv8}	γ ray	Perrimon	<i>ovo</i> ^{D1rG3}	4	<i>svb</i>
<i>ovo</i> ^{D1rv9}	γ ray	Perrimon	<i>ovo</i> ^{D1rG4}	4	<i>svb</i>
<i>ovo</i> ^{D1rv10}	γ ray	Perrimon	<i>ovo</i> ^{D1rG5}	4	<i>svb</i> ; <i>ln(1)4E1-2</i> ; 5A1-6
<i>ovo</i> ^{D1rv11}	γ ray	Perrimon	<i>ovo</i> ^{D1rG8}	4	<i>svb</i>
<i>ovo</i> ^{D1rv12}	γ ray	Perrimon	<i>ovo</i> ^{D1rG9}	4	<i>svb</i>
<i>ovo</i> ^{D1rv13}	γ ray	Oliver	<i>ovo</i> ^{D1rG10}	4	<i>svb</i>
<i>ovo</i> ^{D1rv14}	γ ray	Oliver	<i>ovo</i> ^{D1rG11}	4	<i>svb</i>
<i>ovo</i> ^{D1rv15}	γ ray	Oliver	<i>ovo</i> ^{D1rG12}	4	<i>svb</i>
<i>ovo</i> ^{D1rv16}	γ ray	Oliver	<i>ovo</i> ^{D1rG14}	4	<i>svb</i> ; regains <i>ovo</i> ⁺ function
<i>ovo</i> ^{D1rv17}	γ ray	Oliver	<i>ovo</i> ^{D1rG19}	4	<i>svb</i>
<i>ovo</i> ^{D1rv18}	γ ray	Oliver	<i>ovo</i> ^{D1rG20}	4	<i>svb</i>
<i>ovo</i> ^{D1rv19}	γ ray	Oliver	<i>ovo</i> ^{D1rG21}	4	<i>svb</i>
<i>ovo</i> ^{D1rv20}	γ ray	Oliver	<i>ovo</i> ^{D1rG23}	4	<i>svb</i> ; probably hypomorphic
<i>ovo</i> ^{D1rv21}	γ ray	Oliver	<i>ovo</i> ^{D1rG25}	4	<i>svb</i>
<i>ovo</i> ^{D1rv22}	spont	Gans	<i>lzl</i> ^G	1, 4	<i>lzl</i>
<i>ovo</i> ^{D1rv23}	spont	Oliver	<i>ovo</i> ^{D1rS1}	4	
<i>ovo</i> ^{D1rv24}	spont	Gans	<i>ovo</i> ^{D1rS14}	3	<i>lzl</i> ; <i>gypsy</i> at 0.9 kb
<i>ovo</i> ^{D1rv25}	spont	Gans	<i>ovo</i> ^{D1rS22}	3	<i>lzl</i> ; <i>gypsy</i> at 0.9 kb
<i>ovo</i> ^{D1rv26}	spont	Gans	<i>ovo</i> ^{D1rS32}	3	<i>gypsy</i> at 0.9 kb
<i>ovo</i> ^{D1rv27}	spont	Gans	<i>ovo</i> ^{D1rS35}	3	<i>lzl</i> ; <i>gypsy</i> at 0.9 kb
<i>ovo</i> ^{D1rv28}	spont	Gans	<i>ovo</i> ^{D1rS36}	3	<i>gypsy</i> at 0.9 kb
<i>ovo</i> ^{D1rv29}	spont	Gans	<i>ovo</i> ^{D1rS37}	3	<i>gypsy</i> at 0.9 kb
<i>ovo</i> ^{D1rv30}	spont	Gans	<i>ovo</i> ^{D1rS38}	3	<i>lzl</i> ; <i>gypsy</i> at 0.9 kb
<i>ovo</i> ^{D1rv31}	spont	Gans	<i>ovo</i> ^{D1rS78}	3	<i>lzl</i>
<i>ovo</i> ^{D1rv32}	spont	Gans	<i>ovo</i> ^{D1rS80}	3	<i>svb</i>
<i>ovo</i> ^{D1rv33}	spont	Gans	<i>ovo</i> ^{D1rS85}	3	
<i>ovo</i> ^{D1rv34}	spont	Gans	<i>ovo</i> ^{D1rS86}	3	

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>ovo</i> ^{D1rv35}	spont	Gans	<i>ovo</i> ^{D1rS88}	3	
<i>ovo</i> ^{D1rv36}	spont	Gans	<i>ovo</i> ^{D1rS89}	3	
<i>ovo</i> ^{D1rv37}	spont	Gans	<i>ovo</i> ^{D1rS124}	3	<i>lzl</i> ; <i>gypsy</i> at 0.9 kb; 0.6 kb deletion at insertion site
<i>ovo</i> ^{D1rv38}	P	Gans	<i>ovo</i> ^{D1rHD71}	3	<i>svb</i>
<i>ovo</i> ^{D1rv39}	P	Gans	<i>ovo</i> ^{D1rHD72}	3	lesion in 4.0 to 4.9 kb
<i>ovo</i> ^{D1rv40}	P	Gans	<i>ovo</i> ^{D1rHD81}	3	<i>svb</i> ; 5-kb insert in 1.1 to 3.6 kb
<i>ovo</i> ^{D1rv41}	P	Gans	<i>ovo</i> ^{D1rHD82}	3	<i>svb</i>
<i>ovo</i> ^{D1rv42}	P	Gans	<i>ovo</i> ^{D1rHD90}	3	<i>svb</i>
<i>ovo</i> ^{D1rv43}	P	Gans	<i>ovo</i> ^{D1rHD91}	3	<i>svb</i> ; <i>copia</i> at 1.3 kb
<i>ovo</i> ^{D1rv44}	P	Gans	<i>ovo</i> ^{D1rHD93}	3	<i>svb</i>
<i>ovo</i> ^{D1rv45}	P	Gans	<i>ovo</i> ^{D1rHD101}	3	<i>svb</i>
<i>ovo</i> ^{D1rv46}	P	Gans	<i>ovo</i> ^{D1rHD104}	3	<i>svb</i>
<i>ovo</i> ^{D1rv47}			31	3	DNA lesion in 1.1 to 3.6 kb
<i>ovo</i> ^{D1rv48}			51	3	DNA lesion in 1.1 to 3.6 kb
<i>ovo</i> ^{D1rv49}			81	3	DNA lesion in 1.1 to 3.6 kb
<i>ovo</i> ^{D1rv50}			118	3	DNA lesion in 1.1 to 3.6 kb
<i>ovo</i> ^{D1rv51}			119	3	DNA lesion in 1.1 to 3.6 kb
<i>ovo</i> ^{D1rv52}			121	3	DNA lesion in 1.1 to 3.6 kb
<i>ovo</i> ^{D3rv53}	spont	Gans	<i>ovo</i> ^{D3rS54}	3	<i>gypsy</i> at 0.9 kb
<i>ovo</i> ^{D3rv54}	spont	Gans	<i>ovo</i> ^{D3rS57}	3	<i>gypsy</i> at 0.9 kb
<i>ovo</i> ^{D3rv55}	spont	Gans	<i>ovo</i> ^{D3rS58}	3	<i>gypsy</i> at 0.9 kb
<i>ovo</i> ^{D3rv56}	spont	Gans	<i>ovo</i> ^{D3rS59}	3	<i>copia</i> at 0.9 kb
<i>ovo</i> ^{D3rv57}	γ ray	Oliver	<i>ovo</i> ^{D3rG1}	3	<i>svb</i>
<i>ovo</i> ^{M1}	EMS	Mohler	<i>ovo</i> ^{rM1}	4	hypomorph; permeable eggs
<i>ovo</i> ^{M2}	EMS	Mohler	<i>ovo</i> ^{rM2}	4	hypomorph; permeable eggs

^α 1 = Busson, Gans, Komitopoulou, and Masson, 1983, Genetics 105: 309-25; 2 = Komitopoulou, Gans, Margaritis, Kafatos, and Masson, 1983, Genetics 105: 897-920; 3 = Mével-Ninio, Mariol, and Gans, EMBO J. 8: 1549-58; 4 = Oliver, Perrimon, and Mahowald, 1987, Genes Dev. 1: 913-23.

^β Mutant alleles associated with *ovo*^D revertants; coordinates from chromosomal walk of Mével-Ninio *et al.*; origin designated as *Sall* site just to the left of the *gypsy* insert of *ovo*^{D1rv30}; positive values to the right.

cytology: Placed in 4E2 based on its inclusion in *Df(1)ovo6* = *Df(1)4C5-6,4E2-3* and *Df(1)bi-DL3* = *Df(1)3C7-12,4E1-2* but not *Df(1)bi-DL5* = *Df(1)3C7-12,4E1-2* (Oliver).

molecular biology: 32 kb of wild-type DNA containing *ovo*⁺ has been cloned, using the transposing element *gypsy* as a tag; the position of the *ovo* locus was limited distally on the molecular map by the proximal breakpoint of *Df(1)bi-D2* = *Df(1)4B6-C1,4D7-E1* between coordinates -5 and -4 (Mével-Ninio *et al.*, 1989). Reversions of the dominant alleles *ovo*^{D1} and *ovo*^{D3} to recessive *ovo* alleles occurred in crosses to females carrying *gypsy* and the majority of these revertants carried insertions of *gypsy* and/or *copia* and other transposable elements; eleven spontaneous revertants contained a *gypsy*-element insert and one *copia* insert at coordinate 0.9; of the seven *gypsy*-induced revertants of *ovo*^{D1}, four inserted in one orientation were associated with *lzl* mutations and the three inserted in the opposite orientation were not; *gypsy*-induced revertants of *ovo*^{D3} were *lzl*⁺ irrespective of orientation.

p: pink

location: 3-48.0 (proximal to *hb* and *dsx*).

phenotype: Eye color varies from pink to dull ruby with purplish tone depending on allele. Larval Malpighian tubules colorless. Color autonomous in *p^P* optic disk allowed to undergo metamorphosis in a wild-type host (Beadle and Ephrussi, 1936, *Genetics* 21: 230).

alleles: Mutant alleles of *p* are listed in the following table, their phenotype in regard to eye color included if described in the literature.

allele	origin	discoverer	ref ^α	eye color ^β	cytology
<i>p¹</i>	spont	Morgan, 10g	3, 4, 11, 15	1	
* <i>p³</i>	spont	Bridges, 16d21	5		
* <i>p⁴</i>	spont	Richards, 17a7	5	2	
* <i>p⁵</i>	spont	Bridges	5		
* <i>p^{21a}</i>	spont	L.V. Morgan	5		
* <i>p^{25g}</i>	spont	Bridges	5		
* <i>p^{53h}</i>	spont	Thoday	14	2	
* <i>p⁵⁶</i>	spont	Williams	16	2	
* <i>p^{100.290}</i>	X ray	Alexander	15	3	<i>In(3)85B3-4;85D12-15</i>
* <i>p^{100.48}</i>	X ray	Alexander	15	3	<i>In(3)80-81;85A6-B1</i>
* <i>p^{100.88}</i>	X ray	Alexander	15	3	<i>In(3)80-81;94D11-E1</i>
<i>p⁴¹⁹</i>	X ray		9		<i>In(3R)84D4-6;86A3</i>
<i>p⁷¹²</i>	X ray		9		<i>T(2;3)25D;84D4-6;85B6</i>
<i>p^P</i>	spont	Bridges, 13a24	1, 2, 4, 6, 8, 12	2	
* <i>p^{p56}</i>	spont	Williams	16	4	
* <i>p^{snb}</i>		Dobzhansky	13	1	
<i>p^{XT14}</i>		Porter	7		<i>T(Y;3)85A</i>
<i>p^{XT15}</i>	X ray		10		
<i>p^{XT27}</i>	X ray		10		
<i>p^{XT104}</i>	X ray		10, 12		
<i>p^{XT117}</i>	X ray		10		<i>In(3LR)62E;85A-B</i>
<i>p^{XT126}</i>	X ray		10		<i>T(2;3)44F;85A</i>

^α 1 = Alexander, 1975, *Genetics* 81: 493-500; 2 = Beadle, 1937, *Genetics* 22: 587-611; 3 = Brehme and Demerec, 1942, *Growth* 6: 651-56; 4 = Bridges and Morgan, 1923, *Carnegie Inst. Washington Publ. No. 327: 44*; 5 = CP552; 6 = Duncan and Kaufman, 1975, *Genetics* 80: 733-52; 7 = Jones and Rawls, 1988, *Genetics* 120: 733-42; 8 = Judd, 1955, *DIS* 29: 126; 9 = Kempthues, Raff, and Kaufman, 1983, *Genetics* 105: 345-56; 10 = Lehmann and Nüsslein-Volhard, 1987, *Dev. Biol.* 119: 402-17; 11 = Nolte, 1959, *Heredity* 13: 233-41; 12 = Tautz, Lehmann, Schnürch, Schuh, Seifert, Kienlin, Jones, and Jäckle, 1987, *Nature*, (London) 327: 383-89; 13 = Silva, 1989, *DIS* 67: 72; 14 = Thoday, 1954, *DIS* 28: 78; 15 = Ward and Alexander, 1957, *Genetics* 42: 42-54; 16 = Williams, 1956, *DIS* 30: 80.

^β 1 = Dull ruby with purplish tone; 40% normal red and 33% normal brown pigment; 2 = Lighter and more orange than *p*; 9% normal red and 15% normal brown pigment (Beadle, 1937); 3 = Pink; 4 = Light ruby with orange tone.

^γ Females heterozygous for *p^P* and a white allele (*w*, *wh*, *w^{bf}*) have brownish eyes (Judd, 1955).

cytology: Located in 85A6 on the basis of its association with *Df(3R)p25 = Df(3R)85A5-7;85A11* and by *in situ* hybridization (Jones and Rawls, 1988).

molecular biology: The 85A region including *p* has been cloned by chromosome walking and a restriction map of the region constructed (Jones and Rawls, 1988).

p^{Gr}: see *Pu*

P: Pale

location: 2- or 3- (rearrangement).

origin: Spontaneous.

discoverer: Bridges, 17j16.

references: Bridges, and Morgan, 1923, *Carnegie Inst. Washington Publ. No. 327: 184* (fig.).

phenotype: Heterozygote a specific dilutor of the *w^e* series of white alleles; tends to darken eye color of *w^a* series. Homozygous lethal. RK2A.

cytology: Associated with *Tp(2;3)P = Tp(2;3)58E3-F2;60D14-E2;96B5-C1* (Morgan, Bridges, and Schultz, 1934, *Year Book - Carnegie Inst. Washington* 33: 278).

P1: see *Fbp1*

P6: see *Fbp2*

P37 (J. C. Hall)

location: 1-.

origin: Induced by ethyl methanesulfonate.

discoverer: Deland and Pak.

references: Heisenberg and Götz, 1975, *J. Comp. Physiol.* 98: 217-41.

phenotype: Fast phototaxis not completely absent at high light levels. Optomotor response half as strong as in wild type. In flight, response to movement from front to back is disturbed. Poor fixation.

P43 (J. C. Hall)

location: 1-.

origin: Induced by ethyl methanesulfonate.

discoverer: Deland and Pak.

references: Heisenberg and Götz, 1975, *J. Comp. Physiol.* 98: 217-41.

Hall, 1982, *Quart. Rev. Biophys.* 15: 223-479.

phenotype: Holes in optic lobes and brain; lamina abnormal, but with general structure preserved (Pak, Heisenberg and Hengstenberg). Defective phototaxis and optomotor responses. No fixation. No light-on or light-off transient spikes.

P48 (J. C. Hall)

location: 1-.

origin: Induced by ethyl methanesulfonate.

discoverer: Deland and Pak.

references: Heisenberg and Götz, 1975, *J. Comp. Physiol.* 98: 217-41.

phenotype: Variable. Rotatory optomotor response almost as big as in wild type, but absent when striped pattern used.

pa: patulous

location: 2-101.0.

origin: Spontaneous.

references: Edmondson and Meyer, 1949, *DIS* 23: 61.

phenotype: Wings spread wide apart. Excellent viability; fair fertility. RK1.

cytology: Placed in 58F2-60E2 on the basis of its being covered by *Dp(2;3)P* from *Tp(2;3)P = Tp(2;3)58E3-F2;60D14-E2;96B5-C1*.

pa: see *pt*

***pads: pads**

location: 2-55.

origin: Spontaneous.

discoverer: Bridges, 17e9.

references: Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 212 (fig.), 232.

Stern, 1934, *DIS* 1: 36.

phenotype: Wings malformed, often remain in condition of those of newly emerged flies. RK2.

***pads²**

origin: Spontaneous.
discoverer: Mohr, 20b15.
references: 1929, Z. Indukt. Abstamm. Vererbungsl. 50: 126.
phenotype: Like *pads*. RK2.

pads-b: see *pu*

paired: see *prd*

Paired box: see *Pox*

pal: paternal loss

location: 2-35.7 (between *Sp* and *J*).
origin: Induced by ethyl methanesulfonate.
discoverer: Sandler.
synonym: *mei-W5*.
references: Sandler, 1971, DIS 47: 68.

Baker, 1972, DIS 49: 55.

1975, Genetics 80: 267-96.

Hall, Gelbart, and Kankel, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 265-314.

Baker and Hall, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 351-434.

phenotype: Behaves as a recessive meiotic mutant in males; causes the loss of paternally-derived chromosomes among the progeny of homozygous males. Different chromosomes are lost at different rates. Loss may occur in the zygote and affect the whole body or during early mitotic divisions giving rise to mosaicism. The site responsible for the sensitivity of the X chromosome to *pal* has been located at or near the X centromere. *pal*⁺ function seems to be necessary during meiosis for normal transmission of paternal chromosomes in the early divisions of the fertilized egg.

cytology: Located in either 28C-D or 29F-30F since it is uncovered by *Dp(2;Y)B231 = Dp(2;Y)27C;31E* and is not deleted by deficiencies for 27D-28C, 28D-29F, 30F-31D, and 31C-D in segmental aneuploids (Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84).

other information: Mosaics with large patches can be generated by early chromosome loss in the progeny of *pal* males and allows the construction of fate maps of the blastoderm.

Palat: see *Fs(3)Sz19*

pale: see *ple*

Pale: see *P*

pale ocelli: see *po*

pale wing: see *plw*

palld: see *pld*

palsied: see *pls*

pannier: see *pnr*

par: paralog

location: 1-1.4.

origin: Recovered after ethyl methanesulfonate treatment of a ν ²⁴ chromosome; may have been spontaneous in the stock.

discoverer: Gans.

synonym: *fs(1)A1122^{ts}*.

references: Gans, Audit, and Masson, 1975, Genetics 81: 683-704.

Zalokar, Audit, and Erk, 1975, Dev. Biol. 47: 419-32.

Thierry-Mieg, 1982, Genetics 100: 209-37.

phenotype: Recessive temperature-sensitive female-sterile mutant. At 29°, homozygous females have small ovaries and abnormal egg chambers, the few eggs that are laid dying before hatching; at 23° *par* females are viable and fecund, but 60% of their progeny die without forming a complete blastoderm and the remainder form a blastoderm lacking in pole cells and develop into sterile adults with cuticular defects; at 16°, only 20-30% of the progeny of *par* females are agametic and few of these show cuticular defects. May act as a dominant enhancer of *spl* causing a *spl* phenotype in *par spl/FM3* heterozygotes. A modifier *Su(par)* (mapping to right of ν on X) decreases in a semidominant way the frequency of agametic flies (from 95-100% to 51-61%) and abdominal defects (from 26-49% to 5-14%) (Thierry-Mieg, 1982). The temperature-sensitive period is restricted to the mother's lifespan and the frequency of progeny defects is independent of the genotype of the zygote. The mutation interacts zygotically *in trans* with loci in the neighboring regions 3A2, 3A3, 3C1-2, 3C4, and 3C6-8. When z^a and/or w^a (wild-type eye color) are combined with *par*, a zygotic semidominant temperature-sensitive interaction takes place resulting in $z^a par$ heterozygotes that are brown-eyed and $z^a par w^a$ heterozygotes that are white-eyed. Interactions between *par* and *N* are also reported by Thierry-Mieg, 1982, such as the zygotic dominant suppression of Notch wing in *par N⁺/par⁺ N⁻* females (the Notch mutants being *Df(1)N-8* and *Df(1)N-264-105* but not *N²⁶⁴⁻⁶⁹*, a point mutant). An interaction between *par* and a deficiency for 3A2 results in almost complete sterility in females with a paternal *par X* and in aging females with a maternal *par X*.

alleles: One temperature-sensitive allele, *par^{X1122}* (Gans et al., 1975).

cytology: Placed in 3B3 based on its inclusion in both *Df(1)64f1 = Df(1)3A9-B1;3B2-3* and *Df(1)w258-45 = Df(1)3B2-3;3C2-3*.

other information: Probably represents a new complementation group in the *z-w* region since allelism tests at 23° and 29° show full complementation for all *par* characteristics by *l(1)3Bb [l(1)zw6]*, *l(1)3Bc [l(1)zw12]*, and *l(1)3Bd [l(1)zw7]* (Thierry-Mieg, 1982).

para: paralytic (J.C. Hall)

location: 1-52.1 (between *m* and *f*; Homyk and Pye, 1989).

origin: Induced by ethyl methanesulfonate or hybrid dysgenesis.

discoverer: R. Williamson.

references: Grigliatti, Williamson, and Suzuki, 1970, Genetics 64: s27.

Suzuki, 1970, Science 170: 695-706.

Suzuki, Grigliatti, and Williamson, 1971, Proc. Nat. Acad. Sci. USA 68: 890-93.

- Grigliatti, Suzuki, and Williamson, 1972, *Dev. Biol.* 28: 352-71.
- Grigliatti, Hall, Rosenbluth, and Suzuki, 1973, *Mol. Gen. Genet.* 120: 107-14.
- Wu and Ganetzky, 1980, *Nature (London)* 286: 814-16.
- Falk, Roselli, Curtiss, Halliday, and Klufas, 1984, *Mut. Res.* 126: 25-34.
- Ganetzky, 1984, *Genetics* 108: 897-911.
- Kyriacou and Hall, 1985, *Nature (London)* 314: 171-73.
- Ganetzky, 1986, *J. Neurogenet.* 3: 19-31.
- Jackson, Wilson, and Hall, 1986, *J. Neurogenet.* 3: 1-17.
- Homyk and Pye, 1989, *J. Neurogenet.* 5: 37-48.
- Loughney, Kreber, and Ganetzky, 1989, *Cell* 58: 1143-54.

phenotype: Exposure to 29-30° causes rapid paralysis that is quickly reversed on shift to 22-25°. Larvae are paralyzed, too, at somewhat higher temperatures. Flies of some *para* strains seem sluggish at lower temperatures. When these mutants are still paralyzed (*i.e.* at high temperatures), they appear to retain many of their "vital functions," their heart still beats (Grigliatti, Suzuki and Williamson, 1972), and they quickly regain normal behavior when shifted to 22° after several hours at 29° (Suzuki *et al.*, 1971); in fact, after a less prolonged exposure (30 min) to high temperature, the still-heated mutant flies regain weak mobility and are even able to right themselves and walk (Suzuki *et al.*, 1971). *para^{ts1}/+* adults become paralyzed at 40° within one min [10 min required to paralyze wild-type (Hall, 1973, *DIS* 50: 103-04)]. *para^{ts1}* larvae stop "tracking" at high temperature (Wu, Ganetzky, Jan, Jan and Benzer, 1978, *Proc. Nat. Acad. Sci. USA* 75: 4047-51). *para^{ts1}* is nearly unconditionally lethal when uncovered by a deletion or other *para*-locus aberration, whereas other alleles lead to reduced viability (unconditional) when heterozygous with chromosomal aberrations at the locus (Ganetzky, 1984). Action potentials in larval nerves are reversibly heat-sensitive (Wu and Ganetzky, 1980), and the same can be inferred for at least some adult neurons (indicated by brain stimulation and recording of responses in thoracic muscles [Siddiqi and Benzer, 1976, *Proc. Nat. Acad. Sci. USA* 73: 3253-57; Benshalom and Dagan, 1981, *J. Comp. Physiol.* 144: 409-17]). Other "excitable phenomena," such as the electroretinogram responses and synaptic transmission, appear to be normal in *para^{ts}* adults at high temperature (Suzuki *et al.*, 1971; Siddiqi and Benzer, 1976); also *para^{ts1}* does not block action potentials in the cervical giant fiber at high temperature (Nelson and Baird, 1985, *Neurosci. Abstr.* 11: 313) in contrast to results of recording from larval motor neurons (Wu and Ganetzky, 1980); other studies of the giant fiber pathway (involving adult mosaics bilaterally split, externally, for *para^{ts1}* and *para⁺*) indicate that at least certain elements of the pathway (if not the giant fiber itself) fail to fire action potentials at elevated temperature (Benshalom and Dagan, 1981), and recordings from mosaics of this type also suggest "functional coupling" between left and homologous right sides of this giant fiber pathway (Benshalom and Dagan, 1985, *J. Comp. Physiol.* 156: 13-23). *para^{ts1}* causes first larval instar death when in combination with *nap^{ts}* (Wu and Ganetzky, 1980; Ganetzky, 1984); similar lethality occurs when *para⁺* dosage is decreased in a *nap^{ts}* background (Ganetzky, 1984). Other *para* alleles, in combi-

nation with *nap^{ts}*, lead to reduced viability, with *para^{ts115}* having the strongest effect, followed by *para^{ST76}* and *para^{ST109}* (Ganetzky, 1984). In combination with the *tipE* mutation, *para* mutations again cause decreased viability, but the allele-specific interactions are different from those of the series just noted [*i.e.* with respect to *nap^{ts}* (Ganetzky, 1986)]. Surviving *para*; *tipE* double mutants are weak, and show accentuated heat-sensitivity (in regard to mobility and nerve conduction); *para* alleles are dominant for behavioral defects in a homozygous *tipE* background (Ganetzky, 1985). In adults doubly mutant for *para^{ts1}* and *nap^{ts}*, sensory cells (developing from imaginal discs in mosaics) appear to have no nerve conduction (Burg and Wu, 1984, *Neurosci. Abstr.* 10: 513). In mosaics involving *para* mutations only one allele (*para^{ST109}*) causes all legs to be either paralyzed or normal in different individual gynandromorphs (Siddiqi and Benzer), in contrast to independent paralysis of legs in mosaics constructed with respect to *para^{ts1}* [Grigliatti *et al.*, 1972; Siddiqi and Benzer, 1978, *Genetic Mosaics and Cell Differentiation* (Gehring, ed.), Springer-Verlag, Berlin, pp: 259-305]. These results (and others, Benshalom and Dagan, 1985, for example), reveal poor correlation of the externally mutant genotype (in mosaics) and behavioral or physiological malfunctions [consistent with internal (no doubt neural) "foci" for *para*'s action]. In other studies, *para^{ts1}* mosaics with mutant heads (scored externally) usually are immobile at high temperature, but maintain normal posture (Suzuki *et al.*, 1971; Grigliatti *et al.*, 1972). Exposure of *para^{ts1}* males to high temperature causes arrest of the oscillator underlying rhythmic component of courtship song (Kyriacou and Hall, 1985). At permissive temperatures, *para^{ts1}* neurons (unlike those influenced by *nap^{ts}*) seem normal, except that there is slightly increased resistance of cultured *para^{ts1}* larval neurons to killing effects of veratridine, shift to high temperature causing that resistance to increase (Suzuki and Wu, 1984, *J. Neurogenet.* 1: 225-38). Other pharmacological studies (tetrodotoxin sensitivity of action potentials or binding assays involving that toxin) have revealed no *para*-induced abnormalities (Ganetzky and Wu, 1980; Kauvar, 1982, *Molec. Gen. Genet.* 187: 172-73). It was reported (based on injection of *para^{ts1}* adults with picrotoxin and the ensuing inhibition of paralysis at high temperatures) that the mutation is involved in a generalized augmentation of inhibition mediated by gamma-amino-butyric acid [Williamson, Kaplan and Dagan, 1974, *Nature (London)* 252: 224-26] but further physiological data (involving administration of picrotoxin) dispute this hypothesis. *para^{ts1}* is reported to cause an anomalous inflection point in Arrhenius plots, with respect to activity of the mitochondrial enzyme succinate cytochrome c reductase, at a temperature close to that which induces paralysis (Søndergaard, 1976, *Hereditas* 82: 51-56).

alleles: Mutant alleles of *para* are included in the following table.

alleles	discoverer	ref ^α	comments	molecular biology
<i>para^{cs}</i>		2	ts (cold)	
<i>para^{hd1}</i>		11	ts; no P at 14C-D; no wild-type revertants	2.5 kb insert at 45-45.6 kb
<i>para^{hd2}</i>		10, 11	lethal; P at 14C-D; HD→wild-type	0.9 kb insert at 2.0-2.6 kb

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alleles	discoverer	ref ^α	comments	molecular biology
<i>para</i> ^{hd5}		11	revertants lethal; HD→wild-type revertants	1.0 kb insert at 1-2.6 kb
<i>para</i> ^{hd8}		11	<i>P</i> at 14C-D; HD→partial reversion with loss of insert	2.6 kb insert at 42.3-43 kb (2nd insert at -3.3 to -2.8 kb retained in revertants)
<i>para</i> ^{hd9}		11	<i>ts</i> ; no <i>P</i> at 14C-D; no wild-type revertants	3.5 kb insert at 14.3-16.8 kb
<i>para</i> ^{ID30}		11	lethal; <i>In(1)14C6;14D1</i>	break at 0-1 kb
<i>para</i> ^{lk1}			T(1;2)14C7-D1;41A	
<i>para</i> ^{lk2}		11	lethal; <i>In(1)14C6;14D1</i>	break at 20.5-23 kb
<i>para</i> ^{lk4}			<i>In(1)13E9-14;14C7-8</i>	
<i>para</i> ^{lk5}	Kreber	3		
<i>para</i> ^{ST42}		12		
<i>para</i> ^{ST76}		4, 12		
<i>para</i> ^{ST109}		4, 12		
<i>para</i> ^{TP3}	Siddiqi			
<i>para</i> ^{TP8}	Siddiqi			
<i>para</i> ^{TP10}	Siddiqi			
<i>para</i> ^{TP11}	Siddiqi			
<i>para</i> ^{ts1 β}		1, 4-7, 9, 11, 13-15	<i>ts</i>	
<i>para</i> ^{ts2 β}		4, 5	<i>ts</i>	
<i>para</i> ^{ts3}		8	<i>ts</i>	
<i>para</i> ^{ts4}		4	<i>ts</i>	
<i>para</i> ^{ts115}			<i>ts</i>	insertion and deletion? break at 8-9 kb

^α 1 = Benshalom and Dagan, 1985, J. Comp. Physiol. 156: 13-23; 2 = Falk and DeBoer, 1980, Mol. Gen. Genet. 180: 419-24; 3 = Ganetzky, 1984, Genetics 108: 897-911; 4 = Ganetzky, 1986, J. Neurogenet. 3: 19-31; 5 = Grigliatti, Hall, Rosenbluth, and Suzuki, 1973, Mol. Gen. Genet. 120: 107-114; 6 = Grigliatti, Suzuki, and Williamson, 1972, Dev. Biol. 28: 352-71; 7 = Grigliatti, Williamson, and Suzuki, Genetics 64: s27; 8 = Homyk and Pye, 1989, J. Neurogenet. 5: 37-48; 9 = Kyriacou and Hall, 1985, Nature (London) 314: 171-73; 10 = Loughney and Ganetzky, 1985, Neurosci. Abstr. II: 782; 11 = Loughney, Kreber, and Ganetzky, 1989, Cell 58: 1143-54; 12 = Siddiqi and Benzer, 1976, Proc. Nat. Acad. Sci. USA 73: 3253-57; 13 = Suzuki, Grigliatti, and Williamson, 1971, Proc. Nat. Acad. Sci. USA 68: 890-93; 14 = Williamson, Grigliatti, and Suzuki, 1970, Can. J. Genet. Cytol. 12: 400-401; 15 = Williamson, Kaplan, and Dagan, 1974, Nature (London) 252: 224-26.

^β Marked reduction in number of embryonic neurons in cell culture expressing sodium currents in these alleles (O'Dowd, Germeraad, and Aldrich, 1987, Soc. Neurosci. Abstr. 13: 91; 1989, Neuron 2: 1301-11).

cytology: Located at 14C7-8 by *in situ* hybridization; included in *Df(1)80-Df(1)82* (Falk *et al.*, 1984) and covered by *Dp(1;4)r⁺ = Dp(1;4)14A1-2; 16A1-2; 102F2-3*.

molecular biology: Genomic DNA from the *para* region was cloned from a library of *para*^{hd2} using P element DNA as a probe (Loughney and Ganetzky, 1985, Neurosci. Abstr. II: 782). The P element in this mutant was found to reside in a 4.5 kb *EcoRI* fragment; the 14C6-D1 breakpoint of *In(1)D30* (which behaves as *para*⁻) is also within this 4.5 kb fragment. Another *para*⁻ inversion breakpoint, 14C7-8 [in *In(1)para*^{lk4}], has been localized to a site approximately 20 kb distal to the 4.5 kb fragment. Seven other alleles were distributed over a region of 45 kb. The locus includes a minimum of 26 exons. The complete nucleotide sequence of five *para* cDNAs was obtained. There are 5461 nucleotides in one open reading frame (Loughney *et al.*, 1989). Sequence

analysis of the cDNAs indicates that *para* encodes a protein with extensive amino acid identity to membrane spanning domains in the rat, implicating the sodium channels (Loughney and Ganetzky, 1988; Loughney *et al.*, 1989) in *Drosophila* neurons. The *para* transcript appears to produce several subtypes of the sodium channel by alternative splicing.

paralog: see *par*

paralytic: see *para*

parched: see *pch*

parted: see *ptd*

parted: see *ab*²

pas: see *shakB*

pat: see *ptc*

***pat: patchytergum**

location: 1-32.4.

origin: Induced by triethylenemelamine (CB. 1246).

discoverer: Fahmy, 1952.

references: 1958, DIS 32: 73.

phenotype: Wings divergent. Pigmentation of anterior border of fifth tergite patchy. Ocelli light. Male sterile; viability about 10% wild type. RK3.

other information: One allele induced by CB. 3007.

patch: see *ptc*

***patch: patched**

location: 2- (not located).

origin: Spontaneous.

discoverer: Bridges, 13k25.

references: Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 241.

phenotype: Abdominal sclerites fewer or sharply cut into triangular segments obliquely fitted together. Overlaps wild type. RK3.

patched: see *tuf*

***patched:** see *patch*

patchytergum: see *pat*

paternal loss: see *pal*

patulous: see *pa*

pawn: see *pwn*

pb: see *ANTC*

pbl: pebble

location: 3-26.

origin: Induced by ethyl methanesulfonate.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

phenotype: Homozygous lethal in embryo. Mutant embryos have rudimentary head skeleton, denticle bands and filzkörper; posterior end is on dorsal side. Mitosis defective resulting in fewer and much larger cells in embryo.

alleles: Five ethyl-methanesulfonate-induced cold-sensitive

- ^α 1 = Carroll, Laymon, McCutcheon, Riley, and Scott, 1986, Cell 47: 113-22; 2 = Denell, 1973, Genetics 75: 279-97; 3 = Denell, 1978, Genetics 90: 277-89; 4 = Denell, 1982, Dev. Genet. (Amsterdam) 3: 103-13; 5 = Denell and Frederick, 1983, Dev. Biol. 97: 34-47; 6 = Hannah-Alava, 1969, DIS 44: 75; 7 = Haynie, 1983, Dev. Biol. 100: 399-411; 8 = Kennison and Russell, 1987, Genetics 116: 75-86; 9 = Kennison and Tamkun, 1988, Proc. Nat. Acad. Sci. USA 85: 8136-40; 10 = Lewis, E.B., 1978, Nature (London) 276: 565-70; 11 = Lewis, E.B., 1980, DIS 55: 207-08; 12 = Lewis, P.H., 1947, DIS 21: 69; 13 = Puro and Nygrén, 1975, Hereditas 81: 237-48; 14 = Puro, Nygrén, and Nuutila, 1973, DIS 50: 108; 15 = Sato and Denell, 1985, Dev. Biol. 110: 53-64; 16 = Sato, Hayes, and Denell, 1984, Dev. Genet. (Amsterdam) 4: 185-98; 17 = Sato, Russell, and Denell, 1983, Genetics 105: 357-70; 18 = Struhl, 1981, Nature (London) 293: 36-41.
- ^β Sex combs of males are larger and resemble those of *Scx*.
- ^γ Homozygous viable?
- ^δ Two kb deletion.
- ^ε Enhances extra sex combs phenotype of *Pc*⁴.
- ^ζ Cytology: *In(3L)76C;78B + In(3L)78C-D;78E5-6*.

cytology: Located in 78D7-8 by molecular methods and in 78E by deficiency mapping (Duncan and Lewis, 1982); uncovered by *Df(3L)Pc = Df(3L)78D12;79A4-C1* of Jürgens (Haynie, 1983, Dev. Biol. 100: 399-411).

molecular biology: *Pc* region cloned from salivaries (Paro, Lauer, and Hogness) by microexcision. Breakpoints of rearrangements associated with mutant alleles located on the molecular map (Paro *et al.*, 1984). Three transcripts found by Paro, Zink, Messmer, Franke, and Roddewig (Crete, 1988), a 2.5 kb transcript found throughout development, a 2.0 kb maternal and zygotic transcript, and a 1.0 kb transcript abundant in third instar larvae. The polycomb protein binds to 60 sites on the salivary chromosomes, including those of the *ANTC*, *BXC*, and *Pc*-group genes (Zink and Paro, 1989).

other information: Later embryonic stages of homozygous *Pc* embryos show a decline in the transcript levels of *Antp* and *Ubx* and a depression in *Antp* protein expression in the nervous system (Wedeen, Harding, and Levine, 1986, Cell 44: 739-48; Carroll, Laymon, McCutcheon, Riley, and Scott, 1986, Cell 47: 113-22).

PC13: see *t*^{PC13}

PC79: see *soc*

PC80: see *eas*

***pcb*: posterior cell blister (F. Waddle).**

location: 1-1.2.

origin: Spontaneous.

discoverer: Waddle, 1985.

phenotype: Blistering in one or both wings, usually in second posterior cell, but may be elsewhere. Wings tend to be warped upward around affected area. RK1.

***pch*: parched**

location: 1-0.0.

synonym: *dmd*; *doomed*.

references: Benzer, 1971, J. Am. Med. Assoc. 218: 1015-22.

Flanagan, 1977, Genetics 85: 587-607.

Kimura, Shimozawa, and Tanimura, 1985, J. Insect Physiol. 31: 573-80.

phenotype: *pch*² flies appear normal at eclosion but commence dying immediately; all dead within 12 hr. *pch*² progeny recovered about 60% as frequently as expected owing to pre-eclosion mortality in the pupal stage. Approximately 15% of *pch*² individuals die before late

pupa, 25% in late pupa, and 60% during first 24 hr after eclosion. Adults exhibit uncoordinated leg movement and then lose use of legs. In gynandromorphs, only *pch*² tissue shows loss of leg coordination; such mosaics are doomed. *pch*² flies killed by light etherization but not by prolonged CO₂ narcosis. Fate mapping by method of focusing locates focus of lethal action to thoracic neural ganglia and not to thoracic musculature.

Adult *pch*³ and *pch*⁴ flies have higher rates of water loss than wild-type flies, the rapid water loss causing early death in a desiccated environment. In an attempt to compensate for the loss, the mutants drink much more water than wild-type flies. Even dead *pch* flies lose water more rapidly than their wild-type counterparts, suggesting that a defect in the integument rather than in the digestive or trachea-spiracle systems is the source of the abnormality. *pch* larvae and pupae are not affected by desiccation. The survival of flies mosaic for *pch*⁺ and *pch*⁰ in a desiccated environment depends on the ratio of wild-type to mutant cuticle. No difference in the chemical composition of the cuticle between *pch*³ and the wild type detected. The relationship between mutants isolated as parched and doomed remains unclear, considering the reported differences in fate-mapping results (Flanagan vs. Kimura *et al.*).

alleles: Mutations recovered under a number of lethal designations including the adult-lethal mutants called *dmd* and *pch*. Complementation tests by Tanimura and Shimozawa show that *dmd* fails to complement *pch*³, and Schalet demonstrated that several of the lethal alleles listed below fail to complement *dmd = pch*².

allele	origin	discoverer	synonym	ref ^α	comments
<i>pch</i> ¹	EMS	Benzer		1	adult lethal
<i>pch</i> ²		Sandler	<i>dmd</i>	3	adult lethal
<i>pch</i> ³	EMS	Kimura	<i>pch</i> ^{KK78}	4	adult lethal
<i>pch</i> ⁴	EMS	Tanimura	<i>pch</i> ^{TT362}	4	adult lethal
<i>pch</i> ⁵	spont	Bridges, '28	<i>l(1)7e</i> , <i>l(1)d</i>	2,7,8	
<i>pch</i> ⁶		Novitski	<i>l(1)149</i>	7	
<i>pch</i> ⁷		Novitski	<i>l(1)152</i>	7	
<i>pch</i> ⁸		Novitski	<i>l(1)1403</i>	7	
<i>pch</i> ⁹	EMS	Lim	<i>l(1)EC1</i>	5	
<i>pch</i> ¹⁰	EMS	Lim	<i>l(1)EC1</i> ⁰⁰²		complementing allele ^β
<i>pch</i> ¹¹	EMS	Lim	<i>l(1)EC1</i> ⁰⁰⁹		
<i>pch</i> ¹²	EMS	Lim	<i>l(1)EC1</i> ⁹¹		noncomplementing allele
<i>pch</i> ¹³	EMS	Lim	<i>l(1)EC1</i> ¹¹¹		
<i>pch</i> ¹⁴	EMS	Lim	<i>l(1)EC1</i> ¹²⁴		
<i>pch</i> ¹⁵	EMS	Lim	<i>l(1)EC1</i> ¹⁴⁰		
<i>pch</i> ¹⁶	DNA ^γ	Fox	<i>l(1)EC1F</i> ²²⁵		
<i>*pch</i> ¹⁷	X ray	Meyer	<i>l(1)HM1</i>		
<i>*pch</i> ¹⁸	X ray	Meyer	<i>l(1)HM2</i>		
<i>pch</i> ¹⁹	X ray	Meyer	<i>l(1)HM5</i>		
<i>*pch</i> ²⁰	X ray	Lefevre	<i>l(1)HC246</i>	5	<i>In(1)1A4;4C9-10</i>
<i>*pch</i> ²¹	X ray	Lefevre	<i>l(1)HC310</i>	5	
<i>pch</i> ²²	EMS	Lefevre	<i>l(1)EF463</i>	6	adult lethal; no maternal effect; noncomplementing allele
<i>pch</i> ²³	EMS	White			complementing allele
<i>pch</i> ²⁴	X ray	White			complementing allele
<i>pch</i> ²⁵	ENU	Voelker	<i>l(1)A33</i>		noncomplementing allele
<i>pch</i> ²⁶	MR		<i>l(1)32/51B</i>	1	noncomplementing allele
<i>pch</i> ²⁷	X ray	van Zeeland	<i>l(1)8913-1E</i>		noncomplementing allele
<i>pch</i> ²⁸	X ray	van Zeeland	<i>l(1)8919-37E</i>		noncomplementing

allele	origin	discoverer	synonym	ref ^α	comments
pch²⁹	<i>mei9</i> ^δ	Schalet	<i>l(1)3-2D-82</i>		allele complementing allele
pch³⁰	<i>mei9</i>	Schalet	<i>l(1)7-1B-4</i>		allele complementing allele

^α 1 = Benzer, 1971, J. Am. Med. Assoc. 218: 1015-22; 2 = CP 627; 3 = Flanagan, 1977, Genetics 85: 587-607; 4 = Kimura, Shimozawa, and Tanimura, 1985, J. Insect Physiol. 31: 573-80; 5 = Lefevre, 1981, Genetics 99: 461-80; 6 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 7 = Maddern, 1972, DIS 49: 48; 8 = Maddern, 1977, DIS 52:82.

^β Complementation with *pch²* in every instance.

^γ From DNA-injected egg.

^δ Spontaneous in the paternal X chromosome of a cross between wild-type males and *mei9* females, such that the F₁ females were *pch/mei9*.

cytology: Provisionally the most distal locus on the X chromosome. Located in the interval between 1A1 and 1B1 since uncovered by *Df(1)260-1 = Df(1)1A1;1B4-6* and covered by *Df(1)sc19 = Df(1)1B2;1B4*.

Pch: see *pyd*

***Pcl*: Polycomblike**

location: 2-84.

discoverer: Duncan.

references: Duncan and Lewis, 1982, Symp. Soc. Dev. Biol., 40th, pp. 533-34.

Duncan, 1982, Genetics 102: 49-70.

Sato, Russell, and Denell, 1983, Genetics 105: 357-70.

Jürgens, 1985, Nature (London) 316: 153-55.

Kennison and Russell, 1987, Genetics 116: 75-86.

phenotype: *Pcl*⁺, like *Pc*⁺, may be considered a negative regulator of the *BXC* and the *ANTC*. Strong *Pcl* mutant alleles (*Pcl*¹, *Pcl*²) are homozygous lethal, hemizygous lethal, and lethal with each other, the embryos having partial posteriorly-directed transformations of the abdominal segments (segment 1 into segment 2 and segments 2-7 into more posterior segments). These mutants are strong enhancers of *Pc*³, showing *Pc*-like transformations in embryos, and are indistinguishable from *Pcl* deficiencies in complementation behavior. Extreme posteriorly-directed segmental transformations are found in *Pcl*¹/*Df(2R)Pcl11B*; *Pc*³/*Pc*³ lethal embryos (Duncan, 1982); these embryos are considerably more abnormal than the *Pc*³ homozygotes that are *Pcl*⁺. *Pcl*/*Pcl* embryos that are also homozygous for *Asx*, *Psc*, or *Scm* show strong posteriorly-directed transformations of the entire body pattern; the triple mutant *Psc Asx Pcl* has a tandem array of posterior abdominal segments including four abdominal denticle bands in the head region (Jürgens, 1985). *Pcl*¹/*Pcl*¹ clones in the second through the sixth abdominal tergites also show caudal transformations; similar clones in the wing often appear to be partially transformed into haltere tissue.

Weak *Pcl* alleles (*Pcl*³, *Pcl*⁴) survive, at least through eclosion, either as homozygotes or as heterozygotes with strong or weak *Pcl* alleles (Duncan, 1982). *Pcl*¹/*Pcl*³ and *Pcl*¹/*Pcl*⁴ show segmental transformations characteristic of *Pcl*⁺ flies, i.e. antenna to leg, second and third leg to first leg, wing to haltere, and posteriorly-directed abdominal transformations (the most frequent abnormality). Increasing the dosage of the *BXC* in *Pcl*¹/*Pcl*⁴ heterozygotes suppresses the posteriorly-directed transformations, but enhances the transformations from

leg2 and *leg3* to *leg1*; increasing the dosage of the *ANTC* enhances both types of transformations (Duncan, 1982). *Pcl*¹/*Pcl*³ females, which die before or soon after eclosion, show a conversion of parovaria to spermathecae. Both strong and weak *Pcl* alleles are viable as heterozygotes (*Pcl*/+) and may show partial abdominal and leg transformations, the abnormalities increasing in *Pcl*/+; *Pcl*/+ flies or when *Pcl*/+ is combined with *E(Pc)*.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>Pcl</i>^{1β}	X ray	Duncan		2	homozygous lethal; enhances <i>Pc</i> ³
<i>Pcl</i>²	EMS	Duncan		2	homozygous lethal; enhances <i>Pc</i> ³
<i>Pcl</i>³	EMS	Duncan		2	homozygous viable; leg2, leg3 → leg1 (het)
<i>Pcl</i>^{4γ}	EMS	Duncan		2	homozygous viable; leg2, leg3 → leg1 (het)
<i>Pcl</i>⁵	ENU	Breen	<i>Pcl</i> ^{D5}	1	enhances <i>Mcp</i>
<i>Pcl</i>⁶	EMS	Struhl	<i>Pcl</i> ^{E33}	4	leg2, leg3 → leg1 (het)
<i>Pcl</i>⁷	EMS	Struhl	<i>Pcl</i> ^{E90}	4	leg2, leg3 → leg1 (het)
<i>Pcl</i>⁸	EMS		<i>Pcl</i> ^{E501}		
<i>Pcl</i>^{9δ}		E.B. Lewis	<i>Pcl</i> ^{P3}	3	homozygous lethal
<i>Pcl</i>¹⁰	γ ray	Tiong	<i>Pcl</i> ^{T1}	5	
<i>Pcl</i>^{11ε}	γ ray	Williams	<i>Pcl</i> ^{W4}	5, 7, 8	leg2, leg3 → leg1 (het)
<i>Pcl</i>¹²	γ ray	Williams	<i>Pcl</i> ^{W6}	7, 8	leg2, leg3 → leg1 (het)
<i>Pcl</i>¹³	X ray		<i>Pcl</i> ^{X21}	9	
<i>Pcl</i>¹⁴	X ray		<i>Pcl</i> ^{XF21}	9	
<i>Pcl</i>¹⁵	X ray		<i>Pcl</i> ^{XM3}	9	
<i>Pcl</i>¹⁶	X ray		<i>Pcl</i> ^{XT9}	9	
<i>Pcl</i>¹⁷	EMS		<i>Pcl</i> ^{XT56}	4	homozygous viable; leg2, leg3 → leg1
<i>Pcl</i>¹⁸	EMS		<i>Pcl</i> ^{K1}	6	enhances extra sex combs phenotype of <i>Pc</i> ⁴

^α 1 = Breen and Duncan, 1986, Dev. Biol. 118: 442-56; 2 = Duncan, 1982, Genetics 102: 49-70; 3 = Eberlein, 1984, Genetics 107: s27-28; 4 = Jürgens, 1985, Nature (London) 316: 153-55; 5 = Kennison and Russell, 1987, Genetics 116: 75-86; 6 = Kennison and Tamkun, 1988, Proc. Nat. Acad. Sci. USA 85: 8136-40; 7 = Sato, Hayes, and Denell, 1984, Dev. Genet. (Amsterdam) 4: 185-98; 8 = Sato, Russell, and Denell, 1983, Genetics 105: 357-70; 9 = Tearle and Nüsslein-Volhard, 1987, DIS 66: 211-26.

^β Strong interaction with *Pc*³.

^γ Homozygotes almost wild type; weakly temperature-sensitive for leg transformations at 29°C.

^δ Partial transformation of legs 2 and 3 to leg 1. Enhances wing to haltere transformation in *en*¹/*en*²⁸ (Eberlein, 1984).

^ε *In(2R)55A;57A*.

cytology: Located between 55A and 55C1 since *Pcl* is included in *Df(2R)Pcl-W5 = Df(2R)55A-B;55C* as well as in *Df(2R)Pcl7B = Df(2R)54E8-F1;55B9-C1* and *Df(2R)Pcl11B = Df(2R)54F6-55A1;55C1-3* (Duncan, 1982; Sato *et al.*, 1983). These deficiencies act as enhancers of *Pc*³ and show a strong *Pcl* phenotype.

pco: see *E(z)*

***Pcp*: Pupal cuticle protein**

location: 2-20 (based on site of *ade3*).

origin: Sequence analysis of *ade3*.

synonym: *Pcpgart*; *Gart* intronic gene.

references: Henikoff, Keene, Fechtel, and Fristrom, 1986, Cell 44: 33-42.

phenotype: Apparently encodes pupal cuticle protein since in 11 hr prepupae, the *Pcp* mRNA is found primarily in the abdomen in epidermis involved in cuticle formation. The pupal cuticle protein itself has not been identified.

cytology: Placed in 27C.

molecular biology: Computer analysis of *ade3* sequences located *Pcp* in an open reading frame of 184 amino acids in a nested arrangement on the noncoding strand within the first *ade3* intron (interrupting the GARS domain). The *Pcp* gene is made up of single copy DNA and contains its own intron between codons 4 and 5. Its cDNA sequence shows strong homology to regions of the genes *Lcp1* to *Lcp4* (Snyder, Hirsh, and Davidson, 1981, Cell 25: 165-77; Snyder, Hunkapiller, Yuen, Silvert, Fristrom, and Davidson, 1982, Cell 29: 1027-40). A 0.9 kb transcript was detected primarily, if not exclusively, in prepupal stage (Henikoff *et al.*, 1986).

pcv: posterior crossvein

location: 3-.

origin: X ray induced.

references: Puro, 1982, DIS 58: 205-08.

phenotype: Posterior crossvein incomplete. Wings slightly divergent. Not an allele of *cv-d* since *pcv/cv-d* is wild type.

cytology: Associated with *In(3LR)pcv = In(3LR)65B-C;92A*.

pcx: pecanex

location: 1-0.9.

origin: Induced by ethyl methanesulfonate.

discoverer: Engstrom (*pcx*); Romans [*mel(1)R1*].

synonym: *fs(1)pcx; mel(1)R1*.

references: Romans, 1973, Genetics 74: s233.

Romans, Hodgetts, and Nash, 1976, Can. J. Genet. Cytol. 18: 773-81.

Mahowald, 1983, Time, Space, and Pattern in Embryonic Development (Jeffrey, Rand, and Raff, eds.). Alan R. Liss, Inc., New York, pp. 349-63.

Perrimon, Engstrom, and Mahowald, 1984, Genetics 108: 559-72.

Haenlin, Steller, Pirrotta, and Mohler, 1985, Cell 40: 827-37.

La Bonne and Mahowald, 1985a, Dev. Biol. 110: 264-67.

1985b, Genetics 110: s83.

La Bonne, Sunitha, and Mahowald, 1989.

phenotype: Maternal-effect-lethal mutation; homozygous mutant females crossed to mutant males produce lethal embryos showing hypertrophy of the central nervous system at the expense of the epidermis, a small patch of dorsal cuticle remaining but no ventral cuticle. This embryonic phenotype (as well as the reduced eye phenotype of viable *pcx* adults) resembles that of *amx*. Homozygous *pcx* lethal embryos can be rescued genetically; some mutant females (but not *pcx*¹ homozygotes) crossed to wild-type males produce viable adult *pcx*/+ female offspring (Romans *et al.*, 1976; Perrimon *et al.*, 1984); the *pcx*/Y male offspring are lethal. *pcx*/+ heterozygous females, which are fertile, crossed to viable *pcx*/Y males (also fertile) produce viable adult males (*pcx*/Y and +/Y) and viable adult females (*pcx*/*pcx* and *pcx*/+) (Romans *et al.*, 1976). Homozygous *pcx* lethal embryos can be partially rescued almost up to hatching by preblastoderm microinjection of wild-type or *amx* ooplasm (La

Bonne and Mahowald, 1985a). Germ line clonal analysis demonstrates the germ-line dependency of the *pcx* embryonic lethal phenotype (Perrimon *et al.*, 1984).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>pcx</i> ¹	EMS	Romans	<i>mel(1)R1</i> ^o	3	
<i>pcx</i> ²	EMS	Romans	<i>mel(1)R1</i> ^r	3	
<i>pcx</i> ³	EMS	Mohler	<i>12-1012</i>	1	
<i>pcx</i> ⁴	EMS	Mohler	<i>12-1743</i>	1	
<i>pcx</i> ⁵	EMS	Mohler	<i>12-3014</i>	1	
<i>pcx</i> ⁶	EMS	Mohler	<i>12-3102</i>	1	
<i>pcx</i> ⁷	EMS	Mohler	<i>12-3135</i>	1	
<i>pcx</i> ⁸	EMS	Mohler	<i>12-4169</i>	1	
<i>pcx</i> ⁹	EMS	Mohler	<i>14-567</i>	1	
<i>pcx</i> ¹⁰	EMS	Mohler	<i>14-1153</i>	1	
<i>pcx</i> ¹¹	EMS		<i>pcx</i> ¹	2	

^α 1 = Mohler and Carroll, 1984, DIS 60: 236-41; 2 = Perrimon, Engstrom, and Mahowald, 1984, Genetics 108: 559-72; 3 = Romans, Hodgetts, and Nash, 1976, Can. J. Genet. Cytol. 18: 773-81.

cytology: Located in 2E2 since included in *Df(1)pn38 = Df(1)2D3-4;3E3* but not *Df(1)dor2T = Df(2B6;2E1-2*.

molecular biology: *pcx* cloned and transcripts identified (tentatively by Haenlin *et al.*, 1985; confirmed by La Bonne *et al.*, 1989). These mRNAs include a rare 9 kb transcript and minor 3.7 and 2.3 kb transcripts; they are detected early (at a low level) in 0-1 hr embryos. The level is highest in 5-10 hr embryos; lower levels persist throughout pupal and adult life (La Bonne *et al.*, 1989). Nucleotide and predicted protein sequences of the 3' portion of the locus have been determined; these sequences suggest that *pcx* encodes a large transmembrane protein (La Bonne *et al.*).

other information: Named for its phenotypic resemblance to *amx*.

pd: purpleoid

location: 2-106.4.

origin: Spontaneous.

discoverer: Bridges, 16h31.

references: 1937, Cytologia (Tokyo), Fujii Jub., Vol. 2: 745-55.

phenotype: Eye color dark pink or maroon, like *pr* but less extreme. Semidominant; eye color of heterozygote duller than wild type; color autonomous in larval optic disk transplanted into wild-type host (Beadle and Ephrussi, 1936, Genetics 21: 230). Malpighian tubes wild type (Beadle, 1937, Genetics 22: 587-611). Semilethal with *dor* (Lucchesi, 1968, Genetics 59: 37-54). RK2.

cytology: Placed in region between 59E2 and 60B10 by Bridges (1937) on the basis of its being to the right of *In(2R)bw*^{VDel} = *In(2R)41B2-C1;59E2-4* and to the left of *Df(2R)Px = Df(2R)60B8-10;60D1-2*.

pdf: pod foot

location: 1-57.0.

origin: X ray induced.

discoverer: Welshons, 57h6.

references: 1960, DIS 34: 54.

phenotype: Terminal tarsus swollen in one or more legs. Classification, viability, and fertility good. RK2A.

cytology: Associated with *In(1)pdf = In(1)16B;19F-20A*. Tentatively placed in 16A and at 57.0 since *pdf* is covered by *B*^S*Y* but not by *Ymal*⁺*2*.

Pdr: Purpleoider

location: 3-46.

origin: Spontaneous.

discoverer: Bridges, 22f20.

phenotype: The combination *pd/pd; Pdr/+* gives lighter, yellower eye color than *pd* alone. *pd/+; Pdr/Pdr* has eye color like *pd/pd*. *pd/pd; Pdr/Pdr* is lethal. *Pdr/Pdr* is rosier than wild type. *Pdr/Pdr* and *pd/pd; Pdr/+* Malpighian tubes normal (Brehme and Demerec, 1942, Growth 6: 351-56). RK3.

***pe: pett**

location: 3- (not located).

origin: Spontaneous in *In(3L)P*.

discoverer: Mohr, 38k30.

references: 1939, DIS 12: 47.

phenotype: Body small. Eyes small and rough. Viability good; female fertility low. RK2A.

Pearl: see *PI*

peb: pebbled

location: 1-7.5+. Mapped by recombination as proximal to *rb* (Schalet, 1986).

discoverer: Dubinin.

references: Schalet, 1986, Mutat. Res. 163: 115-44.

Oliver, Perrimon, and Mahowald, 1988, Genetics 120: 159-71.

Steinmann-Zwicky, 1988, EMBO J. 7: 3889-98.

phenotype: Eyes markedly rough at 28°-30°, slightly rough (like *S*) at 25°, and wild type at 19°. RK2 (28°-30°).

alleles: Two spontaneous lethal alleles, *peb*² and *peb*³ isolated as *l(1)10-75* and *l(1)20-41*, (Schalet *et al.*, 1986).

cytology: Placed in salivary chromosome region 4C5-7 since included in *Df(1)ovo7 = Df(1)4C5-6;4E2-3* and *Df(1)rb46 = Df(1)4A3-6;4C6-7* (Oliver *et al.*, 1988).

pebble: see *pbl*

pebbled: see *peb*

pebbly: see *pby*

Pec: Pupilla eccentrica

location: 3-65.

origin: Spontaneous in a sex-linked lethal stock kept at 26°.

references: Parkash, 1970, DIS 45: 35.

phenotype: Temperature-sensitive change in the appearance of the pupilla of the eye. At 26°, this pigment-free circular area is enlarged to a diameter of about 2/5 of the long axis of the eye and is demarcated clearly from the pigmented area; it contains scattered pigment spots. The pigmented part of the eye varies in color from dull red to light brown and the surface of the eye is somewhat rough; ocelli are colorless. As the temperature is lowered, the size of the pupilla is reduced and its boundary becomes blurry until at 16° the eye resembles wild-type. The temperature-sensitive period for development of the *Pec* phenotype seems to be the first day of pupal life.

pecanex: see *pcx*

pelle: see *pil*

pentagon: see *ptg*

pep: peppercorn (T. Schüpbach)

location: 2-{104}.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus.

phenotype: Female sterile; homozygous females contain many small egg chambers in their ovaries, which seem blocked in early stages of oogenesis. Follicle cells nevertheless synthesize a very small, round chorion around these egg chambers, giving them the appearance of small round peppercorns.

alleles: *pep*^{QW} = *pep*¹.

cytology: Placed in 59D8-60A7, since uncovered by *Df(2R)bw-S46 = Df(2R)59D8-11,60A7*.

Pepck: Phosphoenolpyruvate-carboxykinase

references: Gundelfinger, Hermans-Borgmeyer, Grenningloh, and Zopf, 1987, Nucleic Acids Res. 15: 6745.

phenotype: Structural gene for the enzyme phosphoenolpyruvate-carboxykinase in *Drosophila melanogaster*.

molecular biology: A cDNA clone of *Pepck* has been isolated and found to encode a polypeptide that is 64% identical to the rodent and 62% identical to the avian enzyme precursors. The predicted *Drosophila* protein consists of 747 amino acid residues.

peppercorn: see *pep*

per: period (J.C. Hall; M. Young)

location: 1-1.4 (between *z* and *w*).

origin: Induced by ethyl methanesulfonate or nitrosoguanidine.

discoverer: Konopka.

references: Konopka and Benzer, 1971, Proc. Nat. Acad. Sci. USA 68: 2112-16.

Young and Judd, 1978, Genetics 88: 723-42.

Handler and Konopka, 1979, Nature (London) 279: 236-38.

Kyriacou and Hall, 1980, Proc. Nat. Acad. Sci. USA 77: 6729-33.

Smith and Konopka, 1981, Mol. Gen. Genet. 183: 243-51.

Kyriacou and Hall, 1982, Anim. Behav. 30: 794-801.

Smith and Konopka, 1982, Mol. Gen. Genet. 185: 30-36.

Bargiello, Jackson, and Young, 1984, Nature (London) 312: 752-54.

Bargiello and Young, 1984, Proc. Nat. Acad. Sci. USA 81: 2142-46.

Reddy, Zehring, Wheeler, Pirrotta, Hadfield, Hall, and Rosbash, 1984, Cell 38: 701-10.

Zehring, Wheeler, Reddy, Konopka, Kyriacou, Rosbash, and Hall, 1984, Cell 39: 752-54.

Shin, Bargiello, Clark, Jackson, and Young, 1985, Nature (London) 317: 445-48.

Young, Jackson, Shin, and Bargiello, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 865-75.

Côté and Brody, 1986, J. Theoret. Biol. 121: 487-503.

Hamblen, Zehring, Kyriacou, Reddy, Yu, Wheeler, Zwiebel, Konopka, Rosbash, and Hall, 1986, J. Neurogenet. 3: 249-91.

James, Ewer, Reddy, Hall, and Rosbash, 1986, EMBO J. 5: 2313-20.

Kyriacou and Hall, 1986, Science 232: 494-97.

Jackson, Bargiello, Yun, and Young, 1986, Nature (London) 320: 185-88.

Reddy, Jacquier, Abovich, Peterson, and Rosbash, 1986,

Cell 46: 53-61.

Bargiello, Saez, Baylies, Gasic, Young, and Spray, 1987, Nature (London) 328: 686-91.

Baylies, Bargiello, Jackson, and Young, 1987, Nature (London) 326: 390-92.

Citri, Colot, Jacquier, Yu, Hall, Baltimore, and Rosbash, 1987, Nature (London) 326: 42-47.

Dowse, Hall, and Ringo, 1987, Behav. Genet, 17: 19-25.

Dowse and Ringo, 1987, J. Biol. Rhythms 2: 65-76.

Hall and Rosbash, 1987a, Trends Genet. 3: 185-91.

1987b, Bio Essays 7: 108-12.

1987c, J. Biol. Rhythms 2: 153-78.

Konopka, 1987, Life Sci. Adv. Series 5: 47-49.

Yu, Colot, Kyriacou, Hall, and Rosbash, 1987a, Nature (London) 326: 765-69.

Yu, Jacquier, Citri, Hamblen, Hall, and Rosbash, 1987b, Proc. Nat. Acad. Sci. USA 84: 784-88.

Crossley, 1988, Anim. Behav. 36: 1098-1109.

Ewing, 1988, Anim. Behav. 36: 1091-97.

Hall and Rosbash, 1988, Ann. Rev. Neurosci. 11: 373-93.

Kyriacou and Hall, 1988, Anim. Behav. 36: 1110.

Liu, Lorenz, Yu, Hall, and Rosbash, 1988, Genes Dev. 2: 228-38.

Saez and Young, 1988, Mol. Cell Biol. 8: 4588-99.

Young, Bargiello, Baylies, Saez, and Spray, 1988, Cellular and Neuronal Oscillators, (M. Dekke, ed.) pp. 507-20.

Hamblen-Coyle, Konopka, Zwiebel, Colot, Dowse, Rosbash, and Hall, 1989, G. Neurogenet. 5: 229-56.

phenotype: The *per* gene is essential for biological clock functions and determines the period length of circadian and ultradian rhythms. The *per* mutants are characterized by aberrant rhythms involving eclosion and locomotor activity (Konopka and Benzer, 1971) and may change the rhythmic component of the male courtship song (Crossley, 1988; Ewing, 1988; Kyriacou and Hall, 1980, 1986, 1988). These mutants also affect the rhythm of the larval heartbeat (Dowse, Ringo, and Kyriacou; Livingstone, 1981, Neurosci. Abstr. 7: 351), the level of tyrosine decarboxylase [Livingstone and Tempel, 1983, Nature (London) 303: 67-70], and fluctuations in membrane potentials in larval salivary glands (Weitzel and Rensing, 1981, J. Comp. Physiol. 143: 229-35), modulate intercellular junctional communication (Bargiello *et al.*, 1987), and alter the location of neural secretory cells in the brain (Konopka and Wells, 1980, J. Neurobiol. 11: 411-15).

In wild-type flies the period length is about 24 hr. In general, increases in *per*⁺ dosage lead to shortened circadian rhythms and decreases lead to lengthened circadian rhythms (Baylies *et al.*, 1987; Côté and Brody, 1986; Hamblen *et al.*, 1986; Smith and Konopka, 1981, 1982; Young *et al.*, 1985). Females heterozygous for *per*⁺ and a deletion of the locus or a *per*⁰ allele show longer-than-normal periods.

per flies can be classified on the basis of their circadian rhythms as: (1) Cryptic period mutants (*per*⁰, *per*⁻) which have a 10-15 hr (ultradian) period and appear

arrhythmic except in special algorithmic tests (Dowse *et al.*, 1987); (2) Long period mutants (*per*^L), 29 hr; (3) Long-period variable mutants (*per*^{Lvar}), which in homozygotes or heterozygotes are arrhythmic but in combination with certain partial deletions of the *per* locus result in a 30-34 hr period. (Konopka, 1987); (4) Short period mutants (*per*^S), 19 hr; (5) Short period variable mutants (*per*^{Svar}), some flies having a 20 hr period and the others a normal 24 hr period for locomotor activity.

In temperature-change experiments on *per*^S and *per*^{L1}, the locomotor activity periods were found to be nearer to 24 hr at low temperatures, but to diverge further from normal upon heating (Konopka, Pittendrigh, and Orr; Hamblen, Ewer, and Hall). *per*^{L2} shows lengthening of the periods at high temperatures.

The mutant types affecting circadian rhythms (*per*⁰, *per*^L, and *per*^S) may cause similar kinds of changes in the rhythmic fluctuations in courtship song intersperse intervals (IPIs) of the male (Crossley, 1988; Ewing, 1988; Kyriacou and Hall, 1980, 1986, 1988). *per*⁰ mutants show nonrhythmic variations in the interval between pulses of wing vibration.

Neural studies show that transplantation of *per*^S brains into *per*⁰¹ adult hosts causes some of the hosts to be "rescued"; *i.e.* to show short-period circadian rhythms for locomotor activity (Handler and Konopka, 1979). Octopamine synthesis occurs at subnormal rates in *per*⁰¹ brains, with a corresponding decrease in the enzyme tyrosine decarboxylase (Livingstone and Tempel, 1983); less severe decrements in tyrosine decarboxylase are found in *per*^S and *per*^{L1} flies.

Physiological studies show that *per* mutations can affect the level of gap junctional communication among cells in a tissue (Bargiello *et al.*, 1987). In salivary glands the *per*⁰ and *per*^{L1} mutations cause a lowering of the level of junctional communication, while *per*^S gives a level of communication higher than wild type. Because electrical synapses are composed of gap junctions, *per* may influence circadian behavioral rhythms through altered conductances at the synapse (Bargiello *et al.*, 1987).

Mosaic analysis of *per*^S mutants indicates that the gene influences the brain with respect to aberrant locomotor rhythms (Konopka, Wells, and Lee, 1983, Mol. Gen. Genet. 190: 284-88); *per*⁰¹ and *per*⁰² (and, to a lesser degree, *per*^S) are said to cause anomalous photonegative behavior in light-response tests (Palmer, Kendrick, and Hotchkiss, 1985, Ann. N.Y. Acad. Sci., pp 323-24), but in general are not defective in visual responses (phototaxis tests, optomotor behavior, and electroretinogram) according to Dushay and Hall.

alleles: Ten mutant alleles of *per* are described in the table below. *per*⁰¹, *per*⁰², and *per*⁰³ may be the same allele originating at different times, (all three carry the same nonsense or stop codon); *per*⁰⁴ is a different allele. See rearrangement section for *T(1;4)JC43* which shows circadian rhythms with a long period.

alleles	origin	discoverer	ref ^α	comments	molecular data
<i>per</i> ^{-β}			1-7, 9, 25	multiple periods; short ultradian rhythms; hyperactive flies	deletion of 10 kb
<i>per</i> ^{01γ}	EMS	Konopka, Benzer	1-10, 12-14, 16-27	multiple periods; short ultradian rhythms	nonsense mutation in exon 4
<i>per</i> ⁰²	EMS	Smith, Konopka	5-7, 19, 23, 26	multiple periods; short ultradian rhythms	same nonsense codon as <i>per</i> ⁰¹
<i>per</i> ⁰³	EMS	Konopka	6, 7, 26	multiple periods; short ultradian rhythms	same nonsense codon as <i>per</i> ⁰¹
<i>per</i> ⁰⁴	EMS/NNG	Konopka	6, 21, 26	multiple periods; short ultradian rhythms; hyperactive flies	<i>per</i> ⁰¹ nonsense codon absent
<i>per</i> ^{L1δ}	EMS	Konopka, Benzer	3, 4, 6, 7, 10-13	circadian rhythms; long period	nucleotide substitution in exon 3
<i>per</i> ^{L2δ}	EMS	Orr	16, 23, 26 9, 23	circadian rhythms; long period	
<i>per</i> ^{Lvar}	EMS/NNG	Konopka	7, 11	circadian rhythms; variable long period	
<i>per</i> ^s	EMS	Konopka, Benzer	3, 4, 6, 8, 10, 12, 13	circadian rhythms; short period	nucleotide substitution in exon 5
<i>per</i> ^{svar}	NNG	Konopka	15, 16, 23, 26 7	circadian rhythms; variable short period	

^α 1 = Bargiello, Jackson, and Young, 1984, Nature (London) 312: 752-54; 2 = Bargiello, Saez, Baylies, Gasic, Young, and Spray, 1987, Nature (London) 328: 686-91; 3 = Bargiello and Young, 1984, Proc. Nat. Acad. Sci. USA 81: 2142-46; 4 = Baylies, Bargiello, Jackson, and Young, 1987, Nature (London) 326: 390-92; 5 = Dowse, Hall, and Ringo, 1987, Behav. Genet. 17: 19-35; 6 = Hall and Rosbash, 1987, J. Biol. Rhythms 2: 153-78; 7 = Hamblen, Zehring, Kyriacou, Reddy, Yu, Wheeler, Zwiebel, Konopka, Rosbash, and Hall, 1986, J. Neurogenet. 3: 249-91; 8 = Handler and Konopka, 1979, Nature (London) 279: 236-38; 9 = Jackson, Bargiello, Yun, and Young, 1986, Nature (London) 320: 185-88; 10 = Jackson, Gailey, and Siegel, 1983, J. Comp. Physiol. 151: 545-52; 11 = Konopka, 1987, Life Sci. Adv. Series C5: 47-49; 12 = Konopka and Benzer, 1971, Proc. Nat. Acad. Sci. USA 68: 2112-16; 13 = Konopka and Orr, 1980, Development and Neurobiology of Drosophila (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York, pp. 409-16; 14 = Konopka and Wells, 1980, J. Neurobiol. 11: 411-15; 15 = Konopka, Wells, and Lee, 1983, Mol. Gen. Genet. 190: 284-88; 16 = Kyriacou and Hall, 1980, Proc. Nat. Acad. Sci. USA 77: 6729-33; 17 = Livingstone, 1981, Neurosci. Abstr. 7: 351; 18 = Livingstone and Tempel, 1983, Nature (London) 303: 67-70; 19 = Palmer, Kendrick, and Hotchkiss, 1985, Ann. N.Y. Acad. Sci. 323-24; 20 = Reddy, Zehring, Wheeler, Pirrotta, Hadfield, Hall, and Rosbash, 1984, Cell 38: 701-10; 21 = Siwicki, Eastman, Petersen, Rosbash, and Hall, 1988, Neuron 1: 141-50; 22 = Smith and Konopka, 1981, Mol. Gen. Genet. 183: 243-51; 23 = Smith and Konopka, 1982, Mol. Gen. Genet. 185: 30-36; 24 = Weitzel and Rensing, 1981, J. Comp. Physiol. 143: 229-35; 25 = Young, Jackson, Shin, and Bargiello, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 865-75; 26 = Yu, Jacquier, Citri, Hamblen, Hall, and Rosbash, 1987, Proc. Nat. Acad. Sci. USA 84: 784-88; 27 = Zehring, Wheeler, Reddy, Konopka, Kyriacou, Rosbash, and Hall, 1984, Cell 39: 369-76.

^β Includes deficiencies for the *per* locus such as *Df(1)TEM202/Df(1)64j4* or *Df(1)62d18/Df(1)64j4*.
^γ *per*⁰ listed as *per*^o in many references.
^δ Synonym: *per*^{L1} = *per*¹;
per^{L2} = *per*¹².

cytology: Located in 3B1-2 since *per*⁰ mutations are uncovered by a variety of deletions in the *zeste-white* region, including *Df(1)64j4* = *Df(1)3A8-9;3B1-2*, *Df(1)62d18* = *Df(1)3B1-2;3C6-7*, *Df(1)64f1* = *Df(1)3A9-B1;3B2-3* and *Df(1)TEM* = *Df(1)3B1-2;3C3-5* and are covered by *w*⁺*Y* and *Dp(3;1)w*⁺*67k27*. The 3B1-2 breakpoint of *T(1;4)JC43* = *T(1;4)3B1-2;3E3-4;102D* "partially" inactivates *per*⁺, making circadian eclosion arrhythmic (Smith and Konopka, 1981; Young and Judd, 1978); in regard to circadian locomotor activity; however, some *T(1;4)JC43/per*⁰ females are weakly rhythmic and have a long period while the rest are arrhythmic. A synthetic deletion of the *X* between 3B1-2 and 3C2 (the 4^P*X*^D segment of *T(1;4)JC43* and the *X*^{P2}^D segment of *T(1;2)RC45*) leads to the same array of long-period/arrhythmic individuals when heterozygous with *per*⁰¹ (Smith and Konopka, 1981).

molecular biology: The *per* locus and its environs have been cloned by chromosomal walking/jumping (Bargiello and Young, 1984) and by micro-excision (Reddy *et al.*, 1984) and the genomic *per* DNA sequenced (Citri *et al.*, 1987; Jackson *et al.*, 1986) Sequencing data and immunochemical studies suggest that the gene product is a proteoglycan (Bargiello *et al.*, 1987; Jackson *et al.*, 1986; Reddy *et al.*, 1986; Shin *et al.*, 1985). Four types of transcripts have been reported (4.5 kb, 0.9 kb, 2.7 kb, and 1.7 kb) from the region of the *per* locus. The 4.5 kb transcript class is complex, comprising three messages produced by

differential splicing which differ in their 3' sequences (Yu *et al.*, 1987b). The 4.5 kb transcript class almost certainly codes for the *per* gene product (Bargiello *et al.*, 1984; Bargiello and Young, 1984; Bayles *et al.*, 1987; Citri *et al.*, 1987; Hamblen *et al.*, 1986; Young *et al.*, 1985; Yu *et al.*, 1987a,b). Three types of cDNA (A, B, and C), corresponding to three species of 4.5 kb RNA, have been cloned and their sequences compared (Citri *et al.*, 1987). In type A cDNA (the eight-exon type and the most abundant), the first (5'-most) and the last exon (to the right of the stop codon) are noncoding; exon 5 encodes a Threonine-Glycine repeat with 17-23 pairs of alternating Thr-Gly residues (Citri *et al.*, 1987; Jackson *et al.*, 1986; Reddy *et al.*, 1986; Shin *et al.*, 1985) and is polymorphic in different wild-type strains (Yu *et al.*, 1987a); *in vitro*-effected deletion of the Thr-Gly repeat in transformation experiments does not appear to affect circadian rhythms, but causes shorter than normal song rhythm periods (Yu *et al.*, 1987a). 4.5 kb RNA has been found in the brain and ventral ganglia of developing *per*⁺ individuals (James *et al.*, 1986; Liu *et al.*, 1988; Lorenz, Hall, and Rosbash; Saez and Young, 1988) as well as in adult heads (Bargiello *et al.*, 1987; James *et al.*, 1986; Liu *et al.*, 1988; Saez and Young, 1988); it can also be detected in the salivary glands of *per*⁺ embryos and larvae (Bargiello *et al.*, 1987), but is not found in individuals that lack the *per* locus. The *per* gene product has been demonstrated immunologically in *per*⁺ embryos in

the midline of the nervous system, in salivary glands, in *per*⁺ pupae in the optic lobes of the brain and thoracic gland/corpora allata, and, in *per*⁺ adults, in the brain, eyes, gut, male and female reproductive tissues, Malpighian tubules, and most appendages (Bargiello *et al.*, 1987; Liu *et al.*, 1988; Saez and Young, 1988; Siwicki, Eastman, Petersen, Rosbash, and Hall, 1988, Neuron 1: 141-50). Studies involving anti-*per* antibodies show that *per*⁰¹ embryos, larvae, pupae, and adults are "null" at the protein level [in that peptides used to generate the antibodies were downstream of the relevant stop codon (Bargiello *et al.*, 1987; Saez and Young, 1988; Siwicki *et al.*, 1988)]; *per*⁰⁴ adults are antigenically null, whereas *per*^{L1} adults stain poorly and *per*^s adults stain normally (Siwicki *et al.*, 1988). Expression of *per* in adults (embryonic expression bypassed) has been studied by temporal manipulation experiments in which a *Hsp70* promoter has been fused to the *per* gene and the fusion gene turned on in adults only [Ewer, Rosbash, and Hall, 1988, Nature (London) 333: 82-84]. Inducing the expression of *per* after development is completed is necessary for the manifestation of the phenotype; earlier induction of the gene is not necessary or helpful. Circadian rhythmicity can be restored to some of the *per*⁻ and *per*⁰ mutants by *P*-element-mediated transformation with DNA fragments homologous to all or part of the 4.5 kb RNA species (Bargiello *et al.*, 1984; Baylies *et al.*, 1987; Hamblen *et al.*, 1986; Young *et al.*, 1985; Zehring *et al.*, 1984). "Restored" periodicities tend to be longer than normal (Baylies *et al.*, 1987), except when *per*⁰ flies are transformed with a particular 13.2 kb DNA fragment carrying all the coding information flanked by 3.7 kb of 5' and 2.0 kb of 3' sequences (Citri *et al.*, 1987). In the latter case, the periodicity and *per*⁺ penetrance of the transformed flies resembles that of normal wild-type individuals. In the transformation experiments of Baylies *et al.*, 1987, period length was shown to be correlated with abundance of *per* RNA. Transformed flies also show a restoration of cell-to-cell junctional communication. Levels of gap junctional communication are also correlated with abundance of *per* RNA (Bargiello *et al.*, 1987).

The 0.9 kb transcript, located adjacent to the 4.5 kb transcript, does not seem to be involved in the rescue of the clock functions in *per*⁻ and *per*⁰ mutants (Bargiello and Young, 1984; Bargiello *et al.*, 1984; Hamblen *et al.*, 1986). In *per*⁺ flies, however, this transcript shows oscillations in its abundance, being higher at midday than at midnight; in *per*⁰ mutants these oscillations do not occur (Reddy *et al.*, 1984).

Nothing is known at present about the 2.7 kb and the 1.7 kb transcripts. DNA sequences from *per* mutants have been compared and single base-pair changes found in *per*^L (T→A; Val²⁴⁵→Asp), *per*^s (G→A; Ser⁵⁸⁹→Asn), and *per*⁰ (C→T; Gln⁴⁶⁴→Amber) (Baylies *et al.*, 1987; Yu *et al.*, 1987b). In the *per* mutation associated with *T(1;4)JC43* flies, the 4.5 kb transcript has been replaced by a 11.5 kb transcript (Bargiello and Young, 1984; Jackson *et al.*, 1986; Reddy *et al.*, 1984).

other information: *per*⁰¹ is complemented by *l(1)3A* and *l(1)3B* mutants nearby (Young and Judd, 1978; Smith and Konopka, 1981). The mutant *per*⁰¹ of *D. melanogaster* can be rescued (i.e. made to show rhythmic behavior) by transformation with a hybrid gene carrying the coding region of the *D. pseudoobscura per*

gene (Peterson, Hall, and Rosbash, 1988, EMBO J. 7: 3939-47).

***pers*: persimmon**

location: 3- (left arm).

origin: X ray induced.

discoverer: Demerec, 3712.

references: 1940, DIS 14: 40.

phenotype: Eye color dull orange. Larval Malpighian tubes colorless (Brehme, 1942, Genetics 27: 133). Viability and fertility good. RK2A.

cytology: Associated with *In(3L)pers = In(3L)63C2-5;73B2-5*.

***petit*:** see *pe*

***Pfd*: Pufdi**

location: 2-70.8.

discoverer: Brierley, 1935.

references: Shull, 1937, DIS 8: 10.

1938, Proc. Michigan Acad. Sci. 23: 647-49.

Baker, 1950, Am. Naturalist 84: 51-70.

phenotype: Wings spread; fluid often accumulates between membranes. Degree of wing divergence inversely correlated with temperature; wings more divergent in male. In transfers from 19° to 31°, temperature-effective period begins 6-8 hr before eclosion in male and 4-6 hr before eclosion in female and ends with eclosion. In transfers from 31° to 19°, the temperature-sensitive period begins 8-10 hr before eclosion and ends 2-4 hr before eclosion (P. H. Baker, 1950). Homozygous lethal. RK2.

***Pfk*: Phosphofructokinase**

location: 2-85.2.

references: Laurie-Ahlberg, Wilton, Curtsinger, and Emigh, 1982, Genetics 102: 191-206.

Munneke and Collier, 1985, Biochem. Genet. 23: 847-57.

genetics: Produces the glycolytic enzyme phosphofructokinase [PFK (E.C. 2.7.1.11)]. Only one electrophoretic form has been found in larvae and adults.

cytology: Located at 55E.

****pg*: prong**

location: 2-40.

discoverer: Mohr, 19e.

references: 1923, Z. Indukt. Abstamm. Vererbungsl. 32: 218.

phenotype: Extra crossveins distal to anterior crossvein; usually incomplete. Overlaps wild type in at least 10% of flies. RK3.

***pg*:** see *pig*

***Pgd*: Phosphogluconate dehydrogenase**

location: 1-0.6 [between *br* and *pn* (Gvozdev *et al.*, 1970)].

discoverer: Young.

synonym: Lethal allele: *l(1)2Dc*.

references: Kazanian, Young, and Childs, 1965, Science 150: 1601-02.

Young, 1966, J. Hered. 57: 58-60.

Secof, Kaplan, and Futch, 1969, Proc. Nat. Acad. Sci. USA 62: 528-35.

Gvozdev, Birstein, and Faizullin, 1970, DIS 45: 163.

Lucchesi, Hughes, and Geer, 1979, Curr. Top. Cell. Reg. 15: 143-54.

Gutierrez, Christensen, Manning, and Lucchesi, 1989, Dev. Genet. (Amsterdam) 10: 155-61.

phenotype: Structural gene for 6-phosphogluconate dehydrogenase [6PGD (E.C. 1.1.1.44)], the last enzyme in the oxidative part of the pentose phosphate shunt. The electrophoretic variants Pgd^A and Pgd^B have been described in *Drosophila* (Kazanian *et al.*, 1965; Young, 1966), Pgd^A migrating faster in starch gel than Pgd^B . Pgd^A/Pgd^B heterozygous females produce a hybrid band of intermediate mobility. The enzyme 6PGD is a dimer. Its molecular weight was reported by Kazanian [1966, Nature (London) 212: 197-98] to be about 79,000; Williamson, Krochko, and Geer (1980, Biochem. Genet. 18: 87-101), using 6PGD (purified) isolated from Pgd^A homozygotes, found its molecular weight to be 105,000, and that it contains a mixture of equal amounts of subunits having molecular weights of 55,000 and 53,000. Enzyme activity is highest in early third instar larvae and lowest in late third instar larvae and 3-day old pupae, with intermediate levels in 4-day old pupae, newly eclosed adults, and 5-day old adults (Williamson *et al.*, 1980). The enzyme is found mainly in the fat body (Cochrane and Lucchesi, 1980, Genetics 94: s20).

Males with one dose of Pgd^+ and females with two doses have about the same amount of 6PGD, *i.e.* show dosage compensation for enzyme activity (Seecof *et al.*, 1969; Gerasimova and Ananiev, 1972, DIS 48: 93; Bowman and Simmons, 1973, Biochem. Genet. 10: 319-31; Faizullin and Gvozdev, 1973, Mol. Gen. Genet. 126: 233-45). Females heterozygous for a Pgd deficiency show a corresponding reduction in enzyme activity, while males and females with an extra dose of Pgd^+ show increased enzyme activity.

A number of lethal and semilethal Pgd mutants have been induced in Pgd^A or Pgd^B (Bewley and Lucchesi, 1975, Genetics 79: 451-66; Gvozdev, Gostimsky, Gerasimova, Dubrovskaya, and Braslavskaya, 1975, Mol. Gen. Genet. 141: 269-75; Hughes and Lucchesi, 1977, Science 196: 1114-15; Gvozdev, Gerasimova, Rostovsky, Kogan, and Braslavskaya, 1978, DIS 53: 143; Lucchesi *et al.*, 1979). The lethals (Pgd^n) show no enzyme activity unless covered by a Pgd^+ duplication such as w^+Y ; the semilethals (Pgd^{lo}) show low enzyme activity and are completely lethal when heterozygous with Pgd lethals. Flies carrying null alleles for both 6PGD, the first enzyme in the pentose phosphate shunt, and 6PGD, the last enzyme, are viable, presumably because the toxic 6-phosphogluconate is not produced (Lucchesi *et al.*, 1977). Pgd^n/Y males can be rescued by dietary supplements of fructose and linoleate that minimize 6-phosphogluconate production (Hughes and Lucchesi, 1978, Biochem. Genet. 16: 469-75).

alleles: The following table includes electrophoretic (wild-type), semilethal and lethal alleles of Pgd . [Synonyms for Pgd^+ alleles (Pgd^A , Pgd^B) use terminology for allozyme variants from DIS 53: 117].

allele	origin	discoverer	synonym	ref ^α	genetics
Pgd^A	spont	Young	6- Pgd^4	6,12	viable, fertile; fast variant
Pgd^B	spont	Young	6- Pgd^8	6,12	viable, fertile; slow variant
Pgd^I		Lucchesi	6- Pgd^6		viable, fertile; intermediate variant
$Pgd^{lo1} \beta$		Young	Pgd^-	1, 2, 10	semi-lethal,

allele	origin	discoverer	synonym	ref ^α	genetics
Pgd^{lo2}	EMS	Lucchesi		10	female sterile, low 6PGD activity
Pgd^{lo3}	EMS	Lucchesi		10	semi-lethal, female sterile, low 6PGD activity
Pgd^{lo4}	EMS	Lucchesi		10	semi-lethal, female sterile, low 6PGD activity
$Pgd^{lo13} \gamma$	EMS	Gvozdev	Pgd^{13}	3-5	low 6PGD activity
$Pgd^{lo50} \gamma$	EMS	Gvozdev	Pgd^{50}	3-5	semi-lethal, low 6PGD activity
$Pgd^{lo109} \gamma$	EMS	Gvozdev	Pgd^{109}	3-5	semi-lethal, low 6PGD activity
$Pgd^{n1} \beta \gamma$	EMS	Bewley, Lucchesi	$l(1)Pgd-A^{n1}$	1, 10	lethal; Pgd^-
Pgd^{n2}	EMS	Lucchesi		10	lethal; Pgd^-
Pgd^{n3}	EMS	Lucchesi		10	lethal; Pgd^-
Pgd^{n4}	EMS	Lucchesi		10	lethal; Pgd^-
Pgd^{n5}	EMS	Lucchesi		10	lethal; Pgd^-
Pgd^{n6}	EMS	Lucchesi		10	lethal; Pgd^-
Pgd^{n7}	EMS	Lucchesi		10	lethal; Pgd^-
Pgd^{n8}	EMS	Lucchesi		10	lethal; Pgd^-
Pgd^{n9}	X ray	Lefevre	$l(1)JE58$	8	lethal; Pgd^-
Pgd^{n10}	EMS	Lefevre	$l(1)DF958$	9,11	lethal; Pgd^-
Pgd^{n11}	EMS	Lefevre	$l(1)VA55$	9,11	lethal; Pgd^-
Pgd^{n12}	EMS	Lefevre	$l(1)VE618$	9,11	lethal; Pgd^-
$Pgd^{n35} \gamma$	EMS	Gvozdev	Pgd^{35}	3-5	lethal; Pgd^-
$Pgd^{n39} \gamma$	EMS	Gvozdev	Pgd^{39}	3-5	lethal; Pgd^-
$Pgd^{n45} \gamma$	EMS	Gvozdev	Pgd^{45}	3-5	lethal; Pgd^-
$Pgd^{n71} \gamma$	EMS	Gvozdev	Pgd^{71}	3-5	lethal; Pgd^-
$Pgd^{n93} \delta$	EMS	Gvozdev	Pgd^{93}	3-5	lethal; Pgd^-
$Pgd^{n94} \delta$	EMS	Gvozdev	Pgd^{94}	3-5	lethal; Pgd^-
$Pgd^{n100} \delta$	EMS	Gvozdev	Pgd^{100}	3-5	lethal; Pgd^-
$Pgd^{n111} \gamma$	γ ray	Gvozdev	Pgd^{111}	3-5	lethal; Pgd^-
$Pgd^{nHM18} \epsilon$	HMS		$l(1)HM18$	7	lethal; Pgd^-

^α 1 = Bewley and Lucchesi, 1975, Genetics 79: 451-66; 2 = Geer, Lindel, and Lindel, 1979, Biochem. Genet. 17: 881-95; 3 = Gvozdev, Gerasimova, Kogan, and Rostovsky, 1977, Mol. Gen. Genet. 153: 191-98; 4 = Gvozdev, Gerasimova, Rostovsky, Kogan, and Braslavskaya, 1978, DIS 53: 143; 5 = Gvozdev, Gostimsky, Gerasimova, Dubrovskaya, and Braslavskaya, 1975, Mol. Gen. Genet. 141: 269-75; 6 = Kazanian, Young, and Childs, 1965, Science 150: 1601-02; 7 = Kramers, Schalet, Paradi, and Huizer-Hooteyling, 1983, Mutat. Res. 107: 187-201; 8 = Lefevre, 1981, Genetics 99: 461-80; 9 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 10 = Lucchesi, Hughes, and Geer, 1979, Curr. Top. Cell. Reg. 15: 143-54; 11 = Perrimon, Engstrom, and Mahowald, 1985, Genetics 111: 23-41; 12 = Young, 1966, J. Hered. 57: 58-60.

^β Pgd^{lo1}/Pgd^{n1} females are lethal; Pgd^{n1}/Y males are lethal, except for a few escapers.

^γ Induced in Pgd^A .

^δ Induced in Pgd^B .

^ε HMS = hycanthon methanesulfonate.

cytology: Located in 2D3 or 2D4 (Gerasimova and Ananiev, 1972, DIS 48: 93; Gvozdev, Gostimsky, Gerasimova, Dubrovskaya, and Braskavskaya, 1975, Mol. Gen. Genet. 141: 269-75; Slobodyanyuk and Serov, 1983, Mol. Gen. Genet. 191: 372-77) based on the following: (1) Pgd is uncovered by $Df(1)Pgd-kz = Df(1)2D3-4;2F5$ but not by $Df(1)2D6;3C2$ (Lefevre; Seecof *et al.*, 1969) and is covered by w^+Y (2D1;3D4 inserted in Y); (2) It maps to the left of pn (located by Lefevre in 2D5-6). Whereas Pgd^A/Pgd^B heterozygotes show three isozyme bands on starch gels, deficiency heterozygotes show only one band and their 6PGD level is correspondingly reduced (Gerasimova and Ananiev, 1972; Gvozdev *et al.*, 1975).

molecular biology: Using a cDNA clone of the rat 6PGD as a probe, Christensen and Lucchesi (1984, *Genetics* 107: s20; Gutierrez *et al.*, 1989) isolated a *Drosophila* clone (14.7 kb fragment) hybridizing to 2D6, the approximate cytological location of *Pgd*. Another clone hybridizing to 2C1-E3 was obtained by Brock. The lethal *Pgd*⁻ phenotype was rescued by germline transformation (Gutierrez *et al.*, 1989). A single *Pgd*⁺ gene transduced to an autosomal site showed higher enzyme activity in males than in females, as would be expected if sequences responsible for dosage compensation had been transduced into the *Pgd*⁻ host.

Pgi: Phosphoglucose isomerase

location: 2-58.6 (Voelker *et al.*, 1980).

references: Lee, 1979, *J. Biol. Chem.* 254: 6375.

Voelker, Langley, Leigh-Brown, and Ohnishi, 1978, *DIS* 53: 200.

Voelker, Langley, Leigh-Brown, Ohnishi, Dickson, Montgomery, and Smith, 1980, *Proc. Nat. Acad. Sci. USA* 77: 1091-95.

Laurie-Ahlberg, Williamson, Cochrane, Wilton, and Chasalow, 1981, *Genetics* 99: 127-50.

Langley, Voelker, Leigh-Brown, Ohnishi, Dickson, and Montgomery, 1981, *Genetics* 99: 151-56.

Laurie-Ahlberg, Wilton, Curtsinger, and Emigh, 1982, *Genetics* 102: 191-206.

Burkhart, Dickson, Montgomery, Langley, and Voelker, 1984, *Genetics* 107: 295-306.

phenotype: Structural gene for the glycolytic enzyme phosphoglucose isomerase [PGI (E.C. 5.3.1.9)]. There are two electrophoretic variants (fast and slow); also a deficiency for the locus which is CRM negative and probably homozygous lethal (Lee, 1979).

alleles: Two *Pgi*⁺ alleles [*Pgi*⁴ (fast) and *Pgi*² (slow)] and one *Pgi*⁻ allele (*Pgi*^{n^{C80}}) have been reported. Out of a total of 716 alleles collected in North Carolina, one null allele was recovered (Langley *et al.*, 1981).

Pgk: Phosphoglycerate kinase

location: 2-5.9 (between *al* and *dp*) (Voelker *et al.*, 1979).

references: Chew and Cooper, 1973, *Biochem. Genet.* 8: 267-70.

Voelker, Ohnishi, and Langley, 1979, *Biochem. Genet.* 17: 769-83.

Voelker, Langley, Leigh-Brown, Ohnishi, Dickson, Montgomery, and Smith, 1980, *Proc. Nat. Acad. Sci. USA* 77: 1091-95.

Langley, Voelker, Leigh-Brown, Ohnishi, Dickson, and Montgomery, 1981, *Genetics* 99: 151-56.

Laurie-Ahlberg, Wilton, Curtsinger, and Emigh, 1982, *Genetics* 102: 191-206.

phenotype: Structural gene for 3-phosphoglycerate kinase [3-PGK (E.C. 2.7.2.3)]. Three electrophoretic variants (fast, intermediate, slow) isolated by Chew and Cooper (1973), the heterozygotes showing no hybrid band; two electrophoretic variants isolated by Voelker *et al.* (1979).

alleles: Three *Pgk*⁺ alleles, *Pgk*³ (fast), *Pgk*² (intermediate), *Pgk*¹ (slow) of Chew and Cooper (1973). No null alleles reported out of a total of 702 alleles collected in North Carolina (Langley *et al.*, 1981).

cytology: Located between 22D and 23C since included in the 2^D segregant of *T(Y;2)G146 = T(Y;2)h23;23B-C* but not of *T(Y;2)R136 = T(Y;2)h3;h7;22D*.

Pgm: Phosphoglucomutase

location: 3-43.4 (between *th* and *st*; Hjorth, 1970) or 3-43.6 (between *G1* and *st*; Trippa *et al.*, 1970, 1971).

references: Hjorth, 1970a, *DIS* 45: 39.

1970b, *Hereditas* 64: 146-48.

Trippa, Santolamazza and Scozzari, 1970, *Biochem. Genet.* 4: 665-67.

Trippa, 1971, *DIS* 46: 42.

Trippa, Scozzari, and Santolamazza, 1971, *DIS* 46: 44.

Trippa, 1972, *DIS* 49: 35.

Trippa, Danieli, Costa, and Scozzari, 1977, *DIS* 52: 74.

Voelker, Ohnishi, and Langley, 1979, *Biochem. Genet.* 17: 769-83.

phenotype: Structural gene for the glycolytic enzyme phosphoglucomutase [PGM (E.C. 2.7.5.1)], a monomeric protein of 56,000 daltons. Two frequently-occurring electrophoretic variants were isolated and studied by Hjorth (1970a,b) and Trippa *et al.* (1970, 1971). Later five low-frequency variants were found by Trippa's group (see references in table of alleles). One null allele found in a total of 431 alleles collected in Great Britain (Langley, Voelker, Leigh-Brown, Ohnishi, Dickson, and Montgomery, 1981, *Genetics* 99: 151-56).

alleles: The seven *Pgm*⁺ alleles identified by Trippa's group are listed in the following table. The most common allele has been designated *Pgm*⁴ in accordance with the convention of Voelker *et al.* (1979). The slower-migrating alleles are given superscript numbers below four while the faster-migrating alleles have numbers above four.

allele	synonym	ref ^α	comments
<i>Pgm</i> ^{1β}	<i>Pgm E</i> ; <i>Pgm</i> ^{0.55}	2, 3, 5	slowest migration on gel
<i>Pgm</i> ^{2β}	<i>Pgm B</i> ; <i>Pgm</i> ^{0.70} ; <i>Pgm</i> ²	1-6	frequency 0.8 - 20.7%
<i>Pgm</i> ³	<i>Pgm F</i> ; <i>Pgm</i> ^{0.85}	2, 3, 5	
<i>Pgm</i> ^{4γ}	<i>Pgm A</i> ; <i>Pgm</i> ^{1.00} ; <i>Pgm</i> ¹	1-6	frequency 79.3 - 99.2%
<i>Pgm</i> ⁵	<i>Pgm G</i> ; <i>Pgm</i> ^{1.10}	4, 5	
<i>Pgm</i> ^{6β}	<i>Pgm C</i> ; <i>Pgm</i> ^{1.20}	2, 3, 5	
<i>Pgm</i> ⁷	<i>Pgm D</i> ; <i>Pgm</i> ^{1.50}	2, 3, 5	fastest migration on gel

^α 1 = Hjorth, 1970, *DIS* 45: 39; 2 = Trippa, 1972, *DIS* 49: 35; 3 = Trippa, Barberio, Loverre, and Santolamazza, 1972, *DIS* 49: 42; 4 = Trippa, Danieli, Costa, and Scozzari, 1977, *DIS* 52: 2; 5 = Trippa, Danieli, Costa, and Scozzari, 1977, *DIS* 52: 74; 6 = Trippa, Santolamazza, and Scozzari, 1970, *Biochem. Genet.* 4: 665-67.

^β Enzyme heat-sensitive [Loverre and Carmody, 1985, *Biochem. Genet.* 23: 29-36; Trippa, Loverre, and Catamo, 1976, *Nature (London)* 260: 42-44; Trippa, Catamo, Lombardozzi and Cicchetti, 1978, *Biochem. Genet.* 16: 299-305].

^γ Enzyme may be heat-sensitive or heat-resistant (Trippa *et al.*, 1976, 1978).

cytology: Located in 72D1-5 since *Pgm* included in *Df(3L)th117 = Df(3L)72A1;72D5* but not in *Df(3L)th113 = Df(3L)72A2;72D1-2* (Voelker *et al.*, 1978; Burkhart, Dickson, Montgomery, Langley, and Voelker, 1984, *Genetics* 107: 295-306.)

ph: polyhomeotic (P. Santamaria)

location: 1-0.5 (based on 28 y-z recombinants).

synonym: Possibly **rsc*: *reduplicated sex comb* (CP 627; Dura *et al.*, 1985).

references: Brock and Freeman, 1984, *Genetics* 107: s14. Dura, Brock, and Santamaria, 1985, *Mol. Gen. Genet.* 198: 213-20.

Perrimon, Engstrom, and Mahowald, 1985, *Genetics* 111: 23-41.

Dura, Randsholt, Deatrick, Erk, Santamaría, Freeman, Freeman, Weddell, and Brock, 1987, *Cell* 51: 829-39.
 Dura, Deatrick, Randsholt, Brock, and Santamaría, 1988, *Roux's Arch. Dev. Biol.* 197: 239-46.
 Dura and Ingham, 1988, *Development* 103: 733-41.
 Smouse, Goodman, Mahowald, and Perrimon, 1988, *Genes Dev.* 2: 830-42.
 Santamaría, Deatrick, and Randsholt, 1989, *Roux's Arch. Dev. Biol.* 198: 65-77.

phenotype: A member of the Polycomb group of genes; seems to be the only one that is strongly required both maternally and zygotically for normal embryonic development. Two mutagenic events are necessary to produce null mutations, suggesting that the locus is complex, and in fact molecular determinations indicate that the locus comprises a direct repeat. Single-event mutations are viable as males and homozygous females; such mutations produce transformations similar to those of known dominant gain-of-function mutants in the *ANTC* and *BXC*, *i.e.*, transformation of wings to halteres, second and third legs to first legs, and anterior abdominal segments to more posterior segments (mutants may also show loss of the humerus). Two-event lethal mutations die in mid embryogenesis and completely lack ventral and abdominal epidermal derivatives; they show transformations of most of the segments toward the eighth abdominal segment (Dura *et al.*, 1988). There is an alteration in the pattern of axon pathways in the CNS. The axons fail to form commissures or connectives but instead form large bundles in the middle of each hemiganglion (Smouse *et al.*, 1988). Trans heterozygotes between viable and lethal alleles or between viable alleles and a deficiency for *ph* die in late embryogenesis and exhibit posteriorly directed transformations; *i.e.* viable alleles are haplo insufficient. Dura *et al.* (1987) postulate that mutations can occur in either or both of the repeated sequences such that $-/+ = +/- = -/+ =$ viable mutant constitutions ($-/+$ not stated to be mutant); $-/+ = +/- =$ late embryonic lethals; and $-/- =$ mid-embryonic-stage lethals.

The ph^+ product is required autonomously in imaginal cells. A total lack of ph^+ function prevents viability of the cuticular derivatives of these cells, but amorphic *ph* clones induced in late third instar survive. *ph* has a strong maternal effect on segmental identity and epidermal development that cannot be rescued by a single paternally supplied dose of ph^+ in the zygote (Dura *et al.*, 1988).

The expression of *ph* is inversely related to dosage of the *ANTC* and the *BXC*; the gene interacts with *Pc*, *Pcl*, and *esc* (Dura *et al.*, 1985). At the shortened germ band stage (but not at the blastoderm stage), *ph* seems to be involved in the regulation, not only of the homeotic genes *Scr* and *Ubx*, but also in the regulation of the segmentation genes *ftz*, *eve*, and *en* (Dura and Ingham, 1988).

alleles: Almost 50 mutant alleles of polyhomeotic have been isolated, 41 of them being viable as adults and the rest (ph^{500} - ph^{505}) lethal.

allele	origin	discoverer	ref ^α	comments
<i>ph</i> ¹	EMS	Gans and Komitopoulou	1, 3	cytology normal
<i>ph</i> ²	HD		1, 3	cytology normal
<i>ph</i> ^{3β}	HD	Karpen	1, 3	<i>Tp(1;1)2E-F;5B-C;19F-20</i> ^δ ; dominant enhancer of <i>Pc</i> only

allele	origin	discoverer	ref ^α	comments
<i>ph</i> ⁴	HD	Karpen	1, 3	cytology normal
<i>ph</i> ⁵	DEB		3	
<i>ph</i> ⁶	EMS		1, 3	
<i>ph</i> ^{7γ}	HD	Karpen	1, 3	dominant enhancer of <i>Pc</i> only
<i>ph</i> ^{8γ}	EMS		3	
<i>ph</i> ^{9γ}	HD	Karpen	1, 3	dominant enhancer of <i>Pc</i> only
<i>ph</i> ¹⁰	EMS		3	
<i>ph</i> ^{10-1β}	X ray		3	cytology normal
<i>ph</i> ¹¹	X ray		3	cytology normal
<i>ph</i> ¹⁰¹	EMS		3	
<i>ph</i> ¹⁰²	EMS		3	
<i>ph</i> ²⁰³	EMS		3	
<i>ph</i> ²⁰⁴	EMS		3	
<i>ph</i> ²⁰⁵	EMS		3	
<i>ph</i> ²⁰⁷	EMS		3	
<i>ph</i> ²⁰⁸	EMS		3	
<i>ph</i> ²⁰⁹	EMS		2, 3	extra sex combs in females with <i>tra-2</i> OTF
<i>ph</i> ²¹²	EMS		3	
<i>ph</i> ²¹⁴	EMS		3	
<i>ph</i> ²¹⁶	EMS		3	
<i>ph</i> ²¹⁷	EMS		3	
<i>ph</i> ²¹⁸	EMS		3	
<i>ph</i> ²²⁰	EMS		3	
<i>ph</i> ²²²	EMS		3	
<i>ph</i> ³⁰¹	EMS		3	
<i>ph</i> ³⁰³	EMS		3	
<i>ph</i> ³⁰⁵	EMS		3	
<i>ph</i> ⁴⁰⁰	X ray		3	cytology normal
<i>ph</i> ⁴⁰¹	X ray		3	cytology normal
<i>ph</i> ⁴⁰³	X ray		3	cytology normal
<i>ph</i> ⁴⁰⁴	X ray		3	cytology normal
<i>ph</i> ^{409γ}	X ray		3	cytology normal
<i>ph</i> ^{410δ}	X ray		3	<i>In(1)2D;13-14</i>
<i>ph</i> ^{500ε}	EMS		3	lethal amorph
<i>ph</i> ^{501ε}	EMS		2, 3	lethal hypomorph
<i>ph</i> ^{502ε}	EMS		3	lethal amorph
<i>ph</i> ^{503ε}	EMS		3	lethal amorph
<i>ph</i> ^{504ε}	EMS		3	lethal amorph
<i>ph</i> ^{505ε}	EMS		2, 3	lethal amorph
<i>ph</i> ^{brζ}	EMS	Rutledge	2, 3	lethal as pharate adult
<i>ph</i> ^k	EMS	Perrimon	4	
<i>ph</i> ^{rsc}	EMS	B.S. Baker	3	
<i>ph</i> ^{Ting}	EMS	C.T. Wu	3	
<i>ph</i> ^{VA174}	EMS	Perrimon	3	cytology normal

^α 1 = Dura, Brock, and Santamaría, 1985, *Mol. Gen. Genet.* 198: 213-20; 2 = Dura, Deatrick, Randsholt, Brock, and Santamaría, 1988, *Roux's Arch. Dev. Biol.* 197: 239-46; 3 = Dura, Randsholt, Deatrick, Erk, Santamaría, Freeman, Freeman, Weddell, and Brock, 1987, *Cell* 51: 829-39; 4 = Perrimon, Engstrom, and Mahowald, 1985, *Genetics* 111: 23-41.

^β Viable over deficiencies for *ph*. (Dura *et al.*, 1987).

^γ Chromosome mutant for *ph* and *wapl* but not for the intervening *Pgd*; probably carries an inversion with breaks in *ph* and *wapl* (Dura *et al.*, 1987); *ph*³ separable from the associated *Tp(1;1)2E-F;5B-C;19F-20* (Dura *et al.*, 1985).

^δ Carries *In(1)2D;13-14*.

^ε Lethal derivative of *ph*²⁰⁹.

^ζ Extra row of bristles on wing margin; structures like vaginal plates in sixth and seventh sternites of female abdomen (Dura *et al.*, 1988).

cytology: Placed in 2D3-4; uncovered by *Df(1)JA52 = Df(1)2C10-D1;2D3-4* and *Df(1)pn38 = Df(1)2D3-4;2E3* (Perrimon *et al.*, 1985; Dura *et al.*, 1987); *Df(1)Pgd-kz = Df(1)2D3-4;2F5* is broken within *ph* sequences; not covered by *Dp(1;1)dorY18T = Dp(1;1)1A;2D1-2*.

molecular biology: The *ph* gene was localized within a 240 kb chromosome walk, using deficiencies and mutations that result from chromosome rearrangements. Hybridization analyses indicate that *ph* contains a large, highly-conserved direct but not quite contiguous repeat, each element of which contains an opa sequence, and

each is transcribed (Freeman, Randsholt, Deatrick, and Brock). One repeat, distal to the distal breakpoint of *Df(1)Pgd-kz = Df(1)2D3-4;2F5*, is unbroken and contains molecular deletions in mutant alleles, *ph*², *ph*⁴, *ph*^{10a}, and *ph*⁴⁰¹. The proximal element is interrupted by an unrelated sequence and is deleted by *Df(1)Pgd-kz*; mutant alleles *ph*³ and *ph*⁷ contain inserted sequences in the proximal repeat element; *ph*⁴⁰⁹, and *ph*⁴¹⁰ have breakpoints in the element. A restriction site polymorphism is found in the 1.95 kb fragment separating the repeat components (Dura *et al.*, 1987). All of the *ph* lesions map within a 20 kb region between the distal breakpoint of *Df(1)pn38* and the proximal breakpoint of *Df(1)JA52*. DNA fragments from coordinates 122 to 149 on the molecular map of the 2C1-2 to 2E1-2 region (0 being the distal point of the walk) hybridize to two major embryonic poly(A)⁺ RNA's of 6.4 and 6.1 kb (Dura *et al.*, 1987).

ph: see *ms(3)K81*

phantom: see *phm*

phase-angle2: see *psi2*

phase-angle3: see *psi3*

Phb: Photophobe

location: 2R.

origin: Induced by ethyl methanesulfonate in a *sev*^{LY3} strain.

references: Ballinger and Benzer, 1988, Proc. Nat. Acad. Sci. USA 85: 3960-64.

phenotype: Dominant mutation that reverses the color-choice behavior of *sev*^{LY3} mutants - *i.e.* *sev*^{LY3}; *Phb*/+ flies, like wild-type flies, prefer UV over green light, while *sev*^{LY3};+/+ flies prefer green over UV. When tested in a T-maze for preference between darkness and green light (550 nm), both wild type and *sev*^{LY3} flies show positive phototaxis, while *sev*^{LY3}; *Phb*/+ and *sev*^{LY3}; *Phb/Phb* flies show negative phototaxis. Similarly, tests in a countercurrent apparatus for movement toward and movement away from white light indicate marked preference of wild-type flies for light and of *sev*^{LY3}; *Phb*/+ flies for darkness. Both the positive phototactic response of *sev*^{LY3} and the negative phototactic response of *sev*^{LY3}; *Phb*/+ flies increase with age, reaching a maximum at 4-6 days. The interaction between *Phb* and *sev* is allele specific, as is indicated in the following table:

genotype	phototaxis
<i>sev</i> ^{d2} ; <i>Phb</i> /+	+
<i>sev</i> ^{E1} ; <i>Phb</i> /+	+
<i>sev</i> ^{E4} ; <i>Phb</i> /+	+
<i>sev</i> ^{E5} ; <i>Phb</i> /+	+
<i>sev</i> ^{f31E} ; <i>Phb</i> /+	+
<i>sev</i> ^{LY3} ; <i>Phb</i> /+	-
<i>sev</i> ^{P3} ; <i>Phb</i> /+	-
<i>sev</i> ^{x3} ; <i>Phb</i> /+	+

Photoreceptor cell R7, missing in all of the *sev* alleles, was not restored in any mutant combination of *Phb* and *sev*.

Phenol oxidase: see **Phox**

phl: pole hole

location: 1-0.5.

synonym: *l(1)2Fe*; *l(1)ph*.

references: Konrad and Mahowald, 1983, Molecular Aspects of Early Development (Malacinski and Klein, eds.). Plenum Press, New York, pp. 167-88.

Perrimon, Engstrom, and Mahowald, 1984a, Dev. Biol. 105: 404-14.

1984b, Genetics 108: 559-72.

Perrimon and Mahowald, 1986, Symp. Soc. Dev. Biol. 44: 221-35.

Ambrosio, Mahowald, and Perrimon, 1989, Nature (London) 342: 288-91.

Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-52.

phenotype: The wild-type allele of *phl* seems to be involved in the setting up of positional values in embryos and also in the proliferation of diploid cells in imaginal disks (Ambrosio, Perrimon, and Mahowald, 1988). *phl* mutants are recessive early-pupal lethals, which display very small imaginal disks. Embryos derived from germline clones and lacking *phl*⁺ activity show the "torso" or "pole hole" phenotype; structures at the anterior and the posterior end (spiracles, anal tufts, and the entire eighth abdominal segment) fail to develop (Perrimon *et al.*, 1984; Ambrosio, Mahowald, and Perrimon, 1988). The fate map of the blastoderm is shifted posteriorly and fewer segments with more cells result. A partial rescue of these mutants has been obtained with *phl*⁺ sperm (Ambrosio, Engstrom, and Mahowald); all structures posterior to abdominal segment seven are missing (Perrimon and Mahowald, 1986).

alleles:

allele	origin	discoverer	synonym	ref ^α
<i>phl</i> ¹	X ray	Lefevre	<i>l(1)C110</i>	2, 3
<i>phl</i> ^{2β}	X ray	Lefevre	<i>l(1)GA79</i>	2, 3
<i>phl</i> ^{3γ}	X ray	Lefevre	<i>l(1)JC59</i>	2, 3
<i>phl</i> ⁴	EMS	Lefevre	<i>l(1)DA503</i>	2, 3
<i>phl</i> ⁵	EMS	Lefevre	<i>l(1)DC817</i>	2, 3
<i>phl</i> ⁶	EMS	Lefevre	<i>l(1)DF903</i>	2, 3
<i>phl</i> ⁷	EMS	Lefevre	<i>l(1)EA75</i>	2-5
<i>phl</i> ⁸	EMS	Lefevre	<i>l(1)VA88</i>	2, 3
<i>phl</i> ⁹	EMS	Lefevre	<i>l(1)VE733</i>	2, 3
<i>phl</i> ¹⁰	EMS	Lefevre	<i>l(1)VE791</i>	2, 3
<i>phl</i> ¹¹	spont	Schalet	<i>l(1)11-29</i>	4
<i>phl</i> ¹²	HMS δ		<i>l(1)HM7</i>	1

α 1 = Kramers, Schalet, Paradi and Huizer-Hoogteyting, 1983, Mutat. Res. 107: 187-201; 2 = Lefevre, 1981, Genetics 99: 461-80; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 4 = Schalet, 1986, Mutat. Res. 163: 115-44.

β Cytology: *T(1;3)2F-3A;82C*.

γ Cytology: *T(1;3)3A1;92F-93A*.

δ HMS = hycanthon methanesulfonate.

cytology: Located in 2F6 since included in *Df(1)JC19 = Df(1)2F6;3C5*, *Df(1)TEM75 = Df(1)2F5-3A1;3C2-4*, and *Df(1)X12 = Df(2F5-3C1;3B5-C1*, but not in *Df(1)62g18 = Df(1)3A1-2;3A4*.

molecular biology: Three transcripts (3.5, 4.8, and 5.3 kb long) have been reported (Ambrosio, Perrimon, and Mahowald). Gene shows homology to the serine-threonine kinase oncogene *v-raf*; the *Drosophila raf* gene has been shown by molecular analysis to be *phl* (Ambrosio *et al.*, 1989).

phm: phantom

location: 1-64 (average of five alleles).

origin: Induced by ethyl methanesulfonate.

references: Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307.

phenotype: Male lethal. Cuticle of lethal embryos not well differentiated and contracted posteriorly.

alleles: Five *phm* ethyl-methanesulfonate-induced alleles.

cytology: Located in 17A-18A since uncovered by *Df(1)N19 = Df(1)17A1;18A2*.

Phosphoenolpyruvate-

carboxykinase: see *Pepck*

Phosphofructokinase: see *Pfk***Phosphoglucomutase:** see *Pgm***Phosphogluconate dehydrogenase:** see *Pgd***Phosphoglucose isomerase:** see *Pgi***Phosphoglycerate kinase:** see *Pgk***Photophobe:** see *Phb***photorepair:** see *phr***Phox: Phenol oxidase**

location: 2-80.6.

references: Batterham and McKechnie, 1980, Genetica 54: 121-26.

Batterham, 1981, Genetics 97: s8.

Batterham and Chambers, 1981, DIS 56: 18-19.

phenotype: Structural gene for a phenol oxidase component other than A1, A2, or A3. [PHOX (E.C. 1.10.3.1)]. It is a tyrosinase, oxidizing mono- and o-diphenols. Three electrophoretic variants have been described by Batterham and McKechnie (1980), but no null alleles. The molecular weight of the native enzyme is 108,000 daltons. PHOX is believed to be a dimer with a subunit molecular weight of 54,000 (Batterham and Chambers, 1981).

alleles: *Phox^F* (fast), *Phox^I* (intermediate), and *Phox^S* (slow). *Phox^I* is the most common allele in Australasia, occurring with a minimum frequency of 0.85 (Batterham, 1981). No alleles affecting activity levels have been demonstrated, although the possible allelism between *Phox* and the mutant *Bc* [with the same genetic location as *Phox* (2-80.6)] has been suggested (Treat-Clemons and Doane, 1984, DIS 60: 17-42; Wright, 1987, Adv. Genet. 24: 127-222).

other information: *tyr1* and *lz³* show no PHOX activity (Wright, 1987).

phr: photorepair

location: 2-56.8 (between *pr* and *c*).

origin: Spontaneous in a standard laboratory stock.

references: Boyd and Harris, 1985, Genetics 110: s85. 1987, Genetics 116: 233-39.

phenotype: Deficient in photorepair of pyrimidine dimers; partially deficient in excision repair. Since *phr/phr* larvae show little or no photoreactivation after exposure to short wavelength UV light, the *phr⁺* function is thought to play a significant role in UV resistance.

pi: pied

location: 2-17.

origin: Spontaneous.

discoverer: Harnly, 38k31.

phenotype: Eyes like *S* but more extreme, smaller, and rougher; facets jumbled. Wings larger, flimsy, arched, and fringed. Male usually sterile, have abnormal genitalia. Viability erratic, varying from 20 to 80%. RK3.

pic: piccolo

location: 3-52.1 (distal to *ry* and *snk*).

synonym: *l(3)87D*.

references: Schalet, Kernaghan, and Chovnick, 1964, Genetics 50: 1261-68.

Hilliker, Clark, and Chovnick, 1980, Genetics 95: 95-110.

Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72.

Clark and Chovnick, 1986, Genetics 114: 819-40.

phenotype: Lethal or semilethal as homozygotes or heteroallelic heterozygotes. Survivors show the *pic* visible phenotype, *i.e.* short and thin or missing thoracic bristles, abnormal tergite morphology (as in *bb*), and, occasionally, wing defects.

alleles: One semilethal and 32 lethal alleles have been identified. Six of the lethals show some interallelic complementation for viability, but not for the visible phenotype.

allele	origin	discoverer	synonym	ref ^α
<i>pic^{1β}</i>	X ray	Schalet		1, 4, 5, 6
<i>pic^{2γ}</i>	X ray	Schalet	<i>pic²¹</i>	6
<i>pic³</i>	X ray	Schalet	<i>pic³¹</i>	6
<i>pic⁴</i>	EMS	Hilliker, Clark	<i>l(3)3-119</i>	5
<i>pic⁵</i>	EMS	Hilliker, Clark	<i>l(3)8-107</i>	5
<i>pic⁶</i>	EMS	Hilliker, Clark	<i>l(3)8-181</i>	5
<i>pic⁷</i>	EMS	Hilliker, Clark	<i>l(3)12-196</i>	5
<i>pic⁸</i>	EMS	Hilliker, Clark	<i>l(3)33-1</i>	5
<i>pic⁹</i>	EMS	Hilliker, Clark	<i>l(3)A3-3</i>	5
<i>pic¹⁰</i>	EMS	Hilliker, Clark	<i>l(3)A12-3</i>	5
<i>pic¹¹</i>	EMS	Hilliker, Clark	<i>l(3)A19-1</i>	5
<i>pic¹²</i>	EMS	Hilliker, Clark	<i>l(3)A19-2</i>	5
<i>pic¹³</i>	EMS	Hilliker, Clark	<i>l(3)A34-3</i>	5
<i>pic¹⁴</i>	EMS	Hilliker, Clark	<i>l(3)A42-1</i>	5
<i>pic¹⁵</i>	EMS	Hilliker, Clark	<i>l(3)A80</i>	5
<i>pic¹⁶</i>	EMS	Hilliker, Clark	<i>l(3)A111</i>	5
<i>pic¹⁷</i>	EMS	Hilliker, Clark	<i>l(3)A112</i>	5
<i>pic¹⁸</i>	EMS	Hilliker, Clark	<i>l(3)B2-4</i>	5
<i>pic¹⁹</i>	γ ray	Hilliker, Clark	<i>l(3)C-9-2</i>	5
<i>pic²⁰</i>	γ ray	Hilliker, Clark	<i>l(3)C-17-3</i>	5
<i>pic²¹</i>	γ ray	Hilliker, Clark	<i>l(3)C-18-1</i>	5
<i>pic²²</i>	EMS	Hilliker, Clark	<i>l(3)D64</i>	5
<i>pic²³</i>	EMS	Gelbart	<i>l(3)G23</i>	2, 3, 5
<i>pic²⁴</i>	γ ray	Gelbart	<i>l(3)G26</i>	5
<i>pic²⁵</i>	EMS	Hilliker, Clark	<i>l(3)H10</i>	5
<i>pic²⁶</i>	γ ray	Hilliker, Clark	<i>l(3)H19</i>	5
<i>pic²⁷</i>	γ ray	Hilliker, Clark	<i>l(3)H22</i>	5
<i>pic²⁸</i>	EMS	Hilliker, Clark	<i>l(3)H49</i>	5
<i>pic²⁹</i>	EMS	Hilliker, Clark	<i>l(3)H51</i>	5
<i>pic³⁰</i>	EMS	Hilliker, Clark	<i>l(3)H54</i>	5
<i>pic³¹</i>	EMS	Hilliker, Clark	<i>l(3)H59</i>	5
<i>pic³²</i>	EMS	Hilliker, Clark	<i>l(3)H72</i>	5
<i>pic³³</i>	EMS	Hilliker, Clark	<i>l(3)m10</i>	5

^α 1 = Clark and Chovnick, 1986, Genetics 114: 819-40; 2 = Gelbart and Chovnick, 1979, Genetics 92: 849-59; 3 = Gelbart, McCarron, and Chovnick, 1976, Genetics 84: 211-32; 4 = Hilliker, Clark, and Chovnick, 1980, Genetics 95: 95-110; 5 = Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72; 6 = Schalet, Kernaghan, and Chovnick, 1964, Genetics 50: 1261-68.

^β Semilethal.

^γ May show interallelic complementation for viability, but survivors are *pic* in phenotype (Hilliker *et al.*, 1980, 1981; Clark and Chovnick, 1986).

cytology: Located in 87D11-14 since included in *Df(3R)l26d = Df(3R)87D11-13;87E3-5*, *Df(3R)ry75 = Df(3R)87D1-2;87D14-E1*, and *Df(3R)ry614 = Df(3R)87D2-4;87D11-14*, but not in *Df(3R)ry36* (Hilliker *et al.*, 1981; Clark and Chovnick, 1986).

molecular biology: Clark and Chovnick (1986) place *pic* at -152 kb on the molecular map of Bender, Spierer, and Hogness (1983, *J. Mol. Biol.* 168: 17-33); 0 coordinate is the 87E1-2 breakpoint of *In(3R)Cbx^{rvl}*.

pied: see *pi*

*** *pig:*** *pigmy*

location: 1-29.

origin: X ray induced.

discoverer: Muller, 26l8.

synonym: *pg* (preoccupied).

references: 1935, DIS 3: 30.

phenotype: Fly small and melanotic. Viability about 25% wild type. RK3.

Pig1: *Pre-intermolt gene 1*

location: 1- (distal to *Sgs4*).

discoverer: Mathers and Meyerowitz.

references: Chen, Malone, Beckendorf, and Davis, 1987, *Nature* (London) 329: 721-26.

phenotype: Expressed in larval salivary glands, RNA reaching a peak in second larval instar.

cytology: Located in 3C11-12.

molecular biology: Transcript is 630 bp long and lies between coordinates -46.4 and -47, 840 bp distal to *Sgs4* (Rogers and Beckendorf). Encodes a poly(A)⁺ RNA of about 750 bp (an unique genomic sequence; *dnc* or *Sgs4* transcribed on the opposite strand).

Pigmentless: see *Ps*

pigmy: see *pig*

pil: see *plo*

*** *pil3:*** *pilosus on third chromosome*

location: 3- (near or identical with *tra*).

discoverer: Goldschmidt.

references: 1953, *J. Exp. Zool.* 122: 53-96 (fig.).

phenotype: Produces setae on sixth sternite of male or transformed female. Semidominant. Enhanced by *pil-X*. RK3.

Pilis: see *Fs(3)Sz20*

pillow: see *plo*

pilosus on third chromosome: see *pil3*

pilosus on X: see *pilX*

*** *pilX:*** *pilosus on X*

location: 1- (left of *w*).

discoverer: Goldschmidt.

references: 1953, *J. Exp. Zool.* 122: 53-96 (fig.).

phenotype: Produces setae of varying numbers and sizes on the sixth sternite of normal male and of X/X; *tra/tra* female. Effect enhanced by presence of *pil3* and also by Y chromosome of *tra* stock. RK3.

pim: *pimples*

locations: 2-30.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1983, DIS 59: 158-60.

1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

phenotype: Homozygous lethal in embryo. Cuticle poorly differentiated with necrotic patches. Head abnormal.

cytology: Located in 31B-32A (Nüsslein-Volhard *et al.*, 1984); uncovered by *Df(2L)J27 = Df(2L)31B-D;31F-32A*.

alleles: Only one allele was reported by Nüsslein-Volhard *et al.* (1984).

Pin: *Pin*

location: 2-107.3 (to the right of *sp*).

phenotype: Thoracic bristles, especially dorso-centrals and scutellars, shortened and thick at base, sharply tapered at tip. This bristle phenotype is stronger in homozygotes than heterozygotes in the homozygous-viable allele *Pin¹*.

alleles: Five *Pin* alleles have been described. Phenotypes of the homozygous lethal alleles follow the table.

allele	origin	discoverer	ref ^α
<i> Pin¹</i>	spont	Ives, 39a9	4
<i> Pin²</i>	spont	Grell, 57b	3
<i> Pin^B</i>	EMS	Bacher, 66	1
<i> Pin^{Tac}</i>	spont	Weiskettel, 57l	5
<i> Pin^{Yt}</i>	spont	Grell, 57e	2

^α 1 = Craymer, 1980, DIS 55: 197-200; 2 = Grell, 1957, DIS 31: 81; 3 = Grell, 1960, DIS 34: 50; 4 = Ives, 1940, DIS 13: 50; 5 = Kadel, 1958, DIS 32: 80.

cytology: Located between 60C5 and 60D2 since *Pin²* is lethal over *Df(2R)Px = Df(2R)60B8-10;60D1-2* or *Df(2R)Px² = Df(2R)60C5-6;60D9-10*.

Pin²

phenotype: Thoracic bristles of *Pin²/+* flies very short at normal room temperature, but wild type at 17°. Homozygotes almost lethal, the few survivors having virtually no thoracic bristles. *Pin²/Pin* mutants have smaller bristles than *Pin²/+* and lower viability. *Pin²/Pin^{Yt}* is lethal, as are *Df(2R)Px/Pin²*, *Df(2R)Px²/Pin²*, and *Df(2R)Px⁴/Pin²*. *Pin²/+* flies carrying *bw⁺Y* have longer bristles than *Pin²/+* but shorter than wild type.

Pin^B: *Pin-Bacher*

phenotype: Thoracic bristles of *Pin^B/+* flies are pale yellow, thin and twisted (like *Pin^{Yt}/+* flies but more extreme). *Pin^B/Pin¹* has only vestiges of chaetae on the thorax; also has soft, watery-appearing cuticle. Homozygotes lethal.

Pin^{Tac}: *Pin-Tack*

phenotype: Thoracic bristles of *Pin^{Tac}/+* flies very small at 22°, but almost wild type at 18°. Older females hold wings in abnormal position. Homozygous lethal.

Pin^{Yt}: *Pin-Yellow-tip*

phenotype: Thoracic bristles of *Pin^{Yt}/+* flies are pale yellow, thin, and slightly twisted distally; cuticle appears soft. Homozygous lethal and lethal over *Pin¹* and *Pin²*; survives in combination with *Df(2R)Px* and resembles *Pin^{Yt}/+*.

pink: see *p*

pink wing: see *pw*

pink-wing: see *lt^{pk}*

pink wing c: see *pwc*

pinkish: see *pkh*

pinkoid: see *lt^{pk}*

pip: *pipe*

location: 3-47.

origin: Induced by ethyl methanesulfonate.

references: Anderson and Nüsslein-Volhard, 1984, *Nature* (London) 311: 223-27.

Anderson, Bokla, and Nüsslein-Volhard, 1985, *Cell* 42: 791-98.

Carroll, Winslow, Trombly, and Scott, 1987, *Development* 99: 327-32.

phenotype: Maternal-effect lethal; homozygous females are sterile. As in the mutant *dl*, lethal embryos produced by *pip* females lack ventral and lateral elements; all cells differentiate like the dorsal-most cells of normal embryos (Anderson and Nüsslein-Volhard, 1984). Dorsalization is also observed in the pattern of *ftz* stripes in *pip* embryos (Carroll *et al.*, 1987). Embryos are not rescued by injection of wild-type cytoplasm; injection of cytoplasm from ventralized embryos of *Tl^{5/+}* females, however, partially restores the *pip⁺* embryonic pattern, resulting in differentiation of filzkörper in 25-40% of the treated *pip* embryos (Anderson *et al.*, 1985).

alleles: The alleles *pip¹* and *pip²* recovered as *pip²⁸⁶* and *pip⁶⁶⁴*.

pk: *prickle*

location: 2-55.3.

discoverer: Ives, 38k.

references: 1947, *DIS* 21: 68-69.

Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, *DIS* 56: 186-91.

Gubb and García-Bellido, 1982, *J. Embryol. Exp. Morphol.* 68: 37-57.

phenotype: Polarity pattern of wing, haltere, and notum altered in mutants. Chaetae of triple row on anterior wing slanted anteriorly instead of posteriorly. Trichomes near L2 vein arranged in counterclockwise whorl on right wing blade, in clockwise whorl on left blade; trichomes in anterior wing occasionally duplicated (Gubb and García-Bellido, 1982). On the notum, the posterior acrostichals are irregularly erect and whorled. Occasionally, extra dorsocentral and scutellar bristles appear at temperatures above 23°. Mutant flies slightly larger than wild type. Gubb and García-Bellido (1982) describe somatic clones of homozygous *pk* cells.

alleles: All alleles viable and *pk* over *Df(2R)pk78k*, indicating no lethal alleles at locus (Gubb and García-Bellido, 1982).

allele	origin	discoverer	ref ^α
<i>pk¹</i>	spont	Ives	2
<i>pk^{78a} β</i>	X ray	Gubb	1
<i>pk^{78b}</i>	X ray	Gubb	1
<i>pk^{78d} γ</i>	X ray	Gubb	1
<i>pk^{78f}</i>	X ray	Gubb	1
<i>pk^{78g}</i>	X ray	Gubb	1
<i>pk⁷⁸ⁱ</i>	X ray	Gubb	1
<i>pk^{78m} β</i>	X ray	Gubb	1

^α 1 = Gubb and García-Bellido, 1982, *J. Embryol. Exp. Morphol.* 68: 37-57; 2 = Ives, 1947, *DIS* 21: 68-69.
^β Homozygotes indistinguishable from *pk¹/pk¹*.
^γ Homozygotes show slight alteration in polarity pattern of posterior wing (below L5 vein); otherwise same as *pk¹/pk¹*.

cytology: Located between 43A1 and 43A3; included in *Df(2R)pk78k = Df(2R)42E3;43C3*, but not in *Df(2R)ST1 = Df(2R)43B3-5;43E18* (Ashburner *et al.*, 1981).

Pk: *Protein kinase*

Genes in *Drosophila melanogaster* encoding products related (by sequence comparison) to the serine-threonine protein kinases of mammals have been isolated from *Drosophila* clones obtained with mammalian probes. These genes are listed in the following table:

gene	location	synonym	cytology	comments
<i>Pka-C1</i>	2-{34}	<i>DC0</i>	<i>30C</i>	cAMP-dependant
<i>Pka-C2</i>	3-{102}	<i>DC1</i>	<i>100A</i>	cAMP-dependant
<i>Pka-C3</i>	3-{42}	<i>DC2</i>	<i>72A</i>	cAMP-dependant
<i>Pka-R1</i>	3-{47}	<i>RI</i>	<i>77F</i>	cAMP-dependant
<i>Pkc1</i>	2-{78}	<i>dPKC53E(br)</i>	<i>53E</i>	Ca ⁺⁺ -dependant
<i>Pkc2</i>	2-{78}	<i>dPKC53E(ey)</i>	<i>53E4-7</i>	Ca ⁺⁺ -dependant
<i>Pkc3</i>	2-{99}	<i>dPKC98F</i>	<i>98F</i>	Ca ⁺⁺ -dependant
<i>Pkg1</i>	2-{0.2}	<i>DG1</i>	<i>21D</i>	cGMP-dependant
<i>Pkg2</i>	2-{9}	<i>DG2</i>	<i>24A</i>	cGMP-dependant
<i>Pk?1</i>	2-{52}	<i>IC</i>	<i>36A</i>	?
<i>Pk?2</i>	3-{64.6}	<i>IJ</i>	<i>91C</i>	?
<i>Pk?3</i>	2-{59}	3-2	<i>45C</i>	?
<i>Pk?4</i>	1-{60}	3-10	<i>17E</i>	?
<i>Pk?5</i>	3-{42}	5-23	<i>72A</i>	?
<i>Pk?6</i>	3-{18}	7-10	<i>64F</i>	?
<i>Pk?7</i>	2-{78}	8-6	<i>53C</i>	?

The three protein kinase gene families that have been characterized in *Drosophila*, *Pka*, *Pkc*, and *Pkg*, are described in the following sections.

Pka: *Protein kinase-cAMP*

Drosophila c-AMP-dependent protein kinase has been purified from the bodies of adult flies; the enzyme is made up of catalytic and regulatory subunits (Foster, Guttman, Hall, and Rosen, 1984, *J. Biol. Chem.* 259: 13049-55). The catalytic subunit has a molecular weight of 40,000 and the regulatory subunit a molecular weight of 52,000 or 58,000 (based on electrophoretic mobilities in sodium dodecyl sulfate-polyacrylamide gels). This enzyme was found in all developmental stages of *Drosophila melanogaster*. Another c-AMP-dependent protein kinase occurs in larvae and during the first half of pupation, but not in embryos and adults. Genes encoding the protein found throughout the lifetime of *Drosophila* have been identified by molecular methods (Foster *et al.*, 1988; Kalderon and Rubin, 1988). These genes are very similar in their amino acid sequences to their mammalian counterparts. A description of the genes follows.

Pka-C1

references: Foster, Higgins, and Jackson, 1988, *J. Biol. Chem.* 263: 1676-81.

Kalderon and Rubin, 1988, *Genes Dev.* 2: 1539-56.

phenotype: Encodes one of the isoforms of the catalytic subunit form of *Pka*.

molecular biology: *Pka-C1* has been cloned using mammalian probes and its nucleotide and putative amino acid sequences determined (Foster *et al.*, 1988; Kalderon and Rubin, 1988). This gene shows 82% overall sequence identity to the c-AMP-dependent protein kinase catalytic

gene in mouse. *Pka-C1* is the source of at least four different transcripts (Kalderon and Rubin, 1988). The coding portion of the gene contains no introns and encodes a protein of 352 amino acids. An insertion of two amino acids not found in bovine and mouse enzymes has been located near the N terminus of the protein; 273 of the remaining 350 amino acids are identical to those of the bovine and mouse enzymes (Foster *et al.*, 1988).

other information: 11 additional *Drosophila melanogaster* DNA clones whose sequences are similar to that of *Pka-C1* within the kinase domain have been identified by nucleic acid hybridization. cDNAs have been obtained for ten of the genes involved (Kalderon and Rubin, 1988). The putative protein products of two genes (*Pka-C2* and *Pka-C3*) show 45% and 49% amino acid identity to the corresponding mouse protein kinase.

Pka-R1

references: Kalderon and Rubin, 1988, *Genes Dev.* 2: 1539-56.

phenotype: Encodes the type I regulatory subunit form of *Pka*.

molecular biology: A cAMP-dependent protein kinase regulatory subunit gene has been cloned using mammalian probes and its nucleotide and putative amino acid sequences determined (Kalderon and Rubin, 1988). *Pka-R1* is the source of at least three distinct transcripts that originate from different promoters that are spliced to a common coding region. The gene encodes at least three putative RI polypeptide products; it shows 71% amino acid identity to the mammalian RI subunit. Only the RNA class that encodes a full-length RI protein can be detected at all stages of development.

other information: There are two separate genes transcribed in opposite orientation to *Pka-R1* within the first and largest intron; the function of these genes is unknown.

Pkc: Protein kinase-c

The protein kinase C (PKC) enzyme in *Drosophila* is encoded by a gene family made up of three genes which are expressed primarily in adult flies. The deduced amino acid sequences of these genes are quite similar to each other and those of the mammalian PKC family (Schaeffer *et al.*, 1989).

Pkc1: Protein kinase-c1

references: Rosenthal, Rhee, Yadegari, Paro, Ullrich, and Goeddel, 1987, *EMBO J.* 6: 433-41.

Schaeffer, Smith, Mardon, Quinn, and Zuker, 1989, *Cell* 57: 403-12.

phenotype: Structural gene for protein kinase C (PKC) that is expressed primarily in the brain of adult flies.

molecular biology: *Pkc1* has been isolated from Oregon-R DNA clones identified by using bovine PKC cDNA probes and its nucleotide sequence and predicted amino acid sequence obtained (Rosenthal *et al.*, 1987). A single open reading frame from the combined sequences of two overlapping cDNAs encodes a 657-amino acid, 75-kd protein with extensive similarity to bovine, rat, and human protein kinase C. This *Drosophila melanogaster* PKC, like the mammalian forms, carries an amino-terminal cysteine-rich repeat region. The carboxy-

terminal region of the *Pkc1* protein shows about 88% identity to the corresponding region of the mammalian PKC proteins. *Pkc1* contains 13 coding exons of 32-623 pb and at least one untranslated 5' exon. Introns range in size from 54 to more than 8000 bp. The coding regions of *Pkc1* in Oregon-R and Canton-S strains differ by 15 silent changes and one change at amino acid 428 (from isoleucine to methionine); in addition, there are three insertion-deletion changes in the 5' untranslated region. Transcripts of about 4.3, 4, and 2 kb (equally abundant) are found in the head of adult flies, but were not found in 0-3 hr embryos (Rosenthal *et al.*, 1987). Transcription occurs in most neurons of the adult head, including photoreceptor cells (Schaeffer *et al.*, 1989).

other information: Molecular data indicate that *Pkc1* lies approximately 20 kb from *Pkc2*, another PKC gene with 53E cytological location (Schaeffer *et al.*, 1989).

Pkc2: Protein kinase-c2:

references: Schaeffer, Smith, Mardon, Quinn, and Zuker, 1989, *Cell* 57: 403-12.

phenotype: Structural gene for protein kinase C (PKC); expressed primarily in photoreceptors of adult flies.

molecular biology: *Pkc2* has been isolated from Oregon-R DNA clones identified by using bovine PKC cDNA probes and its nucleotide sequence and predicted amino acid sequence obtained (Schaeffer *et al.*, 1989). There is a single open reading frame which encodes a 700 amino acid protein that is similar to other PKCs. The protein encoded by *Pkc2*, like the mammalian forms of the enzyme, carries an amino-terminal cysteine-rich repeat region. The carboxy-terminal region of the *Pkc2* protein (ATP binding site and catalytic domain) shows a high degree of identity to the corresponding region of mammalian PKC proteins. Transcripts are expressed in head (not body) tissue of adult flies. A 2.5 kb transcript accumulates in late pupal stages during the terminal differentiation of photoreceptor cells; it is found in both compound eyes and ocelli of wild type individuals (Schaeffer *et al.*, 1989).

Pkc3: Protein kinase-c3

references: Schaeffer, Smith, Mardon, Quinn, and Zuker, 1989, *Cell* 57: 403-12.

phenotype: Structural gene for protein kinase C (PKC); expressed primarily in adult flies.

molecular biology: *Pkc3* has been isolated from Oregon-R DNA clones identified by using bovine PKC cDNA probes and its nucleotide sequence and predicted amino acid sequence obtained (Schaeffer *et al.*, 1989). There is a single open reading frame which encodes a 634 amino acid protein that shows 61% identity to the mammalian PKC (Ono, Fujii, Ogita, Kikkawa, Igarashi, and Nishizuka, 1988, *J. Biol. Chem.* 263: 6927-32); the *Pkc3* protein, however, diverges markedly from that of *Pkc1*, *Pkc2*, and the classical mammalian PKC genes in its amino-terminal region. *Pkc3* encodes a major 5.5 kb transcript that is found throughout development, although in reduced amounts in embryos; two other transcripts (4.3 and 4.5 kb) show increased embryonic expression. Adult transcription occurs in the cell bodies of the brain (Schaeffer *et al.*, 1989).

Pkg: Protein kinase-cGMP

Genes in *Drosophila melanogaster* encoding products related (by sequence comparison) to cGMP-dependent protein kinases of mammals have been isolated by using a *Drosophila* cAMP-dependent protein kinase catalytic subunit gene as a probe.

Pkg1

references: Kalderon and Rubin, 1989, J. Biol. Chem. 264: 10738-48.

phenotype: Encodes a product with putative cGMP-dependent binding and kinase domains that is 14% larger than the corresponding mammalian enzyme. It is believed to bind cGMP and to undergo changes enabling its catalytic site to interact with appropriate substrates. This gene is unusually polymorphic in various *Drosophila* stocks. The role of the gene and its enzyme in the organism as a whole is unknown at present.

molecular biology: *Pkg1* cloned and three cDNAs isolated with the genomic DNA as a probe; DNA and putative protein sequences determined (Kalderon and Rubin, 1989). A fragment that is part of the cGMP-dependent protein kinase gene (or a close homolog) was also cloned and sequenced (Foster, Higgins, and Jackson 1988, J. Biol. Chem. 263: 1676-81). There are four introns (one in the coding region, another in the regulatory domain, and two in the catalytic domain); the first intron is about 2 kb long. One major transcript of 2.8 kb was found in all developmental stages examined; this transcript is found mainly in the head in adult flies. The protein product differs from the bovine lung sequence at the amino terminus; it shows 61% identity to the mammalian enzyme in the putative cGMP binding domain and 70% identity in the kinase domain; both domains are arranged in consecutive order on the same polypeptide as in the mammalian cGMP-dependent protein kinase.

Pkg2

references: Kalderon and Rubin, 1989, J. Biol. Chem. 264: 10738-48.

phenotype: Encodes products with putative cGMP-dependent protein kinase activities (indicated by sequencing data obtained for all but two of the predicted proteins). The role of the gene and its enzymes in the organism as a whole is unknown at present.

molecular biology: Multiple cDNA clones were isolated and the sequence of representative cDNAs and the corresponding genomic DNAs determined (Kalderon and Rubin, 1989). There are three major RNA species of different sizes (4.6, 4.4, and 3.6 kb). The protein product shows 64% identity to the bovine lung cGMP-dependent protein kinase; one of the *Drosophila* products contains an additional 384 amino acids at the extreme amino terminus. All three of the major *Pkg2* transcripts can be detected throughout development. There are seven introns, three of which are located within two nucleotides of the corresponding introns in *Pkg1*; the positions of these introns do not correspond to the boundaries of the domains in the encoded proteins.

***pkh: pinkish**

location: 2-100.

discoverer: Bridges, 14g27.

references: 1919, J. Exptl. Zool. 28: 365.

Bridges and Morgan, 1919, Carnegie Inst. Washington

Publ. No. 278: 247 (fig.).

phenotype: Specific dilutor of w^e . RK3.

pl: pleated

location: 1-47.9.

origin: X ray induced.

discoverer: Moore, 31c15.

references: 1935, DIS 3: 27.

phenotype: Wings folded lengthwise in pleats. Overlaps wild type at 25°, more extreme at 19°. RK3.

cytology: Placed in salivary chromosome region 13B2-F17 on the basis of its being included in *Dp(1;f)A12 = Dp(1;f)1B-C;13B1-5* but not in the proximal part of the *X* derived from *T(1;4)A4 = T(1;4)13F6-14A1;102F* (inferred from Patterson, 1938, Am. Nat. 72: 193-206, also frontispiece of Texas Univ. Publ. 4032).

pl: see *pld*

***Pl: Pearl**

location: 2-6.

origin: Spontaneous.

discoverer: Rosin, 1948.

references: 1951, DIS 25: 75.

1952, Rev. Suisse Zool. 59: 261-68.

Nef, 1958, Z. Indukt. Abstamm. Vererbungslehre 89: 272-319 (fig.).

phenotype: Heterozygote has pearl-like nodes in wings. Wing margins often snapped; venation disturbed. Bristle pattern defective. Eyes small and rough. At 28°, at least one of these characters always present; at 18°, phenotype virtually normal. Viability good; fertility of male slightly reduced. Fraction of cells die in all imaginal disks. In wing disks, dead cells surrounded by epithelial cells and produce pearl-like structures in adult wing. Homozygote dies as pupa (Tschanz). RK2.

PL: Polygenic Locus

A genetic locus consisting of one or more closely linked genes at which allelic substitutions contribute to variance in a specified quantitative character such as the number of sternopleural bristles (Thompson and Thoday, 1974, Heredity 33: 430-37). The symbol *PL* is followed parenthetically by the chromosome involved and then by a symbol for the locus. A table of polygenic loci from the data of Thoday and his colleagues follows:

locus	location	synonym	ref ^α
<i>PL(1)sp</i> S1 ^β	1-2.4		3, 6
<i>PL(1)sp</i> S2	1-51.5		3, 6
<i>PL(2)L4a</i> γ	2-72.5		5
<i>PL(2)sp</i> G1	2-27.5		1, 6
<i>PL(2)sp</i> G2	2-47.5		1, 6
<i>PL(2)sp</i> S3	2-41.1	<i>ll</i>	3, 6
<i>PL(3)sp</i> T1	3-30.2	<i>3a</i>	4, 6
<i>PL(3)sp</i> T2	3-32.6	<i>3b</i>	4, 6
<i>PL(3)sp</i> W1	3-49	<i>a</i>	6, 7
<i>PL(3)sp</i> W2	3-51	<i>b</i>	6, 7
<i>PL(3)w</i> S	3-13	<i>lllw</i>	2, 6

^α 1 = Gibson and Thoday, 1962, Heredity 17: 1-26; 2 = Spickett, 1963, Nature (London) 199: 870-73; 3 = Spickett and Thoday, 1966, Genet. Res. 7: 96-121; 4 = Thoday, Gibson, and Spickett, 1964, Genet. Res. 5: 1-19; 5 = Thompson, 1976, Genetics 81: 387-402; 6 = Thompson and Thoday, 1974, Heredity 33: 430-37; 7 = Wolstenholme and Thoday, 1963, Heredity 18: 413-31.

^β "sp" = sternopleural bristles.

^γ "L4a" = L4 wing vein.

platinum: see *pt*

***pld: pallid**

location: 1-0.
origin: Found in progeny of flies treated with Janus green.
discoverer: Muller, 28e20.
synonym: *pl*.
references: 1935, DIS 3: 30.
phenotype: Body and wings pale. Viability about 10% wild type. RK3.
other information: Possibly an allele of *svr*.

allele	synonym
<i>pII 3</i>	<i>pII 74</i>
<i>pII 4</i>	<i>pII 122</i>
<i>pII 5</i>	<i>pII 312</i>
<i>pII 6</i>	<i>pII 316</i>
<i>pII 7</i>	<i>pII 385</i>
<i>pII 8</i>	<i>pII 628</i>
<i>pII 9</i>	<i>pII 864</i>
<i>pII 10</i>	<i>pII rm8</i>

ple: pale

location: 3-18.8.
origin: Induced by ethyl methanesulfonate.
references: Jürgens, Wieschhaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.
 Budnick and White, 1987, J. Neurogenet. 4: 309-14.
 Neckameyer and Quinn, 1989, Neuron 2: 1167-75.
phenotype: Homozygous lethal in embryo. Mutant embryos have unpigmented cuticle and head skeleton. Catecholamine levels reduced or absent (Budnick and White, 1987).
alleles:

allele	synonym	comments
<i>ple 1</i>	<i>*ple 7F</i>	
<i>ple 2</i>	<i>ple 70</i>	
<i>ple 3</i>	<i>*ple 11F</i>	temperature-sensitive
<i>ple 4</i>	<i>ple 14A</i>	

cytology: Located in 65A-E based on segmental aneuploidy produced by *Y-3* translocations.
other information: *ple* may encode tyrosine hydrolase since *ple* and *Th* both map to 65B. The ability of *Th* DNA to rescue *ple* mutants has not been tested.

pleated: see *pl*

Plexate: see *Px*

plexus: see *px*

pII: pelle

location: 3-92.
origin: Induced by ethyl methanesulfonate.
references: Anderson and Nüsslein-Volhard, 1984, Nature (London) 311: 223-27.
 Müller-Holtkamp, Knipple, Seifert, and Jäckle, 1985, Dev. Biol. 110: 238-46.
 Carroll, Winslow, Twombly, and Scott, 1987, Development 99: 327-32.
phenotype: Maternal-effect lethal; homozygous females sterile. As in the mutant *dl*, lethal embryos produced by *pII* females lack ventral and lateral elements (Anderson and Nüsslein-Volhard, 1984). Extreme *pII* embryos consist of a long, hollow tube with dorsal cuticular structures (Müller-Holtkamp *et al.*, 1985). Dorsalization is also observed in the pattern of *ftz* stripes in *pII* embryos (Carroll *et al.*, 1987). Injection of wild-type cytoplasm into *pII* embryos partially restores the normal embryonic pattern of development (Müller-Holtkamp *et al.*, 1985).
cytology: Located in 97F.
alleles:

allele	synonym
<i>pII 1</i>	<i>pII 019</i>
<i>pII 2</i>	<i>pII 078</i>

plo: pillow

location: 2- [52].
origin: Induced by ethyl methanesulfonate.
synonym: *pil*.
references: Steward and Nüsslein-Volhard, 1986, Genetics 113: 665-78.
phenotype: Semilethal over *Df(2L)H20*. Alleles, as *trans*-heterozygotes, show a visible recessive phenotype of small-lobed eyes.
alleles: *plo1, plo2, plo3*.
cytology: Placed in 36A8-C1 since included in *Df(2L)H20 = Df(2L)36A8-9;36E1-2*, but not in *Df(2L)H68 = Df(2L)36B2-C1;37A1-B1*.

pIs: palsied

location: 1-14.5 (approximate).
origin: Induced by ethyl methanesulfonate.
references: Singh and Siddiqi, 1981, Mol. Gen. Genet. 181: 400-02.
phenotype: Adults paralyzed by high temperature; shake legs just before paralysis.

Plum: see *bw*^{VI}

***plw: pale wing**

location: 1-37.2.
origin: Spontaneous.
discoverer: Fahmy, 1952.
references: 1959, DIS 33: 88.
phenotype: Body, wings, and bristles pale silvery yellow. Ecdysis delayed; viability low. RK3.

Pm: see *bw*^{VI}

*Pm*²: see *bw*^{V32g}

Pm^{D1}: see *bw*^A

Pm^K: see *Pu*^K

pn: prune

location: 1-0.8.
references: Beadle and Ephrussi, 1936, Genetics 21: 230.
 Demerec, Kaufman, Fano, Sutton, and Sansome, 1942, Year Book-Carnegie Inst. Wash. 41: 191.
 Nolte, 1959, Heredity 13: 233-41.
 Lifschytz and Falk, 1969, Genet. Res. 14: 53-61.
 Orevi and Falk, 1975, Mutat. Res. 33: 193-200.
 Ilyina, Sorokin, Belyaeva, and Zhimulev, 1980, DIS 55: 205.
 Teng, Bender, Engele, Tsubota, and Venkatesh, 1991, Genetics 128: 373-80.
phenotype: Eye color of newly emerged flies transparent brownish red, becoming brownish purple with age. The eyes of *pn* males have about 25% as much drosopterin (red pigment) as the eyes of wild-type males (Gearhart and MacIntyre, 1970, Anal. Biochem. 37: 21-25); the concentrations of xanthopterin and sepiapterin (brown

pigments) are increased to about 110% of wild-type (Nolte, 1959). Control of drosoplerin synthesis seems to be related to the activity of the enzyme GTP cyclohydro-lase (Evans and Howell, 1979, *Biochem. Genet.* 16: 13-26). The *pn* eye color is autonomous in larval optic disks transplanted into wild-type hosts (Beadle and Ephrussi, 1936). Larval Malpighian tube color is normal (Brehme and Demerec, 1942, *Growth* 6: 351-56).

Standard *pn* mutants are homo- and hemizygous viable in a wild-type background, but show a lethal interaction with the third chromosome dominant *awk^K* (Lifschytz and Falk, 1969; Orevi, 1973, *DIS* 50: 77). Some temperature-sensitive *pn* mutants (*pn^{ts-e}*), however, are insensitive to the killing action of *awk^K*; one

temperature-sensitive mutant (*pn^{ts-ek}*) is insensitive to *awk^K* at permissive temperatures (18°, 22°), but sensitive to *awk^K* at restrictive temperatures (25°, 29°) (Orevi, 1973, *DIS* 50: 80; Orevi and Falk, 1975). The TSP for the eye color phenotype occurs during the late pupal stage, while the TSP for the *pn* component of the *pn-awk^K* interaction begins at the late pupal stage and lasts until eclosion (Orevi and Falk, 1975). Homo- and hemizygous *pn* deficiencies and other chromosomal rearrangements have been induced by Ilyina *et al.* (1980), as indicated in the allele table.

alleles: Eye color like *pn¹* except when otherwise indicated. Alleles interact lethally with *awk^K* except for those affected by temperature (Orevi and Falk, 1975).

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	ref ^α	comments	cytology ^β
pn¹	spont	Bridges	3, 4, 15, 16, 22	brownish-red eye	
pn^{1a}	X ray		9, 20	homozygous lethal	<i>Tp(1;2)2D6-E1;20A2-3;20D;43F-44A1</i>
pn^{2γ}	X ray	Demerec	2, 6, 16, 19, 22	light brownish-red eye	
pn^{2a}	X ray		9, 20	homozygous lethal	<i>T(1;4)2D5-6;101F</i>
pn³		Wagenberg, Burdick	22		
pn^{3a}	X ray		9, 20	homozygous lethal	<i>Tp(1;2;3)2E1-2;12E1-2;20A;41A;80C-D</i>
pn⁵	X ray	Glass	7, 8		
pn¹²	X ray		9	homozygous lethal	<i>T(1;3)2E1-2;98A1-2</i>
pn²⁰	X ray		20		<i>T(1;2)2E1-2;40C1-2</i>
pn²⁵	X ray		9, 20	homozygous lethal	<i>Tp(1;3)2E1-2;20A1-2;70A5-6</i>
pn²⁶	X ray		9, 20	homozygous lethal	<i>Tp(1;3)2E1-2;20A1-2;70C1-2</i>
*pn²⁶⁻²⁰	X ray	Sobels	21		
pn^{27-9 δ}	mustard gas	Sobels	21, 22		
*pn²⁷⁻²²	mustard gas	Sobels	21		
pn³⁶	X ray		9, 20	homozygous lethal	<i>Tp(3;1)2E1-2;61A;62C1-2 + In(1)1A;2E1-2</i>
pn⁴⁰	X ray		9, 20	homozygous lethal	<i>T(1;2)2E1-2;41A</i>
pn⁴⁵	X ray		9, 20	homozygous lethal	<i>In(1)2D1-2;20A</i>
*pn^{51b}	p ³²	King	11		
pn^{51h}	X ray	W.K. Baker, 51h8	1		
pn⁵⁵	spont	Kivett	5		
pn^{59j}	spont	Narayanan, Weir	12, 13, 15	light brown eye	
pn⁶²	X ray	Petty			
pn^{63d}	X ray	Mittler	14	eye like <i>pn²</i>	
pn^{68b δ}			22		
pn⁶⁹			10	eye like <i>pn²</i>	
pn^{AA1}	NNG	Kaufman	10		
pn^{FG}	DES	Lifschytz	12, 13		
pn^{FS1}	spont	Falk	13		
pn^{MS2}	EMS	Orevi	18		
pn^{tr ε}	EMS	Lifschytz	13		
pn^{ts-e ζ}	EMS	Orevi	18		
pn^{ts-ek η}	EMS	Orevi	17, 18	<i>awd^K</i> insensitive	
			17, 18	<i>awd^K</i> insensitive at 18°, 22°	

^α 1 = Baker, 1956, DIS 30: 69; 2 = Beadle, 1937, Genetics 22: 587-611; 3 = Beadle and Ephrussi, 1936, Genetics 21: 203; 4 = Brehme and Demerec, 1942, Growth 6: 351-56; 5 = Clancy, 1959, DIS 34: 48; 6 = Evans and Howell, 1979, Biochem. Genet. 16: 13-26; 7 = Glass, 1934, DIS 2: 7; 8 = Glass, 1935, DIS 3: 14; 9 = Ilyina, Sorokin, Belyaeva, and Zhimulev, 1980, DIS 55: 205; 10 = Kaufman, 1970, DIS 45: 34; 11 = King, 1952, DIS 26: 65; 12 = Lifschytz and Falk, 1968, DIS 43: 131; 13 = Lifschytz and Falk, 1969, Genetics 62: 343-52; 14 = Mittler, 1967, DIS 42: 38; 15 = Narayanan and Weir, 1964, Genetics 50: 387-92; 16 = Nolte, 1959, Heredity 13: 233-41; 17 = Orevi, 1973, DIS 50: 80; 18 = Orevi and Falk, 1975, Mutat. Res. 33: 193-200; 19 = Schwinck, 1973, DIS 50: 122; 20 = Slobodyanyuk and Serov, 1983, Mol. Gen. Genet. 191: 372-77; 21 = Sobels, 1958, DIS 32: 84-85; 22 = Wagenberg and Burdick, 1969, DIS 44: 107.

^β For new orders, see rearrangement listing in the chromosome section.

^γ Synonym: *se-like 62*.

^δ *pn²⁷⁻⁹/pn^{68b}* flies are wild type in eye color and insensitive to *awd^K* (Wagenberg and Burdick, 1969).

^ε 40 temperature non-sensitive mutants indistinguishable from standard *pn* alleles (Orevi and Falk, 1975).

^ζ Nine *pn^{ts-e}* alleles, all insensitive to *awd^K* at all temperatures. Eye color light brown at 25° and 29°, wild type at 18° and 22° (Orevi and Falk, 1975).

^η One allele, *pn^{ts-ek}*, sensitive to *awd^K* at 25° and 29°, but insensitive at 18° and 22°. Eye color light brown at 25° and 29°, wild type at 18° and 22° (Orevi and Falk, 1975).

cytology: Located in 2E2-3 since included in *Df(1)pn7a = Df(1)2E1-2;3A4* and *Df(1)pn38 = Df(1)2D3-4;2E3* (Ilyina *et al.*, 1980). Previously located at 2D5-6 by Demerec and Sutton (Demerec *et al.*, 1942) and by J.I. Valencia.

pnr: pannier

location: 3-58.

origin: Induced by ethyl methanesulfonate.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

phenotype: Homozygous lethal. Dorsal anterior of embryo open.

alleles: Two alleles, *pnr¹* and *pnr²*, recovered as *pnr^{7G}* and *pnr^{9L}*.

cytology: Placed in 89B9-10 since uncovered by *Df(3R)sbd45 = Df(3R)89B4;89B10* but not by *Df(3R)sbd105 = Df(3R)88F9-89A1;89B9-10* (Hughes, Nelson, Yanuk, and Szauter).

pnt: pointed

location: 3-79.

origin: Induced by ethyl methanesulfonate.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

Mayer and Nüsslein-Volhard, 1988, Genes Dev. 2: 1496-1511.

phenotype: Homozygous lethal. Zygotic expression in embryo. Head skeleton of embryo pointed; median part of all denticle bands deleted. CNS broad and less dense than wild type. Sensory organs (maxillary, antennal, and Keilin's organs) spread.

alleles: Two alleles, *pnt¹* and *pnt²*, recovered as *pnt^{8B}* and *pnt^{9J}*.

cytology: Located in 94E; covered by *Y^{P3D} of T(Y;3)B27 = T(Y;3)94E* but not *T(Y;3)R13 = T(Y;3)94E*.

po: pale ocelli

location: 2-65.2.

origin: Spontaneous.

discoverer: Bridges, 38d1.

phenotype: Ocelli virtually colorless; some pigment bordering inner margins. Eye color slightly brighter than wild type. RK2.

***po²**

origin: Spontaneous.

discoverer: Bridges, 20j13.

synonym: *do: dilute ocelli*.

references: Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 224.

phenotype: Ocelli pale. RK3.

Po: Pyridoxal oxidase

location: 3-57.1 (0.08 unit to left of *Aldox*).

synonym: *lpo*.

references: Collins and Glassman, 1969, *Genetics* 61: 833-39.

Dickinson and Weisbrod, 1976, *Biochem. Genet.* 14: 709-21.

phenotype: The structural gene for the enzyme, pyridoxal oxidase [PO (EC 1.2.3.8)]; molecular weight approximately 225,000 daltons. Enzymatic characterization by Hanley (1980, *Mol. Gen. Genet.* 180: 455-62). Substrate specificities explored by Cypher, Tedesco, Courtright, and Kumaran (1982, *Biochem. Genet.* 20: 315-32). As PO is a molybdenum-containing protein (Warner and Finnerty, 1981, *Mol. Gen. Genet.* 184: 92-96), its activity is absent from *cin*, *mal*, and *lxd* flies, but cross reacting material is present in the latter two (but not the former) genotypes (Warner, Watts, and Finnerty, 1980, *Mol. Gen. Genet.* 180: 449-53). Enzyme activity present in all major organs except cardia, crop, imaginal discs, ovarian follicle cells, paragonia, pericardial cells, and wreath cells (Cypher *et al.*, 1982). Developmental profile shows dramatic increase in activity beginning in late pupa and continuing into adult life (Dickinson and Weisbrod, 1976). The nearly amorphic allele, *Po^{lpo}*, exhibits 2% normal activity; little if any cross reacting material is detectable in *Po^{lpo}*.

alleles: In addition to *Po^{lpo}*, electrophoretic variants *Po^S* (common) and *Po^F* (rare) have been recorded (Dickinson and Weisbrod, 1976). *In(3R)LVM* reported to carry a hypomorphic allele, *Po^{LVM}*.

cytology: Placed in 89A1-3 based on its inclusion in *Df(3R)Po3 = Df(3R)89A1-2;89A11-13* but not *Df(3R)c3G2 = Df(3R)89A2-3;89A4-5* (Hughes, Nelson, Yanuk and Szauter).

other information: Name changed from *lpo* to *Po* in order to conform to terminology for other structural genes for enzymes.

pod: podgy

location: 2-55.

references: Ashburner, 1991, DIS 69.

pod foot: see pdf

***podG: podoptera of Goldschmidt**

location: Multifactorial.

origin: Spontaneous.

discoverer: Goldschmidt, 1943.

references: 1945, *Science* 101: 389-90.

1945, *J. Morphol.* 77: 71-103 (fig.).

Goldschmidt, Hannah, and Piternick, 1951, *Univ. Calif. Publ. Zool.* 55: 67-294.

phenotype: Wing transformation into legs varies from

almost wild type to three-jointed, leg-like appendages. Penetrance of 1-2% was increased to 2-4% by selection. Scalloped, blistered, and unexpanded wings and various abnormalities of legs are pleiotropic effects. RK3.

other information: Podoptera may be similar to tetraltera effects.

podgy: see *pod*

***podH: podoptera of Hannah**

location: Multifactorial (principal factor on chromosome 2).

origin: Spontaneous.

discoverer: Hannah, 1943.

references: Goldschmidt, Hannah, and Piternick, 1951, *Univ. Calif. Publ. Zool.* 55: 67-294.

phenotype: Wings transformed into leg-like appendages. Legs characteristically changed and parts often duplicated. Average penetrance of 2.5% increases to 5% in selected lines. Somatic elimination of X chromosome produces more than 2% gynandromorphs. RK3.

other information: Claimed to have a maternally inherited component.

***podK: podoptera of Kellen-Piternick**

location: Multifactorial.

origin: Spontaneous.

discoverer: Kellen-Piternick, 1944.

references: Goldschmidt, Hannah, and Piternick, 1951, *Univ. Calif. Publ. Zool.* 55: 67-294.

phenotype: Like *podG*. Wings sometimes replaced by palpus-like structure. Average penetrance 30% in *X/X/Y* females and *X/Y* males. Females without *Y* or *Y^L* do not show podoptera phenotype. Rough eyes, notched wings, and absence of postverticals occur. RK3.

***podM: podoptera in M(3)w-124**

location: Multifactorial.

origin: Spontaneous.

discoverer: Kellen-Piternick, 1944.

references: Goldschmidt, Hannah, and Piternick, 1951, *Univ. Calif. Publ. Zool.* 55: 67-294.

phenotype: Wings transformed into leg-like structures. Penetrance of 15% in selected stocks is increased by presence of *Y^L*. RK3.

podoptera: see *podG*, *podH*, *podK*, *podM*

poi: see *svr^{poi}*

pointed: see *pnt*

Pointed wing: see *Pw*

Pointedoid: see *Bx^J*

pol: see *spa^{pol}*

pole hole: see *phl*

poliert: see *spa^{pol}*

polo: polo

location: 3-46.

references: Sunkel and Glover, 1988, *Jour. Cell Sci.* 89: 25-38.

phenotype: Maternal-effect mitotic mutation and male meiotic mutation. The wild type allele functions in the early embryo and in the imaginal and neuroblast cells of the larva. About 7% of *polo¹* homozygotes (offspring of

*polo*¹/+ females mated to *polo*¹/+ or *polo*¹/*polo*¹ males) survive to adulthood, but most of them die as late third instar larvae, their imaginal disks and neuroblast cells failing to proliferate. *polo*² homozygotes (offspring of *polo*²/+ females mated to *polo*²/+ males) never eclose, dying as early third-instar larvae. Some *polo*¹/*polo*² heterozygotes (less than 7%) survive, but show very abnormal cuticle formation in the abdominal tergites.

Offspring of surviving *polo*¹/*polo*¹ females die very early in development, the embryos showing an abnormal distribution of nuclei that fail to cellularize and then become polyploid before breaking down. Spindles of these embryonic nuclei are highly branched and have broad, barrel-shaped poles. In third instar larvae homozygous for *polo*¹, many of the neuroblast cells show mitotic abnormalities. In anaphase, the chromosomes are often in a circular arrangement and may or may not be polyploid or aneuploid; these chromosomes, however, are never fragmented and seem to undergo normal condensation. In anaphase, chromosomes frequently lie in a random orientation at one or both poles.

Surviving *polo*¹/*polo*¹ males are fertile, but often show meiotic abnormalities. Many of the meiotic spindles are irregular in shape and structure and a high frequency of nondisjunction occurs, mostly in the second meiotic division.

alleles:

alleles	origin	discoverer	comments
<i>polo</i> ¹	EMS	Nüsslein-Volhard	recessive semilethal; a few survivors
<i>polo</i> ²	HD	Karess	recessive lethal

cytology: Located in 76B2-77C (from Ashburner).

polychaetoid: see *pyd*

polychaetous: see *pys*

Polycomb: see *Pc*

polycombeotic: see *E(z)*

Polycomblike: see *Pcl*

Polygenic Locus: see *PL*

polyhomeotic: see *ph*

polymorph: see *ade2*

polyphene: see *pyp*

polyphene 61: see *pph*

polyphenic: see *pph*

polyubiquitin: see *Ubi-p*

ponte thermosensible: see *pts*

***pop: popeye**

location: 1-0.4.

origin: Induced by *p*-N,N-di-(2-chloroethyl)amino-phenylbutyric acid (CB. 1348).

discoverer: Fahmy, 1952.

references: 1958, DIS 32: 73.

phenotype: Eyes small, round, bulging, and rough. Often some central ommatidia protrude. Small body. Wings short, broad, and frequently blistered. Male sterile; via-

bility less than 10% wild type. RK3.

porc: porcupine

location: 1-59.

origin: Hybrid dysgenesis.

references: Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-52.

phenotype: Homo- and hemizygous lethal that affects segmentation pattern. *porc/Y* embryos from *porc/porc* germline clones show a segment polarity phenotype; *porc/+* progeny from the germline clones become normal females.

cytology: Located in 16E-17B from meiotic position.

***port: port**

location: 3- (not located).

discoverer: Morgan, 14c.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 125.

phenotype: Eye color slightly diluted. RK3.

***port-b: port-b**

location: 3- (not located).

discoverer: Bridges, 1911.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 214.

phenotype: Eye color maroon. RK3.

Positive spike II group: see *nona*

posterior cell blister: see *pcb*

posterior crossvein: see *pcv*

Posterior sex combs: see *Psc*

postverticalless: see *pvt*

Pox: Paired box

Two tissue-specific *Drosophila* paired box genes, *Pox-m* and *Pox-n*, both lacking homeodomains, are described in subsequent sections. No mutants have been discovered.

references: Bopp, Jamet, Baumgartner, Burri, and Noll, 1989, EMBO J. 8: 3447-57.

molecular biology: Genomic libraries of *Drosophila melanogaster* were screened with paired box probes from the *prd* gene and the two *gsb* genes (Frigerio, Burri, Bopp, Baumgartner, and Noll, 1986, Cell 47: 735-46; Baumgartner, Bopp, Burri, and Noll, 1987, Genes Dev. 1: 1247-67). The genes *Pox-m* and *Pox-n* were isolated and analyzed; they were found to share DNA and putative amino acid sequences with the paired box regions of *prd* and the two *gsb* genes. *Pox-m* and *Pox-n*, however, have no homeodomains. Both genes are believed to be controlled by *prd*, which has both paired- and homeodomains (Bopp *et al.*, 1989). The proteins of both *Pox-m* and *Pox-n* are located in the cell nucleus and may belong to the same regulatory cascade as the other paired domain genes.

Pox-m: Paired box-meso

location: 3-{48}.

phenotype: *Pox-m* shows tissue-specific, segmentally-repeated expression in the somatic mesoderm, starting at germ band extension. Transcripts are observed posterior to the parasegmental grooves (restricted to the mesodermal layer). The protein has been observed by immunostaining in the somatopleura that gives rise to the somatic

musculature, but is not found in the splanchnopleura and the mesectodermal cells (Jamet). Groups of cells in the clypeolabrum, the cephalic mesoderm, and the primordia of the telson and the proctodeum also express *Pox-m*.

cytology: Located in 84F11-12 by *in situ* hybridization to the salivaries. The distal breakpoint of *Df(3R)dsx5* is located at 84F11-12 in the intron of the *Pox-m* gene.

Pox-n: Paired box-neuro

location: 2-{48}.

phenotype: *Pox-n* shows tissue-specific, segmentally-repeated expression in the central and peripheral nervous system at about 5 hr after fertilization. Transcripts are apparently expressed in neural precursors of the peripheral and the central nervous system. Cells expressing this gene seem to be clonally related.

cytology: Located in 52C9-52D3 by *in situ* hybridization to polytene chromosomes. It is uncovered by *Df(2R)WVG = Df(2R)52C4;52E3* (Gelbart) and is located between the distal breakpoint of *Df(2R)XTE-18* at 52C9-D1 and the proximal breakpoint of *Df(2R)KL-9* at 52D3.

molecular biology: The *Pox-n* gene has an intron with the paired domain preceding the region encoding the helix-turn-helix motif (Bopp *et al.*, 1989).

Pp1: Protein phosphatase 1

location: 3- and 1- (see **molecular biology**).

references: Dombrádi, Axton, Glover, and Cohen, 1989, Eur. J. Biochem. 183: 603-10.

phenotype: Encodes a protein phosphatase 1 catalytic subunit that shows a high degree of similarity to rabbit protein phosphatase 1 α in its enzymatic and physiochemical characteristics and has a predicted protein sequence that is 92% identical to that of rabbit PP-1 α .

cytology: The major site of *Pp1* has been placed at 87B6-12 by *in situ* hybridization to the polytene chromosomes. In addition, three secondary sites have been demonstrated at 96A2-5, 9C1-2, and 13C1-2.

molecular biology: A 1.2-kb cDNA clone carrying the full coding sequence of *Drosophila Pp1* was isolated from a *Drosophila* head library using a 0.76-kb rabbit PP-1 α cDNA as a probe. The nucleotide and predicted amino acid sequences of this *Drosophila Pp1* were determined. A polypeptide of 302 amino acids with a molecular mass of 34.5 kd is encoded. The sequence of the 1.2-kb cDNA contains an open reading frame of 906 nucleotides flanked by 5' and 3' noncoding sequences. Abundant transcripts of 1.6 kb and 2.5 kb were detected in embryos, larvae, pupae, and adults. Another cDNA clone of 0.6 kb was isolated from the *Drosophila* head library with the same probe, and was found to hybridize to 9C1-2, indicating that there are at least two transcriptionally active *Pp1* genes in *Drosophila melanogaster*.

****pph: polyphenic***

location: 1-60.8 (originally located at 61.0 but genetic location arbitrarily interchanged with that of *sby* for consistency with cytological observations).

origin: Induced by D-1:6-dimethanesulfonyl mannitol (CB. 2511).

discoverer: Fahmy, 1959.

synonym: *pph-61: polyphene 61*.

references: 1964, DIS 39: 58.

phenotype: Body small. Eyes brighter than normal. Wing

size and shape slightly altered. Scutellar bristles occasionally kinked. Both sexes viable; fertility of homozygous female low. RK3.

cytology: Not included in deficiency for 18A4 through 18B8 produced by combining left end of *ln(1)y*⁴ = *ln(1)1A8-B1;18A3-4* and right end of *ln(1)sc*⁹ = *ln(1)1B2-3;18B8-9* (Norton and Valencia, 1965, DIS 40: 40).

pph-61 : see *pph*

pr: purple

location: 2-54.5.

references: Bridges, 1919, J. Exp. Zool. 28: 264-305.

Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 169.

Mainz, 1938, Z. Indukt. Abstamm. Vererbungsl. 75: 256-76.

Wright, Hodgetts, and Sherald, 1976, Genetics 84: 267-85.

Wilson and Jacobson, 1977, Biochem. Genet. 15: 321-32.

Yim, Grell, and Jacobson, 1977, Science 198: 1168-70.

Tobler, Yim, Grell, and Jacobson, 1979, Biochem. Genet. 17: 197-206.

Dorsett and Jacobson, 1982, Biochemistry 21: 1238-43.

Wiederreect and Brown, 1984a, J. Biol. Chem. 259: 14121-27.

Wiederreect, Paton, and Brown, 1984b, J. Biol. Chem. 259: 2195-2200.

Searles and Voelker, 1986, Proc. Nat. Acad. Sci. USA 83: 404-08.

phenotype: Eye color ruby at hatching, darkening to purplish ruby with age [Sturtevant and Beadle, 1939, An Introduction to Genetics, W.B. Saunders Co., Philadelphia and London, p. 64 (plate I)]; orange in combination with *st*, reddish brown in combination with *bw* (Mainz, 1938). *pr* eye color autonomous in larval optic disks transplanted into wild-type hosts (Beadle and Ephrussi, 1936, Genetics 21: 230). Larval Malpighian tubules wild-type in color (Beadle, 1937, Genetics 22: 587-611). Mutants deficient in pteridines (Hadorn and Mitchell, 1951, Proc. Nat. Acad. Sci. USA 37: 650-65; Wilson and Jacobson, 1977; Yim *et al.*, 1977); sepiapterin and the drosopterins are markedly reduced relative to wild type in *pr*¹ and *pr*^{bw} (Wilson and Jacobson, 1977; Dorsett and Brown, 1982), the effect being greater in *pr*^{bw} than in *pr*¹. The enzyme sepiapterin synthase A, which is involved in early steps leading to the synthesis of the drosopterins (Wiederreect *et al.*, 1984a, 1984b), is most active in wild-type *Drosophila* in late pupae and young adults when sepiapterin accumulation begins and the eyes become pigmented (Krivi and Brown, 1979, Biochem. Genet. 17: 371-90). When *pr*⁺ has been translocated into the Y chromosome [*Y*(*pr*^{CS}), the *Y*(*pr*^{CS}, *cn/pr*^{C4} *cn* flies show a variegated eye color (Yim *et al.*, 1977; Tobler *et al.*, 1979) and the mutant late larvae and early pupae show a reduction in sepiapterin synthase A activity as compared to both wild-type and *pr*¹ (Tobler *et al.*, 1979). The suppressors *su(s)*² and *su(pr)*^{e3} restore the pyrimidine level of *pr*¹ and *pr*^{bw} to that of wild-type or nearly wild-type (Wilson and Jacobson, 1977; Yim *et al.*, 1977).

alleles: *pr* mutants and rearrangements (other than

deficiencies) are described in the following table:

allele	origin	discoverer	ref ^α	eye color	cytology
<i>pr</i> ^{1β}		Bridges, 12b20	1-4, 6, 14	reddish-purple	
<i>*pr</i> ²		L.V. Morgan	8	redder than <i>pr</i> ¹	
<i>pr</i> ⁴⁰			11		<i>1n(2L)21D-E; 38B</i>
<i>*pr</i> ^{42d}	spont	Nolte, 42d	10	redder than <i>pr</i> ¹	
<i>pr</i> ^{80d}	spont	Najera, 80d	9b		
<i>pr</i> ^{81d}	spont	Najera, 81d	9, 9a		
<i>pr</i> ¹¹²	X ray	Wright	13		
<i>pr</i> ¹⁵⁶	X ray		13		
<i>pr</i> ^{bwβ}	spont	Bridges, 38d20	14	brownish-pink	
<i>pr</i> ^{C4γ}	EMS	Yim	12, 14	<i>pr</i> ^{C4} / <i>pr</i> ¹ : lighter than <i>pr</i> ¹ / <i>pr</i> ¹	
<i>pr</i> ^{C5}	EMS	Yim	12, 14	<i>pr</i> variegated	<i>Tp(2;Y)pr</i> ^{C5}
<i>pr</i> ^{IM60γ}	spont	Meyer, 60g	7	<i>pr</i> ^{IM60} / <i>pr</i> ¹ : purple	small deficiency?
<i>*pr</i> ^{M60}	X ray	Meyer, 60g	7	<i>pr</i> ^{M60} / <i>pr</i> ¹ : dark brown	
<i>*pr</i> ^{sδ}	X ray	Ives, 38k	5	weak <i>pr</i>	
<i>pr</i> ^v	X ray		13	<i>pr</i> variegated	<i>T(Y;2)TW124</i>

^α 1 = Beadle, 1937, *Genetics* 22: 587-611; 2 = Beadle and Ephrussi, 1936, *Genetics* 21: 230; 3 = Bridges, 1919, *J. Exp. Zool.* 28: 264-305; 4 = Bridges and Morgan, 1919, *Carnegie Inst. Washington Publ. No. 278*: 169; 5 = Ives, 1937, *DIS* 13: 50; 6 = Mainx, 1938, *Z. Indukt. Abstamm. Vererbungsleh.* 75: 256-76; 7 = Meyer, 1963, *DIS* 37: 51; 8 = Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 233; 9 = Najera, 1984, *DIS* 60: 241; 9a = Najera, 1985, *DIS* 61: 215; 9b = Najera, 1986, *DIS* 63: 167; 10 = Nolte, 1957, *DIS* 31: 84; 11 = Reuter and Wolfe, 1981, *Mol. Gen. Genet.* 182: 516-19; 12 = Tobler, Yim, Grell, and Jacobson, 1979, *Biochem. Genet.* 17: 197-206; 13 = Wright, Hodgetts, and Seraldi, 1976, *Genetics* 84: 267-85; 14 = Yim, Grell, and Jacobson, 1977, *Science* 198: 1168-70.

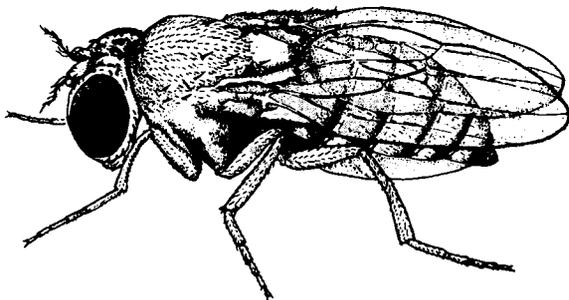
^β *pr*¹ and *pr*^{bw} suppressed by *su(s)*² and *su(pr)*^{e3} (see "phenotype").

^γ Homozygous lethal.

^δ *pr*^s/*pr*^s females sterile, males fertile; viability of males and females good. *pr*^s/*+* and *pr*^s/*pr*¹ females fertile.

cytology: Placed in 38B5-C2 based on its inclusion in the region of overlap of *Df(2L)pr2b = Df(2L)38B5-C1;38D2-E1* and *Df(2L)pr-A20 = Df(2L)38A3-4;38B6-C1*.

molecular biology: The transposable element 412 is inserted in cytological interval 38B4-6 in *pr*¹ and *pr*^{bw}, as indicated by hybridization of cloned 412 to salivary squashes (Searles and Voelker, 1986).



Pr: Prickly

From Muller, 1930, *J. Genet.* 22: 299-334.

Pr: Prickly

location: 3-90.0.

references: Muller, 1930, *J. Genet.* 22: 299-334 (fig.). 1935, *DIS* 3: 30.

Knust, Tietze, and Campos-Ortega, 1987, *EMBO J.* 6: 4113-23.

phenotype: Bristles very short; tips thin and twisted. Post-dorsocentrals and scutellars usually missing; dark granule present beneath normal bristle location. Homozygote has low viability. *Pr*^K complements some dominant traits of *E(spl)* and is in turn enhanced by it, showing severe defects and loss of bristles.

alleles:

allele	origin	ref ^α	molecular biology ^β
<i>Pr</i> ¹	X ray	1, 2	-4.5 to 0
<i>Pr</i> ^K		2	-4.5 to 0
<i>Pr</i> ^{Lγ}	spont	1, 2	-4.8 to -3.8

^α 1 = CP627; 2 = Knust, Tietze, and Campos-Ortega, 1987, *EMBO J.* 6: 4113-23.

^β The positions of DNA polymorphisms; relation to mutations unclear. DNA coordinates (kb) of the walk in region 96F8-13 where *E(spl)* has been mapped ("+" values to the right, "-" values to the left).

^γ Partial revertant of *Pr*¹. Bristles of *Pr*^{L/+} about one-third normal length (longer than bristles of *Pr*^{1/+}). *Pr*^L homozygote viable with small vestiges of bristles. *Pr*^{L/H} resembles *Pr*^{1/+}.

cytology: Located in 96F8-13 from molecular data; also located in 96F10-97C1.

***pra: prawny abdomen**

location: 1-15.2.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).

discoverer: Fahmy, 1954.

references: 1959, *DIS* 33: 88.

phenotype: Thorax narrow. Abdomen slender, often flexed between fourth and fifth segments. Wings short, rather broad, and often held atypically. Eclosion delayed. Viability about 15% wild type. RK3.

prat: pratfall (T. Schüpbach)

location: 2-98.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1989, *Genetics* 121: 101-17.

phenotype: Maternal effect lethal; embryos from homozygous mothers do not hatch. In cuticle preparations they show irregular segmentation and variable segment fusions.

alleles: *prat*^{PD} = *prat*¹.

cytology: Placed in 57B4-14 or in 57D8-58B (Schüpbach and Wieschaus, 1989).

prawy abdomen: see ***pra**

prd: paired

location: 2-45.

references: Nüsslein-Volhard and Wieschaus, 1980, *Nature (London)* 287: 795-801.

Nüsslein-Volhard, Kluding, and Jürgens, 1985, *Cold Spring Harbor Symp. Quant. Biol.* 50: 145-54.

Frigerio, Burri, Bopp, Baumgartner, and Noll, 1986, *Cell* 47: 735-46.

Kilchherr, Baumgartner, Bopp, Frei, and Noll, 1986, *Nature (London)* 321: 493-99.

phenotype: The wild-type allele of *prd* is required for normal segmentation in embryos and larvae. Mutant alleles and deficiencies show no maternal effects and are embryonic lethals with half the normal number of segmental units. In strong mutants, the anterior part of segments T1, T3, A2, A4, A6, and A8 and the posterior part of T2, A1, A3, A5, and A7 (*i.e.*, odd-numbered parasegments) are deleted (Nüsslein-Volhard *et al.*, 1985). Weak mutants such as *prd*² show small and less regular segmental deletions. Structures missing in *prd* mutants include: derivatives of the mandibular segments, labial sense organs, anterior prothorax, posterior mesothorax, anterior metathorax, and alternating posterior and anterior abdominal segments, including the telson and the posterior lateral sense organs (Nüsslein-Volhard *et al.*, 1985). No head fold visible at gastrulation. Experiments with a temperature-sensitive mutant indicate that the TSP occurs during the cellular blastoderm stage (Nüsslein-Volhard *et al.*, 1985; Kilchherr *et al.*, 1986).

alleles: *prd* mutants and rearrangements (except for deficiencies) are described in the following table.

allele	origin	synonym	ref ^α	comments
<i>prd</i> ¹	EMS		4	
<i>prd</i> ²	EMS		4	
<i>prd</i> ³	X ray	<i>prd</i> ^{2.27}	3, 5	<i>Tp(2;3)31B;33D-E;97C-D</i>
<i>prd</i> ⁴	X ray	<i>prd</i> ^{2.45.17}	1, 2, 7	strong allele
<i>prd</i> ⁵	X ray	<i>prd</i> ^{5.12}	3, 5	<i>Tp(2;Y)33A;35B</i>
<i>prd</i> ⁶	EMS	<i>prd</i> ^{6L}	7	strong allele
<i>prd</i> ⁷	X ray	<i>prd</i> ^{32.12}	7	strong allele
<i>prd</i> ^{8β}	EMS	<i>prd</i> ^{FR1}	6, 7	strong allele
<i>prd</i> ⁹	EMS	<i>prd</i> ^{IIB}	7	weak allele
<i>prd</i> ¹⁰	EMS	<i>prd</i> ^{IIN}	7	temperature sensitive
<i>prd</i> ¹¹	EMS	<i>prd</i> ^{IIV}	7	weak allele
<i>prd</i> ¹²	X ray	<i>prd</i> ^{X3}	7	strong allele

^α 1 = Frigerio, Burri, Bopp, Baumgartner, and Noll, 1986, Cell 47: 735-46; 2 = Kilchherr, Baumgartner, Bopp, Frei, and Noll, 1986, Nature (London) 321: 493-99; 3 = Nüsslein-Volhard, Kluding, and Jürgens, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 145-54; 4 = Nüsslein-Volhard and Wieschaus, 1980, Nature (London) 287: 795-801; 5 = Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82; 6 = Sander, Lohs-Schardin, and Baumann, 1980, Nature (London) 287: 841-43. 7 = Tearle and Nüsslein-Volhard, 1987, DJS 66: 209-69.

^β Larva moves in egg case, but fails to hatch. 45% of embryos show holes in the larval cuticle and/or lateral fusion of denticle belts (Sander *et al.*, 1980).

cytology: Located in 33C1-2 by *in situ* hybridization; included in *Df(2L)prd1.25 = Df(2L)33B6-7;33E2-3* as well as in *Df(2L)prd1.7 = Df(2L)33B2-3;34A1-2* (Nüsslein-Volhard *et al.*, 1985).

molecular biology: *prd*⁺ and *prd*⁴ have been cloned by microdissection of salivaries and chromosome walking and restriction-mapped to the DNA at +170 to +180 kb by Kilchherr *et al.* (1986) [coordinate 0 at distal end of a 41 kb *EcoRI* fragment; "+" values to the right, "-" values to the left (Frei, Baumgartner, Edström, and Noll, 1985, EMBO J. 4: 979-87)]. One transcript of 2.5 kb was detected in *prd*⁺ embryos; two transcripts of 2.5 and 3.6 kb were detected in embryos from *prd*⁴/*prd*⁺ parents; a 1.1 kb insertion was found in the mutant DNA (Kilchherr *et al.*, 1986). The 2.5 kb transcript is absent in oocytes, peaks in 2-4 hr embryos, and disappears after gastrulation.

The *prd*⁺ gene was sequenced by Frigerio *et al.* (1986) and the amino acid sequence of a putative *prd*

protein (613 amino acids) determined. The longest open reading frame is interrupted by a 356 bp intron after the first 22 amino acids. The 1.1 kb insertion in the *prd*⁴ DNA has also been sequenced by Frigerio *et al.* Two domains shared with the two closely-linked genes at the *gsb* locus (Bopp, Burri, Baumgartner, Frigerio, and Noll, 1986, Cell 47: 1033-40; Baumgartner, Bopp, Burri, and Noll, 1987, Genes and Development 1: 1247-67).

During the syncytial blastoderm stage in paired embryos, a transcript pattern of 7 bands, with a periodicity corresponding to two primordial segments, appears (Kilchherr *et al.*, 1986). By the time the embryos have reached the cellular blastoderm stage, an additional band appears posterior to band 7, band 1 is narrowed, and bands 2-7 have split, resulting in a 14-banded transcript pattern with single segment periodicity. *prd* is first expressed in a pattern similar to that of *ftz*, *h*, and *eve* (pair-rule genes). However, it shares domains with segment-polarity genes such as *bcd* (Bopp *et al.*, 1986). Expression of *en* absent with a consequent increase of *Ubx* expression in odd-numbered parasegments (Martinez-Arias and White, 1988, Development 102: 325-38).

pre: presto (*T. Schüpbach*)

location: 2-54.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

phenotype: Maternal-effect lethal; homozygous females lay eggs in which periplasmic clearing occurs, but no signs of cellularization are observed.

alleles: Two: *pre*¹ and *pre*² isolated as *PL* and *ADF* respectively.

cytology: Placed in 36E4-37C1 based on its inclusion in both *Df(2L)TW50 = Df(2L)36E4-F1;38A6-7* and *Df(2L)TW137 = Df(2L)36C2-4;37B9-C1*.

Pre-intermolt gene 1: see *Pig1*

presto: see *pre*

prickle: see *pk*

Prickly: see *Pr*

proboscipedia: see *pb*

prong: see *pg*

Pros35: Proteasome 35

location: 3-{59}.

references: Haass, Pesold-Hurt, Multhaup, Beyreuther, and Kloetzel, 1989, EMBO J. 8: 2373-79.

phenotype: Encodes the 35 kd proteasome subunit of *Drosophila melanogaster*. The proteasome is strongly expressed in the central nervous system of embryos and the heart muscle and epithelial cells of the stomach and ovary of adults and is localized in the cytoplasm in some cells and/or the nucleus in others.

cytology: *Pros35* was located by *in situ* hybridization at the border of 89F/90A. It is a single copy gene.

molecular biology: The gene was cloned and its nucleotide sequence and deduced amino acid sequence obtained; the primary translation product is a 31.4 kd protein. This protein is encoded by a mRNA of about 1100 nucleotides which seems to be present in very low abun-

dance. The cDNA is 999 bp long and there is a single open reading frame encoding 279 amino acids. The 35 kd protein subunit carries a consensus sequence for a potential tyrosine phosphorylation site; this subunit may be involved in the regulation of the complete multicatalytic proteinase complex.

Protein kinase: see *Pk*

proximalless: see *pxl*

prune: see *pn*

Ps: Pigmentless

location: 2-57.5 (inseparable from *cn*).

origin: X ray induced.

discoverer: Krivshenko, 56115.

references: 1959, DIS 33: 95.

phenotype: Black strips on last abdominal segments of female reduced; expression variable. Male unaffected. Homozygous lethal. RK2.

cytology: Salivary chromosomes apparently normal.

Ps2a: see *if*

Psc: Posterior sex combs

location: 2-67.

origin: Induced by ethyl methanesulfonate; isolated as embryonic lethal with head defects (Jürgens, 1985).

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

Jürgens, 1985, Nature (London) 316: 153-55.

Wu, Jones, Lasko, and Gelbart, 1989, Mol. Gen. Genet. 218: 559-64.

phenotype: *Psc*⁺ is another gene that may be considered a negative regulator of the *BXC* or the *ANTC*. The mutant is homo- and hemizygous lethal, the embryos showing partial transformation of head and thorax into abdomen and of abdominal segments 1-7 into more posterior ones. *Psc/Psc* embryos that are also homozygous for *Asx*, *Pcl*, or *Scm* show stronger posteriorly-directed transformations; the triple mutant *Psc Asx Pcl* has a tandem array of posterior abdominal segments including four abdominal denticle bands in the head region (Jürgens, 1985). *Psc/+* males may have sex combs on second and third legs. The gene suppresses *z*¹ eye color, which becomes orange-maroon; it is lethal with *Su(z)2*¹ and *Su(z)2*⁵ and semiviable with *Su(z)2*⁴ (Wu *et al.*, 1989). *Psc* and *Su(z)2* are both members of the *Su(z)2* complex.

alleles: One allele isolated: *Psc*^{INN} = *Psc*¹.

cytology: Located in 49D3-E5 since included in *Df(2R)vg-B* = *Df(2R)49D3-4;49F15-50A2-3* and *Df(2R)vg-D* = *Df(2)49C1-2;49E4-5*; at odds with the above determination is the failure of *Df(2R)vg-C* = *Df(2R)49B2-3;49E7-F1* to uncover *Psc* (Jürgens, 1985).

psi2: phase-angle2 (J. C. Hall)

location: 2-72.0.

origin: Induced by ethyl methanesulfonate.

references: Jackson, 1983, J. Neurogenet. 1: 3-15.

phenotype: Semidominant; emerges prematurely in light-dark cycle. Eclosion and locomotor activity rhythms have abnormally long periods (about 25-26 hr instead of normal 24 hr). Mutant males have abnormally long periods of courtship song rhythms (Kyriacou); phenotype recessive. Mutant males also show abnormal conditioning in courtship tests (Jackson, Gailey, and Siegel, 1983,

J. Comp. Physiol. 151: 545-52).

cytology: Located in 49E7-50A2, based on uncoverage of song rhythm defect by *Df(2R)vg-B* = *Df(2R)49D3-4;49F15-50A2* and complementation of this defect by *Df(2R)vg-C* = *Df(2R)49B2-3;49E7-F1* (Kyriacou and Clarkson).

other information: May be allelic to quasi-arrhythmic mutant *gat*, based on similar map positions and lengthened song rhythm periods in *psi2/gat* males (Jackson and Kyriacou).

psi3 (J. C. Hall)

location: 3- (to right of and close to *Sb*).

origin: Induced by ethyl methanesulfonate.

references: Jackson, 1983, J. Neurogenet. 1: 3-15.

phenotype: Semidominant; emerges prematurely in light-dark cycle. Eclosion rhythm exhibits abnormally long period (about 25 hr), as do males in their rhythmic courtship singing behavior (Kyriacou). Mutant males show abnormal conditioning in courtship tests (Jackson, Gailey, and Siegel, 1983, J. Comp. Physiol. 151: 545-52).

pt: platinum

location: 1-23.1.

origin: Deuteron induced.

discoverer: Hildreth, 51h.

references: Hildreth, 1953, DIS 27: 56.

King, Mohler, Riley, Storto, and Nicolazzo, 1986, Dev. Genet. (Amsterdam) 7: 1-20.

phenotype: Body color very pale yellow, almost colorless. Bristles colorless and translucent except for dark bases. Male sterile and short lived. Tyrosinase forms in adult (Horowitz and Fling). One allele (*pt*²) is a zygotic lethal; another (*pt*⁴) is female sterile (King *et al.*, 1986); shown to be a maternal-effect lethal rescuable by a normal paternal allele (Mohler and Carrol, 1984, DIS 60: 236-41).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>pt</i> ¹	deuteron	Hildreth	<i>pa</i>	1	viable
<i>pt</i> ²	X ray	Lefevre	<i>l(1)JC28</i>	2, 3	lethal (zygote)
<i>pt</i> ³	EMS	Lefevre	<i>l(1)VA70</i>	3	viable
<i>pt</i> ⁴	EMS	Mohler	<i>fs(1)M47</i>	2	female sterile

^α 1 = Hildreth, 1953, DIS 27: 56; 2 = King, Mohler, Riley, Storto, and Nicolazzo, 1986, Dev. Genet. (Amsterdam) 7: 1-20; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95.

cytology: Located in the 7F region since included in *Df(1)7F1-2;8C6*; maps to the left of *oc*, which is located at 8A1-2.

pt: see *ab*²

Pt-1: see *Lsp*²

pta: see *sld*^{pta}

ptc: see *tuf*

ptd: parted

location: 1-52.7.

origin: Induced by nitrosoguanidine.

references: Kaufman, 1969, DIS 44: 44.

phenotype: Wings held at 45° angle from body and slightly elevated. In young flies, the wing tips are usually curved up; in old cultures and old flies, the wings are usually shrivelled. The shrivelled condition (but not the cup-

ping) does not occur at 18°. Males viable and fertile, females viable and sterile.

Ptd: see *Bx*^J

**pte*: pterygion

location: 1-1.4.

origin: Induced by 1:4-dimethanesulfonylbut-2-yne (CB. 2058).

discoverer: Fahmy, 1951.

references: 1958, DIS 32: 73.

phenotype: Wings shortened, usually spread, and slightly drooping. Eyes misshapen and somewhat rough. Abdomen disproportionately large. Eclosion slightly delayed and viability about 20% wild type. RK3.

other information: Possible allele of *dwg*.

ptg: pentagon

location: 1-23.2.

phenotype: Thoracic trident darker than wild-type, especially the pentagonal spot just anterior to the scutellum; more extreme at 19°C. Hard to classify in young flies.

alleles: Six pentagon alleles are listed in the following table. (*ptg*², *ptg*³, and *ptg*⁴ are also given a separate description).

allele	origin	discoverer	synonym	ref ^α
<i>ptg</i> ¹		Bridges, 2218		
<i>ptg</i> ²		L.V. Morgan, 24j21		4
<i>ptg</i> ³		Kaliss, 351	<i>cro:crown</i>	1, 3
<i>ptg</i> ⁴	spont	Curry, 38b8		
<i>ptg</i> ⁵	EMS	Craymer	<i>ptg</i> ^{M1.270}	
<i>ptg</i> ⁶	EMS	Craymer	<i>ptg</i> ^{M1.33}	
<i>ptg</i> ^{7β}	EMS	Helfand, Carlson	<i>ptg</i> ^{3D18}	2

^α 1 = Felsenstein, 1937, DIS 7: 21; 2 = Helfand and Carlson, 1989, Proc. Nat. Acad. Sci. USA 86: 2908-12; 3 = Kaliss, 1937, DIS 7: 6,18; 4 = Morgan, L.V., 1935, DIS 3: 14.

^β Fertility of homozygotes low; response to odor of benzaldehyde reduced.

cytology: Located in salivary region 8A5 by Lefevre (1981, Genetics 99: 461-80). Included in the X chromosome insertion of *ct*⁺-*ptg*⁺ in *Dp(1;2)sn*⁺*72d* = *Dp(1;2)7A8;8A5;32C;58E*.

*ptg*²

phenotype: Pentagonal spot sharper and darker than in *ptg*¹. Scutellum often dark and prongs of tridents may be dark also.

*ptg*³

phenotype: Trident darker than in *ptg*¹; dark color extends to head, sides, and abdomen.

other information: Occasionally reverts to wild type or weak *ptg*. Allelism with *ptg* shown by Bridges.

*ptg*⁴

phenotype: Darkness of pentagon intermediate between that of *ptg*¹ and *ptg*².

ptm: see *bw*^{ptm}

pts: ponte thermosensible

location: 3- (near *se* at 3-26.0).

origin: Spontaneous in *se/se* strain.

references: Picard, 1973, Mol. Gen. Genet. 123: 363-68. Anxolabehere, 1980, Genetica 51: 161-65.

phenotype: Temperature-sensitive recessive female sterile (Picard, 1973). When homozygous *pts* females are kept

at 30°, their eggs fail to hatch or hatch in very small numbers at either 20° or 30°; when the females are kept at 20°, the eggs hatch at 20° but not at 30°. *pts/pts* females kept at an intermediate temperature (25°) are fertile, but their eggs take longer to develop than at the permissive temperature (Anxolabehere, 1980). Heterozygous females kept at either 20° or 30° hatch eggs at either temperature; their productivity is greater than that of homozygotes at 20° (Picard, 1973). The temperature-sensitive periods occur at the beginning of oogenesis (for laying) and from 36-60 hr of larval life (for hatching).

pu: pupal

location: 2-51.

synonym: *pads-b*.

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 232.

Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

phenotype: Wings unexpanded or incompletely expanded; more extreme at 19°. Extreme alleles show erect and crossed postscutellar bristles and a distorted thoracic cage (Ashburner). *Df(2L)A400/Df(2L)el*^{4D}*A80*^P, a homozygous *pu* deficiency, is barely viable; escapers are *pu*.

alleles: All alleles are cytologically normal.

allele	origin	discoverer	synonym	ref ^α	25° phenotype
<i>pu</i> ^{1β}	spont	Duncan, 20d		1, 3	strong
<i>pu</i> ²	EMS	Harrington		1	strong
<i>pu</i> ³	EMS	Harrington		1	strong
<i>pu</i> ⁴	EMS	Harrington		1	very weak ^γ
<i>pu</i> ^{5β}	EMS	Harrington		1	intermediate ^δ
<i>pu</i> ⁶	EMS	Littlewood	<i>pu</i> ^{76e}	2, 5	strong
<i>pu</i> ⁷	EMS		<i>pu</i> ^{DM7}	4	strong

^α 1 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91; 2 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95; 3 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 232; 4 = O'Donnell, Mandel, Krauss, and Sofer, 1977, Genetics 86: 553-66; 5 = Woodruff and Ashburner, 1979, Genetics 92: 133-49.

^β *pu*¹/*pu*⁵ flies show some wing expansion (Ashburner *et al.*, 1981).

^γ *pu*⁴ overlaps wild-type, its wings being almost fully expanded but wavy (Ashburner).

^δ *pu*⁵ shows some wing expansion at 25°.

cytology: Placed within 35A1-3 since *Df(2L)el*^{4D}*A80*^P = *Df(2L)35A2-4;35A3-4* [from *T(Y;2)el*⁴ and *T(Y;2)A80*] and *Df(2L)A400* = *Df(2L)35A1-4;35B10* are deficient for *pu*, but *Df(2L)fn2* = *Df(2L)35A3;35B2* is not (Ashburner).

other information: Not allelic to *pads*.

Pu: Punch

location: 2-97.

synonym: *upi*: *unpigmented* (Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82).

references: Mackay and O'Donnell, 1983, Genetics 105: 35-53.

Mackay, Reynolds, and O'Donnell, 1985, Genetics 111: 885-904.

Reynolds and O'Donnell, 1987a, Dev. Biol. 123: 430-41.

1987b, Genetics 116: s15.

1988, Genetics 119: 609-17.

O'Donnell, Boswell, Reynolds, and Mackay, 1989a,

Genetics 121: 273-80.

O'Donnell, McLean, and Reynolds, 1989b, Dev. Biol. 10: 273-86.

phenotype: The structural gene for guanosine triphosphate 7,8-8,9-dehydrolase = GTP cyclohydrolase [GTP CH (EC 3.5.4.16)], which catalyzes the first step in pteridine biosynthesis, the conversion of GTP to dihydroneopterin with the release of formic acid; GTP CH activity proportional to the number of *Pu*⁺ alleles. Purification and characterization by Weisberg and O'Donnell (1986, J. Biol. Chem. 261: 1453-58); the active complex has an apparent molecular mass of 575,000 daltons comprised of 39,000-dalton subunits. Developmentally regulated with a short-lived activity peak at or shortly before eclosion; 80-90% of activity found in the head; activity not detectable in embryos. Dominant alleles are embryonic lethal as homozygotes and in heterozygotes produce dilute purple eye color; Appear to be antimorphic in that GTP CH activity in heterozygotes reduced to less than the 50% normal levels observed in deficiency heterozygotes, at least in adult tissues, and is but 80% normal in genotypes that carry, in addition to the mutant allele, two doses of *Pu*⁺. Most are associated with a chromosome rearrangement with one breakpoint in 57 and the other in or near centric heterochromatin. A few recessive alleles are viable and fertile and exhibit reduced GTP CH activity in the head but normal or near-normal levels in prepupae and adult body; however, the majority are embryonic lethals and have reduced activity in prepupae, adult body, and head when heterozygous with viable alleles; rare lethal alleles are defective in neither eye-pigment production nor in postembryonic enzyme activity. Viable alleles complement lethal alleles for viability, at least partially; in combination with each other and with lethal alleles they display a wide range of eye pigmentation;

some pairs of lethal alleles complement fully or partially for both viability and eye color; prepupal enzyme levels are variably reduced in these combinations in ways that are uncorrelated with survival; heteroallelic survivors are fertile; heteroallelic survival markedly reduced or absent when reared at 16°.

Homozygous deficiencies [*Df(2R)F36*] die as fully formed larvae, but prior to hatching; mouth parts and setae completely unpigmented; setae and sensory structures poorly differentiated; cuticle thin and fragile; head involution and differentiation frequently incomplete, beginning at the time of germ-band shortening. Class II mutants display variably similar phenotypes as do class III alleles, but to a less severe extent. 50% of class V mutants die before blastoderm, the remaining 50% dying during late embryogenesis, but with fully pigmented mouth parts and setae and with normal head development; fusions and deletions of abdominal denticle belts, or the normal number of belts, but all of identically abnormal structure, retaining only the two posterior setal rows; preblastoderm nuclear divisions asynchronous, abnormally distributed in embryos, and nuclei misshapen (Reynolds and O'Donnell, 1987, Genetics 116: s14). Embryos homozygous for class IV alleles resemble class V mutants, but with additional features characteristic of class II embryos, including unpigmented setae and mouth parts. In crosses of class V bearing genotypes to *Df(2R)F36/+*, the *Df(2R)F36/Pu* embryos resemble class V embryos when the deficiency is maternally inherited and deficiency homozygotes when the *Pu* allele is maternally inherited.

alleles: In the following table, recessive alleles are superscripted "r" and revertants "rv". *Pu* deficiencies are listed as rearrangements.

allele	origin	discoverer	synonym	ref ^α	comments
Pu¹	X ray	Oliver, 28k4		5, 7, 8, 10, 12	<i>T(2;3)57B5-C1;79F</i> ; viable, dominant
Pu²	spont	Grell, 57b		4, 5, 6, 12	
Pu^{E18A}	EMS	Boswell		12	dominant, noncomplementing, cytologically normal (Class II) ^β
Pu^{Gr}	X ray	Muller, 29l	<i>Gr:Grape</i>	2, 3	noncomplementing (Class II)
Pu^{IN}	EMS			14	dominant, <i>T(2;3)57C;81F</i>
Pu^{IIK}	EMS			14	
Pu^K	X ray	Krivshenko, 53k24	<i>Pm^K</i>	1, 13	dominant, <i>In(2R)41;57E-F</i>
Pu^{K5-2}	EMS	Boswell		12	noncomplementing (Class II), dominant ^β
Pu^L	γ ray	Lyttle		5	dominant
Pu^{PD16}	EMS			12	noncomplementing (Class II)
Pu^{r1}	spont	Ives		5, 12	viable, <i>In(2R)57C;57E</i> ^β
Pu^{r331}	spont	Finnerty		5, 12	viable, eye color intermediate ^β
Pu^{rAA1}	EMS			5, 6, 12	noncomplementing (Class II)
Pu^{rAA4}	EMS			5, 12	viable
Pu^{rAA17}	EMS			5, 6, 12	noncomplementing (Class II)
Pu^{rFAMI-2}	EMS	Boswell		12	noncomplementing (Class II)
Pu^{rI9}	EMS			5, 6, 12	complementing (Class IVc)
Pu^{rJE5}	EMS			12	complementing (Class III)
Pu^{rK8-2}	EMS	Boswell		11, 12	noncomplementing (Class V), eye color normal
Pu^{rP1}	EMS			5, 6, 12	partially dominant
Pu^{rP11}	EMS			5, 6, 11, 12	noncomplementing (Class II)
Pu^{rP21}	EMS			5, 6, 11, 12	complementing (Class IVa)
Pu^{rP30}	EMS			5, 6, 11, 12	noncomplementing (Class II)
Pu^{rP42}	EMS			5, 6, 11, 12	complementing (Class III)
Pu^{rP43}	EMS			5, 6, 11, 12	complementing (Class III)
Pu^{rPF7}	EMS	Boswell		12	complementing (Class IVb) ^β
Pu^{rPF12}	EMS	Boswell		12	noncomplementing (Class II)
Pu^{rPH30}	EMS			12	noncomplementing (Class II)
Pu^{rPJ15}	EMS			12	noncomplementing (Class II)
Pu^{rS47}	EMS			12	noncomplementing (Class II)
Pu^{rSH8}	EMS	Boswell		5, 6, 11, 12	complementing (Class IVa)
Pu^{rTR1}	EMS			12	noncomplementing (Class II)
Pu^{rTR3}	EMS			12	noncomplementing (Class II)
Pu^{rTR5}	EMS			12	noncomplementing (Class II)
Pu^{rTR7}	EMS			12	noncomplementing (Class II)
Pu^{rv}	X ray	Oliver, 32127		9	complementing (Class IVc)
Pu^{rV3}	EMS			12	noncomplementing (Class II)
Pu^{rV9}	EMS			5, 6, 12	noncomplementing (Class II)
Pu^{rV14}	EMS			5, 6, 12	noncomplementing (Class II)
Pu^{rV15}	EMS			6, 12	noncomplementing (Class II)
Pu^{rX17}	EMS			5, 12	viable
Pu^{rW10}	EMS			6, 12	noncomplementing (Class II)
Pu^{rWE67}	EMS			11, 12	noncomplementing (Class V)
Pu^{rWE75}	EMS			11, 12	eye color normal
Pu^{rWM1}	EMS			12	complementing (Class V)
Pu^{rWM2}	EMS			12	eye color normal
Pu^{rWM3}	EMS			12	noncomplementing (Class II)
Pu^{rWM4}	EMS			12	viable
Pu^{rWM5}	EMS			12	complementing (Class IVc)
Pu^{rWM6}	EMS			12	noncomplementing (Class II)
Pu^{rWM7}	EMS			12	noncomplementing (Class II)
Pu^{rX25}	EMS			12	complementing (Class IVa)
Pu^{rZ2}	EMS			6, 12	complementing (Class IVc)
Pu^{rZ8}	EMS			6, 12	noncomplementing (Class II)
Pu^{rZ19}	EMS			5, 12	viable
Pu^{rZ22}	EMS			6, 11, 12	complementing (Class IVa)
Pu^{SHC}	EMS	Boswell		6, 12	noncomplementing (Class II)
Pu^{TR6}	EMS			5, 12	noncomplementing (Class II), dominant ^β
Pu^W	EMS			12	noncomplementing (Class II)
Pu^W	X ray	Lewis, 55h			dominant, <i>T(2;3)57B-C;80</i>

^α 1 = Krivshenko, 1954, DIS 28: 75; 2 = Glass, 1933, J. Genet. 28: 69-112; 3 = Glass, Am. Nat. 68: 111; 4 = Grell, 1960, DIS 34: 50; 5 = Mackay and O'Donnell, 1983, Genetics 105: 35-53; 6 = Mackay, Reynolds, and O'Donnell, 1985, Genetics 111: 885-904; 7 = Muller, 1930, J. Genet. 22: 326 (fig.); 8 = Oliver, 1932, Z. Indukt. Abstamm. Vererbungsl. 61: 484; 9 = Oliver, 1941, Proc. Int. Congr. Genet. 7th., p. 228; 10 = Oliver, 1935, DIS 3: 14; 11 = Reynolds and O'Donnell, 1987, Dev. Biol. 123: 430-41; 12 = Reynolds and O'Donnell, 1988, Genetics 119: 609-17; 13 = Rowan, 1966, DIS 41: 166-67.

^β 14 = Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-69.
More complete description below.

cytology: Placed in 57C5-6 based on its inclusion in both
Df(2R)F36 = *Df(2R)57B16-17;57C6-7* and

Df(2R)P112 = Df(2R)57C4;57D8-9; also placed in 57C by *in situ* hybridization to the salivaries (McLean, Boswell, and O'Donnell, 1990, Genetics 126: 1007-19).

molecular biology: Genomic clones of region recovered and mutants shown to cause alterations over a 30 kb segment. The region encodes at least 16 developmentally regulated transcripts (O'Donnell *et al.*, 1989b). Northern analysis reveals a 1.7 kb polyadenylated transcript that is abundant in the head and a 9 kb transcript that is transcribed off the same strand, overlaps the 1.7 kb sequence and is expressed at all stages (McLean and O'Donnell, 1987, Genetics 116: s52). The 1.7 kb transcript, which gives rise to a 39 kd polypeptide, is missing from three eye-specific *Pu* mutants (McLean, Lipsky, and O'Donnell, 1979, Genetics 122: s44).

other information: Mutations at this locus comprise a complex of viable and lethal, recessive and dominant, complementing and noncomplementing alleles with different stage and tissue specific defects. A complementation map has been constructed, but recombinational fine structure has not been determined.

***Pu*²**

phenotype: Eye color of *Pu*^{2/+} purplish, resembling *pr*; GTP CH activity reduced to less than 50% normal levels, in prepupae, adult body, and adult head, *i.e.* lower than observed in deficiency heterozygotes. Activity 80% normal in genotypes that carry, in addition to *Pu*², two doses of *Pu*⁺; Homozygous lethal with death occurring in the embryonic stage; slight transient dilution of eye color in such heterozygotes at eclosion.

***Pu*^{K5-2}**

phenotype: Slightly dominant eye-color phenotype. Survives in heterozygous combination with *SMI*, but behaves as a dominant lethal or displays delayed development in combination with most other second chromosomes tested.

***Pu*^{r1}**

phenotype: Homozygous viable recessive allele. GTP CH activity moderately reduced in prepupae and nearly normal in adult bodies of homozygotes; activity appears to be virtually absent in adult heads. Slight transient dilution of eye color in freshly emerged heterozygotes.

***Pu*^{r331}**

phenotype: Homozygous viable recessive allele. GTP CH activity in *Pu*^{r331}/*Pu*⁺ same as wild type; somewhat reduced in homozygotes.

***Pu*^{rP43}**

phenotype: Homozygous lethal; however, GTP CH activity in heterozygotes nearly normal. In heteroallelic combination with *Pu*^{rZ19} the enzyme produced is unstable at 53°; other combinations produce relatively heat-stable enzyme.

***Pu*^{SHC}**

phenotype: Similar to but less severe than that of *Pu*^{K5-2}. Apparent dominant lethal effects more severe when inherited maternally than when inherited paternally.

***pub*: pubescent**

location: 1-63.

origin: Induced by P³².

discoverer: Bateman, 1950.

references: 1950, DIS 24: 55.

phenotype: Hairs and bristles *M*-like; black pigment on terminal abdominal segments nearly absent; male sterile. Tendency toward short, fat, gnarled legs; shortened L2; posterior nicking of wings. After several generations, only bristle effect and male sterility remained. RK3.

****Pub*: Pub**

location: 1- (rearrangement).

discoverer: P. Farnsworth.

references: Lefevre, 1954, DIS 28: 75.

phenotype: Eye size of heterozygote variably reduced, ranging from something like *B*^{1/+} to wild type. Eyes of homozygote greatly reduced, similar to double Bar. Interacts with *B* to give small, glazed, almost facetless eyes. RK2A.

cytology: Associated with *ln(1)Pub*; breakpoints unknown.

***pubescent*: see pub**

Pudur*: see *Fs(3)Sz29

***puf*: puff**

location: 2-58.

origin: Spontaneous.

discoverer: Nichols-Skoog, 35k19.

phenotype: Wings puffed or blistered; effect centering in third posterior cell; wings warped and creased longitudinally along vein L3. Penetrance usually 90-100% in female and 20-40% in male. RK3.

other information: Possibly an allele of *blo* (Ashburner).

Pufdi*: see *Pfd

puff*: see *puf

***pum*: pumilio**

location: 3-48.5.

origin: Induced by ethyl methanesulfonate.

references: Lehmann and Nüsslein-Volhard, 1987, Nature (London) 329: 167-70.

Nüsslein-Volhard, Frohnhofer, and Lehmann, 1987, Science 238: 1675-81.

phenotype: Wild-type allele of *pum* involved in development of the abdomen (embryos) and of the imaginal disks (larvae or pupae), perhaps having a function in signal transport. Embryos derived from *pum/pum* females (strong allele) form two instead of eight abdominal segments; head, thorax, and telson are normal. Pole cells are formed by the *pum* embryos and these cells function normally when transplanted into otherwise sterile embryos. There is no paternal rescue. Partial rescue of the *pum* abdominal phenotype (at the site of the injection) can be obtained with cytoplasm from the posterior pole of (1) wild-type embryos or (2) *pum* embryos. When *pum* is combined with *tsl* (mutant in which the abdominal region is placed next to the pole plasm), a rescue of abdominal segments may occur. *pum* opposite a deficiency or another *pum* allele results in a recessive zygotic visible phenotype; *pum* adult flies have additional postalar, dorso-central, and scutellar bristles and reduced viability.

alleles: The first *pum* allele, *pum*¹², was recovered in a screen for maternal-effect mutations (Nüsslein-Volhard, Jürgens, Anderson, and Lehmann) and 12 more alleles were located on the basis of failure to complement the maternal phenotype (Lehmann, Frohnhofer, Anderson, Mayer, and Nüsslein-Volhard). All were induced by

ethyl methanesulfonate; most are semilethal and have abnormal bristles when homozygous.

allele	synonym
pum 1	<i>pum</i> ^{ET1}
pum 2	<i>pum</i> ^{ET2}
pum 3	<i>pum</i> ^{ET3}
pum 4	<i>pum</i> ^{ET4}
pum 5	<i>pum</i> ^{ET5}
pum 6	<i>pum</i> ^{ET6}
pum 7	<i>pum</i> ^{ET7}
pum 8	<i>pum</i> ^{ET8}
pum 9	<i>pum</i> ^{ET9}
pum 10	<i>pum</i> ^{ET10}
pum 11	<i>pum</i> ^{ET11}
pum 12	<i>pum</i> ²¹
pum 13	<i>pum</i> ⁶⁸⁰

cytology: Placed in region 85C-D since included in *Df(3R)p-XT9 = Df(3R)84F14;85C-D* but not in *Df(3R)p-XT103 = Df(3R)82A;85C1-2*.

pun: puny

location: 1-41.1.

origin: Induced by triethylenemelamine (CB. 1246).

discoverer: Fahmy, 1950.

references: 1958, DIS 32: 73.

phenotype: Body small. Wings slightly shorter than normal. Eyes occasionally deformed. Eclosion delayed. Both sexes fertile; viability about 50% wild type. RK3.

other information: One allele each induced by CB. 1356 and CB. 3025.

Punch: see *Pu*

punt: see *put*

puny: see *pun*

pupal: see *pu*

Pupal cuticle protein: see *Pcp*

Pupilla excentrica: see *Pec*

pur: purplish ruby

location: 3-39.5.

origin: Spontaneous in a natural population in Spain.

references: Aparisi and Najera, 1987, DIS 66: 12-13.

Najera and Aparisi, 1987, DIS 66: 191.

1988, DIS 67: 4-5, 5-6.

phenotype: Eye color purplish ruby. Red pigment at 75% wild-type level and brown pigment at 122% normal level.

pur1: purine requiring

location: 1-32.35.

origin: Induced by ethyl methanesulfonate.

references: Falk and Nash, 1974, Genetics 76: 755-66.

Johnson, Woloshyn, and Nash, 1979, Mol. Gen. Genet. 174: 287-92.

Nash, Woloshyn, Mehl, and Janca, 1981, Can. J. Genet. Cytol. 23: 411-23 (fig.).

phenotype: Purine nucleoside auxotroph; lethal on unsupplemented medium at 29° and poorly viable at 25°; rescuable by adenosine or guanosine supplementation. Slight oblique truncation of the wing occasionally observed as a small concavity of the wing margin at the distal end of vein L4 in *pur1* males and females; *pur1/Df(1)ras* females display stronger expression; hairs on wing margin regularly disposed, unlike *r*.

alleles: Two alleles, *pur1*¹ and *pur1*²; development of *pur1*¹ but not *pur1*² delayed when supplemented with adenosine; guanosine supports normal development of both.

cytology: Placed in 9E1-4 on the basis of its inclusion in the region of overlap between *Df(1)rasP14 = Df(1)9E1-2;9F3-4* and *Dp(1;2)9E1;10A11;56A* (Zhimulev, Pokholkova, Bgatov, Semeshin, and Belyaeva, 1981, Chromosoma 82: 25-40).

other information: *pur1* is but one of four types of mutations belonging to an interrelated complex involved some way in purine metabolism: *ras*, a nonauxotrophic eye-color mutation; two purine auxotrophs, *pur1* and *gual*, which complement *ras* and one another; *ras-l*, lethal mutations that fail to complement completely the other three types; genetic map order not determined. The complex shares many genetic features with rudimentary.

Pur-r: Purine-resistant (A. Chovnick)

location: 2-82 (approximate).

discoverer: Duck, 1973.

origin: Induced by ethyl methanesulfonate.

references: Dutton and Chovnick, 1990, Mol. Gen. Genet. 220: 172-76.

phenotype: Exhibits elevated resistance to purine [7H-imidazo (4,5-d) pyrimidine] in association with normal levels of xanthine dehydrogenase. Also confers resistance to 2,6-Diaminopurine. *Pur-r* is associated with elevated activity of adenosine deaminase (ADA); however, gene dosage experiments are inconsistent with the notion that *Pur-r* represents the structural locus for ADA, and indicate instead that *Pur-r* encodes a negative regulator of ADA. This view is supported by biochemical studies, which indicate that *Pur-r*⁺ encodes a thermolabile component that inhibits ADA activity, presumably analogous to the ADA-binding protein known in mammalian systems (Daddona and Kelley, 1980, Mol. Cell Biochem. 29: 91-101).

purple: see *pr*

purpleoid: see *pd*

Purpleoider: see *Pdr*

purplish ruby: see *pur*

put: punt

location: 3-58.

origin: Induced by ethyl methanesulfonate.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

phenotype: Homozygous lethal in embryo. Dorsal part of embryo open.

cytology: Located in 88C3-E2 based on ability of aneuploid segregants of both *Tp(3;1)XM54 = Tp(3;1)20;87C7-D1;88E2-3* and *Tp(3;2)kar51 = Tp(3;2)88C2-3;96B11-C1* to cover it.

***pvt: postverticalless**

location: 1-20.9.

origin: Induced by ethyl methanesulfonate.

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 88.

phenotype: Wings either divergent or slightly held up. Thoracic hairs sparse, and one or both postvertical bristles

tles almost invariably absent. Shape of head and eyes varies from almost normal to anteroposterior flattening of head and deep grooving of eyes. Male viable and fertile; female sterile. RK2.

***pw: pink wing**

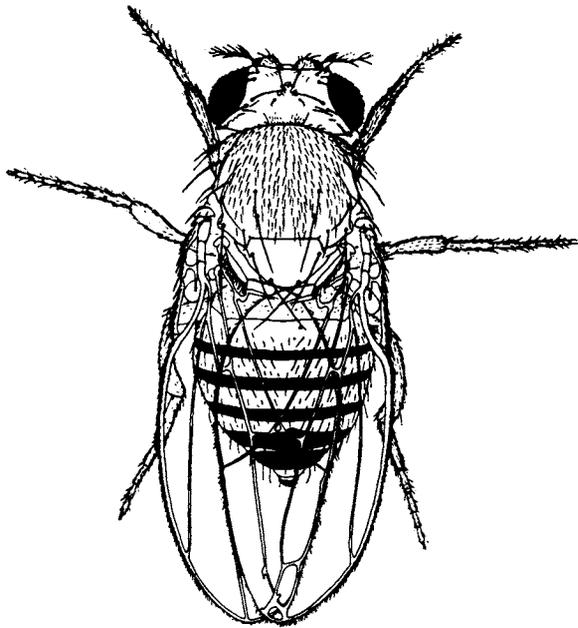
location: 2-14.
discoverer: Bridges, 20b17.
references: Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 213.
 1931, *Eos* 7: 229-48.
phenotype: Eye color like pink. Wings shorter than normal and crumpled. Viability low. RK3.

***Pw: Pointed wing**

location: 3-94.1.
discoverer: Bridges, 21c29.
references: Bridges and Morgan, 1923, *Carnegie Inst. Wash. Publ. No.* 327: 238 (fig.).
phenotype: Wings narrowed slightly at tips; extra venation near tips of L3 and L4. Homozygous lethal. RK3.
other information: Not an allele of *Bd* (3-93.8).

pwc: pink wing c

location: 2-79.
discoverer: Bridges, 31c18.
phenotype: Eye color lighter than normal. Wings short and blunt. Overlaps wild type. RK3.



Pw: Pointed wing

pwn: pawn

location: 2-55.4 [between *pk* and *cn* (Ashburner *et al.*, 1981)].
origin: Induced by ethyl methanesulfonate.
references: García-Bellido and Dapena, 1973, *DIS* 50: 179.
 1974, *Mol. Gen. Genet.* 128: 117-30.
 Lawrence and Morata, 1976, *Dev. Biol.* 50: 321-37.
 Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, *DIS* 56: 186-91.

Gubb and García-Bellido, 1982, *J. Embryol. Exp. Morphol.* 68: 37-57.

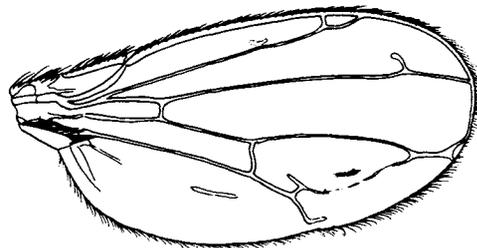
phenotype: Homozygous semilethal as zygote (survival 2%); escapers have dark brown eyes and show tanning of cornea cuticle (García-Bellido and Dapena, 1973). Cell viable in homozygous clones, which have truncated bristles with pale tips and pin-shaped trichomes with basal spurs and thin, transparent hairs (García-Bellido, 1973, 1974; Lawrence and Morata, 1976). Useful as cell marker.

cytology: Located in 42E3-43C3; included in *Df(2R)pk78k = Df(2R)42E3;43C3* (Ashburner *et al.*, 1981).

px: plexus

location: 2-100.5.
references: Bridges and Morgan, 1919, *Carnegie Inst. Wash. Publ. No.* 278: 251.
 Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 212, 233.
 Waddington 1940, *J. Genet.* 41: 75-139.
 Thompson, 1974, *Heredity* 33: 389-401.

phenotype: Wings have network of extra veins, especially towards tips and margins; L4 vein bent near tip. Semi-dominant with some Minutes. Suppressed by *S* (Bedichek, 1936, *DIS* 4: 24); suppresses expression of *ve* (Thompson, 1973, *DIS* 50: 59). Venation effect caused by inadequate contraction of wing during pupal stage, leaving spaces between epithelial layers (Waddington, 1940). Vein patterns of *px* flies studied in lines selected for increased or decreased expression (Thompson, 1974).



px: plexus

From "Edith M. Wallace, unpublished."

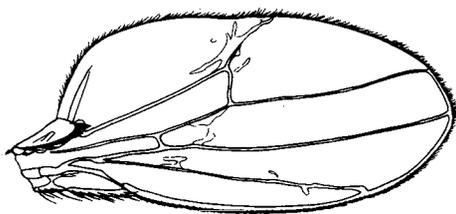
alleles: Phenotype same as *px*¹.

allele	origin	discover	ref ^α	cytology
<i>px</i> ¹	spont	Bridges, 14h20	1, 6	
<i>*px</i> ²	spont	Villee, 40a	7	
<i>px</i> ^{52g}	X ray	Iyengar, 52g	2, 4	<i>In(2LR)30A;58F-59B</i> (Lewis)
<i>*px</i> ^{54h}	spont	Meyer, 54h	5	
<i>*px</i> ^{55k}	spont	Williams, 55k	8	
<i>px</i> ⁷⁰	spont	Gooskov	3	

^α 1 = Bridges and Morgan, 1919, *Carnegie Inst. Wash. Publ. No.* 278: 251; 2 = Craymer, 1980, *DIS* 55: 197-200; 3 = Gooskov, 1971, *DIS* 46: 41; 4 = Iyengar and Meyer, 1956, *DIS* 30: 73; 5 = Meyer, 1954, *DIS* 28: 77; 6 = Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 212, 233; 7 = Villee, 1942, *Univ. Calif. Publ. Zool.* 49: 125-84; 8 = Williams, 1956, *DIS* 30: 80.

cytology: Placed in 58F on the basis of its inclusion in *Df(2R)M-1 = Df(2R)57F11-58A1;58F8-59A1* and *Dp(2;3)P* from *Tp(2;3)P = Tp(2;3)58E3-F2;60D14-E2;96B5-C1* [Bridges, 1937, *Cytologia* (Tokyo), Fujii *Jub. Vol.* 2: 745-55]. Also, *In(2LR)px*³²⁸ =

In(2LR)30A;58F-59B (Lewis) is mutant for *px*.



Px: Plexate

From "Edith M. Wallace, unpublished."

Px: Plexate

location: 2-107.2 (approximate).

origin: Spontaneous.

discoverer: Bridges, 22f6.

references: 1937, *Cytologia*, Fujii Jub. Vol. 2: 745-55.

Díaz-Benjumea, González-Gaitán, and García-Bellido, 1989, *Genome* 31: 612-19.

phenotype: Wing veins of heterozygote with plexuslike or deltalike thickenings, most often near posterior crossvein, and free fragments of vein, most often in third posterior cell; L4 vein bent near margin. Wings smaller and narrower than wild type and dusky textured. Closely resembles *bs*. Expression more extreme in female; enhanced by cold (19°). Homozygous *Df(2R)Px* lethal as embryo (Li, 1927, *Genetics* 12: 1-58); *Df(2R)Px2* cell lethal (Ripoll and García-Bellido, 1979, *Genetics* 91: 443-53). Mutants associated with deficiencies near tip of 2R.

alleles: Eight (Díaz-Benjumea *et al*, 1989).

cytology: A haplo-insufficient locus; placed in 60C6-D1 on basis of the region of overlap between *Df(2R)Px2* = *Df(2R)60C5-6;60D9-10* and *Df(2R)Px* = *Df(2R)60B8-10;60D1-2*.

other information: May be part of a pseudoallelic complex with *ba* and *bs*.

pxl: proximalless

location: 2R (5 units proximal to *bw*).

discoverer: Ransom.

phenotype: Temperature-sensitive lethal. Male and female sterile. Lacks proximal region of the retinal photoreceptors.

pyd: polychaetoid

location: 3-39.

origin: Spontaneous.

discoverer: Spencer, 39h31.

synonym: *Pch*; *xvt*.

references: Spencer, 1935, DIS 3: 28.

1937, DIS 7: 15.

Neel, 1939, *Genetics* 24: 81.

1941, *Genetics* 26: 52-68.

1943, *Genetics* 28: 49-68.

phenotype: Extra bristles present in homozygote at or near almost all normal bristle locations but most frequently in dorsocentral and scutellar regions. Heterozygote in some stocks occasionally shows extra bristles, especially vibrissae. Character expressed better at low temperatures and in large flies. Combinations with *h* and *Hw* generally superadditive for bristle number. RK3.

alleles: In addition to *pyd*¹, there are two mutants that resemble the original allele, both in chromosome location and bristle phenotype; they have been named *pyd*^{xvt} (synonym: *xvt*) and *pyd*⁶⁶. *pyd*^{xvt} appeared in an Australian line selected for an increased number of scutellar bristles (Miller and Fraser, 1968, *Aust. J. Biol. Sci.* 21: 61-74; Fraser, Erway, and Brenton, 1968, *Aust. J. Biol. Sci.* 21: 75-87; Fraser, 1970, *Genetics* 65: 305-09). *pyd*⁶⁶ appeared in a Spanish line selected for an increased number of dorsocentral bristles; also shows many verticals, orbitals, scutellars, interocellars, sternopleurals, and abdominals (Ménsua, 1972, DIS 49: 40), the mutant phenotype occurring with a high penetrance.

Pyk: Pyruvate kinase

location: 1-[43].

references: Rust and Collier, 1985, *J. Hered.* 76: 39-44.

phenotype: Structural gene for the glycolytic enzyme pyruvate kinase [PYK (EC 2.7.1.40) = ATP: pyruvate phosphotransferase]. Enzyme activity low in larvae and pupae, but it increases in young adults, becoming relatively stable in two-day-old flies. Most of the pyruvate kinase activity of adults is found in the thorax.

cytology: Located in the 12A-C region of the X chromosome on the basis of the dosage sensitivity of the region for pyruvate kinase as shown by segmental aneuploids.

pym: see *ade2*

***pyp: polyphene**

location: 1-53.5.

origin: Spontaneous.

discoverer: Bridges, 37l26.

phenotype: Wings spread, yellowish, and have uneven surface. Trace of extra vein in third posterior cell near second crossvein. Eyes rough, pitted, bulging, and smaller than wild type. Trident more darkly pigmented in male. Female sterile. Viability about 70% wild type. RK3.

pyr2: see *nuc1*

Pyridoxal oxidase: see *Po*

Pyruvate kinase: see *Pyk*

pys: polychaetous

location: 2-52.

discoverer: Curry, 37k15.

phenotype: Extra or double bristles present; most easily seen are scutellars, dorsocentrals, orbitals, and vibrissae. Extra bristles on scutellum curve upward. Overlaps wild type at 19°C; classification good at 28°-30°. RK3.

***Q: Queer wing**

location: 2- (not located).
discoverer: E. M. Wallace, 1931.
phenotype: Wings irregularly incised; marginal bristles irregular. Heterozygote has low penetrance; homozygote better. RK3.

Qd: Quadroon

location: 1-6.8.
origin: Spontaneous.
references: Thompson, 1959, DIS 33: 99.
phenotype: Broad dark band on margins of all abdominal tergites, giving abdomen superficial appearance of uniform darkness. Viability of heterozygous female normal, of homozygous female < 10% normal, and of male 30% normal. *Qd/Df(1)bi4* exhibit homozygous *Qd* phenotype as well as *bi*-like phenotype; *Qd/bi* and *Qd/+* are normal with respect to *bi* (Banga, Bloomquist, Brodberg, Pye, Larrivee, Mason, Boyd, and Pak, 1986, Chromosoma 93: 341-46). RK2.

cytology: Placed in 4C5-6 based on its inclusion in *Df(1)rb13 = Df(1)4C5-6;4D3-E1* and the localization of the X break in *T(1;3)bi^{D1} = T(1;3)4C5-6;65C3-5* (Banga *et al.*, 1986).

qf: quetas-finas

location: 3-60.7.
origin: Spontaneous.
references: Ribo, 1968, DIS 43: 59.
phenotype: Macro- and microchaetae thinner and shorter; low fertility; viability good.

qs: quicksilver

location: 1-39.5 (based on mapping of *qs²*).
references: Craymer, 1984, DIS 60: 234-36.
 Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307.
 Wright, 1987, Adv. Genet. 24: 127-222.
 Pentz, Black, and Wright, 1990, Biochem. Genet. 28: 151-71.
phenotype: Recessive embryonic lethal; denticles and mouthparts unpigmented; cell viable in gynandromorphs causing depigmentation of cuticle, including chaetae; viability reduced owing to weakened cuticle. Used as marker in the analysis of mosaic embryos (Gergen and Wieschaus, 1985, Dev. Biol. 109: 321-35). *qs¹*, *qs²*, and *qs³* hypomorphic in that expression varies with temperature and nutrition; some survival when reared on enriched medium; survivors smaller than normal and incompletely pigmented with a yellowish tinge; some extremely pale with bristles with very little pigmentation and with wings that when expanded are glassy clear and very fragile. The extremely pale *qs²/Y* never exhibit melanotic wound reaction, and puparia underpigmented. *qs* embryos derived from homozygous germ-line clones do not hatch and exhibit head defects and abnormalities of germ-band shortening; heterozygous embryos from such clones exhibit 75% survival; homozygous clones behave as meiotic mutant yielding 8% patroclinous males (Wieschaus and Noel, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 63-73). Phenol oxidase activity of any surviving *qs* flies significantly depressed, in some cases being undetectable; all three components, A1, A2, and A3, coordinately reduced; activator activity normal. Phenol oxidase deficiency leads to significant accumulations of

catecholamine pools in *qs²* males 60 to 80 min after eclosion: three-to-eight-fold increases in N- β -alanyldopamine and N-acetyldopamine and two-fold increases in dopamine (Pentz, Black, and Wright).

alleles:

allele	origin	discoverer	synonym	ref ^{α}	comments
<i>qs¹</i>	ENU	Crosby, 1982		1, 4	lethal
<i>qs²</i>	EMS	Sherald	<i>su¹⁸</i>	2, 4	recovered as suppressor of <i>b</i> ; cold sensitive in males and females ^{β}
<i>qs³</i>	EMS		<i>fid^{N1}</i>	3, 4	embryonic lethal
<i>qs⁴</i>	EMS		<i>fid^{N9}</i>	3	embryonic lethal
<i>qs⁵</i>	EMS		<i>fid</i>	3	embryonic lethal
<i>qs⁶</i>	EMS		<i>fid</i>	3	embryonic lethal
<i>qs⁷</i>	EMS		<i>fid</i>	3	embryonic lethal
<i>qs⁸</i>	EMS		<i>fid</i>	3	embryonic lethal
<i>qs⁹</i>	EMS		<i>fid</i>	3	embryonic lethal

^{α} 1 = Craymer, 1984, DIS 60: 234-36; 2 = Sherald, 1981, Mol. Gen. Genet. 183: 102-06; 3 = Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307; 4 = Wright, 1987, Adv. Genet. 24: 127-222.

^{β} At 25°, *qs²/qs²* females only 1% viable but *qs²/Y* males as viable as wild type (Pentz *et al.*, 1990).

cytology: Placed in 10F1-10 based on its inclusion in *Df(1)RA47 = Df(1)10F1;10F10* (Wieschäus *et al.*, 1984).

qua: quail (T. Schüpbach)

location: 2-53.
origin: Induced by ethyl methanesulfonate.
discoverer: Wieschaus and Nüsslein-Volhard.
references: Steward and Nüsslein-Volhard, 1986, Genetics 113: 665-78.
phenotype: Female sterile; nurse-cell contents not transported into the egg; follicle cells produce normal chorion around tiny eggs, which remain unfertilized.
alleles: *qua¹* isolated as *WP* by Wieschaus and Nüsslein-Volhard; *f2qua²-qua⁷* by Steward; *qua⁸-qua¹²* by Schüpbach and Wieschaus as *HK, HM, PX, QE, and QT*.
cytology: Placed in 36C2-C4 by Steward and Nüsslein-Volhard; uncovered by *Df(2L)TW137 = Df(2L)36C2-4;37B9-C1*, but not by *Df(2L)VA18 = Df(2L)36C4-D1;37C2-D1*.

Quadroon: see *Qd*

quail: see *qua*

Queer wing: see *Q*

quetas finas: see *qf*

qui: quit (T. Schüpbach)

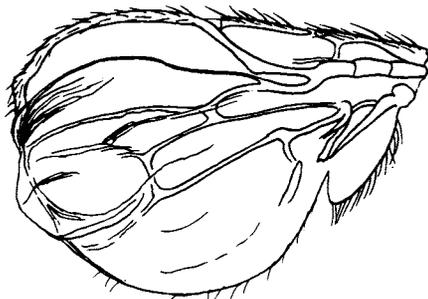
location: 2-105.
origin: Induced by ethyl methanesulfonate.
references: Schüpbach and Wieschaus.
phenotype: Female sterile; ovaries of homozygous females contain egg chambers with normal and abnormal numbers of nurse cells, which start degenerating at early stages of oogenesis.
alleles: Two: *qui^{QL} = qui¹* and *qui^{PX}*.
cytology: Localized to 59D8-60A7, since *qui⁻¹* and *qui⁻²* recovered as *QL* and *PX*, respectively, uncovered by *Df(2R)bw-S46 = Df(2R)59D8-11;60A7*.

quicksilver: see *qs*

quit: see *qui*

***r*: rudimentary**

location: 1-54.5 [Revised to 55.6 by Colaianne and Bell (1972, DIS 48: 20-21) and to 55.3-55.4 by Nørby (1970, DIS 45: 41) based on large scale measurements of *r-f* recombination].



phenotype: Homozygotes and hemizygotes are pyrimidine auxotrophs (Nørby, 1969, Hereditas 66: 205-14). A complex locus encoding a 220 kd polypeptide containing the first three enzyme activities in the pyrimidine synthetic pathway: glutamine-dependent carbamyl phosphate synthetase [CPS (EC.2.7.2.5)] (Jarry and Falk, 1974, Mol. Gen. Genet. 135: 113-22), aspartate transcarbamylase [ATC (EC.2.1.3.2)] (Nørby, 1969), and dihydroorotase [DHO (EC.3.5.2.3)] (Rawls and Fristrom, 1975, Nature 255: 738-40). Probably exists as a homomultimer. These three activities cosediment and copurify (Brothers, Tsubota, Germeraad, and Fristrom, 1978, Biochem. Genet. 16: 321-32); also the developmental profiles of the three activities are the same, maximal in the egg, dropping until the time of hatching, increasing again during the first larval instar, and then leveling off at a low level (Mehl and Jarry, 1978, Dev. Biol. 67: 1-10); high activity in egg attributable to maternal expression. Wings of homozygous females and hemizygous males obliquely truncated posteriorly; phenotype varies from wings that are wrinkled and blistered and do not extend beyond the tip of the abdomen to normal, with intermediate phenotypes having wings truncated to various degrees but not wrinkled or blistered, or normal wings with irregularly spaced marginal hairs. Wing cells smaller than normal (Fausto-Sterling and Hsieh, 1975,

Dev. Biol. 51: 269-81); oblique truncation attributed to cell death in distal portion of presumptive wing blade (Fausto-Sterling, 1980, J. Exp. Zool. 213: 383-90).

Homozygous females usually sterile when crossed to *r* male; occasionally give a few offspring, virtually all daughters plus a few exceptional males, when outcrossed. *r*³⁹ females produce many malformed eggs and unfertilized eggs with normal morphology; ovarian development often retarded or fails; yolk deposition affected; lethal effect in progeny results from generalized disturbance in differentiation 13-16 hr after fertilization at 25°; surviving embryos hatch late and may produce larvae that neither move nor feed (Counce, 1956, Z. Indukt. Abstamm. Vererbungsl. 87: 482-92). Eggs produced by *r*⁹ parents fail to hatch, but can be rescued by the injection of preblastoderm eggs with cytoplasm from either fertilized or unfertilized wild-type eggs or with pyrimidine nucleosides (Okada, Kleinman, and Schneiderman, 1974, Dev. Biol. 37: 55-62). *r/0* tissue in gynandromorphs produced by *r/r* females confined to abdomen; no such constraint when produced by *r/+* females (Fausto-Sterling, 1971, Dev. Biol. 26: 452-63). Mosaic studies of Falk (1977, Dev. Biol. 58: 134-47) indicate nonautonomy of *r*⁺ within the wing and the ovary, and that normal wing and ovarian development depend on pyrimidine synthesis within those organs. Female-sterile but not truncated-wing aspect of phenotype partially alleviated by administration of cytidine during development (Bahn, 1970, DIS 45: 99); administration of 6-azauracil or 6-azauridine, competitive inhibitors of pyrimidine synthesis, causes rudimentary phenocopies [Rizki and Rizki, 1965, Science 150: 222-23; Strømman, Bahn, Nørby, and Sick, 1973, Hereditas 73: 239-46 (fig.)].

Sex-linked recessive mutants selected on the basis of inability to survive on medium deficient in pyrimidines all map to the rudimentary locus; the majority have the rudimentary phenotype, but some are phenotypically normal and are designated subliminal alleles in the following table. Subliminal alleles exhibit strongly depressed survival on pyrimidine-free medium; standard alleles do not survive in the absence of pyrimidine; relative survival of *r/+* heterozygotes varies from 5 to 70% depending on severity of allele (Falk and Nash, 1974, Mol. Gen. Genet. 131: 339-49).

alleles:

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	synonym α	ref β	complementation C????BA A?B??CD 1234567 γ	comments δ
<i>r</i> 1		Morgan, 10f		25, 26, 27		
<i>r</i> 2		Bridges, 14g		10		
<i>r</i> 2L		Lancefield		19		
<i>r</i> 3		Sturtevant, 17j30				
<i>r</i> 4	spont	Wallace				
<i>r</i> 5	spont	Bridges		8		
<i>r</i> 6	spont	Bridges		8		
<i>r</i> 7	spont	Bridges		8		
<i>r</i> 8		Sturtevant		8		
<i>r</i> 9		Bridges, 20b3	<i>r</i> 7, 8	7, 25	++++++	
<i>r</i> 10	spont	Bridges		8		
<i>r</i> 11		Sturtevant		8		
<i>r</i> 12	spont	Wallace, 22k8		25		
<i>r</i> 13	spont	Bridges		8		
<i>r</i> 13A	chemical?	Fahmy	<i>r</i> MAH13A, 45	7	-----	
<i>r</i> 14	spont	Bridges, 24d4		25		
<i>r</i> 15	spont	Bridges		8		
<i>r</i> 15	chemical?	Fahmy	6	6	-----	
<i>r</i> 16		Stern		8		
<i>r</i> 17	spont	Bridges		8		
<i>r</i> 32k		Ball		8		
<i>r</i> 35	spont	Gottschewski, 1935				
<i>r</i> 35a	X ray	Oliver, 35a10		27		
<i>r</i> 39	sulfur mustard	Auerbach, 1951		11		
<i>r</i> 39k	spont	L. V. Morgan, 39k9	<i>r</i> sl, 9	7, 22	++++++	
<i>r</i> 39l	spont	Buzzati-Traverso, 39l12		6		
<i>r</i> 41	sulfur mustard	Auerbach, 1951		11		
<i>r</i> 53l	CB. 3025	Fahmy, 53l	29	7, 13	++++++	
<i>r</i> 54c	CB. 3026	Fahmy, 54c	36	7, 13	++++++	
<i>r</i> 54d	CB. 3025	Fahmy, 54d	30	7, 13	++++++	
<i>r</i> 54j	CB. 3007	Fahmy, 54j	37	7, 13	-----	
<i>r</i> 55a	CB. 3025	Fahmy, 55a	31	7, 13	++++++	
<i>r</i> 55k	CB. 3034	Fahmy, 55k	19	7, 13	++++++	
<i>r</i> 56d	CB. 1528	Fahmy, 56d	25	7, 13	++++++	
<i>r</i> 56j	CB. 1540	Fahmy, 56j	38	7, 13	++++++	
<i>r</i> 56k	CB. 1540	Fahmy, 56k	4	7, 13	++++++	
<i>r</i> 58a	X ray	Burdick, 1958	32	7, 5	++++++	
<i>r</i> 61	spont	Green	18	7	++++++	
<i>r</i> 61l	spont	Green, 61l8	44	7	-----	
<i>r</i> 61j	spont	Green, 61j26	11	7	++++++	
<i>r</i> 62j	chemical?	Fahmy	22	7	++++++	
<i>r</i> 63c	spont	Clancy, 63c		10		
<i>r</i> 64k	chemical?	Fahmy	33	7	++++++	
<i>r</i> 68b	colchicine	Gethmann		32		
<i>r</i> 68g	chemical?	Carlson, 68g18	14	7	++++++	
<i>r</i> 70b	X ray	Lefevre, 70b26		29	-?-?-?	In(1)7B;15A1-2
<i>r</i> 71j	spont	Mohler, 71j26		29	-?1??+	
<i>r</i> 75	chemical?	Fahmy	7	7	-----	
<i>r</i> 81k	P	Osgood, 81k24		28		
<i>r</i> 1996	chemical?	Fahmy	3	7	++++++	
<i>r</i> 2291	chemical?	Fahmy	23	7	++++++	
<i>r</i> 2381	chemical?	Fahmy	10	7	++++++	
<i>r</i> 2622	chemical?	Fahmy	16	7	++++++	
<i>r</i> 2696	chemical?	Fahmy	35	7	-----	
<i>r</i> 3433	chemical?	Fahmy	34	7	++++++	
<i>r</i> 3455	chemical?	Fahmy	39	7	++++++	
<i>r</i> 3463	chemical?	Fahmy	40	7	++++++	
<i>r</i> 3484	chemical?	Fahmy	5	7	++++++	
<i>r</i> 3589	chemical?	Fahmy	20	7	++++++	
<i>r</i> 3718	chemical?	Fahmy	15	7	++++++	
<i>r</i> 3719	chemical?	Fahmy	1	7	++++++	
<i>r</i> 3720	chemical?	Fahmy	13	7	++++++	
<i>r</i> 3721	chemical?	Fahmy	17	7	++++++	
<i>r</i> 3722	chemical?	Fahmy	24	7	++++++	
<i>r</i> bs1	EMS			4		
<i>r</i> bs2	EMS			4		
<i>r</i> bs3	EMS			4		
<i>r</i> C	spont	Nørby		3, 26		
<i>r</i> cCS1	EMS	Gans		2, 15		cold sensitive lethal
<i>r</i> E1	EMS			3		
<i>r</i> E2	EMS			3		
<i>r</i> E3	EMS			3		
<i>r</i> E4	EMS			3		

allele	origin	discoverer	synonym α	ref β	complementation		comments δ
					C???BA	A?B??CD	
					1234567 γ		
r E5	EMS			3			
r E6	EMS			3			
r E7	EMS			3			
r E8	EMS			3			
r E9	EMS			3			
r E10	EMS			3			
r G	spont	Goldschmidt	r px bl	16			
r H	H2CO	Auerbach		11			
r hdt	P	Tsubota		33			
r hd2	P	Tsubota		33			
r hd3	P	Tsubota		33			
r hd4	P	Tsubota		33			
r hd5	P	Tsubota		33			
r K		Krivshenko		1			In(1)20F
r LE1	EMS	Rawls		29	+?+??+		
r LE2	EMS	Rawls		29	+?+??+		
r LE3	EMS	Rawls		29	+?+??1+		subliminal allele
r LE4	EMS	Rawls		29	+?+??+		
r LE5	EMS	Rawls		29	+?+??+		
r LE6	EMS	Rawls		29	+?+??+		
r LE7	EMS	Rawls		29	+?+??+		
r LE8	EMS	Rawls		29	-?-??-		
r LE9	EMS	Rawls		29	+?+??+		
r LE10	EMS	Rawls		29	+?+??1+		subliminal allele
r LE11	EMS	Rawls		29	-?+??+		
r LE12	EMS	Rawls		29	+?1??+		
r LE13	EMS	Rawls		29	+?+??1-		
r LE14	EMS	Rawls		29	-?-??-		
r LE15	EMS	Rawls		29	+?+??+		
r LE16	EMS	Rawls		29	-?-??-		
r LE17	EMS	Rawls		29	-?-??-		
r LE18	EMS	Rawls		29	+?+??1+		subliminal allele
r LE19	EMS	Rawls		29	+++++		
r LE20	EMS	Rawls		29	+++++		
r LE21	EMS	Rawls		29	+?+??+		
r LE22	EMS	Rawls		29	+?+??+		
r LE23	EMS	Rawls		29	-?-??-		
r LE24	EMS	Rawls		29	+?+??+		
r LE25	EMS	Rawls		29	-?-??-		
r LI1	ICR170	Rawls		29	-?-??-		
r LI2	ICR170	Rawls		29	-?-??-		
r LI3	ICR170	Rawls		29	-?-??-		
r LI4	ICR170	Rawls		29	-?-??-		
r LI5	ICR170	Rawls		29	-?-??-		
r LI6	ICR170	Rawls		29	-?-??-		
r LI7	ICR170	Rawls		29	-?-??-		
r LI8	ICR170	Rawls		29	+?+??+		
r LI9	ICR170	Rawls		29	-?-??-		
r LI10	ICR170	Rawls		29	-?-??-		
r LI11	ICR170	Rawls		29	-?-??-		
r LI12	ICR170	Rawls		29	-?-??-		
r LI13	ICR170	Rawls		29	-?-??-		
r LI14	ICR170	Rawls		29	-?-??-		
r LI15	ICR170	Rawls		29	-?-??-		
r LI16	ICR170	Rawls		29	-?-??-		
r LX1	X ray	Rawls		29	+?+??+		
r LX2	X ray	Rawls		29	+?+??1-		
r LX3	X ray	Rawls		29	-?-??-		
r LX4	X ray	Rawls		29	+?+??+		
r LX5	X ray	Rawls		29	-?-??-		
r LX6	X ray	Rawls		29	-?-??-		
r LX7	X ray	Rawls		29	-?-??-		
r LX8	X ray	Rawls		29	-?-??-		
r LX9	X ray	Rawls		29	-?-??-		
r LX10	X ray	Rawls		29	-?-??-		
r LX11	X ray	Rawls		29	+?-??+		
r LX12	X ray	Rawls		29	+?1??+		
r M1	EMS	Mohler	fs(1)11-722	21			
r M2	EMS	Mohler	fs(1)11-836	21			
r M3	EMS	Mohler	fs(1)11-992	21			
r M4	EMS	Mohler	fs(1)11-007	21			
r M5	EMS	Mohler	fs(1)12-779	21			
r M6	EMS	Mohler	fs(1)12-829	21			

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	synonym ^α	ref ^β	complementation		comments ^δ
					C????BA	A?B??CD 1234567 ^γ	
<i>r^{M7}</i>	EMS	Mohler	<i>fs(1)12-1247</i>	21			
<i>r^{M8}</i>	EMS	Mohler	<i>fs(1)12-2088</i>	21			
<i>r^{M9}</i>	EMS	Mohler	<i>fs(1)12-2502</i>	21			
<i>r^{M10}</i>	EMS	Mohler	<i>fs(1)12-3642</i>	21			
<i>r^{M11}</i>	EMS	Mohler	<i>fs(1)12-4331a</i>	21			
<i>r^{M12}</i>	EMS	Mohler	<i>fs(1)13-873</i>	21			
<i>r^{M13}</i>	EMS	Mohler	<i>fs(1)13-1977</i>	21			
<i>r^{M14}</i>	EMS	Mohler	<i>fs(1)14-693</i>	21	+++++		
<i>r^{M15}</i>	EMS	Mohler	<i>fs(1)14D-73</i>	21			
<i>r^{ntg}</i>	NNG	Kaufman		18			
<i>r^{pyr1}</i>	EMS			14	----+		subliminal allele
<i>r^{pyr2}</i>	EMS			14	----+		subliminal allele
<i>r^{pyr3}</i>	EMS			14	----+		temperature-sensitive
<i>r^{pyr4}</i>	EMS			14	+++++		subliminal allele
<i>r^{pyr5}</i>	EMS			14			temperature-sensitive
<i>r^{pyr6}</i>	EMS			14	+++++		subliminal allele
<i>r^{pyr7}</i>	EMS			14	----+		temperature-sensitive
<i>r^{pyr8}</i>	EMS			14			subliminal allele
<i>r^{pyr9}</i>	EMS			14			
<i>r^{pyr10}</i>	EMS			14			
<i>r^{pyr11}</i>	EMS			14			female fertile
<i>r^{pyr12}</i>	EMS			14			
<i>r^{pyr13}</i>	EMS			14			female fertile
<i>r^{pyr14}</i>	EMS			14	+++++		
<i>r^{pyr15}</i>	EMS			14			
<i>r^{pyr16}</i>	EMS			14	----+		female fertile
<i>r^{pyr17}</i>	EMS			14			
<i>r^{pyr18}</i>	EMS			14			
<i>r^{pyr19}</i>	EMS			14			
<i>r^{pyr20}</i>	EMS			14			subliminal allele
<i>*r^S</i>	spont	Hadorn, 59d		30			
<i>r^{Sch}</i>	spont	Schalet		31			inserted DNA element
<i>r^{s2}</i>	spont	Green, 59k22	41	7, 17	+++++		
<i>r^{s3}</i>	spont	Green, 58b	21	7, 17	++----		
<i>*r^{s4}</i>	spont	Green, 59b		17			
<i>*r^{s5}</i>	spont	Green, 60i7		17			
<i>*r^{s6}</i>	spont	Green, 60i12		17			
<i>r^{s7}</i>	spont	Green, 60i12		17			
<i>*r^{s8}</i>	spont	Green, 61g2		17			
<i>*r^{Sn}</i>		Silberman		1			
<i>r^{sP1}</i>	spont	Craymer, 77k		12, 20			
<i>r^{sP2}</i>	spont			20			
<i>r^{tsB}</i>	EMS	Baker		12			
<i>r^{X1}</i>	X ray	Green, 60b13		17			
<i>r^{X2}</i>	X ray	Green, 60c15		17			
<i>r^{X3}</i>	X ray	Green, 60c15	28	7, 17	+++++		
<i>r^{X4}</i>	X ray	Green, 60c15		17			
<i>r^{X5}</i>	X ray	Green, 60c15		17			
<i>r^{X6}</i>	X ray	Green, 60dt	42	7, 17	-----		
<i>r^{X7}</i>	X ray	Green, 60e24		17			
<i>r^{X8}</i>	X ray	Green, 58a		17			
<i>r^{X9}</i>	X ray	Gloor, 57a	27	7, 17	+++--++		
<i>r^{X10}</i>	X ray	Gloor, 57a	26	7, 17	+++--++		
<i>r^{X11}</i>	X ray	Green, 60k27	43	7, 17	-----		
<i>r^{X12}</i>	X ray	Green, 60k27		17			
<i>r^{X13}</i>	X ray	Green, 60k27	12	7, 17	++++++		
<i>r^{X14}</i>	X ray	Green, 62j7		17			
<i>r^{X917}</i>	chemical?	Fahmy	2	7	+++++-		

^α Carlson (1971) ordered 45 alleles by means of genetic recombination; the order, from left to right, of alleles thus localized is represented by numbers in bold face. In some publications these numbers are used as allelic designations.

^β 1 = Agol, 1936, DIS 5: 7; 2 = Azou, Mehl, and Jarry, 1981, Dev. Biol. 84: 157-63; 3 = Bahn, Nørby, and Sick, 1971, Hereditas 69: 187-92; 4 = Bahn and Sondergaard, 1983, Hereditas 99: 309-10; 5 = Burdick, 1961, DIS 35: 45; 6 = Buzzati-Traverso, 1940, DIS 13: 51; 7 = Carlson, 1972, Genet. Res. 19: 129-32; 8 = Carnegie Publication 552; 9 = Carnegie Publication 627; 10 = Clancy, 1964, DIS 39: 65; 11 = Counce, 1956, Z. Indukt. Abstamm. Vererbungsl. 87: 482-92; 12 = Craymer, 1980, DIS 55: 197-200; 13 = Fahmy, 1959, Nature 184: 1927-29; 14 = Falk and Nash, 1974, Mol. Gen. Genet. 131: 339-47; 15 = Gans, Audit, and Masson, 1975, Genetics 81: 683-704; 16 = Goldschmidt, 1945, Univ. Calif. (Berkeley) Publ. Zool. 49: 501-03; 17 = Green, 1963, Genetica 34: 242-53; 18 = Kaufman, 1970, DIS 45: 34; 19 = Lancefield, 1918, Am. Nat. 52: 264-69; 20 = Modolell, Bender, and Meselson, 1983, Proc. Nat. Acad. Sci. USA 80: 1678-82; 21 = Mohler and Carroll, 1984, DIS 60: 236-41; 22 = Morgan, L.V., 1940, DIS 13: 51; 23 = Morgan, T.H., 1915, Am. Nat. 49: 240-50; 24 = Morgan and Bridges, 1916, Carnegie Inst. Wash. Publ. No. 237: 25 (fig.); 25 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 234; 26 = Nørby, 1970,

Hereditas 66: 205-14; 27 = Oliver, 1939, DIS 12: 48; 28 = Osgood, 1983, Genetics 104: s55; 29 = Rawls and Porter, 1979, Genetics 93: 143-61; 30 = Rohr, 1962, DIS 36: 39; 31 = Schalet, 1985, Genetics 110: s99; 32 = Thompson, 1969, DIS 44: 44; 33 = Tsubota, Ashburner, and Schedl, 1985, Mol. Cell. Biol. 5: 2567-74.

γ Complementation results according to Carlson (1972), Rawls and Porter (1979) and Falk (1976, Mol. Gen. Genet. 148: 1-8; Falk, McCaughin, and Cogley, 1977, Genetics 85: 765-77); arabic numbers are the equivalent of Roman numerals used by Carlson; upper case letters used by Rawls and Porter, and by Falk, where A of Rawls and Porter corresponds to C of Falk and to 1 of Carlson, B of Rawls and Porter to 3 of Carlson, C of Rawls and Porter to B of Falk to 6 of Carlson, and D of Rawls and Porter to A of Falk to 7 of Carlson. Lines with seven + or - symbols from Carlson (many confirmed by Rawls and Porter); lines with four + or - symbols from Rawls and Porter only; and lines with but three are from Falk.

δ Subliminal alleles exhibit normal-wing phenotype and female fertility, but survive very poorly on pyrimidine deficient medium. Fully mutant derivatives of subliminal alleles induced with EMS by Falk and deBoer (1980, Mol. Gen. Genet. 180: 219-24): six from r^{pyr1} , thirteen from r^{pyr2} , sixteen from r^{pyr3} , 21 from r^{pyr4} , and 29 from r^{pyr11} .

ϵ More detailed description below.

cytology: Tentatively placed in 15A1 by Lefevre [Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 32-66] and in 15A1-2 on the basis of *in situ* hybridization (Rawls, Freund, Jarry, Louis, Seagraves, and Schedl, 1986, Mol. Gen. Genet. 202: 493-99).

molecular biology: *r* transcription unit selected using a hamster *pyr1-3* probe; walk in either direction produced 90 kb of cloned sequence from 14F-15A region (Seagraves, Louis, Schedl, and Jarry, 1983, Mol. Gen. Genet. 189: 34-40). Genomic probe hybridizes to a 7.3 kb polyadenylated RNA, which is large enough to encode the 220 kd polypeptide. Direction of transcription from left to right, beginning with the domain for DHO on the left, continuing through the domain for CPS in the middle and ATC on the right; the region transcribed is approximately 12.5 kb in length and contains at least three introns, one of which is over 4 kb in length.

other information: Fine structure of the locus examined by both recombination and complementation (Carlson, 1971, Genet. Res. 17: 53-81). Carlson defines seven complementing regions, whereas Fahmy and Fahmy (1959, Nature 184: 1927-29) recognized six, Green (1963, Genetica, 34: 242-53) at least three, Falk (1976, Mol. Gen. Genet. 148: 1-8) three, and Rawls and Porter (1979, Genetics 93: 143-61) four. Region D of Rawls and Porter corresponds to regions VII and C of Carlson and Falk respectively, and identifies the DHO domain; region C of Rawls and Porter corresponds to regions VI and B of Carlson and Falk and identifies the CPS domain; and region D of Rawls and Porter corresponds to region I and C of Carlson and Falk and identifies the ACT domain. The genetic map and the complementation map are roughly colinear. Noncomplementing alleles map to both the 5' and 3' ends of the gene (Carlson's numbers 5 and 6 at the 5' end and numbers 35, 37, and 42-45 at the 3' end, where numbers refer to approximate linear order of alleles on the genetic map). Complementation maps constructed using wing phenotype and survival on unsupplemented medium not identical (Falk, 1976).

r^{CS1} : rudimentary-cold sensitive

phenotype: Homozygotes raised at 16° are lethal, at 20° are rudimentary, and at 25° are wild type in phenotype; the same results are observed in $r^{CS1}/Df(1)r-D17$ and r^{CS1}/r^1 heterozygotes; r^1 is deficient for DHO only. Heterozygotes with r^{11} , which is deficient for CPS, are rudimentary at all temperatures, and heterozygotes with r^{29} and r^{38} , which lack ATC activity, are wild type at all temperatures. Cold-sensitive periods during embryogenesis and during organogenesis in the early pupal stage; larval stages cold stable. Activities of DHO iso-

lated from early embryos cold sensitive; CPS exhibits reduced activity at all temperatures; ATC cold stable (Azou, Mehl, and Jarry, 1981, Dev. Biol. 84: 157-63).

R: Roughened

location: 3-1.4.

discoverer: E. M. Wallace, 35i.

phenotype: Eyes of $R/+$ rough, have some large dark facets. Photoreceptor cell 7 frequently absent (Carthew). Male genitalia frequently rotated and male sometimes sterile; viability about 80% wild type. Homozygote semi-lethal; wings spread. Thorax short; acrostichal hairs deranged, some missing; eyes small. Homozygous female fertile. RK1.

alleles: R^2 , a hybrid-dysgenesis-induced revertant of R , which is hemizygous lethal (Sliter, Henrich, Tucker and Gilbert, 1989, Genetics 123: 327-36).

cytology: Placed in 62B8-12 based on reversion of R by $Df(3L)R-G2 = Df(3L)62B2-4;62B11-12$ and $Df(3L)R-G7 = Df(3L)62B8-9;62F2-5$ (Sliter, Henrich, Tucker, and Gilbert, 1989, Genetics 123: 327-36).

other information: Recently determined to be an allele of *Rap1* (Hariharan).

* R^{51b}

origin: Recovered among progeny of female treated as embryo with cold shock.

discoverer: Mickey, 51b21.

references: 1951, DIS 25: 74.

1951, Genetics 36: 565-66.

phenotype: Eyes of heterozygote small, oblong, and rough; facets and eye hairs irregular. Viability good. Homozygote lethal. R^{51b}/R has very small eyes, much fusion of facets, and resembles *gl* and *Gl*. RK1.

$R^3-55.4$: see *M(GPDH)*

$R^3(+)$: see *T(2;3;4)ci+3*

$R^{26}-43$: see *M(ADH)*

r-l: rudimentary-like

location: 3-70.4 (based on 240 *gl-e* recombinants).

references: Lastowski and Falk, 1980, Genetics 96: 471-78.

Rawls, 1980, Mol. Gen. Genet. 178: 43-49.

Rawls, 1981, Mol. Gen. Genet. 184: 174-79.

Conner and Rawls, 1982, Biochem Genet. 20: 607-19.

phenotype: Encodes a polypeptide with the enzyme activities that catalyze the fifth and sixth steps of *de novo* pyrimidine synthesis: orotate phosphoribosyl transferase [OPRT (EC. 2.4.2.10)] and orotidylate decarboxylase [ODC (EC. 4.1.1.23)] respectively. Thought to exist as a homodimer (Rawls, 1979, Comp. Biochem. Physiol. 62B: 207-16). The first three enzyme activities encoded

by *r* and the fourth by *Dhod*. OPRT and ODC activities drastically reduced except that OPRT moderately reduced in *r-l*^{K7} and ODC above normal in *r-l*^{K6}. All mutants accumulate orotic acid, the substrate of ODC. Flies hemizygous or homozygous for mutants at this locus resemble *r* in that they have obliquely truncated wings with deranged marginal hairs and virtual female sterility; they differ from *r* in having faintly mottled eyes and severely impaired viability (viability of hypomorphs moderately reduced). *r* is epistatic to *r-l* in that *r*; *r-l* flies display the *r* rather than the *r-l* phenotype, suggesting that mottled eyes and reduced viability are the consequence of orotic acid accumulation rather than pyrimidine deficiency.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>r-l</i> ¹	EMS		<i>ral</i> ^{α1a}	2	
<i>r-l</i> ^{K1}	EMS	Rawls		1, 3, 4	noncomplementing
<i>r-l</i> ^{K2}	EMS	Rawls		1, 3, 4	hypomorph; complements <i>r-l</i> ^{K6} , <i>r-l</i> ^{K7} , and <i>r-l</i> ^{K8}
<i>r-l</i> ^{K3}	EMS	Rawls		1, 4	noncomplementing
<i>r-l</i> ^{K4}	EMS	Rawls		1, 4	noncomplementing
<i>r-l</i> ^{K5}	EMS	Rawls		1, 4	hypomorph; complements <i>r-l</i> ^{K6} and <i>r-l</i> ^{K8}
<i>r-l</i> ^{K6}	EMS	Rawls		1, 4	complements <i>r-l</i> ^{K2} , <i>r-l</i> ^{K3} , and <i>r-l</i> ^{K7}
<i>r-l</i> ^{K7}	EMS	Rawls		1, 4	complements <i>r-l</i> ^{K2} , <i>r-l</i> ^{K6} , and <i>r-l</i> ^{K8}
<i>r-l</i> ^{K8}	EMS	Rawls		1, 4	complements <i>r-l</i> ^{K2} , <i>r-l</i> ^{K3} , and <i>r-l</i> ^{K7}

^α 1 = Conner and Rawls, 1982, Biochem. Genet. 20: 607-19; 2 = Las-towski and Falk, 1980, Genetics 96: 471-78; 3 = Rawls, 1980, Mol. Gen. Genet. 178: 43-49; 4 = Rawls, 1981, Mol. Gen. Genet. 184: 174-79.

cytology: Tentatively placed in 93B4-13 based on its inclusion in *Df(3R)e-R6 = Df(3R)93B4-5;94A5-16* (B.S. Baker) and *Df(3R)e-R1 = Df(3R)93B3-5;93D2-4* (B.S. Baker) but not *Df(3R)e-F2 = Df(3R)93A6-B1;93D7-10* (Mohler and Pardue) = *Df(3R)93B8-13;93F9-10* (Scalenghe and Ritossa).

r(αGPDH): regulator of α glycerol phosphate dehydrogenase

location: 3-55.4.

origin: Spontaneous.

synonym: *m(GPDH)*, *r*^{3-55.4}.

references: King and McDonald, 1982, DIS 58: 91-92. 1983, Genetics 105: 55-69.

phenotype: Recessive allele of a locus that affects levels of αglycerol phosphate dehydrogenase activity. In the presence of the dominant allele, which we designate *r(αGPDH)*⁺, αGPDH levels in the adult are 60% higher than they are in *r(αGPDH)* homozygotes; the increase is observed in the thorax but not the abdomen. Rates of enzyme synthesis the same in the two genotypes; differences in activity attributed to reduced rates of degradation in the presence of *r(αGPDH)*⁺; no evidence for conformational differences in αGPDH. Not a general regulator of enzyme activity; however one rapidly migrating polypeptide is much more abundant in *r(αGPDH)* homozygotes than in the presence of *r(αGPDH)*⁺. Larvae carrying *r(αGPDH)*⁺ have 25% lower levels of αGPDH activity than *r(αGPDH)* homozygotes.

r(ADH)

location: 3- (between *h* and *th*).

origin: Naturally occurring allele.

synonym: *r*²⁶⁻⁴³.

references: King and McDonald, 1987, Genetics 115: 693-99.

phenotype: *r(ADH)* homozygotes exhibit approximately 70% of the level of alcohol dehydrogenase seen in heterozygotes and *r(ADH)*⁺ homozygotes; parallel differences in the levels of immunologically cross-reacting material (CRM) also observed. The effect of *r(ADH)* is constant over development and is not attributable to differences in charge, conformation, or rate of synthesis of ADH. Activities of GPDH and PGI levels insensitive to *r(ADH)* genotype.

ra: rasé

location: 3-97.3.

origin: Spontaneous.

discoverer: Beadle, 34d.

references: 1935, DIS 4: 10.

phenotype: Bristles and hairs small and irregularly absent, especially from head and thorax. Viability good; developmental time normal. RK2.

cytology: Associated with *ln(3R)P* (Craymer).

****ra*²**

origin: Spontaneous in *ln(3R)P*.

discoverer: Mossige, 36k21.

synonym: *bd: bald*.

references: 1937, DIS 8: 9.

phenotype: Homozygote lacks all head bristles and some scutellars. Heterozygote has extra anterior scutellars in about 30% of flies. RK2A.

cytology: Occurred in and probably inseparable from *ln(3R)P = ln(3R)89C2-4;96A18-19*.

****rab: rabbit***

location: 1-58.

origin: Induced by P³².

references: Bateman, 1950, DIS 24: 55.

phenotype: Hairs on mesonotum near dorsocentral bristles turned inward toward midline. Air bubbles occasionally in thorax, beneath dorsocentrals, and scutellum. Wings rarely held up. Viability and fertility normal. RK2(A).

other information: Slight disturbance of crossing over proximally.

rad201: radiation sensitive

location: 2-59.9.

synonym: *rad(2)201*^{G1}.

references: Levina, Malinovsky, and Zakharov, 1980, Genetika (Moscow) 16: 285-89.

Khromykh and Zakharov, 1981, Genetika (Moscow) 17: 658-66.

Khromykh, 1981, Genetika (Moscow) 17: 667-76.

Tikhomirova and Rasheva, 1983, Genetika (Moscow) 19: 628-34.

phenotype: Survival of homozygous larvae more sensitive to exposure to γ radiation than controls; chromosome- and chromatid-aberration induction increased over control but ratio of the two types same as control. X-ray-induced X-chromosome loss in immature oocytes greater in *rad201* than in control; mature oocytes show no effect of *rad201*. Post-treatment hyperthermia enhances the effect of irradiation in mature oocytes and control imma-

ture oocytes, but not in immature oocytes of *rad201*.

radioresistant: see *rar1*

radius incompletus: see *ri*

raf: see *phl*

rag: rags (*T. Schüpbach*)

location: 2-32.

origin: Induced by ethyl methanesulfonate.

references: Schupbach and Wieschaus, 1989, *Genetics* 121: 101-17.

phenotype: Maternal effect lethal; embryos from homozygous mothers form a fragmented cuticle with large ventral holes and head defects.

alleles: One: *rag^{QI}* = *rag¹*.

***rag: ragged**

location: 3-37 (Steinberg).

discoverer: Charles, 1932.

references: Dunn, 1934, *DIS* 1: 30.

phenotype: Hairs missing from sections of wing margin. RK3.

rags: see *rag*

***rai: raisin**

location: 3-17 (Stanley).

origin: Spontaneous.

discoverer: Hersh.

references: 1953, *DIS* 27: 55.

phenotype: Eye color deep brown, like *se*. Ecdysis delayed 1 or 2 days. RK2.

raised: see *rsd*

raised wing: see *rw*

raisin: see *rai*

ral: see *r-l*

rap: retina aberrant in pattern (*J.C. Hall*)

location: 1- {7.5}.

references: Karpilov, Kolodkin, Bork, and Venkatesh, 1989, *Genes Dev.* 3: 1834-44.

phenotype: Eyes grossly abnormal: rough surface texture, and deep pseudopupil (DPP) has anomalous appearance; finer level observations show ommatidia to be aberrant in size, shape, and alignment; in sections, numbers of photoreceptor cells per ommatidium vary greatly, and positions of such cells plus rhabdomere arrangements are altered. Developmentally, recruitment of presumptive photoreceptors behind morphogenetic furrow in third larval instar eye disc is abnormal: *e.g.*, arrangement of five-cell preclusters is very disorganized, and the normal increase in numbers of cells within clusters does not occur, so that, in regions where eight-cell clusters are found in wild-type, there are small clusters in *rap*, intermingled with others than can have >8 cells; cell bodies of developing photoreceptor cells do not become apically positioned in the disc, as in wild-type. Temperature-shift experiments on *rap⁴* show a temperature-sensitive period for roughened eye is in third larval instar. Mosaic experiments, involving induced mitotic recombination in *rap¹/+* larvae, showed that normal adult ommatidia always had a *rap⁺* R8 cell, whereas R1-7 could readily be genotypically *rap¹* in such facets; also, certain pheno-

typically mutant ommatidia, near borders separating mutant and wild-type tissue, contained all *rap⁺* cells (in clusters of 3, 4, 6, or 8 photoreceptor cells).

alleles:

allele	origin	discoverer	synonym	comments
<i>rap¹</i>	P			hypomorph?
<i>rap²</i>	P			hypomorph?
<i>rap³</i>	P			amorph
<i>rap⁴</i>	P		<i>rap^{R22ts}</i>	temperature-sensitive hypomorph ^α

^α Rearing at 17° leads to normal eye-surface morphology (albeit 36% penetrance of *rap¹*-like DPP phenotype); rearing at 29° causes all flies to have relatively mild eye roughening.

cytology: Maps to 4C7-16, based on its inclusion in *Df(1)rb¹³* = *Df(1)4C5-6;4D3-E1* but not *Df(1)rb¹* = *Df(1)3F6-4A1;4C7-8* or *Df(1)JC70* = *Df(1)4C15-16;5A1-2*.

other information: *rap¹* was shown to complement the nearby rough-eye mutation, *rg*.

Rap1: Ras3

location: 3- 1.4 (based on mapping of *R*).

origin: Isolated from genome library using v-Ha-ras probe.

synonym: *Dras3*, *Ras3*.

references: Neuman-Silberberg, Scheifer, Hoffmann, and Shilo, 1984, *Cell* 37: 1027-33.

cytology: Cloned sequence hybridizes to 62B.

molecular biology: Transcripts of 1.5, 1.9, and 2.9 kb detected (Lev, Kimchie, Hessel, and Segev, 1985, *Mol. Cell. Biol.* 5: 1540-1542); larger transcript present throughout development; shorter ones more abundant during embryogenesis.

other information: Isolated as a *Ras* homologue, but recently found to share high homology with human *Rap*.

rapid exhaustion: see *rex*

rar1: radioresistant

location: 1- (not mapped).

origin: Spontaneous in line selected for radioresistance.

references: Nöthel, 1980, *DIS* 55: 208-09.

Nöthel, 1981, *Mutat. Res.* 80: 105-20.

Nöthel, 1982, *Mutat. Res.* 103: 87-90.

Nöthel and Abdalla, 1982, *Mutat. Res.* 92: 123-32.

phenotype: Semidominant; reduces sensitivity of prestage-14 vitellarial oocytes to the dominant- and recessive-lethal-inducing effects of X irradiation or exposure to ethyl methanesulfonate. 1.3 X the dose to produce a given effect in controls required to produce the same effect in *radl* (*i.e.*, Dose Reduction Factor = 1.31). Effect of mutation inhibited by caffeine.

rar2

location: 2- (near centromere).

origin: Spontaneous in line selected for radioresistance.

references: Nöthel, 1980, *DIS* 55: 208-09.

Nöthel, 1981, *Mutat. Res.* 80: 105-20.

Nöthel, 1982, *Mutat. Res.* 103: 87-90.

phenotype: Semidominant; produces relative resistance of immature oocytes to X-ray and ethyl-methanesulfonate induction of dominant and sex-linked recessive lethals (Dose Reduction Factor = 1.31) and X-chromosome loss (Dose Reduction Factor = 1.72).

rar3

location: 3-49.8.

origin: Spontaneous in line selected for radioresistance.

references: Nöthel, 1980, DIS 55: 208-09.

Nöthel, 1981, Mutat. Res. 80: 105-20.

Nöthel, 1981, Mutat. Res. 84: 291-304, 305-13, 315-19.

Nöthel, 1982, Mutat. Res. 103: 87-90.

phenotype: Recessive mutant conferring resistance to the genetic effects of irradiation on oögonia and immature oocytes. Confers a dose reduction factor of 1.58 on the induction of dominant lethals, nondisjunction of major chromosomes and homologous exchange; and of 1.87 on the induction of sex-linked recessive lethals. Without effect on male germ line or somatic cells. Resistant to ethyl methanesulfonate as well.

ras: raspberry

location: 1-32.35 (Lefevre).

phenotype: Defective in pteridine synthesis; eye color dark ruby. Appears to affect the level of guanosine triphosphate cyclohydrolase activity; *ras* alleles exhibit increased activity compared to wild type at pupariation, but decreased activity at eclosion (Evans and Howell, 1979, Biochem. Genet. 16: 13-26). Not a purine auxotroph. Color autonomous in larval optic disks transplanted into wild-type hosts (Beadle and Ephrussi, 1936, Genetics 21: 230). *ras/ras¹* display *ras* eye color. Larval Malpighian tubes nearly wild type; not useful for classification (Brehme and Demerec, 1942, Growth 6: 351-56). *ras* is but one of four types of mutations belonging to an interrelated complex involved some way in purine metabolism: *ras*, a nonauxotrophic eye-color mutant; two purine auxotrophs, *pur1* and *gual*, which complement *ras* and one another; and *ras¹*, lethal alleles that fail to complement completely the other three types.

The complex shares genetic features with rudimentary.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>ras¹</i>	heat	Muller, 28d17		9	
<i>ras²</i>		Grossman, 1932		2	
<i>ras³</i>	spont	Ives, 37b18			eye color lighter than <i>ras¹</i> ;
<i>ras⁴</i>	spont	Ives, 38f			darkens with age
<i>ras⁵</i>	P	Green	<i>ras^{f2}</i>	3	female sterile
<i>ras⁶</i>	P	Green	<i>ras^{f26}</i>	3	
<i>ras⁷</i>	P	Green	<i>ras^{f26}</i>	3	
<i>ras⁸</i>	fast	Lewis, 1953	<i>ras^v</i>	1	variegated;
<i>ras¹¹</i>	neutron				<i>Tp(1;3)9E;13C;81F</i>
<i>ras¹²</i>	EMS	Grigliatti, 1970	<i>l(1)E6^{1S}</i>	4	temperature-sensitive lethal
<i>ras¹³</i>	X ray	Lefevre	<i>l(1)HC199</i>	6,7	lethal
<i>ras¹⁴</i>	X ray	Lefevre	<i>l(1)KC12</i>	7	lethal
<i>ras¹⁵</i>	X ray	Lefevre	<i>l(1)RC1</i>	7	lethal
<i>ras¹⁶</i>	X ray	Lefevre	<i>l(1)S27</i>	7	lethal
<i>ras¹⁷</i>	X ray	Lefevre	<i>l(1)S89</i>	7	lethal
<i>ras¹⁸</i>	EMS	Lefevre	<i>l(1)DA589</i>	6,8	lethal
<i>ras¹⁹</i>	EMS	Lefevre	<i>l(1)DC819</i>	6,8	lethal
<i>ras¹⁰</i>	EMS	Lefevre	<i>l(1)DF955</i>	6,8	lethal
<i>ras¹¹</i>	EMS	Lefevre	<i>l(1)EA140</i>	6,8	lethal
<i>ras¹²</i>	EMS	Lefevre	<i>l(1)VA212</i>	6,8	lethal
<i>ras¹³</i>	EMS	Lefevre	<i>l(1)VD224</i>	6,8	lethal
<i>ras¹⁴</i>	EMS	Lefevre	<i>l(1)VE432</i>	6,8	lethal
<i>ras¹⁵</i>	EMS		<i>l(1)D12</i>	5,10	lethal
<i>ras¹⁶</i>	EMS		<i>l(1)F1</i>	5,10	lethal ^β
<i>ras¹⁷</i>	EMS		<i>l(1)F19</i>	5,10	lethal ^β
<i>ras¹⁸</i>	EMS		<i>l(1)H6</i>	5,10	lethal
<i>ras¹⁹</i>	EMS		<i>l(1)H25</i>	5,10	lethal
<i>ras²⁰</i>	EMS		<i>l(1)J5</i>	5,10	haplo-specific

allele	origin	discoverer	synonym	ref ^α	comments
<i>ras¹²⁰</i>	EMS		<i>l(1)K27</i>	5,10	lethal ^γ
<i>ras¹²¹</i>	EMS		<i>l(1)N23</i>	5,10	lethal

^α 1 = Brokaw, 1954, DIS 28: 73; 2 = Dunn, 1934, DIS 1: 30; 3 = Green, 1977, Proc. Nat. Acad. Sci. USA 74: 3490-93; 4 = Grigliatti and Suzuki, 1970, Proc. Nat. Acad. Sci. USA 67: 1101-08; 5 = Janca, Woloshyn, and Nash, 1986, Genetics 112: 43-64; 6 = Johnson, Woloshyn, and Nash, 1979, Mol. Gen. Genet. 174: 287-92; 7 = Lefevre 1981, Genetics 99: 461-80; 8 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 9 = Muller, 1935, DIS 3: 30; 10 = Nash and Janca, 1983, Genetics 105: 957-86.

^β Complements *ras²* eye color but not lethality of other *ras¹* alleles.

^γ Survives weakly as homozygous female, but completely lethal as male and hemizygous female.

cytology: Placed in 9E1-4 on the basis of its inclusion in the region of overlap between *Df(1)ras-P14* = *Df(1)9E1-2;9F3-4* and *Dp(1;2)9E1;10A11;56A* (Zhimulev, Pokholkova, Bgatov, Semeshin, and Belyaeva, 1981, Chromosoma 82: 25-40).

Ras1: Ras proto-oncogene sequence

location: 3- {49}.

origin: Isolated from genomic library using v-Ha-ras probe.

synonym: *Dras1*.

references: Neuman-Silberberg, Scheifer, Hoffmann, and Shilo, 1984, Cell 37: 1027-33.

phenotype: Codes for a polypeptide of 21.6 kilodaltons; residues 1-121 and 137-64 exhibit 75% homology with vertebrate H-ras protein.

alleles: Loss-of-function mutations are homozygous lethal; enhance *sev* and *Egfr^E* in heterozygotes (M. Simon).

cytology: Cloned sequence hybridizes to 85D.

molecular biology: Gene cloned and sequenced. Contains four introns. Open reading frame in cDNA codes for 189 amino acids corresponding to a polypeptide of 21 kilodaltons. Transcripts of 1.3 and 2.0 kb detected by Lev, Kimchie, Hessel, and Segev (1985, Mol. Cell. Biol. 5: 1540-42); the larger persists throughout development, the smaller being primarily in unfertilized eggs and embryos.

Ras2

location: 3- {15}.

origin: Isolated from genome library using v-Ha-ras probe.

synonym: *Dras2, Dmras64B*.

references: Neuman-Silberberg, Scheifer, Hoffmann, and Shilo, 1984, Cell 37: 1027-33.

Moser, Marlör, Parkhurst, and Corces, 1985, Mol. Cell. Biol. 5: 885-89.

phenotype: Encodes a 22.7 kilodalton, 195 amino acid, polypeptide, p21.

cytology: Cloned sequence hybridizes to 64B.

molecular biology: Entire gene sequenced. Contains two more amino-terminal codons than the homologous gene from humans and four fewer than the yeast gene. *Drosophila*, human, and yeast sequences highly homologous up to amino-acid residue 90, less so for the next 80 amino acids, and not at all for residues 170-190. *Ras2* contains two introns in positions different from those in the homologous human gene. Appears to produce transcripts of 1.6, 2.1, and 2.6 kb, which are present in the same proportions throughout development (Wadsworth, Madhavan, and Bilodeau-Wentworth, 1985, Nucl. Acids

Res. 13: 2153-70; Mozer, Marlo, Parkhurst, and Corces, 1985, Cell. Biol. 5: 885-89). Claimed by Lev, Kimchie, Hessel, and Segev (1985, Mol. Cell. Biol. 5: 1540-42) to produce transcripts of 1.4 and 1.8 kb, the larger of which is expressed at all stages and the smaller in unfertilized eggs and early embryos.

Ras3: see *Rap1*

Ras proto-oncogene sequence: see *Ras1*

rasé: see *ra*

rasberry: see *ras*

rasberry-lethal: see *ras-l*

rauhig: see *gl*³

raven: see *rv*

raw: raw

location: 2-19.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Homozygous lethal; dorsal closure and cuticle differentiation defective.

alleles: *raw*¹ and *raw*² recovered as IG and IIF.

rb: ruby

location: 1-7.5.

discoverer: Bridges, 14j18.

phenotype: Eye color clear ruby, white in combination with *w*^a, orange with *st*, and brownish red with *bw* (Mainx, 1938, Z. Indukt. Abstamm. Vererbungsl. 75: 256-76). Development of pigment autonomous in *rb* eye disks transplanted into wild-type hosts (Beadle and Ephrussi, 1936, Genetics 21: 230). Larval Malpighian tubes pale yellow (Beadle, 1937, Genetics 22: 587-611). RK1.

alleles:

allele	origin	discoverer	ref ^α
<i>rb</i> ¹		Bridges, 14j18	
<i>rb</i> ^{48a}	X ray	Fox, 48a7	2, 3
<i>rb</i> ^{64f}	X ray	Ives, 64f14	
<i>rb</i> ^{66a}	X ray	Becker	1
<i>rb</i> ^{m48}	X ray		5
<i>rb</i> ^{ntg}	NNG	Kaufman	4

^α 1 = Becker, 1968, DIS 43: 59; 2 = Fox, 1948, DIS 22: 53; 3 = Fox, 1949, Genetics 34: 647-64; 4 = Kaufman, 1970, DIS 45: 34; 5 = Valencia, 1966, DIS 41: 58.

cytology: Located in 4C6 based on the overlap of *Df(1)rb13 = Df(1)4C5-6;4D3-E1* and *Df(1)rb46 = Df(1)4A3-6;4C6-7* (Banga, Bloomquist, Brodberg, Pye, Larrivee, Mason, Boyd, and Pak, 1983, Chromosoma 93: 341-46).

rbc: see *rc*

rb1: reduced bristle

location: 2-82.3.

origin: Spontaneous; natural population.

references: Watanabe, 1969, Jpn. J. Genet. 44: 15-22.

Watanabe and Oshima, 1970, Genetics 64: 93-106.

phenotype: Homozygotes show reduced dorsal bristles; both sexes fertile, but development delayed by 1.5 days

and viability reduced.

rbp: see *BRC*

rc: red cells

location: 2-36.8 (between *d* and *J*).

origin: Spontaneous.

discoverer: E. B. Lewis, 1946.

synonym: *rbc*: red blood cells.

references: 1950, DIS 24: 59.

Jones and Lewis, 1957, Biol. Bull. 112: 220-24 (fig.).

Grell, 1961, Genetics 46: 925-33.

phenotype: *rc/rc* normal; in *lys rc/lys rc*, fat cells of head and thorax acquire brownish red pigment. Effect most prominent in one or more rows of pigmented cells along mid-dorsal line of thorax just beneath chitin. Pigment is ommochrome since *lys rc bw* cells are pigmented, whereas *v; lys rc* cells are colorless except in kynurenine-fed flies. RK3.

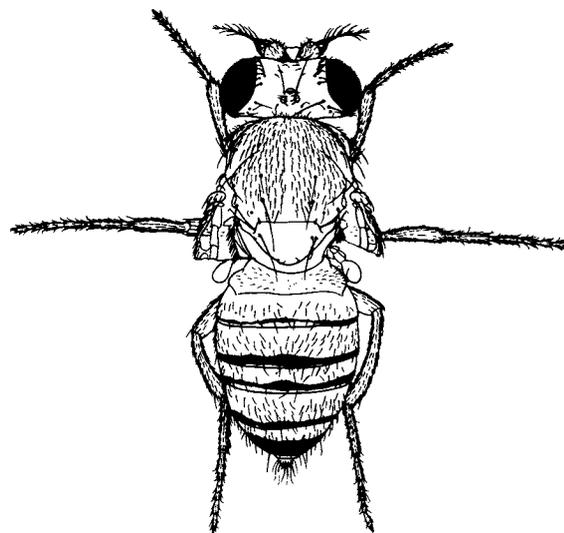
rc²

origin: Spontaneous.

discoverer: R. F. Grell, 1957.

references: Grell, 1961, Genetics 46: 925-33.

phenotype: Wild type at 25° on standard medium; at 17°, a few red fat cells are visible. Early third instar larvae placed on glucose-agar medium produce flies with numerous red cells. *lys rc²* has red cells under any conditions. RK3.



rd: reduced

From Bridges and Brehme, 1944, Carnegie Inst. Washington Publ. No. 552: 157.

rd: reduced

location: 2-51.2.

origin: Spontaneous.

discoverer: Bridges, 17g15.

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 233-35.

phenotype: Bristles reduced in length and thickness, sometimes twisted; best seen on sternopleurals; male more extreme than female (*rd*¹); female may have abnormal abdominal banding (*rd*^s). Males fertile, but females may

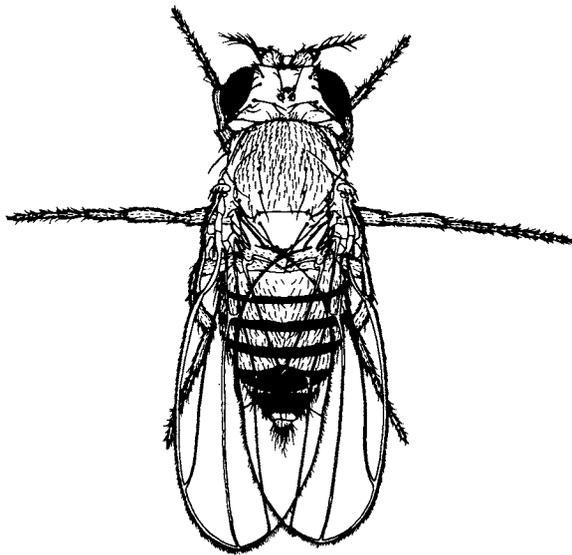
be sterile (*rd*¹) (Lynch, 1919, Genetics 4: 501-33).
alleles:

allele	origin	discoverer	ref ^α	comments
<i>rd</i> ¹	spont	Bridges, 17g15	2	
<i>rd</i> ⁴	γ ray		1	on <i>Sco</i>
<i>rd</i> ⁷	γ ray		1	on <i>Sco</i>
<i>rd</i> ⁹	γ ray		1	on <i>Sco</i> β
<i>rd</i> ^s	spont	Bridges, f8j2	2	reduced-scraggly

^α 1 = Angel, Ashburner, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186; 2 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 233-35.

^β *rd*⁹ is lethal in combination with *Df(2L)osp18* and *Df(2L)TE35BC-3* but not with *Df(2L)nNXF1* or *Df(2L)A72*; complemented by all known lethal mutants in the region.

cytology: Placed in 35C2-5 based on its inclusion in *Df(2L)osp18* = *Df(2L)35B1-2;35C4-5* but not *Df(2L)el81i1* = *Df(2L)34E1-2;35C2-3*; also claimed to lie within deficiencies that include bands from 35B but not 35C [*Df(2LA215; Df(2L)A400, Df(2L)Sco-1*].



***rd*^s: reduced-scraggly**

Edith M. Wallace, unpublished.

RD(1): Recovery Disrupter (1)

location: 1-62.9 [10% of the distance between *car* and *su(f)*].

origin: Found in a chronically irradiated population obtained from B. Wallace.

discoverer: Hanks, 1957.

references: Novitski and Hanks, 1961, Nature 190: 989-90.

Erickson and Hanks, 1961, Am. Naturalist 95: 247-50.

Hanks, 1964, Genetics 50: 123-30.

phenotype: Males containing this factor, *RD(2)*, and certain other factors produce approximately 67% female and 33% male progeny. The effect is not produced by zygotic mortality but by a mechanism that operates during meiosis, leading to fragmentation of the *Y* chromosome and production of fewer than 64 sperm heads per sperm bundle (Erickson, 1965, Genetics 51: 555-71). The effect is maximal at 25° and less pronounced at both

18° and 27°. Viability good but fertility reduced in both sexes. RK3.

RD(2)

location: 2- (not located).

origin: Found in a chronically irradiated population obtained from B. Wallace.

discoverer: Hanks, 1960.

references: Novitski and Hanks, 1961, Nature 190: 989-90.

phenotype: Males with this factor, *RD(1)*, and certain other factors produce about 67% female progeny. RK3.

****rdb: reddish brown***

location: 1-21.7.

origin: Induced by methyl methanesulfonate (CB. 1540).

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 89.

phenotype: Eye color deep reddish brown. Wings frequently curve slightly upward at tips. Body somewhat small. Male sterile. Viability about 30% wild type. RK3.

rde: reduced eye

location: 1-20 (inferred from Fig. 1 of Homyk and Shepard and the known map position of *hypoB*).

origin: Induced by ethyl methanesulfonate.

references: Homyk and Sheppard, 1977, Genetics 87: 95-104.

Homyk, 1977, Genetics 87: 105-28.

phenotype: Selected on basis of reduced ability to fly; other behavioral traits normal; also exhibits reduced eye size and frequently missing palpi.

rdgA: retinal degeneration A (J.C. Hall)

location: 1-26.3.

origin: Induced by ethyl methanesulfonate.

synonym: *receptor degeneration-1* (Hotta and Benzer, 1970); *x35* (Pak).

references: Hotta and Benzer, 1970, Proc. Nat. Acad. Sci. USA 67: 1156-63.

Harris and Stark, 1977, J. Gen. Physiol. 69: 261-91.

Johnson, Frayer, and Stark, 1982, J. Insect Physiol. 28: 23-42.

Stark and Carlson, 1985, Int. J. Insect Morphol. Embryol. 14: 243-54.

phenotype: Photoreceptors degenerate during first week of adult life, although electroretinogram is small at time of eclosion when photoreceptors are morphologically normal; exception: *rdg*¹ which is already aberrant then (Stark and Carlson, 1985); degeneration of outer photoreceptors (R1-6) in each eye facet is more severe (*i.e.*, is essentially complete) than that of inner two cells (R7,8), with the most severe central-cell degeneration caused by *rdgA*¹ and *rdgA*², *rdgA*⁴ being less severe, and *rdg*³ still less (Harris and Stark, 1977); the ocelli degenerate, too, with *rdgA*¹ causing particularly severe effect (Johnson *et al.*, 1982); degeneration of compound eye photoreceptors is not light- or temperature-dependent (Harris and Stark, 1977); such degeneration is photoreceptor-autonomous in mosaics (Hotta and Benzer, 1970; Harris and Stark, 1977); degenerating photoreceptor axon terminals, especially those projecting from R1-6 into the lamina optic ganglion, are phagocytosed by glia in that optic lobe (Stark and Carlson, 1985); in general, though, the gross and internal structure of the lamina seems quite

normal, even after photoreceptor degeneration caused by most severe *rdgA* alleles is complete (Meyerowitz and Kankel, 1978, Dev. Biol. 62: 112-42; Johnson *et al.*, 1982; Stark and Carlson, 1985); two polypeptide spots in 2-d gel analysis of eye-specific proteins are reduced in intensity under influence of *rdgA*⁴ [Hotta, 1979, Mechanisms of Cell Change (Ebert and Okada, eds.). John Wiley, New York, pp. 169-82]; *rdgA* mutations said to cause hypoactive behavior, and one of them also leads to shaking on exposure to ether plus premature death after exposure to 29° (Homyk, Pye and Pak, 1981, Genetics 97: s50).

cytology: Placed in 8A4-C6 based on its inclusion in *Df(1)KA14 = Df(1)7F1-2;8C6* but not *Df(1)RA2 = Df(1)7D10;8A4-5*.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>rdgA</i> ¹	EMS		<i>rdgA</i> ^{BS12}	4	
<i>rdgA</i> ²	EMS		<i>rdgA</i> ^{KO14}	4	
<i>rdgA</i> ³	EMS		<i>rdgA</i> ^{PC47}	4	
<i>rdgA</i> ⁴	EMS		<i>rdgA</i> ^{KSI99}	1	
<i>rdgA</i> ⁵	EMS	Benzer			
<i>rdgA</i> ⁶	EMS		<i>rdgA</i> ^{P35}	3	
<i>rdgA</i> ⁷	EMS		<i>rdgA</i> ^{P36}	3	
<i>rdgA</i> ⁸	EMS		<i>rdgA</i> ^{P38}	3	
<i>rdgA</i> ⁹	EMS		<i>rdgA</i> ^{P59}	3	
<i>rdgA</i> ¹⁰	EMS		<i>rdgA</i> ^{P63}	3	
<i>rdgA</i> ¹¹	EMS		<i>rdgA</i> ^{P65}	3	
<i>rdgA</i> ¹²	EMS	Heisenberg	<i>opm3</i>	2	
<i>rdgA</i> ¹³	EMS	Heisenberg	<i>opm4</i>	2	
<i>rdgA</i> ¹⁴	EMS	Heisenberg	<i>opm5</i>	2	
<i>rdgA</i> ¹⁵	EMS	Heisenberg	<i>opm9</i>	2	
<i>rdgA</i> ¹⁶	EMS	Heisenberg	<i>opm10</i>	2	
<i>rdgA</i> ¹⁷	EMS	Heisenberg	<i>opm14</i>	2	
<i>rdgA</i> ¹⁸	EMS	Heisenberg			
<i>rdgA</i> ¹⁹	EMS	Heisenberg			
<i>rdgA</i> ²⁰	EMS	Heisenberg			
<i>rdgA</i> ²¹	P	Hardy			

^α 1 = Harris and Stark, 1977, J. Gen. Physiol. 69: 261-91; 2 = Heisenberg, 1971, DIS 46: 68; 3 = Homyk, Pye, and Pak, 1981, Genetics 97: s50; 4 = Hotta and Benzer, 1970, Proc. Nat. Acad. Sci. USA 67: 1156-63.

***rdgB* (J.C. Hall)**

location: 1-42.7.

origin: Induced by ethyl methanesulfonate.

synonym: *receptor degeneration II* (Hotta and Benzer, 1970); *x36* (Pak).

references: Hotta and Benzer, 1970, Proc. Nat. Acad. Sci. USA 67: 1156-63.

Harris and Stark, 1977, J. Gen. Physiol. 69: 261-91.

Stark and Carlson, 1982, Cell Tissue Res. 225: 11-22.

Stark, Chen, Johnson, and Frayer, 1983, J. Insect Physiol. 29: 123-31.

Stark and Carlson, 1985, DIS 61: 162-64.

phenotype: Photoreceptors in each facet of compound eye show light-induced degeneration; morphology is essentially normal on eclosion, but maintenance of mutant adults on diurnal light regime causes severe degeneration within approximately one week (Harris and Stark, 1977); the cell bodies and axons of photoreceptors begin to look ultrastructurally abnormal after three days (Stark and Carlson, 1982); the various *rdgB* mutations tend to cause the outer photoreceptors (R1-6) in each facet to degenerate more than the inner two cells (R7,8), such that *rdgB*⁹ and *rdgB*¹ have R7,8 preserved in nearly all ommatidia, *rdgB*⁶ and *rdgB*⁸ retain most central cells,

and *rdgB*⁷ plus *rdgB*⁵ show progressively worse degeneration of R7,8 with *rdgB*⁵ retaining these cells in only 10% of the facets according to Harris and Stark (1977); degeneration of R7,8 not confirmed, however, by Stark *et al.* (1983). After R1-6 have degenerated and the central cells have or have not, depending on the allele, R7,8 retain at least quasi-normal function as indicated by electroretinograms (Stark, 1977, J. Comp. Physiol. 115: 47-59); degeneration is photoreceptor autonomous in mosaics (Hotta and Benzer, 1970; Harris and Stark, 1977); autonomous in mixed ommatidia at the electron-microscope level (Hofbauer and Campos-Ortega, 1976, Wilhelm Roux's Arch. Dev. Biol. 179: 275-89); ultrastructural details of degeneration include electron-dense cytoplasm with liposomes, lysosome-like bodies, myeloid bodies and vacuoles, and electron-dense reticulum and degenerate mitochondria, electron opaque photoreceptor axons lacking synaptic vesicles and containing seemingly none of the typical presynaptic structures (Stark and Carlson, 1982). Degenerating photoreceptors are associated with degeneration in the first order optic ganglion (the lamina), though the second order medulla is spared (Stark *et al.*, 1983); high temperature treatments accelerate degeneration, whereas homozygosity for an *AcpH-1*-null mutation delays it slightly (Harris and Stark, 1977). After rearing of *rdgB* animals in room light, the R1-6 cells are physiologically non-functional at eclosion (revealed by ERGs), in spite of apparently normal cellular structure (Harris and Stark, 1977). Prolonged depolarizing afterpotentials (induced by strong blue light) are abnormally short-lived and cannot be reversed by orange light, unlike the response of wild-type (Harris and Stark, 1977); degeneration of the photoreceptors is dramatically retarded by rearing in the dark followed by maintenance of adults under this condition (Harris and Stark, 1977); the same retardation of the mutations' effects occur when an *rdgB* mutation is linked to an ERG-minus *norPA* mutation (Harris and Stark, 1977), although degeneration does eventually occur in the double mutants (Stark *et al.*, 1983); three *norPA* mutations were induced based on their inhibition of *rdgB*-induced degeneration (Harris and Stark, 1977), with two of the new mutations leading to very small ERGs but the third, *norPA*^{sull}, allowing for apparently normal retinal physiology (also see Stark *et al.*, 1983); this mutation revealed as a *norPA* allele, based on uncoverage of its degeneration-suppressing effects by an ERG-minus *norPA* mutation (Harris and Stark, 1977); *norPA*⁵⁶'s suppressing effects are allele-specific, to the extent that *rdgB*⁹-induced degeneration is retarded (and this was the *rdgB* allele used to isolate this suppressor), but the effects of another allele, *rdgB*¹, are not (Harris and Stark, 1977); *ort*¹ *ninaE*¹ (formerly *ora*^{JK84}), an opsin-deficient genotype, also blocks light induced degeneration of receptor cells in *rdgB*⁹ (Stark and Carlson, 1985, DIS 61: 162-64). Two *rdgB* alleles (unspecified) are associated with premature death of adults exposed to 29° (Homyk, Pye and Pak, 1981, Genetics 97: s50); further indications of pleiotropic action of this gene come from isolation of a hypoactive *rdgB* allele [originally *hypoF* (Homyk, Szidonya and Suzuki, 1980, Mol. Gen. Genet. 177: 553-65)]; this mutation (mapping between *v* and *f*, as does *rdgB*) was shown to be an *rdgB* allele by Homyk and is now called *rdgB*²³; it causes adults to be somewhat inactive in their

general movements and their jumping ability to be weak; there are no light-on or light-off transient spikes in the ERG, and optomotor responses are eliminated; two polypeptide spots observed in two-dimensional gel analysis of eye-specific proteins are reduced in intensity in an *rdgB* mutant -- the same spots as affected by *rdgA* [(Hotta, 1979, Mechanisms of Cell Change (Ebert and Okada, eds.). John Wiley, New York, pp. 169-82)].

cytology: Placed in 12A6-D3 based on its inclusion in *Df(1)HA92 = Df(1)12A6-7;12D3* (Deak, Bellamy, Bienz, Dubuis, Fenner, Gollin, Rähmni, Ramp, Reinhardt, and Cotton, 1982, J. Embryol. Exp. Morphol. 69: 61-81).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>rdgB</i> ¹	EMS		<i>rdgB</i> ^{KO45}	5	
<i>rdgB</i> ²	EMS	Benzer			
<i>rdgB</i> ³	EMS	Benzer			
<i>rdgB</i> ⁴	EMS	Benzer			
<i>rdgB</i> ⁵	EMS		<i>rdgB</i> ^{EE170}	1	
<i>rdgB</i> ⁶	EMS		<i>rdgB</i> ^{KS16}	1	
<i>rdgB</i> ⁷	EMS		<i>rdgB</i> ^{KS100}	1	
<i>rdgB</i> ⁸	EMS		<i>rdgB</i> ^{KS200}	1	
<i>rdgB</i> ⁹	EMS		<i>rdgB</i> ^{KS222}	1	
<i>rdgB</i> ¹⁰	EMS		<i>rdgB</i> ^{P6}	3	
<i>rdgB</i> ¹¹	EMS			3	
<i>rdgB</i> ¹²	EMS			3	
<i>rdgB</i> ¹³	EMS			3	
<i>rdgB</i> ¹⁴	EMS	Heisenberg	<i>rdgB</i> ^{H6}		
<i>rdgB</i> ¹⁵	EMS	Heisenberg	<i>rdgB</i> ^{H11}		
<i>rdgB</i> ¹⁶	EMS	Heisenberg	<i>rdgB</i> ^{H12}		
<i>rdgB</i> ¹⁷	EMS	Heisenberg			
<i>rdgB</i> ¹⁸	EMS	Heisenberg			
<i>rdgB</i> ¹⁹	EMS	Heisenberg			
<i>rdgB</i> ²⁰	EMS	Heisenberg			
<i>rdgB</i> ²¹	EMS	Heisenberg			
<i>rdgB</i> ²²	EMS	Heisenberg			
<i>rdgB</i> ²³	EMS		<i>rdgB</i> ¹⁰¹	4	
<i>rdgB</i> ²⁴	EMS	Gerresheim	<i>rdgB</i> ^{hypoF} <i>rdgB</i> ^{a31}	2	

^α 1 = Harris and Stark, 1977, J. Gen. Physiol. 69: 261-91; 2 = Gerresheim, 1988, Behav. Genet. 18: 222-46; 3 = Homyk, Pye, and Pak, 1981, Genetics 97: s50; 4 = Homyk, Szidonya, and Suzuki, 1980, Mol. Gen. Genet. 177: 553-65; 5 = Hotta and Benzer, 1970, Proc. Nat. Acad. Sci. USA 67: 1156-63.

other information: *rdgB* mutations, especially those causing R1-6-specific degeneration (notably *rdgB*⁹), frequently used in experiments aimed at assessing behavioral and physiological significance of light input through central photoreceptors only; hence, phototaxis mediated by R7,8 (Jacob, Willmund, Folkers, Fischbach and Spatz, 1977, J. Comp. Physiol. 118: 261-71; Broda and Willmund, J. Insect Physiol. 27: 789-92; Hu and Stark, 1980, J. Comp. Physiol. 135: 85-95; Miller, Hansen and Stark, 1981, J. Insect Physiol. 27: 813-19); optomotor responses eliminated by degeneration of R1-6 (Heisenberg and Buchner, 1977, J. Comp. Physiol. 117: 127-62); visual learning not impaired; intensity discrimination less acute than in wild type; no positive indication of color discrimination (Bicker and Reichert, 1978, J. Comp. Physiol. 127: 29-38). Visual pigment specific to outer photoreceptors (Harris, Stark and Walker, 1976, J. Physiol. 256: 415-39); absence of physiological effects of *rdgB* on ocelli (Hu, Reichert and Stark, J. Comp. Physiol. 126: 15-24); and physiological effects of a *trp* mutation on the central photoreceptors (Chen and Stark, 1983, J. Insect Physiol. 29: 133-40).

***rdm: reduced macros**

location: 1-59.8.
origin: Induced by 2-fluoroethyl methanesulfonate (CB. 1522).
discoverer: Fahmy, 1957.
references: 1959, DIS 33: 89.
phenotype: Most bristles thin and short. Eye shape slightly abnormal. Body short; wings short, broad, and frequently pleated. Male fertile. Viability about 10% wild type. RK3.

rdο: reduced ocelli

location: 2-53.
origin: Spontaneous.
discoverer: E. M. Wallace, 37113.
phenotype: Ocelli small and colorless, often missing, leaving top of head smooth and sometimes pigmented. Hairs between ocelli fewer than wild type. Eye surface irregular. RK2.
alleles: *rdο*², spontaneous, Bridges, 38b10.
cytology: Placed in 36E4-F1 based on its inclusion in the region of overlap of *Df(2L)TW203 = Df(2L)36E4-F1;37B9-C1* and *Df(2L)H20 = Df(2L)36A8-9;36E4-F1* (Wright).

***rdp: reduplicated**

location: 1-34.7.
discoverer: Hoge-Richards, 12k.
references: Hoge, 1915, J. Exp. Zool. 18: 241-97.
phenotype: At low temperatures, most flies have malformed or branched legs, often with mirror image reduplication. At 25°, most flies normal. RK3.

***rdt: reduced thorax**

location: 1-54.4.
origin: Induced by *p*-N,N-di-(2-chloroethyl)amino-phenylethylamine (CB. 3034).
discoverer: Fahmy, 1955.
references: 1959, DIS 33: 89.
phenotype: Head and thorax disproportionately small compared to abdomen. Wings short, reaching only to tip of abdomen; frequently incompletely expanded or misheld. Male shows reduced viability and usually sterile. RK3.

re: reduced eyes

location: 3- (not located).
origin: Spontaneous.
discoverer: Rapoport.
references: 1940, Dokl. Acad. Nauk SSSR 27: 1030-32.
phenotype: Eye size reduced from the normal 750 to about 180 facets. Reduction more extreme in combination with *B*; some flies have no facets and are sterile. RK2.

re: see *rey*

***re-b: reduced eyes-b**

location: 3-45.
origin: Spontaneous.
discoverer: Whittinghill, 53g.
references: Schacht, 1954, DIS 28: 78.
phenotype: Eyes reduced in 80% of homozygotes. Expression varies independently in each eye from absence of facets to wild type. RK2.
other information: Possibly allelic to *re*.

rea: rearranged tergites**location:** 1-25.4.**origin:** Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).**discoverer:** Fahmy, 1954.**references:** 1958, DIS 32: 73.**phenotype:** Tergites highly abnormal, partly missing, and have different segments united. Expression variable. Viability and fertility inversely related to tergite abnormality. RK2.**other information:** One allele induced by CB. 3025.**rec: recombination defective (R.F. Grell)****location:** 3-58 (between 57.0 and 58.2).**references:** Grell, 1978, Proc. Nat. Acad. Sci. USA 75: 3351-54.

Grell and Generoso, 1980, Chromosoma 81: 339-48.

Grell, 1984, Genetics 108: 425-33.

Grell, 1985, Aneuploidy (Dellarco, Voytek, and Hollaender, eds.). Plenum Press, New York and London, pp. 317-36.

phenotype: Recombination in homozygous females drastically reduced; total cross over between *y* and *f* in the *X* chromosome reduced from 56% to 2% with distal recombination being more severely affected than proximal. *X* nondisjunction increased from virtually 0 to 27%. In *rec*¹/*rec*², females show a similar reduction for ten regions between *al* and *sp*, where crossing over decreases to 9.1%, corresponding to 9% of normal. The number of progeny per homozygous female is reduced 75%; attributed to lethality of nondisjunctional products arising from distributive pairing between noncrossover heterologues. Phenotype of *rec*/+ is the same as wild-type; *rec*¹ and *rec*² homozygotes and hemizygotes have equivalent effects on recombination.**alleles:**

allele	origin	discoverer	synonym	comments
<i>rec</i> ¹	EMS	R.F. Grell & E.E. Generoso, 72k	<i>rec-1</i> ⁶	null allele
<i>rec</i> ²	EMS	R.F. Grell & E.E. Generoso, 74d	<i>rec-1</i> ¹⁶	null allele
<i>rec</i> ³	EMS	R.F. Grell & E.E. Generoso, 74i	<i>rec-1</i> ²⁶	temperature sensitive allele ^α

^α See below for description.**cytology:** Placed in 88F9-89B4 based on its inclusion in *Df(3R)sbd105* = *Df(3R)88F9-89A1;89B9-10* (Lewis, 1948, DIS 22: 72-73) but not *Df(3R)sbd45* = *Df(3R)89B3-4;89B10-11* (Grell, 1984, Genetics 108: 425-43).**other information:** Not allelic to *c(3)G*, since *rec* and *c(3)G* fully complement, also unlike *c(3)G*, *rec* has no discernable effect on the synaptonemal complex.**rec³****phenotype:** At 17° recombination in *rec*³/+, *rec*³/*rec*³, and *rec*³/*Df(3R)sbd105* is elevated. At 25°, the homozygote and the heterozygote display normal values, but recombination in the hemizygote is significantly reduced. At 31°, *rec*³/+ remains normal but *rec*³/*rec*³ recombination is drastically reduced. The hemizygote is sterile above 28°. In *rec*²/*rec*³ females which show the most extreme temperature responses, application of the restrictive temperature at sequential days during development at 25° shows control value activity to be reduced only withtreatment on days six and seven, and more precisely during a 36-hour period beginning at the time of pro-oocyte formation between 126 and 132 hours and terminating at 162 hours. This 36-hour period coincides with the boundaries of the S period as well as those of the heat-sensitive period for enhancing recombination in the normal genotype [Grell and Day, 1974, Mechanisms in Recombination (R. F. Grell, ed.). Plenum Press, New York and London, pp. 327-49]. Activity of the *rec* gene product in the range of 17° to 31° shows a sharp decline between 28° and 31° typical of a protein denaturation curve. If denaturation of a *rec* protein by the restrictive temperature marks its active phase, then recombination must terminate at the end of S, when the restrictive temperature becomes ineffective. Electron microscopy of serially sectioned oocyte nuclei from *rec*²/*rec*³ females maintained at the restrictive or at the permissive temperature reveals synaptonemal complexes indistinguishable in length and fine structure, implicating recombination and not synapsis as the target of the *rec* gene product.**Receptor:** see *Rsp***receptor degeneration:** see *rdg***recombination defective:** see *rec***Recovery Disrupter:** see *RD***red: red Malpighian tubules****location:** 3-53.6.**origin:** Spontaneous.**discoverer:** Muller, 49a.**synonym:** *bw-l*: brown-like.**references:** Oster, 1954, DIS 28: 77-78.

Aslaksen and Hadorn, 1957, Arch. Julius Klaus-Stift. Vererbungsforsch. Sozialanthropol. Rassenhyg. 32: 464-69.

phenotype: Malpighian tubes of larva and adult rusty red. Eye color brown, darkening with age. *kar*², *red* flies have orange eyes (Henikoff, 1979, Genetics 93: 105-15); *cn*; *red* colorless (Paton and Sullivan, 1975, Genetics 80: s63). Malpighian tubes of *v*; *red* and *cn*; *red* are colorless and tubes of *bw*; *red* are red; therefore, pigment an ommochrome. Eyes contain less drosoperin and isoxanthopterin and more of the other pteridines than normal. Eye color autonomous in *red* eye disks transplanted into wild-type host. Wild-type Malpighian tubes acquire some red pigment after transplantation into *red* hosts. RK1.**cytology:** Placed in 88B1-3 based on its inclusion in *Df(3R)red21* = *Df(3R)88B1-2;88B2-3* (Spillman and Nöthiger, 1978, DIS 53: 163).**red blood cells:** see *rc***red cells:** see *rc***red Malpighian tubules:** see *red***red wine:** see *rwi***reddish brown:** see *rdb***reduced:** see *rd***reduced bristle:** see *rbl***reduced eye:** see *rde*

reduced eyes: see *re*

reduced macros: see *rdm*

reduced mushroom body: see *mbmB*

reduced ocelli: see *rdo*

reduced optic lobes: see *rol*

reduced pigment: see *rgt*

reduced size: see *rsi*

reduced tarsi: see *rta*

reduced thorax: see *rdt*

reduplicated: see *rdp*

reduplicated sex combs: see *rsc*

ref: refractaire

Mutants at several loci that interfere with the normal multiplication of some strains of sigma virus, as indicated by delayed acquisition of CO₂ sensitivity following viral injection. They are designated *ref* followed by parenthetical indication of the chromosome on which they reside and then a locus designating letter; the permissive or normal allele is designated by superscript *o* indicating standard allele from Oregon-R; restrictive alleles are superscripted with a letter indicating origin: *b* = Belinga; *e* = ebony stocks; *h* = Hikone; *m* = Moiré stock; *n* = Nagasaki; *p* = Paris. Except for the case of *ref(3)O*, in which the restrictive allele is dominant, permissive and restrictive alleles are codominant.

locus	genetic location	restrictive allele	ref ^α	comments ^β
ref(1)H	1-14	<i>ref(1)H^h</i> <i>ref(1)H^b</i>	1, 2	viral replication
ref(2)M	2-40	<i>ref(2)M^m</i>	1, 2	viral replication
ref(2)P	2-54.0	<i>ref(2)P^p</i> <i>ref(2)Pⁿ</i>	1, 2, 4	viral replication
ref(3)D	3-90	<i>ref(3)D^p</i>	1, 2	viral replication
ref(3)O	3-69.8	<i>ref(3)O^e</i>	1, 2, 3	viral maturation
ref(3)V	3-96.7	<i>ref(3)V^p</i>	1, 2	viral replication in male germ line

^α 1 = Brun and Plus, 1980, Genetics and Biology of *Drosophila* (Ashburner and Wright, eds.). Academic Press, London, New York, San Francisco, Vol. 2d, pp. 625-702; 2 = Gay, 1978, Mol. Gen. Genet. 159: 269-83; 3 = Herforth, 1978, Genetics 88: 505-13; 4 = Ohanessian-Guillemain, 1953, DIS 27: 59.

^β Mechanism inferred to be affected.

ref: see *rfr*

ref(2)P

synonym: *ref*.

phenotype: *ref(2)P^p* is the first refractaire mutant found and the most thoroughly studied. Allele frequency about 0.2 in Paris population and approaches the same incidence in caged populations with or without the presence of sigma virus (Fleuriet, 1980, Genetics 95: 459-65; 1981, Genetics 97: 415-25). Refractaire phenotype expressed in cultured cells (Richard-Molard, 1975, Arch. Virol. 47: 139-46) and in imaginal disks transplanted into stably infected hosts (Bernard, 1968, Exp. Cell Res. 50: 117-26). Inhibition of viral multiplication increases with increased doses of *ref(2)P^p*, with added doses of *ref(2)P^o* having an antagonistic effect (Nakamura, 1978, Mol. Gen. Genet. 159: 285-92). Strains of sigma virus

capable of multiplication in restrictive strains can be selected; *P⁺* virus strains multiply in *ref(2)P^p* hosts, whereas *P⁻* strains do not; no such distinction in *ref(2)P^o* hosts. Also 5-FU-induced host-adapted strains of virus (haP) capable of replication in *ref(2)P^p*; many are temperature sensitive, suggesting that *ref(2)P^p* interacts with a viral protein (Coulon and Contamine, 1982, Virology 123: 381-92). Males homozygous for null alleles sterile; elongating spermatids display degeneration of axonemes (Dezelee, Bras, Contamine, Lopez-Ferber, Segretain, and Teninges, 1989, EMBO J. 8: 3437-46).

alleles: In addition to naturally occurring *ref(2)P^p* and *ref(s)P^o*, X-ray-induced (Nakamura) and three hybrid-dysgenesis-induced null alleles, *ref(2)P^{hd1}*, *ref(2)P^{hd2}*, and *ref(2)P^{hd3}* (Contamine, Petitjean, and Ashburner, 1989, Genetics 123: 525-33).

cytology: Placed in 37E2-F4 based on its being localized between *Df(2L)TW158 = Df(2L)37B2-8;37E2-F4* and *Df(2L)TW12 = Df(2L)37E2-F4;39D1-2*.

molecular biology: *ref(2)P* cloned (Contamine, Petitjean and Ashburner, 1989, Genetics 123: 525-33). Genomic DNA isolated by transposon tagging; sequencing indicates a structural gene with three exons and conceptual amino-acid sequence of 599 residues containing internal PEST repeats and interesting structural motifs, such as zinc fingers and amphiphilic helices; no homology with known proteins (Dezelee, Bras, Contamine, Lopez-Ferber, Segretain, and Teninges, 1989, EMBO J. 8: 3437-46).

refrangent: see *rfr*

regulator of αglycerol phosphate dehydrogenase: see *r(αGPDH)*

Regulator of bithorax: see *trx*

Regulator of post bithorax: see *hb*

rem: see *mbmB*

rem: remnants (T. Schüpbach)

location: 2-32.

origin: Induced by ethyl methanesulfonate.

references: Schupbach and Wieschaus, 1989, Genetics 121: 101-17.

phenotype: Maternal effect lethal; eggs laid by females homozygous for *rem¹* show no visible sign of development when observed under transmitted light in stereomicroscope; eggs derived from *rem²* often allow embryonic development; those embryos form fragmented cuticle with variable holes and head defects.

alleles: *rem¹* and *rem²* recovered as A and HG.

cytology: Located in 30A-C since included in *Df(2L)30A;C*.

Resistance (): see *Rst()*

Responder: see *Rsp*

ret: reticent (T. Schüpbach)

location: 2-62.

origin: Induced by ethyl methanesulfonate.

references: Schupbach and Wieschaus.

phenotype: Maternal effect lethal; embryos from homozygous mothers develop into larvae with no visible cuticular abnormalities, but do not hatch out of the egg case.

alleles: $ret^{PK} = ret^1$.

***ret: reticulated**

location: 1- (rearrangement).

origin: Induced by L-p-N,N-(2-chloroethyl)aminophenylalanine (CB. 3025).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 73.

phenotype: Wing veins increased to anastomosing reticulated areas. Wings shortened, deformed, and blistered. Eyes large and rough. Postvertical bristles usually absent. Male sterile; viability about 20% wild type. RK2A.

cytology: Associated with $T(1;2)ret = T(1;2)20A5-B2;2R$.

reticent: see *ret*

reticulated: see **ret*

retina aberrant in pattern: see *rap*

retinal degeneration: see *rdg*

retroactive: see *rtv*

Rev: Revolute

location: 2- (rearrangement).

origin: X ray induced.

discoverer: Dobzhansky, 31b5.

phenotype: Wings of heterozygote spread at 45° from midline; edges curled, giving spoon shape. Sense organs along veins enlarged. Eyes mottled in *Rev/lt*. Homozygote viable and fertile, somewhat more abnormal than heterozygote. Phenotype suppressed by extra Y's, probably a variegated position effect. RK2A.

cytology: Associated with $In(2LR)Rev = In(2LR)40F;52D10-E1$ [Bridges and Li, in Morgan, Bridges, and Schultz (1936, Year Book – Carnegie Inst. Washington 35: 293)].

Rev^B: Revolute of Bridges

origin: Spontaneous as a single homozygous female in a culture with no heterozygote.

discoverer: Bridges, 36e22.

synonym: *Rvd: Revolutoid*.

references: Morgan, Bridges, and Schultz, 1936, Year Book – Carnegie Inst. Washington 35: 293.

phenotype: Wings spread and curved. A variegated position effect based on enhanced phenotype by rearing at low temperature and Y suppressibility (Wargent, 1972, DIS 49: 50-51). Expression suppressed by $In(1)m^K$, and variegation of m^K enhanced by $In(2LR)Rev^B$ (Wargent and Hartmann-Goldstein, 1974, Heredity 33: 317-26); incidence of chromosomes heterochromatinized in vicinity of breakpoints shows parallel interactions (Hartmann-Goldstein and Wargent, 1975, Chromosoma 52: 349-62). Homozygous lethal; Rev/Rev^B viable (E. B. Lewis). RK2A.

cytology: Associated with $In(2LR)Rev^B = In(2LR)40F;52D5$ (Wargent).

Revolute: see *Rev*

Revolutoid: see *Rev^B*

rex: rapid exhaustion (J.C. Hall)

location: 1-17.0.

origin: Induced by ethyl methanesulfonate.

references: Grigliatti, Hall, Rosenbluth and Suzuki, 1973,

Mol. Gen. Genet. 120: 107-14.

Homyk, Sinclair and Suzuki, 1979, Genetics 91: s49-50.

phenotype: Adults briefly paralyzed after movement, especially when they are induced to move rapidly; recovery occurs within one min, followed by uncoordinated movements and a period refractory to induction of further paralysis of at least one hr (Grigliatti *et al.*, 1973); *rex* is temperature sensitive for lethality, *i.e.*, as induced by holding adults about ten days at 29° (Homyk *et al.*, 1979); also there are semi-lethal effects on development [*i.e.*, *ca.* 50% viability after rearing at 29° (Homyk, Sinclair, Wong, and Grigliatti, 1986, Genetics 113: 367-89)]. *rex* also hypersensitive to killing effects of caffeine (Homyk *et al.*, 1979). Causes an abnormally slow recovery from blue-light induction of prolonged depolarizing afterpotential (PDA) of photoreceptors; recovery induced rapidly by orange light (Homyk and Pye). Mosaic analysis of leg paralysis induced by *rex* suggests mesodermal (possibly muscle) foci, separate focus for each leg (Homyk *et al.*, 1986).

cytology: Placed in 7C1-4 based on its inclusion in $Df(1)ct4b1 = Df(1)7B2-4;7C3-4$ but not in $Df(1)ct268-42 = Df(1)7A5-6;7B8-C1$ or $Df(1)ct-J4 = Df(1)7A2-3;7C1$ (Homyk).

alleles: One mutant allele, sometimes called *rex^{ts}* (Homyk *et al.*, 1979).

Rex: Ribosomal exchange (L. Robbins)

location: 1-66 [proximal to *su(f)* (Rasooly)].

origin: Spontaneous? Discovered in $Df(1)w^{rJ1}$ bearing chromosome.

references: Robbins, 1981, Genetics 99: 443-59.

Swanson, 1984, Ph.D. dissertation, Michigan State University.

Swanson, 1987, Genetics 115: 271-76.

phenotype: Semi-dominant, maternal-effect locus which causes an early mitotic exchange between blocks of rDNA in 1 to 10% of the offspring of *Rex*-bearing females. *Rex* affects chromosomes with two separated, complete or partial ribosomal-DNA regions, such as *YSX.YL* or $In(1)sc^{SILsc^{4R}}$. Chromosomes having separated blocks of heterochromatin that do not both include ribosomal genes, such as $In(1)w^{m4}$, are insensitive. Affected chromosomes may pair in either spiral or hairpin configurations; the former results in loss of the intervening material (*e.g.*, Ybb^{Rex} , $Dp(1;f)sc^{SILsc^{4R}}$, $Dp(1;f)w^{m51bl_w^{m4R}}$, the latter in the inversion of that material ($Dp(1;1)sc^{SILsc^{4R}}$, $Dp(1;1)w^{m51bl_w^{m4R}}$) (Robbins and Swanson, 1988, Genetics 120: 1053-59). Exchanges usually occur early enough to yield whole-body recombinant genotypes, but a minority of events yield half-half mosaics. The exchange event generally results in partial deletion of rDNA (Robbins).

***rey: rough eye**

location: 1-0.6 (from combined measurements on *rey*, rey^2 , and rey^3).

phenotype: Eyes extremely small and rough in male, less extreme in female. Areas of thorax often underdeveloped, sometimes hemithoracic. (rey^2). Homozygous female viable and infertile (rey^3).

alleles: Complementation tests not performed; allelism

inferred from similarity of phenotype and map position.

allele	origin	discoverer	synonym	ref ^α
<i>*rey¹</i>	spont	Neel, 41g7	<i>re</i>	2
<i>*rey²</i>	spont	Sturtevant, 1948	<i>rey²</i>	3
<i>rey³</i>	CB 3025	Fahmy, 1953	<i>re²</i> ; <i>rougheye-like</i>	1
<i>rey⁴</i>	CB 3007	Fahmy		1

^α 1 = Fahmy, 1958, DIS 32: 73; 2 = Neel, 1942, DIS 16: 52; 3 = Sturtevant, 1948, DIS 22: 55-56.

cytology: Outside of region 1E1-2B9 based on complementation of *rey³* by both *Df(1)sta = Df(1)ID3-E1;2A* and *Df(1)RA19 = Df(1)IE3-4;2B9-10* (Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90).

other information: Not recognized as such in recent studies of polytene region 2 by Lefevre or by Perrimon.

***rf: roof wings**

location: 2-81.

discoverer: Bridges, 1921.

references: Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 233.

phenotype: Wings rotated on long axis so that inner margins are raised and costal margins lowered. Overlaps wild type. RK3.

rf²

origin: Spontaneous.

discoverer: Redfield, 1926.

references: Franke, 1933, Ph.D. Thesis, Univ. Berlin.

phenotype: Like *rf*. RK3.

Rf: Roof

location: 3-59.

origin: Spontaneous.

discoverer: Waddington, 38a.

references: 1939, DIS 12: 48-49.

phenotype: Wing position normal at eclosion, becomes rooflike in 12-hr imagos. RK1.

cytology: Tentatively placed in 89C-90E based on the observation that a third chromosome deficiency combining the left portion of *T(Y;3)L142 = T(Y;3)89C* and the right portion of *T(Y;3)B116 = T(Y;3)90E* has a *Rf*-like phenotype (Rendel, 1977, DIS 52: 86).

***Rf-c: Roof-c**

location: 3- (to the left of *se*).

discoverer: Bridges, 20a1.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 228 (fig.).

phenotype: Wings slanted at roof-like angle. RK3.

rfd: see *sas*

rfr: refringent

location: 1- 67.9[to the left of *ot* at 1-65.7, since it is not covered by *y⁺Ymal106* which does cover *ot* (Schalet, 1972, DIS 49: 36)].

origin: Induced by D-*p*-N,N-di-(2-chloroethyl)aminophenyl-alanine (CB. 3026).

discoverer: Fahmy, 1955.

synonym: *ref* (preoccupied).

references: 1959, DIS 33: 89.

phenotype: Wing surface yellowish and iridescent; occasionally, one or both wings held out; inner margins may

be incised. Expression more extreme in male than in female. Male viable and fertile; female has reduced viability and is sterile. RK2.

alleles: One allele each induced by CB.3026 and CB. 3034.

rg: rugose

location: 1-11.0.

references: Renfranz and Benzer, 1989, *Dev. Biol.* 136: 411-29.

phenotype: Eyes variably rough depending on strength of allele; stronger alleles display reduced viability. Additional traits include wings thin, curled upward (*rg^P*), margins somewhat frayed (*rg^I*); eclosion delayed (*rg⁷*); body pale (*rg^P*).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>*rg¹</i>		Bridges, 21c	<i>roughish</i>	5	
<i>rg²</i>		Demerec, 28f23	<i>rough-64</i>		
<i>*rg³</i>	spont			4	
<i>*rg⁴</i>		Ives, 33g22	<i>rg^{33g}</i>	6, 7	
<i>*rg⁵</i>	spont	Bridges, 38c9		4	
<i>*rg⁶</i>	spont	Bridges	<i>rox</i>	4	
<i>*rg⁷</i>	X ray	Cantor, 46d20	<i>rg^C</i>	3	<i>In(1)4E;7A</i> (Valencia)
<i>*rg^P</i>	32 P	Bateman, 1950		1, 2	<i>In(1)3C;4E</i> (Darby)

^α 1 = Bateman, 1950, DIS 24: 54; 2 = Bateman, 1951, DIS 25: 77-78; 3 = Cantor, 1946, DIS 20: 64; 4 = CP552; 5 = Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 234; 6 = Plough, 1934, DIS 2: 34; 7 = Plough and Ives, 1935, *Genetics* 20: 42-69.

cytology: Locus at 4E1-3 (Demerec, Kaufmann, Fano, Sutton, and Sansome, 1942, *Year Book - Carnegie Inst. Wash.* 41: 191). Placed in 4E2 based on its inclusion in *Df(1)ovo6 = Df(1)4C5-6;4E2-3* and *Df(1)bi-DL3 = Df(1)3C7-12;4E1-2* but not *Df(1)bi-DL5 = Df(1)3C7-12;4E1-2* (Oliver).

Rg-bx: see *trx*

Rg-pbx: see *hb*

***rgt: reduced pigment**

location: 1-11.5.

origin: Induced by L-*p*-N,N-di-(2-chloroethyl)aminophenylalanine (CB. 3025).

discoverer: Fahmy, 1954.

references: 1959, DIS 33: 89.

phenotype: Characteristic pigmentation of fifth tergite reduced or absent in male. Body color yellowish. Eyes bright red. Male sterile. RK2.

rh: roughish

location: 2- 54.7[on 2R based on its recovery in newly generated C(2R) chromosomes and placed distal to *ap* (2-55.2) on the basis of its being complemented by *Df(2R)M41A4* (Hilliker, Gibson, Yeomans, and Holm, 1977, DIS 52: 32)].

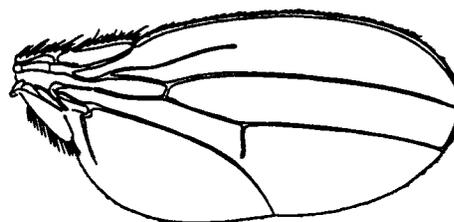
origin: Spontaneous.

discoverer: Bridges, 21a3.

phenotype: Eyes moderately rough. At 19°, bristles slightly wavy and wings broad. RK2.

rh: see *gl³*

Rh1: see *ninaE*

Rh2: Rhodopsin 2**location:** 3- {65}.**references:** Cowman, Zuker, and Rubin, 1986, Cell 44: 705-10.**phenotype:** Encodes the opsin moiety of the rhodopsin specific to the ocelli. Not expressed in the eye nor is it expressed in Bolwig's organ, the larval photoreceptor [Pollock and Benzer, 1988, Nature (London) 333: 779-82)]. *Rh2* under the control of the *ninaE* promoter in a *ninaE* background functions in R1-6 cells (Zuker, Mismar, Hardy, and Rubin, 1988, Cell 55: 475-82) but with different spectral characteristics and physiology (Feiler, Harris, Kirschfield, Wehrkan, and Zuker, 1988, Nature (London) 333: 737-41). RH2 is a 381-amino-acid protein that is 67% homologous to the opsin found in R1-6 encoded by *ninaE*, but only 35% homologous to RH3 and RH4.**cytology:** Located in 91D1-2 by *in situ* hybridization.**molecular biology:** Genomic clone isolated by cross homology with a clone containing the *ninaE* gene. Sequence analysis indicates the presence of three introns, the position of the second of which is shared with that of the fourth intron of *ninaE*; positions of the other introns are not conserved. The conceptual amino-acid sequence reveals many features that characterize opsins: seven hydrophobic domains separated by hydrophilic sequences, a putative retinal-binding site in the seventh transmembrane domain, a glycosylation site in the extra cytoplasmic face, and a series of potential phosphorylation sites in the C-terminal region. Head specific transcription depends on elements between -183 and -112 (Mismar, Michael, Laverty, and Rubin, 1988, Genetics 120: 173-80); contains an eleven-base-pair sequence common to *ninaC* and *ninaE* (Mismar *et al.*).**Rh3****location:** 3- {70}.**references:** Zuker, Montell, Jones, Laverty, and Rubin, 1987, J. Neurosci. 7: 1550-57.**phenotype:** Encodes the opsin moiety of a rhodopsin specific to the rhabdomere of the seventh photoreceptor cell. Also expressed in Bolwig's organ [Pollock and Benzer, 1988, Nature (London) 333: 779-82)]. Transcript first appears during the last 48 hr of the pupal stage and is found in newly eclosed adult heads. RH3 is a 383 amino acid protein that is 35% homologous to the opsins found in R1-6 encoded by *ninaE* and in ocelli encoded by *Rh2* but 72% homologous to the *Rh4*-encoded protein.**cytology:** Located in 92D1 by *in situ* hybridization.**molecular biology:** Genomic clone isolated by hybridization to oligonucleotide probes containing sequences highly conserved between *ninaE* and *Rh2*. The gene contains no introns. The conceptual amino-acid sequence reveals features characteristic of opsin molecules (see *Rh2*).**Rh4****location:** 3- {45}.**references:** Montell, Jones, Zuker, and Rubin, 1987, J. Neurosci. 7: 1558-66.**phenotype:** Encodes the opsin moiety of a rhodopsin specific to the rhabdomere of the seventh photoreceptor cell; *Rh3* and *Rh4* are expressed in nonoverlapping subsets of ommatidia; the distribution of the two types of R7 cells within the eye is irregular. Transcript first appearsduring the last 48 hr of the pupal stage and is found in newly eclosed adult heads. Also expressed in Bolwig's organ [Pollock and Benzer, 1988, Nature (London) 333: 779-82)]. RH4 is a 378 amino acid protein that is 35% homologous to the opsins found in R1-6 encoded by *ninaE* and in the ocelli, encoded by *Rh2*, but 72% homologous to the *Rh3*-encoded protein.**cytology:** Located in 73D3-5 by *in situ* hybridization.**molecular biology:** Genomic clone isolated by hybridization to the *Rh3* gene. Sequence analysis reveals the presence of a single 9.0 kb intron, which subdivides the gene into exons of 676 and 719 nucleotides. The conceptual amino-acid sequence reveals features characteristic of opsin molecules (see *Rh2*).*RH11*: see *eas**rho*: see *ve**Rho*: see *Lcp10***Rhodopsin:** see *Rh**rhomboid*: see *ve***ri: radius incompletus**

From Edith M. Wallace, unpublished.

ri: radius incompletus**location:** 3-46.8. (Arajarvi and Hannah-Alava, 1969, DIS 44: 73).**phenotype:** Vein L2 interrupted. Wings slightly warped and blunt. Acts during contraction period in *D. simulans*, inhibiting fusion of small spaces into a vein (Waddington, 1940, J. Genet. 41: 75-139). RK1.**alleles:**

allele	origin	discoverer	synonym	ref ^α	comments
<i>ri</i> ¹	spont	Tshetverikov, 1926			
<i>*ri</i> ²		Nordenskiöld, 1935		3	
<i>*ri</i> ⁵ ^{1k}	spont	Meyer, 51k		1	
<i>ri</i> ^{53j}	spont	Meyer, 53j		2	

^α 1 = Meyer, 1952, DIS 26: 67; 2 = Meyer, 1953, DIS 27: 58; 3 = Nordenskiöld, 1937, DIS 7: 18.**cytology:** Tentatively placed in salivary region 77E-F (Arajarvi and Hannah-Alava, 1969, DIS 44: 73).*RI*^{DDT}: see *Rst(2)DDT**RI*^{II}: see *Rst(2)DDT***rib: ribbon****location:** 2-88.**origin:** Induced by ethyl methanesulfonate.**references:** Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82. Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Homozygous embryonic lethal; lateral extents of belts narrow; fusion of adjacent denticular bands in ventral midline; dorsal closure defective.

alleles: Nine; *rib*¹ recovered as *IK* and weak allele *rib*² recovered as *IIB*; seven alleles discarded.

Ribosomal exchange: see *Rex*

Ribosomal protein 49: see *M(3)99D*

Ribosomal protein A1: see *RpA1*

ricketts: see *rk*

rimy: see *rm*

Ring magnifier: see *Rm*

rk: *ricketts* (*M. Ashburner*)

location: 2-48.8 (immediately to the right of *j*).

phenotype: Strong alleles have legs, especially metathoracic ones, flattened and bent. Femora and tibiae bowed in middle; first two tarsal joints shortened, bent, and flattened; last three tarsal joints almost fused. Wings unexpanded, resembling those of *pu*. Posterior scutellar bristles erect and crossed. Viability may be reduced. Weak alleles do not show the leg phenotype, or they overlap wild type; may have partially expanded wings and normal posterior scutellar bristles. Even strong alleles more extreme when hemizygous with *rk* deficiencies.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>rk</i> ¹	UV	Edmondson, 48h		2	strong allele
<i>rk</i> ²	UV	Erickson, 50a		7	weak allele, stronger at 29° than 17°
<i>rk</i> ³	UV	Meyer, 54d		4	strong allele, body small, bristles fine ^β
<i>rk</i> ⁴	spont	Jackson, 54c	<i>cq: creeper</i>	3, 5	β
<i>rk</i> ⁵	spont	Mischaikow, 59a		8	weak allele ^β
<i>rk</i> ⁶	X ray	Thomas, 60g		6	β
<i>rk</i> ^{<i>cyl</i>}	spont	Stroher, 58		9	β
<i>rk</i> ⁷⁶	EMS			1	intermediate allele; cytologically normal
<i>rk</i> ⁸⁰¹¹	EMS	Bodmer			strong allele
<i>rk</i> ⁸⁰¹²	EMS	Bodmer			weak allele
<i>rk</i> ⁸¹¹²	EMS	Harrington			strong allele, on <i>In(2LR)O</i> , induced with <i>b^{81f2}</i>
<i>rk</i> ¹²⁵⁰	EMS	Lindsley			intermediate allele
<i>rk</i> ^{EMS}	EMS	Grell			on <i>Dp(2;2)Adh3</i>

^α 1 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193; 2 = Edmondson, 1948, DIS 22: 53; 3 = Jackson, 1954, DIS 28: 74; 4 = Meyer, 1955, DIS 29: 74; 5 = Meyer, 1958, DIS 32: 83; 6 = Meyer, 1963, DIS 37: 51; 7 = Meyer, Edmondson, Byers, and Erickson, 1950, DIS 24: 60; 8 = Mischaikow, 1959, DIS 33: 98; 9 = Mainx, 1958, DIS 32: 82.

^β Additional description below.

cytology: Placed in 31E1-5 based on its inclusion in *Df(2L)b-L = Df(2L)34D3;34E3-5* and *Df(2L)el82fl = Df(2L)34E1-2;35C3-5*, but not *Df(2L)b82a2 = Df(2L)34D1-2;34E1-2*.

rk⁴

phenotype: Wings unexpanded, spread, and drooping. Posterior legs malformed. Both sexes fully viable and fertile. *rk*⁴ male mates with wild-type female only if wings removed from female. Viability 60% of wild type.

rk⁵

phenotype: Wings sometimes fully expanded and held out.

rk⁶

phenotype: Legs weak, wings unexpanded. Viability higher at higher temperature.

rk^{*cyl*}

phenotype: Abdomen cylindrical; terminal segments thickened; halteres small and melanotic; legs like those of *bal*, but less deformed. Viability low but fertility good.

rl: *rolled*

location: 2-55.1 [between centromere and *stw* (Sturtevant); 0.03 unit to the left of *stw* (Tano, 1966, J. J. Genet. 41: 299-308)].

phenotype: Most alleles lethal. The viable allele, *rl*¹, has wing edges rolled downward; margins somewhat frayed; L4 interrupted distal to posterior crossvein. Eyes small, dark, and rough. Most extreme at 26°, less extreme above and below that temperature (Lakovaara, 1963, Proc. Intern. Congr. Genet., 11th., Vol. 1: 175). Temperature sensitive period for eye phenotype during larval stages with most sensitive stage about 60 hr after hatching, *i.e.*, at the beginning of the third-instar (Hackman and Lakovaara, 1966, DIS 41: 92). Effects of dosage of *rl* and *rl*⁺ on eye pigment deposition investigated by Lakovaara (1966, Hereditas 56: 1-19). *rl*¹ lethal in combination with all lethal alleles except *rl*⁶, with which it is fully viable and exhibits a *rl* phenotype. Hemizygotes for lethal alleles, except for *rl*⁶, die as third-instar larvae completely devoid of imaginal disks; when heterozygous for *rl*¹ or *rl*⁶, the lethal alleles lead to pupal lethality (Hilliker, 1976, Genetics 83: 765-82).

allele	origin	discoverer	synonym	ref ^α	comments
<i>rl</i> ¹	spont	Bridges, 22f23		4	viable, RK2
<i>rl</i> ²	EMS	Hilliker	<i>l(2)EMS34-29</i>	3	L3
<i>rl</i> ³	EMS	Hilliker	<i>l(2)EMS43</i>	3	L3
<i>rl</i> ⁴	EMS	Hilliker	<i>l(2)EMS45-32</i>	3	L3
<i>rl</i> ⁵	EMS	Hilliker	<i>l(2)EMS45-39</i>	3	L3
<i>rl</i> ⁶	EMS	Hilliker	<i>l(2)EMS45-52</i>	3	P
<i>rl</i> ⁷	EMS	Hilliker	<i>l(2)EMS45-54</i>	3	L3
<i>rl</i> ⁸	EMS	Hilliker	<i>l(2)EMS45-95</i>	3	L3
<i>rl</i> ⁹	EMS	Hilliker	<i>l(2)EMS64</i>	3	L3
<i>rl</i> ¹⁰	EMS	Hilliker	<i>l(2)EMS698</i>	3	L3
<i>rl</i> ^G	heat	Goldschmidt, 1929		1, 2	viable

^α 1 = Goldschmidt, 1929, Biol. Zentralbl. 49: 437-48; 2 = Goldschmidt, 1939, Am. Nat. 73: 547-59; 3 = Hilliker, 1976, Genetics 83: 765-82; 4 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 233.

cytology: Placed in 41A on the basis of its inclusion in *Df(2R)M41A10 = Df(2R)41A* (Morgan, Schultz, and Curry, 1941, Year Book - Carnegie Inst. Washington 40: 284). Further restricted to the middle of the proximal heterochromatin on the basis of its inclusion in *Df(2R)A* but not *Df(2R)B* (Hilliker, 1976, Genetics 93: 765-82).

***rlu: rolled up**

location: 1- (rearrangement).
origin: Spontaneous in *In(1)sc^{S1}+dl-49*.
discoverer: Reddi.
references: 1963, DIS 37: 53.
phenotype: Wings rolled. Good viability and fertility. RK2A.

rm: rimy

location: 1-48.1.
origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).
discoverer: Fahmy, 1956.
references: 1958, DIS 32: 74.
phenotype: Eyes often dull brownish red with conspicuous white hairs between ommatidia. Wings longitudinally pleated. Viability and fertility good. RK2.
other information: One allele each induced by CB. 1540 and CB. 1592.

rm: see *rmp*

Rm: Ring magnifier

location: Autosomal; not mapped.
origin: Spontaneous.
references: Endow, Komma, and Atwood, 1984, Genetics 108: 969-83.
phenotype: Existence of such a mutant inferred from the behavior of ring chromosomes in magnifying genotypes in different autosomal backgrounds. In the absence of *Rm*, magnifying conditions cause the meiotic loss of *bb*-bearing ring chromosomes from *R(1)*, *bb/Ybb⁻* males, as detected by both a shift in the sex ratio among the progeny and the production of offspring with no paternally inherited sex chromosome; some *bb^r* (bobbed-reduced) offspring are observed, but there are no *bb^m* (bobbed magnified) offspring. In the presence of *Rm*, the incidence of ring-chromosome loss is elevated and *bb^m* offspring occur. Postulated that sister-chromatid exchange outside of the ribosomal cistrons correlated with magnifying sister-chromatid exchanges within the ribosomal cistrons may occur in the presence of *Rm* but not *Rm⁺*.

Rm2: Ring magnifier on chromosome 2

location: 2-48.
origin: Spontaneous.
discoverer: Komma and Atwood.
references: Endow, Komma, and Atwood, 1984, Genetics 108: 969-83.
phenotype: Dominant mutant that permits recovery of magnified *bb* alleles in ring chromosomes. Also increases incidence of dicentric rings as seen in the primary spermatocyte division. Postulated to engender sister chromatid exchanges outside the ribosomal DNA.

Rm4

location: 4-.
origin: Spontaneous.
discoverer: Komma.
phenotype: Similar to that of *Rm2*.

***rmp: rumped**

location: 1-14.4.
origin: Induced by *L-p-N,N-di-(2-chloroethyl)amino-phenylalanine* (CB. 3025).

discoverer: Fahmy, 1955.
synonym: *rm* (preoccupied).
references: 1959, DIS 33: 89.
phenotype: Wings variably unexpanded. Bristles deranged; postverticals frequently crossed. Derangement of bristles correlated with degree of wing abnormality. Viability and fertility good in both sexes. RK2.

rn: rotund (S. Kerridge)

location: 3-47.6 [from location of *rn³* (Puro and Nygrén, 1975, Hereditas 81: 237-48); distal to *roe* (Agnel *et al.*)].
references: Cavener, Otteson, and Kaufman, 1986, Genetics 114: 111-23.
 Kerridge and Thomas-Cavallin, 1988, Roux's Arch. Dev. Biol. 197: 19-26.
 Agnel, Kerridge, Vola, and Griffin-Shea, 1989, Genes Dev. 3: 85-95.

phenotype: All alleles are viable as homozygotes. Both males and females are sterile; sterility is not germ line dependent. Two transcripts from the rotund region of *Drosophila* show similar positional specificities in imaginal disc tissues. Adult defects are restricted to homologous distal parts of the appendages, *i.e.*, the antennae, legs, wings, halteres, and proboscis. In the antenna the basal capsule is missing and the third antennal segment is reduced in size; all other antennal and eye disc derivatives are normal. In all three pairs of legs abnormalities caused by the lack of *rn⁺* product are localized specifically in the tarsus; instead of five individual tarsal segments, a single tarsus-like segment differentiates. The distal claw and proximal leg articulations (tibia, femur, trochanter, and coxa) are unaffected. Incomplete and duplicated joints seen at the presumptive positions of the tarsus 1-2 and 4-5 joints; intermediate joints virtually absent (Held, Duarte, and Derakhshanian, 1986, Wilhelm Roux's Arch. Dev Biol. 195: 145-57). Of the wing disc derivatives, the medial and distal costa are fused and the corresponding region in the posterior wing, the alula, is smaller, making the wing as a whole appear shorter; distal wing and mesonotum are formed as in wild type. Vein L5 and to a lesser extent L2 interrupted (*rn³*). Specific and localized deficiencies of labial and haltere disc derivatives are also evident. Genital and abdominal patterns are indistinguishable from wild-type patterns; however, seminal receptacles shorter than in wild-type females. Examination of the imaginal discs from third-instar *rn* larvae shows localized cell death in regions determined from fate maps to give rise to distal appendage parts (Cavallin).

alleles: No interallelic complementation detectable.

allele	origin	discoverer	synonym	ref ^α	comments
<i>rn¹</i>	X ray	Glass, 1929		1, 3	<i>T(2;3)40-41;80-81;84D3-4</i>
<i>*rn²</i>	spont	Carlson		2	
<i>rn³</i>	X ray	Hannah-Alava		1, 5	cytology normal
<i>rn⁴</i>	EMS	Kaufman		1	<i>roe⁻ scr⁻</i>
<i>rn⁵</i>	γ ray	Williams		1	aberration at 84D3-4
<i>rn⁶</i>	X ray	Kerridge		1	
<i>rn⁷</i>	EMS	Ait-Ahmed		1	<i>roe⁻</i>
<i>rn⁸</i>	EMS	Kerridge		1	<i>roe⁻</i>
<i>rn¹⁰</i>	EMS	Kerridge		1	<i>roe⁻</i>
<i>rn¹¹</i>	EMS	Kerridge		1	lesion in 20.5 to 22.8
<i>rn¹⁴</i>	EMS	Kerridge		1	
<i>rn¹⁵</i>	EMS	Kerridge		1	
<i>rn¹⁶</i>	DEB	Kerridge		1	1 kb deletion between -6.5 and -2.1 kb; <i>roe⁻</i>

allele	origin	discoverer	synonym	ref ^α	comments
<i>rn</i> ¹⁸	DEB	Kerridge		1	<i>roe</i> ⁻
<i>rn</i> ¹⁹	DEB	Kerridge		1	sequences distal to 12.3 deleted
<i>rn</i> ²⁰	DEB	Kerridge		1	all cloned sequences missing <i>roe</i> ⁻
<i>rn</i> ²¹	DEB	Kerridge		1	<i>roe</i> ⁻
<i>rn</i> ²³	DEB	Kerridge		1	<i>roe</i> ⁻
<i>rn</i> ^D	X ray	Kerridge	<i>Dipr</i>	1, 4	<i>ln(3R)84D3-4</i> ; <i>84F6-11 β</i>
<i>rn</i> ^{FC8}	X ray	Lehman		1	<i>Tp(3;1)84D3-4:85</i>
<i>rn</i> ^{X49}	X ray	Jürgens	<i>rn roeX49</i>	1	<i>roe</i> ⁻
<i>rn</i> ^{X130}	X ray	Jürgens	<i>rn XT130</i>	1	<i>ln(3R)81;84D9-10</i>

^α 1 = Agnel, Kerridge, Vola, and Griffin-Shea, 1989, *Genes Dev.* 3: 85-95; 2 = Carlson, 1956, *DIS* 30: 70, 109; 3 = Glass, 1934, *DIS* 2: 8; 4 = Kerridge, 1981, *Mol. Gen. Genet.* 184: 519-25; 5 = Puro and Nygrén, 1975, *Hereditas* 81: 237-48.
^β More detailed description below.

cytology: Placed in 84D3 on the basis of its inclusion in the region of overlap between *Df(3R)dsx10M = Df(3R)84D3;84F1* and *Df(3R)Scx4 = Df(3R)84B1-2;84D3-4*. The heterozygote between the two deficiencies survives and is rotund in phenotype.

molecular biology: Gene localized within a 70 kb walk on the basis of the clustering of restriction-fragment-length differences associated with *rn* mutations. Mutational lesions are arrayed over a 50 kb region from -6.5 to +44 kb. 0 is defined as the distal breakpoint of *Df(3R)Scx4*, positive values extending to the right. Two transcripts identified from the region, a 5.3 kb transcript hybridizing to a genomic fragment from 23.8 to 34.4 kb which accumulates steadily from the embryonic stage until the end of pupation and a 1.7 kb transcript between coordinates 6.3 and 12.0 kb, which appears during the third larval instar and peaks during the first two days of pupation. Both sequences are transcribed from left to right. Spatial distribution of expression of the two sequences in white prepupae highly concordant. Found in the distal-forming regions of the wing, haltere, leg, and labial discs. Adepithelial cells of the wing discs thought to contribute to thoracic musculature also exhibit expression as does a narrow strip of tissue in the genital disc destined to give rise to the internal ducts.

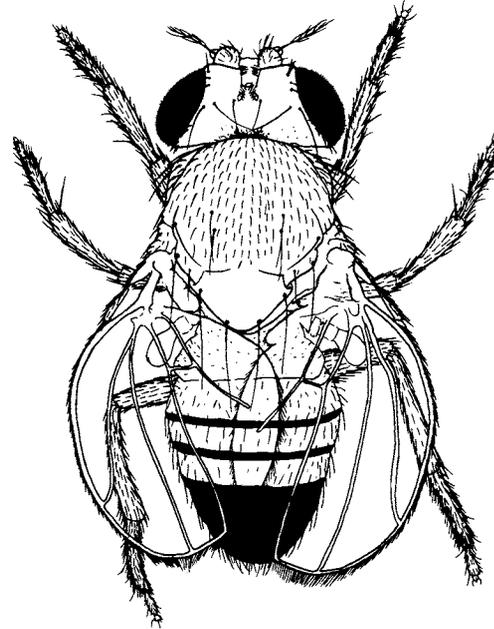
***rn*^D: rotund-Dominant**

synonym: *Dipr*: Distal into proximal.

phenotype: Dominant mutation with complete penetrance and high expressivity. In *Dipr/+* flies the distal wing blade reduced to two-thirds normal length; triple row replaced by two to three rows of irregularly sized bristles resembling those found on costa; occasionally these bristles may be bracted as are those of proximal costa. Posterior row of marginal bristles absent distally. Venation in proximal portion of wing irregular and crowded but more nearly normal distally. Trichomes of wing blade organized into whorls. Capitellum of haltere covered with small adventitious bristles and sometimes pedicellar-like sensilla. Pleura, coxa, trochanter, femur, tibia, and fifth tarsal segment unaffected; other tarsal segments reduced; tarsal segments two to four variably fused; ectopic sex combs form on second tarsal segment in 70-90% of forelegs. Basal cylinder of antenna reduced or missing; replaced by bristle elements at third-segment-arista junction resembling those on third segment. Proboscis con-

tains five to seven rows of pseudotracheae rather than normal twelve; replaced by bristles laterally. Kerridge suggests transformation of distal into more proximal appendage elements. Homozygotes survive and are more extreme.

other information: Probably a hypermorphic allele of *rn*. X-ray-induced revertants designated *Dipr^{+R1}-Dipr^{+R9}* (lacking +R3 and +R6); the three that are visibly deleted are deficient for the more distal inversion realignment of chromosome segments.



rn: rotund

From Bridges and Brehme, 1944, *Carnegie Inst. Washington Publ. No. 552*: 159.

RNA: Genes described below:

RNA polymerase: see *RpII*

rRNA5s: see *min*

rRNA5.8s: see *bb*

rRNA18s: see *bb*

rRNA28s: see *bb*

snRNA: small nuclear RNA

RNA molecules ranging in size from about 100 to 300 nucleotides, which in association with polypeptides comprise a family of nuclear ribonucleoprotein (snRNP) complexes. The polypeptides are recognized by antibodies produced by patients with systemic lupus erythematosus; in *Drosophila* such sera recognize a 26 kd polypeptide, which is conserved between flies and mammals, as well as an 18 kd polypeptide (Wooley, Cone, Tartof, and Chung, 1982, *Proc. Nat. Acad. Sci. USA* 79: 6762-66). Significant sequence homology is found between *Drosophila* snRNA molecules and those isolated from mammals. Some snRNA genes have been cloned and sequenced, but as yet not localized by *in situ* hybridization; in other cases, the RNA's have been isolated and

complementary genomic sequences located by hybridization to polytene chromosomes. Where *in situ* hybridizations have been carried out, single snRNA species hybridize to a fixed number of specific sites, and with the exception of 39B no two hybridize to the same site.

locus	location	synonym	ref ^α	comments
snRNA1			2	homologous to mammalian U1; cloned and sequenced; ca 164 nucleotides
snRNA2	39B 40A-B	<i>snRNA1</i>	1, 3, 4	3 copies; homologous to mammalian U2; cloned and sequenced; ca 186 nucleotides
snRNA3	22A 82E 95C	<i>snRNA2</i>	1, 3	7 copies; homologous to mammalian U3
snRNA4	14B 23D 34A 35E-F 39B 63A	<i>snRNA3</i>	3	7 copies; homologous to mammalian U4
snRNA6	96A	<i>snRNA4</i>	3	1-3 copies; homologous to mammalian U6

^α 1 = Alonso, Jorcano, Beck, and Spiess, 1983, J. Mol. Biol. 169: 691-705; 2 = Mount and Steitz, 1981, Nucl. Acids Res. 9: 6351-68; 3 = Saluz, Schmidt, Dudler, Altwegg, Stumm, Zolinger, Kubli, and Chen, 1983, Nucl. Acids Res. 11: 77-90; 4 = Wooley, Cone, Tartof, and Chung, 1982, Proc. Nat. Acad. Sci. USA 79: 6762-66.

other information: Since the numerical designations applied by Saluz *et al.* are generally offset from those of the mammalian numbering system, we here reconcile the two systems by renumbering the *Drosophila* snRNA species.

tRNA: transfer RNA

As many as 90 different tRNA sequences have been identified in the *Drosophila melanogaster* genome, each being present in from eight to twelve copies. tRNA species have been differentiated and designated according to the chromatographic mobility of their aminoacylated derivatives and without regard to their anticodon content (White, Tener, Holden, and Suzuki, 1973, Dev. Biol. 33: 185-95). The number of isoacceptor forms resolvable by column chromatography varies from two for histidine to eleven for asparagine. Several forms show evidence of post-transcriptional modification in that an earlier form (δ) decreases during development while a form that elutes from the column at reduced ionic strength (γ) displays a concomittant increase (White, Tener, Holden, and Suzuki, 1973, J. Mol. Biol. 74: 635-51). The several copies of a particular isoform need not occur together on the chromosome, although numerous cases of clustering of copies of the same isoform as well as of different tRNA's have been recorded. In cases of clustered tRNA genes, transcription may take place in both directions. Dosage compensation has been observed for *tRNA-Ser4* (Birchler, Owenby, and Jacobson, 1982, Genetics 102: 525-37) as well as in the case of *tRNA-Val3b* in heterozygotes for *Df(3R)Antp17 = Df(3R)84A6;84D13-14* [Tener, Hayashi, Dunn, Delaney, Gillam, Grigliatti, Kaufman, and Suzuki, 1980, Transfer RNA (Abelson, Schimmel, and Söll, eds.). Cold Spring Harbor Laboratory, Cold Spring Harbor, NY, pp. 295-

307]; however, these observations are based on specific amino acylation activity and need not reflect increased transcription from 12D-E or 84D alone. In the table below, each of the isoforms is listed and, where available, the cytological locations, as determined by *in situ* hybridization with purified samples or cloned sequences, are indicated; where localizations do not specify isoform, a numerical designation is omitted. In only a few cases has crossreaction in *in situ* hybridizations been ruled out by competition experiments. Where the numbers of copies are indicated, these are minimum estimates based on cloned segments from the regions involved; other indications of copy number are available from grain counts in autoradiograms; for example the grain-number ratio in 84D:90B-C:92D for tRNA-Val3b is 6:3:1 (or 5:4:1) and in 42A:42E:50B:62A:63B for tRNA-Lys2 is 5:3:1:1:1 (Tener *et al.*, 1980). Sequencing, where indicated, has been performed either on purified tRNA samples or on cloned DNA sequences.

locus	location	ref ^α	comments
tRNA-Ala	63A 90C	9, 24 9, 24	
tRNA-Ala1			
tRNA-Ala2			
tRNA-Ala3			
tRNA-Ala4			
tRNA-Ala5			
tRNA-Arg1			
tRNA-Arg2	42A	9, 12, 18, 24, 29, 30, 33	8 copies; sequenced
(CGU binding)	56E-F 84E-F	12 7, 9, 12, 24	5 copies; cloned
tRNA-Arg3			
tRNA-Arg4			
tRNA-Arg5			
tRNA-Asn1			δ and γ forms
tRNA-Asn2			δ and γ forms
tRNA-Asn3			δ and γ forms
tRNA-Asn4			δ and γ forms
tRNA-Asn5	42A	9, 12, 18, 24, 30, 32	δ and γ forms; (AAC binding) cloned and sequenced
	59F	24	
	60C	24	
	84F	7, 9, 12, 24	3 copies; cloned
tRNA-Asn6			
tRNA-Asn7			
tRNA-Asp1			
tRNA-Asp2	25D 29D-E 70A 96A 96B	12, 26 9, 12, 26, 25, 30 7, 9, 25 12 12	δ and γ forms cloned and sequenced
tRNA-Asp3			
tRNA-Asp4			
tRNA-Cys1			
tRNA-Cys2			
tRNA-Cys3			
tRNA-Cys4			
tRNA-Gln1			
tRNA-Gln2			
tRNA-Gln3			
tRNA-Gln4			
tRNA-Gln5			
tRNA-Glu1			
tRNA-Glu2			
tRNA-Glu3			
tRNA-Glu4	52F	9, 20	
(GAA binding)	56E-F	9, 19, 20, 30	3 copies, cloned and sequenced
	62A	4, 9, 16, 20	5 copies; cloned and sequenced
tRNA-Glu5			

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locus	location	ref ^α	comments
tRNA-Glu6			
tRNA-Gly1			
tRNA-Gly2	58A	13	
(GGA binding)	84C	13	
	90E	13	
tRNA-Gly3	22B-C	12, 23	
(GGC binding)	28D?	12	
	35B-C	9, 12	
	53E?	12	
	55E?	12	
	56E-F	9, 11, 15, 20, 30	2 copies; cloned and sequenced
	57B-C	12	
tRNA-His1	48F	4, 7, 9, 24	δ and γ forms; (CAC binding) cloned and sequenced
	56F	24	
tRNA-Ile	42A	9, 12, 17, 18, 24, 30, 33	1 copy
(AUU binding)	50A-B	22	5 copies; cloned and sequenced
tRNA-Ile1			
tRNA-Ile2			
tRNA-Ile3			
tRNA-Ile4			
tRNA-Leu1			
tRNA-Leu2	44F	13, 14	
	50A?	13	
	66B5-8	9, 12, 13, 14, 30	
	70A?	13	
	79F	13	
	95A	13	
tRNA-Leu3	50A-B	22	2 copies, cloned and sequenced; both have introns
tRNA-Lys1			
tRNA-Lys2	42A	9, 10, 12, 18, 24, 27, 30, 32, 33	5 copies; cloned and (AAG binding) sequenced
	42E1-2	9, 10, 11, 12, 30	4 copies
	44E	13	
	50B5-8	9, 12, 30	
	56E-f	11, 12	
	62A1-2	9, 12, 16, 20	
	63B1-2	12, 30	
	66B5-8	13	
	79F	13	
tRNA-Lys3			
tRNA-Lys4			
tRNA-Lys5	29A	5, 6, 12	may not be transcribed
(AAA binding)	84A-B	5, 6, 9, 12, 30	cloned and sequenced
	85B	12	
	87B	12	
tRNA-Lys6			
tRNA-Met1			
tRNA-Met2	48B5-7	9, 12, 30	2 copies
	63A?	9, 12, 24	
	72F1-2	9, 12, 30	2 copies
	83F3-84A2	9, 12, 30	
tRNA-Met3	19-20	12	<i>tRNA-Met3</i> is the initiator sequence; cloned and sequenced
(AUG binding)	42A?	9	
	42E?	9	
	46A1-2	12, 30	
	56E-F	12	
	61D1-2	9, 12, 28, 30	
	70F1-2	9, 12, 30	
tRNA-Phe1			
tRNA-Phe2	56F	3, 9, 20, 30	four posttranscriptionally modified forms
(UCC binding)	89B-C	1	cloned and sequenced
tRNA-Phe3			
tRNA-Pro1			
tRNA-Pro2			

locus	location	ref ^α	comments
tRNA-Pro3			
tRNA-Ser1			
tRNA-Ser2b	86A	13, 14, 31	
	88A9-12	13, 14	
	94A6-8	13, 14	
tRNA-Ser3			
tRNA-Ser4	12D-E	8, 9, 12, 31	cloned
	23E	8, 9, 12	cloned
	56D	12	
	64D	12	
tRNA-Ser5			
tRNA-Ser6			
tRNA-Ser7	12D-E	8, 9, 12, 31	cloned
	23E	8, 9, 12	cloned
	56D	12	
	64D	12	
tRNA-Thr1			
tRNA-Thr2			
tRNA-Thr3	47F	13, 14	
	87B	13, 14	
	93A1-2	13, 14	
tRNA-Thr4			
tRNA-Thr5			
tRNA-Thr6	56E-F	14	
	61F	14	
tRNA-Thr7			
tRNA-Thr8			
tRNA-Trp1			
tRNA-Trp2			
tRNA-Trp3			
tRNA-Tyr1	19F	13, 14	δ and γ forms
	22F-23A	13, 14	
	28C	13	
	41	13	
	42A	9, 12, 18, 24, 30, 33	
	42E	9, 30	
	50C1-4	13, 14	polymorphic
	85A	13, 14	
tRNA-Tyr2			
tRNA-Tyr3			
tRNA-Val1			
tRNA-Val2			
tRNA-Val3a	64D1-2	2, 9, 12, 30	cloned and sequenced
(GUA binding)			
tRNA-Val3b	84D3-4	2, 8, 9, 12, 21, 30	5 copies; cloned and sequenced
	90B-C	8, 9, 13, 24, 30	cloned
	92B1-11	9, 12, 30	4 copies
tRNA-Val4	56D3-7	9, 12, 30	
	70B-C	1, 8, 9, 12, 30	cloned and sequenced
	89B	1, 8, 9, 30	cloned and sequenced
	90B-C	1, 13, 21	cloned and sequenced
tRNA-Val5			
tRNA-Val6			
tRNA-Val7			

^α 1 = Addison, Astell, Delaney, Gillam, Hayashi, Miller, Rajpur, Smith, Taylor, and Tener, 1982, *J. Biol. Chem.* 257: 670-73; 2 = Addison, Gillam, Hayashi, and Tener, 1985, *Canadian J. Biochem. and Cell Biol.* 63: 176-82; 3 = Altweg and Kubli, 1979, *Nucl. Acids Res.* 7: 93-105; 4 = Altweg and Kubli, 1980, *Nucl. Acids Res.* 8: 215-23; 5 = Cribbs, Gillam, and Tener, 1982, *Nucl. Acids Res.* 10: 6393-6400; 6 = DeFranco, Burke, Hayashi, Tener, Miller, and Söll, 1982, *Nucl. Acids Res.* 10: 5799-5808; 7 = Dudler, Egg, Kubli, Artavanis-Tsakonas, Gehring, Steward, and Schedl, 1980, *Nucl. Acids Res.* 8: 2921-27; 8 = Dunn, Delaney, Gillam, Hayashi, Tener, Grigliatti, Misra, Spurr, Taylor, and Miller, 1979, *Gene* 7: 199-215; 9 = Elder, Szabo, and Uhlenbeck, 1980, *J. Mol. Biol.* 142: 1-17; 10 = Gergen, Loewenberg, and Wensink, 1981, *J. Mol. Biol.* 147: 475-99; 11 = Hayashi, Addison, Gillam, Grigliatti, and Tener, 1981, *Chromosoma* 82: 385-97; 12 = Hayashi, Gillam, Delaney, Dunn, Tener, Grigliatti, and Suzuki, 1980, *Chromosoma* 76: 65-84; 13 = Hayashi, Gillam, Grigliatti, and Tener, 1982, *Chromosoma* 86: 279-92; 14 = Hayashi, Gillam, Tener, Grigliatti, and Suzuki, 1980, *Genetics* 94: s42; 15 = Hershey and Davidson, 1980, *Nucl. Acids Res.* 8: 4899-4910; 16 = Hosbach, Silberklang, and

McCarthy, 1980, Cell 21: 169-78; 17 = Hovemann, Schmidt, Yamada, Silverman, Mao, DeFranco, and Söll, 1980, Transfer RNA (Abelson, Schimmel, and Söll, eds.). Cold Spring Harbor Laboratory, Cold Spring Harbor, NY, pp. 325-38; 18 = Hovemann Sharp, Yamada, and Söll, 1980, Cell 19: 889-95; 19 = Indik and Tartof, 1982, Nucl. Acids Res. 10: 4159-72; 20 = Kubli and Schmidt, 1978, Nucl. Acid Res. 5: 1465-78; 21 = Miller, Tener, Bradley, and Scraba, 1981, Gene 15: 361-64; 22 = Robinson and Davidson, 1981, Cell 23: 251-59; 23 = Schedl and Donelson, 1978, Biochim. Biophys. Acta 520: 539-54; 24 = Schmidt and Kubli, 1980, Chromosoma 80: 277-87; 25 = Schmidt, Egg, and Kubli, 1978, Mol. Gen. Genet. 164: 249-54; 26 = Schmidt, Mao, Silverman, Hovemann, and Söll, 1978, Proc. Nat. Acad. Sci. USA 75: 4819-23; 27 = Silverman, Gillam, Tener, and Söll, 1979, Nucl. Acids Res. 6: 435-42; 28 = Silverman, Heckman, Cowling, Delaney, Dunn, Gillam, Tener, Söll, and Ray, 1979, Nucl. Acids Res. 6: 421-33; 29 = Silverman, Schmidt, Söll, and Hovemann, 1979, J. Biol. Chem. 254: 10290-94; 30 = Tener, Hayashi, Dunn, Delaney, Gillam, Griati, Kaufman, and Suzuki, 1980, Transfer RNA (Abelson, Schimmel, and Söll, eds.). Cold Spring Harbor Laboratory, Cold Spring Harbor, NY, pp. 295-307; 31 = White, Dunn, Gillam, Tener, Armstrong, Skoog, Frihart, and Leonard, 1975, J. Biol. Chem. 251: 515-21; 32 = Yen and Davidson, 1980, Cell 19: 889-95; 33 = Yen, Sodja, Cohen, Conrad, Wu, Davidson, and Ilgen, 1977, Cell 11: 763-77.

ro: rough

location: 3-91.1.

references: Pilkington, 1941, Proc. Zool. Soc. London A 111: 199-222.

Ready, Hanson, and Benzer, 1976, Dev. Biol. 53: 217-40 (fig.).

Meyerowitz and Kankel, 1978, Dev. Biol. 62: 112-42 (fig.).

Garen and Kankel, 1983, Dev. Biol. 96: 445-66.

Tomlinson, Kimmel, and Rubin, 1988, Cell 55: 771-84.

Renfranz, and Benzer, 1989, Dev. Biol. 136: 411-29.

phenotype: Homozygotes have rough eyes; slightly smaller and narrower than wild type. Approximately normal numbers of facets but arrangement irregular; frequently have fewer and sometimes more than eight retinula cells; some facets missing such that three ommatidial bristles juxtaposed; other facets fused (Stemm-Tegethoff and Dicke, 1974, Theoret. Appl. Genet. 44: 262-65; Ready *et al.*). Fiber pathways through lamina and into medulla in considerable disarray; optic chiasma between the two replaced by parallel fibers; laminar cartridge and medullar columns deranged, ventral epithelial nuclear row absent; medulla displaced from normal position and rotated anteriorly. Mosaic studies demonstrate that phenotype is eye autonomous; *i.e.*, the genotype of the eye dictates that of the underlying nervous elements. Expression, as determined by *in situ* hybridization found in the eye-antenna imaginal disk, especially in the region of the morphogenetic furrow, as well as in a specific area of the brain. No evidence of embryonic expression (Saint, Kalionis, Lockett, and Elizur, 1988, Nature (London) 334: 151-54). RK1.

alleles:

allele	origin	discoverer	ref ^α	comments
<i>ro</i> ^{1β}	spont	Muller, 13f	1, 5	insertion of middle-repetitive DNA into intron 5' to homeobox
<i>ro</i> ²	X ray	Brosseau	2	
<i>ro</i> ³	X ray	Brosseau	2	
<i>ro</i> ⁴	X ray	Brosseau	2	
<i>ro</i> ^{64c}	X ray	Puro	3, 4	recombination normal
<i>ro</i> ^{π13γ}	P-element		6	
<i>ro</i> ^{v1}	X ray	Brosseau	2	heterochromatin,

allele	origin	discoverer	ref ^α	comments
<i>ro</i> ^{v2}	X ray	Brosseau	2	91D rearrangement heterochromatin,
<i>ro</i> ^w	EMS		5	91E rearrangement nonsense mutation in homeobox

^α 1 = Bridges and Morgan, 1923, Carnegie Inst. Wash. Publ. No. 327: 93 (fig.). 2 = Brosseau, 1970, DIS 45: 100; 3 = Puro, 1980, DIS 58: 205; 4 = Puro and Nygrén, 1982, Hereditas 81: 231-48; 5 = Saint, Kalionis, Lockett, and Elizur, 1988, Nature (London) 334: 151-54 (fig.); 6 = Tomlinson, Kimmel, and Rubin, 1988, Cell 55: 771-84.

^β Contains a >10 kb insertion homologous to *412* in intron 1 (Tomlinson *et al.*, 1988).

^γ Contains a P-element insertion in exon 3 (Tomlinson *et al.*, 1988). P-element excised in a revertant of this allele.

cytology: Placed in 97D1-9 based on its inclusion in *Df(3R)ro-XB3 = Df(3R)97D1-2;97D9*. Restricted to 97D5-7 by *in situ* hybridization (Tomlinson *et al.*, 1988).

molecular biology: Gene or part thereof isolated from a genomic library using a 33 base-pair homeobox consensus sequence as a probe. Isolated homeobox sequence shows 57 and 58% homology to those of *Antp* and *Scr* (Saint *et al.*). The complete *ro* gene was isolated by Tomlinson *et al.* (1988) by P-element transposon tagging. A mutant *ro*^{π13} was rescued by transformation with an 8.6 kb genomic fragment carrying the gene. The *ro* locus contains three exons and two introns and the transcription unit spans about 4.3 kb. The homeobox domain is encoded in exon 2 and *opa*-like repeats are encoded in exons 1 and 3. The complete sequence of the *ro* gene was obtained, and the putative sequence of its 350-amino-acid homeobox protein determined (Tomlinson *et al.*, 1988).

ro-63: see *un*³

rod: rough deal

location: 3-105.1 (to right of *ca*).

origin: Induced by hybrid dysgenesis (*rod*^{H4.8}) or γ rays (*rod*^{X1-4}).

references: Karess and Glover, 1989, J. Cell Biol. 109: 2951-61.

phenotype: Mutants show mitotic abnormalities in larval neuroblasts; there is a high frequency of both aneuploid cells and abnormal anaphase figures, with lagging chromatids, anaphase bridges, and stretched chromatid arms. Abnormal chromosome behavior also occurs in the second meiotic anaphase in male meiosis, although motile sperm are produced. All surviving homozygous females and 90% of surviving homozygous males are sterile and have roughened eyes, sparse abdominal bristles, and notched wings.

alleles:

allele	% abnormal anaphases	%homozygous viable
<i>rod</i> ^{H4.8}	20.2	25
<i>rod</i> ^{X-1}	42.5	0
<i>rod</i> ^{X-2}	20.7	27
<i>rod</i> ^{X-3}	15.0	49
<i>rod</i> ^{X-4}	13.9	73

cytology: Placed in 100B5-D1 based on the presence of the normal allele in *Dp(3;1)48 (=Df(3R)ca48) = Dp(3;1)100B7-8;100F* as well as in *Df(3R)ill-e = Df(3R)100A1-2;100B5-9* and *Df(3R)Kpn-A =*

Df(3R)100D1;100E and a synthetic deficiency for 100D-F.

roe: roughened eye

location: 3-47.6 [proximal to *rn* (Agnel, Kerridge, Vola, and Griffin-Shea, 1989, Genes Dev. 3: 85-95)].

references: Cavener, Otteson, and Kaufman, 1986, Genetics 114: 111-23 (fig.).

Renfranz and Benzer, 1989, Dev. Biol. 136: 411-29.

phenotype: Produces a slight roughening of the eye owing to the irregular disposition of the facets; interommatidial bristles lost from posterior portion of the eye. Extreme expression in mutant/deficiency heterozygotes and more so in deficiency homozygotes; enhanced roughening and posterior loss of ommatidial bristles; in addition the eye is reduced in size and remaining interommatidial bristles are clumped. RK1.

alleles: All alleles appear to be hypomorphic in that the phenotype of mutant/deficiency is more severe than of homozygous mutants.

allele	origin	discoverer	synonym	ref ^α	comments
<i>roe</i> ¹	spont	Markowitz, 61g		1, 2	
<i>roe</i> ²	EMS	Grigliatti		1	
<i>roe</i> ³	EMS	Grigliatti	<i>roe</i> ^{5.15}	1	

^α 1 = Lewis, Kaufman, Denell, and Talerico, 1980, Genetics 95: 367-81; 2 = Markowitz, 1963, DIS 38: 31.

cytology: Placed in 84D2 based on its inclusion in *Df(3R)D6 = Df(3R)84D2-3;84F13-16* but not *Df(3R)dsx10M = Df(3R)84D3;84F1-2*. *Df(3R)D6/Df(3R)Scx4*, which is deficient for both *roe* and *rn*, survives with an extreme *roe* phenotype.

Roi: Rough eye

location: 2- [left arm, not separated from *ln(2L)t*].

origin: Spontaneous in *ln(2L)t*.

discoverer: Ives, 47k18.

references: 1952, DIS 26: 65.

1956, DIS 30: 72.

Renfranz and Benzer, 1989, Dev. Biol. 136: 411-29.

phenotype: Eye facets of *Roi/+* irregularly rounded, sometimes enlarged; eyes sometimes bulge. *Roi/Roi* lethal; *Roi/S* viable. Acts as a partial suppressor of *B* (E. H. Grell). Viability good. RK2A.

cytology: Tentatively placed in 36F7-37B8 on the basis of its failure to survive in heterozygous combination with *Df(2L)TW3 = Df(2L)36F7-37A1;37B2-8* (Voelker and Langley, 1978, DIS 53: 185).

rol: reduced optic lobes (J.C. Hall)

location: 1-52.

origin: Induced by ethyl methanesulfonate.

discoverer: Heisenberg.

references: Wolf and Heisenberg, 1986, Nature (London) 323: 154-56.

Coombe, 1986, J. Comp. Physiol. 159: 655-65.

Heisenberg and Wolf, 1988, J. Comp. Physiol. 163: 373-88.

phenotype: Relatively proximal portion of optic lobes, except for lamina, reduced in volume to approximately 40-50% normal; less severe reduction than in *sol*; *rol sol* double mutant has extremely small visual system (connecting eye to central brain), whose volume is some 12% of normal (Wolf and Heisenberg, 1986) and whose lobula plate optic ganglion is absent (Heisenberg, unpublished);

a triple mutant, with *mnb* added to the two just noted, has diminished amplitudes of light-on and light-off transient spikes in electroretinogram (Coombe, 1986). Behavioral experiments reported for *rol sol* [a single mutant involving only the former exhibits only subtle defects in visual behavior (Heisenberg, unpublished)]; the double mutant is blind in terms of standard optomotor responses to rotating vertical stripes, but it can respond to the positions of landmarks by making turning maneuvers (Wolf and Heisenberg, 1986); these optic-lobe-depleted flies can make use of the magnitudes, though not the directions, of moving patterns to stabilize the panorama presented to them; they do so apparently by sampling the optomotor torque value which has the effect of putting them in optomotor balance; thus, it is concluded that the double mutant's visual orientation is an operant behavior (Wolf and Heisenberg, 1986); same conclusion arrived at in experiments testing this double mutant's visually mediated course control during tethered flight (Heisenberg and Wolf, 1988).

allele: One allele, *rol*¹, isolated as *rol*^{KS221}.

rolled: see *rl*

rolled up: see *rlu*

Roof: see *Rf*

roof wings: see *rf*

rosd: see *Fs(3)Sz30*

rose: see *rs*

rosy: see *ry*

Rosy: see *bw*^{V4}

rotated abdomen: see *rt*

rotated penis: see *rp*

rotund: see *rn*

rough: see *ro*

rough deal: see *rod*

rough eye: see *rey*

Rough eye: see *Roi*

rough III: see *dft*

Rough wing: see *Rw*

rough-64: see *rg*²

roughened eye: see *roe*

Roughened: see *R*

roughest: see *rst*

roughestlike: see *rstl*

roughex: see *rux*

rough-eye-like: see *rey*³

roughish: see *rg*¹

roughish: see *rh*

roughoid: see *ru*

rox: see *rg*⁶

***rp: rotated penis**

location: 3-41.7.

origin: Spontaneous.

discoverer: Bridges, 29c15.

references: Morgan, Sturtevant, and Bridges, 1929, Year Book – Carnegie Inst. Washington 28: 339.

phenotype: As viewed from behind, external genitalia of male rotated counterclockwise from 0° to 270°, usually about 180°; overlaps wild type in 30% of flies. Eyes rough. Fly small; legs weak; tergites ridged; abdomen narrowed. Male sterile, even when genitalia not rotated. RK3.

Rp21: Ribosomal protein 21

location: 3-{47}.

references: Biessmann, Kuger, Schropfer, and Spindler, 1981, Chromosoma 82: 493-503.

Kay, Zhang, and Jacobs-Lorena, 1988, Mol. Gen. Genet. 213: 354-58.

phenotype: Cloned *Rp21* DNA encodes a ribosomal protein of 26,000 daltons (Kay *et al.*, 1988). When transformed into the *Drosophila* germ line using *P* elements, the *Rp21* gene is expressed in the transgenic flies as an increase in the level of the corresponding mRNA.

cytology: *Rp21* located in 3L at 80 by *in situ* hybridization of the transformed copy to the salivaries.

molecular biology: Gene coding for an abundant small RNA cloned by Biessman *et al.* (1981) and Kay *et al.* (1988) (see discussion under phenotype). Although mapped to the same region as *M(3)80*, the transformed *Rp21* gene did not complement this Minute (Kay *et al.*, 1988).

Rp49: see *M(3)99D*

RpA1: Ribosomal protein A1

location: 2-{78}.

references: Kay and Jacobs-Lorena, 1985, Mol. Cell. Biol. 5: 3583-92.

Qian, Zhang, Kay, and Jacobs-Lorena, 1987, Nucl. Acids Res. 15: 987-1003.

Qian, Hongo, and Jacobs-Lorena, 1988, Proc. Nat. Acad. Sci. USA 85: 9601-05.

phenotype: Encodes protein homologous to "A" family of eucaryotic ribosomal proteins involved in the initiation and elongation steps of protein synthesis (Qian *et al.*, 1987). mRNA of the *RpA1* gene is regulated at the level of translation; it is associated with polysomes during oogenesis and late embryonic stages (Kay and Jacobs-Lorena, 1985). An antisense *RpA1* gene (carrying a heat shock promoter), transformed into the fly genome, disrupts oogenesis and results in female sterility (Qian *et al.*, 1988).

cytology: Located in 53C1-6 by *in situ* hybridization to the salivaries (Socolitch). Single copy gene indicated.

molecular biology: Cloned DNA sequence coding for *RpA1* protein identified; complete nucleotide sequence and deduced amino acid sequence determined (Qian *et al.*, 1987). *RpA1* gene has no introns. The amino acid sequence of *RpA1* shows significant identity to the amino acid sequences of the r-proteins of rat, shrimp, and yeast;

all of these r-proteins are acidic.

other information: Not allelic to *M(2)53*.

RpII140: RNA polymerase II-140 kd subunit

location: 3- {54}.

origin: Recovered by cross hybridization with yeast gene clone.

synonym: *wimp*.

references: Faust, Renkawitz-Pohl, Falkenburg, Gasch, Bialojan, Young, and Bautz, 1986, EMBO J. 5: 741-46.

phenotype: The structural gene for the 140-kd subunit of RNA polymerase II [RNA nucleotidyl transferase (EC 2.7.7.6)]. The gene is highly conserved judging from shared sequence homology with the yeast 150-kd subunit gene.

alleles: Mutants selected by Mortin, (1990, Proc. Nat. Acad. Sci. USA 87: 4864-68) as extragenic suppressors of *RpII215^{K1}*.

cytology: Placed in 88A-B by *in situ* hybridization.

molecular biology: Clone isolated from a genomic library using as a probe the gene for the yeast 150-kd subunit of RNA polymerase II. Antibodies raised to a fusion protein made in an expression vector react with *Drosophila* RNA polymerase II, and the fusion protein has spots in common with RNA polymerase II in peptide maps. Polyadenylated RNA of 3.9 kb transcribed by gene; very little space for untranslated sequences. High degree of homology in conceptual amino-acid sequence between yeast and *Drosophila* subunits.

RpII215: RNA polymerase II-215 kd subunit

location: 1-35.66 [based on 320 recombinants between *v* and *m* (Greenleaf *et al.*, 1980)].

synonym: *l(1)L5*.

references: Greenleaf, Borsett, Jiamachello, and Coulter, 1979, Cell 18: 13-22.

Greenleaf, Weeks, Voelker, Ohmishi, and Dickson, 1980, Cell 21: 785-92.

Greenleaf, 1983, J. Biol. Chem. 258: 13403-06.

phenotype: The structural gene encoding the 215 kd subunit of RNA polymerase II [RNA nucleotidyl transferase (EC 2.7.7.6)]. This subunit highly conserved as inferred from the cross reaction of antiserum from the large subunit of calf RNA polymerase II with enzyme isolated from *Drosophila* as well as that from yeast and wheat germ (Carrol and Stollar, 1983, J. Mol. Biol. 170: 777-90); also amino-acid sequence homology detected with the β' subunit of *E. coli* RNA polymerase (Biggs, Searles, and Greenleaf, 1985, Cell 42: 611-21). Dosage compensated at the level of transcription (Faust, Penkawitz-Pohl, Falkenberg, Gasch, Biabjah, Young, and Bautz, 1986, EMBO J. 5: 741-46).

alleles: Most alleles are recessive lethals; however several viable alleles and others with dominant phenotypes are described at the end of this entry. These alleles all enhance expression of *Ubx* in *RpII215/+* heterozygotes with the effect of + (no effect) < *RpII215^{H1}* < *RpII215⁷* < *RpII215^{K2}* < *RpII215⁴* < *RpII215^{Ubl}*. Heteroallelic combinations of these mutants produce either a reduced effect when *RpII215^{Ubl}* is involved or no effect in other combinations.

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allele	origin	discoverer	synonym α	ref β	comments γ
Rpl1215¹	X ray	Lefevre	<i>l(1)L5, Ublⁿ</i>	5, 10	
Rpl1215² δ	EMS		<i>Rpl1215^{A9}</i>	12	heat sensitive, haplospecific
Rpl1215³ δ	EMS		<i>Rpl1215^{A11}</i>	12, 4	haplospecific
Rpl1215⁴	EMS		<i>Rpl1215^{C4}</i>	1, 3, 4, 12	viable
Rpl1215⁵	EMS		<i>Rpl1215^{C8}</i>	12, 4	
Rpl1215⁶	EMS		<i>Rpl1215^{C9}</i>	12, 4	
Rpl1215⁷	EMS	Baker	<i>Rpl1215^{C11-138}</i>	12	cold sensitive
Rpl1215⁸ δ	EMS		<i>Rpl1215^{C20}</i>	12, 4	heat sensitive, haplospecific
Rpl1215⁹ ζ	dysgenesis		<i>Rpl1215^{D50}</i>	11	P insert at 0.1
Rpl1215¹⁰ ϵ	EMS		<i>Rpl1215^{D150}</i>	12	
Rpl1215¹¹ ϵ	EMS		<i>Rpl1215^{D308}</i>	12	
Rpl1215¹² δ	EMS		<i>Rpl1215^{E28}</i>	12	heat sensitive, haplospecific
Rpl1215¹³	EMS		<i>Rpl1215^{F4}</i>	12	
Rpl1215¹⁴ δ	EMS		<i>Rpl1215^{F6}</i>	12	
Rpl1215¹⁵	EMS		<i>Rpl1215^{F7}</i>	12	
Rpl1215¹⁶ δ	EMS		<i>Rpl1215^{F8}</i>	12	
Rpl1215¹⁷	EMS		<i>Rpl1215^{F9}</i>	12	
Rpl1215¹⁸	EMS		<i>Rpl1215^{F12}</i>	12	
Rpl1215¹⁹ δ	EMS		<i>Rpl1215^{F36}</i>	12	
Rpl1215²⁰	EMS		<i>Rpl1215^{F48}</i>	12	
Rpl1215²¹ δ	EMS		<i>Rpl1215^{F50}</i>	12	
Rpl1215²²	EMS		<i>Rpl1215^{F51}</i>	12	
Rpl1215²³ δ	EMS		<i>Rpl1215^{F60}</i>	12	
Rpl1215²⁴	EMS		<i>Rpl1215^{F69}</i>	12	
Rpl1215²⁵	EMS		<i>Rpl1215^{F76}</i>	12	
Rpl1215²⁶	EMS		<i>Rpl1215^{F89}</i>	12	
Rpl1215²⁷ δ	EMS		<i>Rpl1215^{F90}</i>	12	
Rpl1215²⁸	EMS		<i>Rpl1215^{F103}</i>	12	
Rpl1215²⁹	EMS		<i>Rpl1215^{F106}</i>	12	
Rpl1215³⁰	EMS		<i>Rpl1215^{F118}</i>	12	
Rpl1215³¹	EMS		<i>Rpl1215^{F125}</i>	12	
Rpl1215³²	EMS		<i>Rpl1215^{F128}</i>	12	
Rpl1215³³	EMS		<i>Rpl1215^{F129}</i>	12	
Rpl1215³⁴	EMS		<i>Rpl1215^{F134}</i>	12	
Rpl1215³⁵	EMS		<i>Rpl1215^{G1}</i>	12	
Rpl1215³⁶	EMS		<i>Rpl1215^{G2}</i>	12	
Rpl1215³⁷	EMS		<i>Rpl1215^{G3}</i>	12	
Rpl1215³⁸	EMS		<i>Rpl1215^{G8}</i>	12	
Rpl1215³⁹	ENU		<i>Rpl1215^{H2}</i>	12	heat sensitive
Rpl1215⁴⁰	ENU		<i>Rpl1215^{H14}</i>	12	heat & cold sensitive
*Rpl1215⁴¹	ENU		<i>Rpl1215^{H19}</i>	12	
Rpl1215⁴²	ENU		<i>Rpl1215^{H20}</i>	12	
Rpl1215⁴³	ENU		<i>Rpl1215^{H21}</i>	12	
Rpl1215⁴⁴	ENU		<i>Rpl1215^{H22}</i>	12	
Rpl1215⁴⁵	ENU		<i>Rpl1215^{H24}</i>	12	
Rpl1215⁴⁶	ENU		<i>Rpl1215^{H25}</i>	12	
Rpl1215⁴⁷	ENU		<i>Rpl1215^{H26}</i>	12	
Rpl1215⁴⁸	ENU		<i>Rpl1215^{H33}</i>	12	
Rpl1215⁴⁹	ENU		<i>Rpl1215^{H34}</i>	12	
Rpl1215⁵⁰	ENU		<i>Rpl1215^{H35}</i>	12	
Rpl1215⁵¹	ENU		<i>Rpl1215^{H36}</i>	12	
Rpl1215⁵²	ENU		<i>Rpl1215^{H37}</i>	12	
Rpl1215⁵³	ENU		<i>Rpl1215^{H38}</i>	12	
Rpl1215⁵⁴	ENU		<i>Rpl1215^{H101}</i>	12	
Rpl1215⁵⁵	ENU		<i>Rpl1215^{H445}</i>	12	
Rpl1215⁵⁶	ENU		<i>Rpl1215^{H1239}</i>	12	
Rpl1215⁵⁷	ENU		<i>Rpl1215^{H1717}</i>	12	
Rpl1215⁵⁸	ENU		<i>Rpl1215^{H3006}</i>	12	
Rpl1215⁵⁹	ENU		<i>Rpl1215^{H3116}</i>	12	
Rpl1215⁶⁰	EMS		<i>Rpl1215^{K6}</i>	12	
Rpl1215⁶¹	EMS		<i>Rpl1215^{K8}</i>	12	heat & cold sensitive
Rpl1215⁶²	EMS		<i>Rpl1215^{K12}</i>	12	
Rpl1215⁶³	EMS		<i>Rpl1215^{K17}</i>	12	
Rpl1215⁶⁴	EMS		<i>Rpl1215^{K18}</i>	12	
Rpl1215⁶⁵	EMS		<i>Rpl1215^{K21}</i>	12	
Rpl1215⁶⁶	EMS		<i>Rpl1215^{K26}</i>	12	heat sensitive
Rpl1215⁶⁷	ENU		<i>Rpl1215^{M1}</i>	12	
Rpl1215⁶⁸	ENU		<i>Rpl1215^{M3}</i>	12	
Rpl1215⁶⁹	ENU		<i>Rpl1215^{M8}</i>	12	
Rpl1215⁷⁰	ENU		<i>Rpl1215^{M17}</i>	12	
Rpl1215⁷¹	ENU		<i>Rpl1215^{M18}</i>	12	
Rpl1215⁷²	ENU		<i>Rpl1215^{M42}</i>	12	
Rpl1215⁷³	ENU		<i>Rpl1215^{M43}</i>	12	

allele	origin	discoverer	synonym ^α	ref ^β	comments ^γ
<i>RpII215</i> ⁷⁴	ENU		<i>RpII215</i> ^{M45}	12	
<i>RpII215</i> ⁷⁵	ENU		<i>RpII215</i> ^{M47}	12	
<i>RpII215</i> ⁷⁶	ENU		<i>RpII215</i> ^{M48}	12	heat & cold sensitive
<i>RpII215</i> ⁷⁷	ENU		<i>RpII215</i> ^{M49}	12	
<i>RpII215</i> ⁷⁸	ENU		<i>RpII215</i> ^{M54}	12	
<i>RpII215</i> ⁷⁹	ENU		<i>RpII215</i> ^{M66}	12	
<i>RpII215</i> ⁸⁰	ENU		<i>RpII215</i> ^{M69}	12	
<i>RpII215</i> ⁸¹	ENU		<i>RpII215</i> ^{M81}	12	
<i>RpII215</i> ⁸² ζ	dysgenesis		<i>RpII215</i> ^{W21}	11, 12	P insert 0 to 0.9
<i>RpII215</i> ⁸³ ζ	dysgenesis		<i>RpII215</i> ^{W38}	11, 12	P insert -5 to -7
<i>RpII215</i> ⁸⁴ ζ	dysgenesis		<i>RpII215</i> ^{W42}	11, 12	P insert 0 to 0.9
* <i>RpII215</i> ⁸⁵	dysgenesis		<i>RpII215</i> ^{W49}	12	
<i>RpII215</i> ⁸⁶ ζ	dysgenesis		<i>RpII215</i> ^{W81}	11, 12	P insert -5 to -7
<i>RpII215</i> ⁸⁷ ζ	dysgenesis		<i>RpII215</i> ^{W83}	12	
<i>RpII215</i> ⁸⁸	dysgenesis		<i>RpII215</i> ^{W86}	12	
<i>RpII215</i> ⁸⁹ ζ	dysgenesis		<i>RpII215</i> ^{W94}	12	
<i>RpII215</i> ⁹⁰	dysgenesis		<i>RpII215</i> ^{W105}	12	
<i>RpII215</i> ⁹¹ ζ	dysgenesis		<i>RpII215</i> ^{W110}	12	
<i>RpII215</i> ⁹² ζ	dysgenesis		<i>RpII215</i> ^{W151}	12	
<i>RpII215</i> ⁹³	dysgenesis		<i>RpII215</i> ^{W173}	12	
<i>RpII215</i> ⁹⁴ ζ	dysgenesis		<i>RpII215</i> ^{W182}	12	
<i>RpII215</i> ⁹⁵ ζ	dysgenesis		<i>RpII215</i> ^{W183}	12	
<i>RpII215</i> ⁹⁶	EMS		<i>l(1)v8</i>	2	
<i>RpII215</i> ⁹⁷	EMS		<i>l(1)v9</i>	2	
<i>RpII215</i> ⁹⁸	EMS		<i>l(1)v10</i>	2	
<i>RpII215</i> ⁹⁹	EMS		<i>l(1)v83</i>	2	
<i>RpII215</i> ¹⁰⁰	EMS		<i>l(1)v84</i>	2	
<i>RpII215</i> ¹⁰¹	EMS		<i>l(1)v216</i>	2	
<i>RpII215</i> ¹⁰²	EMS		<i>l(1)v219</i>	2	
<i>RpII215</i> ¹⁰³	EMS		<i>l(1)v221</i>	2	
<i>RpII215</i> ¹⁰⁴	X ray	Lefevre	<i>l(1)HC212</i>	6	
<i>RpII215</i> ¹⁰⁵	X ray	Lefevre	<i>l(1)N40</i>	6	
<i>RpII215</i> ¹⁰⁶	EMS	Lefevre	<i>l(1)DC783, Ubl^h</i>	7, 10	
<i>RpII215</i> ¹⁰⁷	EMS	Lefevre	<i>l(1)DC811</i>	7	
<i>RpII215</i> ¹⁰⁸	EMS	Lefevre	<i>l(1)DF912, Ublⁿ²</i>	7, 10	
<i>RpII215</i> ¹⁰⁹	EMS	Lefevre	<i>l(1)DF940, Ubl^m</i>	7, 10	
<i>RpII215</i> ¹¹⁰	EMS	Lefevre	<i>l(1)VA113</i>	7	
<i>RpII215</i> ¹¹¹	EMS	Lefevre	<i>l(1)VE778</i>	7	
<i>RpII215</i> ¹¹²	EMS	Lefevre	<i>l(1)VE811</i>	7	
<i>RpII215</i> ¹¹³	EMS	Lefevre	<i>l(1)VE819</i>	7	
<i>RpII215</i> ¹¹⁴	EMS	Lefevre	<i>l(1)VE851</i>	7	
<i>RpII215</i> ¹¹⁵	EMS	Lefevre	<i>l(1)VE895</i>	7	
<i>RpII215</i> ¹¹⁶	EMS	Lefevre	<i>l(1)VE919</i>	7	
<i>RpII215</i> ^{H1}	EMS	Huang	<i>RpII215</i> ^{JH1}	9	viable
<i>RpII215</i> ^{K1}	EMS	Kim	<i>RpII215</i> ^{WJK1}	9	t.s. lethal
<i>RpII215</i> ^{K2}	EMS	Kim	<i>RpII215</i> ^{WJK2}	9	viable; female sterile
<i>RpII215</i> ^{ts}	EMS		<i>l(1)Fb40, Ubl^{ts}</i>	1, 8	
<i>RpII215</i> ^{Ubl}	EMS		<i>l(1)MGM179, Ubl</i>	10	

^α Alleles with synonymic designations A through F, except *RpII215*⁴ and *RpII215*⁷, are derivatives of *RpII215*⁴.

^β 1 = Coulter and Greenleaf, 1982, J. Biol. Chem. 257: 1945-52; 2 = Geer, Lischwe, and Murphy, 1983, J. Exp. Zool. 225: 107-18; 3 = Greenleaf, Borsett, Jiamachello, and Coulter, 1979, Cell 18: 613-22; 4 = Greenleaf, Weeks, Voelker, Ohnishi, and Dickson, 1980, Cell 21: 785-92; 5 = Lefevre, 1971, Genetics 67: 497-513; 6 = Lefevre, 1981, Genetics 99: 461-80; 7 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 8 = Mortin and Kaufman, 1982, Mol. Gen. Genet. 187: 120-25; 9 = Mortin, Kim, and Huang, 1988, Genetics 119: 863-73; 10 = Mortin and Lefevre, 1981, Chromosoma 82: 237-47 (fig.); 11 = Searles, Jakerst, Bingham, Voelker, and Greenleaf, 1982, Cell 31: 585-92; 12 = Voelker, Wisely, Huang, and Gyurkovic, 1985, Mol. Gen. Genet. 201: 437-45.

^γ Haplo-specific lethals survive, at least with a low frequency, as homozygotes, but are lethal as hemizygotes.

^δ Lethal derivatives of *RpII215*⁴ that retain alpha-amanitin resistance.

^ε Derivatives of *RpII215*⁴ chosen for reduced resistance to alpha amanitin.

^ζ P-element insertion in 10B-C.

cytology: Placed in 10C2-5 based on its being in the region of overlap between *Df(1)m259 = Df(1)10C2-3;10E1-2* and *Df(1)vN48 = Df(1)9F;10C3-5* (Voelker, Wisely, Huang, and Gyurkovic, 1985, Mol. Gen. Genet. 201: 437-45).

molecular biology: Region cloned and restriction mapped (Searles, Jakerst, Bingham, Voelker, and Greenleaf, 1982, Cell 31: 585-92) and a 7 kb transcript identified as that encoding *RpII215* polypeptide (Ingles, Biggs, Wong, Weeks, and Greenleaf, 1983, Proc. Nat. Acad. Sci. USA 80: 3396-3400); four exons separated by three relatively small introns; 5' exon of 500 bp followed by 600 bp intron; the next three introns larger (2.3, 2.1, and 1.8 kb)

separated by introns of 100 and 50 bp. 0 coordinate of walk is *SstI* site within the small 5' exon of *RpII215*; polarity with respect to chromosome not determined. *RpII215* transcript extends from 0.2 at the 5' end to -7.2; first large exon sequenced (Biggs, Searles, and Greenleaf, 1985, Cell 42: 611-21).

*RpII215*⁴

synonym: *RpII215*^{C4}.

references: Greenleaf, Borsett, Jiamachello, and Coulter, 1979, Cell 18: 13-22.

Greenleaf, Weeks, Voelker, Ohnishi, and Dickson, 1980, Cell 21: 785-92.

Mortin, Kim, and Huang, 1988, Genetics 119: 863-73.

phenotype: This allele was selected as an amanitin resistant mutation; *Rpl1215*^{4/+} females produce equal amounts of amanitin-sensitive and -resistant enzyme and are themselves amanitin resistant. Generally a viable allele, but survival in combination with *Rpl1215*⁶³ and *Rpl1215*⁸⁶ reduced, and with *Rpl1215*⁵¹ and *Rpl1215*⁷⁸ survival is zero. RNA polymerase isolated from embryos homozygous for this allele is 250 times less sensitive to inhibition by alpha amanitin than that from wild type; resistance attributable to reduced amanitin binding; otherwise RNA polymerase normal in all respects except that there is some reduction in activity in the presence of Mg²⁺; stable to thermal denaturation (Coulter and Greenleaf, 1982, J. Biol. Chem. 157: 1945-52). Flies carrying both *Rpl1215*⁴ and *Rpl1215*⁺ display an Ultrabithorax-like phenotype, with enlarged halteres, and an enhancing effect on expression of *Ubx* [Greenleaf *et al.*, 1980; Voelker, Wisely, Huang, and Gyurkovics, 1985, Mol. Gen. Genet. 201: 437-45 (fig.)]. Measurements of haltere size in *TM6/+* females of various constitutions produced the following: *4/+/+* > *4/+* > *4/4/+* > *4/4* = *4/0* = *+/+*. The haltere effect of *Rpl1215*⁴ is abolished in heteroallelic combination with *Rpl1215*⁷, *Rpl1215*^{H1}, and *Rpl1215*^{K2}. *Rpl1215*⁴ decreases the *Ubx* effect of *Rpl1215*^{Ubl}. Heterozygotes also exhibit increased numbers of duplicated bristles in *Dll/+* (Mortin *et al.*). Males carrying *Rpl1215*⁴ on their X plus a normal allele on *Dp(1;Y)B*^{S⁻v⁺y⁺} are nearly sterile, and those carrying the normal allele instead on *Dp(1;2)v*^{65b} are sterile (Voelker *et al.*).

***Rpl1215*⁷**

synonym: *Rpl1215*^{C11-138}, *Ubl*^{C11}.

references: Voelker, Wisely, Huang, and Gyurkovics, 1985, Mol. Gen. Genet. 201: 437-45.

Mortin, Kim, and Huang, 1988, Genetics 119: 863-73.

phenotype: Cold-sensitive lethal; survives at 28° with nearly wild-type viability, but is lethal at lower temperatures. Surviving males are fertile, whereas homozygous females are sterile. Displays slight increase in haltere size in *Rpl1215*^{7/+}; *Ubx/+* females. Lethal in combination with *Rpl1215*^{Ubl}. *Ubx* effect inhibited in *trans* heterozygotes with other interacting alleles. In viable heterozygotes, causes an increase in the number of duplicated bristles in *Dll/+* and, in males carrying a duplicated normal allele, produces sex combs on the middle legs.

***Rpl1215*¹⁰⁶**

synonym: *Ubl*^h.

references: Mortin and Lefevre, 1981, Chromosoma 82: 237-47.

phenotype: Homozygous lethal; *Rpl1215*¹⁰⁶/*Rpl1215*¹⁰⁶ females with a duplication for *Rpl1215*⁺ display about 50% normal viability, and are fertile when crossed to wild type, but nearly sterile in crosses to *Rpl1215*^{Ubl} males with a duplication for *Rpl1215*⁺; all surviving progeny of the latter cross display a hyperabdominal-like phenotype; the females lack metathoracic legs and/or halteres in addition to having extra abdominal tissue; this phenotype barely detectable in offspring of crosses to normal males.

***Rpl1215*¹⁰⁹**

synonym: *Ubl*^m.

references: Mortin and Lefevre, 1981, Chromosoma 82: 237-47.

phenotype: Homozygous lethal; *Rpl1215*¹⁰⁹/*Rpl1215*¹⁰⁹/*Dp(1;1)Rpl1215*⁺ females display about 50% normal viability, are fertile when crossed to wild type, but fertility only 50% normal in crosses to *Rpl1215*^{Ubl}/*Dp(1;1)Rpl1215*⁺ males; offspring from the latter cross have darkened eyes, thin bristles, and are sterile.

***Rpl1215*^{H1}**

phenotype: Homozygous and hemizygous viable. Exhibits slight enhancement of *Ubx* when heterozygous to + but not in heteroallelic combination with any of the interacting mutant alleles; it decreases the enhancing effects of *Rpl1215*^{Ubl}. Increases the number of duplicated bristles of *Dll/+* in all genotypes.

***Rpl1215*^{K1}**

phenotype: Temperature-sensitive recessive lethal; flies survive at 19° but not at 29°. Although not displaying the *Ubx*-enhancing effect in combination with +, it does inhibit the effect of *Rpl1215*⁴ at 29° but not at 25° or 19°.

***Rpl1215*^{K2}**

phenotype: Homozygous and hemizygous viable; homozygous females sterile. Exhibits slight enhancement of *Ubx* when heterozygous to + but not in heteroallelic combination with any of the interacting mutant alleles; it decreases the enhancing effects of *Rpl1215*^{Ubl}. Increases the number of duplicated bristles of *Dll/+* in all genotypes. Temperature-sensitive period for effects on both *Ubx* and *Dl* from third larval instar until mid-pupal stage.

***Rpl1215*^{ts}**

synonym: *Ubl*^{ts}.

references: Mortin and Kaufman, 1982, Mol. Gen. Genet. 187: 120-25.

Mortin and Kaufman, 1984, Dev. Biol. 103: 343-54 (fig.).

phenotype: Encodes a heat labile RNA polymerase subunit, as measured *in vitro* (Coulter and Greenleaf, 1982, J. Biol. Chem. 157: 1945-52) and by sterilizing effects on females and lethal effects on embryos. Females shifted from 22° to 29° become sterile, although their eggs laid during the first 24 hr appear normal morphologically; after 24 hr, embryonic development is visibly abnormal. Embryonic abnormalities include holes in the ventral cuticle and abnormal pharyngeal structures. Partial rescue of the sterility can be achieved by shifting newly laid eggs to 22° or by fertilization of eggs of *Rpl1215*^{ts} females with wild-type sperm; the degree of rescue decreases as the time that the females have been held at 29° increases. Abnormalities of embryos dying despite rescue attempts mimic the phenotypes of pair-rule and segment-polarity mutants; surviving adults resemble *Hab* in lacking one or both halteres and metathoracic legs. In *Rpl1215*^{ts/+}/*Rpl1215*^{ts/0} gynandromorphs, the *Rpl1215*^{ts/0} tissue appears to occupy more territory than expected, as though it had a proliferative advantage over the *Rpl1215*^{ts/+} tissue (Mortin, Perrimon, and Bonner, 1985, Mol. Gen. Genet. 201: 437-45).

RpII215^{Ubl}: Ultrabithorax-like

synonym: *Ubl*.

references: Mortin and Lefevre, 1981, *Chromosoma* 82: 237-47.

Mortin, Kim, and Huang, 1988, *Genetics* 119: 863-73.

phenotype: Homozygous and hemizygous lethal. Heterozygotes mimic *Ubx* in adding several hairs to and enlarging the capitellum of the haltere; females homozygous for *RpII215^{Ubl}*, surviving by virtue of a duplication for *RpII215⁺*, more severely affected with capitellum approximately three times the size of that observed in *RpII215^{Ubl/+}* and with two or more rows of bristles; about 10% normal viability; poorly fertile in crosses to wild type; sterile when crossed to *RpII215^{Ubl}* males with a duplication of *RpII215⁺*. Similarly males with one mutant and one normal allele are more extreme than females with one mutant and two normal alleles. Acts as a dosage sensitive enhancer of *Ubx*, transforming halteres into wing-like structures; enhancement by *RpII215^{Ubl/RpII215^{Ubl/+}}* > *RpII215^{Ubl/+}* > *RpII215^{Ubl/+/+}*. *RpII215^{Ubl/+}* interacts with heterozygotes for *bx³* and *bx⁷* but not *bx¹* to produce enlarged capitellum, and with *bx^{d100/+}* to transform halteres into wing-like structures; extra doses of *Ubx⁺* counteract the enhancing effects of *RpII215^{Ubl}*. A second interaction with *Ubx/+* is the production of miscadestral-like pigmentation. *Ubx* enhancement by *RpII215^{Ubl}* reduced in *trans* heterozygotes with other interacting alleles (Mortin *et al.*). Furthermore in *RpII215^{Ubl/+//RpII215^{Ubl/0}}* gynandromorphs, the *X0* tissue is without any *RpII215^{Ubl}* phenotype, displaying neither enlarged halteres nor enhancement of *Ubx* expression, whereas the *XX* tissue exhibits both enlarged halteres and *Ubx* enhancement (Mortin, Perrimon, and Bonner, 1985, *Mol. Gen. Genet.* 201: 437-45). *RpII215^{Ubl/+}* also display increased frequencies of duplicated bristles in *Dll/+*, and in some crosses causes *Sb/+* flies to exhibit shortened and broadened wings whose longitudinal veins fail to reach the margin (Mortin *et al.*). *RpII215^{Ubl}* in heterozygous combination with deficiencies for either *ct* or *sno* is lethal and with lethal alleles of *ct* produces a strong cut phenotype; produces a mutant phenotype of allele specific severity in heterozygous combination with deficiencies for or lethal alleles of *br*, *N*, *dm*, *slc*, *bi*, *oc*, *m*, *sd*, and *sw*; interacts with heterozygotes for mutant alleles of *oc*, *sno*, and *sw*, but not the others; no interaction with *dor*, *sn*, *ras*, or *g* deficiencies. Influence of the maternal genotype apparent since patroclinous *RpII215^{Ubl/Y}* and *RpII215^{Ubl/0}* males from non mutant mothers survive at 20% the expected rate; they are phenotypically normal but are sterile (Voelker *et al.*).

***rs: rose**

location: 3-35.0.

phenotype: Eye color translucent purplish pink and ocelli colorless immediately after hatching; darken with age becoming sepia like, at least in the case of *rs^{66j}*. Further, *rs^{66j}* displays markedly decreased xanthopterin levels, reduced drosoppterins, and higher than normal levels of sepiapterin and bioppterin (Thörig and Scharloo, 1971, *DIS* 46: 40). *rs* very clear when combined with *v* (Akam), also interacts strongly with *ltd* (Silva and Mensua, 1985, *DIS* 51: 156). Larval Malpighian tubes pale yellow (Brehme and Demerec, 1942, *Growth* 6: 351-56).

alleles:

allele	origin	discoverer	ref ^α	comments
<i>*rs¹</i>	spont	Bridges, 23c10	1	male sterile; RK2
<i>rs²</i>	spont	Bridges, 35d5		fertile; RK1
<i>rs^{66j}</i>	spont		2	heat sensitive

^α 1 = Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 234; 2 = Thörig and Scharloo, 1971, *DIS* 40: 46.

cytology: Placed 67F3-68A3 on the basis of its inclusion in *Df(3L)vi²* = *Df(3L)67F2-3;68D6* but not *Df(3L)vi⁵* = *Df(3L)68A3;69A1-2* (Akam, Roberts, Richards, and Ashburner, 1978, *Cell* 13: 215-26).

***rsc: reduplicated sex combs**

location: 1- (between *y* and *cv*).

origin: X ray induced.

discoverer: Yanders, 56f6.

references: 1957, *DIS* 31: 85.

phenotype: Sex combs present on all six legs of males. Overlaps wild type in crowded cultures. Wings droop. Male fertile; viability only 15% wild type. Female lethal. RK2.

other information: Possible allele of *ph*.

rsd: raised

location: 3-95.4 (Ives); location in *Act88F⁸ rsd* stocks more proximal (Mahaffey *et al.*, 1985).

origin: Spontaneous.

discoverer: Ives, 40i5.

synonym: *mod: modification*.

references: 1945, *DIS* 19: 46.

1947, *DIS* 21: 69.

Lang, Wyss, and Eppenberger, 1981, *Nature* (London) 291: 506-08.

Mahaffey, Coutu, Fyrberg, and Inwood, 1985, *Cell* 40: 101-10.

phenotype: Wings held straight up, nearly meeting over thorax. Appears to interfere with processing of certain muscle proteins (Mahaffey *et al.*). *rsd* strains analyzed in recent years also carry *Act88F⁸*. Viability and fertility normal. RK1.

other information: *Act88F⁸ rsd* referred to as *Act88F⁸ mod = rsd* by Mahaffey *et al.*

rsi: reduced size

location: 1-0.28 (no crossovers with *br* in 1038 flies).

origin: Induced by D-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3026).

discoverer: Fahmy, 1954.

references: 1959, *DIS* 33: 89.

phenotype: Body small; eclosion delayed; viability reduced. RK3.

cytology: Not in *Df(1)RA19* = *Df(1)1E3-4;2B9-10* or *Df(1)sta* = *Df(1)1D3-E1;2A* (Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, *DIS* 58: 184-90).

other information: One allele each induced by CB. 1506 and CB. 3026.

Rsp: Responder

location: 2-56.61 (based on 122 *pr-cn* recombinants).

synonym: *Ac-SD: Activator of SD* (Hiraizumi and Nakazima).

Dr: Director (Hartl).

Receptor (Sandler and Carpenter).

references: Hartl, 1974, *Genetics* 76: 477-86.
 Ganetzky, 1977, *Genetics* 86: 321-55.
 Brittnacher and Ganetzky, 1984, *Genetics* 107: 423-34.
 Wu, True, and Johnson, 1989, *Nature* (London) 341: 248-51.

phenotype: The region of the chromosome upon which *Sd* acts to cause dysfunction of sperm that receive it; the sensitivity of *Rsp* is unaffected by its position in the genome, being equally responsive to *Sd* either in repulsion or coupling with *Sd*, or when located ectopically as in *Dp(2;f)Rsp* (Brittnacher and Ganetzky, 1989, *Genetics* 121: 739-50) or in *Dp(2;Y)Rsp* (Lyttle and Ault, 1985, *Genetics* 110: s23; Lyttle, 1989, *Genetics* 121: 751-63).

alleles: Both sensitive and insensitive alleles occur, and evidence of their existence appeared in the earliest articles on *Sd*. By monitoring the relative recoveries of homologous second chromosomes from males carrying *Dp(2;Y)B10-4 = Dp(2;Y)YL;36D2-3;40F*, in which *Sd* and *E(Sd)* are transposed from the base of *2L* into *YL*, Lyttle, Brittnacher, and Ganetzky (1986, *Genetics* 114: 183-202) were able to assign relative sensitivities to the *Rsp* alleles carried by different laboratory chromosomes; Hiraizumi and Martin had made similar assessments based on the different degrees of distortion in response to heterozygosity to an *SD* chromosome. The degree of distortion as seen in genotypic ratios among the progeny of *Sd/+* males depends on the sensitivity differential of *Rsp* on the two homologues.

alleles:

allele	synonym	ref ^α	comments
<i>Rspⁱ</i>	<i>Rsp, Rsp^{ins}</i>		insensitive
<i>Rsp^{116β}</i>		2	
<i>Rsp^{132β}</i>		2	
<i>Rsp^s</i>	<i>Rsp⁺, Rsp^{s2}</i>	3, 4	sensitive
<i>Rsp^{si}</i>	<i>Rsp^{s3}</i>	3, 4	intermediate sensitivity
<i>Rsp^{ss}</i>	<i>Rsp^{s1}</i>	1, 3, 4	super sensitive

^α 1 = Brittnacher and Ganetzky, 1984, *Genetics* 197: 423-34; 2 = Ganetzky, 1977, *Genetics* 86: 321-55; 3 = Hiraizumi, Martin, and Eckstrand, 1980, *Genetics* 95: 693-706; 4 = Lyttle, Brittnacher and Ganetzky, 1986, *Genetics* 114: 183-202.

^β X-ray-induced derivatives of *Rsp^s* (Ganetzky).

cytology: Placed in 41A proximal to *Df(2R)M41*, *Df(2R)B*, and *Df(2R)A'* based on the observation that these deficiencies, all of which include the most proximal known lethally mutable locus on *2R*, distort in *Df/Sd* males. Other proximal deficiencies have lost sensitivity to *Sd* [*Df(2R)A''*] (Ganetzky; Sharp, Hilliker, and Holm, 1985, *Genetics* 110: 671-88). X-ray-induced deficiencies for *Rsp* delete the most proximal *2R* lethal loci (Ganetzky). Insensitive chromosomes are characterized by the absence of *2R* heterochromatic region h39; there is also one fewer n-band brought about by fusion of n bands in h38 and h40; supersensitive Responders are characterized by a duplication of h39 and a wholly heterochromatic pericentric inversion (Pimpinelli and Dimitri, 1989, *Genetics* 121: 765-72).

molecular biology: Responder activity correlated with the dosage of a tandemly arrayed 120-base-pair, AT-rich repeat that maps to the base of *2R* and is transposed to the *Y* in *Yrsp^s* chromosomes of Lyttle. In one case in which reduced sensitivity is transposed to the *Y* leaving residual activity on *2R*, repeated sequences are found on both elements of the transposition (Wu, Lyttle, Wu and Lin,

1988, *Cell* 54: 179-89).

other information: In flies without *SD*, deletion of the *Rsp* region results in flies (both male and female) that are less fit than *Rsp⁺* flies (Wu *et al.*, 1989).

rss: reduced scutellars and sternitals

location: 1-17.4.

origin: Spontaneous in a selection line for reduced bristle number.

references: Sheldon and Evans, 1984, *Aust. J. Biol. Sci.* 37: 277-301.

phenotype: Shows specific reduction in the number of scutellar bristles without appreciable effect on other microchaetae. Also shows a reduction in the number of sternital microchaetae.

rst: roughest

location: 1-2.2 (based on 132 *w-spl* recombinants).

phenotype: Eyes rough and bulging; facets irregular in size and arrangement.

alleles: X-ray-induced *rst* mutations associated with chromosome rearrangements; those induced by ethyl methanesulfonate appear to be point mutations, although some also affect *vt*; *rst⁶* appears to be a point mutation that does not affect *vt*, is not rearranged and has normal viability in both sexes.

allele	origin	discoverer	synonym	ref ^α	comments
<i>rst¹</i>	X ray	Bell, 32b25			<i>T(1;3)rst</i> ; reduced viability
<i>rst³</i>	X ray	Grüneberg, 33116		4, 5, 6, 7, 8	<i>ln(1)3C3-5;h28</i>
<i>rst⁴</i>	EMS		<i>rst⁶⁸ⁱ¹⁹</i>	9	<i>vt</i> effect
<i>rst⁵</i>	EMS		<i>rst⁶⁸ⁱ²⁵</i>	9	<i>vt</i> effect
<i>rst⁶</i>	EMS	E. B. Lewis	<i>rst^{16172.646}</i> <i>rst^{CT}</i>	9	no <i>vt</i> effect
<i>rst⁷</i>	X ray	Demerec, 38d	<i>rst²⁶⁴⁻⁵⁷</i>	2	<i>ln(1)N²⁶⁴⁻⁵⁷</i> ; not variegated
<i>rst⁸</i>	X ray	Demerec, 39i	<i>rst²⁶⁵⁻⁸⁶</i>	1, 3, 10	<i>ln(1)N²⁶⁴⁻⁸⁶</i>

^α 1 = Demerec, 1940, *Genetics* 25: 618-27; 2 = Demerec, 1941, *Proc. Intern. Congr. Genet.*, 7th, pp. 99-102; 3 = Demerec and Sutton, 1940, *Proc. Nat. Acad. Sci. USA* 26: 532-36; 4 = Emmens, 1937, *J. Genet.* 34: 191-202; 5 = Grüneberg, 1935, *DIS* 3: 27; 6 = Grüneberg, 1935, *J. Genet.* 31: 163-84; 7 = Grüneberg, 1937, *J. Genet.* 34: 169-89; 8 = Kaufman, 1942, *Genetics* 27: 537-49; 9 = Lefevre and Green, 1972, *Chromosoma* 36: 391-412; 10 = Sutton, 1940, *Genetics* 25: 534-40.

cytology: Placed in 3C5 by Lefevre and Green (1972, *Chromosoma* 36: 391-412); formerly placed in 3C4, which apparently does not exist.

Rst: Resistance

A term used to denote genes that confer resistance to the killing effects of insecticides and other noxious agents. The symbol *Rst* is followed by parenthetical designation of the chromosomal location of the gene and then by an indication of the insecticide. Both dominant and recessive genes for insecticide resistance are conceivable. Several investigators have exposed populations to insecticides for numerous generations and selected resistant lines. In most cases, the genetic basis of resistance is multigenic, and unless a major factor has been identified, these strains are not included in this list.

Rst(1)JH: Resistance (1) Juvenile Hormone

location: 1-35.4.

origin: Induced by ethyl methanesulfonate.

synonym: *Met: Methoprene tolerant.*

references: Arking and Vlach, 1970, *J. Insect Physiol.* 22: 1143-51.

Wilson, 1985, *Genetics* 100: s84.

Wilson and Fabian, 1986, *Dev. Biol.* 118: 190-201.

phenotype: Semidominant mutation conferring 50-100 fold increase in resistance to juvenile hormone III or its analogue methoprene over that of wild type. Also resistant to methoprene-induced tumors and abnormalities in adult cuticle; action of *Rst(1)JH* autonomous in gynandromorphs. Authors speculate that gene may affect juvenile-hormone receptor.

alleles: Six ethyl-methanesulfonate-induced, X-linked mutations resistant to juvenile hormone reported by Arking and Vlach (now lost); resistance factors varied from 1.4 to 21.5 by their methods. Presumed to be alleles of the mutation described by Wilson and Fabian.

cytology: Placed in 10C2-D4 based on increased survival of flies raised on methoprene food when *Rst(1)JH* combined with *Df(1)M259-4 = Df(1)10C2;10E2* and *Df(1)N71 = Df(1)10B5;10D4*, compared to *Rst(1)JH/+*.

Rst(1)mth: Resistance (1) malathion

location: Thought to be polygenic.

references: Sing and Morton, 1981, *Can J. Genet. Cytol.* 23: 355-69.

phenotype: Both larvae and adults show resistance to malathion.

Rst(1)str: Resistance (1) streptomycin

location: 1- (not mapped).

synonym: *str-R.*

references: Lambertsson and Rasmuson, 1971, *Hereditas* 69: 299.

phenotype: Dominant; ribosomes from homozygotes and hemizygotes bind about one-tenth the amount of streptomycin bound by wild-type ribosomes. Resistant flies appear to lack one ribosomal protein found in wild type.

Rst(2)amd: Resistance (2) alpha methyl dopa

location: 2- (not mapped).

references: Sparrow and Wright, 1974, *Mol. Gen. Genet.* 130: 127-41.

Sherald and Wright, 1974, *Mol. Gen. Genet.* 133: 35-36.

phenotype: Partially dominant; resistant to levels of alpha methyl dopa in the medium that are lethal to wild type.

Rst(2)DDT: Resistance (2) DDT

location: 2-65 (64.5-66).

origin: Naturally occurring allele.

discoverer: Tsukamoto and Ogaki, 1953.

synonym: *RI^{DDT}: Resistance to Insecticide-DDT;*
RI^{II}: Resistance to Insecticide on chromosome 2.

references: 1954, *Botyu-Kagaku* 19: 25.

Tsukamoto, 1958, *DIS* 32: 87.

Kikkawa, 1961, *Ann. Rept. Sci. Works, Fac. Sci., Osaka Univ.* 9: 1-20.

Ogaki, Nakashima-Tanaka, and Murakami, 1967, *Jpn. J. Genet.* 42: 387-94.

phenotype: *Rst(2)DDT* is a major dominant gene responsible for the phenotype of a line selected for resistance to DDT. Median lethal dose of DDT for *Rst(2)DDT* lines is about 4000 µg/cc of medium; that for sensitive lines is

50-100 µg/cc. Also resistant to BHC (benzene hexachloride) and organophosphorus insecticides such as parathion and malathion. Median lethal dose of parathion is 2 ppm for resistant line and 0.08 ppm for sensitive. Sensitive to phenylthiourea (Ogita, 1958, *Botyu-Kagaku* 2: 188-204) and phenylthiocarbamide (Däuring and Sunner, 1971, *Hereditas* 68: 115-22). Shows maternal effect in that progeny of *Rst(2)DDT/+* female crossed to *+/+* male are more resistant than those of reciprocal cross. Larva more resistant than adult. RK3.

other information: Possible to select new resistant alleles by growing the offspring of irradiated wild-type flies on parathion (Kikkawa, 1968, *DIS* 43: 161).

Rst(3)amd: Resistance (3) alpha methyl dopa

location: 3- (between *h* at 25.6 and *th* at 43.2).

origin: Induced by ethyl methanesulfonate.

references: Bishop and Sherald, 1981, *DIS* 56: 21-22.

phenotype: Homozygous lethal. LD₅₀ concentration of α methyl dopa for heterozygotes more than three times that of wild type.

alleles: Two alleles (PR40 and PR45) exhibiting weak complementation for lethality.

Rst(Eth): see *Eth*

Rst(3)FU: Resistance (3) Fluorouracil

location: 3- (not mapped).

origin: Spontaneous.

references: Duke and Glassman, 1968, *Nature (London)* 220: 588-89.

phenotype: Partially dominant; homozygote survives 0.003% fluorouracil, whereas 0.0008% lethal to most strains.

****Rst(3)ns: Resistance (3) nicotine sulfate***

location: 3-49.5.

origin: Spontaneous.

discoverer: Tsukamoto, 1954.

references: 1955, *Botyu-Kagaku* 20: 73.

1956, *Botyu-Kagaku* 21: 71.

1958, *DIS* 32: 87.

phenotype: Median lethal dose to homozygote is 600 ppm of nicotine sulfate added to culture medium (from first instar larva through eclosion); to heterozygote, it is 300 ppm; to susceptible strains, 40 ppm. RK3.

****rstl: roughestlike***

location: 1- (rearrangement).

origin: X ray induced.

discoverer: Oliver, 29d3.

synonym: *lz-1: lozenge-like.*

references: 1935, *DIS* 3: 28.

phenotype: Eyes rough; more extreme than *lz*. Viability low. RK2A.

cytology: Associated with *In(1)rstl*; breakpoints unknown.

****rt: rotated abdomen***

location: 3-37 (based on location of *rt*²).

discoverer: Bridges, 18g28.

references: Bridges and Morgan, 1923, *Carnegie Inst. Washington Publ. No. 327*: 190 (fig.).

Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 54 (fig.).

phenotype: Abdomen twisted clockwise through 60° to

90°, as viewed from behind. Both sexes sterile. Viability low. RK2.

cytology: Placed in 68C8-11 based on inclusion in *Df(3L)vin7 = Df(3L)68C8-11;69B4-5* but not *Df(3L)vin6 = Df(3L)68C8-11;69A4-5* (Akam, Roberts, Richards, and Ashburner, 1978, Cell 13: 215-25).

rt²

origin: Spontaneous.

discoverer: Bridges, 25114.

phenotype: Abdomen twisted, as is *rt*. Viability erratic, usually about 50% wild type. Male fertile; female not tested. RK2.

***rt^w: rotated abdomen of Wallbrunn**

origin: γ ray induced.

discoverer: Wallbrunn, 61i26.

references: 1964, DIS 39: 59.

phenotype: Like *rt*. RK2.

***rta: reduced tarsi**

location: 1-4.5.

origin: Induced by methyl methanesulfonate (CB. 1540).

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 89.

phenotype: Tarsi short and sometimes deformed. Body small. Eyes and wings small and abnormal. Bristles often waved or bent; postscutellars often held upright. Male sterile. RK2.

rtv: retroactive

location: 1-38.

origin: Induced by ethyl-methanesulfonate.

references: Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307.

phenotype: Recessive embryonic lethal; mouth parts darkly sclerotized. Embryo sometimes reversed in egg case (due to hyperactivity at late stages?). Zygotes from homozygous germ-line clones in females develop normally (Wieschaus and Noell, 1986, Roux's Arch. Dev. Biol. 195: 63-73).

alleles: Allelism with Lefevre lethals reported by Perrimon, Engstrom, and Mahowald, 1989, Genetics 21: 333-52.

allele	origin	discoverer	synonym	ref ^α	comments
<i>rtv 1</i>	X ray	Lefevre	<i>l(1)JL1</i>	3	
<i>rtv 2</i>	X ray	Lefevre	<i>l(1)HC236</i>	4	
<i>rtv 3</i>	X ray	Lefevre	<i>l(1)JA10</i>	4	
<i>rtv 4</i>	X ray	Lefevre	<i>l(1)JC65</i>	4	
<i>rtv 5</i>	EMS	Lefevre	<i>l(1)VE701</i>	5	
<i>rtv 6</i>	EMS	Lefevre	<i>l(1)VE745</i>	5	embryonic lethal; no maternal effect
<i>rtv 7</i>	EMS	Lefevre	<i>l(1)VE800</i>	5	
<i>rtv 8</i>	EMS		<i>l(1)jv24</i>	2	
<i>rtv 8</i>	EMS		<i>l(1)jv68</i>	2	
<i>rtv 9</i>	EMS		<i>l(1)jv152</i>	2	
<i>rtv 10</i>	MR	Eeken	<i>l(1)D40</i>	1	
<i>rtv 11</i>	EMS		<i>l(1)YA</i>	6	
<i>rtv 12</i>	EMS			6	
<i>rtv 13</i>	EMS			6	

^α 1 = Eeken, Sobels, Hyland, and Schalet, 1985, Mutat. Res. 150: 261-75; 2 = Geer, Lischwe, and Murphy, 1983, J. Exp. Zool. 225: 107-18; 3 = Lefevre, 1971, Genetics 67: 497-513; 4 = Lefevre, 1981, Genetics 99: 461-80; 5 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 6 = Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307.

cytology: Placed in 10A6-B1 based on its inclusion in

Df(1)RA37 = Df(1)10A6;10B15-17 but not *Df(1)GA112 = Df(1)10A11-B1;10C2*.

ru: roughoid

location: 3-0.0 [actually 2.57 units to the right of the end of the chromosome, based on recombination with *y⁺* of *T(1;3)sc^{JA}*] in a sample of 9106 flies (Strommer and Falk, 1981, DIS 56: 196)].

references: Renfranz and Benzer, 1989, Dev. Biol. 136: 411-29.

phenotype: Eyes small and rough, have irregular facets and hairs, and have black specks from erupted facets. Expression variable; sometimes overlaps wild type. SEM study by Stumm-Tegethoff and Dick (1974, Theor. Appl. Genet. 44: 262-65).

alleles:

allele	origin	discoverer	ref ^α	comments
<i>ru 1</i>		Sturtevant, 19b14	1, 3, 5	may overlap wild type
<i>*ru 40k</i>	spont	Steinberg, 40k	4	
<i>*ru 100.392</i>	X ray	Alexander	6	<i>Df(3L)61E;62A10-B1</i>
<i>*ru 100.393</i>	X ray	Alexander	6	<i>Df(3L)61F;62A4-6</i>
<i>*ru 300.234</i>	X ray	Alexander	6	<i>Df(3L)61E;62A2-4</i>
<i>ru^g</i>	spont	Glass	2	RK1

^α 1 = Bridges and Morgan, 1923, Carnegie Inst. Wash. Publ. No. 327: 212 (fig); 2 = Glass, 1934, DIS 2: 8; 3 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 215 (fig.), 234; 4 = Steinberg, 1942, DIS 16: 54; 5 = Strong, 1920, Biol. Bull. 38: 33-37; 6 = Ward and Alexander, 1957, Genetics 42: 42-54.

cytology: Placed in 61F5-62A3 on the basis of its inclusion in *Df(3L)ru-K2 = Df(3L)61F4-5;61A10-B1* (Krivshenko, 1958, DIS 31: 81) and *Df(3L)ru1300.234 = Df(3L)61E;62A2-4* (Ward and Alexander, 1957, Genetics 42: 42-54).

rub: rubroad

location: 2-5.0 (to the right of *dpp^{d-ho}*).

origin: Spontaneous.

discoverer: Mohr, 31k20.

phenotype: Eyes rough and kidney shaped. Wings broad and somewhat arclike. Abdomen short and bloated; tergites irregular. External genitalia of male rotated in varying degrees. Overlaps wild type. RK3.

***rub^{48d}**

origin: Spontaneous.

discoverer: Chute, 48d.

references: Sturtevant, 1948, DIS 22: 56.

phenotype: Like *rub*; wings also show slight network of extra veins and thickening between L3 and L4. RK3.

rubb: rubbish (T. Schüpbach)

location: 2-89.

origin: Induced by ethyl methanesulfonate.

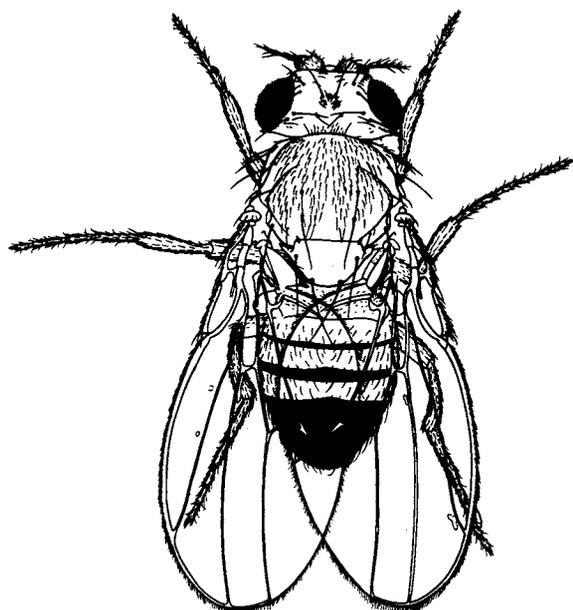
references: Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

phenotype: Maternal effect lethal; embryos from homozygous mothers form a fragmented cuticle with variable holes and head defects.

alleles: One: *rubb^{QD} = rubbish¹*.

rubroad: see rub

ruby: see rb



rub: rubroad

Edith M. Wallace, unpublished.

rud: ruddle

location: 1-3.3.

origin: Induced by L-p-N,N-di-(2-chloroethyl)aminophenylalanine (CB. 3025).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 74.

phenotype: Eye color dull reddish brown. Classification best in newly eclosed flies. Good viability and fertility. RK2.

other information: One allele each induced by CB. 1528, CB. 3026, and X rays.

rudimentary: see *r*

rudimentary-like: see *r-l*

rue: ruffed eye

location: 3- {47}.

origin: Induced by ethyl methanesulfonate.

discoverer: Kaufman.

references: Lewis, Kaufman, Denell, and Tellerico, 1980, Genetics 95: 367-81.

Cavener, Otteson, and Kaufman, 1986, Genetics 114: 111-23.

phenotype: Roughened eyes and fine bristles at 25°; lethal at 28°.

cytology: Placed in 84C1-6 based on its inclusion in *Df(3R)dsx2M = Df(3R)84C1-2;84E1* and *Df(3R)Antp-X2 = Df(3R)84B6-C1;84C5-6*.

rugose: see *rg*

rumpled: see *rmp*

run: runt

location: 1- {65.8}.

synonym: *leg: legless*.

references: Nüsslein-Volhard and Wieschaus, 1980, Nature (London) 287: 795-801 (fig.).

Gergen and Wieschaus, 1986, Cell 45: 289-99 (fig.).

Gergen, 1987, Genetics 117: 477-85 (fig.).

Gergen and Butler, 1988, Genes Dev. 2: 1179-93.

phenotype: A pair-rule embryonic lethal; causes deletions of the dentical belts of the mesothoracic and the first, third, fifth, and seventh abdominal segments, extending through the more anterior naked cuticle and into the denticle belts of the next most anterior segments. Deleted regions appear to be replaced by mirror image duplications of the remaining more anterior pattern elements. Deleted regions exceed duplications in size, resulting in shorter embryos. The amount of material deleted varies among segments, alleles, and among animals with the same alleles. Hypomorphic alleles do not remove as much tissue as amorphs, and weak hypomorphic alleles produce occasional survivors missing methathoracic legs or halteres or both and are frequently missing one or more abdominal tergites. *run*³¹, among the weakest alleles, survives to adulthood. Deficiencies for *run* have discernable dominant effects on embryonic development; extra doses of *run*⁺ produce anti-runt phenotype, *i.e.*, 30% of males with two doses of *run*⁺ display deletions of portions of the dentical belts of A6 and less frequently of A2 and A8; males with three *run*⁺ alleles more severely affected with 70% penetrance. *run*⁺ postulated to repress *eve* function and positively regulate *ftz*; *run* mutants show expansion of stripes of *eve* expression and premature disappearance of stripes of *ftz* expression in the embryo (Frasch and Levine, 1987, Genes Dev. 1: 981-95). *ftz*⁺ expression in cellular blastoderm reduced in four anterior stripes; A5-A7 expression abnormal, possibly reflecting pattern duplication; nuclear shape abnormal [Carroll and Scott, 1986, Cell 45: 113-26 (fig.)]. For expression pattern later in development see Kania, Bonner, Duffy, and Gergen (1990, Genes Dev. 4: 1701-13). *run* is autonomous in gynandromorphs both for missing and for mirror-image duplicated phenotypes, suggesting that the duplication does not result from proliferation following cell death (Gergen and Wieschaus, 1985, Dev. Biol. 109: 321-35). Also, the earliest embryonic phenotypes are dosage compensated (Gergen, 1987). *run* embryos produced by homozygous ovarian clones not different from those produced by heterozygous mothers (Wieschaus and Noell, 1986, Roux's Arch. Dev. Biol. 195: 63-73).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>run</i> ¹		Novitski	<i>l(1)152</i>		
<i>run</i> ²	EMS	Lifschytz	<i>l(1)AA33</i>	3, 8, 11	weak hypomorph
<i>run</i> ³	mit C	Baldwin	<i>l(1)LB5</i>	3, 9	amorph
<i>run</i> ⁴	EMS	Baldwin	<i>l(1)LB19</i>	9	
<i>run</i> ⁵	EMS	Lifschytz	<i>l(1)P235</i>	3, 8	intermediate hypomorph
<i>run</i> ⁶	EMS	Lifschytz	<i>l(1)P425</i>	8	
<i>run</i> ⁷	EMS	Lifschytz	<i>l(1)P515</i>	8	
<i>run</i> ⁸	EMS	Lifschytz	<i>l(1)W1</i>	3, 8	amorph; also <i>unc</i> ⁸
<i>run</i> ⁹	EMS	Lifschytz	<i>l(1)W3a</i>	7	also mutant for <i>unc</i>
<i>run</i> ¹⁰	X ray	Lefevre	<i>l(1)HA14</i>	5	<i>T(1;3)19E;80</i>
<i>run</i> ¹¹	X ray	Lefevre	<i>l(1)RF45</i>	5	<i>T(1;2)19E-20F;52A</i>
<i>run</i> ¹²	EMS	Lefevre	<i>l(1)DC839</i>		
<i>run</i> ¹³	EMS	Lefevre	<i>l(1)EA14</i>	3, 6	intermediate hypomorph
<i>run</i> ¹⁴	EMS	Lefevre	<i>l(1)EF535</i>		
<i>run</i> ¹⁵	EMS	Lefevre	<i>l(1)VE726</i>	3, 6	strong hypomorph
<i>run</i> ¹⁶	EMS	Lefevre	<i>l(1)VE751A</i>	3, 6	amorph
<i>run</i> ¹⁷	spont	Schalet	<i>l(1)16-137</i>		
<i>run</i> ¹⁸	neutron	Muños	<i>l(1)17-26</i>	3-10	amorph; deletion with one breakpoint at -5.4 and extends in + direction
<i>run</i> ¹⁹	neutron	Muños	<i>l(1)17-169</i>	3, 9, 10	weak hypomorph; breakpoint between -7.9 and -6.4 also <i>vao</i> mutant
<i>run</i> ²⁰	HMS	Kramers	<i>l(1)HM48</i>	4	
<i>run</i> ²¹	HMS	Kramers	<i>l(1)HM449</i>	4	
<i>run</i> ²²	EMS	Wieschaus	<i>l(1)XA06</i>	3, 10	amorph
<i>run</i> ²³	EMS	Wieschaus	<i>l(1)XD106</i>	3, 10	amorph
<i>run</i> ²⁴	EMS	Wieschaus	<i>l(1)XK52</i>	1, 3, 10	intermediate hypomorph
<i>run</i> ²⁵	EMS	Wieschaus	<i>l(1)YC28</i>	2, 3, 10	intermediate hypomorph
<i>run</i> ²⁶	EMS	Wieschaus	<i>l(1)YC47</i>	3, 10	strong hypomorph
<i>run</i> ²⁷	EMS	Wieschaus	<i>l(1)YD24</i>	3, 10	intermediate hypomorph
<i>run</i> ²⁸	EMS	Wieschaus	<i>l(1)YE96</i>	1, 3, 10	strong hypomorph
<i>run</i> ²⁹	EMS	Wieschaus	<i>l(1)YP17</i>	3, 10	intermediate hypomorph temperature sensitive
<i>run</i> ³⁰	P	Gergen	<i>l(1)34A</i>	3, 11	intermediate hypomorph; breakpoint near -0.9
<i>run</i> ³¹	P	Gergen	<i>l(1)50-2</i>	3, 11	weak hypomorph; P-insert site defined as coordinate 0
<i>run</i> ³²	P	Gergen	<i>l(1)PV1</i>	3, 11	amorph; 5 kb deletion between -6.5 and 8.7

^α 1 = Carroll and Scott, 1986, Cell 45: 113-26 (fig.); 2 = Gergen, 1987, Genetics 117: 477-85; 3 = Gergen and Wieschaus, 1986, Cell 45: 289-99; 4 = Kramers, Schalet, Paradi, and Huizer-Hoogteyling, 1983, Mut. Res. 107: 187-201; 5 = Lefevre, 1981, Genetics 99: 461-80; 6 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 7 = Lifschytz and Falk, 1968, Mut. Res. 6: 235-44; 8 = Lifschytz and Falk, 1969, Mut. Res. 8: 147-55; 9 = Schalet and Lefevre, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902; 10 = Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307; 11 = Zusman, Coulter, and Gergen, 1985, DIS 61: 217-18.

cytology: Placed in 19E1-3 on the basis of its inclusion in the region of overlap of *Df(1)16.3.35 = Df(1)19D2-D3;E3* and *Df(1)B57 = Df(1)19E1-2;19F1* (Miklos, Kelly, Coombe, Leeds, and Lefevre, 1987, J. Neurogenet. 4: 1-19).

molecular biology: Gene cloned by transposon tagging; 50 kb cloned. Mutational lesions define a segment of at least 8.5 kb required for *run* function; transposition of a 14.5 kb segment that includes the 8.5 kb sequence elicits partial rescue of *run* mutants. Genomic sequences identify a 2.6 kb poly-adenylated mRNA on northern blots. Transcript uniformly distributed in early embryo; most abundant during blastoderm when it is expressed in the seven stripes characteristic of other pair-rule genes; switching to every-segment expression during gastrulation and showing expression in the head and proctodeal primordium at that time [Gergen and Butler, 1988, Genes Dev. 2: 1179-93 (fig.)].

run^t: see *run*

rut: *rutabaga* (J.C. Hall)

location: 1- {46}.

references: Duerr and Quinn, 1982, Proc. Nat. Acad. Sci. USA 79: 3646-50.

Dudai, Uzzan, and Zvi, 1983, Neurosci. Lett. 42: 207-12.
Tempel, Bonini, Dawson, and Quinn, 1983, Proc. Nat. Acad. Sci. USA 80: 1482-86.

Gailey, Jackson, and Siegel, 1984, Genetics 106: 613-23.
Kyriacou and Hall, 1984, Nature (London) 308: 62-65.
Livingstone, Sziber, and Quinn, 1984, Cell 37: 205-15.
Livingstone, 1985, Proc. Nat. Acad. Sci. USA 82: 5992-96.

Tully and Quinn, 1985, J. Comp. Physiol. 157: 263-77.
Mariath, 1985, J. Insect Physiol. 31: 779-87.

phenotype: Mutant males and homozygous females impaired in several types of learning and memory; associative conditioning defective in tests using either reward (Tempel *et al.*, 1983) or aversive unconditioned stimuli (e.g. Dudai, 1983, Proc. Nat. Acad. Sci. USA 80: 5445-48; Dudai, Svi, and Segel, 1984, J. Comp. Physiol. 155: 569-76; Livingstone *et al.*), including tests of "classical" (e.g. Tully and Quinn, 1985) and "operant" conditioning (Mariath, 1985); able to learn in associative conditioning tests involving visual cues, but at subnormal levels (Folkers, 1982, J. Insect Physiol. 28: 535-39), and memory appears to be normal. Learning scores subnormal when measured immediately after certain types of training; then either scores decay rapidly with time (Tem-

pel *et al.*, 1983; Tully and Quinn, 1985) or there is no indication of memory (Mariath). Although defective in some aspects of learning, heterozygous females behave essentially normally in shock/odor tests (Dudai *et al.*, 1983). Courtship also defective; unlike wild-type males, *rut* males court inseminated and virgin females with equal vigor; they may be unable to distinguish them (Gailey *et al.*). In tests of non-associative conditioning, *rut* shows aberrant habituation and sensitization to sugar stimuli (Duerr and Quinn); *rut* males subnormal in learning to avoid courtship of immature males; and homozygous or hemizygous *rut* females defective in "priming" of mating behavior by prestimulations with artificial courtship songs; effects of such acoustical prestimulations decay more rapidly than normal (Kyriacou and Hall). In either the homozygous or heterozygous condition *rut* acts as a partial suppressor of the sterility of homozygous *dnc* females inversely related to degree of rescue, suggesting both a maternal and a zygotic role of *rut* (Bellen, Gregory, Olsson, and Kiger, 1987, Dev. Biol. 121: 432-44; Bellen and Kiger, 1988, Roux's Arch. Dev. Biol. 197: 258-68). Double mutant females mated to Canton-S males lay many eggs, but most of the eggs fail to hatch.

Biochemically, *rut* influences adenylate cyclase activity (Dudai *et al.*, Livingstone *et al.*); it seems to abolish a calcium or calmodulin stimulated component of adenylate cyclase activity (Livingstone, Dudai, and Zvi, 1984, Neurosci. Lett. 47: 119-24), while leaving intact a component of activity stimulated by guanyl nucleotides, fluoride, or monoamines, suggesting that *rut* may directly affect the catalytic subunit of the adenylate cyclase complex (Livingstone *et al.*, Dudai *et al.*, 1984); consistent with this hypothesis is the observation that cyclase activity in *rut* is lower than normal, even in the presence of forskolin (Dudai *et al.*, 1984; Dudai, Sher, Segal, and Yovell, 1985, J. Neurogenet. 2: 365-80). *rut* primarily affects total cyclase activity in the adult abdomen, with progressively milder effects on thoracic and head cyclase (Livingstone *et al.*, Dudai and Svi, 1985, J. Neurochem. 45: 355-64); reduction of abdominal adenylate cyclase activity of *rut*¹>*rut*²>*rut*³ (Bellen *et al.*); the majority of adenylate cyclase activity in wild type is in a particulate fraction, and *rut* lacks up to 35% of total particulate activity (Dudai and Zvi, 1985). That *rut* may in fact encode a component of the fly's adenylate cyclase catalytic subunit is suggested by altered Km of enzyme activity in mutant flies (*e.g.* Dudai *et al.*, 1983, 1985) and by the fact that hypoploidy of *rut*⁺ in females leads to approximately half normal levels of that cyclase activity specifically affected by the *rut* mutations (Livingstone *et al.*), and hyperploidy for the normal allele leads to increased activity (Livingstone). The biochemical results suggest that *rut*¹ could be a null mutation.

alleles: Three alleles: *rut*² and *rut*³ recovered as partial suppressors of the female sterility of homozygous *dnc* females.

allele	origin	discoverer	synonym	ref ^α	comments
<i>rut</i> ¹	EMS	Sziber			
<i>rut</i> ²	EMS	Gregory	BG2	1	partial suppressor of <i>dnc</i>
<i>rut</i> ³	EMS	Gregory	BG3	1	partial suppressor of <i>dnc</i>

^α 1 = Bellen, Gregory, Olsson, and Kiger, 1987, Dev. Biol. 121: 4332-44.

cytology: Placed in 12F5-13A1 based on its inclusion in the region of overlap of *Df(1)KA9 = Df(1)12E2-3;12F5-13A1* and *Df(1)RK4 = Df(1)12F5-6;13A9-B1*.

***rut*: roughex**

location: 1-15.0.

references: Renfranz and Benzer, 1989, Dev. Biol. 136: 411-29.

phenotype: Eyes smaller than wild type and uniformly rough. Male sterile. RK2.

alleles:

allele	origin	discoverer	ref ^α	comments
<i>rut</i> ¹		Bridges, 33d24	1	male sterile
<i>rut</i> ²		Curry, 3711	1	
<i>rut</i> ^{60d}	spont.	Rolfes, 1960	1, 2, 3	60% viability

^α 1 = CP627; 2 = Hollander, 1960, DIS 34: 50; 3 = Hollander and Festing, 1962, DIS 36: 79.

cytology: Placed in 5C5-D6, possibly in 5D2-6 (Ashburner).

***rv*: raven**

location: 1-4.4.

origin: Induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1953.

references: 1959, DIS 33: 89.

phenotype: Body small and heavily melanized. Eye color dark. Wings short and frequently divergent or not fully expanded. Male fertile but viability reduced; female more inviable and infertile. RK2.

Rvd: see *Rev*^B

***rw*: raised wing**

location: 2-93.2.

origin: Spontaneous.

discoverer: Gomes, 55a.

references: Burdick, 1955, DIS 29: 70.

phenotype: Wings held vertically; venation normal. Legs morphologically normal, but fly has difficulty walking. Penetrance and expressivity good. Viability poor. RK2.

rw: see *rwi*

****Rw*: Rough wing**

location: 2-56 [locus from crossing over in triploids (Schultz)].

discoverer: Hamly.

phenotype: Wings notched and veins irregular. An occasional extra antenna. *Rw*/+/+ triploid female slightly fertile. *Rw*/+ female sterile. RK3.

rwg: see *hdp*^{*rwg*}

***rwi*: red wine**

location: 3-22.7.

origin: Spontaneous.

synonym: *rw*.

references: Mostashfi and Kolianz, 1970, DIS 45: 34.

phenotype: Eye color wine like.

ry: rosy (*A. Chovnick and colleagues*)**location:** 3-52.0.

phenotype: The structural gene for xanthine dehydrogenase [XDH (EC.1.2.1.37)]; it is a homodimer with subunit molecular weight estimated from its DNA sequence as 146,898 daltons (Keith, Riley, Kreitman, Lewontin, Curtis, and Chambers, 1987, *Genetics* 116: 67-73). Enzyme level responds to dose of *ry*⁺ alleles (Grell, 1962, *Z. Indukt. Abstamm. Vererbungsl.* 93: 371-77). XDH is a molybdenum hydroxylase and requires the activity of *cin*⁺, *lxd*⁺, *mal*⁺ for normal activity, though not for normal levels of CRM (Glassman, Shinoda, Duke, and Collins, 1968, *Ann. N. Y. Acad. Sci.* 151: 263-73). CRM (cross reacting material) contains bound molybdenum in the presence of *mal*; however, enzyme activity inhibited (Andres 1976, *Eur. J. Biochem.* 62: 591-600). Homozygotes for null alleles lack XDH activity (Forrest, Glassman, and Mitchell, 1956, *Science* 124: 725-26; Glassman and Mitchell, 1959, *Genetics* 44: 153-62; Hubby and Forrest, 1960, *Genetics* 45: 211-24) and have reddish brown eyes; accumulate enzyme's substrates, xanthine and 2-amino-4-hydroxypteridine as larvae plus hypoxanthine in the adult; precursors collect as solid granules in Malpighian tubules (Bonse, 1967, *Z. Naturforsch.* 22B: 1027-29); lack enzyme products uric acid and isoxanthopterin (Mitchell, Glassman, and Hadorn, 1959, *Science* 129: 268-69). Mutant homozygotes are also sensitive to administration of purine to the medium (Glassman, 1965, *Fed. Proc.* 24: 1243-51); survival on purine supplemented medium can be used to select for rare *ry*⁺ recombinants (Chovnick, Ballantyne, Baillie, and Holm, 1970, *Genetics* 66: 315-29) and unequal crossovers producing tandem duplications (Gelbart and Chovnick, 1979, *Genetics* 92: 849-59). Hypomorphic alleles that have normal eye color are also sensitive to appropriate levels of purine supplementation; furthermore, both wild types and hypomorphs can be made to display mutant eye color by administration of appropriate levels of the XDH inhibitor, HPP (allopurinol) [4-hydroxypyrazolo-(3,4-d)pyrimidine] (Glassman, 1965, *Fed. Proc.* 24: 1243-51; Boni, DeLerma, and Parisi, 1967, *Experientia* 23: 186-87; McCarron and Chovnick, 1981, *Genetics* 97: s70-71); *in vitro* and *in vivo* complementation between *mal* and *ry* products was demonstrated by Glassman (1952, *Proc. Nat. Acad. Sci. USA* 48: 1491-97; Glassman and McLean, 1962, *Proc. Nat. Acad. Sci. USA* 48: 1712-18). Pigmentation is nonautonomous in *ry* eye disks trans-

planted into wild-type hosts (Hadorn and Schwink, 1956, *Nature* 177: 940-41). Enzyme levels climb from low levels in the zygote to a peak at puparium formation; the level then falls but increases again to a maximum a few days after eclosion (Chovnick, McCarron, Hilliker, O'Donnell, Gelbart, and Clark, 1978, *Cold Spring Harbor Symp. Quant. Biol.* 42: 1011-21). Enzyme derived from the paternal genome appears during gastrulation; activity at time zero is low in *ry*⁺ zygotes produced by *ry*/+ females but undetectable in those produced by *ry* females (Sayles, Browder, and Williamson, 1973, *Dev. Biol.* 33: 213-17). Enzyme activity present in larval and adult fat bodies, larval and adult Malpighian tubules, and, in smaller amounts, in various regions of the larval and adult gut [Ursprung and Hadorn, 1961, *Experientia* 17: 230-31; Munz, 1964, *Z. Indukt. Abstamm. Vererbungsl.* 95: 195-210; Reaume, Clark, and Chovnick, 1989, *Genetics* 123: 503-09; Reaume (unpublished observations)]. XDH is not synthesized in the adult eye, but is transported there [Reaume *et al.*, 1989].

alleles: *ry* alleles have been detected by several criteria, including electrophoretic mobility of XDH, purine sensitivity, and rosy eye color, either in the absence or presence of allopurinol. Alleles are presented in two tables; the first table includes the wild-type variants, and the second the mutant alleles. Chovnick and his colleagues (Chovnick, Gelbart, and McCarron, 1980, *Cell* 11: 1-10) identify at least seven different electromorphs in laboratory stocks; using a highly discriminating series of gel conditions, Buchanan and Johnson (1983, *Genetics* 104: 301-15) identified, among 62 wild-type chromosomes isolated from nature, fourteen electromorphs, two of which corresponded to those contained among the earlier seven. Both induced mutations and natural variable sites are designated by the number of the + progenitor followed by specific derivative numbers, *e.g.* *ry*¹⁰² is the second mutant derivative of *ry*⁺¹. Not all mutants with allelic designations with values less than 100 are known to be derivatives of *ry*⁺⁰; however, those numbered from 3a to 54 (excepting 17, 20, and 21) are known to be so derived. However, those mutations discovered by Girton, Green, Daniels, Lewis, Spradling and Rubin do not use this system of nomenclature. Also, those alleles marked with an asterisk are no longer available. Finally, Chovnick's laboratory maintains several hundred mutants not reported here, including a group generated on a *ry*⁺¹¹ background.

Table I

allele ^α	seq. data avail. ^β	rel. mob.	tentative constitution ^γ	ref. ^δ	origin of line
<i>ry</i> ⁺⁰	+	1.00	0 0/0 0 0	1, 3-7, 9, 10	<i>cu kar</i> stock ^ε
<i>ry</i> ^{+1d}	-	1.02	0 0/0 0 -	3, 4, 7, 9, 10	conversion of Oregon-R <i>ry</i>
<i>ry</i> ⁺²	+	1.03	0 0/- 0 0	1, 3-5, 7, 9, 10	Oregon-R iso-3
<i>ry</i> ⁺³	-	1.05	0 0/- 0 -	3, 4, 7, 9, 10	Amherst iso-3
<i>ry</i> ⁺⁴	+	1.02	0 +/0 0 -	2-7, 9, 10	Pacific iso-3
<i>ry</i> ⁺⁵	+	1.05	0 0/- 0 -	3-5, 7-9, 10	zeste; <i>ry</i> ⁺ iso-3
<i>ry</i> ⁺⁶	+	1.00	0 0/0 0 0	3-7, 9, 10	Hikone iso-3
<i>ry</i> ⁺¹⁰	+	0.97	- 0/0 + 0	2-8, 10	Bethulie iso-3
<i>ry</i> ⁺¹¹	+	1.02	0 0/0 0 -	3-5, 7, 10	<i>kar</i> ² <i>ry</i> ⁺ stock
<i>ry</i> ^{+12d}	-	0.90	0 0/0 + 0	3, 4, 10	conversion of <i>ry</i> ^{16608.19} (Lewis) against <i>ry</i> ⁸
<i>ry</i> ^{+13d}	-	0.90	0 0/0 + 0	3, 4, 10	(see <i>ry</i> ^{+12d})
<i>ry</i> ^{+14d}	-	0.94	0 0/0 + -	3, 4, 10	(see <i>ry</i> ^{+12d})
<i>ry</i> ⁺¹⁵	+	1.02	0 0/0 0 -	5	Kalahari iso-3
<i>ry</i> ⁺¹⁶	-	1.02	0 0/0 0 -	10	Kalahari iso-3
<i>ry</i> ^{+19d}	-	1.00	0 0/0 0 0	4	conversion of Weymouth <i>ry</i> against <i>ry</i> ⁸
<i>ry</i> ^{+21d}	-	1.03	0 0/- 0 0	4	conversion of <i>ry</i> ²¹⁰¹ (<i>copia</i>)
<i>ry</i> ⁺³¹	+	1.00	0 0/0 0 0	5	Okanogon iso-3

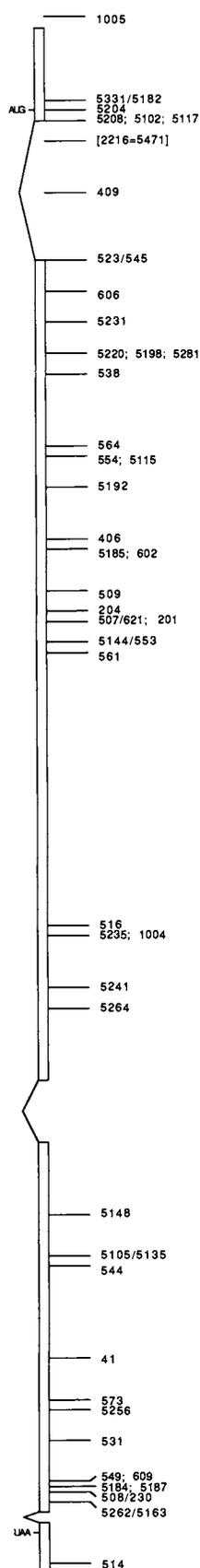
^α *ry*⁺ allele designation refers to the entire rosy DNA sequence. Electrophoretic markers and control variants characteristic of a given allele represent only a few of the bp polymorphisms distinguishing one *ry*⁺ allele from another. *ry*⁺ alleles originated as iso-3 stocks from wild populations, with the exception of those marked (derivative) which are conversions to wild type of a unique rosy mutant allele. These may carry bp polymorphisms within the conversion segment not common to the original rosy mutant.

^β Sequence data have been submitted to the EMBL/Gen Bank Data Libraries under the accession number Y 00307 and Y 00308. Data on other wild-type sequence are available (5, 6).

^γ Polymorphic sites segregating in wild type alleles; the digits to the left of the slash bar represent the phenotype with respect to the 5' cis-acting control elements, 1005 and 409, with "0" indicating the CRM levels of *ry*⁺⁰, "+" representing higher CRM, and "-" indicating lower CRM. The remaining digits designate the electrophoretic charge relative to that of XDH produced by *ry*⁺⁰ attributable to the amino-acid residues inferred to correspond to the three sites inferred from mapping and sequencing results; the sites are indicated in order and are located at +736, +1551, and +3557 in the gene sequence (5); "0" indicates the relative charge at the three sites of *ry*⁺⁰; "-" indicates a more negative charge, i.e. less anodically migrating; and "+" a more positive charge.

^δ 1 = Buchanan and Johnson, 1983, Genetics 104: 301-15; 2 = Clark, Daniels, Rushlow, Hilliker, and Chovnick, 1984, Genetics 108: 953-68; 3 = Chovnick, Gelbart, and McCarron, 1980, Cell 11: 1-10; 4 = Coté, Bender, Chovnick, 1986, Genetics 112: 769-83; 5 = Curtis, personal communication; 6 = Curtis, Clark, Chovnick, and Bender, 1989, Genetics 122: 653-61; 7 = Gelbart, McCarron, Pandey, and Chovnick, 1974, Genetics 78: 869-86; 8 = Lee, Curtis, McCarron, Love, Gray, Bender, and Chovnick, 1987, Genetics 116: 55-66; 9 = McCarron, Gelbart, and Chovnick, 1974, Genetics 76: 289-99; 10 = McCarron, O'Donnell, Chovnick, Bhullar, Hewitt, and Candido, 1979, Genetics 91: 275-93.

^ε The only extant non-derivative *ry*⁺⁰ sequence is carried in the multiple break rearrangement *In(3LR)Ubx*^A.



rosy molecular map

Data of Chovnick and colleagues.

Table II

allele	origin	discoverer	synonym	ref ^α	characterization ^β
<i>ry</i> ¹	spont	Bridges		2, 3, 8, 14, 17, 20, 22, 24	0.1 kb deletion including <i>Sst</i> I site at +0.95 kb; non-complementing allele; null
<i>ry</i> ²	spont	Hadorn & Schwinck		3, 5, 6, 8, 14, 19, 20, 24	<i>B104</i> insertion at +3.2 kb; complementing allele; null
<i>ry</i> ³	spont	Hubby		7, 8, 20, 22, 24	<i>B104</i> insertion at +2.2 kb; leaky
<i>ry</i> ^{3a}	X ray	Schalet		2, 3, 14, 21, 24	Non-complementing allele; null
<i>ry</i> ⁴	X ray	Schalet		2, 3, 8, 14, 15, 24, 29	Apparent point; non-complementing allele; null
<i>ry</i> ⁵	X ray	Schalet		2, 3, 8, 14, 18, 20, 24, 29	19 bp deletion from +294 to +312; frameshift; missing <i>Sst</i> I site at -1.0 kb; non-complementing allele; null
<i>ry</i> ⁶	X ray	Schalet		2, 8, 14, 20, 24, 29	Apparent point; non-complementing allele; null
<i>ry</i> ⁷	X ray	Schalet		2, 8, 14, 24, 29	0.6 kb deletion between +2.9 and +4.2 kb; non-complementing allele; null
<i>ry</i> ⁸	X ray	Schalet		1-3, 8, 14, 15, 18, 20, 24, 29	17 bp deletion from +1283 to +1299; frameshift; null
<i>ry</i> ⁹	X ray	Schalet		2, 3, 8, 14, 24, 29	Apparent point; non-complementing allele; null
* <i>ry</i> ¹⁰	X ray	Schalet		24, 29	Null
* <i>ry</i> ¹¹	X ray	Schalet		24, 29	Null
* <i>ry</i> ¹²	X ray	Schalet		24, 29	Null
* <i>ry</i> ¹³	X ray	Schalet		24, 29	Null
* <i>ry</i> ¹⁴	X ray	Schalet		24, 29	Null
* <i>ry</i> ¹⁵	X ray	Schalet		24, 29	Null
* <i>ry</i> ¹⁶	X ray	Schalet		24, 29	Null
* <i>ry</i> ¹⁷	X ray	Schalet		8, 14, 24, 29	Apparent point; non-complementing allele; null
* <i>ry</i> ¹⁸	X ray	Schalet		14, 24, 29	Non-complementing allele; null
* <i>ry</i> ¹⁹	X ray	Schalet		14, 24, 29	Non-complementing allele; null
<i>ry</i> ²⁰	X ray	Schalet		8, 14, 24, 29	Apparent point; non-complementing allele; null
<i>ry</i> ²¹	X ray	Schalet		8, 14, 24, 29	Apparent point; non-complementing allele; null
* <i>ry</i> ²²	X ray	Schalet		24, 29	Null
<i>ry</i> ²³	X ray	Schalet		2, 3, 8, 14, 24, 29	Apparent point; non-complementing allele; null
<i>ry</i> ²⁴	X ray	Schalet		2, 3, 8, 14, 24, 29	Apparent point; non-complementing allele; null
* <i>ry</i> ²⁵	X ray	Schalet		2, 24, 29	Null
<i>ry</i> ²⁶	X ray	Schalet		2, 3, 8, 14, 18, 20, 24, 29	GG→T (frameshift, Amber) at +2804-5; non-complementing allele; null
* <i>ry</i> ³⁷	X ray	Schalet		24, 29	Null
* <i>ry</i> ³⁸	X ray	Schalet		24, 29	Null
<i>ry</i> ⁴⁰	X ray	Schalet		8, 14, 24, 29	Apparent point; non-complementing allele; null
<i>ry</i> ⁴¹	X ray	Schalet		2, 8, 9, 14, 15, 20, 24, 29	Apparent point; non-complementing allele; null
<i>ry</i> ⁴²	X ray	Schalet		2, 14, 18, 20, 24, 29	Complementing allele; null; 16 bp deletion and insertion of 7 bp of non-rosy DNA at +2030, yielding a net 9 bp "in frame" deletion
* <i>ry</i> ⁴³	X ray	Schalet		24, 29	Null
* <i>ry</i> ⁴⁴	X ray	Schalet		24, 29	Null
<i>ry</i> ⁴⁵	X ray	Schalet		14, 24, 29	Non-complementing allele; null
* <i>ry</i> ⁴⁶	X ray	Schalet		24, 29	Null
* <i>ry</i> ⁴⁷	X ray	Schalet		24, 29	Null
<i>ry</i> ⁴⁸	X ray	Schalet		8, 14, 24, 29	Apparent point; non-complementing allele; null
* <i>ry</i> ⁴⁹	X ray	Schalet		24, 29	Null
* <i>ry</i> ⁵⁰	X ray	Schalet		24, 29	Null
* <i>ry</i> ⁵³	X ray	Schalet		24, 29	Null
<i>ry</i> ⁵⁴	X ray	Schalet		8, 24, 29	<i>In(3R)81;87D8-12</i> ; break between +1.9 and +2.6 kb; null
* <i>ry</i> ⁵⁵	X ray	Kernaghan		24, 29	Null
<i>ry</i> ⁵⁶	X ray	Kernaghan		8, 14, 24, 29	Apparent point; non-complementing allele; null

allele	origin	discoverer	synonym	ref ^α	characterization ^β
<i>ry</i> ⁵⁷	X ray	Kernaghan		8, 14, 24, 29	Apparent point; non-complementing allele; null
<i>ry</i> ⁵⁸	X ray	Kernaghan		8, 14, 24, 29	Apparent point; non-complementing allele; null
<i>ry</i> ⁵⁹	X ray	Kernaghan		8, 14, 24, 29	Apparent point; non-complementing allele; null
<i>ry</i> ⁶⁰	X ray	Kernaghan		7, 8, 14, 20, 24, 29	1.1 kb deletion between +0.95 and +2.6 kb; complementing allele; null
<i>ry</i> ⁶¹	X ray	Kernaghan		14, 24, 29	Non-complementing allele; null
<i>ry</i> ⁶²	X ray	Kernaghan		8, 14, 24, 29	Apparent point; missing <i>Ssr</i> I site at +0.5 kb; non-complementing allele; null
<i>ry</i> ⁶³	X ray	Kernaghan		14, 24, 29	Non-complementing allele; null
<i>ry</i> ⁶⁴	X ray	Kernaghan		8, 14, 24, 29	<i>In(3LR)64E;87D</i> ; breakpoint in +1.5 to +3.6; 1.9 to 3.0 kb deleted; non-complementing allele; null
<i>*ry</i> ⁶⁵	X ray	Kernaghan		24, 29	Null
<i>*ry</i> ⁶⁷	X ray	Kernaghan		24, 29	Null
<i>*ry</i> ⁶⁸	X ray	Kernaghan		24, 29	Null
<i>*ry</i> ⁶⁹	X ray	Kernaghan		24, 29	Null
<i>*ry</i> ⁷¹	X ray	Kernaghan		24, 29	Null
<i>*ry</i> ⁷²	X ray	Kernaghan		24, 29	Null
<i>*ry</i> ⁷³	X ray	Kernaghan		24, 29	Null
<i>*ry</i> ^{77y6}	P	Green		8	5 kb insertion (<i>copia</i> ?) between +1.9 and +2.6 kb; null
<i>ry</i> ¹⁰²	γ ray	Chovnick		8, 14, 15, 20, 26	Apparent point; non-complementing allele; null
<i>ry</i> ¹⁰³	γ ray	Chovnick		8, 14, 15, 20, 26	Apparent point; non-complementing allele; null
<i>ry</i> ¹⁰⁵	γ ray	Chovnick		8	Apparent point; null
<i>ry</i> ¹⁰⁶	γ ray	Chovnick		8, 14, 15, 20, 26	5 kb insertion between +1.3 and +2.6 kb; non-complementing allele; null
<i>ry</i> ¹¹⁰	γ ray	Chovnick		8, 14, 15, 20, 26	Apparent point; non-complementing allele; null
<i>ry</i> ¹¹¹	spont	McCarron	<i>ry</i> ^{e111}	9, 15, 26	Electrophoretic variant at 3' end, probably at +3557
<i>ry</i> ²⁰¹	γ ray	Gelbart		8, 14, 15, 18, 20	Frameshift insert TT at +737; non-complementing allele; null
<i>ry</i> ²⁰³	γ ray	Gelbart		8, 14, 15, 20	Apparent point; non-complementing allele; null
<i>ry</i> ²⁰⁴	γ ray	Gelbart		8, 14, 15, 18, 20	Frameshift deletion of GCC → GC at +685; non-complementing allele; null
<i>ry</i> ²⁰⁵	γ ray	Gelbart		8, 14, 15, 20	Apparent point; non-complementing allele; null
<i>ry</i> ²⁰⁶	EMS	Gelbart		8, 14	Apparent point; non-complementing allele; null
<i>ry</i> ²⁰⁷	EMS	Gelbart		8, 14, 20	Apparent point; complementing allele; null
<i>ry</i> ²⁰⁸	EMS	Gelbart		8, 14	Apparent point; non-complementing allele; null
<i>ry</i> ²⁰⁹	EMS	Gelbart		8, 14	Apparent point; non-complementing allele; null
<i>ry</i> ²¹⁰	EMS	Gelbart		8, 14	Apparent point; non-complementing allele; null
<i>ry</i> ²¹¹	EMS	Gelbart		8	Apparent point; leaky ^ε
<i>ry</i> ²¹³	EMS	Gelbart		8	Apparent point; leaky
<i>ry</i> ²¹⁴	EMS	Gelbart	<i>ry</i> ^{ps214}	1, 8, 14, 20	Apparent point; leaky; XDH activity 24% normal
<i>ry</i> ²¹⁷	spont	McCarron	<i>ry</i> ^{e217}	9, 14, 15, 20	Electrophoretic variant at +736
<i>ry</i> ²¹⁸	EMS	Gelbart	<i>ry</i> ^{ps218}	5, 8, 20	Apparent point; leaky
<i>ry</i> ²¹⁹	EMS	Gelbart	<i>ry</i> ^{ps219}	8, 20	Apparent point; leaky
<i>ry</i> ²²⁰	EMS	Gelbart		8	Apparent point; null
<i>ry</i> ²²²	EMS	Gelbart	<i>ry</i> ^{ps222}	8	Apparent point; 2% XDH activity; resistant to HPP and purine
<i>ry</i> ²²³	EMS	Gelbart	<i>ry</i> ^{ps223}	5, 8, 20	Apparent point; leaky
<i>ry</i> ²²⁴	EMS	Gelbart		8	Apparent point; leaky
<i>ry</i> ²²⁵	EMS	Gelbart		8	Apparent point; leaky
<i>ry</i> ²²⁶	EMS	Gelbart		8	Apparent point; leaky
<i>ry</i> ²²⁷	EMS	Gelbart		8	Apparent point; leaky
<i>ry</i> ²²⁸	EMS	Gelbart	<i>ry</i> ^{ps228}	8	Apparent point; leaky
<i>ry</i> ²³⁰	EMS	Gelbart	<i>ry</i> ^{ps228}	5, 8	Apparent point; leaky
<i>ry</i> ³⁰¹	spont	McCarron	<i>ry</i> ^{e230}	9	Electrophoretic variant at +3557
<i>ry</i> ³⁰²	EMS	Gelbart		6-8, 14, 15, 20	7.6 kb insertion (<i>calypso</i>) at +0.5 kb; non-complementing allele; null
<i>ry</i> ³⁰³	spont	McCarron	<i>ry</i> ^{e302}	9, 15	Electrophoretic variant at +3557
<i>ry</i> ⁴⁰²	spont	McCarron	<i>ry</i> ^{e303}	15	Electrophoretic variant at +736
<i>ry</i> ⁴⁰⁴	γ ray	Gelbart		1, 8, 15, 20	Apparent point; null
<i>ry</i> ⁴⁰⁴	EMS	Gelbart		14	Non-complementing allele; null
<i>ry</i> ⁴⁰⁵	EMS	Gelbart		1, 8, 14, 20	Apparent point; non-complementing allele; null
<i>ry</i> ⁴⁰⁶	EMS	Gelbart		1, 7, 14, 27	Complementing allele: G → A at +451; null
<i>ry</i> ⁴⁰⁷	EMS	Gelbart	<i>ry</i> ^{ps407}	8	Apparent point; leaky

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allele	origin	discoverer	synonym	ref ^α	characterization ^β
<i>ry</i> 408	spont	McCarron	<i>ry</i> ^{e408}	9, 15	Electrophoretic variant at +3557
<i>ry</i> 409H ^γ	spont	McCarron	<i>ry</i> ⁱ⁴⁰⁹	1, 4, 10, 27	<i>cis</i> -acting control element site in intron 1 at -1145; normal → fatbody specific overproduction
<i>ry</i> 501	γ ray	Gelbart		8, 14, 15, 20	Apparent point; complements <i>ry</i> ⁶⁰⁶ ; null
<i>ry</i> 502	γ ray	Gelbart		8, 14, 15, 18, 20	3 bp deletion from +683 to +685 and insertion of an "A"; frameshift; non-complementing allele; null
<i>ry</i> 503	γ ray	Gelbart		8	Apparent point; null
<i>ry</i> 506	γ ray	Gelbart		7, 8, 14, 15, 20	3.4 kb deletion between +1.1 and +5.0 kb; null
<i>ry</i> 507	spont	McCarron		9, 14, 15, 20	Electrophoretic variant at +736
<i>ry</i> 508	spont	McCarron	<i>ry</i> ^{e508}	9, 15	Electrophoretic variant at +3557
<i>ry</i> 509	HN2	McCarron		18	Deletion of bases +626 through +698; frameshift; non-complementing allele; null
<i>ry</i> 516	ENU	McCarron		18, 25	C → T (Amber) at +1521; non-complementing allele; null
<i>ry</i> 523	ENU	McCarron		23, 25	G → A in 3' splice dinucleotide of intron 1 at -551; non-complementing allele; null
<i>ry</i> 531	DEB	McCarron		18	G → A at +3312; complements <i>ry</i> ⁶⁰⁶ ; null
<i>ry</i> 537	ENU	McCarron		7, 25	Deletion of 200-250 bp in <i>Pvu</i> II 2.55 kb fragment; non-complementing allele; null
<i>ry</i> 538	DEB	McCarron		23	Deletion of 56 bp at -168 to -111; non-complementing allele; null
<i>ry</i> 544	ENU	McCarron	<i>G1011</i> → <i>E</i>	18, 25	G → A at +2721; 75% CRM; non-complementing allele; null
<i>ry</i> 545	ENU	McCarron		23, 25	G → A in 3' splice dinucleotide of intron 1; non-complementing allele; null
<i>ry</i> 549	ENU	McCarron		18, 25	T → C at +3498; non-complementing allele; null
<i>ry</i> 553	ENU	McCarron		18, 25	C → T (Opal) at +816; non-complementing allele; null
<i>ry</i> 554	ENU	McCarron		18, 25	G → A (Amber) at +109; non-complementing allele; null
<i>ry</i> 556	TEM	McCarron			Non-complementing allele; null
<i>ry</i> 561	ENU	McCarron		18, 25	T → C at +846; non-complementing allele; null
<i>ry</i> 564	ENU	McCarron		18, 25	C → T (Amber) at +75; non-complementing allele; null
<i>ry</i> 569	ENU	McCarron		25	85% CRM; non-complementing allele; null
<i>ry</i> 573	ENU	McCarron		18, 25	G → A at +3179; complements <i>ry</i> ⁶⁰⁶ ; null
<i>ry</i> 601	EMS	Gelbart		8, 14	Apparent point; null
<i>ry</i> 602	EMS	Gelbart	<i>S357</i> → <i>F</i>	8, 14, 18, 20	C → T at +478; complementing allele; leaky
<i>ry</i> 603	EMS	Gelbart		14	Non-complementing allele; null
<i>ry</i> 604	EMS	Gelbart		8, 14	Apparent point; null
<i>ry</i> 605	EMS	Gelbart		8, 14	Apparent point; new <i>Sst</i> I site at +1.7 or +4.1; null
<i>ry</i> 606	EMS	Gelbart		1, 5, 7, 8, 10, 14, 18, 20	G → A at -468; complementing allele; null
<i>ry</i> 607	EMS	Gelbart		8, 14	Apparent point; null
<i>ry</i> 608	EMS	Gelbart		8, 14	Apparent point; null
<i>ry</i> 609	EMS	Gelbart		8, 14, 18, 20	G → A at +3506; complementing allele; null
<i>ry</i> 610	EMS	Gelbart		8	Apparent point; leaky
<i>ry</i> 611	EMS	Gelbart	<i>ry</i> ^{ps611}	8, 14	Apparent point; leaky; XDH activity 1% normal
<i>ry</i> 612	EMS	Gelbart	<i>ry</i> ^{ps612}	5, 8, 14, 20	Apparent point; leaky; XDH activity 5% normal
<i>ry</i> 613	EMS	Gelbart		8	Apparent point; leaky
<i>ry</i> 621	spont	McCarron	<i>ry</i> ^{e621}	9	Electrophoretic variant at +736
<i>ry</i> 1001	EMS	Gelbart		14	Non-complementing allele; null
<i>ry</i> 1002	EMS	Gelbart		8, 14	Apparent point; null
<i>ry</i> 1003	EMS	Gelbart		8, 14, 27	Apparent point; complementing allele; null
<i>ry</i> 1004	spont	McCarron	<i>ry</i> ^{e1004}	9, 27	Electrophoretic variant at +1551
<i>ry</i> 1005 ^γ	spont	McCarron	<i>ry</i> ⁱ¹⁰⁰⁵	4, 10, 27	T → C at -1701; normal → underproducer; <i>cis</i> -acting control element
<i>ry</i> 1009	X ray	O'Donnell		8	1.0 deletion between 0.95 and 2.6 kb; null
<i>ry</i> 1012	X ray	O'Donnell		8	Apparent point; null
<i>ry</i> 1202	γ ray	Gelbart		8	Apparent point; null
<i>ry</i> 1302	γ ray	Gelbart		8	Apparent point; null
<i>ry</i> 1401	EMS	Gelbart		8, 20	Apparent point; null
<i>ry</i> 1404	spont	McCarron	<i>ry</i> ^{e1404}		Electrophoretic variant; slower than <i>ry</i> ⁺¹⁰
<i>ry</i> 1407	spont	McCarron	<i>ry</i> ^{e1407}	9	Electrophoretic variant at +3557
<i>ry</i> 1901	spont	McCarron		8	Apparent point; null
<i>ry</i> 2101	P	Chovnick		7, 8	<i>cop</i> ia insert at +0.3 kb; null

allele	origin	discoverer	synonym	ref ^α	characterization ^β
<i>ry</i> 5102	ENU	McCarron		18, 25	G → A at -1365; splice donor mutation; non-complementing allele; null
<i>ry</i> 5105	ENU	McCarron		18, 25	G → A (Opal) at +2683; non-complementing allele; null
<i>ry</i> 5106	ENU	McCarron		25	20% CRM; non-complementing allele; null
<i>ry</i> 5107	ENU	McCarron		25	80% CRM; non-complementing allele; null
<i>ry</i> 5115	ENU	McCarron		18, 25	G → A (Opal) at +110; non-complementing allele; null
<i>ry</i> 5117	ENU	McCarron		18, 25	T → C at -1364; splice donor mutation; non-complementing allele; null
<i>ry</i> 5122	ENU	McCarron		18, 25	Deletion of 500-700 in 602 region plus 300 bp insertion of unknown material; non-complementing allele; null
<i>ry</i> 5135	ENU	McCarron		18, 25	G → A (Opal) at +2683; non-complementing allele; null
<i>ry</i> 5144	ENU	McCarron		18, 25	C → T (Opal) at +816; non-complementing allele; null
<i>ry</i> 5148	ENU	McCarron		18, 25	C → T (Amber) at +2573; non-complementing allele; null
<i>ry</i> 5163	ENU	McCarron		18, 25, 28	C → T (Amber) at +3626; 23 amino acids from 3' end; temperature sensitive; leaky
<i>ry</i> 5182	ENU	McCarron		9, 23, 25	G → A (new start) at -1435; 35% CRM; leaky
<i>ry</i> 5184	ENU	McCarron		18, 25	C → T at +3513; leaky
<i>ry</i> 5185	ENU	McCarron		18, 25	G → A at +466; 100% CRM; leaky
<i>ry</i> 5187	ENU	McCarron		18, 25	G → A at +3524; leaky
<i>ry</i> 5192	ENU	McCarron		18, 25	A → G at +213 (altered mobility 1.07); complementing allele; leaky
<i>ry</i> 5198	ENU	McCarron		23, 25	C → T at -225; 40% CRM; leaky
<i>ry</i> 5204	ENU	McCarron		23, 25	T → C at -1388 and a frameshift deletion of a T at -1386; leaky
<i>ry</i> 5205	ENU	McCarron	<i>ry</i> ^{ps5205}	5, 18, 25	G → A at +3486; 25% CRM; complementing allele; leaky
<i>ry</i> 5207	ENU	McCarron		23, 25	rearrangement breakpoint near -1200
<i>ry</i> 5208	ENU	McCarron		23, 25	G → A at -1366 (alters splice efficiency at exon 1/intron 1 junction); leaky
<i>ry</i> 5214	ENU	McCarron		18, 25	T → C at +3850 (mRNA processing?); 100% CRM; leaky
<i>ry</i> 5215	ENU	McCarron		18, 25	C → A at +43; about 35% CRM
<i>ry</i> 5220	ENU	McCarron		18, 25	T → C at -226; 15% CRM; leaky
<i>ry</i> 5231	ENU	McCarron	<i>E89 → K</i>	18, 25	G → A at -328; altered mobility -- slower than <i>ry</i> ⁺⁵ ; 50% CRM; leaky
<i>ry</i> 5235	ENU	McCarron		18, 25, 28	T → C at +1539; 10% CRM; temperature sensitive; leaky
<i>ry</i> 5241	ENU	McCarron		18, 25, 28	C → T (Amber) at +1772; temperature sensitive; null ^δ
<i>ry</i> 5252	ENU	McCarron		18, 25	T → C at -123; leaky
<i>ry</i> 5256	ENU	McCarron		18, 25	C → T (Amber) at +3221; null ^δ
<i>ry</i> 5262	ENU	McCarron		18, 25, 28	C → T (Amber) at +3626 (23 amino acids from 3' end); 5% CRM; temperature sensitive; leaky
<i>ry</i> 5264	ENU	McCarron		18, 25	G → A at +1807; 10% CRM; leaky
<i>ry</i> 5281	ENU	McCarron		18, 25	C → T at -214; 50% CRM; leaky
<i>ry</i> 5322	ENU	McCarron		18, 25	G → A at +180; about 100% CRM; leaky
<i>ry</i> 5331	ENU	McCarron		18, 25	G → A (new start) at -1435; 40% CRM; leaky
<i>ry</i> a1	EMS	Girton		16	Null
<i>ry</i> a2	EMS	Girton		16	Null
<i>ry</i> a3	EMS	Girton		16	Complementing allele; null
<i>ry</i> a4	EMS	Girton		16	Complementing allele; null
<i>ry</i> a5	EMS	Girton		16	Complementing allele; null
<i>ry</i> a6	EMS	Girton		16	Null
<i>ry</i> a7	EMS	Girton		16	Null
<i>ry</i> a8	EMS	Girton		16	Complementing allele; null
<i>ry</i> a9	EMS	Girton		16	Complementing allele; null
<i>ry</i> a10	EMS	Girton		16	Complementing allele; null
<i>ry</i> a11	EMS	Girton		16	Null
<i>ry</i> L.12	EMS	E. B. Lewis	<i>L.16608.12</i>	14	non-complementing allele; null
<i>ry</i> L.14	EMS	E. B. Lewis	<i>L.16608.14</i>	14	non-complementing allele; null
<i>ry</i> L.18	EMS	E. B. Lewis	<i>L.16608.18</i>	14	non-complementing allele; null
<i>ry</i> L.19	EMS	E. B. Lewis	<i>L.16608.19</i>	7, 14, 20	G → A at +3332; complementing allele; null
[<i>ry</i> ^{+14-1a}]	P	Daniels		12, 13	<i>ry</i> ⁺ P-element transposition to 57F; very reduced expression
[<i>ry</i> ^{+14-1a-4}]	P	Daniels		12, 13	<i>ry</i> ⁺ P-element transposition to 68A; near normal expression
[<i>ry</i> ⁺⁷²⁻¹]	P	Daniels		12, 13	<i>ry</i> ⁺ P-element transposition to 100D; reduced

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allele	origin	discoverer	synonym	ref ^α	characterization ^β
[<i>ry</i> ⁺¹⁷⁷⁻¹]	P	Daniels		12, 13	expression <i>ry</i> ⁺ P-element transposition to 16D; reduced expression
[<i>ry</i> ⁺²⁰¹⁻⁴]	P	Daniels		12, 13	expression <i>ry</i> ⁺ P-element transposition to 43EF; elevated expression
[<i>ry</i> ⁺²⁴¹⁻⁸]	P	Daniels		12, 13	expression <i>ry</i> ⁺ P-element transposition to 76F; normal expression
[<i>ry</i> ^{+401.1}]	P	Spradling and Rubín		12, 13, 30	<i>ry</i> ⁺ P-element transposition to 101; variegated expression
[<i>ry</i> ^{+403.1}]	P	Spradling and Rubín		12, 13, 30	<i>ry</i> ⁺ P-element transposition to 7D; normal expression
[<i>ry</i> ⁺²²¹⁶]	P	Daniels		11, 12, 13	<i>ry</i> ⁺ P-element transposition to <i>sd</i> at 13F; normal expression
[<i>ry</i> ⁺²²¹⁶⁻⁵⁴⁷]	P	McCarron		9, 11, 13	deletion internal to intron 1 from -1263 to -809; reduced fat body activity; leaky

^α 1 = Chovnick, Gelbart, McCarron, Osmond, Candido, and Ballie, 1976, *Genetics* 84: 233-55; 2 = Chovnick, Schalet, Kernaghan, and Krauss, 1964, *Genetics* 50: 1245-59; 3 = Chovnick, Schalet, Kernaghan, and Talsma, 1962, *Am. Nat.* 96: 281-96; 4 = Clark, Daniels, Rushlow, Hilliker, and Chovnick, 1984, *Genetics* 108: 953-68; 5 = Clark, Hilliker, and Chovnick, 1986, *Genet. Res.* 47: 109-16; 6 = Clark, Hilliker, and Chovnick, 1988, *Genetics* 118: 261-66; 7 = Clark, McCarron, Love, and Chovnick, 1986, *Genetics* 112: 755-67; 8 = Coté, Bender, Curtis, and Chovnick, 1986, *Genetics* 112: 769-83; 9 = Curtis and Bender (unpublished results); 10 = Curtis, Clark, Chovnick, and Bender, 1989, *Genetics* 122: 653-61; 11 = Daniels, McCarron, Love, and Chovnick, 1985, *Genetics* 109: 95-117; 12 = Daniels, McCarron, Love, Clark, and Chovnick, 1986, *Genetics* 113: 265-85; 13 = Dutton and Chovnick, 1988, *Dev. Biol.* 5: 267-316; 14 = Gelbart, McCarron, and Chovnick, 1976, *Genetics* 84: 211-32; 15 = Gelbart, McCarron, Pandey, and Chovnick, 1974, *Genetics* 78: 869-86; 16 = Girton, Lo, and Bell, 1979, *Can. J. Genet. Cytol.* 21: 379-89; 17 = Glassman and Mitchell, 1959, *Genetics* 44: 153-62; 18 = Gray and Bender (unpublished results); 19 = Hadorn and Schwinck, 1956, *Z. Indukt. Abstamm. Vererbungsleh.* 87: 528-53; 20 = Hilliker and Chovnick, 1981, *Genet. Res.* 38: 281-96; 21 = Hubby, 1961, *DIS* 35: 46; 22 = Hubby and Forrest, 1960, *Genetics* 45: 211-24; 23 = Lee, Curtis, McCarron, Love, Gray, Bender, and Chovnick, 1987, *Genetics* 116: 55-66; 24 = Lindsley and Grell, 1968, *Carnegie Inst. Wash. Publ. No.* 627; 25 = McCarron and Chovnick, 1981, *Genetics* 97: s70-71; 26 = McCarron, Gelbart, and Chovnick, 1974, *Genetics* 76: 289-99; 27 = McCarron, O'Donnell, Chovnick, Bhullar, Hewitt, and Candido, 1979, *Genetics* 91: 275-93; 28 = Reaum and Chovnick (unpublished results); 29 = Schalet, 1964, *DIS* 39: 62-64; 30 = Spradling and Rubín, 1982, *Science* 218: 341-47.

^β +1 of the nucleotide sequence defined as the second base pair in an *EcoRI* site in the second exon; transcription from left to right.

^γ Additional description below.

^δ Adult eye color is not the same as in most null mutants. Some residual XDH activity may be present.

^ε Leaky = hypomorph.

cytology: Placed in 87D8-12 based on *Df(3R)ry74 = Df(3R)87D4;87D12* and in 87D11-12 based on *in situ* hybridization with radioactively-labelled *ry* DNA probe (Lefevre, 1971, *DIS* 46: 40; Spierer, Spierer, Bender, and Hogness, 1983, *J. Mol. Biol.* 168: 35-50) and with biotin labelled *ry* DNA probe [Duttaroy, 1988, (unpublished results)].

molecular biology: Gene sequence originally included in a 315-kb walk in region 87D-E (Bender, Spierer, and Hogness, 1983, *J. Mol. Biol.* 168: 17-33). Location of *ry* restricted to region between the right breaks of *Df(3R)kar*^{1G27} between coordinates -191 and -187 kb and *Df(3R)ry*³⁶ between -166 and -163.5 kb (Spierer, Spierer, Bender, and Hogness, 1983, *J. Mol. Biol.* 168: 35-50). A group of *ry* mutants with molecular lesions were confined to 4 kb between -171 and -167 kb; 0 on the nucleotide map is within an *EcoRI* restriction site at -171 kb (Clark, McCarron, Love, and Chovnick, 1986, *Genetics* 112: 755-67; and Coté, Bender, Curtis, and Chovnick, 1986, *Genetics* 112: 769-83). The genomic sequence has been cloned and sequenced (Lee, Curtis, McCarron, Love, Gray, Bender, and Chovnick, 1987, *Genetics* 116: 55-66; Keith, Riley, Kreitman, Lewontin, Curtis, and Chambers, 1987, *Genetics* 116: 67-73). The gene is transcribed from the centromere (proximal to distal) and comprises four exons and three introns; exon one is at least 179 bp in length; it

contains the ATG codon and encodes the first fourteen amino acids. The codon for Lys¹⁴ is followed by an 815 bp intron (coordinates -1365 to -551). Exon 2 begins at nucleotide -550 with the codon for Val¹⁵ and extends for 2601 base pairs encoding 867 amino acids ending in Ser⁸⁸¹. Intron 2 contains 281 base pairs from 2052 to 2332. Exon 3 is 1314 base pairs in length and encodes 438 amino acids from Val⁸⁸² to Leu¹³¹⁹. Finally, intron 3 extends from base pair 3647 through 3711 and is followed by the 3' exon beginning with Leu¹³²⁰ and terminating at the carboxyl end with Pro¹³³⁵ at nucleotide 3759. Seven conservative nucleotide substitutions noted between the Canton-S genomic sequence and Oregon-R cDNA sequences.

The molecular biology of *ry* alleles (listed according to their position on the DNA map of the locus) is summarized in the table on the next page.

other information: The locus has been extensively mapped by reciprocal recombination and conversion studies (see appended maps). Seven different classes of complementing or partially complementing alleles described; complementation map circular (Gelbart, McCarron, and Chovnick, 1976, *Genetics* 84: 211-32). *ry*⁺ commonly used as a marker in P-element transformation experiments (Spradling and Rubín, 1982, *Science* 218: 341-47; Rubín and Spradling, 1982, *Science* 218: 348-53).

allele	position ^α	molecular biology ^β	allele	position ^α	molecular biology ^β
<i>ry</i> ¹⁰⁰⁵	-1701	TC; normal→underproducer; 50% CRM	<i>ry</i> ²⁰¹	+737	electrophoretic variant; slower than <i>ry</i> ⁵⁰⁷
<i>ry</i> ⁵³³¹	-1435	G→A; GTG→ATG (new start); 40% CRM; leaky	<i>ry</i> ⁵¹⁴⁴	+816	TT insertion; frameshift; null
<i>ry</i> ⁵¹⁸²	-1435	G→A; GTG→ATG (new start); 35% CRM; leaky	<i>ry</i> ⁵⁵³	+816	C→T; CGA→TGA (Arg→Opal); null
<i>ry</i> ⁵²⁰⁴	(-1382/-1386)	TTTTT→TTTT (frameshift deletion of and 1388 one base) and T→G; produces TGA stop in the new frame two codons downstream; leaky	<i>ry</i> ⁵⁶¹	+846	C→T; CGA→TGA (Arg→Opal); null
<i>ry</i> ⁵²⁰⁸	-1366	G→A; AAG→AAA (no amino acid change); alters splice efficiency of exon 1/intron 1 junction; leaky	<i>ry</i> ¹		T→C; TGG→CGG (Trp→Arg); null
<i>ry</i> ⁵¹⁰²	-1365	G→A; GT→AT (splice donor mutation); null	<i>ry</i> ⁸	+1283	deletion of 0.1 kb <i>Sst</i> I site at +940
<i>ry</i> ⁵¹¹⁷	-1364	T→C; GT→GC (splice donor mutation); null	<i>ry</i> ⁵³⁷	+1283	deletion through +1299; frameshift; null
<i>ry</i> ^[2216-547]	-1263	deletion internal to intron 1 from -1263 1263 to -809; reduced fat body activity	<i>ry</i> ⁵¹⁶	+1521	deletion of 200-250 bp in <i>Pvu</i> II 2.55 kb fragment; null
<i>ry</i> ⁵²⁰⁷		rearrangement breakpoint near -1200	<i>ry</i> ⁵²³⁵	+1539	C→T; CAG→TAG (Gln→Amber); null
<i>ry</i> ⁴⁰⁹	-1145	G→C; normal→overproducer; fat body specific	<i>ry</i> ¹⁰⁰⁴	+1551	T→C; TCC→CCC (Ser→Pro); 10% CRM leaky; temperature sensitive
<i>ry</i> ⁵⁴⁵	-551	G→A; AG→AA (3' splice dinucleotide); null	<i>ry</i> ⁵²⁴¹	+1722	G→A; GAC→AAC (Asp→Asn); electrophoretic variant
<i>ry</i> ⁵²³	-551	G→A; AG→AA (3' splice dinucleotide); null	<i>ry</i> ⁵²⁶⁴	+1807	C→T; CAG→TAG (Gln→Amber); null; ^γ temperature sensitive
<i>ry</i> ⁶⁰⁶	-468	G→A; GGA→GAA (Gly→Glu); null; complementing allele	<i>ry</i> ⁴²	+2030	G→A; GGA→GAA (Gly→Glu); 10% CRM; leaky
<i>ry</i> ⁵²³¹	-328	G→A; GAG→AAG (Glu→Lys); electrophoretic variant; slower than <i>ry</i> ⁺⁵ ; leaky			deletion of 16 bp and insertion of 7 bp of non-rosy DNA yielding a net 9 bp "in frame" deletion; no CRM; null; complementing allele
<i>ry</i> ⁵²²⁰	-226	T→C; TTC→CCC (Ser→Pro); 15% CRM; leaky	<i>ry</i> ³		<i>B104</i> insert at 2.2 kb; leaky
<i>ry</i> ⁵¹⁹⁸	-225	C→T; TCC→TTC (Ser→Phe); 40% CRM; leaky	<i>ry</i> ⁵¹⁴⁸	+2573	C→T; CAG→TAG (Gln→Amber); null
<i>ry</i> ⁵²⁸¹	-214	C→T; CTT→TTT (Leu→Phe); 50% CRM; leaky	<i>ry</i> ⁵¹⁰⁵	+2683	G→A; TGG→TGA (Trp→Opal); null
<i>ry</i> ⁵³⁸	-168	deletion through -111; frameshift; null	<i>ry</i> ⁵¹³⁵	+2683	G→A; TGG→TGA (Trp→Opal); null
<i>ry</i> ⁵²⁵²	-123	T→C; CTC→CCC (Leu→Pro); leaky	<i>ry</i> ⁵⁴⁴	+2721	G→A; GGA→GAA (Gly→Glu); null
<i>ry</i> ⁵²¹⁵	+43	C→A; CCG→CAG (Pro-Gln); 35% CRM; leaky	<i>ry</i> ²⁶	+2804-5	GG→T; GGAG→TAG [Gly→(frameshift) Amber]; null
<i>ry</i> ⁵⁶⁴	+75	C→T; CAG→TAG (Gln→Amber); null	<i>ry</i> ⁴¹	+3095	deletion through +3097; Gly codon lost; null
<i>ry</i> ⁵⁵⁴	+109	G→A; TGG→TAG (Trp→Amber); null	<i>ry</i> ⁵⁷³	+3179	G→A; GGA→AGA (Gly→Arg); null
<i>ry</i> ⁵¹¹⁵	+110	G→A; TGG→TGA (Trp→Opal); null	<i>ry</i> ²		<i>B104</i> insert at +3.2 kb; null; complementing allele
<i>ry</i> ⁵³²²	+180	G→A; GGC→AGC (Gly→Ser); 100% CRM; leaky	<i>ry</i> ⁵²⁵⁶	+3221	C→T; CAG→TAG (Gln→Amber); null ^γ
<i>ry</i> ⁵¹⁹²	+213	A→G; AAG→GAG (Lys→Glu); electrophoretic variant; faster than <i>ry</i> ⁺⁵ ; leaky	<i>ry</i> ⁵³¹	+3312	G→A; GGC→GAC (Gly→Asp); null; complementing allele
<i>ry</i> ⁵	+294	deletion through +312; frameshift yielding two downstream stop codons; null; non-complementing allele	<i>ry</i> ^{L19}	+3332	G→A; GAG→AAG (Glu→Lys); null; complementing allele
<i>ry</i> ²¹⁰¹		<i>calypso</i> insert at +0.3 kb; null	<i>ry</i> ⁵²⁰⁵	+3486	G→A; GGT→GAT (Gly→Asp); 25% CRM; leaky; complementing allele
<i>ry</i> ⁴⁰⁶	+451	G→A; GGA→GAA (Gly→Glu); null; complementing allele	<i>ry</i> ⁵⁴⁹	+3498	T→C; CTC→CCC (Leu→Pro); null
<i>ry</i> ⁵¹⁸⁵	+466	G→A; GGC→GAC (Gly→Asp); 100% CRM; leaky	<i>ry</i> ⁶⁰⁹	+3506	G→A; GGA→AGA (Gly→Arg); null
<i>ry</i> ⁶⁰²	+478	C→T; TCC→TTC (Ser→Phe); leaky	<i>ry</i> ⁵¹⁸⁴	+3513	C→T; TC→TTT (Ser→Phe); leaky
<i>ry</i> ⁵¹²²	+500	deletion of 500-700 bp in 602 region, plus insertion of 300 bp of unknown material; null	<i>ry</i> ⁵¹⁸⁷	+3524	G→A; GCC→ACC (Ala→Thr); leaky
<i>ry</i> ³⁰¹		7.6 kb <i>calypso</i> insert at 0.5 kb; null; non-complementing allele	<i>ry</i> ⁵⁰⁸	+3557	G; GAT (Asp); electrophoretic variant; faster than <i>ry</i> ²³⁰
<i>ry</i> ⁶²		apparent point mutation; missing <i>Sst</i> I site at +508; null; non-complementing allele	<i>ry</i> ³⁰²	+3557	electrophoretic variant; same mobility as <i>ry</i> ⁵⁰⁸
<i>ry</i> ⁵⁰⁹	+626	deletion through +698; frameshift; null	<i>ry</i> ⁴⁰⁸	+3557	electrophoretic variant; same mobility as <i>ry</i> ⁵⁰⁸
<i>ry</i> ⁵⁰²	+683	deletion through +685 and A insertion yielding a net two base deletion frameshift; null; non-complementing allele	<i>ry</i> ¹⁴⁰⁷	+3557	electrophoretic variant; same mobility as <i>ry</i> ⁵⁰⁸
<i>ry</i> ²⁰⁴	+685	deletion of C; GCC→GC; frameshift; null	<i>ry</i> ²³⁰	+3557	G→A; GAT→AAT (Asp→Asn); electrophoretic variant; slower than <i>ry</i> ⁵⁰⁸
<i>ry</i> ⁵⁰⁷	+736	A; CAC (His); electrophoretic variant; faster than <i>ry</i> ⁶²¹	<i>ry</i> ⁵¹⁶³	+3626	C→T; CAG→TAG (Gln→Amber) 23 amino acids from 3' end; leaky
<i>ry</i> ²¹⁷	+736	electrophoretic variant, same mobility as <i>ry</i> ⁵⁰⁷	<i>ry</i> ⁵²⁶²	+3626	C→T; CAG→TAG (Gln→Amber) 23 amino acids from 3' end; 5% CRM; leaky; temperature sensitive
<i>ry</i> ³⁰³	+736	electrophoretic variant, same mobility as <i>ry</i> ⁵⁰⁷	<i>ry</i> ⁵²¹⁴	+3850	T→C; ATGTTTT→ATGCTTT; 100% CRM; leaky
<i>ry</i> ⁶²¹	+736	A→G; CAC→CGC (His→Arg);			

^α^βPositions are relative to the *Eco*RI site in exon 2.The molecular biology was referenced from one or more of the following: Curtis and Bender (unpublished results); Curtis, Clark, Chovnick and Bender, 1989, *Genetics* 122: 653-61; Gray and Bender (unpublished results); Lee, Curtis, McCarron, Love, Gray, Bender, and Chovnick, 1987, *Genetics* 116: 55-66.^γ

Adult eye color is not the same as in most null mutants. Some residual XDH activity might be present.

***ry*⁴⁰⁹**

phenotype: Designation applied to the site at -1145 in *ry*⁺⁴ that is responsible for the higher than normal XDH CRM of that allele (*i.e.*, *ry*^{409H} vs. *ry*^{409N}, the normal alternative; Curtis, Clark, Chovnick, and Bender, 1989, *Genetics* 122: 653-61). Enzyme activity two to three times that of other *ry*⁺ alleles (Chovnick, Gelbart, McCarron, Osmond, Candido, and Baillie, 1976, *Genetics* 84: 223-55); large tissue-specific increase in specific activity observed in late third-instar larval fat body, but not Malpighian tubules; mRNA levels 3.2 times higher than normal (Covington, Fleenor, and Devlin, 1976, *Genetics* 84: 211-32; see also Clark, Daniels, Rushlow, Hilliker, and Chovnick, 1984, *Genetics* 108: 953-68). Maps genetically to the right of *ry*¹⁰⁰⁵ (Clark *et al.*, 1984).

***ry*¹⁰⁰⁵**

phenotype: Designation applied to the site at -1701 in *ry*⁺¹⁰ that is responsible for the lower than normal XDH CRM of that allele (*i.e.*, *ry*^{1005L} vs. *ry*^{1005N}, the normal alternative) (Curtis, Clark, Chovnick, and Bender, 1989, *Genetics* 122: 653-61). Enzyme levels 50% those of other normal alleles (McCarron, O'Donnell, Chovnick, Bhullar, Hewitt, and Candido, 1979, *Genetics* 91: 275-93); mRNA levels 52% normal (Covington, Fleenor, and Devlin, 1976, *Genetics* 84: 211-32). Maps genetically to the left of *ry*⁴⁰⁹ (Clark, Daniels, Rushlow, Hilliker, and Chovnick, 1984, *Genetics* 108: 953-68).

molecular biology: *ry*⁺¹⁰ sequenced from nucleotides -2105 to -260; 25 differences from the corresponding sequence of *ry*⁺⁵ noted; none could be associated with the difference in transcription levels (Lee *et al.*, 1987).

s: sable**location:** 1-43.0.**discoverer:** Bridges, 12g19.**references:** Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. No. 237: 34.**phenotype:** Body color dark with prominent trident. Classification good at 19°, overlaps wild type increasingly with higher temperature. ERG normal (Hotta and Benzer, 1969, Nature 222: 354-56). Viability sometimes reduced. *s* is nonautonomous in gynandromorphs containing both *s* and + tissue (Lewis, 1955, DIS 29: 134). Tyrosinase formed in adult (Horowitz and Fling). β -alanine pools only 39% normal (Wright, 1987, Adv. Genet. 24: 127-222). *s/s*² easily classified. Females hemizygous for *s* very dark and sterile (Craymer and Roy, 1980, DIS 55: 200-04). Suppressed by *su(s)*. RK1 at 19°.**cytology:** Placed in 11F (Lefevre).**other information:** Neither transposable element 412 nor *roo* associated with *s* mutation as indicated in *in situ* hybridization experiments (Searles and Voelker, 1986, Proc. Nat. Acad. Sci. USA 83: 404-08).**s²****discoverer:** Bridges, 17e9.**references:** Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 234.**phenotype:** Body color less dark than *s* and trident more prominent. Expression best at 19°, overlaps wild type at 25° and 30°. Viability excellent. RK1 at 19°.**s^{eb}: sable-ebonized****origin:** Induced by ethyl methanesulfonate.**discoverer:** Fahmy, 1956.**synonym:** *eb*.**references:** Fahmy, 1959, DIS 33: 86.**phenotype:** Fly heavily pigmented with trident pattern and scutellum very dark. Wings slightly shortened; membrane often slightly concave; wing tips occasionally truncate. Males viable and fertile; females sterile. RK2.**S: Star****location:** 2-1.3 (0.02 unit to the left of *ast*).**references:** Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 259 (fig.).

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 213 (fig.).

Lewis, 1945, Genetics 30: 137-66.

1951, Cold Spring Harbor Symp. Quant. Biol. 16: 159-74 (fig.).

Mayer and Nüsslein-Volhard, 1988, Genes Dev. 2: 1496-1511.

Renfranz and Benzer, 1989, Dev. Biol. 136: 411-29.

phenotype: Eyes slightly smaller and narrower than wild type; texture somewhat rough from rounded, irregular facets. Arrangement of hairs on surface of eye irregular. *S/ast* has small rough eyes; *S ast/+* + is like *S/+*. Enhanced by *E(S)*; partially suppresses *px* and *net* (Bedichuk, 1936, DIS 5: 24; Lewis, 1945). Homozygote dies in late embryonic stage (Sivertzev-Dobzhansky, 1927, Wilhelm Roux's Arch. Entwicklungsmech. Organ. 109: 535-48; Sonnenblick and Huettner, 1938, Genetics 23: 169). A member of the so-called spitz group of mutants; embryos lack structures derived from ventral-lateral region of blastoderm. Denticle bands narrow and

ventral arms of head skeleton fused. Anal pads reduced. Transverse commissures of ventral nervous system reduced; Keilin organs, maxillary and antennal sense organs strongly reduced. Lethal in homozygous clones in female germ line (Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82; Mayer and Nüsslein-Volhard). RK1.

alleles: Many deficiencies formerly described as alleles listed under *Df(2L)S*.

allele	origin	discoverer	synonym	ref ^α	comments
S¹	spont	Bridges, 15b21		1, 5	
S²	spont	Redfield, 25k	<i>S^R</i>	8	<i>In(2L)Cy</i>
S⁵¹	UV	Meyer, 51b		4	
S⁵⁴	EMS			6	
S^{11N}	EMS			6	
*S^K		Krivshenko		2	<i>In(2LR)SK</i>
*S^L	X ray	Lewis, 1940		3	<i>T(2;3)21E2-3;88D6-8</i>
*S^M		Muller, 1928		7	<i>T(2;3)21E2-3;79D2-E1</i>
S^r			ast		
*S^W	X ray	Whittinghill, 47b			<i>In(2L)Cy</i>
S^X	X ray	Lewis		3	induced simultaneously with <i>ast^X</i>

^α 1 = Bridges and Morgan, 1919, Carnegie Inst. Wash. Publ. 278: 279 (fig.); 2 = Krivshenko, 1936, DIS 5: 8; 3 = Lewis, 1945, Genetics 30: 147-51; 4 = Meyer, 1952, DIS 26: 67; 5 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 213 (fig.); 6 = Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82; 7 = Painter and Muller, 1928, J. Hered. 20: 287-98; 8 = Stern and Bridges, 1926, Genetics 11: 507-08;

cytology: Placed in the 21E1-2 doublet on the basis of its being covered by neither Y^{P2D} of $T(Y;2)21E = T(Y;2)21D4-E1$ nor 2^{P4D} of $T(2;4)ast^v = T(2;4)21E2-3;101$; the synthetic deficiency formed by combining these two elements produces a Star phenotype in combination with a normal second chromosome (Lewis, 1941, Proc. Nat. Acad. Sci. USA 27: 31-35).**other information:** A pseudoallele, *i.e.*, probably a gain-of-function allele of *ast*. In crossover tests, *S* localizes to the left of *ast* (Lewis, 1941, Proc. Nat. Acad. Sci. USA 27: 31-35; Lewis, 1945, 1951).*S15-S38*: see *Cp15-Cp38**S100*: see *nofA**S-i*: see *e(S)**sa*: see *crm^{sa}***sallimus**: see *sls**salmon*: see *g****Sa: Salmon****location:** 2- or 3- (rearrangement).**origin:** X ray induced.**discoverer:** Van Atta, 30k1.**references:** 1932, Am. Naturalist 66: 93-95.

1932, Genetics 17: 637-59.

1935, DIS 3: 15.

phenotype: Eye color wine at eclosion, becomes dark salmon with age. Homozygous lethal. RK1A.**cytology:** Associated with *T(2;3)Sa*; breaks proximal in 2L and 3L.

sab: straight abdomen*location:** 1-58.9.**origin:** Induced by D-1:6-dimethanesulfonyl mannitol (CB. 2511).**discoverer:** Fahmy, 1958.**references:** 1964, DIS 39: 58.**phenotype:** Abdomen long, narrow, and straight. Bristles somewhat fine. Male viable and fertile. RK3.**Sab:** see **Sab** under **BXC****sable:** see **s****sable duplication:** see **su(s)****sad: shadow (C. Nüsslein-Volhard)****location:** 3-51.**origin:** Induced by ethyl methanesulfonate.**synonym:** *karmoisin ghost*.**references:** Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.**phenotype:** Embryonic lethal; no differentiation of cuticle or head skeleton; posterior of embryo condensed.**alleles:** Two.**cytology:** Placed in 86F7-87A7 based on inclusion in *Df(3R)E229 = Df(3R)86F6-7;87B1-2* but not *Df(3R)kar-D1 = Df(3R)87A7-8;87D1-2*.**safranin:** see **sf****sal: spalt****location:** 2-44.**references:** Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82. Jürgens, 1988, EMBO J. 7: 189-96.

Frei, Schuh, Baumgartner, Burri, Noll, Jürgens, Seifert, Nauber, and Jäckle, 1988, EMBO J. 7: 197-204.

phenotype: Embryonic lethal. Shows partial homeotic transformation of labium to prothorax and of A9 and 10 toward A8. Point mutants judged to be amorphic or nearly so judging from the phenotype of *sal/Df* compared with *sal/sal*. *sal; Abd-B*-double mutants exhibit thoracic structures in parasegments 14 and 15; similarly, *sal* and *Scr* seem to act independently on head structures in parasegments 2 and possibly 1.**alleles:**

allele	origin	discoverer	synonym	ref ^α
sal¹	EMS		<i>sal IIA55</i>	1
sal²	EMS		<i>sal IIB57</i>	1
sal³	EMS	Anderson	<i>sal 445</i>	

^α 1 = Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.**cytology:** Placed in 32F2-33A2 based on its inclusion in *Df(2L)esc-P2-0 = Df(2L)32F2-3;33A1-2* but not *Df(2L)esc10 = Df(2L)33A1-2;33B2-3* (Frei *et al.*).**molecular biology:** *sal* lies in the region of a chromosomal walk (Frei, Baumgartner, Edström, and Noll, 1985, EMBO J. 4: 979-87; Kilchherr, Baumgartner, Bopp, Frei, and Noll, 1986, Nature 321: 493-99); an early zygotic 0.8 kb transcript identified from the region between -385.25 and -377.5 of the walk (described under molecular biology of *prd*); injection of antisense RNA from this sequence is able to induce *sal* phenocopy formation. Transcript first appears at 0-2 h, is prevalentfrom 2-8 h after which it declines. Rescue of embryonic lethality by transformation requires 14 kb of 5' flanking sequence in addition to the 0.8 kb coding region; such transformants, however, do not complete larval development, implying later *sal* function. Sequencing reveals the presence of a single 57 base pair intron between codons 4 and 5 and that transcription is from left to right; the conceptual polypeptide comprises 142 amino acids and has a molecular weight of 14,533 daltons; the amino acid sequence reveals a series of three-amino-acid repeats and a high incidence of Gly residues; it consists of a larger hydrophobic amino-terminal and a hydrophilic carboxy-terminal part. *In situ* hybridization of labelled cDNA to sections of wild-type embryos reveals that *sal* transcript accumulates to high levels in the segmental Anlagen affected in mutant embryos but is also found in regions of the embryo where no functional requirement has been demonstrated. Homology detected in DNA of *D. simulans* and *D. orena*, but not of *D. hydei* or *D. virilis* (Reuter, Schuh, and Jäckle, 1989, Proc. Nat. Acad. Sci. USA 86: 5483-86).**salivary gland secretion:** see **sgs****sallimus:** see **sls****salmon:** see **g****Salmon:** see **Sa****sam: sperm amotile****location:** 1-3.7 (0.24 unit distal to *dnc*).**references:** Salz, Davis, and Kiger, 1982, Genetics 100: 587-96.**phenotype:** Males sterile with amotile sperm; homozygous females fertile.**alleles:** Two ethyl-methanesulfonate-induced alleles, *sam¹* and *sam²*.**cytology:** Placed in 3D4; covered by *Dp(1;2)w^{+51b7} = Dp(1;2)3C2;3D6-E1* but not *w^{+Y} = Dp(1;Y)2D1-2;3D3-4*; also maps to the left of *dnc* in 3D4.**molecular biology:** Included in the chromosome walk around *dnc* (Davis and Davidson, 1984, Mol. Cell Biol. 4: 358-67). Molecular and genetic mapping data suggest that *sam* lies in the 79 kb intron that separates exons 1 and 2 of *dnc*. May be responsible for the 2.0-kb transcript from coordinates 17-20 (Chen, Malone, Beckendorf and Davis, 1987, Nature 329: 721-24).**sandpaper:** see **sdp****sas: stranded-at-second (D. R. Cavener)****location:** 3-{48}.**synonym:** *l(3)84Cd* and *rfd*.**references:** Cavener, Corbett, Cox, and Whetten, 1986, EMBO J. 5: 2939-48.

Cavener, Otteson, and Kaufman, 1986, Genetics 114: 111-23.

Schneuwly, Kuroiwa, Gehring, 1987, EMBO J. 6: 201-6. Schonbaum, 1990, Ph.D. Dissertation, Vanderbilt University.

phenotype: Homozygous lethal; homozygotes die at the first to second instar molt or at second instar. Larvae do not grow and exhibit a segmentally repeated pattern of tanned spots on the ventral cuticle between the fourth and fifth row of setae and sometimes between the first and second row of setae. Tracheae are convoluted.

alleles:

allele	origin	discoverer	synonym
<i>sas</i> ¹¹	EMS	Grigliatti	<i>l(3)3.6</i>
<i>sas</i> ¹²	spont.	Green	<i>l(3)73b</i>
<i>sas</i> ¹³	X ray	Denell	<i>l(3)d2</i>
<i>sas</i> ¹⁴	EMS	Cavener	<i>l(3)g7</i>
<i>sas</i> ¹⁵	EMS	R. Lewis	<i>l(3)r16</i>
<i>sas</i> ¹⁶	EMS	R. Lewis	<i>l(3)r17</i>
<i>sas</i> ¹⁷	EMS	R. Lewis	<i>l(3)r27</i>

cytology: Placed in 84C1-6 on the basis of *In(3R)Antp*^{73b} being a *sas* allele and complementation tests with *Df(3R)Antp1* and *Df(3R)Scx2*.

molecular biology: Genomic clone (Cavener, *et al.*) and cDNA clone isolated (Schneuwly, Kuroiwa, and Gehring). *sas* RNA's are expressed at all stages of development in a variety of cuticle secreting ectodermally derived tissues. A 1348-amino-acid sequence (Schonbaum) is inferred for the *sas* protein. The inferred protein sequence is similar to a variety of cell receptor proteins. *In(3R)Antp*^{73b} results in the formation of a fusion gene containing 5' exons of *sas* and the 3' end of *Antp*, beginning with exon 2 (Frischer, Hagen, and Garber, 1986, Cell 47: 1017-23).

***saw: sawtooth**

location: 1-0.0 (very close to right of *sc*).

origin: Ultraviolet induced.

discoverer: Edmondson, 51g.

references: 1952, DIS 26: 60.

phenotype: Hairs along wing edge arranged so that edge appears serrated. Wings may warp, especially in female. Fertility and viability excellent. Classification originally easy, but stocks apparently accumulate modifiers so that they came to appear nearly wild type. RK2.

other information: Not separated from *sc* in two crosses between *ac* and *sc* or in 60 crossovers between *sc* and *pn*. Not covered by *Dp(1;2)sc*¹⁹ = *Df(1;2)1B1-2;1B4-7;25-26*. Locus must be slightly to the right of *sc*.

***saw²**

origin: Ultraviolet induced.

discoverer: Edmondson, 51f.

references: 1952, DIS 26: 61.

phenotype: More extreme than *saw*. Wing margins as in *saw*, but wings are strongly warped up or down; thin textured, especially in female. Viability reduced. Fly often becomes stuck in food owing to warped wings. Fertility good; classification easy. RK2.

sax: saxophone

(*T. Schüpbach and E. Wieschaus*)

location: 2- {57}.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

phenotype: Maternal effect lethal, female sterile. Embryos from homozygous mothers gastrulate abnormally. The cephalic furrow is more pronounced than in wildtype. The phenotype is reminiscent of the zygotic embryonic lethal mutation twisted gastrulation (*twg*).

alleles: *sax*¹ and *sax*² originally *sax*^{WO} and *sax*^{HB}, respectively.

cytology: Placed in 41C1-43F8, since uncovered by *Df(2R)pk78s* = *Df(2R)42C1-7;43F5-8*.

sb: soft brown

location: 3-37.7.

origin: Spontaneous.

references: Aparisi and Nájera, 1987, DIS 66: 13-14. 1988, DIS 67: 4-5 and 5-6.

phenotype: Eye color soft brown, darkening with age.

sb: see *ml*

Sb: Stubble

location: 3-58.22.

origin: Spontaneous.

discoverer: Bridges, 23d21.

references: Dobzhansky, 1930, Z. Indukt. Abstamm. Vererbungsl. 54: 427-57 (fig.).

Beaton, Kiss, Fristrom, and Fristrom, 1988, Genetics 120: 453-64.

phenotype: Bristles of *Sb*/+ less than one-half normal length and somewhat thicker than wild type. Fiber bundles in bristle shafts smaller and more numerous than in wild type; occupy a third as much of the bristle cross-sectional area in *Sb* as in wild type (Overton, 1967, J. Morph. 122: 367-80). Developmental studies by Lees and Waddington [1943, Proc. Roy. Soc. (London), Ser. B. 131: 87-110 (fig.)] show that trichogen is shifted to lie more or less on the level of the tormogen. Most alleles viable as homozygotes; the only exceptions are *Sb*¹ and its derivative *Sb*^v; they may be defective for an adjacent locus. Many homoallelic and heteroallelic combinations, involving both *Sb* and *sbd* alleles, display, in addition to shortened thickened bristles, what is termed by Beaton *et al.*, the malformed syndrome (Moore, 1935, DIS 3: 27); it consists of wings greatly reduced in size and short, thick, and twisted mesothoracic and metathoracic legs. *Sb* genotypes defective in elongation of appendage imaginal discs (Beaton *et al.*). This effect is exaggerated in a temperature-sensitive fashion in many genotypes that contain mutants in *BRC*; for example, females that are heterozygous both for *Sb*¹ or *Sb*⁶³ and any of a number of *br* alleles or a *br* deficiency display a strong enhancement of the short-wing deformed-leg syndrome. Similarly the malformed syndrome is engendered in *br*/Y males by the presence of *Sb*/+ (Beaton *et al.*). *Sb*¹/*sbd*² more extreme than *Sb*¹/+. *sbd*²/*Sb*¹ behaves as a recessive *sbd* allele but is homozygous lethal; rare escapers more extreme and easy to recognize (Davis, 1971, Mol. Gen. Genet. 113: 251-72). Classifiable in single dose in triploids. RK1.

alleles: Alleles differ in strength; phenotypes described at end of entry.

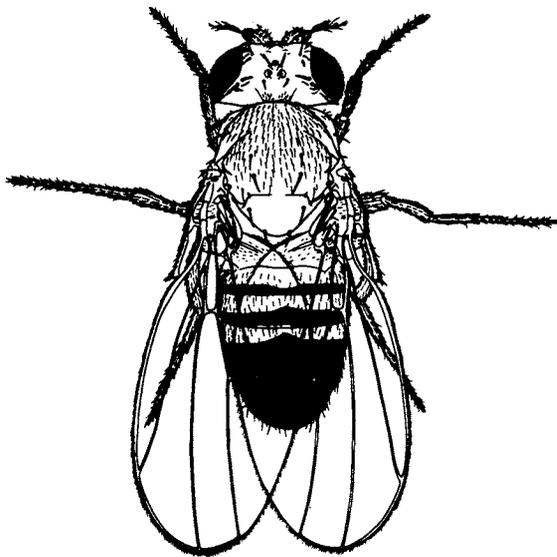
allele	origin	discoverer	synonym	ref ^α	comments
<i>Sb</i> ¹	spont	Bridges, 23d21		2, 3	intermediate allele, homozygous lethal
<i>Sb</i> ⁶³	spont	Merriam, 63b		1, 2	strong allele, homozygous viable
<i>Sb</i> ⁷⁰	spont			2	strong allele, homozygous viable
<i>Sb</i> ^r			<i>sbd</i>	2	
<i>Sb</i> ^{Spi}	X ray	Moore, 31d5		2, 7	intermediate allele, homozygous viable
<i>Sb</i> ^v	X ray	Lewis, 1948		2, 6	weak allele, homozygous lethal
<i>Sb</i> ^W	spont	Crow		4, 5	

^α 1 = CP627; 2 = Beaton, Kiss, Fristrom, and Fristrom, 1988, Genetics 120: 453-64; 3 = Dobzhansky, 1930, Z. Indukt. Abstamm. Vererbungsl. 54: 427-57 (fig.); 4 = Friedenberg, 1963, Hereditas 50: 89-

115; 5 = Friedenberg, 1964, Hereditas 51: 31-66; 6 = Lewis, 1948, DIS 30: 76-77; 7 = Moore, 1935, DIS 3: 27.

cytology: Salivary chromosomes normal (Morgan, Bridges, and Schultz, 1937, Year Book - Carnegie Inst. Washington 36: 301). Placed in 89B4-5, probably in 89B4, by Lewis (1951, Cold Spring Harbor Symp. Quant. Biol. 16: 159-74). This probably corresponds to 89B9-10 on Bridges's revised map.

other information: *Sb* is pseudoallelic to and lies 0.01-0.03 unit to the right of *sbd*². A gain-of-function mutation. Deficiency for the *Sb* locus produces no dominant phenotype (Lewis, 1951).



Sb: Stubble

Edith M. Wallace, unpublished.

Sb^{63b}

phenotype: Bristles of *Sb*^{63/+} somewhat shorter and thicker than *Sb*. Wings and legs normal. Homozygote shows reduced viability, short, thick bristles, small wings, and short deformed legs (Beaton *et al.*). *Sb*^{63b}/*Sb* viable and fertile, more extreme than either heterozygote. RK1.

other information: Allelism inferred from failure to recover recombinants among 100 progeny of *Sb*^{63b}/*Sb*.

Sb^{Sp1}: Stubble-Spike

phenotype: Bristles of *Sb*^{Sp1/+} about two-thirds normal length. Wings and legs normal. Bristles of homozygote one-fourth normal length. Wings reduced, crumpled, or blistered. Legs often short and bowed. *Sb*^{Sp1}/*Sb* viability about 30% wild type. Bristles and wings shorter than homozygous *Sb*^{Sp1}. RK1.

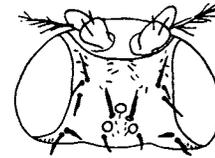
Sb^V: Stubble-Variigated

phenotype: *Sb*^{V/+} has mixture of wild-type and *Sb* bristles. In *X/X/Y* female and *X/Y/Y* male, bristles nearly all *Sb*. In *X/0* male, bristles usually all wild type. *Sb*^V/*Sb* and homozygous *Sb*^V are lethal. RK1A.

cytology: Associated with *T(2;3)Sb^V = T(2;3)41A-C;88;89B*. Superimposed on *In(3R)Mo = In(3R)93D;98F2-6*.

Sb^W: Stubble-Wisconsin

phenotype: Heterozygotes have short stout bristles; homozygotes die as larvae. Tends to persist in laboratory populations (Friedenberg and Chung, 1967, Genetics 57: 957-67).



sbd: stubbloid

From Dobzhansky, 1930, Z. Indukt. Abstamm. Vererbungsbl. 54: 427-57.

sbd: stubbloid

location: 3-58.2.

synonym: *stb*.

references: Beaton, Kiss, Fristrom, and Fristrom, 1988, Genetics 120: 453-64.

phenotype: Bristles short but usually slightly longer than in *Sb/+*. One or both wings often shortened and crumpled at base. Tibia and femur often shortened, thickened, and bowed. *sbd* genotypes interact synergistically with *br* genotypes to exaggerate reductions in wing length and short gnarled legs (Beaton *et al.*). Viability somewhat low. RK2.

alleles: *sbd*¹, *sbd*², and *sbd*^{20l} homozygous viable; other listed alleles are recessive embryonic-larval-boundary lethals. Complementation for lethality but not *sbd* phenotype observed in some heteroallelic combinations; for example *sbd*³² complements, at least partially, all lethal alleles except *sbd*⁴⁷; *sbd*¹⁷ fully complements all lethal alleles except *sbd*¹² and *sbd*⁴⁷, which are partially complemented. *sbd* deficiencies listed under deficiencies.

allele	origin	discoverer	synonym	ref ^α	comments
<i>sbd</i> ¹		Sturtevant, 1926	<i>Sb</i> ^r	3, 7	
<i>sbd</i> ²	spont	Harnly, 271	<i>Sb</i> ^{r2}	1, 2	
<i>sbd</i> ¹²	X ray			5	<i>In(3R)88B2-C1;89B3-16</i>
<i>sbd</i> ¹³	X ray			5	
<i>sbd</i> ¹⁷	X ray			5	<i>In(3R)81;89B10-12</i>
<i>sbd</i> ¹⁸	X ray			5	
<i>sbd</i> ^{20l}	EMS			2	
<i>sbd</i> ²¹	X ray			5	<i>In(3R)86D2-E1;89B3-12</i>
<i>sbd</i> ³²	X ray			5	
<i>sbd</i> ³⁵	X ray			5	
<i>sbd</i> ⁴³	X ray			5	
<i>sbd</i> ⁴⁷	X ray			5	<i>T(2;3)41;88F-89A;89B4-4</i>
<i>sbd</i> ^l	X ray	Lewis		4	associated with <i>T(2;3)Me</i>

^α 1 = CP627; 2 = Beaton, Kiss, Fristrom, and Fristrom, 1988, Genetics 120: 453-64; 3 = Dobzhansky, 1930, Z. Indukt. Abstamm. Vererbungsbl. 4: 4237-57 (fig.); 4 = Lewis, 1949, DIS 23: 92; 5 = Spillmann and Nöthiger, 1978, DIS 53: 164-65; 6 = Stern, 1929, Biol. Zentralbl. 49: 261-90.

cytology: Placed in region 89B4-5 by Lewis (1951, Cold Spring Harbor Symp. Quant. Biol. 16: 159-74). This probably corresponds to 89B9-10 on Bridges's revised map.

other information: Pseudoallelic to *Sb* and lies to the left

of it (Lewis, 1951).

***sbd*²**

phenotype: Most bristles about three-fourths normal length although some (*i.e.*, posterior postalar) are shorter. Less extreme than *sbd*. *sbd*²/*Sb* has shorter bristles than homozygous *sbd*² or *Sb*/. *sbd*² *Sb*/+ + has wild-type bristles (Lewis, 1951, Cold Spring Harbor Symp. Quant. Biol. 16: 159-74). RK1.

***sbd*¹: stubbloid-lethal**

phenotype: *sbd*¹/*sbd* is *sbd*; *sbd*¹/*Sb* is lethal. RK2A.

cytology: Associated with *T(2;3)Me = T(2;3)48C1-2; 59D2-3;80-81 + In(3L)63C;72E1-2 + In(3LR)69E;91C + In(3R)89B;97D*.

***sbl*: smellblind (J.C. Hall)**

location: 1-54.7 (based on mapping lethality; Lilly and Carlson, 1990).

synonym: *olfD*.

references: Rodrigues and Siddiqi, 1978, Proc. Ind. Acad. Sci. 87B: 147-60.

Aceves-Piña and Quinn, 1979, Science 206: 93-96.

Rodrigues, 1980, Development and Neurobiology of *Drosophila* (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, NY, pp. 371.

Tompkins, Hall, and Hall, 1980, J. Insect Physiol. 26: 689-97.

Tompkins, Gross, Hall, Gailey, and Siegel, 1982, Behav. Genet. 12: 295-307.

Tompkins, Siegel, Gailey, and Hall, 1983, Behav. Genet. 13: 565-78.

Technau, 1984, J. Neurogenet. 1: 113-126.

Gailey, Lacaillade, and Hall, 1986, Behav. Genet. 16: 375-405.

Markow, 1987, Proc. Nat. Acad. Sci. USA 84: 6200-04.

Monte et al., 1989, Behav. Genet. 19: 267-83;

Lilly and Carlson, 1990, Genetics, 124: 293-302.

phenotype: Responds poorly, or apparently not at all, to a variety of volatile compounds; hence, may be generally olfaction defective, as opposed to responding poorly to only certain classes of synthetic or natural compounds; *sbl*¹ was isolated with respect to abnormal shock-avoidance learning tests of adults, which involve odor cues (Aceves-Piña and Quinn, 1979); *sbl*² was isolated with regard to odor response tests *per se* (Rodrigues and Siddiqi, 1978); *sbl*¹ and *sbl*² defective in larval olfaction (Aceves-Piña and Quinn, 1979; Monte et al., 1989) and fail to complement in this regard (Lilly and Carlson, 1990); the latter report showed *sbl*² to respond poorly to 3 separate odorants; both of these mutants also behave subnormally in tests of larval contact chemosensory responses, but each is normal in visually mediated larval behavior (Lilly and Carlson, 1990); *sbl*¹ and *sbl*² are temperature-sensitive near-lethals in 29° rearing tests, and they fail to complement in this regard (Lilly and Carlson, 1990); a post eclosion shift from 25° to the higher temperature does not appear to affect viability, but adults under these conditions have not been tested behaviorally--recall that *sbl*² flies are defective in odor responses (Rodrigues and Siddiqi, 1978; Gailey et al., 1986), and *sbl*¹ adults are as well (Aceves-Piña and Quinn, 1979; Tompkins et al., 1980; J. Carlson, unpublished--the latter determination used testing procedures as reported by Woodward, Huang, Sun, Helfand,

and Carlson, 1989, Genetics 123: 315-326). Other alleles are unconditionally lethal. In courtship experiments, *sbl*¹ (Tompkins et al., 1980) or *sbl*² (Gailey et al., 1986) males make little or no responses to pheromonal materials extracted from adult females; the former mutant, as a male, also performs subnormally in terms of quantitative measurements of overall courtship interactions with females (Tompkins et al., 1980) as well as by specification of individual elements of such sexual behavior (Markow, 1987). seemingly as a consequence, *sbl*¹ males exhibit mediocre mating success (Tompkins et al., 1980; Markow, 1987); the latter is observed when the mutant males are with females only (Tompkins et al., 1982) or are also in competition with wild-type males (Markow, 1987). *sbl*² males are rather wild-type-like, with respect to short-term monitoring of their behavior in presence of females and also in terms of mating success (Gailey et al., 1986); a *sbl*² male, however, makes almost no odor-mediated responses to a courtship object in his presence, as stimulated by a female placed nearby, whereas wild-type males are rather vigorously stimulated to court under these circumstances (Gailey et al., 1986). Females homozygous for *sbl*¹ (Tompkins et al., 1982; Markow, 1987) or *sbl*² (Gailey et al., 1986) exhibit relatively poor receptivity to male courtship/mating activities; this could be explained by the fact that they tend not to slow their movements in response to the courting male, whereas wild-type females do (Tompkins et al., 1982; Gailey et al., 1986). Courtship-experience-dependent after-effects, which appear to be in part mediated olfactorily, are subnormal or absent in tests of *sbl*¹ males with fertilized females (Tompkins et al., 1983). In experiments involving counts of post-eclosion mushroom-body fiber (axon) numbers in young imagoes, deantennated vs. intact *sbl*¹ flies were found to exhibit same degree of reductions in fiber numbers (Technau, 1984)--suggesting that deantennation, which leads to such decreases in wild-type adults, effects these anatomical changes because of sensory deprivation, instead of surgery-induced injury *per se*.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>sbl</i>¹		Sziber	<i>sbl</i> ^{PSS42}	1, 3, 7	
<i>sbl</i>²		Rodrigues	<i>olfD</i>	3, 4, 5	
<i>sbl</i>³	P		<i>l(1)D23</i>	2	unconditionally lethal
<i>sbl</i>⁴	spont	Schalet	<i>l(1)20-137</i>	6	unconditionally lethal
<i>sbl</i>⁵	EMS	Rodrigues	<i>sbl</i> ^{L1}		unconditionally lethal
<i>sbl</i>⁶	EMS	Rodrigues	<i>sbl</i> ^{L2}		unconditionally lethal

^α 1 = Aceves-Piña and Quinn, 1979, Science 206: 93-96; 2 = Eeken, Sobels, Hyland, and Schalet, 1985, Mutat. Res. 150: 261-75; 3 = Lilly and Carlson, 1990, Genetics, in press; 4 = Rodrigues and Siddiqi, 1978, Proc. Ind. Acad. Sci. 87B: 147-60; 5 = Rodrigues, 1980, Development and Neurobiology of *Drosophila* (Siddiqi, Babu, Hall, and Hall, eds.). Plenum, NY, pp. 371. 6 = Schalet, 1986, Mutat. Res. 163: 115-44; 7 = Tompkins, Gross, Hall, Gailey, and Siegel, 1982, Behav. Genet. 12: 295-307.

cytology: Placed in 14B13-15A9 based on its being covered by *Dp(1;2)r⁺75c = Dp(1;2)14B3;15A9;35D-E* (Carlson).

other information: Regarding lethality associated with each of the six mutant alleles (at least under high-temperature conditions), *inter se* crosses show all combinations not to complement at 25°-29°, meaning that *sbl*³ or *sbl*⁴, over *sbl*¹ or *sbl*², are lethal even at 25° (Lilly

and Carlson, 1990).

sbr: small bristles

location: 1-32.6 [between 32.53 and 32.67 (Zhimulev *et al.*, 1981, 1982)].

references: Zhimulev, Belyaeva, Pokholkova, Kotchneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1981, DIS 56: 192-96; 1982, DIS 58: 210-14.

Zhimulev, Pokholkova, Bgatov, Umbetova, Solovjeva, and Belyaeva, 1987, DIS 66: 194-97.

Zhimulev, Pokholkova, Bgatov, Umbertova, Solovjeva, Khudyakov, and Belyaeva, 1987, Biol. Zentralbl. 106: 699-720.

phenotype: Most alleles homozygous and hemizygous lethal; *sbr*¹ viable but displays reduced viability in hemizygous females. Bristles small, with one or more missing, especially scutellars and especially in hemizygous females (Zhimulev and Ilyina, 1980, DIS 55: 146). *sbr*¹⁶ semilethal and male sterile; females sterile in heteroallelic combination with other lethal alleles. *sbr*⁹ is a temperature-sensitive lethal; semilethal with small body at 25°, *sbr*¹/*sbr*⁹ viable and normal at 30°.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>sbr</i> ¹	spont	Curry		2	viable allele, complements <i>sbr</i> ⁹
<i>*sbr</i> ²	CB.3007	Fahmy, 1954	<i>sbt</i>	3	escapers
<i>sbr</i> ³	X ray	Kotchneva	<i>l(1)K2</i>	7	
<i>sbr</i> ⁴	X ray	Kotchneva	<i>l(1)K3</i>	7	
<i>sbr</i> ⁵	X ray	Kotchneva	<i>l(1)K4</i>	7	
<i>sbr</i> ⁶	X ray	Kotchneva	<i>l(1)K5</i>	7	
<i>sbr</i> ⁷	X ray	Kotchneva	<i>l(1)K6</i>	7	
<i>sbr</i> ⁸	X ray	Kotchneva	<i>l(1)K7</i>	7	
<i>sbr</i> ⁹	X ray	Kotchneva	<i>l(1)K11</i>	7	complements <i>sbr</i> ¹
<i>sbr</i> ¹⁰		Arking, 1975	<i>l(1)ts403</i>	1, 7	temperature-sensitive lethal
<i>sbr</i> ¹¹	HMS	Kramers	<i>l(1)HM424</i>	5	
<i>sbr</i> ¹²	dysgenesis ^β	Eeken	<i>l(1)24/45A</i>	7	
<i>sbr</i> ¹³	EMS		<i>l(1)v1</i>	4	
<i>sbr</i> ¹⁴	EMS		<i>l(1)v14</i>	4	
<i>sbr</i> ¹⁵	EMS		<i>l(1)v19</i>	4	
<i>sbr</i> ¹⁶	EMS		<i>l(1)v107</i>	4	
<i>sbr</i> ¹⁷	EMS		<i>msv7</i>	4	11% survival; male sterile
<i>sbr</i> ¹⁸	spont	Schalet	<i>l(1)7-27</i>	6	
<i>sbr</i> ¹⁹	spont	Schalet	<i>l(1)18-14-1</i>	6	

^α 1 = Arking, 1975, Genetics 80: 519-37; 2 = CP627; 3 = Fahmy, 1959, DIS 33: 90; 4 = Geer, Lischwe, and Murphy, 1983, J. Zool. 225: 107-18; 5 = Kramers, Schalet, Paradi, and Huiser-Hoogteyling, 1983, Mutat. Res. 107: 187-201; 6 = Schalet, 1986, Mutat., Res. 163: 115-44; 7 = Zhimulev, Belyaeva, Pokholkova, Kotchneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1981, DIS 56: 192-96.

^β Induced by male recombination factor, *MR*^{h12} in *y mei9*^a *mei41*^{D5}.

cytology: Placed in 9F5-11 based on its inclusion in the region of overlap of *Df(1)v-LA = Df(1)9F5-6;10A1-2* and *Df(1)ras59 = Df(1)9E1;9F10-11*.

***sbs: stubs**

location: 1-0.9.

origin: Induced by ethyl methanesulfonate.

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 90.

phenotype: Wing abnormalities vary from extreme reduction in size to partial incision of margin with L2 and L3 closer together. Eyes small and slightly rough. Male viable and fertile; female sterile. RK2.

**sbt*: see *sbr*²

***sby: small body**

location: 1-60.8.

origin: γ ray induced.

discoverer: Fahmy, 1958.

synonym: *sby-61*.

references: 1964, DIS 39: 58.

phenotype: Extremely small, lightly pigmented fly. Viability and fertility reduced. RK3.

cytology: Placed in salivary region 18A4 to 18B8 on the basis of its inclusion within deficiency resulting from recombining left end of *In(1)y*⁴ = *In(1)1A8-B1;18A3-4* with right end of *In(1)sc*⁹ = *In(1)1B2-3;18B8-9* (Norton and Valencia, 1965, DIS 40: 40).

sc: see *ASC*

Sc: Scotched eye

location: 1-4.5 (about 4 or 5).

origin: X ray induced.

references: Muller, 1946, DIS 20: 67.

phenotype: Ommatidia disarranged near posterior margin of eye. Resembles *spa*^{Cat}. Good viability and fertility in heterozygous female. Male lethal. RK2.

Sc²

origin: X ray induced.

discoverer: Craymer, 1971.

references: 1980, DIS 55: 198.

phenotype: *Sc*^{2/+} has glazed eyes, which are slightly reduced in size. Abdomen of female fails to expand with eggs; female seems to have impaired fertility. Hemizygous lethal. RK2.

other information: Allelism inferred from location (1-3 or 4) and phenotype.

Sc^{2rv}

origin: Spontaneous.

discoverer: Craymer, 1977.

references: 1980, DIS 55: 199.

phenotype: Revertant of dominant phenotype of *Sc*². Still recessive lethal.

sc: see *Scp*

sc-Dp: see *Dp(1,f)100*

sc-Inh-3: see *Su(sc)*

sca: scabrous

location: 2-66.7.

origin: Spontaneous.

discoverer: Ives, 34j2.

references: 1935, DIS 4: 10.

phenotype: Eyes large and rough. Ocellar bristles 85% absent at 25° and 10% absent at 18°. Postverticals occasionally missing. Bristle effect more extreme in male at 21° and in female at 28°. Most bristles subject to twinning. May be extra rows of acrostichal hairs. Mosaic experiments suggest that *sca* affects the spacing of R8 cells in the eye (Baker, Mlodzik, and Rubin, 1990, Science 250: 1370-77). RK1.

alleles:

allele	origin	discoverer	ref ^α	comments
sca ¹	spont	Ives, 34j2	2	
sca ²	spont	Ives, 65131	3, 4	no reduction in bristle number
sca ^{dc}	spont		1, 5	adds 3.5 bristles (scutellum); sternoplurals and third coxals reduced

^α 1 = Frankham and Nurthen, 1980, DIS 55: 204; 2 = Ives, 1935, DIS 4: 10; 3 = Ives, 1967, DIS 42: 39; 4 = Ives, 1972, DIS 48: 16; 5 = MacBean, McKenzie, and Parsons, 1971, Theor. Appl. Genet. 41: 227-35.

cytology: Placed in region 49C2-D4 *Df(2R)vg*^I = *Df(2R)49C2-D1;50A2-3* but not in *Df(2R)vg*^B = *Df(2R)49D3-4;50A2-3* (Morgan, Bridges, and Schultz, 1938, Year Book – Carnegie Inst. Washington 37: 205).

molecular biology: *sca* cloned, the transcription unit identified, genomic DNA sequenced and putative amino acid sequence determined (Baker *et al*, 1990). The predicted protein of 774 amino acids appears to encode a secreted protein with similarity to the β and γ chains of fibrinogens in the carboxyl-terminal portion; also, the *sca* protein shows 39% identity to the protein encoded by a gene in the human adrenal medulla.

scab: see *scb*

scal: *scabrous like*

location: 2-11.7.

origin: Spontaneous.

references: Barker and Hollingdale, 1970, DIS 45: 39.

phenotype: Eyes rough, slightly bulging; increased numbers of abdominal and scutellar bristles; wings broad and curved with irregular L2 and posterior crossvein. Semilethal; females nearly sterile.

scalloped: see *sd*

***Scar:** *Scarred*

location: 2- or 3- (rearrangement).

origin: X ray induced.

discoverer: Yu, 48h.

references: 1949, DIS 23: 65.

phenotype: Eyes elliptical with indented, glassy posterior margin. Wings spread at 45° from body axis. Enhanced at 28°. Homozygous lethal. RK1A.

cytology: Associated with *T(2;3)Scar = T(2;3)27E;95A + In(3)91F;96A*.

scarlet: see *st*

scarp: see *scrp*

Scarred: see *Scar*

scb: *scab* (C. Nüsslein-Volhard)

location: 2-73.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

phenotype: Embryonic lethal. Embryos have small mid-dorsal hole with necrotic rim.

alleles: Four alleles *scb*¹ = IID; *scb*² = IIG; other two discarded.

scd: *sex comb distal* (J.A. Kennison)

location: 1-30.6.

origin: Spontaneous.

discoverer: Kennison, 1983.

phenotype: Exhibits 1-2 sex comb teeth on the second tarsal segment of the prothoracic leg in hemizygous males. Homozygous females viable and fertile. Interacts with *Sco* in that *scd;Sco*+/+ males have reduced viability and lack many bristles, including many of the sex comb teeth on the first tarsal segment of the prothoracic leg. Resembles description of rare *Sco* homozygotes.

Sce: *Sex combs extra*

location: 3-92.

origin: Induced by ethyl nitrosourea.

references: Breen and Duncan, 1986, Dev. Biol. 118: 442-56.

phenotype: A dominant enhancer of *Mcp* in the *BXC*. *Sce/Sce* zygotes from *Sce*+/+ mothers die as first instar larvae with weak posteriorly directed transformations, *i.e.*, A7 displays some A8 characteristics. Heterozygous offspring normal. *Sce/Sce* embryos from clones of homozygous oocytes produced by pole-cell transplantation display extreme posteriorly directed segmental transformation. Ventral setal belts of all abdominal and thoracic segments transformed toward A8; head involution blocked; abdominal type denticle belts also found anterior to T1 in the presumptive labial and maxillary segments; in addition an extensive belt of abdominal denticles of unknown derivation forms on the anterodorsal surface of the embryo. Keilin's organs and ventral pits suppressed in thoracic segments; wart-like sensilla normally found in A8 formed anteriorly as far as A2. Tracheal branches in A1-7 resemble those normally found more posteriorly. *Sce*+/+ offspring from homozygous germ line clones in the mothers may survive to adulthood or die as pharate adults; they show patches of tissue transformed toward A8.

scg: *scrambled-egg*

location: 3-{41}.

references: Berg and Spradling.

phenotype: Germ cell differentiation affected.

cytology: Located in 70C.

***sch:** *slender chaetae*

location: 1-21.1.

origin: Induced by D-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3026).

discoverer: Fahmy, 1955.

references: 1959, DIS 33: 90.

phenotype: Bristles thin and slightly shortened. Eyes slightly smaller and brownish. Body small. RK2.

schlaff: see *slf*

schmal: see *sim*

Scm: *Sex combs on midleg*

location: 3-48.5.

phenotype: Presumably *Scm*+/+ males form sex combs on the mesothoracic legs; does not appear to have been explicitly described. Homozygous embryos show posteriorly directed transformations; A1 resembles A2 and A2-7 transformed into more posterior segments, head and thorax normal. *Pcl Scm* double homozygotes exhibit

transformation of all segments toward A8; Keilin's organs retained in thoracic segments; head involution does not occur; prominent abdominal denticle band seen on dorsal surface of head. Homozygous embryos derived from transplanted homozygous maternal germ-cell precursors display A8 morphology in all thoracic and abdominal segments; head seems to resemble that of *Pcl Scm* double homozygotes produced by heterozygous mothers; heterozygous adults from homozygous oocytes show patchy transformations of A4 to A5 and of A6 and A7 to A8; similar transformation infrequently seen in heterozygous offspring of heterozygous mothers.

alleles:

allele	origin	synonym	ref ^α	comments
Scm 1	EMS	<i>E502</i>	2	
Scm 2	EMS	<i>ET16</i>	2	
Scm 3	EMS	<i>ET19</i>	2	
Scm 4	X ray	<i>XF24</i>	2	
Scm 5		<i>KM23</i>		
Scm D1	X ray		1	enhancer of <i>Ubx Cbx</i>
Scm D2	X ray		1	enhancer of <i>Mcp</i>
Scm K1	EMS		3	β

^α 1 = Breen and Duncan, 1986, Dev. Biol. 118: 442-56; 2 = Jürgens, 1985, Nature 316: 153-55; 3 = Kennison and Tamkun, 1988, Proc. Nat. Acad. Sci. USA 85: 8136-40.
^β Fuller description follows.

cytology: Placed in 85E1-10 based on its inclusion in *Df(3R)by62 = Df(3R)85D11-14;85F16* and *Df(3R)GB104 = Df(3R)85D11-13;85E10*, but not *Df(3R)by416 = Df(3R)85D10-12;85E1-3*.

Scm K1

phenotype: Strong dominant enhancer of the extra sex combs phenotype of *Pc* heterozygotes. Allelism to *Scm* inferred from phenotype and location.

cytology: Associated with *In(3R)85F;89AB*.

***Scn: Scutenick**

location: 4- [included in *Df(4)M*].

discoverer: Padoa, 1931.

references: Bridges, 1935, Biol. Zh. (Moscow) 4: 401-20. Padoa, 1938, Monit. Zool. Ital. 49: 279-84.

phenotype: Scutellum shortened, has nick at posterior edge; scutellar bristles missing. Ocelli reduced, has disturbed hairs and bristles. One or both eyes often small or absent. All characters overlap wild type. Eye effect is strongest at 19° but other effects weaker. Scutellum effect best at 28° but eyes normal. Homozygous lethal. Claimed by Robertson (1980, DIS 55: 130) to have been reisolated from a selection line for sternopleural-bristle number. RK2.

cytology: Placed in salivary chromosome region 101E through 102B16 on the basis of its inclusion in *Df(4)M = Df(4)101E-F;102B6-17*.

Sco: Scutoid

location: 2-51.0.

origin: X ray induced.

discoverer: Krivshenko, 56115.

references: 1959, DIS 33: 96.

1960, DIS 34: 55.

Ashburner, Tsubota, and Wooduff, 1982, Genetics 102: 401-20.

Ashburner and Harington, 1984, Chromosoma 89: 329-37.

phenotype: *Sco*+ flies exhibit the loss of 10-15 bristles, scutellars, notopleurals, upper humerals and anterior postalars being most frequently affected; removes both trichogen and tormogen cell derivatives. Homozygotes nearly lethal; rare homozygous or hemizygous escapers have only eight or so bristles per fly; eyes small and very rough. Escapers short lived and sterile. RK1A.

cytology: Associated with a pair of reciprocal transpositions that are tentatively postulated to result from a pair of inversions, one included within the other; i.e., *In(2L)34A4-5;35C5-D1 + In(2L)35B3-4;35C1-2*. Based on reversion analysis *Sco* postulated to result from the juxtaposition of *noc*⁺ and *sna*⁺ at the 34A5|35D1 junction.

other information: Numerous *Sco* revertants recovered; some are simple deficiencies and are listed under *Df(2L)Sco*, whereas others are rearrangements with a deletion for *Sco* associated with the breakpoint in 35D1-2 and are designated for example as *T(2;3)Sco*⁻; revertants without apparent loss of material are listed below. Breakpoints of the rearrangements indicated are superimposed on the four breakpoints of the original *Sco* chromosome; the rearrangements listed have in common a break in 35D1-2 at the proximal end of the *Sco* rearrangement.

allele	origin	comments
Sco rv1	X ray	<i>In(2LR)35D1-2;44C3-5</i>
Sco rv2	X ray	<i>In(2L)35D1-2;36D3</i>
Sco rv5	X ray	<i>In(2L)35D1-2;38A3-8</i>
Sco rv8	X ray	<i>In(2L)34C1-2;35D1-2</i>
Sco rv9	X ray	<i>In(2LR)35D1-2;41</i>
Sco rv11	X ray	<i>In(2L)24C3-9;35D1-2</i>
Sco rv12	X ray	<i>Tp(2;2)34A8-B1;35A4-B1;35D1-2</i>
Sco rv13	X ray	<i>T(2;3)35D1;71B;81</i>
Sco rv15	EMS	
Sco rv17	X ray	<i>In(2L)25D3-7;35D1-2</i>
Sco rv21	X ray	<i>In(2L)35D1-2;36E1-2</i>
Sco rv23	X ray	<i>Tp(2;1)20F;34F1-2;35B1-D1</i>
Sco rv24	X ray	<i>In(2L)34B1-2;35D1-2</i>
Sco rv26	X ray	<i>In(2L)35D1-2;40</i>
Sco rv27	X ray	

molecular biology: Distal breakpoint of the *Sco* rearrangement occurs within the *noc* gene between components A and B; coordinate -107.8 from *noc* is brought to within 16 kb of *sna* by the realignment responsible for the *Sco* phenotype; the region from -107.8 to -102.3 is duplicated on opposite sides of the left-most breakpoint of the *Sco* rearrangement (McGill, Chia, Karp, and Ashburner, 1988, Genetics 119: 647-61) (coordinate 0 an *Eco*RI restriction site 1321 bp to the left of the start of transcription of the larval *Adh* transcript; positive values to the right).

Scoop: see *Scp*

scooped: see *scp*

scooped thickvein: see *sct*

Scotched eye: see *Sc*

scp: scooped

location: 1-19.3.

discoverer: Muller, 1926.

phenotype: Wings turn up slightly; classification fairly reliable. RK2.

cytology: Placed between 6A3-4 and 6F10-11 (Demerec, Kaufmann, Fano, Sutton, and Sansome, 1942, Year Book

– Carnegie Inst. Washington 41: 191).

***Scp: Scoop**

location: 3- (not located).

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Reddi.

synonym: *Sc* (preoccupied).

references: 1963, DIS 37: 53.

phenotype: Wing size reduced; proximal one-third of wing compressed laterally; distal two-thirds spoonlike. Three furrows run length of wing, and surface is wrinkled. Abdomen cylindrical and untapered posteriorly. Pigmented abdominal bands darkened. Excellent viability and fertility. RK3.

***scr: scruff**

location: 1-22.0.

origin: Spontaneous.

discoverer: Neel, 41b22.

references: 1942, DIS 16: 52.

1942, Genetics 27: 532.

phenotype: Hairs and bristles missing or doubled, and deranged. Eyes small and rough. Scutellum more convex than wild type. Wing margins, especially posterior, often incised. Wings occasionally blistered. All characters variable; a few flies appear normal. RK3.

cytology: Salivary chromosomes appear normal.

Scr: see **ANTC**

scra: scraps (T. Schüpbach)

location: 2 - {57}.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus.

phenotype: Maternal-effect lethal. Embryos from homozygous females begin to cellularize at the blastoderm stage, but cellularization is often not completed; further development very abnormal; at final differentiation embryos form only fragmented pieces of cuticle.

alleles: *scra*¹ - *scra*⁶ recovered as *HP*, *PB*, *PE*, *PQ*, *RS*, and *RV* respectively.

cytology: Placed in 42C1-43F8, since uncovered by *Df(2R)pk78s = Df(2R)42C1-7;43F5-8*.

other information: *scra*¹, *scra*⁴, *scra*⁵ are homozygous viable but lethal over the deficiency.

scrambled-egg: see **scg**

scratched eyes: see **sey**

screw: see **scw**

scrp: scarp

location: 2-74 (to the left of *c*; not an allele of *L*).

origin: Spontaneous.

discoverer: Hansen and Gardner, 1960.

references: 1962, DIS 36: 38.

1962, Genetics 47: 587-98 (fig.).

phenotype: Ventral one-third of eye flattened and separated from dorsal two-thirds by a furrow. Penetrance 80% at 30°; at 25°, eyes are wild type. Temperature-effective period from forty-second to sixty-eighth hour of development. RK3.

scruff: see **scr**

***sct: scooped thickvein**

location: 1-16.0.

origin: Induced by methyl methanesulfonate (CB. 1540).

discoverer: Fahmy, 1956.

references: 1960, DIS 34: 49.

phenotype: Wings short and scooped; inner margin frequently incised in several places; veins thickened. Eyes darker and slightly altered in shape. Abdominal tergites slightly ridged. Male sterile; viability about 40% normal. RK2?

scute: see **sc** under **ASC**

scute Inhibitor on chromosome 3: see **Su(sc)**

Scutenick: see **Scn**

scutex: see *sc*¹⁵

Scutoid: see **Sco**

scw: screw

location: 2-53.

origin: Induced by ethyl methanesulfonate.

synonym: *l(2)IG76*.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Embryonic lethal; partially ventralized; cephalic furrow shifted dorsally with defects in germ band extension. Ventral denticles extended laterally; spiracles and two to three terminal segments retracted into embryo (Arora and Nüsslein-Volhard).

alleles: Five ethyl-methanesulfonate-induced alleles.

allele	synonym	comments
scw ¹	<i>C13</i>	
scw ²	<i>IG</i>	weak allele
scw ³	<i>N5</i>	
scw ⁴	<i>O5</i>	
scw ⁴	<i>S12</i>	

cytology: Placed in 37F5-38C1 based on inclusion in *Df(2L)TW50 = Df(2L)37F5-38A1;38B2-C1* but not *Df(2L)E55 = Df(2L)37D2-E1;37F5-38A1*.

Scx: see **Antp**^{Scx} under **ANTC**

sd: scalloped (S. D. Campbell and A. Chovnick)

location: 1-51.5.

phenotype: Wing margins scalloped and veins thickened.

Eyes slightly roughened. Does not overlap wild type.

Additional defects noted are uplifting of posterior scutellar bristles, haltere diminution, and ectopic bristles on the wing blade. RK1.

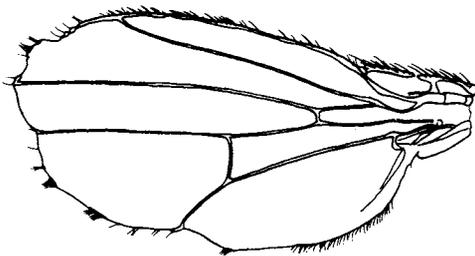
allele	origin	discoverer	synonym	ref ^α	comments
sd ¹	X ray	Grüneberg, 28j20		4,5	
sd ²	X ray	Panshin, 33g7		11	
sd ³	<i>P</i>	McCarron	<i>sd</i> ^{2A3}	2	
sd ⁴	<i>P</i>	McCarron	<i>sd</i> ^{11D}	2	
sd ⁵	<i>P</i>	McCarron	<i>sd</i> ⁹³	2	≥10-kb deletion
sd ⁶	<i>P</i>	McCarron	<i>sd</i> ¹⁸⁹	2	deletion
sd ⁷	<i>P</i>	McCarron	<i>sd</i> ^{299A}	2	
sd ⁸	<i>P</i>	McCarron	<i>sd</i> ^{299D}	2	deletion
sd ⁹	EMS	Katzen	<i>sd</i> ^{3L}	2	larval lethal
sd ¹⁰	EMS	Katzen	<i>sd</i> ^{11L}	2	pupal lethal
sd ¹¹	EMS	Katzen	<i>sd</i> ^{31H}	2	pupal lethal
sd ¹²	EMS	Katzen	<i>sd</i> ^{47M}	2	larval lethal

allele	origin	discoverer	synonym	ref ^α	comments
<i>sd</i> ¹³	EMS	Katzen	<i>sd</i> ^{68L}	2	pupal lethal
<i>sd</i> ¹⁴	P	Moore	<i>sd</i> ^{JM}	2	
<i>sd</i> ¹⁵	P	Orr-Weaver	<i>sd</i> ^{Ag2014}	2	
<i>*sd</i> ³⁵		Hollander, 1935		6	
<i>*sd</i> ^{56j}	X ray	Clark, 56j		1	
<i>sd</i> ^{58d}	γ ray	Ives, 58d14		7	
<i>sd</i> ^[ry+]	P	Daniels	<i>sd</i> ^[ry+2216]	3	
<i>*sd</i> ^s	X ray	Muller		10	
<i>sd</i> ^{sp}	X ray	R. M. Valencia, 1959		14,15	<i>ln(1)scS1</i> Lsc8R+dl-49
<i>sd</i> ^{ts}	EMS			12,13	
<i>*sd</i> ^{UCI}				8,16	
<i>sd</i> ^[w+]	P	Hazelrigg	<i>sd</i> ^{[w,ryE]3}	9	

^α 1 = Andrew, 1959, DIS 33: 82; 2 = Campbell, Duttaroy, Katzen, and Chovnick, unpublished; 3 = Daniels, McCarron, Love, and Chovnick, 1986, Genetics 109: 95-117; 4 = Grüneberg, 1929, Biol. Zentralbl. 49: 680-94; 5 = Grüneberg, 1934, DIS 2: 9; 6 = Hollander, 1935, DIS 8: 8; 7 = Ives, 1961, DIS 35: 46; 8 = James and Bryant, 1981, Dev. Biol. 85: 39-54; 9 = Levis, Hazelrigg, and Rubin, 1985, EMBO J. 4: 3489-99; 10 = Muller, 1946, DIS 20: 67-78; 11 = Panshin, 1935, DIS 3: 28; 12 = Simpson and Schneiderman, 1974, DIS 51: 23 (fig); 13 = Simpson and Schneiderman, 1975, Wilhelm Roux's Arch. Dev. Biol. 178: 247-75; 14 = Valencia, 1959, DIS 33: 99; 15 = Valencia, 1965, DIS 40: 37; 16 = Vyse and James, 1972, DIS 49: 39.

cytology: Placed in 13F (Daniels, McCarron, Love, and Chovnick, 1986, Genetics 109: 95-117) by *in situ* hybridization; in 14A by Lefevre [Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, p. 59].

molecular biology: A *P*[*ry+*] transposition into 13F associated with *sd*^[*ry+*] (Daniels *et al.*, 1985) was the starting point of a 41 kb chromosomal walk. Molecular lesions associated with viable and lethal *sd* alleles were localized within a 15 kb region. Structurally related and developmentally regulated transcripts were detected in the genomic region where several *sd* lethal alleles were localized. 32 cDNA clones have been isolated from the region. Several are characterized that map across the 15 kb interval where *sd* lesions map. The strong possibility that these transcripts are products of the *sd* gene is being investigated. If true, the *sd* gene includes genomic sequences extending over at least 13 kb of the chromosomal walk described, and it appears to be subject to alternative splicing (Campbell, Duttaroy, Katzen and Chovnick, unpublished).



sd: scalloped

From Edith M. Wallace, unpublished.

sd: see *sprd*

***sd²**

phenotype: More extreme than *sd*. Wings small and scalloped. Like *vg* at high temperatures. Crossing over inhibited. RK2A?

cytology: Associated with *ln(1)sd*²; breakpoints unknown. No visible X-chromosome rearrangement in vicinity of 13F (Campbell, Duttaroy, Katzen, and Chovnick).

***sd^{56j}**

phenotype: More extreme than *sd*. Expression enhanced by high temperature. Visible in prepupal wing buds. Interacts with *Bx* and *bi*. RK1.

cytology: No gross chromosomal abnormality.

sd^{58d}

phenotype: Wings reduced to vestiges, like *vg*. Halteres and bristles also like *vg*. *sd*^{58d}/*sd* has strap-shaped wing. Temperature sensitive; effects of temperature pulses at different developmental stages suggest that wing areas eliminated in a specific order (Simpson, Lawrence, and Maschat, 1981, Dev. Biol. 84: 206-11). RK2A.

cytology: Associated with *ln(1)sd*^{58d} = *ln(1)11F;13F* (Campbell, Duttaroy, Katzen, and Chovnick).

sd^[ry+]

phenotype: Extreme allele of *sd* associated with the insertion of a *P* element carrying *ry*⁺ at 13F. In hemizygous males, wings reduced to mere vestiges similar to *vg*; in homozygous females the wings are narrow and strap-like. One or both scutellar bristles are sometimes truncated, and halteres appear somewhat reduced in males. Severity of wing defect may vary with temperature. Under dysgenic conditions, excision of the *P* element leads to the loss of *ry*⁺ and amelioration of the *sd* phenotype (*i.e.*, wild type or nibbled wings); the molecular lesions of a number of such derivatives have been characterized (Daniels, McCarron, Love, and Chovnick, 1985, Genetics 109: 95-117). One derivative was used as a "tag" for cloning the *sd* region (Campbell *et al.*).

***sd^s: scalloped-sterile**

phenotype: Wings divergent and slightly nicked. Male sterile. RK2.

other information: Allelism inferred from position and phenotype. No evidence of chromosome rearrangement.

sd^{sp}: scalloped-spatula

phenotype: Wings cut at tips and along both margins. *sd*^{sp} +/+ *Bx*^r give slight nicking of wings. RK1A.

cytology: No gross rearrangement in addition to *ln(1)sc*^{SIL_{sc}8R}+*dl-49* but possibly a local disturbance in pairing.

sd^{ts}

phenotype: Wings vestigial-like at 29°; also capitellum of haltere extremely reduced; some crippling of legs. At 22° wings have parts of wing blade or wing margin or both missing; halteres normal. Viability and fertility good at 22°. Increases incidence of pattern duplications and deficiencies in *l(1)ts504*.

***sd^{UCI}**

phenotype: Extreme vestigial-like wing (as in *sd*^{58d}) with erect postscutellar bristles. Halteres greatly reduced in size. In some flies, outgrowths of tissue found on one side of the metanotum; in extreme cases, these outgrowths can be recognized as mirror-image pattern dupli-

cations of the notum and ventral hinge and reduction of the wing blade. Pupation delayed until about 168 hr after oviposition, with four days being spent as third-instar larvae. Average size of mature wing disk about 56% normal; reduction in size and pattern duplication attributable to extensive cell death in the wing imaginal disk. Wing margins of *sd^{UC1}/Basc* females frequently incised; such females also produce significant numbers of patroclinous males. (Vyse and James, 1972, DIS 49: 39; James and Bryant, 1981, Dev. Biol. 85: 39-54).

***Sd*: Segregation distorter**

location: 2-54 (between *hk* and *pr*).

origin: Naturally occurring abnormality found at low levels in many natural populations.

discoverer: Hiraizumi.

references: Sandler, Hiraizumi, and Sandler, 1959, Genetics 44: 233-50.

Hartl and Hiraizumi, 1976, Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 615-66.

Ganetzky, 1977, Genetics 86: 321-55.

Brittnacher and Ganetzky, 1983, Genetics 103: 659-73.

1984, Genetics 107: 423-34.

phenotype: *Sd*-bearing second chromosomes are referred to as *SD*; they carry, in addition to *Sd*, other components of the segregation distortion system; they may carry inversions and recessive lethal alleles as well. The constitutions of various *SD* chromosomes are tabulated below. Other components of the segregation distortion system are *E(SD)* (Enhancer of *SD*) at the base of *2L*; and *Rsp* (Responder) at the base of *2R*; *St(SD)* (Stabilizer of *SD*) located more distally in *2R* appears to comprise several weak enhancers of distortion (Miklos, 1972, Genetics 70: 405-18). All naturally occurring *SD* chromosomes carry *Sd* and *E(SD)* but lack *Rsp* [sometimes said to carry *Rspⁱ* (Responder insensitive)]; most normal chromosomes carry a *Rsp* element. *SD/+* males transmit *SD*-bearing, to the virtual exclusion of *+*-bearing, homologues; as many as 99% of the functional sperm may carry *SD* (defined as *k* = .99). The sex ratio of the minority class of offspring, in diverse crosses, is skewed in proportion to *k* in such a way as to indicate that within this class the probability of recovery of *XY* < *X* < *Y*-bearing < nullo-*X*, nullo-*Y* sperm (Denell, Judd, and Richardson, 1969, Genetics 61: 129-39; Denell and Miklos, 1971, Mol. Gen. Genet. 110: 167-77). Distortion by *Sd* requires heterozygosity at the *Rsp* locus; when *Rsp* is on the homologue, the *Sd*-bearing chromosome is preferentially recovered. and when *Rsp* and *Sd* are in coupling, the homologue is recovered preferentially. Extensive

electron microscopic studies of spermatogenesis (Tokuyasu, Peacock, and Hardy, 1972, Z. Zellforsch. 124: 479-506; 127: 492-525; 1977, J. Ultrastruct. Res. 48: 284-303) demonstrate that spermiogenesis of *SD/+* males is defective; chromatin in half of the spermatid nuclei fails to condense properly, leading in some cases to a failure of the spermatids to become individually invested in membrane, remaining syncytial instead, and in all cases to incomplete maturation of half the sperm. In addition, the transition from lysine-rich to sperm-specific arginine-rich histone, which normally occurs in late spermiogenesis, does not appear to take place in half the spermatids of *SD/+* males (Hauschteck-Jungen and Hartl, 1978, Genetics 101: 57-69). Temperature-sensitive period of distortion said to be in the primary spermatocyte (Mange, 1968, Genetics 58: 399-413); however, see Matthews and Mortin (1983, Canad. J. Genet. Cytol. 25: 662-67). *k* values also reported to increase with time of storage of sperm by females (Hartl, 1973, Genetics 74: 619-31), to decrease with the age of *SD/+* males (Hiraizumi and Watanabe, 1969, Genetics 63: 212-31), and to be influenced by the genotype of the female to which such males are crossed (Denell and Judd, 1969, Mol. Gen. Genet. 105: 262-74). Segregation distortion is subject to modification by numerous genetic factors throughout the genome; in addition various abnormal chromosome constitutions have been reported to reduce *k* values (Novitski and Erlich, 1970, DIS 45: 102; Enns, 1970, DIS 45: 136; Fowler, 1971, DIS 46: 74). Homozygous (when viable) and heteroallelic constitutions exhibit variably reduced male fertility, but the contribution of *Sd vis á vis* other components of the *SD* chromosomes involved not easily ascertainable. *SD* chromosomes without effect in females.

cytology: Placed in 37D2-5 based on reversions of *Sd* by *Df(2L)Sd2 = Df(2L)37D1-2;38D2-E1*, *Df(2L)Sd14 = Df(2L)37D1-2;38C1-2*, *Df(2L)Sd57 = Df(2L)37D1-2;38C1-2*, and *Df(2L)Sd77 = Df(2L)37D1-2;38C1-2*, but by neither *Df(2L)hk39 = Df(2L)36F6-37A1;37D1-2* nor *Df(2L)pr26 = Df(2L)37D5-6;38C8-10*, all of which were induced in *SD* chromosomes (Brittnacher and Ganetzky, 1983).

molecular biology: A 5 kb *Sd*-specific tandem duplication found in a restriction fragment that hybridizes at 37D5; an *Sd*-specific mRNA associated with the duplication (Powers).

other information: Analysis of the distribution of *k* values among and within genotypes by the use of probit transformation described by Miklos and Smith-White (1971, Genetics 67: 305-17) and by Miklos (1972, Genetics 70: 405-18).

chromosome	inversions	homozygote	origin	ref ^α	comments
SD5	<i>In(2R)45C-F;49A</i> <i>In(2R)NS</i>	lethal	Madison, Wisconsin	4	
SD36	same as <i>SD5</i>	lethal	Madison, Wisconsin	4	
SD72	<i>In(2LR)39-40;42A</i> <i>In(2R)NS</i>	viable	Madison, Wisconsin	4	
SD-BG12	same as <i>SD72</i>		Bowling Green, Ohio	6	also → male recombination
SD-NH	<i>In(2R) NS</i> <i>In(2R)55E;60E</i>		Ohdate, Japan	2	
SD-Ra	<i>In(2L)32A-C;35B-C</i>	female sterile male sterile	Ranna, Sicily	5	
SD-Roma	none	viable	Rome, Italy	3	
SD-S90	<i>In(2R)NS</i>		Northern California	1	also → male recombination

^α 1 = Bencze and Slatko, 1984, Genet. Res. 43: 149-58; 2 = Hiraizumi and Nakazima, 1965, DIS 40: 72; 3 = Nicoletti and Trippa, 1967, Atti Assoc. Genet. Ital. 12: 361-65; 4 = Sandler, Hiraizumi, and Sandler, 1959, Genetics 44: 233-50; 5 = Trippa, Loverre, and Cicchetti, 1980, Genetics 95: 399-412; 6 = Woodruff and Lyman, 1980, Am. Nat. 116: 297-304.

sdby: see *flil*⁸

Sdh: Succinic dehydrogenase

location: 2-89 [based on 199 *M(2)53-bw* recombinants].

origin: Identified by finding a naturally occurring isoallele that produced a slightly more temperature labile enzyme than that produced by other alleles.

references: Lawrence, 1981, J. Embryol. Exp. Morphol. 64: 321-32.

phenotype: The structural gene for succinic dehydrogenase (EC 1.3.99.1). Null mutations are homozygous lethal and lethal in heteroallelic combinations; however clones of homozygous cells survive and develop normally. Detected in clones by a cytochemical test for enzyme activity; cell autonomous with low perdurance; an excellent cell marker.

alleles: Mutant alleles show reduced or missing autonomous activity; some are heat sensitive.

allele	origin	discoverer	comments
Sdh 1	spont	de Jong	heat labile enzyme
Sdh 2	EMS	Lawrence	viable allele
Sdh 3	EMS	Lawrence	lethal
Sdh 4	EMS	Lawrence	lethal
Sdh 5	EMS	Lawrence	lethal
Sdh 6	EMS	Lawrence	lethal
Sdh 7	EMS	Lawrence	lethal
Sdh 8	EMS	Lawrence	lethal at 25° viable at 18°

sdp: sandpaper

location: 2-83 (based on 53 *pr-px* recombinants).

origin: Induced by ethyl methanesulfonate.

references: Garcia-Bellido and Dapena, 1974, Mol. Gen. Genet. 128: 117-30.

phenotype: Homozygous lethal, but survives as clones of homozygous cells; useful as a cell marker in the cuticle. Produces depigmentation and changes in trichome pattern only in tergites; extra processes per epidermal cell on tergite surface; densely packed uncombed. Not detectable in thorax.

sdt: stardust

location: 1-23.

references: Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307 (fig.).

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Embryonic lethal. Hypoderm almost totally absent; only small remains of cuticle found. *sdt*⁺ function not required in female germline (Wieschaus and

Noell, 1986, Roux's Arch. Dev. Biol. 195: 63-73).

alleles: Six ethyl methanesulfonate-induced alleles recorded; two weak; one temperature sensitive.

allele	synonym	ref ^α
sdt 1	<i>XM</i>	2
sdt 2	<i>NO</i>	1, 2
sdt 3	<i>P9</i>	1, 2
sdt 4		2
sdt 5		2
sdt 6		2

^α 1 = Wieschaus and Noell, 1986, Roux's Arch. Dev. Biol. 195: 63-73; 2 = Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Roux's Arch. Dev. Biol. 193: 296-307.

cytology: Placed in 7D10-F2 based on its inclusion in *Df(1)RA2 = Df(1)7D10;8A45* but not *Df(1)KA14 = Df(1)7F1-2;8C6*.

sdx: spreadex

location: 1- (rearrangement).

origin: X ray induced.

discoverer: Muller.

synonym: *spx* (preoccupied).

references: 1965, DIS 40: 35.

phenotype: Wings spread widely apart and often directed somewhat downward, as in *Dichaete*. Abdomen of female tends to be narrow and shrunken. Fertility sufficient for maintaining homozygous stock. RK2A.

cytology: Associated with *In(1)sdx*; breakpoints unknown.

se: sepia

location: 3-26.0.

phenotype: Eye color brown at eclosion, darkening to sepia, and becoming black with age. Thought to be the structural gene for the enzyme PDA synthetase which catalyzes the conversion of 6-pyruvoyltetrahydropterin to 2-amino-4-oxo-6-acetyl-7,8-dihydro-3H,9H-pyrimido [4, 5, 6]-[1, 4] diazepine (=PDA), which is a precursor of the red drosoprotein pigments. The enzyme has been partially purified and has a molecular weight of 48,000 daltons (Wiederrecht and Brown, 1984, J. Biol. Chem. 259: 14121-27). Pigment of ocelli normal. Chromatographically, *se* eyes characterized by having no red pigment [e.g. drosoprotein and isodrosoprotein (McKay, 1972, DIS 48: 62)] and an accumulation of yellow pigment (Hadorn and Mitchell, 1951, Proc. Nat. Acad. Sci. USA 37: 650-65; Ziegler-Günder and Hadorn, 1958, Z. Vererbungsl. 89: 235-45). Three yellow pigments identified are 6- derivatives of 2-amino-4-hydroxy-7,8-

dihydropteridine; the most abundant is sepiapterin, the 6-lactyl derivative; minor species are isosepiapterin (Viscontini and Möhlmann, 1959, *Helv. Chim. Acta* 42: 836-41; Forrest, VanBaalen, and Myers, 1959, *Arch. Biochem. Biophys.* 83: 508-20) and neosepiapterin (Takikawa, 1973, *DIS* 50: 158; Sugiura, Takikawa, Tsusue, and Goto, 1973, *Bull. Chem. Soc. Jpn.* 46: 3312-13), which are the 6-propionyl and 6-acetyl derivatives respectively; the latter compound shown to be D-erythro-neopterin by Katoh and Arai (1974, *DIS* 51: 70). Other pteridines present in greater-than-normal amounts [*e.g.* isoxanthopterin II, xanthopterin I, and biopterin (McKay)]. Eye color autonomous in *se* eye disks transplanted into wild-type hosts (Beadle and Ephrussi, 1936, *Genetics* 21: 230). *pn se* flies have 10% amount of sepiapterin of *se* alone (Lifschytz and Falk, 1969, *Genet. Res.* 14: 53-61); *dor se* displays reduced viability (Lucchesi, 1968, *Genetics* 59: 37-44). RK1.

alleles:

allele	origin	discoverer	ref ^α	comments
<i>se</i> ¹	spont	E. M. Wallace, 13e10	3	
<i>se</i> ^{51j}	spont	Hungerford, 51j	10	
<i>*se</i> ^{51k}	spont	Clark, 51k	5	
<i>*se</i> ⁵⁸	spont	Andrew, 58k	1	
<i>se</i> ⁶¹	spont	Clancy, 61c	4	
<i>se</i> ⁷²	spont	Anxolabehre, 72l	2	
<i>se</i> ⁷⁹	spont	Najera, 79i	7, 8	
<i>se</i> ^{80l}	spont	Najera, 80i	9	
<i>se</i> ⁸¹	spont	Najera, 81d	7	
<i>*se</i> ^v	X ray		6	T(Y;3) variegated

^α 1 = Andrew, 1959, *DIS* 33: 82; 2 = Anxolabehre and Periquet, 1973, *DIS* 50: 21; 3 = Bridges and Morgan, 1923, *Carnegie Inst. Wash. Publ. No. 327*: 86 (fig); 4 = Clancy, 1964, *DIS* 39: 65; 5 = Clark, 1952, *DIS* 26: 60; 6 = Jeffery, Stephans, and Giddings, 1974, *Genetics* 77: s32; 7 = Najera, 1984, *DIS* 60: 241; 8 = Najera, 1985, *DIS* 61: 215; 9 = Najera, 1986, *DIS* 63: 167; 10 = Redfield, 1952, *DIS* 26: 68.

se-like 62: see *pn*²

sed: see *Hn*^{r3}

Segregation distorter: see *Sd*

seg: short egg

location: 1- (proximal to *B*).

synonym: *l(1)SE*.

references: Wieschaus, 1978, *Symp. Soc. Dev. Biol.* 36: 23-43.

phenotype: Eggs of homozygous females 20% shorter and 10% wider than normal; volume and density of blastoderm nuclei unchanged. Defect in somatic tissue inferred from normal phenotype of germ-line clones (Wieschaus).

sei: seizure (J.C. Hall)

location: 2-106 [Ganetzky; probably a short distance (<1 m.u.) distal to this, as inferred from cytology].

origin: Induced by ethyl methanesulfonate.

discoverers: Wu and Ganetzky.

references: Jackson, Wilson, Strichartz, and Hall, 1984, *Nature* 308: 189-91.

Jackson, Gitschier, Strichartz, and Hall, 1985, *J. Neurosci.* 5: 1144-51.

Kasbekar, Nelson, and Hall, 1987, *Genetics* 116: 423-31.

phenotype: Paralyzed fairly rapidly at temperatures above 38°; adults seem to have a fit before becoming immobile, as opposed to immediately ceasing their movements;

increasing times of exposure to high temperature leads to progressively longer times necessary for recovery when temperature is lowered (Jackson *et al.*, 1985); *sei*¹ is recessive for heat-sensitive paralysis (Jackson *et al.*, 1985); *sei*² is semi-dominant in this respect, and behavior of hyperploid adults exhibits dose dependence: *sei*²/*sei*²/+ flies become paralyzed, at 40°, slightly less rapidly than homozygous mutants; *sei*²/+ paralyzed more slowly than *sei*²/+ but faster than wild-type (Jackson *et al.*, 1985); biochemically, head membrane extracts from *sei*² adults exhibit anomalously high K_d of saxitoxin (STX) binding activity, and a different pH dependence of this activity from wild-type; these abnormalities observed when the incubations done at high, but not low, temperatures (Jackson *et al.*, 1984); in flies carrying three *sei* alleles, apparent reductions in the amounts of STX bound are observed, which are proportional to the relative number of mutant alleles (Jackson *et al.*, 1985); when these kinds of biochemical experiments are performed on head extracts from *sei*¹, K_d values are not appreciably different from wild-type at all assay temperatures, but the concentrations of STX binding activity are lower than wild-type under the influence of this allele, with such reductions being more marked at higher assay temperatures; recovery times, after exposure to high temperature and subsequent return to mild conditions, are shortened when *sei*² is combined with *nap*^{ts} (Jackson *et al.*, 1985). Recordings of action potentials in the adult giant-fiber pathway reveal no decrement in *sei*² at 40° (Nelson and Baird, 1985, *Neurosci. Abstr.* 11: 313); at temperatures >40°, *sei*² causes spontaneous activity in recordings from dorsal longitudinal flight muscles, to appear coincidentally with the heat-induced paralysis (Kasbekar *et al.*, 1989); from "tight-seal whole-cell" recordings, using cultured neurons from *sei*¹ embryos, this mutation found to cause sodium current density to be about half-normal; yet, voltage dependence and channel-gating properties were not different from wild type (O'Dowd and Aldrich, 1988); for other aspects of *sei*-associated defects in physiology, see enhancer of seizure.

alleles: Two temperature sensitive alleles; *sei*¹ and *sei*².

Some new ones recently isolated (Deak and Hall, 1989, *Neurosci. Abstr.* 15: 196), but not specified as to how many, the mutagenizing agent, or the allele names; certain of these, however, were found to be small deletions of the locus, which were used to generate apparently viable overlapping, *Df/Df* genotypes; these adults are paralyzed at 38°, suggesting that the null phenotype is temperature-sensitive paralytic (Deak and Hall, 1989).

cytology: Placed in 60A7-B10 based on its position between *Df(2R)bw-S46 = Df(2R)59D8-11;60A7* and *Df(2R)Px = Df(2R)60B8-10;60D1-2* (Jackson *et al.*, 1986; L.M. Hall, unpublished).

other information: Though some of the data mentioned above suggest *sei* might code for a voltage-sensitive sodium-channel component, this gene could not be colocalized with a known sodium-channel-encoding factor in 2R, which on the basis of clone and sequence plus *in situ* hybridization, maps within 60D-E (Salkoff, Butler, Wei, Scavarda, Griffen, Ifune, Goodman, and Mandel, 1987, *Science* 237: 744-749); see *NaCP*.

***semi-f: semiforked**

location: 3- (not located).
origin: Spontaneous.
discoverer: Lancefield, 18b.
references: 1918, Am. Naturalist 52: 462-64.
phenotype: Homozygotes that are also heterozygous for *f* have slightly forked bristles. RK3.

sens(3)str: sensitivity (3) streptomycin

location: 3-.
origin: Spontaneous.
synonym: *str*^S.
references: Duke and Glassman, 1968, Nature (London) 220: 588-89.
phenotype: Homozygous larvae, but not adults, show 96% lethality when cultured on media containing 1% streptomycin, a concentration tolerated by wild type. Development of homozygotes arrested in early larval stages.

sep: separated

location: 3- (rearrangement).
discoverer: Muller.
phenotype: Most of posterior crossvein absent, one-third usually remaining attached to vein L5. RK2A.
cytology: Associated with *In(3LR)sep = In(3LR)65D2-3;85F2-4* (Craymer, 1981, Genetics 99: 75-97).

sepia: see *se*

sepiacid: see *Hn*^{r3}

sepiapterin synthetase: see *pr*

Ser: see *Bd*^S

Ser1: Serine protease 1

location: 3-{101}.
references: Yun and Davis, 1989, Mol. Cell. Biol. 9: 692-700.
phenotype: Expressed abundantly in larval gut. Mutants *dnc*^{M11} and *dnc*^{M14} show lower levels of *Ser1* RNA than normal flies; levels of cAMP, however, are elevated in the mutants. The *Ser1*-related genes *Ser2* and *Ser3* (with same location and cytology) are not expressed differentially in *dnc* mutants.
cytology: Located in 99C-D by *in situ* hybridization to salivaries.
molecular biology: *Ser1*, *Ser2*, and *Ser3* cloned and their nucleotide and putative amino acid sequences determined (Yun and Davis, 1989); amino acid sequences homologous to serine protease enzyme family.

Serendipity: see *Sry*

Serendipity cognate: see *Sryc*

Serine protease 1: see *Ser1*

serpent: see *srp*

Serrate: see *Bd*^S

sesA: stress-sensitive: A (J.C. Hall)

location: 1-23.3 (Homyk).
origin: Induced by ethyl methanesulfonate.
discoverer: Homyk.
references: Homyk and Sheppard, 1977, Genetics 87: 95-104.
phenotype: Paralyzed briefly by mechanical shock, effected by banging a container of the flies (this barely,

or not at all, stuns wild-type flies); mutant's paralysis is accompanied by wing beating and followed by erratic movements; when stimulated to jump and fly, does so abnormally for short distances. Lethal when raised at 29°.

sesB (J.C. Hall)

location: 1-32.4 (Homyk).
origin: Induced by ethyl methanesulfonate.
discoverer: Homyk.
references: Homyk and Sheppard, 1977, Genetics 87: 95-104.
 Homyk, Szidonya, and Suzuki, 1980, Mol. Gen. Genet. 177: 553-65.

phenotype: *sesB*¹ causes adults to be extremely sensitive to mechanical shock (Homyk and Sheppard, 1977); paralysis lasts many seconds, and recovery is slow; the mutant is also generally inactive and uncoordinated; when stimulated to jump and fly, does so abnormally for short distances; when reared at 29°, after being raised at 22°, *sesB*² is not only stress-sensitive, but it also becomes debilitated after several bouts of induced to hopping and flying; in addition, *sesB*² is heat sensitive for female sterility (after rearing at low temperature).

alleles: Lack of complementation of lethal alleles and *sesB* alleles reported by Zhimulev, Pokholkova, Bgatov, Umbetova, Solovjeva, Khudyakov, and Belyaeva (1987, Biol. Zentralbl. 106: 699-720). Janca, Woloshyn, and Nash (1986, Genetics 112: 43-64) report lack of full complementation between *sesB*³ and *l(1)9Ed* alleles.

allele	origin	discoverer	synonym	ref ^α	comments
ses B1	EMS	Homyk		1,2	viable allele
ses B2	EMS	Homyk		1,2	viable allele
ses B3	X ray	Schalet	<i>l(1)S12</i>	3	lethal allele
ses B4	X ray	Lefevre	<i>l(1)HF380</i>	4	lethal allele;
					<i>In(1)9E-F;20A4-5</i>
ses B5	EMS	Lefevre	<i>l(1)EA79</i>	5	lethal allele

^α 1 = Homyk and Sheppard, 1977, Genetics 87: 95-104; 2 = Homyk, Szidonya, and Suzuki, 1980, Mol. Gen. Genet. 177: 553-65; 3 = Lefevre, 1971, Genetics 67: 497-513; 4 = Lefevre, 1981, Genetics 99: 461-80; 5 = Lefevre and Watkins, 1986, Genetics 113: 869-95.

cytology: Placed in 9E7-F4 based on its inclusion in the region of overlap between *Df(1)ras-P14 = Df(1)9E1-2;9F3-4* and *Df(1)v64f = Df(1)9E7-8;10A1-2* (Zhimulev *et al.*).

sesC: see *stn*

sesD (J.C. Hall)

location: 1-23.8 (Homyk and Pye, 1989).
origin: Induced by ethyl methanesulfonate.
references: Homyk, Szidonya, and Suzuki, 1980, Mol. Gen. Genet. 177: 553-65;
 Homyk and Pye, 1989, J. Neurogenet. 5: 37-48.
phenotype: Paralyzed by mechanical shock; one allele (*sesD*²) causes constitutive inactivity, poor jumping and flying ability, abnormal landing responses, and feeble male courtship (Homyk *et al.*, 1980); this allele also leads to longer stress-induced paralysis than does *sesD*¹; the latter is a heat-sensitive developmental lethal. In addition, *sesD*¹ is reversibly temperature sensitive for the following visual phenotype, shown by electroretinogram recordings (Homyk and Pye, 1989): at temperatures <30°, orange-light-elicited recovery from blue-induced prolonged depolarizing after potential (PDA) is slow, and

orange responses induced after recovery are aberrantly low-amplitude; at higher temperatures, orange-induced recovery from PDA involves incomplete repolarization, so that potential levels off below baseline extant before exposure to blue light; this persists even after continued orange stimulation or in the dark; under these conditions, orange light also leads to light-on and light-off ERG transient spikes with anomalously low amplitudes; these heat-induced effects are quickly reversible (to normal ERG/PDA-associated potentials) when temperature lowered to 25°.

alleles: Two alleles, *sesD*¹ and *sesD*².

cytology: Maps within 7D13-14, based on assessment of ERG abnormalities of heterozygotes between *sesD*¹ and *Df(1)HA11 = Df(1)7D13-14;7D22*, which uncovers the defects and *Df(1)γa2 = Df(1)7D14;8A3*, which does not (Homyk and Pye, 1989).

other information: Complements adult-lethal-1, which maps nearby; rapid exhaustion does, too, but it maps to 7C1-3 (Homyk and Pye, 1989); mosaic analysis suggests stress-induced paralysis of legs associated with *sesD* maps to separate foci, since the individual appendages can be paralyzed independently in such gynandromorphs (Homyk, unpublished); *sesD*¹'s developmental temperature-sensitive lethality originally said (Homyk *et al.*, 1980) to be caused by separate mutation on the X chromosome, but turned out not to be the case (Homyk, unpublished).

***sesE* (J.C. Hall)**

location: 1-29.8 (Homyk).

origin: Induced by ethyl methanesulfonate.

discoverer: Homyk.

references: Homyk, Szidonya, and Suzuki, 1980, *Mol. Gen. Genet.* 177: 553-65.

Homyk, Sinclair, Wong, and Grigliatti, 1986, *Genetics* 113: 367-89.

phenotype: Severely paralyzed by mechanical shock and also constitutively inactive; adults become hyperactive under crowded conditions (Homyk *et al.*, 1980). A heat-sensitive developmental lethal, with two temperature-sensitive periods, one primarily during second larval instar and another ranging from late third instar to early pupation (Homyk *et al.*, 1986); also an adult lethal, such that about half the flies are dead after five days exposure to 29° (Homyk *et al.*, 1986). From analysis of high-temperature-induced adult death and leg paralysis in mosaics, the foci of the former concluded to be diffuse, "domineering," and near posterior region of ventral blastoderm; regarding the behavioral phenotype, individual legs observed to be paralyzed, or not, in these gynandromorphs, and from these data it was also inferred that the paralysis foci are rather near that for lethality.

***sesF* (J.C. Hall)**

location: 1-49.6 (Homyk).

origin: Induced by ethyl methanesulfonate.

discoverer: Homyk.

references: Homyk, Szidonya, and Suzuki, 1980, *Mol. Gen. Genet.* 177: 553-65.

phenotype: Adults have small bristles and are heat-sensitive for death during development; temperature-sensitive period for lethality includes at least larval stages (Homyk); when cultures shifted to 29° after pupariation, adults eclose but are very uncoordinated (Homyk); the

originally stress-sensitive character seems to have vanished from the strain, though jumping ability of adults may still be impaired.

cytology: Placed in 12E1-13A5, on the basis of its inclusion in *Df(1)KA9 = Df(1)12E1;13A5* (Homyk, unpublished).

other information: Complements nearby *sesG* and *sesH* mutations.

***sesG* (J.C. Hall)**

location: 1-50.8 (Homyk).

origin: Induced by ethyl methanesulfonate.

discoverer: Homyk.

references: Homyk, Szidonya, and Suzuki, 1980, *Mol. Gen. Genet.* 177: 553-65.

phenotype: Flies become hyperactive when mechanically shocked, especially in crowded conditions; when in uncrowded containers, the adults tend to be inactive after coming to rest from flight.

other information: Complements the nearby *sesF* and *sesH* mutations.

***sesH* (J.C. Hall)**

location: 1-51.7 (Homyk).

origin: Induced by ethyl methanesulfonate.

discoverer: Homyk.

references: Homyk, Szidonya, and Suzuki, 1980, *Mol. Gen. Genet.* 177: 553-65.

phenotype: Briefly paralyzed after mechanical shock; during tethered flight, wing beating anomalously stops and starts again spontaneously; adults become hyperactive during various kinds of behavioral testings (*e.g.*, assessments of flying, climbing, running abilities; ability to hop when wings removed; landing responses; male tracking of, and vibration at, females).

other information: Complements the nearby *sesF* and *sesG* mutations.

sev: sevenless

location: 1-33.38.

references: Harris, Stark, and Walker, 1976, *J. Physiol.* 256: 127-62.

Tomlinson and Ready, 1986, *Science* 231: 400-02.

Banerjee, Renfranz, Hinton, Rabin, and Benzer, 1987a, *Cell* 51: 151-58.

Banerjee, Renfranz, Pollock, and Benzer, 1987b, *Cell* 49: 281-91.

Hafen, Basler, Edström, and Rubin, 1987, *Science* 236: 55-63.

Tomlinson, Bowtell, Hafen, and Rubin, 1987, *Cell* 51: 143-50.

Tomlinson and Ready, 1987, *Dev. Biol.* 123: 264-75.

Basler and Hafen, 1988, *Cell* 54: 299-311.

Bowtell and Rubin, 1988, *Genes Dev.* 2: 620-34.

Tomlinson, Kimmel, and Rubin, 1988, *Cell* 55: 771-84.

Bowtell, Simon, and Rubin, 1989, *Cell* 56: 931-36.

Bowtell, Kimmel, Simon, and Rubin, 1989, *Proc. Nat. Acad. Sci. USA* 86: 6245-49.

Renfranz and Benzer, 1989, *Dev. Biol.* 136: 411-29.

Rubin, 1989, *Cell* 57: 519-20.

phenotype: Homozygotes and hemizygotes lack the R7 rhabdomere in all ommatidia; the R7 photoreceptor cell instead develops as an accessory lens-secreting cell, the equatorial cone cell (Tomlinson and Ready, 1986, 1987). Cell autonomous in mosaics (Harris *et al.*). Mosaic

analysis demonstrates that *sev*⁺ product is required only in the presumptive photoreceptor 7 cell and is therefore involved in receiving signals from neighboring cells. Electroretinograms normal; defective in response to ultraviolet; in T-maze tests *sev* flies prefer visible to ultraviolet wavelengths and green light to darkness; slight preference for blue wavelengths over ultraviolet used in selecting mutant alleles (Gerresheim, 1988, Behav. Genet. 18: 227-46). In the presence of *Phb* the preference of green light to darkness is reversed for *sev*¹ and *sev*¹⁰ but not for six other unspecified alleles; the absence of R7 is unaffected by *Phb* (Ballinger and Benzer, 1988, Proc. Nat. Acad. Sci. 85: 3960-64). Gene expression demonstrated by *in situ* hybridization and immune staining to take place in developing eye imaginal disc, near and posterior to the morphogenetic furrow, during ommatidial differentiation. Protein present transiently in at least nine cells in each developing ommatidium and is detectable before any overt differentiation of R7 observed (Tomlinson *et al.*, 1987; Banerjee *et al.*, 1987a). Transient expression of *sev*⁺ by means of a transformed construct under control of the *Hsp70* promoter in a *sev* background results in a narrow stripe of ommatidia that contain photoreceptor cell 7 in eyes that are otherwise sevenless; the position and time of expression required for normal function are highly restricted (Bowtell, Simon, and Rubin, 1989, Cell 56: 931-36).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>sev</i> ¹	EMS	Harris	<i>sev</i> ^{Ly3}	3	
<i>sev</i> ²	EMS		<i>sev</i> ^{dr1}	1	
<i>sev</i> ³	EMS		<i>sev</i> ^{E1}	1	
<i>sev</i> ⁴	EMS		<i>sev</i> ^{E2}	1	
<i>sev</i> ⁵	EMS		<i>sev</i> ^{E3}	1	
<i>sev</i> ⁶	EMS		<i>sev</i> ^{E4}	1	
<i>sev</i> ⁷	EMS		<i>sev</i> ^{E5}	1	
<i>sev</i> ⁸	EMS	Ready	<i>sev</i> ^{elm}	1	
<i>sev</i> ⁹	EMS	Ready	<i>sev</i> ^{fig}	1	
<i>sev</i> ¹⁰	EMS	Ready		1	
<i>sev</i> ¹¹	EMS	Gerresheim, 1988	<i>sev</i> ^{a27}	2	
<i>sev</i> ¹²	EMS	Gerresheim, 1988	<i>sev</i> ^{a32}	2	4.3kb insertion
<i>sev</i> ¹³	EMS	Gerresheim, 1988	<i>sev</i> ^{b8}	2	
<i>sev</i> ¹⁴	EMS	Gerresheim, 1988	<i>sev</i> ^{d2}	2	
<i>sev</i> ¹⁵	EMS	Gerresheim, 1988	<i>sev</i> ^{f1}	2	
<i>sev</i> ¹⁶	EMS	Gerresheim, 1988	<i>sev</i> ^{f3}	2	
<i>sev</i> ¹⁷	EMS	Gerresheim, 1988	<i>sev</i> ⁱ³	2	
<i>sev</i> ¹⁸	P		<i>sev</i> ^{P1}	1	53kb deletion; female sterile
<i>sev</i> ¹⁹	P		<i>sev</i> ^{P2}	1	
<i>sev</i> ²⁰	P		<i>sev</i> ^{P3}	1	11kb deletion

^α 1 = Banerjee, Renfranz, Pollock, and Benzer, 1987, Cell 49: 281-91; 2 = Gerresheim, 1988, Behav. Genet. 18: 227-46; 3 = Harris, Stark, and Walker, 1976, J. Physiol. 256: 127-62.

cytology: Placed in the right edge of the 10A1-2 doublet on the basis of its inclusion in *Df(1)ras-vCc8 = Df(1)9D1-2;10A2-3* but not *Df(1)sbr-K8 = Df(1)9B1-2;10A1-2* or *Df(1)v64f = Df(1)9E7-8;10A1-2* (Zhimulev, Semeshin, and Belyaeva, 1981, Chromosoma 82: 9-23).

molecular biology: Locus cloned by transposon tagging (Banerjee *et al.*, 1987b) and by microdissection followed by chromosome walking (Hafen *et al.*, 1987). cDNA sequencing (Hafen *et al.*, 1987; Basler and Hafen, 1988) reveals a gene with twelve exons, the first four of which are separated by long introns; exons 4-7 are short and separated by short introns; exon 8 contains the majority of the gene; exons 9-12 are again short and separated

from exon 8 and from each other by short introns. The conceptual amino-acid sequence indicates a protein of 2554 amino acids of calculated molecular weight 290 kd. Two putative membrane-spanning domains indicated-22 and 24 amino acids, beginning with residues 102 and 2124, respectively. These hydrophobic domains flank closely two short hydrophilic stretches (Basler and Hafen, 1988). The 2000 residues between the membrane-spanning domains are the extracellular domain (Bowtell, Simon, and Rubin, 1988). Transcription from right to left; produces an 8.2 kb mRNA, which is found in isolated third-instar imaginal eye discs, but not in wing discs or pre-third-instar discs, at low levels in adult heads, but not bodies, and in 5-12 h, but not 22-30 h pupae. The carboxy-terminal cytoplasmic domain shows homology with the tyrosine kinase domains of such proteins as *c-ras*, *v-src*, and the EGF receptor (Hafen *et al.*). A single amino acid substitution in the ATP-binding site of the putative kinase inactivates the gene as determined in transformation experiments (Basler and Hafen). Expression of *sev*⁺-β-galactosidase fusion protein confined to the apical surface of developing retina; expression not restricted to R7 cell; expressed in presumptive photoreceptor cells, cone cells, and possibly others. Protein localizes to all membrane of the apical tips and their microvilli, away from the bulk of the cell-cell contacts (Banerjee *et al.*, 1987a). 967 nucleotides of 5' promoter region capable of conferring the correct pattern but not level of *sev* expression on a *lacZ* reporter gene in some transformants. Enhancer sequences in the genomic segment comprising exons 2-7 able to confer both correct pattern and level of sevenless expression on either *sev* or heterologous promoters (Bowtell, Kimmel, Simon, and Rubin, 1989, Proc. Nat. Acad. Sci. USA 86: 6245-49). *sev* minigenes lacking introns expressed at low levels but in correct pattern (Bowtell *et al.*, 1988).

seven up: see *svp*

seven-in-absentia: see *sina*

sex comb distal: see *scd*

Sex combs extra: see *ScE*

Sex combs on midleg: see *Scm*

Sex combs reduced: see *Scr* under *ANTC*

Sex lethal: see *Sxl*

sexcombless: see *sx*

sey: scratched eyes

location: 3-17.0 (to the left of *Me*).

origin: Spontaneous.

discoverer: Grell.

phenotype: At 25° homozygotes have patches of eroded and blackened eye tissue; phenotype normal at 18°.

sf: safranin

location: 2-71.5.

phenotype: Eye color soft dark brown. More easily classified in male and in aged fly. Eye color autonomous in larval optic disks transplanted into wild-type host (Beadle and Ephrussi, 1936, Genetics 21: 230). Larval Malpighian tubes of *sf*¹ pale yellow; classifiable (Brehme and Demerec, 1942, Growth 6: 351-56); those

of *sf*² bright yellow like wild type (Beadle, 1937, *Genetics* 22: 587-611). RK2.

alleles:

allele	origin	discoverer	synonym	ref ^α
<i>sf</i> ¹	spont	Bridges 16a6		3
<i>sf</i> ²	spont	Spencer, 25k	<i>bronze</i>	7, 8, 9
<i>*sf</i> ³	spont	Ives, 39c	<i>dark eye</i>	1
<i>*sf</i> ^{32e}	heat	Ives, 32e28		2, 6
<i>sf</i> ⁷⁹	spont	Najera		4
<i>sf</i> ^{80d}	spont	Najera		5
<i>sf</i> ⁸⁰ⁱ	spont	Najera		5
<i>sf</i> ⁸¹	spont	Najera		4

^α 1 = Curry, 1939, *DIS* 12: 45; 2 = Plough and Ives, 1934, *DIS* 1: 33; 3 = Morgan, Bridges, and Sturtevant, 1925, *Bibliogr. Genet.* 2: 235; 4 = Najera, 1985, *DIS* 61: 215; 5 = Najera, 1986, *DIS* 63: 167; 6 = Plough and Ives, 1935, *Genetics* 20: 42-69; 7 = Spencer, 1934, *DIS* 1: 35; 8 = Spencer, 1935, *Am. Nat.* 69: 223-38; 9 = Spencer, 1937, *DIS* 7: 21.

***sf-3: safranin in chromosome 3**

location: 3- (not located).

discoverer: Bridges, 15a15.

references: Bridges and Morgan, 1923, *Carnegie Inst. Washington Publ. No.* 327: 126.

phenotype: Eye color dull brown. RK3.

***sfc: stiff chaetae**

location: 1-3.2.

origin: Induced by D-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3026).

discoverer: Fahmy, 1955.

references: 1958, *DIS* 32: 74.

phenotype: Bristles short and stiff; occasionally one missing. Fertility and viability good. RK2.

alleles: One allele induced by CB. 1592.

***sg: shortened wing**

location: 3- (left arm).

origin: Spontaneous.

discoverer: Herskowitz, 47118.

references: 1949, *DIS* 23: 57.

phenotype: Wings abnormal at base; veins interrupted, missing, or thickened. Many flies have short, rounded wings that curve upward slightly. RK3.

SGA62: see *iab7*^{SGA} under *BXC*

***sge: shifted genitals**

location: 1-48.4.

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1956.

references: 1958, *DIS* 32: 74.

phenotype: Male genitalia and anal plates rotated to various degrees (up to 90°). Wings slightly divergent and drooping, occasionally one outheld. Eyes slightly dark. Male sterile. Viability about 70% normal. RK2.

sgg: shaggy

location: 1-1.32.

synonym: *l(1)zw3*.

references: Simpson, El Messal, Moscoso del Prado, and Ripoll, 1988, *Development* 103: 391-401.

Ripoll, El Messal, Laran, and Simpson, 1988, *Development* 103: 757-67.

Simpson and Carteret, 1989, *Development* 106: 57-66.

Bourouis, Heitzler, El Messal, and Simpson, 1989,

Nature 341: 442-44 (fig.).

phenotype: Larval growth protracted, ceasing in the first [*sgg*², *sgg*⁹], second [*sgg*⁵], or third [*sgg*³ and *sgg*¹⁰] larval instar; death follows. *sgg*¹⁰ may survive to puparium formation. Mutant cuticle tissue survives only in tergites; lacks bristles and appears etched; some deformed mutant wing tissue also observed in *sgg*³. *sgg*³, but no other allele produces viable and fertile males in combination with *Dp(1;4)w^{m65g}* (Shannon, Kaufman, Shen, and Judd, 1972, *Genetics* 72: 615-38). *sgg*¹ and *sgg*¹⁰ males exhibiting maternal-zygotic interaction with *Dp(1;4)mg* display deletion-mirror-image duplication homeotic transformation (eye-antenna, wing, and leg discs) (Robbins, 1983, *Genetics* 103: 633-48). Same noted in male tissue of gynandromorphs (Kaufman), *sgg*⁹, and *sgg*¹⁰ (García-Bellido and Robbins, 1983, *Genetics* 103: 235-47). More recent studies with *sgg*³², an amorphic allele, (Bourouis *et al.*; Ripoll *et al.*; Simpson *et al.*; Simpson and Carteret) indicate that *sgg* is involved in the developmental choice between the epidermal pathway and that of the nervous system. In homozygous clones of *sgg*³² formed 48 h or more before puparium formation all trichomes are replaced by chaetae in the ratio of one chaeta to four trichomes reflecting the number of cells involved in the elaboration of each structure; chaetae formed conform to positional information and genotype; *e.g.*, wing clones resemble dorsal or ventral costal bristles or triple or double row bristles depending on their position; clones are linear when near the margins and form clumps of chaetae when distant from the margins; adventitious veins often formed in association with wing clones. In the notum scutellar clones form macrochaetae in + but not *sc* genotypes and microchaetae in *h* but not + genotypes. Clones formed later than 48 h before puparium formation form trichomes, either more densely than normal (10-21 trichomes) or in normal density (up to 10 trichomes). *sgg* embryos from homozygous *sgg* female germ line cells exhibit delayed and disordered cellularization at blastoderm; gastrulation does not occur; no differentiation of ectoderm, mesoderm, or endoderm occurs; cell division continues and the embryo becomes filled with small round cells, most or all of which stain with neuronal-specific antibodies. *sgg*⁺ embryos from *sgg* female germinal tissue show nearly normal blastoderm formation and gastrulation; however they are short with complete cuticles and neural hyperplasia as seen in neurogenic mutants; a lawn of hairs is seen dorsally, and ventrally most of the denticle belts are lacking. Two doses of *sgg*⁺ in such embryos lead to more nearly normal cuticular development, but with odd-numbered denticle bands appearing before even-numbered ones. *sgg* progeny of females that carry *sgg*⁺ in their germ lines die as defective larvae with underdeveloped central nervous systems; the degree of CNS development is positively correlated with the number of maternal doses of *sgg*⁺.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>sgg</i> ¹	X ray	Judd	<i>l(1)zw3^{b12}</i>	1, 2	<i>In(1)b12,MZI</i>
<i>sgg</i> ²	"	Elequin	<i>l(1)zw3^{b24}</i>	2	
<i>sgg</i> ³	"	Elequin	<i>l(1)zw3^{b25}</i>	2	
<i>sgg</i> ⁴	"	Judd	<i>l(1)zw3^{d13}</i>	2	with <i>l(1)3Ac¹⁴</i>
<i>sgg</i> ⁵	X ray+EI	Alexander	<i>l(1)zw3^{g5}</i>	2	
<i>sgg</i> ⁶	EI	Alexander	<i>l(1)zw3^{g13}</i>	2	

allele	origin	discoverer	synonym	ref ^α	comments ^β
sgg⁷	EMS	Judd	<i>l(1)zw3^{h2}</i>	2	
sgg⁸	X ray	Judd	<i>l(1)zw3^{h15}</i>	2	
sgg⁹	X ray	Judd	<i>l(1)zw3^{h22}</i>	2	MZI
sgg¹⁰	EI	Alexander	<i>l(1)zw3^{k22}</i>	1, 2	MZI
sgg¹¹	EMS		<i>l(1)zw3^{e6}</i>	5	
sgg¹²	EMS		<i>l(1)zw3^{e28}</i>	5	
sgg¹³	EMS		<i>l(1)zw3^{e29}</i>	5	
sgg¹⁴	EMS		<i>l(1)zw3^{e56}</i>	5	
sgg¹⁵	EMS		<i>l(1)zw3^{e60}</i>	5	
sgg¹⁶	MMS		<i>l(1)zw3^{m9}</i>	6	
sgg¹⁷	MMS		<i>l(1)zw3^{m17}</i>	6	
sgg¹⁸	MMS		<i>l(1)zw3^{m41}</i>	6	
sgg¹⁹	MMS		<i>l(1)zw3^{m62}</i>	6	
sgg²⁰	MMS		<i>l(1)zw3^{m69}</i>	6	with <i>l(1)3Ag⁶⁵</i>
sgg²¹	MMS		<i>l(1)zw3^{m72}</i>	6	
sgg²²	MMS		<i>l(1)zw3^{m80}</i>	6	
sgg²³	MMS		<i>l(1)zw3^{m98}</i>	6	
sgg²⁴	X ray	Lefevre	<i>l(1)GE228</i>	3	<i>ln(1)3B;20A</i>
sgg²⁵	X ray	Lefevre	<i>l(1)GE257</i>	3	<i>ln(1)3B3;20F</i>
sgg²⁶	X ray	Lefevre	<i>l(1)HA41</i>	3	
sgg²⁷	X ray	Lefevre	<i>l(1)JA131</i>	3	
sgg²⁸	X ray	Lefevre	<i>l(1)KA22</i>	3	
sgg²⁹	EMS	Lefevre	<i>l(1)VE762</i>	4	
sgg³⁰	EMS	Lefevre	<i>l(1)VE805</i>	4	
sgg³¹	mei9 ^γ	Schalet	<i>l(1)zw3^{SIM}</i>		
sgg³²	EMS	Ripoll	<i>l(1)zw3^{sgg}</i> <i>D127</i>	1, 7, 8, 9	

^α 1 = Bourouis, Heitzler, El Messal, and Simpson, 1989, Nature (London) 341: 442-44 (fig.); 2 = Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 5 = Lim and Snyder, 1974, Genet. Res. 24: 1-10; 6 = Liu and Lim, 1975, Genetics 79: 601-11; 7 = Ripoll, El Messal, Laran, and Simpson, 1988, Development 103: 757-67; 8 = Simpson and Carteret, 1989, Development 106: 57-66; 9 = Simpson, El Messal, Moscoso del Prado, and Ripoll, 1988, Development 103: 391-401.

^β MZI = maternal-zygotic interaction; demonstrated in males carrying mutant allele from heterozygous mother and variegating allele on a paternally derived duplication (Robbins, 1983, Genetics 103: 663-48).

^γ Spontaneous in the paternal X chromosome of a cross between wild-type males and *mei9* females, such that the F1 females were *sgg/mei9*.

cytology: Placed in 3B1 based on its inclusion in *Df(1)64fl* = *Df(1)3A9-B1;3B2-3* but not *Df(1)62d18* = *Df(1)3B1-2;3C6-7*.

molecular biology: Cloning and sequencing indicates gene product to be homologous to serine-threonine proteases (Siegfried, Perkins, Capaci, and Perrimon, 1990, Nature 345: 825-29).

Sgs: Salivary gland secretion proteins

A group of seven genes encoding proteins that are components of the secretion produced by the larval salivary glands during the third instar for the purpose of attaching the larva to the substrate preparative to pupariation. Initiation of transcription of these genes is coincident with the formation of the intermolt puffs in early to mid third instar (Korge, 1975, Proc. Nat. Acad. Sci. USA 72: 4550-54). The genes colocalize with the most prominent intermolt puffs. Synthesis of the glue proteins begins about 106 h after egg deposition and ceases abruptly within a few minutes after the glue is released 14 h later (Beckendorf and Kafatos, 1976, Cell 9: 365-73). Initiation of transcription, but not of intermolt-puff formation seems to depend on the presence of suitable levels of ecdysterone in early third instar larvae (Hansson and Lambertsson, 1983, Mol. Gen. Genet. 192: 395-401). The subsequent cessation of transcription and puff

regression also ecdysterone dependent (Crowley and Meyerowitz, 1984, Dev. Biol. 102: 110-21). Loci designated *Sgs1* and *Sgs3* through *Sgs8* in order of increasing electrophoretic mobility, except that *Sgs6* is out of order.

Sgs1

location: 2-13.9 (based on 64 *dp-cl* recombinants).

references: Velissariou and Ashburner, 1980, Chromosoma 77: 13-27.

phenotype: Structural gene for the most slowly migrating of the salivary-gland glue proteins, SGS1 (Zhimulev and Kolesnikov, 1975, Wilhelm Roux's Arch. Entwicklungsmech. Org. 178: 15-28).

alleles: Nine electrophoretic variants designated *Sgs1^a* through *Sgs1ⁱ*; no naturally occurring null alleles recorded.

cytology: Placed in region 25A2-D2 based on increased product in salivary glands of larvae carrying the duplication produced by combining elements of *T(Y;2;4)J96* = *T(Y;2;4)25A2-3* and *T(Y;2)D110* = *T(Y;2)25D1-2*. Associated with the intermolt puff in 25B3-7, which comes up coincidentally with the other glue-protein puffs.

Sgs3

location: 3-35.0 (based on 100 *se-ri* recombinants; Kokoza, Kazakova, Karakin, 1982, DIS 58: 94-95).

references: Korge, 1975, Proc. Nat. Acad. Sci. USA 72: 4550-54.

Akam, Roberts, Richards, and Ashburner, 1978, Cell 13: 215-25.

Meyerowitz and Hogness, 1982, Cell 28: 165-76.

Garfinkle, Pruitt, and Meyerowitz, 1983, J. Mol. Biol. 168: 765-89.

phenotype: The structural gene for glue protein, SGS3. Dependence of initiation and cessation of *Sgs3* expression on ecdysterone levels studied by Crowley and Meyerowitz (1984, Dev. Biol. 102: 110-21). Expression of *Sgs3* as well as *Sgs7* and *Sgs8* does not take place in the presence of the nonpupariating lethal mutation, *npr*, which is a member of the Broad Complex, *BRC*; expression cannot be rescued by the administration of ecdysterone; *npr* does not inhibit formation of intermolt puff 67C, thus dissociating transcription from puff formation (Crowley, Mathers, and Meyerowitz, 1984, Cell 39: 149-56).

alleles: Four electrophoretic variants designated *Sgs3^a* through *Sgs3^d* identified by Akam *et al.*. In addition the allele from the Formosa wild strain produces a smaller polypeptide than other alleles.

cytology: Placed in 68C3-5 by in situ hybridization; associated with intermolt puff at 68C (Korge; Akam *et al.*).

molecular biology: Sequence recovered as a third-instar-salivary-gland-specific cDNA (Wolfner, 1980, PhD thesis, Stanford University). Hybridizes to an mRNA 1120 nucleotides in length (in Oregon R; smaller in some other strains; *e.g.*, Formosa). Nucleotide sequence and conceptual amino acid sequence of translation product of cDNA clone determined by Garfinkle *et al.*; 6751 nucleotides of genomic DNA, containing the coding sequences of the cluster, *Sgs3*, *Sgs7*, and *Sgs8*, were sequenced; The sequence encoding *Sgs3* extends from approximately nucleotide 4457 to 5646, with an intervening sequence of 73 nucleotides from nucleotides 4514 to 4586, between the first and second nucleotides of codon 10. The conceptual amino-acid sequence comprises three domains;

the first 23 amino acids are rich in hydrophobic residues and constitute a signal peptide which is removed from the mature polypeptide; the next 234 amino acids contain an amino terminal 49 amino acids of threonine-rich sequences followed by 185 amino acids that are composed entirely of 37 tandem repeats of minor variants of a five amino acid unit (basic unit = Pro-Thr-Thr-Thr-Lys); twenty of these repeats are deleted in the Formosa wild-type allele (Mettling, Bourouis, and Richards, 1985, Mol. Gen. Genet. 201: 265-68); the carboxy-terminal fifty amino acids of SGS3 are similar to those found in SGS7 and SGS8. *i.e.*, 19 identities including eight cysteines. The SGS3 polypeptide is heavily glycosylated (Beckendorf and Kafatos, 1976, Cell 9: 365-73), probably on the threonine residues (Garfinkle *et.al.*). Transformation experiments show that as little as 2.3 kb of 5' plus 1.1 kb of 3' sequence is sufficient for normal regulation of *Sgs3* expression (Crosby and Meyerowitz, 1986, Dev. Biol. 118: 593-607); 980 base pairs of 5' sequence allow for appropriate stage and tissue specificity of expression, but the levels of expression are greatly reduced; 27 5' base pairs are insufficient for *Sgs3* expression (Bourouis and Richards, 1985, Cell 40: 349-57). Finally, functional *Sgs7 Sgs8 Sgs3* inserts may form ectopic intermolt puffs, but they need not, thus separating puff formation from *Sgs* expression (Crosby and Meyerowitz).

Sgs4

location: 1-3.0 (based on 97 *w^a-rb* recombinants; Kokoza, Kazakova, and Karakin, 1982, DIS 58: 94-95).

references: Korge, 1977, Chromosoma 62: 155-74.

phenotype: Encodes the salivary-gland glue protein, SGS4; this protein not required for viability, since non producers eclose normally; it varies in size owing to a variable number of copies of a heptapeptide repeat in an N-terminal tandem array. Expression of the virtually inactive allele, *Sgs4^H* increased four fold in trans heterozygotes with the normally active allele, *Sgs4^{OR}*, and nine fold with a tandem duplication [*Dp(1;1)Co*] containing two copies of a normally active allele; neither *Sgs4^H/Sgs4^{Ber}* nor *Sgs4^{Ber}/Sgs4^{OR}* exhibits enhanced activity; enhancement decreased by disruption in pairing, *e.g.*, in *Sgs4^H/FM6* (Kornher and Brutlag, 1986, Cell 44: 879-83).

alleles: So-called electrophoretic alleles, which are probably size variants, have been designated *Sgs4^a*, *Sgs4^b*, *Sgs4^c*, and *Sgs4^d* (see Korge, 1977); the correspondence between these designations and those tabulated below is not available.

allele	origin	ref ^α	express- ion (%) ^β	repeat number ^γ	5' sequences ^δ
<i>Sgs4^{Ber}</i>	Berkeley	1, 2	0	22	-486 to -444 and -440 to -392 deleted
<i>Sgs4^{Ch}</i>	Chieti	2	100	22	
<i>Sgs4^{CS}</i>	Canton-S	1, 2	40	21	C→T at -344
<i>Sgs4^{D232}</i>	Davis	1, 2	40	34	C→T at -344
<i>Sgs4^{Dk}</i>	Daekwan- ryeong	1	<1		9 single base-pair alterations
<i>Sgs4^H</i>	Hikone	2	~2	31	-356 to -305 deleted
<i>Sgs4^K</i>	Kochi	2	~2	101	
<i>Sgs4^{OR}</i>	Oregon-R	2	100	19	
<i>Sgs4^S</i>	Seto	2	~2	28	-356 to -305 deleted
<i>Sgs4^U</i>	Urbana	2	100	27	

^α 1 = McGinnis, Shermoen, Heemskirk, and Beckendorf, 1983, Proc. Nat. Acad. Sci. USA 80: 1063-67; 2 = Muskavitch and Hogness,

1980, Cell 29: 1041-51.

^β mRNA level compared to Oregon-R.

^γ The number of 21-base-pair repeats about 100 nucleotides downstream from the 5' end of coding sequence = seven-amino-acid tandem repeats near the amino-terminal end of polypeptide.

^δ Comparison of upstream sequences with those of Oregon R; nucleotide 1 is the first base transcribed, the first T of an *EcoRI* restriction site.

cytology: Placed in 3C11-12 based on its inclusion in *Df(1)N-69h9* = *Df(1)3C6;3D1* but not *Df(1)N-5419* = *Df(1)3C5-6;3C10-11*; activity associated with formation of intermolt puff in same bands (Korge, 1977, Chromosoma 62: 155-74).

molecular biology: *Sgs4* resides within the first intron of *dnc* (Chen, Malone, Beckendorf, and Davis, 1984, Nature 329: 721-24). Transcription is from left to right. The entire *Sgs4* region cloned and the gene and its flanking regions sequenced (Muskavitch and Hogness, 1980, Proc. Nat. Acad. Sci. USA 77: 7362-66; 1982, Cell 29: 1041-51); gene contains no introns and is characterized by a tandem array of 21-base-pair repeats occupying the 5' 45% of the sequence. The repeating unit appears to be (Arg or Thr)-Cys-(Glu, Lys, or Arg)-Thr-Glu-Pro-Pro. The number of repeats is uncorrelated with gene activity; rather regions some 300 to 500 base pairs upstream of the start of transcription are abnormal or deleted in alleles with little or no activity. Fifteen transpositions of *Sgs4* with 2.6 kb of 5' and 1.3 kb of 3' sequences display normal expression with respect to quantity, stage and tissue specificity, and dosage compensation but not puff formation (Krumm, Roth, and Korge, 1985, Proc. Nat. Acad. Sci. USA 82: 5055-59); reduction of the 5' flanking sequences to -840 but not to -392 retains normal regulation (McNabb and Beckendorf, 1986, EMBO J. 5: 2331-40).

Sgs5

location: 3- {60}.

references: Guild, 1984, Dev. Biol. 102: 462-70.

Guild and Shore, 1984, J. Mol. Biol. 179: 289-314.

Shore and Guild, 1986, J. Mol. Biol. 190: 149-58.

Shore and Guild, 1987 Genes Dev. 1: 829-39.

phenotype: The structural gene for the salivary-gland glue protein, SGS5, a polypeptide of 163 amino-acid residues, 13% of which are serine and threonine. SGS5 lightly glycosylated (Beckendorf and Kafatos, 1976, Cell 9: 365-73). The first 18 amino acids are highly hydrophobic as expected for a signal peptide.

cytology: Located in 90B3-8 by *in situ* hybridization; associated with the intermolt puff at 90B (Wolfner, 1980, PhD Thesis, Stanford University, Stanford, CA).

alleles: One null allele, *Sgs5^{nl}*, found in a strain designated C2 from Wallace.

molecular biology: Genomic clone isolated; 1 kb including the *Sgs5* transcription unit sequenced; extends from nucleotide -173 to +839, with the transcribed region beginning at +1 and ending at the site of poly(A) addition at +769. Upstream sequences resemble those of other *Sgs* genes; information necessary for *Sgs5* expression contained within 109 base pairs upstream and 69 base pairs downstream of the transcribed region. Two introns, one extending from +302 to +357 and the other from +547 to +605; both introns interrupt a proline codon after the first nucleotide. *Sgs5^{nl}* differs from wild type in seven base-pair substitutions between nucleotides -84 and

+75.

Sgs6**location:** 3-42.0 (based on 42 *h-th* recombinants).**references:** Velissariou and Ashburner, 1981, Chromosoma 84: 173-85.**phenotype:** A component of the glue-protein mixture that is not present in all stocks. Electrophoretic mobility between that of SGS1 and SGS3, close to SGS3.**cytology:** Placed in 71C1-F5 based on dosage studies with segmental aneuploids involving $T(Y;3)A60 = T(Y;3)71C1-2$ and $T(Y;3)B99 = T(Y;3)71F3-5$. Associated with intermolt puff at 71C3-4, which is absent in flies lacking SGS6.**Sgs7****location:** 3-35.0.**references:** Crowley, Bond, and Meyerowitz, 1983, Mol. Cell. Biol. 3: 623-34.**phenotype:** Encodes the 5481 kd salivary-gland glue protein, SGS7; the protein contains, in common with SGS3 and SGS8, a 23-residue amino-terminal signal peptide and a carboxy-terminal region of about 50 amino acids that shows considerable homology with that of SGS3 and SGS8; it is not glycosylated. Expressed coordinately with *Sgs3* and *Sgs8*.**cytology:** Placed in 68E3-5 by *in situ* hybridization.**molecular biology:** Cloned by Meyerowitz and Hogness (1982, Cell 28: 165-76); located approximately 2 kb distal to *Sgs3* and 500 base pairs to the right of *Sgs8*; transcribed from left to right, as is *Sgs3*. Has a small intervening sequence between the first and second bases of the tenth codon; mRNA contains 320 nucleotides. Expression absent in *su(f)^{ts67g}* (rescuable by administration of ecdysterone) (Hansson and Lambertsson, 1983, Mol. Gen. Genet. 192: 395-401) and in *npr* flies (not rescued by ecdysterone) (Crowley, Mathers, and Meyerowitz, 1984, Cell 39: 149-56).**Sgs8****location:** 3-35.0.**references:** Crowley, Bond, and Meyerowitz, 1983, Mol. Cell. Biol. 3: 623-34.**phenotype:** Encodes the 5592 kd salivary-gland glue protein, SGS7; the protein contains, in common with SGS3 and SGS7, a 23-residue amino-terminal signal peptide and a carboxy-terminal region of about 50 amino acids that shows considerable homology with that of SGS3 and SGS8; it is not glycosylated. Expressed coordinately with *Sgs3* and *Sgs7*.**cytology:** Placed in 68E3-5 by *in situ* hybridization.**molecular biology:** Cloned by Meyerowitz and Hogness (1982, Cell 28: 165-76); located approximately 500 base pairs to the left of *Sgs7*; transcribed from right to left from the opposite strand transcribed by *Sgs7*. Has a small intervening sequence between the first and second bases of the tenth codon; mRNA contains 360 nucleotides. Expression absent in *su(f)^{ts67g}* (rescuable by administration of ecdysterone) (Hansson and Lambertsson, 1983, Mol. Gen. Genet. 192: 395-401) and in *npr* flies (not rescued by ecdysterone) (Crowley, Mathers, and Meyerowitz, 1984, Cell 39: 149-56).**Sgs9****location:** 3-60.8 (based on eight *bx-sr* recombinants).**synonym:** *GP5*.**references:** Hoshizaki, Dlott, Joslyn, and Beckendorf, 1987, Genet. Res. 49: 111-19.**phenotype:** Controls the quantity of SGS9 (P5 of Beckendorf and Kafatos, 1976, Cell 9: 365-73), which migrates ahead of SGS5 in SDS polyacrylamide gel electrophoresis. Not demonstrated to encode SGS9. Protein levels inordinately sensitive to background genotype.**alleles:** A null allele exists in a stock labeled Stromsvreten 10; used in mapping experiments.**cytology:** Genetic mapping places it close to *Sgs5* and the intermolt puff at 90 B-C.**sh: short winged****location:** 3-56.**origin:** Spontaneous.**discoverer:** Bridges, 23d3.**synonym:** *short wing*.**references:** Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 235. 1935, DIS 3: 16.**phenotype:** Wings small; similar to *dy*. Appears to be epistatic to *vg* in that *vg sh* flies have *sh* phenotype; by same argument *px* epistatic to *sh* (Rudy and Duffy, 1972, J. Hered. 62: 387-88). RK2.***sh-5: short-5****location:** 3-93.2 (Koliantz, 1969, DIS 44: 52).**origin:** Spontaneous.**discoverer:** Spencer, 26j.**references:** 1934, DIS 1: 35.

1935, Am. Naturalist 69: 223-38.

phenotype: Wing veins L5 and L3 short and do not reach wing margin. Expression variable; overlaps wild type. RK3.**Sh: Shaker (M. Tanouye)****location:** 1-57.6.**origin:** X ray induced.**discoverer:** Catsch, 1944.**references:** Catsch, 1944, Z. Indukt. Abstamm. Vererbungslehre 82: 64-66.

Trout and Kaplan, 1973, J. Neurobiol. 4: 495-512.

Jan, Jan, and Dennis, 1977, Proc. R. Soc. London Ser. B 198: 87-108.

Tanouye, Ferrus, and Fujita, 1981, Proc. Nat. Acad. Sci. USA 78: 6548-52.

Salkoff and Wyman, 1981, Nature (London) 293: 228-30.

Tanouye, Kamb, Iverson, and Salkoff, 1986, Ann. Rev. Neurosci. 9: 255-76.

phenotype: Under moderate ether anesthesia, legs shake abnormally, antennae twitch, abdomen pulsates; wings scissor in some alleles; very little effect in deeply etherized flies; unetherized mutants twitch and shudder occasionally; severed legs shake (Kaplan and Trout, 1969, Genetics 61: 399-409; Trout and Kaplan, 1973; Tanouye, Ferrus, and Fujita, 1981; Ganetzky and Wu, 1982a, Genetics 100: 597-614; Tanouye and Ferrus, 1985, J. Neurogenet. 2: 253-71). Structural gene for several types of potassium channel (Iverson, Tanouye, Lester, Davidson, and Rudy, 1988, Proc. Nat. Acad. Sci. USA 85: 5723-27; Timpe, Schwarz, Tempel, Papazian,

Jan, and Jan, 1988, *Nature* 331: 143-45). Abnormal action potential repolarization of adult giant fiber; repetitive firing of action potentials in larval nerves; prolonged transmitter release at larval neuromuscular junction (Jan, Jan, and Dennis, 1977; Tanouye, Ferrus, and Fujita, 1981; Ganetzky and Wu, 1982b, *J. Neurophysiol.* 47: 501-14; Tanouye and Ferrus, 1985). Abnormal in one class of potassium channel (A channel) present in embryonic myocytes, larval and pupal muscle (Salkoff and Wyman, 1981; Salkoff, 1983, Cold Spring Harbor Symp. Quant. Biol. 48: 221-31; Wu and Haugland, 1985, *J. Neurosci.* 5: 2626-40; Timpe and Jan, 1987, *J. Neurosci.* 7: 1307-17; Haugland and Wu, 1990, *J. Neurosci.*). *Sh* mutations do not affect four other distinct potassium-channel types (K_D , K_1 , A_2 , Calcium-gated) (Salkoff and Wyman, 1981; Salkoff, 1983, *Nature* 302: 249-51; Wu, Ganetzky, Haugland, and Liu, 1983, *Science* 220: 1076-78; Solc, Zagotta, and Aldrich, 1987, *Science* 236: 1094-98; Solc and Aldrich, 1988, *J. Neurosci.* 8: 2556-70). Males carrying hemizygous deletions of *Sh* are viable (Tanouye, Ferrus, and Fujita, 1981). Abnormal associative learning in some paradigms (Tully); activity patterns high, but show normal circadian rhythmicity (Konopka). RK1.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>*Sh 1</i>	X ray	Catsch		1	1
<i>*Sh 2</i>	X ray	Novitski		2	1
<i>*Sh 3</i>	X ray	Novitski		2	1
<i>*Sh 4</i>	CB3025	Fahmy		2	
<i>Sh 5</i>	EMS	Kaplan		6, 7, 9, 12, 14	4
<i>Sh 6</i>	EMS	Homyk	<i>Sh</i> ¹⁰¹	4	1
<i>Sh 7</i>	EMS	Homyk	<i>Sh</i> ¹⁰²	4, 12, 13, 14	4
<i>Sh 8</i>	X ray	Merriam	<i>Sh</i> ^{B55}	10, 12	3; <i>T(1;Y)B55</i>
<i>Sh 9</i>	EMS	Ferrus/Ganetzky	<i>Sh</i> ^{E62}	3, 5, 12, 13	4
<i>Sh 10</i>	P	Kreber/Ganetzky	<i>Sh</i> ^{HD1}	8	1
<i>Sh 11</i>	γ ray	Kreber	<i>Sh</i> ^{K82a}	3, 8, 13	2
<i>Sh 12</i>	γ ray	Kreber	<i>Sh</i> ^{K82b}	8, 13	2
<i>Sh 13</i>	P	Kreber	<i>Sh</i> ^{K83f}	8, 13	2
<i>Sh 14</i>	EMS	Searcy	<i>Sh</i> ^{KS133}	6, 10, 12	4
<i>Sh 15</i>	EMS	Crosby	<i>Sh</i> ^{LC}	12, 13	3; <i>T(1;3)16F1-2;80</i>
<i>Sh 16</i>	spont.	Ganetzky	<i>Sh</i> ^M	11, 14	4
<i>Sh 17</i>	γ ray	Paulus	<i>Sh</i> ^P	8, 13	2
<i>Sh 18</i>	γ ray	Paulus	<i>Sh</i> ^{RP1}	8	1
<i>Sh 19</i>	γ ray	Paulus	<i>Sh</i> ^{RP2}	8	1
<i>Sh 20</i>	γ ray	Paulus	<i>Sh</i> ^{RP2}	8	1
<i>Sh 21</i>	EMS	O'Hara	<i>Sh</i> ^{rKO120}	3, 6, 9, 12, 14	4
<i>Sh 22</i>	spont.	Barbel	<i>Sh</i> ^{SB2}	5, 13	4
<i>Sh 23</i>	P	Barbel	<i>Sh</i> ^{SB3}	5, 13	4
<i>Sh 24</i>	spont.	Barbel	<i>Sh</i> ^{SB13}	5, 13	4
<i>Sh 25</i>	X ray	Merriam	<i>Sh</i> ^{W32}	10, 12	3; <i>T(1;Y)16F3-6</i>
<i>Sh 26</i>	spont.	Kreber	<i>Sh</i> ^X	8	1

^α 1 = Catsch, 1944, *Z. Indukt. Abstamm. Vererbungsl.* 82: 64-66; 2 = CP627; 3 = Haugland and Wu, 1990, *J. Neurosci.*; 4 = Homyk, 1977, *Genetics* 87: 105-28; 5 = Jan, Barbel, Timpe, Laffer, Salkoff, O'Farrell, and Jan, 1983, Cold Spring Harbor Symp. Quant. Biol. 48: 233-45; 6 = Jan, Jan, and Dennis, 1977, *Proc. R. Soc. London Ser. B* 198: 87-108; 7 = Kaplan and Trout, 1969, *Genetics* 61: 399-409; Trout and Kaplan, 1973, *J. Neurobiol.* 4: 495-512; 8 = Kreber and Ganetzky; 9 = Salkoff and Wyman, 1981, *Nature (London)* 293: 228-30; 10 = Salkoff, 1983, Cold Spring Harbor Symp. Quant. Biol. 48: 221-31; 11 = Tanouye and Ferrus, 1985, *J. Neurogenet.* 2: 253-71; 12 = Tanouye, Ferrus, and Fujita, 1981, *Proc. Nat. Acad. Sci. USA* 78: 6548-52; 13 = Timpe and Jan, 1987, *J. Neurosci.* 7: 1307-17; 14 = Wu and Haugland, 1985, *J. Neurosci.* 5: 2626-40.

^β *Sh* phenotype has been assessed in four ways: (a) abnormal leg shaking under ether anesthesia; (b) abnormal A-type potassium currents in larval muscle and/or pupal flight muscle; (c) abnormal action potentials in the adult cervical giant fiber; and (d) abnormal synaptic transmission at the larval neuromuscular junction and multiple firing of larval motoneurons. Numbers above indicate the tests that have been performed on each allele: 1 = a; 2 = a + b; 3 = a + b + c; and 4 = a + b + c + d. All tests performed produced mutant results.

cytology: Located in 16F1-8 (Tanouye, Ferrus, and Fujita, 1981). Associated with *T(1;Y)B55 = T(1;Y)16F1-4*, with *T(1;3)Sh15 = T(1;3)16F1-2;80*, and with *T(1;Y)W32 = T(1;Y)16F3-6*; covered by *Dp(1;3)16E2-4;17A-B,99D*.

molecular biology: Structural gene for a potassium channel cloned by chromosomal walking; large transcription unit spans >110 kb of chromosomal DNA with multiple transcripts generated by differential splicing; at least twelve different transcripts formed from at least 25 different exons; all transcripts contain a conserved central portion (863 bp) built from six common exons; the transcripts contain variable 5' and 3' domains; Northern blots show a heterogeneous pattern of transcripts with a broad band of 5.5-6.5 kb and major bands at 7.8, 8.5, and 9.5 kb (Kamb, Iverson, and Tanouye, 1987, *Cell* 50: 405-13; Baumann, Krah-Jentgens, Mueller, Mueller-Holtkamp, Seidel, Kecskemethy, Casal, Ferrus, and Pongs, 1987, *EMBO J.* 6: 3419-29; Papazian, Schwarz, Tempel, Jan, and Jan, 1987, *Science* 237: 749-53; Tempel, Papazian, Schwarz, Jan, and Jan, 1987, *Science* 237: 770-75; Kamb, Tseng-Crank, and Tanouye, 1988, *Neuron* 1: 421-30; Pongs, Kecskemethy, Muller, Krah-Jentgens, Baumann, Kiltz, Canal, Llamazares, and Ferrus, 1988, *EMBO J.* 7: 1087-97; Schwarz, Tempel, Papazian, Jan, and Jan, 1988, *Nature (London)* 331: 137-42).

Two forms of gene product are deduced: a small-protein form with three hydrophobic segments has unknown function; a larger form with six potential membrane-spanning segments has potassium channel function as demonstrated electrophysiologically by expression in *Xenopus* oocytes, mammalian cell lines, and insect cell lines; at least six different transcript variants express potassium channels with different physiological properties; the channel may be a tetramer formed from four *Sh* subunits; a heteromultimer may be indicated from results on heterozygous combinations and coinjections into *Xenopus* oocytes; conserved region may be responsible for voltage dependence of activation, voltage dependence of inactivation, potassium selectivity, and toxin sensitivity which are similar among all products; variable amino termini are responsible for different potassium channel inactivation rates; variable carboxyl termini are responsible for differences in inactivation recovery rates; a current loss in myotubes may be rescued by germline transformation (Iverson, Tanouye, Lester, Davidson, and Rudy, 1988; Timpe, Schwarz, Tempel, Papazian, Jan, and Jan, 1988; Timpe, Jan, and Jan, 1988, *Neuron* 1: 659-67; Leonard, Karschin, Jayashree-Aiyar, Davidson, Tanouye, Thomas, Thomas, and Lester, 1989, *Proc. Nat. Acad. Sci. USA* 86: 7629-33; Zagotta, Geraaad, Garber, and Aldrich, 1989, *Soc. Neurosci. Abstracts* 15: 338; Iverson and Rudy, 1990, *J. Neurosci.*; Wu and Haugland, 1990; Iverson and Rudy; Stuhmer; Isakoff; Miller).

An S4 motif, consisting of 7 repeats of the triplet, R-X-X, where R is sometimes replaced by K, and X is a

hydrophobic residue, is present in all *Sh* channels and is highly conserved in virtually all other known voltage-gated ion channels; mutagenesis of S4 results in alterations of voltage sensitivity; the motif is thought to be a voltage-sensor (Tempel, Papazian, Schwarz, Jan, and Jan, 1987; Kamb, Tseng-Crank, and Tanouye, 1988; Pongs, Kecskemethy, Muller, Krah-Jentgens, Baumann, Kiltz, Canal, Llamazares, and Ferrus, 1988; Papazian, Timpe, Jan, and Jan, 1989, Soc. Neurosci. Abstracts 15: 337). A leucine-heptad repeat located adjacent to the S4 motif may participate in the channel gate; mutagenesis causes alterations in voltage sensitivity and subunit interactions (McCormack, Campanelli, Ramaswami, Mathew, Tanouye, Iverson, and Rudy, 1989, Nature 340: 103; McCormack, Ramaswami, Mathew, Tanouye, Iverson, McCormack, Rudy, 1989, Soc. Neurosci. Abstracts 15: 337).

A truncated product that acts as an antimorph is associated with *Sh*⁷; an amino acid substitution is associated with the *Sh*¹⁴ product, which acts as an antimorph; an amino acid substitution in the leucine-heptad repeat motif is associated with the *Sh*⁵ product; an alteration of a splice site is associated with the *Sh*⁹ mutation; *Sh*¹⁶ contains an insertion into coding sequence (Kamb, Iverson, Tanouye, 1983; Gisselmann, Sewing, Madsen, Mallart, Angaut-Petit, Mueller-Holtkamp, Ferrus, and Pongs, 1989, EMBO J. 8: 2359-64; Pongs; Gautam and Tanouye). The *Sh* product is highly homologous to three other potassium channel genes: *Shab*, *Shal*, and *Shaw*, isolated by DNA crosshybridization using *Sh* probes (Butler, Wei, Baker, and Salkoff, 1989, Science 243: 943-47).

Tissue *in situ* hybridization shows *Sh* transcript in the cell bodies of retina, optic lobe, central brain, thoracic ganglion, and muscle; antibody studies show a major polypeptide of about 70-80 kd; differential temporal and spatial expression may be indicated (Pongs, Kecskemethy, Muller, Krah-Jentgens, Baumann, Kiltz, Canal, Llamazares, and Ferrus, 1988; Tseng-Crank and Tanouye; Schwarz, Papazian, Carretto, Jan, and Jan, 1988, Soc. Neurosci. Abstracts 14: 454).

allele	molecular biology ^α	ref ^β
<i>Sh</i> ⁵	phe ³⁷¹ to ile	1, 2
<i>Sh</i> ⁷	trp ⁴⁰⁴ to stop	1, 3
<i>Sh</i> ⁸	break at 33.4 to 34.9	4, 5, 6
<i>Sh</i> ⁹	splicing defect at gly ⁴⁸⁰	2
<i>Sh</i> ¹¹	break at 61	4
<i>Sh</i> ¹³	insertion at 44	4
<i>Sh</i> ¹⁴	val ⁴¹³ to asp	2
<i>Sh</i> ¹⁵	break at 54.1 to 59.1	4, 5, 6
<i>Sh</i> ¹⁶	insertion at 45.5 to 46.1	5
<i>Sh</i> ²⁵	break at 95.2 to 98.7	4, 5, 6
<i>T(1;Y)B27</i>	break at -22 to -21	6

^α Locations are relative to the *Sh* chromosomal walk in kb (Kamb, Iverson, and Tanouye, 1987). Amino acid changes are relative to the deduced H37 protein (Kamb, Tseng-Crank, and Tanouye, 1988).

^β 1 = Gautam and Tanouye; 2 = Pongs; 3 = Gisselmann, Sewing, Madsen, Mallart, Angaut-Petit, Muller-Holtkamp, Ferrus, and Pongs, 1989, EMBO J. 8: 2359-64; 4 = Papazian, Schwarz, Tempel, Jan, and Jan, 1987, Science 237: 749-53; 5 = Kamb, Iverson, and Tanouye, 1987, Cell 50: 405-13; 6 = Baumann, Krah-Jentgens, Mueller, Mueller-Holtkamp, Seidel, Kecskemethy, Casal, Ferrus, and Pongs, 1987, EMBO J. 6: 3419-29.

other information: Some *Sh* phenotypes are suppressed by *nap* and *para* alleles; *i.e.*, in double mutant combinations,

abnormal leg-shaking, repetitive firing of larval action potentials, and transmitter release at larval neuromuscular junction are nearly normal; the interactions are not allele-specific (Ganetzky and Wu, 1982a,b). Some *Sh* phenotypes are enhanced by *eag*; *i.e.*, in double mutant combinations, abnormal leg-shaking, repetitive firing of larval action potentials, and transmitter release are more extreme; also, adults have down-turned wings, and dented-in thoraces at the sites of the dorsal longitudinal muscle insertions; the interactions are not allele-specific (Ganetzky and Wu, 1983, J. Neurogenet. 1: 17-28). Some *Sh* phenotypes are enhanced by *dnc*; *i.e.*, in double mutant combinations, abnormal leg-shaking is more extreme; abnormal spontaneous activity is seen in the giant fiber (Ferrus and Tanouye). The breakpoint of *T(1;3)B27 = T(1;3)16E3-5;36D-F*, induced in a *Sh*¹⁴ background, causes an alteration in the pattern of leg-shaking (Tanouye and Ferrus).

sha: shavenoid

location: 2-62.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82. Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Trichomes missing or very short. Flies cannot fly or walk on glass. In larvae, the number of denticles is reduced with remaining denticles thin and bent. Hairs are largely absent, but sensory hairs not affected. Autonomous in nuclear transplants. Causes disoriented and abbreviated hairs on larval cuticle; useful as a larval-cuticle marker (Struhl and Lawrence, 1982, Cell 31: 285-92).

alleles: *sha*¹ and *sha*² (originally designated *IN* and *IIP*) plus two discarded alleles.

cytology: Placed in 47E3-48A6 based on its location in the area of overlap between *Df(2R)en-A = Df(2R)47D3; 48A5-6* and *Df(2R)en-B = Df(2R)47E3;48B2*.

Shab: Shaker cognate b (J.C. Hall)

location: 3- {15}.

origin: Isolated by screening a cDNA library with a *Sh* cDNA probe.

references: Butler, Wei, Baker, and Salkoff, 1989, Science 243: 943-47.

cytology: Mapped to 63A by *in situ* hybridization.

phenotype: Expression in *Xenopus* oocytes reveals a delayed-rectifier type of potassium current expressed by *Shab*; the rate of current activation in *Shab* is somewhat faster than in *Shaw*.

molecular biology: Isolated by screening a cDNA library with a *Sh* cDNA probe containing all of the presumptive membrane-spanning domains, which form the core of the potassium channel proteins encoded by *Sh*, and which thus very likely characterize the *Shab* gene product as well. Sequencing of six *Shab* cDNAs identifies alternatively spliced mRNAs from this locus; this involves, at least in part, the coding portion of the gene (*e.g.*, one cDNA contains an additional exon not found in two others). Sequencing data reveal, in addition to homologies to *Sh*, as expected, homologies to two other *Sh*-type genes, *Shaw* and *Shal*. Similarities among products of these four genes are greater than those between *D. melanogaster*'s *Sh* gene family and sodium or calcium channel proteins. *Shab* shares opa (poly-Gln) repeats with some of the alternatively spliced *Sh* products (rela-

tively near N- and C-termini of the respective proteins), and there are possible cAMP-dependent phosphorylation sites in one of the corresponding regions of the two gene products (C-terminal to the intramembrane portions of the proteins); in *Shab*, but not *Shaw* conceptual protein, there is a large sequence N-terminal to the first membrane-spanning region, which includes Ser-Gly and Thr-Gly repeats (possible sites of O-linked glycosylation); the "S4" region of *Shab*, which is the presumed voltage sensor of the channel, contains a string of five positive charges (*vs.* seven and four in *Sh* and *Shaw* proteins, respectively). *Shab* cDNA probes found to detect two late embryonic transcripts, 4.3 and 6.8 kb, the latter being more abundant; in pupae, relative abundances of these two switch; these RNA species not detected in adults.

other information: A rat potassium channel gene (*drkl*), isolated by expression cloning in a frog oocyte system, has greater sequence similarity to *Shab* than to *Sh* (Frech, VanDongen, Schuster, Brown, and Joho, 1989, *Nature* (London) 340: 642-645); *drkl* is not the same as two other mammalian K⁺ channel genes, cloned by homology to *Sh*.

shade: see *shd*

shadow: see *sad*

shaggy: see *sgg*

shakA: shaking A (J.C. Hall)

location: 1-38.2 (Homyk).

origin: Induced by ethyl methanesulfonate.

discoverer: Homyk.

references: Homyk, Szidonya, and Suzuki, 1980, *Mol. Gen. Genet.* 177: 553-65.

phenotype: Vigorous shaking of legs when flies under ether; both mutant alleles cause temperature-sensitive phenotypes: *shakA*¹ exhibits generally hyperactive behavior (irrespective of anesthetic) when raised at 22°; after rearing at 29°, flying and jumping abilities severely subnormal, and males show abnormal wing usage in courtship; *shakA*² causes lethality when raised at 29°; after rearing at 22°, adults are hypoactive and uncoordinated.

alleles: Two alleles, *shakA*¹ and *shakA*².

shakB: shaking B (J.C. Hall)

location: 1-64.

synonym: *nj-156: non-jumper; pas: passover.*

references: Homyk, Szidonya, and Suzuki, 1980, *Mol. Gen. Genet.* 177: 553-65.

Thomas, 1980, *Neurosci. Abstr.* 6: 742.

Wyman and Thomas, 1983, *Cold Spring Harbor Symp. Quant. Biol.* 48: 641-52.

Thomas and Wyman, 1984, *J. Neurosci.* 4: 530-38.

Baird and Hillis, 1985, *Neurosci. Abstr.* 11: 627.

Baird, 1986, *Neurosci. Abstr.* 12: 1164.

Miklos, Kelly, Leeds, and Lefevre, 1987, *J. Neurogenet.* 4: 1-19.

Perrimon, Engstrom, and Mahowald, 1989, *Genetics* 121: 313-31.

Baird, Schalet, and Wyman, 1990, *Genetics* 126: 1045-59.

phenotype: Some of the *shakB* mutants are viable but defective in their neural phenotypes as homo-, hemi-, or

heterozygotes, but other mutants are homozygous lethals that may or may not complement the viable *shakB* alleles. The viable mutants have difficulty in controlling leg movements and show leg tremors under ether anesthesia (Homyk *et al.*, 1980). They show no escape response; the flies are unable to jump into the air and fly away at a light off stimulus (Thomas, 1980; Thomas and Wyman, 1984). Unlike the mutant *Sh*, the leg tremors of *shakB* are weak and end when the legs are severed from the body (indicating a central nervous system defect).

In wild-type flies, the thoracic muscles involved in the escape response are driven by the giant fiber (GF) neuron pathway connecting the brain and thoracic ganglia. In the mutant *shakB*, the synapse between the GF axon and the postsynaptic interneuron (PSI) or between the PSI and the dorsal longitudinal muscle (DLM) seems to be defective; thus the DLM does not respond to visual stimulation by depressing the wings in flight. The synapse between the GF axon and the motor neuron of the tergotrochanter muscle (TTM) also seems to be defective, resulting in a weak response or no response from the TTM, the muscle that extends the leg in jumping. The motor neurons "pass over" the midline of the thoracic central nervous system and send aberrant branches into each contralateral mesothoracic ganglion. The abnormal neural phenotype is more pronounced if *shakB* is uncovered by a deficiency (Wyman and Thomas, 1983; Baird and Hillis, 1985; Baird *et al.*, 1990). The muscles themselves and their neuro-muscular junctions are not abnormal (Thomas and Wyman, 1984).

Viable *shakB* mutants are also characterized by electroretinogram (ERG) abnormalities; the corneal negative component is reduced and the on- and off- transients are reduced or absent. Neurons in the brain are affected, as indicated by failure of one of the superoesophageal brain commissures to fill with cobalt when the antennal nerve is backfilled (Aceves-Piña).

*shakB*³ (= *Pas*) is partially dominant to wild type in regard to the mutant's elimination of the jump response, but the other viable alleles are recessive. +/Df(1)16-3-35 and +/Df(1)A118 are behaviorally normal, but Df(1)16-3-35/Df(1)A118 females are *shakB* in phenotype.

alleles: A number of homozygous lethal alleles have been located in the *shakB* region. Six of them do not complement the *shakB* neural phenotype; two of the remainder have been tested and found to complement this neural phenotype, but do not complement the lethality of the other lethal alleles. The six noncomplementing lethals also fail to complement Df(1)16-3-35 (distal deficiency) and Df(1)A118 (proximal deficiency), while the two complementing lethals complement Df(1)A118 but not Df(1)16-3-35. 25 alleles, viables and lethals, are listed in the following table.

allele	origin	discoverer	synonym	ref ^α	comments
<i>*shakB</i> ¹	EMS	Homyk		2	viable; neurological defect
<i>shakB</i> ²	EMS	Homyk		1,2	viable; neurological defect
<i>shakB</i> ³	EMS	Thomas	<i>Pas</i>	1,9	viable; neurological defect
<i>shakB</i> ⁴	EMS	Homyk	<i>pas</i> ^{TH73}		viable; neurological defect
<i>shakB</i> ⁵	X ray	Lifschytz	<i>l(1)B220</i>	4,6	
<i>shakB</i> ^{6 β}	EMS	Lifschytz	<i>l(1)E81</i>	1,5,6,8	
<i>shakB</i> ⁷	EMS	Lifschytz	<i>l(1)P81</i>	5,7	

allele	origin	discoverer	synonym	ref ^α	comments
shakB⁸	EMS	Lifschytz	<i>l(1)P525</i>	5,7	
shakB⁹	EMS	Lifschytz	<i>l(1)Q212</i>	5,7	
shakB¹⁰	EMS	Lifschytz	<i>l(1)R-9-21</i>	5,7	
shakB¹¹ β	EMS	Lifschytz	<i>l(1)R-9-29</i>	1,5,7,8	
shakB¹²	EMS	Lifschytz	<i>l(1)R10-3</i>	5,7	
shakB¹³	EMS	Lifschytz	<i>l(1)R10-7</i>	5,7	
shakB¹⁴	EMS	Lifschytz	<i>l(1)R10-14</i>	5,7	
shakB¹⁵	EMS	Lifschytz	<i>l(1)N36</i>	5	
shakB¹⁶	EMS	Lifschytz	<i>l(1)YT7</i>	5	
shakB¹⁷	EMS	Lifschytz	<i>l(1)YT14</i>	5	
shakB¹⁸	neutrons	Muños	<i>l(1)17-96</i>	8	
shakB¹⁹ β	neutrons	Muños	<i>l(1)17-189</i>	1	
shakB²⁰ β	neutrons	Muños	<i>l(1)17-360</i>	1	
shakB²¹ γ	X ray	Lefevre	<i>l(1)L41</i>	1,4	
shakB²² β	EMS	Lefevre	<i>l(1)EC201</i>	1,5	
shakB²³	EMS	Lefevre	<i>l(1)EF481</i>	5	
shakB²⁴ γ	EMS	Lefevre	<i>l(1)EF535</i>	1,5	
shakB²⁵ β	HMS	Kramers	<i>l(1)HM437</i>	1,3	

^α 1 = Baird, Schalet, and Wyman, 1990, Genetics 126: 1045-59; 2 = Homyk, Szidonya, and Suzuki, 1980, Mol. Gen. Genet., 177: 533-65; 3 = Kramers, Schalet, Paradi, and Huizer-Hoogteyting, 1983, Mutat. Res. 107: 187-201; 4 = Lefevre, 1981, Genetics 99: 461-80; 5 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 6 = Lifschytz and Falk, 1968, Mutat. Res. 6: 235-44; 7 = Lifschytz and Falk, 1969, Mutat. Res. 8: 147-55; 8 = Schalet and Lefevre, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902; 9 = Thomas, 1980, Neurosci. Abst. 6: 742.

β Fails to complement *shakB²*.

γ Complements *shakB²*.

cytology: Placed in 19E3-4 based on the mutant neurological phenotype of heterozygotes for *Df(1)16-3-35 = Df(1)19D2-3;19E3-4* and *Df(1)A118 = Df(1)19E3-4;19E7-8* (Baird *et al.*).

Shaker cognate: see *Shab, Shal, and Shaw*

shaking: see *shak*

Shal (J.C. Hall)

location: 3- {45}.

origin: Isolated by screening a cDNA library with a *Sh* cDNA probe.

references: Butler, Wei, Baker, and Salkoff, 1989, Science 243: 943-47.

phenotype: Expression of *Shal* cDNA-derived mRNA in *Xenopus* oocytes (Wei, Covarrubias, Butler, Baker, Pak, and Salkoff, unpublished) leads to potassium currents intermediate in kinetic properties between those associated with *Sh* (using the heterologous egg system) and *Shaw* (same kind of experiment), *i.e.*, between very rapid activation/inactivation (*Sh*-encoded "A"-type channels) and quite slow kinetics (*Shaw*-encoded, delayed-rectifier-type channels).

cytology: Mapped to 76B by *in situ* hybridization.

molecular biology: Identified, as was *Shab*, via screening for *Sh*-like cDNAs; mixed probe of these two types plus a *Shaw* probe was used to isolate *Shal*. No structural details of the latter's coding sequence explicitly presented by the authors cited above, yet there are inferences about homology domains among the four known members of *Drosophila*'s *Sh* family, including *Shal*, *e.g.*, they are conserved at least insofar as each one's encoding of six similar putative membrane-spanning segments and the putative S4 voltage sensor. Also, whereas *Sh*, *Shab*, and *Shaw* conceptual proteins have strings of seven, five, and four positively charged amino acids in the presumed

gating charge region, *Shal* has five such charges.

shaven: see *svb*

shaven baby: see *svb*

shavenoid: see *sha*

Shaw (J.C. Hall)

location: 2- {10}.

origin: Isolated by screening a cDNA library with a *Shab* cDNA probe.

references: Butler, Wei, Baker, and Salkoff, 1989, Science 243: 943-47.

phenotype: In *Xenopus* oocyte mRNA injection experiment, protein encoded by *Shaw* transcripts leads to potassium currents with slow activation and inactivation kinetics (Wei, Covarrubias, Butler, Baker, Pak, and Salkoff, unpublished).

cytology: Mapped to 24B-C by *in situ* hybridization.

molecular biology: Identified by screening a cDNA library for *Sh*-like cDNAs, using a *Shab* cDNA probe. Sequencing the *Shaw* cDNA resulted in conceptual protein of approximately 500 amino acids (some 400 residues smaller than that deduced from *Shab*'s longest correctly spliced cDNA); hence *Shaw* polypeptide more similar in size to that of *Sh* than other putative potassium channel proteins; differs in length of charged amino acid string, in voltage-sensor region, from other members of the *Sh* family; for example, the position corresponding to the first (positive) gating charge in *Sh* occupied by negatively charged Asp in *Shaw* and *Shab*; *Shaw* also has a negative residue (Glu) at second position (defined by a positive amino acid in *Sh*). Northern blotting experiment using a *Shaw* cDNA probe showed one 4.9 kb transcript, which was similar in abundance in late embryos, pupae, and adults.

***shb: shortened bristles**

location: 1-39.0.

origin: Induced by S-2-chloroethylcysteine (CB. 1592).

discoverer: Fahmy, 1957.

references: 1959, DIS 33: 90.

phenotype: Bristles slightly short and thin. Wings broad, often convex or concave. Fly somewhat large. Male fertile; viability about 50% wild type. Female sterile. RK3.

shd: shade

location: 3-41.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

Roark, Mahoney, Graham, and Lengyel, 1985, Dev. Biol. 109: 476-88.

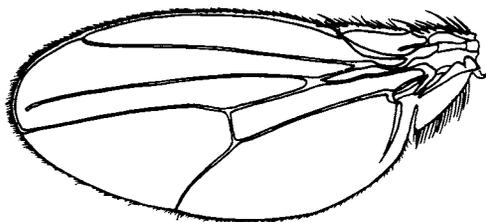
Tearle and Nüsslein-Volhard, 1987, Dis 66: 209-26.

phenotype: Embryonic lethal. No differentiation of cuticle or head skeleton.

alleles: *shd¹*, *shd²*, and *shd³* (originally designated 6J, 7C, and 19K) plus two discarded alleles; all induced by ethyl methanesulfonate.

cytology: Placed in 70D-71C; covered by *Dp(3;Y)B162 = Dp(3;Y)65E;71A-C* but not by *Dp(3;Y)G145 = Dp(3;Y)68D;70D*.

shd: see *spl*

she: sherry*location:** 3-0.**origin:** Kaliss, 36a13.**references:** 1937, DIS 8: 9.**phenotype:** Eye color sherry. Sterile *inter se* but both sexes crossfertilable. RK3.*Shell*: see *Cpl5****shf: shifted****location:** 1-17.9.**discoverer:** Bridges, 13a.**references:** Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. No. 237: 63.**phenotype:** Vein L3 fails to reach wing margin and is shifted toward L4. Anterior crossvein usually lacking. Wings divergent. Postscutellar bristles small and erect. Body small. Viability 60% wild type. Female often sterile. RK2.**cytology:** Placed between 6A3 and 6F11 based on deficiency analysis using *shf*² (Demerec, Kaufmann, Fano, Sutton, and Sansome, 1942, Year Book – Carnegie Inst. Washington 41: 191).***shf*²****origin:** X ray induced.**discoverer:** Oliver, 29j29.**references:** 1935, DIS 3: 28.
1935, DIS 4: 10.**phenotype:** Veins closer together than in wild type. L3 and L4 tend to fuse near anterior crossvein; anterior crossvein shortened, knotted, or absent. Phenotypic effect visible in prepupal wing bud, the two longitudinal veins diverging at a smaller-than-normal angle [Waddington, 1940, J. Genet. 41: 75-139 (fig.)]. Eyes sometimes slightly rough. Scutellar bristles often absent. Scutellum short. Wings narrow and often warped downward. Fertility and viability good. RK2.***shf³****origin:** Spontaneous.**discoverer:** Curry, 37d26.**phenotype:** Like *shf*² but more extreme. Viability about 70% wild type. Frequently infertile. RK2.***shf*³: shifted-3**

From Bridges and Brehme, 1944, Carnegie Inst. Washington Publ. No. 552: 173.

shf*^{ov}: shifted-oval*origin:** Induced by P³².**discoverer:** Bateman, 1950.**references:** 1950, DIS 24: 55.**phenotype:** Eyes rough and narrow. First basal wing cell absent because L3 and L4 veins close. Wings narrow

and pointed. Viability and fertility low. RK2.

other information: On basis of phenotype and position, could be an allele of either *ov* or *shf* or both; not tested.***shg*: shotgun****location:** 2-92.**references:** Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Embryonic lethal. Embryonic cuticle has many small holes with necrotic rims. Head grossly distorted. Weak alleles show head defects and irregular flaws in segmentation pattern.**alleles:** Two retained alleles, *shg*¹ and *shg*² (originally designated *IG* and *IH*) plus sixteen discarded alleles.***shi*: shibire (C. A. Poodry)****location:** 1-51.5.**discoverer:** Grigliatti, 1971.**references:** Grigliatti, Hall, Rosenbluth, and Suzuki, 1973, Mol. Gen. Genet. 120: 107-14.**phenotype:** The shibire locus is characterized by its temperature-sensitive alleles, which are reversibly paralyzed by exposure to 29°, but are essentially normal at 22° (Grigliatti *et al.*). Exposure of developing animals to the restrictive temperature for pulses of one to several hours leads to a plethora of developmental defects, which are specific for the stage treated (Poodry, Hall, and Suzuki, 1973, Dev. Biol. 66: 442-56) (see following table). Short exposures to restrictive temperatures at the time of delamination of the neuroblasts from the neurogenic ectoderm leads to excess neurogenesis at the expense of epidermogenesis, as seen in the neurogenic mutants (Poodry, 1990, Dev. Biol., in press). Differentiation of myoblasts and neuroblasts is inhibited in *shi*¹ embryonic cells *in vitro* at 30° (Buzin, Dewhurst, and Seecof, 1978, Dev. Biol. 66: 442-56). Embryonic neurons cultured at 30° show reduced adhesion to the substrate, retardation of growth cone formation and suppressed neuron formation and elongation; reversed by shift to permissive temperature (Kim and Wu, 1987, J. Neurosci. 7: 3245-55). Lethal embryos disorganized by the restrictive temperature can be cultured *in vivo* as tumorous masses (Poodry). Eye-antenna discs can also be cultured as tumorous masses for several transfer generations (Williams, 1981, DIS 56: 158-61). Primary *in vivo* culture of cut leg imaginal discs leads to an exceptionally high rate of transdetermination (Poodry).

temperature-sensitive period	developmental phenotype
1.5-3 hr	loss of pole cells
3-4 hr	fusion of cell membranes leading to syncytium
5-12 hr	disorganized proliferation of cells leading to transplantable tumorous masses
late third instar	stubby legs; joints missing;
12 hr heat pulse	clipped wings
48 hr before pupariation	eye scar (loss of pigment cells and cone cells). The later the heat pulse, the more anterior the position of the scar on eye
pupariation to pupation	animals die and fail to undergo pupation
14-24 hr after pupariation	supernumerary microchaetae on head and thorax; the temperature sensitive period for each bristle site precedes

temperature-sensitive period	developmental phenotype
	the final cell division of bristle precursor; loss of macrochaetae on head and thorax. Disruption of giant-fiber pathway development (Hummon and Costello, 1987, J. Neurosci. 7: 3633-38). Reduced numbers of dorsal-longitudinal flight muscles (Hummon and Costello, 1988, Roux's Arch. Dev. Biol. 197: 383-93)
24-36 hr after pupariation	loss of head and thoracic microchaetae; supernumerary abdominal macrochaetae and microchaetae
28-42 hr after pupariation	loss of abdominal macrochaetae and microchaetae
32-48 hr after pupariation	loss of abdominal microchaetae
48 hr after pupariation	scimitar-shaped bristles
adult	eggs fail to mature

The temperature-sensitive alleles differ in the severity of their paralysis, recovery period, the restrictive temperature for developmental effects, and in their viability as hemizygotes. They are all hypomorphs, being recessive and having a more extreme expression in combination with a deficiency than when homozygous. A wild-type paternal gene can rescue an egg from a homozygous mother only after 10 hr of development (Swanson and Poodry, 1976, Dev. Biol. 48: 205-11). Of the developmental effects tested, all are autonomous in mosaics generated by somatic recombination or in gynandromorphs (Poodry). The developmental effects on bristles is not enhanced or suppressed by the presence of temperature-sensitive alleles of *N*; *shi* is epistatic to *N* (Lujan, 1981, DIS 56: 86).

Physiological studies of *shi* have revealed the loss of transients in electroretinograms (Kelley and Suzuki, 1974, Proc. Nat. Acad. Sci. USA 71: 4906-09) and failure of neuromuscular transmission at the restrictive temperature (Ikeda, Ozawa, and Hagiwara, 1976, Nature 259: 489-91; Siddiqi and Benzer, 1976, Proc Nat. Acad. Sci. USA 73: 3253-57), though axonal conduction and muscle membrane excitability are unimpaired (Ikeda *et al.*). Exposure of *shi*¹ adults to 29° causes the depletion of synaptic vesicles from the neuromuscular synapse and their replacement with large cisternae (Poodry and Edgar, 1979, J. Cell Biol. 81: 520-27; Koenig, Saito, and Ikeda, 1983, J. Cell Biol. 96: 1517-22). Accumulation of acetyl choline is reduced at the restrictive temperature, not because of reduced synthesis but because of an abnormally rapid rate of release from the cell, which is not reduced by inhibiting tetrodotoxin-sensitive nerve activity (Wu, Merneking, and Barker, 1983, J. Neurochem. 40: 1386-96). Endocytosis is reversibly blocked in the nerve terminus (Kosaka and Ikeda, 1983, Neurobiol. 14: 207-25; Masur, Kim, and Wu, 1990, J. Neurosci.) and may limit the ability of nerves to regenerate synaptic vesicles. Neuromuscular transmission temperature is sensitive in mosaics in which the neuron but not the muscle is mutant, but not in the converse situation (Koenig and Ikeda, 1983, J. Neurobiol. 14: 411-19). During recovery from exposure to 30° *shi*^{ts1} muscles display a multimodal distribution of miniature excitatory junction potential amplitudes never seen in wild type (Ikeda and Koenig, 1987, J. Physiol. 406: 215-23). Further, as the temperature is increased the amplitude of evoked excita-

tory junction potentials decreases; the numbers of vesicles per synapse displays a correlated decrease (Koenig, Kosaka, and Ikeda, 1989, J. Neurosci. 9: 1937-42). Endocytosis is also blocked in the garland cells (Kosaka and Ikeda, 1983, J. Cell Biol. 97: 499-507). Vesiculation of cell membranes results in fusion of blastoderm cells (Swanson and Poodry, 1981, Dev. Biol. 84: 465-70) and vesiculation of surface membranes accompanies secretion of protein epicuticle (Poodry).

alleles: The first alleles recovered were recognized as temperature-sensitive paralytic mutations. These show different temperature responses as summarized in the second table below.

allele	origin	discoverer	synonym	ref ^α	comments
<i>shi</i> ¹	EMS	Grigliatti		1	temperature sensitive
<i>shi</i> ²	EMS	Grigliatti		1	temperature sensitive
<i>shi</i> ³	EMS	Grigliatti		1	temperature sensitive
<i>shi</i> ⁴	EMS	Grigliatti		1	temperature sensitive
<i>shi</i> ⁵	EMS	Grigliatti		1	temperature sensitive
<i>shi</i> ⁶	EMS	Grigliatti		1	temperature sensitive
<i>shi</i> ⁷	EMS	Lindsley & Poodry		2	temperature sensitive
* <i>shi</i> ⁸	γ ray	Poodry	<i>shi</i> ²⁻¹¹	4	<i>T(1;4)14A1</i> ?
<i>shi</i> ⁹	γ ray	Poodry	<i>shi</i> ^{4-14C}	4	
<i>shi</i> ¹⁰	γ ray	Poodry	<i>shi</i> ^{4-14D}	4	
<i>shi</i> ¹¹	γ ray	Poodry	<i>shi</i> ⁶⁻¹	4	
<i>shi</i> ¹²	γ ray	Poodry	<i>shi</i> ⁶⁻⁷	4	
<i>shi</i> ¹³	γ ray	Poodry	<i>shi</i> ⁶⁻¹³	4	
<i>shi</i> ¹⁴	γ ray	Poodry	<i>shi</i> ⁸⁻⁹	4	
<i>shi</i> ¹⁵	γ ray	Poodry	<i>shi</i> ⁸⁻¹³	4	
<i>shi</i> ¹⁶	γ ray	Poodry	<i>shi</i> ⁸⁻¹⁸	4	<i>ln(1)14A;16A</i>
* <i>shi</i> ¹⁷	γ ray	Poodry	<i>shi</i> ¹²⁻⁶	4	<i>ln(1)12D-E;14A1</i>
<i>shi</i> ¹⁸	γ ray	Poodry	<i>shi</i> ^{12-12A}	4	
<i>shi</i> ¹⁹	γ ray	Poodry	<i>shi</i> ¹²⁻¹³	4	<i>T(1;3)14A1;94D</i>
<i>shi</i> ²⁰	γ ray	Poodry	<i>shi</i> ¹²⁻¹⁸	4	<i>T(1;2)14A1;24C</i>
<i>shi</i> ²¹		Siddiqi & Benzer	<i>shi</i> ^{ST139}	3	
<i>shi</i> ²²	EMS	Katzen			

^α 1 = Grigliatti, Hall, Rosenbluth, and Suzuki, 1973, Mol. Gen. Genet. 120: 107-14; 2 = Dale Lindsley and Poodry, Dev. Biol. 56: 213-18; 3 = Siddiqi and Benzer, 1976, Proc Nat. Acad. Sci. USA 73: 3253-57; 4 = Poodry.

allele	temperature of adult paralysis	temperature of larval paralysis	temperature causing developmental defects	viability of allele over deficiency
<i>shi</i> ¹	29°	29°	29°	weak
<i>shi</i> ²	29°	31°	31°	strong
<i>shi</i> ³	29°	29°	29°	very weak
<i>shi</i> ⁴	29°		none	lethal
<i>shi</i> ⁵	29°			
<i>shi</i> ⁶	29°	31°	31°	strong
<i>shi</i> ⁷	29°	31°	31°	strong
<i>shi</i> ²¹	29°	29°	29°	

cytology: Located in 13F to 14A (Austin and Poodry, 1991; Morgan, 1991).

molecular biology: Cloned and sequenced by van der Blik and Meyerowitz (1991, Nature 351: 411-14); homologous to rat dynamin.

shifted genitals: see *sgc*

****shi*: shorter legs**

location: 1-36.3.

origin: Induced by 2-fluoroethyl methanesulfonate (CB. 1522).

discoverer: Fahmy, 1957.

references: 1959, DIS 33: 90.

phenotype: Small fly with short legs. Male viability and fertility low. RK3.

shm: short macros

location: 1-22.4.

origin: Induced by triethylenemelamine (CB. 1246).

discoverer: Fahmy, 1953.

references: 1959, DIS 33: 90.

phenotype: Bristles short and stiff. Eclosion delayed. Male sterile and viability reduced. RK2.

shn: schnurri

location: 2-62.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Embryonic lethal. Embryos lack dorsal hypoderm. Internal organs appear normal and extruded through the open dorsal side of the embryo. Ventral hypoderm contracted.

alleles: Two ethyl-methanesulfonate-induced alleles *shn*¹ and *shn*² (formerly designated *IB* and *IM*), the latter being heat sensitive, and one hybrid-dysgenesis-induced allele, *shn*³, (formerly *TD5*) isolated by Gergen. In addition fifteen discarded alleles.

cytology: Placed in 47E3-48A6 based on its location to the area of overlap between *Df(2R)en-A = Df(2R)47D3; 48A5-6* and *Df(2R)en-B = Df(2R)47E3;48B2*.

***sho: shovel**

location: 2- (not located).

origin: Spontaneous in *In(2L)t*.

discoverer: GoodSmith, 49k.

references: Ives, 1952, DIS 26: 65.

phenotype: Wings short and rounded. Viability good. RK2A.

short bristle: see *stb*

short egg: see *seq*

short macros: see *shm*

short tarsi: see *sht*

short vein: see *shv* under *dpp*

short wing: see *sw*

short winged: see *sh*

short-5: see *sh-5*

short-bristle: see *ml*

shortened wing: see *sg*

shortened bristles: see *shb*

shortened veins: see *svs*

shorter bristles: see *sbt*

shorter legs: see *shl*

shotgun: see *shg*

shovel: see *sho*

***shp: shrimp**

location: 1-47.5.

origin: Induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1955.

references: 1958, DIS 32: 74.

phenotype: Small fly. Eclosion delayed. Male viability about 30% wild type. Both sexes fertile. RK3.

shr: shrunken

location: 2-2.3.

discoverer: Bridges.

phenotype: Body small and dark. Viability and fertility good. Overlaps wild type unless combined with *abb*, where mutual enhancement occurs. RK3.

cytology: Placed between 22A3 and 22B1 on the basis of its inclusion in *Df(2L)S2 = Df(2L)21C6-D1;22A6-B1* but not in *Df(2L)S5 = Df(2L)21C2-3;22A3-4* (Lewis, 1945, Genetics 30: 137-66).

shrew: see *sw*

shrimp: see *shp*

shroud: see *sro*

shrunken: see *shr*

Shrunken thorax: see *Sht*

shrunken-3: see *wz*

***sht: short tarsi**

location: 1-20.9.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).

discoverer: Fahmy, 1953.

references: 1959, DIS 33: 90.

phenotype: Legs extremely short; reduction in length most pronounced in metatarsal and tarsal regions. Some tarsi fused, others absent. Bristles thin and short. Adult short lived. RK3.

Sht: Shrunken thorax

location: 2-54.7 (2.8±0.4 map units to the left of *cn*).

origin: Induced by ethyl methanesulfonate.

references: Kulkarni and Babu, 1981, DIS 56: 192.

phenotype: Heterozygotes exhibit an indentation across the dorsal mesothorax giving the appearance of shrunken thorax. Typically a groove runs across the thorax in a V shape. There is some variability in the expressivity; a small fraction of flies have only a marginal phenotype, but the penetrance is nearly complete. Newly emerged flies do not often show the phenotype or have only a faint line on the thorax; the groove becomes visible as the cuticle hardens. Mutant flies have good viability and fertility. Homozygous lethal.

other information: Possibly an allele of *Mhc*.

shu: shut down (T. Schüpbach)

location: 2-105.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus.

phenotype: Female sterile; homozygous females contain few developing egg chambers in their ovaries, which may contain abnormal numbers of nurse cells and usually degenerate before yolk uptake begins. Rarely a few very small and abnormal eggs are produced.

alleles: *shu*¹ and *shu*² recovered as *WQ* and *WM* respectively.

cytology: Placed in 59D8-60A2, since uncovered by both *Df(2R)bw-S46 = Df(2R)59D8-11;60A7* and *Df(2R)bw-D23 = Df(2R)59D4-5;60A1-2*.

Shu: Shudderer (J.C. Hall)

location: 1-55.1.

origin: Induced by ethyl methanesulfonate.

discoverer: R.L. Williamson.

references: Williamson, 1982, *Psychopharmacology* 76: 265-68.

phenotype: Sudden leg jerks are frequent and enough to topple the fly; negative geotaxis is sluggish and seemingly not a consequence of shuddering bouts during climbing toward top of glass vials; neither of these behavioral abnormalities is pronounced in young adults, but they become maximal after about a week of adult life; lithium or ammonium ions placed in medium on which the mutant is grown reduce the severities of the eventual adult phenotypes. Homozygous females are not observed.

shut down: see *shu*

shv: see under *dpp*

shv: see *svs*

Shw: see *Sh*⁴

shy: shy (T. Schüpbach)

location: 2-62.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1989, *Genetics* 121: 101-17.

phenotype: Maternal effect lethal. Embryos from homozygous mothers develop into larvae with no visible cuticular abnormalities, but which do not hatch out of the egg case.

alleles: *shy*^{RU} = *shy*¹.

***Si: Ski**

location: 2-36.

discoverer: Clausen, 1911.

references: Clausen and Collins, 1922, *Genetics* 7: 385-426.

Bridges and Morgan, 1923, *Carnegie Inst. Washington Publ. No. 327*: 149 (fig.).

phenotype: Homozygous or heterozygous *Si* combined with homozygous *si-3* produces wings with turned up tips. Double homozygote also has a crimped costal vein. Other genotypes wild type. RK3.

***si-3: ski-3**

location: 3-46.5.

discoverer: Clausen, 1911.

references: Clausen and Collins, 1922, *Genetics* 7: 385-426.

Bridges and Morgan, 1923, *Carnegie Inst. Washington Publ. No. 327*: 149.

phenotype: *si-3/si-3* fly has upturned wing tips when homozygous or heterozygous for *Si*, otherwise normal. RK3.

sic: sichel

location: 3-48.8.

references: Mayer and Nüsslein-Volhard, 1988, *Genes Dev.* 2: 1496-1511 (fig.).

Tearle and Nüsslein-Volhard, 1987, *DIS* 66: 209-26.

phenotype: Maternal effect lethal; embryos produced by homozygous mothers show narrow dentical bands and head skeleton with fused ventral arms. Embryos may also be normal and hatch or die early with irregular cell cleavage patterns. Distribution of syncytial blastoderm nuclei normal in some embryos and irregular in others. Germ line dependent but may also have a somatic component. Mutant expression autonomous in mutant pole cells transplanted into wild type recipients. Embryonic phenotypes of progeny of *sic/sic* and *sic/Df(3R)by10* are similar indicating null alleles.

alleles: Four ethyl methanesulfonate-induced alleles; all cold sensitive: *sic*¹ (weak allele), *sic*², *sic*³, and *sic*⁴ (originally designated 215, 256, 371, and 612).

cytology: Placed in 85D8-12 based on its inclusion in the region of overlap of the synthetic deficiency for 84F2-85E in *In(3R)Scr^{MscL}Antp^{BR}* and *Df(3R)by10 = Df(3R)85D8-12;85E7-F1*.

side wings: see *siw*

sie: sieve (T. Schüpbach)

location: 2-68.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1989, *Genetics* 121: 101-17.

phenotype: Maternal-effect lethal. Embryos from homozygous females show variable defects and irregularities at cellularization; at final differentiation embryos form only fragmented pieces of cuticle.

alleles: *sie*¹, *sie*², and *sie*³ recovered as *HA*, *RF*, and *III E* respectively.

cytology: Placed in 49E7-50A3, since uncovered by *Df(2R)vgB = Df(2R)49D3-4;49F15-50A3*, but not by *Df(2R)vgC = Df(2R)49B2-3;49E7-F1*.

other information: *sie*³ has a semi-dominant effect, causing 60-80% embryonic lethality when mother heterozygous.

***Sil: Skilike**

location: 2- (not located).

discoverer: Goldschmidt.

references: 1947, *J. Exptl. Zool.* 104: 216.

phenotype: Wings turned up at tips. Semidominant. Poor viability. RK3.

other information: Not an allele of *Si*.

silver: see *svr*

silver tips: see *stp*

sim: single minded

location: 3-52.2 (just distal to *pic*).

synonym: *l(3)S8; schm; l(3)87Ea*.

references: Hilliker, Clark, Gelbart, and Chovnick, 1981, *DIS* 56: 65-72.

Thomas, Crews, and Goodman, 1988, *Cell* 52: 133-41 (Fig.).

Crews, Thomas, and Goodman, 1988, *Cell* 52: 143-51 (Fig.).

Mayer and Nüsslein-Volhard, 1988, *Genes Dev.*

2: 1496-1511 (Fig.).

phenotype: Embryonic lethal. Denticle bands of all segments narrow. Both head skeleton ventral arms and anal plates fused. In the ventral nervous system, transverse commissures lacking entirely. Midline neurons and supportive mesectodermal cells missing. Germline viable with no maternal component. Deficiency test indicates that *sim*¹ is amorphic. In normal genotypes, transcript first noted at cellular blastoderm in a pair of longitudinal rows of cells at the interface between the presumptive mesoderm and the neurogenic ectoderm. At the end of gastrulation, the mesoderm has invaginated into the ventral furrow and the two rows of expressing cells have come together in the ventral midline and both transcript and protein expressed in a row of cells in the midline and in an annulus around the presumptive anterior midgut. By eleven hours of development, protein is found in the cell types missing in the midline of the central nervous system of mutant embryos, more in mesectodermal cells than neuronal elements, and in a subset of cells of the foregut. Antibody staining confined to nuclei.

alleles:

allele	origin	discoverer	synonym	ref ^α
<i>sim</i> ¹	X ray	Schalet	<i>l(3)S8</i>	3
<i>sim</i> ²	EMS	Hilliker, Clark	<i>l(3)H9</i>	1, 2
<i>sim</i> ³	EMS	Hilliker, Clark	<i>l(3)H66</i>	1, 2
<i>sim</i> ⁴	EMS	Hilliker, Clark	<i>l(3)H79</i>	1, 2
<i>sim</i> ⁵	EMS	Hilliker, Clark	<i>l(3)B13-4</i>	1, 2
<i>sim</i> ⁶	EMS	Hilliker, Clark	<i>l(3)B21-2</i>	1, 2
<i>sim</i> ⁷	EMS	Hilliker, Clark	<i>l(3)B30-1</i>	1, 2
<i>sim</i> ⁸	EMS	Nüsslein-Volhard	<i>E320</i>	
<i>sim</i> ⁹	EMS	Nüsslein-Volhard	<i>RD</i>	

^α 1 = Hilliker, Clark, and Chovnick, 1980, Genetics 95: 95-110;
2 = Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72;
3 = Schalet, Kernaghan, and Chovnick, 1964, Genetics 50: 1261-68.

cytology: Placed in 87E1 based on its being between *Df(3R)ry75 = Df(3R)87D1-2;87D14-E1* and *Df(3R)l26c = Df(3R)87D14-E1;87E3-5*.

molecular biology: The 3' end of the gene has been cloned, a cDNA containing the 3' end sequence, and the conceptual sequence of 655 C-terminal amino acids determined. The only identity encountered in protein-sequence data bases is to the *Drosophila per* gene; a 239-amino-acid sequence exhibits 23% identity and contains two 51-amino-acid repeats separated by 115 amino acids in *sim* and 99 amino acids in *per*. The sequence contains no transmembrane, homeobox, or zinc-finger motifs. A transcript of 3.0 kb is present from 0-3 hr post fertilization, increases in 3-6 hr and then disappears; a 3.5 kb transcript appears strongly from 3-6 hr, declining over the next six hours, and then persisting at low levels throughout embryogenesis.

***sina*: seven in absentia (R. Carthew)**

location: 3-45.5.

origin: Induced with ethyl methanesulfonate.

discoverer: Carthew, 1988.

phenotype: Photoreceptor cell R7 of eye transformed into a cone cell, resulting in the absence of R7 in adult ommatidia. In amorphic alleles, 40% of ommatidia also missing an additional photoreceptor cell and 10% of ommatidia also missing two photoreceptor cells. Eyes weakly rough; ocelli appear normal. Adult bristles sometimes missing and sometimes duplicated, arising from a com-

mon socket; occasionally three bristles arise from a common socket. Wings sometimes held outstretched. Adults eclose normally but fail to survive after 24 h; display lethargic behavior and are not fertile, though mature sperm and ova are produced.

alleles: Six, designated *sina*¹ through *sina*⁶. *sina*¹, *sina*⁴, *sina*⁵, and *sina*⁶ hypomorphic; *sina*² and *sina*³ amorphic based on the phenotypes of hemizygous [*sina/Df(3L)st-g18*] versus that of homozygous flies.

cytology: Located in 73D1-7 by deficiency mapping.

***sine oculis*:** see *so*

***singed*:** see *sn*

***singed wing*:** see *l(1)nfw*

***single minded*:** see *sim*

***sis-a*: sisterless a (T.W. Cline)**

location: 1-34.3 (based on 4496 *v-m* recombinants).

origin: Induced by ethyl methanesulfonate.

references: Cline, 1986, Genetics 113: 641-63, corrigendum 114: 345.

Cline, 1988, Genetics 119: 829-62.

phenotype: Homozygous females die but hemizygous males and heterozygous females fully viable; females die as embryos and larvae; rare morphologically normal and fertile escapers observed at lower temperatures. Single extant allele hypomorphic; locus also defined by dominant behavior of deficiencies and duplications. Dominant lethal for females simultaneously heterozygous for *sis-b*⁻ or *Sxl*⁻ or whose mothers are heterozygous for *da*⁻. Magnitude of female-lethal dominant synergism sensitive to genetic background, but can be very high. Lethal interactions are generally less severe at lower culture temperatures. Constitutive allele, *Sxl*^{M1}, suppresses female lethality of *sis-a* homozygote or of any heterozygous combination of mutant alleles or deficiencies of these four genes. Duplication of *Sxl*⁺ also suppresses, but less effectively. Female-lethal interactions between *sis-a* and *Sxl* mutations display remarkably similar *Sxl* allele specificity to those between maternal *da* and zygotic *Sxl* mutations, indicating that *da* and *sis-a* disrupt the same aspect of *Sxl* regulation. Oogenesis normal for homozygous *sis-a* germ-line clones induced by mitotic recombination; no maternal effect. Sexual phenotype of 2X:3A animals extremely sensitive to *sis-a*⁺ dose (more male at lower temperatures), and like *da* and *Sxl*⁻, shows masculinizing interaction with autosomal male-specific lethal mutations but no increase in viability of escapers; nevertheless, sexual phenotype of homozygous *sis-a* clones generated by mitotic recombination normal. Female-lethal effects caused by decrease in *sis-a* function have their complement in male-lethal interactions caused by increase in *sis-a* function (duplications). Male lethality is increased as *Sxl*⁺ dose or *sis-b*⁺ dose is increased, and is suppressed by loss-of-function *Sxl* mutations. The dose-dependent interactions of this gene with *Sxl*⁺ identify it as part of the numerator of what has been called the X/A balance, the primary sex-determination signal—a character it shares with *sis-b*.

cytology: Placed in extreme distal position in 10B4-9 based on its failure to complement *Df(1)N71 = Df(1)10B4-5;10D4* and its genetic position distal to *l(1)10Bb* and within <0.004 cM of *l(1)10Ba*.

sis-b: see under **Asc**

***siw: side wings**

location: 1-58.5.

origin: Induced by L-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1955.

references: 1958, DIS 32: 74.

phenotype: Wings rotated on long axis so that inner margin is higher than costal margin. Male sterile; viability about 50% wild type. jK2.

sk: stuck (J.C. Hall)

location: 4-?.

origin: Spontaneous.

references: Beckman, 1970, DIS 45: 36.

Hall, Siegel, Tompkins, and Kyriacou, 1980, Stadler Genet. Symp. 12: 43-82.

phenotype: Males become stuck in females after copulation; ca. 50% of such matings exhibit this phenotype (data shown in Hall *et al.*, 1980; Beckman, 1970, quoted 100% penetrance originally, which had fallen to 78% by the time of her report); stuck males not infrequently are able to disengage eventually on their own; if so, appendages of their terminalia are held in aberrantly protruding positions (Hall *et al.*, 1980); Beckman mentions anatomical abnormality in the strain (near time of isolation): affected males showing narrowing of abdomen (not observable in current versions of stock and was never consistent enough to allow discrimination of affected individuals from those who would behave normally); if mutant males are unable to withdraw after copulating, he and the female die within a few days (Beckman 1970, quotes 14% as mortality frequency for mated pairs); a stuck *vs.* non-stuck mating result is stochastic (as opposed to being a pure penetrance problem, as it were), given that (for example) re-testing a non-stuck case can readily lead to stuck outcome in a subsequent mating (Hall *et al.*, 1980); mutant male-female pairs are more difficult to separate (by vortexing) than are normal copulating pairs (Hall *et al.*, 1980); females unaffected by the genetic variant(s).

other information: Beckman (1970) reported that *sk* maps on chromosome 3; Hall *et al.* (1980) unable to confirm this, but their mapping to 4 was somewhat problematical, in that other factors in the strain seem necessary for full mutant phenotype to be realized.

Sk: Streak

location: 2-16.0.

origin: Spontaneous.

discoverer: Bridges, 12k27.

references: Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 222 (fig.).

phenotype: Dark streak extends down middle of thorax from neck to tip of scutellum. Wings may diverge and droop. Overlaps wild type. Enhanced by *b* or *e^s*. Homozygous lethal. RK2.

cytology: Salivary chromosomes apparently normal (Bridges).

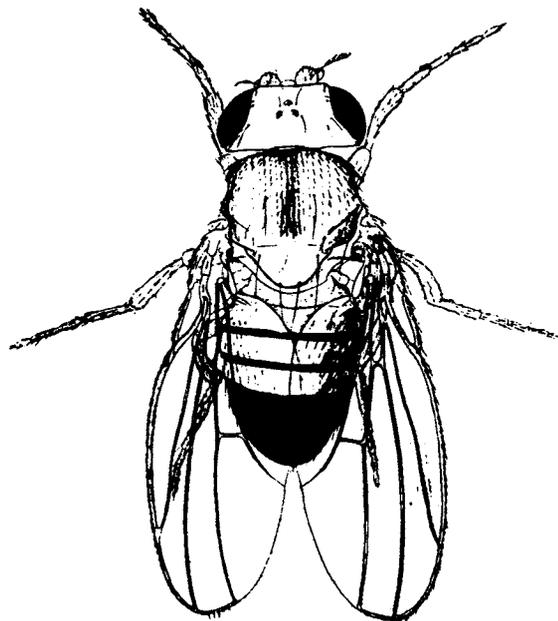
skd: skuld (J.A. Kennison)

location: 3-51.

origin: Induced by ethyl methanesulfonate.

discoverer: Kennison, 1984.

references: Kennison and Tamkun, 1988, Proc. Nat. Acad.



Sk: Streak

From Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 216.

Sci. USA 85: 8136-40.

phenotype: Isolated as a dominant suppressor of *Pc* mutations. Associated with recessive lethality at the larval-pupal transition. Also interacts with *Pcl*, *Scr*, and *Ubx* mutations. May weakly suppress *Antp^{Ns}*.

alleles: *skd¹* induced by ethyl methanesulfonate and *skd²* induced by gamma irradiation.

cytology: Proximal to 87B15 on basis of exclusion from *Df(3R)ry615 = Df(3R)87B12-15;87E8-11*.

Ski: see *Si*

ski-3: see *si-3*

Skilike: see **Sil**

skuld: see **skd**

sl: small wing

location: 1-53.5.

phenotype: Wings about 80% normal length, straight edged, and blunt tipped. Crossveins rather close. Eyes large and slightly rough.

alleles:

allele	origin	discoverer	ref ^α	comments
<i>sl¹</i>	spont	Bridges, 15121		
<i>sl²</i>	X ray	Dobzhansky, 31b3	2	
<i>sl³</i>	X ray	Lefevre	1	associated with <i>Tp(1;2)r⁺75c</i>
* <i>sl³⁴</i>	spont	Gottschewski, 1934		eyes normal

^α 1 = Schalet, 1986, Mutat. Res. 163: 115-44. 2 = Sivertzev-Dobzhansky and Dobzhansky, 1933, Genetics 18: 173-92.

cytology: Placed in 14B13 based on probable association of *sl³* with distal X break of *Tp(1;2)r⁺75c = Tp(1;2)14B13;15A9;35D-E*. *sl²/Tp(1;2)r⁺75c* and *sl²/Dp(1;2)r⁺75c* have small

wings and rough eyes; *sl²/Df(1)r75c* has very rough eyes. *sl²* has a normal phenotype in combination with *Dp(1;4)r⁺ = Dp(1;4)14A1-2;16A1-2;102F2-3* (Schalet, 1986, Mutat. Res. 163: 115-44).

***Sl: Splotched**

location: 1-56.9 (to the right of *f*).
origin: X ray induced.
discoverer: Muller, 26111.
references: 1935, DIS 3: 30.
phenotype: Wing hairs disarranged in small patches. Male infertile. Viability excellent. RK1.

sla: slimma

location: 1-48.6.
origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).
discoverer: Fahmy, 1954.
references: 1958, DIS 32: 74.
phenotype: Fly slim with very narrow abdomen. Body length normal. Eclosion delayed slightly. Wings curve slightly. *sla/slb* and *sla/sld* wild type. Male fertile and viable. Female sterile; viability about 50% wild type. RK3.
other information: Two alleles each induced by CB. 3007 and CB. 3025.

slater: see *tkv*

***slb: slim body**

location: 1-45.3.
origin: Induced by ethyl methanesulfonate (CB. 1528).
discoverer: Fahmy, 1956.
references: 1958, DIS 32: 74.
phenotype: Body narrow but of normal length. *slb/sla* and *slb/sld* wild type. Viability and fertility good in both sexes. RK3.

slc: slim chaetae

location: 1-3.6.
origin: Induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).
discoverer: Fahmy, 1954.
references: 1959, DIS 33: 90-91.
phenotype: Bristles thin and short. Inner wing margins occasionally incised. Both sexes viable and fertile. RK1.

sld: slender

location: 1-50.1.
origin: Induced by *p*-N,N-di-(2-chloroethyl)amino-phenylethylamine (CB. 3034).
discoverer: Fahmy, 1957.
references: 1959, DIS 33: 91.
phenotype: Fly rather small and slim with narrow abdomen. *sld/sla* and *sld/slb* wild type. Male fertile but shows delayed eclosion and reduced viability. Female very inviable. RK3.
other information: One allele induced by CB. 3025.

***sld^{pta}: slender-pointed abdomen**

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).
discoverer: Fahmy, 1956.
synonym: *pta*.
references: 1959, DIS 33: 88.
phenotype: Fly small, has narrowed abdomen and slightly altered eye and wing shape. Male sterile; viability about

25% wild type. RK3.

slender chaetae: see *sch*

slf: schlaff

location: 2-15.
references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.
 Roarke, Mahoney, Graham, and Lengyel, 1985, Dev. Biol. 109: 476-88.
 Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.
phenotype: Embryonic lethal. Cuticular differentiation normal but arrangement of cuticle around embryo abnormal. Cuticle is detached from the body. Head skeleton tilted backwards. Posterior segments contracted, anterior segments stretched around egg tip.
alleles: Two ethyl methanesulfonate-induced alleles retained, *slf¹* and *slf²* (originally designated *IG* and *IJ*) plus four discarded alleles.

slgA: sluggish-A (J.C. Hall)

location: 1- {65}.
origin: Induced by ethyl methanesulfonate.
discoverer: Eichenberger and Benzer.
synonym: *EE85*.
references: Markow and Merriam, 1977, Behav. Genet. 7: 447-55.
 Hall, 1978, Genetic Mosaics and Cell Differentiation, (W.J. Gehring, ed.). Springer-Verlag, Berlin, pp. 259-305.
 Miklos, Kelly, Coombe, Leeds, and Lefevre., 1987, J. Neurogenet. 4: 1-19.

phenotype: Isolated as defective in fast phototaxis, using counter-current-distribution (CCD) machine; also turned out to have poor (negative) geotaxis in this machine; yet mutant males showed some positive optomotor responses and had a normal electroretinogram; hence termed sluggish (as opposed to phototaxis-defective *per se*); Markow and Merriam (1977) confirmed aberrant CCD phototactic response; in maze tests the mutant was found to be quite photonegative (more so than wild type) and seemingly normal in geotaxis (Markow and Merriam, 1977). *slgA* causes poor viability, as well as aberrant behavior in hemizygous females (Miklos *et al.*, 1987); in mosaics studied for sluggishness, a mutant leg seemed to be debilitated independently of behavior of other legs, with the six foci for such movement deficits mapping to the ventral blastoderm (A. Ghysen, cited in Hall, 1978).

cytology: Mapped to 19F4, along with *sol*, these two mutations being flanked by *l(1)19Fe* and *l(1)19Ff*; this determination (Miklos *et al.*, 1987) was based on the poor phototaxis of *slg* in heterozygous combination with *Df(1)GA104*, but not with *Df(1)16-129* or *Df(1)JA117*, none of which have breakpoints determined cytologically.

molecular biology: Gene located in a 25 kb subsegment of 19F. Three transcription units detected by Delaney *et al.* (unpubl.).

other information: Complements *sol* (Miklos *et al.*, 1987). Also complements *PC16*, another phototaxis/geotaxis-defective mutation in the region, isolated in laboratory of S. Benzer and studied by Markow and Merriam (1977).

slgB (J.C. Hall)

location: 1- {1}.

origin: γ ray induced.

references: Sharma, 1977, *Experientia* 33: 171-77.

phenotype: Isolated with regard to poor response to light in fast phototaxis tests; both sexes are sluggish in this regard. Males in this strain do not mate with females in darkness; in the light, males court and mate with females (40-50%, within 10 m) and other males with equal vigor; the latter behavior includes formations of chains and rings of intermale courters, as well as pseudo-copulation attempts; courtships in these circumstances do not exhibit wing-flick repelling responses characteristic of wild-type males. Spectral sensitivity studies of the light-elicited courtship activities showed 420-515 nm to be effective (thus orange/red range ineffective); yellow light was perhaps the most stimulatory; quick turn-ons and turn-offs of intermale courtships could be effected by intermittent exposures to yellow and red light, respectively.

cytology: Associated with a reciprocal *T(1;3)*, whose *X* breakpoint is in 2C; appears to cause the aberrant behaviors; the other breakpoint is said to be in 97A10 (but also indicated as being in 3L in Sharma, 1977).

other information: *fru* causes somewhat similar courtship abnormalities, but its locus (polytene 90-91) is not near the third-chromosome breakpoint in this *T(1;3)*.

sli: slit

location: 2-77.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 267-82.

Rothberg, Hartley, Walther, and Artavanis-Tsakonas, 1988, *Cell* 55: 1047-59.

phenotype: Homozygotes die as embryos. Head involution abnormal. In the ventral nervous system, transverse commissures lacking entirely. Midline neurons and supportive mesectodermal cells missing. Gene expression confined to ectoderm in cellular blastoderm (*i.e.*, no expression in presumptive mesoderm) and to the midline neuroepithelium at gastrulation. Antibodies raised against a *TrpE* fusion protein fail to stain mutant embryos. More severe abnormality in *Df/sli* than *sli/sli* indicates that *sli* is hypomorphic in nature. In combination with *Pc*-like mutants abdominal transformations occur (Jürgens).

alleles: Two ethyl-methanesulfonate-induced alleles, *sli*¹ and *sli*² (originally designated *IG2 = IG23* and *IG7 = IG107*); also a *P*-induced allele mentioned by Rothberg *et al.*

cytology: Localized to 52D by *in situ* hybridization.

molecular biology: Isolated from genomic library using a 7 kb *EcoRI* fragment from the Notch locus that contains the epidermal-growth-factor-like repeats. A 2.5 kb *HindIII* genomic fragment sequenced; cDNA sequence indicates presence of 63 base-pair intron. Within exon sequence a single large ORF can code for six adjacent and one noncontiguous EGF-like repeat separated by 99 amino acids. EGF-like repeats show 59% identity to those of *N*.

slight: see *sli*

slim: see *slm*

slim body: see *slb*

slim bristle: see *smb*

slim chaetae: see *slc*

slimma: see *sla*

slimmer abdomen: see *sln*

slip: slipshod (T. Schüpbach)

location: 2-42.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1989, *Genetics* 121: 101-17.

phenotype: Maternal-effect lethal. Embryos from homozygous mothers do not hatch; in cuticle preparations they show irregular segmentation and variable segment fusions.

alleles: *slip*^{PX} = *slip*¹.

slit: see *sli*

slm: slim

location: 1-33.7.

origin: Induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1955.

references: 1958, *DIS* 32: 75.

phenotype: Small fly with narrow abdomen. Viability and fertility good. RK3.

other information: One allele induced by CB. 1506.

***sln: slimmer abdomen**

location: 1-53.5.

origin: Induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1953.

references: 1959, *DIS* 33: 91.

phenotype: Rather small fly with narrow abdomen. Occasionally, wings slightly upheld and eyes small or misshapen. Male infertile; viability about 15% wild type. Female sterile. RK3.

slo: slowpoke (J.C. Hall)

location: 3-86.

origin: Induced by ethyl methanesulfonate.

references: Elkins, Ganetzky, and Wu, 1986, *Proc. Nat. Acad. Sci. USA* 83: 8415-19.

Elkins and Ganetzky, 1988, *J. Neurosci.* 8: 428-34.

phenotype: Isolated in screen for third chromosomal temperature-sensitive paralytic mutants (Elkins *et al.*, 1986); this one is uncoordinated and unable to climb when exposed to 38° but not completely paralyzed; four-minute exposure to that high temperature causes several minutes of motionlessness on return to 22°. Legs shake when under ether anesthesia, as in *Shaker* mutants but less extreme. Unconditionally defective in behavior (*e.g.*, diminished flight ability; tends to walk or fly in anomalous short hops when in large open container). Physiologically, the mutation abolishes Ca²⁺-dependent potassium current (*I_C*), as shown, and analyzed further, in a variety of experiments involving recordings from dorsal longitudinal flight muscles (DLMs) of adults (Elkins *et al.*, 1986; Elkins and Ganetzky, 1988): DLM spikes abnormally broadened after stimulation of giant fiber nerve pathway (one of whose endpoints is DLMs) or of motor neurons synapsing on these muscles. From voltage clamp analyses, a peak of early outward current fol-

lowing a step pulse from -80 to -40 mV, as revealed by a *Sh* mutation, is absent in *slo*, though inward Ca^{2+} currents in same traces are normal; no early outward current seen when *slo* is treated with 4-aminopyridine (which phenocopies *Sh*) or combined with *Sh*¹⁴. (This double mutant has very low viability and severe impairment in locomotor activity); charybdotoxin, which blocks I_C channels in wild-type DLMs, had no effect on outward currents in *slo*; delayed excitation of DLMs, observed in response to depolarizing currents delivered to wild-type (or *Sh*), absent in *slo*, and spike amplitudes increased; these abnormalities phenocopied by reducing I_C in normal muscle by injecting EGTA into such cells or using low- Ca^{2+} saline; the lengthened muscle action potentials in *slo* indicate importance of I_C in effecting repolarization of such potentials.

alleles: Several alleles; *slo*¹ and *slo*⁴ have been mentioned (Atkinson, Robertson, and Ganetzky, 1989, Neurosci. Abstr. 15: 541).

cytology: Localization defined in part by *In(3R)slo*⁴, with one breakpoint in the *E(spl)* region (in 96F) and the other at or very near *slo* locus, which is indicated as proximal to *E(spl)* (Atkinson *et al.*, 1989).

slope wing: see *slw*

sloppy paired: see *slp*

slow receptor potential: see *slrp*

slowpoke: see *slo*

slp: sloppy paired

location: 2-8.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Embryonic lethal. Embryos lack parts of naked cuticle of T2, A1, A3, A5, and A7 in an irregular fashion. No effect of *ftz* expression (Carroll and Scott, 1986, Cell 45: 113-26).

alleles: Three ethyl methanesulfonate-induced alleles, *slp*¹ (weak) and *slp*² and *slp*³ (both strong) (original designations *11M*, *L12*, and *7L*, respectively).

cytology: Placed in 24A3-D4 based on its being in the segment deleted by *Df(2L)ed1 = Df(2L)24A3-4;24D3-4*.

slrp: slow receptor potential (J.C. Hall)

location: 1-51.4 (Pak, 1975); 49.7 (Homyk and Pye, 1988).

origin: Induced by ethyl methanesulfonate.

discoverer: Pak.

references: Pak, 1975, Handbook of Genetics (R.C. King, ed.). Plenum Press, New York, Vol. 3, pp. 703-33.

Homyk and Sheppard, 1977, Genetics 87: 95-104.

Homyk and Pye, 1989, J. Neurogenet. 5: 37-48.

phenotype: In electroretinogram recordings, there is an abnormally slow return of photoreceptor potential to baseline after stimulus is turned off; also, reduced light-on and light-off transient spikes; these phenotypes reported for the first three alleles isolated (Pak, 1975); action spectrum of mutant ERG (Pak, 1975) same as in wild-type; hence, opsins unlikely to be altered. Intracellular recordings reveal some penetrated photoreceptors to have potentials with abnormally slow decay and others to show normal receptor potentials (Pak, 1975). *slrp* mutants are generally hypoactive (Homyk, Pye, and Pak,

1981, Genetics 97: s50) and show ether-induced leg shaking, cold-induced leg paralysis, and the defects in ERG; *slrp*⁴ exhibits, in its permissive temperature range (20°-30°), the phenotypes just listed; in addition it is difficult to arouse for flight, has abnormally short jumps elicited, and, as a male, shows abnormal courtship wing displays (Homyk and Sheppard, 1977); in the temperature range noted above, this allele causes ERG phenotypes like those associated with the original mutations (slow rate of repolarization, transient amplitude deficits); as the temperature is lowered from 20° to 15°, *slrp*⁴ becomes (<17°) sensitive to mechanical stress (Homyk and Sheppard, 1977), and eventually completely paralyzed (Homyk and Pye, 1989); the ERG abnormalities are accentuated at these low temperatures (Homyk and Pye, 1989); for example, the off-transient spike can be completely eliminated in 15° recordings; this effect is reversed on raising the temperature (Homyk and Pye, 1989).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>slrp</i> ¹	EMS	Pak	<i>slrp</i> ^{P28}	p	
<i>slrp</i> ²	EMS	Pak	<i>slrp</i> ^{P29}	p	
<i>slrp</i> ³	EMS	Pak	<i>slrp</i> ^{P67}	p	
<i>slrp</i> ⁴	EMS		<i>hypoD</i>	h	strongest allele

^α h = Homyk and Sheppard, 1977, Genetics 87: 95-104; p = Pak, 1975, Handbook of Genetics (R.C. King, ed.). Plenum Press, New York, Vol. 3, pp. 703-33.

cytology: Maps to 13F10-14B1, based on inclusion of *slrp*⁴ in both *Df(1)sd72b = Df(1)13F1;14B1* and *Dp(1;4)r⁺ = Dp(1;4)13F10;16A1-2;102F2-3* (Homyk and Pye, 1988).

sls: sallimus (J.A. Kennison)

location: 3 (unmapped).

origin: Induced by ethyl methanesulfonate.

discoverer: Kennison, 1984.

synonym: *sam* (preoccupied).

references: Kennison and Tamkun, 1988, Proc. Nat. Acad. Sci. USA 85: 8136-40.

phenotype: Dominant suppressor of the extra-sex-combs phenotype of heterozygous *Pc* alleles. Also suppresses the dominant extra-sex-combs phenotype of *Pcl* alleles.

slt: slight

location: 2-106.3.

origin: Spontaneous.

discoverer: Curry, 39b20.

references: 1939, DIS 12: 45.

phenotype: Fly small. Bristles short and thin. Enhances *px*. Viability and fertility good. RK3.

sluggish: see slg

slv: see *svr*

***slw: slope wing**

location: 1-51.2.

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1956.

references: 1958, DIS 32: 75.

phenotype: Wings usually slightly upheld or spread. Viability and fertility good. RK3.

sm: smooth

location: 2-91.5.
origin: Spontaneous.
discoverer: Bridges, 35c14.
phenotype: Abdomen partially denuded of bristles and shrunken. Wings usually warped and semierect. Acrostichal hairs disarranged. Tendency for erect postscutellars. Male genitalia often disturbed. Anal protuberance of female bent down. Viability 30% wild type. Both sexes entirely sterile. RK2.

sm²

references: Frankham and Nurthen, 1980, DIS 80: 204.
synonym: *2sm^{lab}*: low abdominal bristle number.
phenotype: Reduced numbers of abdominal bristles; alteration of abdominal bristle pattern and a reversal of the sexual dimorphism for abdominal bristle number. Temperature sensitive with a TSP in the pupal period. Both sexes fertile.

sm: see *smk*

sma: smaller

location: 1-29.9.
origin: Induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).
discoverer: Fahmy, 1953.
references: 1958, DIS 32: 75.
phenotype: Body small. Eye color frequently dark. Viability and fertility good. RK2.
other information: One allele each induced by CB. 1528, CB. 1540, CB. 2511, CB. 3007, CB. 3025, CB. 3026, CB. 3034. Two alleles induced by CB. 1414.

small: see *sml*

small body: see *sby*

small body 62: see *srb*

small bristles: see *sbr*

small eye: see *os^s*

small narrow: see *smn*

small optic lobes: see *sol*

small pallid: see *smp*

small round: see *srd*

small thin: see *sth*

small thorax: see *smt*

small tumoroid: see *stu*

small wing: see *sl*

smaller: see *sma*

smaller body: see *srb*

smaller eye: see *sme*

smaller thinner: see *smh*

smalloid: see *smd*

smb: slim bristle

location: 1-23.1.
origin: Induced by ethyl methanesulfonate (CB. 1528).
discoverer: Fahmy, 1956.

phenotype: Bristles thin and rather short. Male viable and fertile; female sterile. RK2.
other information: One allele induced by CB. 1540.

smd: smalloid

location: 1-61.1.
origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).
discoverer: Fahmy, 1954.
references: 1958, DIS 32: 75.
phenotype: Rather small body. Eyes frequently dark. Viability and fertility good. RK2.
cytology: Placed in salivary chromosome region 18A4-18B8 on the basis of its inclusion within the deficiency resulting from recombining left end of *In(1)y⁴* = *In(1)IA8-B1;18A3-4* with right end of *In(1)sc⁹* = *In(1)IB2-3;18B8-9* (Norton and Valencia, 1965, DIS 40: 40).
other information: One allele each induced by CB. 1414, CB. 1540, CB. 1592, and CB. 3007. Two alleles each induced by CB. 1506 and CB. 1528. Seven alleles induced by CB. 3025 and 10 by X rays.

***sme: smaller eye**

location: 1-68.9.
origin: Induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).
discoverer: Fahmy, 1955.
references: 1959, DIS 33: 91.
phenotype: Small fly with small, round, and slightly dark eyes. Wings occasionally diverge. Male sterile; viability about 50% wild type. RK2.
other information: One allele induced by CB. 3051.

smell blind: see *sbl*

***smh: smaller thinner**

location: 1-1.5.
origin: Induced by methyl methanesulfonate (CB. 1540).
discoverer: Fahmy, 1956.
references: 1959, DIS 33: 91.
phenotype: Rather small fly with thin bristles. Both sexes viable and fertile. RK2.

***smk: smoky**

location: 2-58.6.
origin: Ultraviolet induced.
discoverer: Edmondson and Meyer, 49d.
synonym: *sm* (preoccupied).
references: 1949, DIS 23: 61.
phenotype: Body color dark, especially along sides of thorax. Similar to *e^s* but somewhat lighter. At 27°, female sterile and male fertile; at 17°, both sexes fertile. Viability and classification good. RK2.

***sml: small**

location: 1-25.
origin: Induced by P³².
discoverer: Bateman, 1950.
references: 1950, DIS 24: 56.
phenotype: Body small; wings short; eyes small, rough, and bulging. Thoracic hairs irregular. Eclosion delayed. Ten percent normal viability. RK3.

***smn: small narrow**

location: 1-45.7.

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1955.

references: 1959, DIS 33: 91.

phenotype: Fly weak and inviable, usually dies within 48 hr of eclosion. Wings frequently upheld slightly. Abdomen narrow. RK3.

smo: smoothened

location: 2-4.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Embryonic lethal. All denticles in abdominal segments point posteriorly. At 18° naked cuticle deleted and denticle belts of adjacent segments fused and locally arranged as mirror-image duplications.

alleles: Three ethyl-methanesulfonate-induced cold-sensitive alleles, *smo*¹, *smo*², and *smo*³ (originally designated *IIG*, *IIX*, and *Q14*).

smoky: see *smk*

smooth: see *sm*

smoothened: see *smo*

***smp: small pallid**

location: 1-25.6.

origin: X ray induced.

discoverer: Fahmy, 1954.

references: 1959, DIS 33: 91.

phenotype: Fly quite small and lightly pigmented. Bristles slightly thin. Occasional eye misshapen. Male viable and fertile. Female sterile. RK2.

***smt: small thorax**

location: 1-51.9.

origin: Induced by L-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 75.

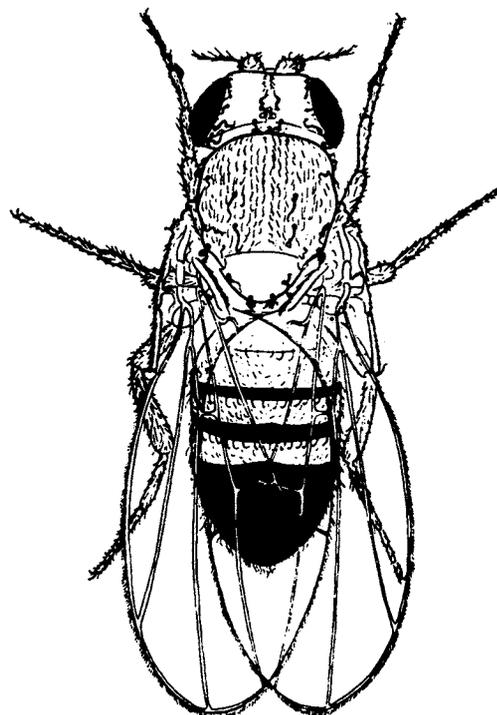
phenotype: Thorax and head small. Wings correspondingly short but of normal width and frequently wavy. Both sexes fertile. Viability about 50% wild type. RK2.

sn: singed

location: 1-21.0.

references: Bender, 1960, Genetics 45: 867-83.

phenotype: Macrochaetae deformed, from short and gnarled to wavy, depending on allele. Similarly, microchaetae may be straight or wavy. Electron-microscope examination of developing bristle shaft shows flattened fiber bundles around periphery, which occupy but 5% of cross-sectional profile, compared to wild type, which have fiber bundles that are circular in cross section and occupy 20% of cross-sectional area [Overton, 1967, J. Morph. 122: 367-80 (fig.)]. Females homozygous for the most extreme alleles are completely sterile; vitellogenesis defective. Eggs laid by *sn*^f homozygotes are normal in number, but are short, blunt, and wrinkled with small blunt dorsal appendages [Mohr, 1922, Z. Indukt.



sn: singed

From Mohr, 1922, Z. Indukt. Abstamm. Vererbungs. 28: 1-22.

Abstamm. Vererbungs. 28: 1-22 (fig.)). Sterility autonomous in transplants (*sn*^f; Clancy and Beadle, 1937, Biol. Bull. 72: 47-56; Perrimon and Gans, 1983, Dev. Biol. 100: 365-73). Heterozygotes between female-sterile and fertile alleles are fertile, between female sterile alleles are sterile.

alleles: The *sn* locus seems to be a very favorable site for *P*-element insertion; a number of the alleles listed below were isolated from natural populations in the USSR or are derivatives thereof. These alleles are highly mutable, generating innumerable derivative mutable normal-appearing and extreme singed alleles; some have been shown to carry *P*-element sequences at *sn* (Golubovsky, Ivanov, and Green, 1977, Proc. Nat. Acad. Sci. USA 74: 2973-75; Green, 1977, Proc. Nat. Acad. Sci. USA 74: 3490-93). Also some display slight increases in the rate of nondisjunction from *Basc* (Golubovsky, 1983, DIS 59: 40-42). Bender has classified *sn* alleles into four classes: class 1 = female sterile with gnarled macrochaetae and kinky microchaetae; class 2 = female fertile with kinky macrochaetae only; class 3 = female fertile with gnarled macrochaetae and kinky microchaetae; class 4 = female sterile with gnarled macrochaetae only. To these Golubovsky and Kozlovskaya (1978, DIS 53: 141-42) have added class 5 = kinky microchaetae only. Derivative alleles have sometimes been designated generically as *sn*^f, *sn*^m, *sn*^s, and *sn*^{ex}, for faint, moderate, strong, and extreme phenotypes; these symbols do not specify particular alleles (e.g., Zakharov and Golubovsky, 1984, Genetika 20: 1117-24). Unstable alleles also classed as *sn*^A and *sn*^B, *sn*^A alleles mutating to normal and back to the original phenotype only and *sn*^B alleles mutating to an array of intermediate states as well as to normal.

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	ref ^α	comments ^β
sn (+) γ	P	Engels	11, 12, 43	0.95 kb <i>P</i> insert at -0.589 to 0.582; <i>sn</i> ^W derivative; unstable
sn 1	spont	Mohr, 18j25	7, 35, 43	class 1; sequence normal
sn 2	spont	Bridges, 1912	7, 37, 43	class 2; lesion at 8.0 to 10.5 kb
sn 2-2	spont		17	class 1
sn 3	spont	Mohr, 22f11	7, 36, 43	class 3; 0.3 kb deletion in -0.9 to 0.0 kb
sn 4	spont	Bridges, 30126	7, 43	class 2; sequence normal
sn 4	spont		17	class 3
sn 5	spont	Bridges 30b5	7	class 1
sn 5S	spont	Skinner, 42c18	26, 43	class 1; 2 kb insert in -0.9 to 0.0 kb
sn 7	spont	Berg	8, 17	class 3; unstable
sn 7-27	spont		17	class 5
sn 7-55	spont		17	class 3
sn 8	spont	Berg	8, 17	class 3; stable
sn 9	spont		17	class 2
sn 9-152	spont		17	class 3, unstable
sn 9e	P	Green	20	functional <i>P</i> element insert
sn 11	spont	Berg	8, 17	class 2; unstable
sn 13a1	X ray, R(1)2	Hannah, 1947	45	<i>Tp(1;3)6C;7C9-10;79E</i> ; breakpoint at 0.0 to 5.5 kb
sn 14e	P	Green	20	functional <i>P</i> element insert
sn 15	P	Green	20	functional <i>P</i> element insert
sn 17	spont		13	<i>P</i> sequences at 7D
sn 19Bb5	X ray	Valencia	45	<i>Tp(1;3)3C1-2;7C9-10;72A-B</i>
sn 26-7	spont		16, 17, 24, 47	class 3; unstable
sn 27-10	mustard	Sobels, 57j	44	
sn 27-49	mustard	Sobels, 57j	44	
sn 29-1	X ray	Sobels, 57l	44	
sn 29-11	spont		17	class 3; in <i>Basc</i>
sn 31f	X ray	Patterson	39	class 1
sn 33-13	spont		16, 17, 47	class 2, unstable
sn 33-13a	spont		17	class 5
sn 34e		Duncan, 34e20	10, 43	class 2; complex lesion 2.4 to 4.3 kb
sn 34p	HD		12a	
sn 36a	spont	Spencer, 36a21	7, 43	class 4; 5.5 kb insert in -1.2 to 0.0 kb
sn 37b	spont	Poulson, 37b	40, 41	class 1
sn 39k		Buzzati-Traverso, 39k19	9	class 1
sn 41l	spont	Oliver, 41i25	38	class 1
sn 42	spont		17	class 5
sn 42-5	spont		16, 17	class 3; unstable
sn 44-12	spont		17	class 1
sn 45-7	spont	Ivanov, 73j	23	stable; in <i>Basc</i>
sn 46a	X ray	Belgovsky	6	class 2
sn 48h	X ray	Lindsley, 48h11	31	class 2
sn 49 γ	spont		18, 25, 46	unstable; also <i>chw</i> mutant
sn 49h	32 P	King, 49h	42	class 1
sn 49-5	spont		17, 24	class 1; unstable
sn 50-18	spont		16, 17, 24	class 2; unstable
sn 50k	Ives		7, 27	class 1; sequence normal
sn 55a	spont	Hillman, 55a	22	class 3
sn 57c	spont	Kadel	28	class 1
sn 61k	γ ray	Mickey, 61k	33	class 1
sn 61k2	γ ray	Mickey, 61k	33	class 3
sn 63-15 γ	spont	Ivanov	16, 17, 23, 24, 47	class 5; unstable
sn 63-15a	spont		17	class 5
sn 63a	radio waves	Mickey, 63a	32	class 1
sn 63b	radio waves	Mickey, 63b19	32	
sn 65a	X ray	Becker	5	class 4
sn 68	NNG	Kaufman	29	class 1
sn 73-8	spont		17, 24	class 1; in <i>Basc</i>
sn 77-27 γ	spont	Ivanov, 73j	17, 23, 24, 47	class 1; unstable
sn 78	spont	Anxolabéhère, 73b	3	synonym: <i>frisé</i>
sn 79-15	spont		16, 17, 47	class 2; stable
sn 79-22	spont		17, 47	class 1
sn 79b10	P	Green	43	class 1; 1 kb <i>P</i> insert at -0.667 to -0.660 kb
sn 79f22	P	Green	43	class 1; 1 kb <i>P</i> insert at -.667 to -.660 kb
sn 79h2	P	Green	43	class 1; 1 kb <i>P</i> insert at -.667 to -.660 kb
sn 8317(1)	P	Green	43	class 1; 2.9 kb <i>P</i> insert at -.667 to -.660 kb
sn 8317(2)	P	Green	43	class 1; 2.9 kb <i>P</i> insert at -.667 to -.660 kb
sn 8317(3)	P	Green	43	class 1; 2.9 kb <i>P</i> insert at -.667 to -.660 kb
sn 8317(15)	P	Green	43	2.9 kb <i>P</i> insert
sn 8317(17)	P	Green	43	class 1; 2.9 kb <i>P</i> insert at -.667 to -.660 kb
sn 84-6	spont		16, 17, 24, 47	class 3; unstable
sn 84-6a	spont		17	class 5
sn 88-9	spont		16, 17, 47	class 5; stable
sn 90-9	spont		24, 16, 17, 47	class 2; unstable

allele	origin	discoverer	ref ^α	comments ^β
<i>sn</i> ¹¹⁰	spont		13	<i>P</i> element insert at 7D
<i>sn</i> ⁶³³⁻⁴⁵				
<i>sn</i> ^A	spont	Golubovsky	14, 15	a class of mutable alleles; e.g., <i>sn</i> ⁷⁷⁻²⁷
<i>sn</i> ^{A1}	EMS		4	class 1
<i>sn</i> ^{A2.4}	P	Engels	43	class 1; 1.2 kb insert at -0.684 to -0.677 kb
<i>sn</i> ^B	spont	Golubovsky	14, 15	a class of mutable alleles; e.g., <i>sn</i> ⁶³⁻¹⁵
<i>sn</i> ^{B332.1}				<i>In(1)7D1-2;17C</i> ; breakpoint at -0.6.
<i>sn</i> ^{B337.2}				<i>In(1)7D1-2;17C</i> ; breakpoint at -0.6.
<i>sn</i> ^C	spont	Muller	7	class 1
<i>sn</i> ^{CFL3}	P	Osgood	43	class 1; 2.9 kb <i>P</i> insert at -0.667 to -0.660 kb
<i>sn</i> ^{CFL5}	P	Osgood	43	1.3 kb <i>P</i> insert
<i>sn</i> ^{cm γ}	P	Hawley	21	class 3; 628 base pair <i>P</i> insert at -0.667 kb
<i>sn</i> ^{e γ}			11, 12, 43	1.3 kb <i>P</i> insert at -0.589 to 0.582; <i>sn</i> ^w derivative; unstable derivative of <i>sn</i> ^{cm}
<i>sn</i> ^{ext γ}	P		21	
<i>sn</i> ^{h12-1}	spont, MR	Green	19	unstable
<i>sn</i> ^{h12-2}	spont, MR	Green	19	unstable
<i>sn</i> ^{h12-3}	spont, MR	Green	19	unstable
<i>sn</i> ^{h12-4}	spont, MR	Green	19	unstable
<i>sn</i> ^{h12-5}	spont, MR	Green	19	unstable
<i>sn</i> ^{h12-6}	spont, MR	Green	19	unstable
<i>sn</i> ^K		Krivshenko	1	class 1
<i>sn</i> ^{K1}	EMS		30	class 1
<i>sn</i> ^{K2}	EMS		30	class 1
<i>sn</i> ^{K3}	EMS		30	class 1
<i>sn</i> ^{K4}	EMS		30	class 1
<i>sn</i> ^{KH36}	P	Osgood	43	0.4 kb <i>P</i> insert
<i>sn</i> ^{KH40}	P	Osgood	43	1.0 kb <i>P</i> insert
<i>sn</i> ^{KHL1}	P	Osgood	43	0.4 kb <i>P</i> insert
<i>sn</i> ^{KHL3}	P	Osgood	43	0.4 kb <i>P</i> insert
<i>sn</i> ^{KHL4}	P	Osgood	43	0.6 kb <i>P</i> insert
<i>sn</i> ^{KHL5}	P	Osgood	43	0.4 kb <i>P</i> insert
<i>sn</i> ^{M1}	EMS	Mohler	34	class 1
<i>sn</i> ^{M2}	EMS	Mohler	34	class 1
<i>sn</i> ^{M3}	EMS	Mohler	34	class 1
<i>sn</i> ^{M4}	EMS	Mohler	34	class 1
<i>sn</i> ^{MJ19.3}	P	Engels	43	0.5 kb <i>P</i> insert
<i>sn</i> ^{MK7}	P	Engels	43	2.9 kb <i>P</i> insert at -0.589 to -0.582
<i>sn</i> ^{MH18.2}	P	Engels	43	1.0 kb <i>P</i> insert
<i>sn</i> ^{MH26.5}	P	Engels	43	class 1; 2.9 kb <i>P</i> insert at -0.667 to -0.660
<i>sn</i> ^{MI17}	P	Engels	43	0.5 kb <i>P</i> insert
<i>sn</i> ^{MR2}	spont		13	<i>P</i> element at 7D
<i>sn</i> ^{qr}	spont	Ménsua	2	class 3; to the right of <i>sn</i> ¹ ; <i>qr</i> = <i>quetas recordatas</i>
<i>sn</i> ^{w γ}	P	Engels	11, 12, 43	class 3; hypermutable; 2.25 kb <i>P</i> insert at -0.589 to 0.582
<i>sn</i> ^{X2}	X ray	Muller	7, 43	class 1; 0.1 kb insert in -1.9 to -1.2 same insert present in parental chromosome

^α 1 = Agol, 1936, DIS 5: 7; 2 = Alvarez, 1980, DIS 55: 193; 3 = Anxolabèhère and Périquet, 1973, DIS 50: 21; 4 = Gans, Audit, and Masson, 1975, Genetics 81: 683-704; 5 = Becker, 1967, DIS 42: 40; 6 = Belgovsky, 1946, DIS 20: 63; 7 = Bender, 1960, Genetics 45: 867-83 (fig.); 8 = Berg, 1974, DIS 51: 100; 9 = Buzzati-Traverso, 1940, DIS 13: 49; 10 = Duncan, 1935, DIS 4: 10; 11 = Engels, 1979, Proc. Nat. Acad. Sci. USA 76: 4011-15; 12 = Engels, 1981, Genetics 98: 565-87; 12a = Furman and Zabanov, 1988, DIS 67: 37; 13 = Gerasimova and Ilyn, 1984, DIS 60: 111-12; 14 = Golubovsky, 1978, DIS 53: 171; 15 = Golubovsky, 1977, Genetika 16: 1605-11; 16 = Golubovsky, Ivanov, and Green, 1977, Proc. Nat. Acad. Sci. USA 74: 2973-75; 17 = Golubovsky and Kozlovskaya, 1978, DIS 53: 141-42; 18 = Golubovsky and Zakharov, 1980, DIS 55: 49-51; 19 = Green, 1977, Proc. Nat. Acad. Sci. USA 74: 3490-93; 20 = Green, 1986, Proc. Nat. Acad. Sci. USA 83: 1036-40; 21 = Hawley, Steuber, Marcus, Sohn, Baronas, Cameron, Zitron, and Chase, 1988, Genetics 119: 85-94; 22 = Hillman, 1957, DIS 31: 82; 23 = Ivanov, 1974, DIS 51: 71; 24 = Ivanov, 1978, DIS 53: 119; 25 = Ivanov and Golubovsky, 1977, Genetika 13: 655-66; 26 = Ives, 1943, DIS 17: 50; 27 = Ives and Noyes, 1951, Anat. Rec. 111: 565; 28 = Kadel, 1957, DIS 37: 83; 29 = Kaufman, 1969, DIS 44: 44; 30 = Komitopoulou, Gans, Margaritis, Kafatos, and Masson, 1983, Genetics 105: 897-920; 31 = Lindsley, 1949, DIS 23: 60; 32 = Mickey, 1963, DIS 38: 29; 33 = Mickey, 1963, DIS 38: 31; 34 = Mohler and Carrol, 1984, DIS 60: 236-41; 35 = Mohr, 1922, Z. Indukt. Abstammungs. Vererbungs. 28: 1-22 (fig.); 36 = Mohr, 1923, Hereditas 4: 142-60 (fig.); 37 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 235; 38 = Oliver, 1942, DIS 16: 53; 39 = Patterson, 1934, DIS 2: 59; 40 = Poulson, 1938, DIS 10: 55; 41 = Poulson, 1939, DIS 12: 49; 42 = Poulson and King, 1949, DIS 23: 63; 43 = Roiha, Rubin, and O'Hare, 1988, Genetics 119: 75-83; 44 = Sobels, 1958, DIS 32: 85; 45 = Valencia, 1966, DIS 41: 58; 46 = Yurcheuko, Zakharov, and Golubovsky, 1984, Mol. Gen. Genet. 194: 279-85; 47 = Zakharov and Golubovsky, 1984, Genetika, 20: 1117-24.

^β Class according to Bender (1960); coordinates according to Roiha, Rubin, and O'Hare (1988).

^γ More detailed information given below.

cytology: Placed in 7D1-2 by *in situ* hybridization (Spradling; Engels).

molecular biology: 40 kb cloned; from coordinates -15 to +25 with the origin at an arbitrary *EcoRI* site and positive values to the right. Six out of nine alleles tested have lesions between -1.9 and +10.5 kb. Three breakpoints [*Dp(1;2)sn*^{13a}, *In(1)sn*^{B332.1} and *In(1)sn*^{B337.2} (Roiha,

Rubin, and O'Hare, 1988, Genetics 119: 75-83)] between -0.6 and +5.5; *P* element hotspot at -0.7 to 0.0 with at least four different insertion sites. Gene consists of six exons with three polyadenylation sites resulting in three sizes for the 3' exon and three sizes of RNA, 3.6, 3.3, and 3.0 kb. Structure of gene-exon 1 from -0.7 to 0.0 kb; exon 2 from 11.0 to 11.3 kb; exon 3 from 12.3 to 12.7

kb; exon 4 from 12.8 to 13.3 kb; exon 5 from 13.4 to 13.6 kb; exon 6 from 13.7 to 14.4 or 14.7 or 15.0 kb. A single 59-kd protein predicted.

other information: Locus divided into three recombinationally different sites from distal to proximal: (*sn*³ and *sn*^{36a}) (*sn*² and *sn*⁴) (*sn*¹, *sn*⁵ and *sn*^{50k}) (Ives and Noyes, 1951, Anat. Rec. 111: 565; Hexter, 1955, Proc. Nat. Acad. Sci. USA 41: 921-25; Genetics 42: 376). Of these, *sn*³ and *sn*^{36a} show lesions between -0.9 and 0.0 kb, whereas *sn*² has a lesion between 8.0 and 10.5. The others show no detectable molecular lesions, and are presumed to be point mutations within the coding region between 11.1 and 13.8 kb.

***sn*^{36a}**

phenotype: Macrochaetae gnarled in a fairly extreme manner. Microchaetae wild type. *sn*^{36a} is only allele to cause pronounced reduction in replication of oocyte nurse cell DNA [King and Burnett, 1957, Growth 21: 263-80 (fig.)]. Also causes more extreme retardation of vitellogenesis than other female-sterile *sn* alleles (Bender). *sn*^{36a} *sn*⁴ homozygote has nearly normal bristles and is sterile. RK1.

***sn*⁴⁹**

phenotype: A strong allele of *sn* recovered from a natural population. Associated with simultaneous mutation to club wing, *clw*, a defect in wing expansion with low penetrance. *sn*⁴⁹ is unstable, producing an array of derivatives that are in turn stable or unstable. and the expression of *clw* differs among them. It mutates to *sn*⁺ and back at a rate of approximately 10⁻³; a rare moderate singed derivative exhibits an approximately ten-fold elevation in mutation frequency, mutating either back to the strong allele or to an unstable normal allele; a single extreme singed derivative of a normal derivative of the moderate allele produces strong-singed and non-singed derivatives, which can in turn revert to the extreme allele and in the case of the strong derivative to non singed (Yurchenko, Zakharov, and Golubovsky, 1984, Mol. Gen. Genet. 194: 279-85).

***sn*⁶³⁻¹⁵**

phenotype: Moderate *sn* phenotype. Prototype type B mutable allele; mutation rate 0.1 to 1.2%. Produces both extreme singed and normal-appearing derivatives as well as an array of intermediate phenotypes. In addition strongly reversible alleles (25-50 x more mutable than parental allele) are produced. This allele also associated with increased rate of mutation to *fw*.

***sn*⁷⁷⁻²⁷**

phenotype: Extreme *sn* phenotype. Prototype type A mutable allele; mutates to an unstable *sn*⁺, which mutates back to extreme alleles; no intermediate alleles recovered.

***sn*^{cm}**

phenotype: An allele of *sn* that is mutable in dysgenic but not in non-dysgenic genotypes. Mutation takes place in two directions; one is to apparent stable reversions and the other to a more extreme phenotype, *sn*^{ex}, which is in turn unstable.

molecular biology: Contains a 628-bp defective *P* element inserted at coordinate -667. *sn*⁺ derivatives characterized by precise or nearly precise excision of the element.

sn^{ex} derivatives contain duplications of the defective *P* element located close to the right of the original *P* in inverted orientation.

***sn*^w: singed-weak**

phenotype: Weak singed phenotype, probably class 2. Highly mutable in dysgenic genotypes; 40% to 60% of offspring are either normal (*sn*⁽⁺⁾) or extreme singed (*sn*^e) in phenotype. These derivatives are in turn mutable, but at much lower levels. Completely stable in non-dysgenic genotypes.

molecular biology: Contains a pair of adjacent defective *P* elements of 0.95 kb and 1.15 kb inserted in head-to-head orientation between -0.589 and -0.582 kb. All *sn*^e derivatives tested retain only the 1.15 kb element and all normal-appearing derivatives retain only the 0.95 kb element, thus the symbol *sn*⁽⁺⁾.

***sna*: snail**

location: 2-51.

synonym: *l(2)br28 = l(2)35Db*.

references: Simpson, 1983, Genetics 105: 615-32.

Grau, Carteret, and Simpson, 1984, Genetics 108: 347-60.

phenotype: Embryonic lethal. Partially dorsalized. In homozygotes for strong alleles, ventral furrow not formed at gastrulation; however, endoderm invaginates, cephalic furrow formed, and germ-band elongation takes place. Embryo has few if any mesodermally derived internal tissues. Homozygotes for weak alleles gastrulate normally, but die as late embryos without differentiating normal internal tissues. Many embryos make normal ectodermal derivatives: larval hypoderm with normal or reduced denticle belts, mouth hooks, and spiracles, but tracheae seen only in weaker alleles. Head involution abnormal and anterior end of embryo twisted in the egg owing to extra length. Some embryos fail to make normal cuticle and resemble long folded tubes filled with yolk. *sna* heterozygotes produced from mothers heterozygous for *dl* or a deficiency for *dl* show reduced viability; this effect is more extreme at elevated temperatures. Defect in *sna* embryos enhanced by heterozygosity for *Df(2L)75c* or *Df(2L)fn2*. No maternal effect in germ-line clones. No effect on pattern of *fz* expression (Carroll, Winslow, Twombly, and Scott, 1987, Development 99: 327-32). *sna*/+ embryos have delayed ventral furrow formation.

alleles: Alleles of varying strengths reported. Weak alleles appear to be hypomorphic based on their phenotypes over deficiencies versus in homozygotes. Strong alleles behave as amorphs. *Trans* heterozygotes of weak alleles produce few escapers, which may exhibit missing halteres or more rarely hemithorax.

allele	origin	discoverer	synonym	ref ^α	comments
<i>sna</i> ¹	EMS	Harrington	<i>HG31</i>	1, 3, 5	strong allele
<i>sna</i> ²	EMS	Grau	<i>EY1</i>	3	strong allele
<i>sna</i> ³	EMS	Grau	<i>EY2</i>	3	
<i>sna</i> ⁴	EMS	Grau	<i>EY3</i>	3	weak allele
<i>sna</i> ⁵	X ray	Simpson	<i>RI</i>	3, 5	weak allele
<i>sna</i> ⁶	X ray	Simpson	<i>RY1</i>	2, 3, 5	strong allele; 1.6 kb deletion
<i>sna</i> ⁷	X ray	Simpson	<i>RY2</i>		<i>T(2;3)24B-C</i> ; <i>35C-D;41;81</i>
<i>*sna</i> ⁸	X ray	Simpson	<i>RY3</i>		
<i>sna</i> ⁹	EMS	Simpson	<i>VI</i>		

allele	origin	discoverer	synonym	ref ^α	comments
<i>sna</i> ¹⁰	EMS	Simpson	V2		
<i>sna</i> ¹¹	EMS	Simpson	V3		
<i>sna</i> ¹²	EMS	Simpson	V4		
<i>sna</i> ¹³	EMS	Simpson	V5		
<i>sna</i> ¹⁴	EMS	Simpson	V6		
<i>sna</i> ¹⁵	EMS	Simpson	V7		
<i>sna</i> ¹⁶	EMS	El Messal	VD1		
<i>sna</i> ¹⁷	EMS	El Messal	VD2		
<i>sna</i> ¹⁸	EMS	Nüsslein-Volhard	IIG05	3, 4, 5	strong allele
<i>sna</i> ¹⁹	X ray	Nüsslein-Volhard	4.26		
<i>sna</i> ²⁰	X ray	Nüsslein-Volhard	18.19		

^α 1 = Ashburner, Tsubota, and Woodruff, 1982, Genetics 102: 401-20; 2 = Boulay, Dennefeld, and Alberga, 1987, Nature (London) 330: 395-98; 3 = Grau, Carteret, and Simpson, 1984, Genetics 108: 347-60; 4 = Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82; 5 = Simpson, 1983, Genetics 105: 615-32.

cytology: Placed in 35C4-D2 based on its inclusion in *Df(2L)b80e4* = *Df(2L)35C4;35D1-2* but not in *Df(2L)osp18* = *Df(2L)35B1-2;35C4-5*.

molecular biology: Gene isolated from walk on the basis of DNA lesions identified in five independent *sna* alleles. Sequences from the region identify a 1.7 mRNA on Northern blots. A 1.7 kb cDNA of nearly full length cloned and sequenced; injection of antisense RNA into early embryos promotes phenocopy of *sna* development. Transcription from proximal to distal on 2L. cDNA contains a single open reading frame of 1,170 nucleotides, corresponding to 390 amino-acid residues with a calculated relative molecular mass of 43,000. The carboxy terminus of the conceptual polypeptide contains five zinc-finger motifs (Boulay, Dennefeld, and Alberga, 1987, Nature (London) 330: 395-98).

other information: The *Sco* transposition brings a portion of the *noc* gene into juxtaposition with *sna*; subsequent rearrangements with breakpoints within the *sna* gene are able to revert the *Sco* phenotype (McGill, Chia, Karp, and Ashburner, 1988, Genetics 119: 647-61).

snake: see *snk*

snap: see *snp*

snb: see *p^{snb}*

***Snb*: Snow blind (B. Ondek)**

location: 3-104.7.

origin: Induced by ethyl methanesulfonate.

discoverer: Ondek and Hardy.

phenotype: Deep pseudopupil absent in white-eyed flies raised in either light or dark. Electroretinogram displays no receptor potential. Light and electron microscopy reveal no organized ommatidial structure; widespread degeneration and no apparent photoreceptor cells.

snf: see *fs(1)A1621*

***snk*: snake**

location: 3-52.1.

references: Anderson and Nüsslein-Volhard, 1984, Nature 311: 223-27.

Anderson and Nüsslein-Volhard, 1986, Gametogenesis and the Early Embryo, Alan R. Liss, Inc., pp. 177-94.

phenotype: Maternal effect lethal. Embryos produced by homozygous mothers show extreme dorsalized phenotype; only dorsally derived cuticle remains, resulting in a

hollow tube of dorsal ectoderm. No paternal rescue, even by two doses of *snk*⁺; dorsalizes the expression of *ftz* such that entire *ftz* stripes in the blastoderm exhibit the wide dorsal conformation and do not narrow ventrally as is normal (Carroll, Winslow, Twombly, and Scott, 1987, Development 99: 327-32). Responds most strongly of all maternal dorsalized mutants to rescue by injection of cytoplasm and poly A⁺ RNA from wild-type embryos. Injection of wild-type cytoplasm or cytoplasm from other dorsalizing maternal-effect mutants into pre-pole-cell snake embryos fully restores the normal dorsal-ventral pattern, and more than 20% can develop into normal adults; the site of injection appears to be unimportant. Partial rescue also achieved by the injection of poly A⁺ but not poly A⁻ RNA isolated from cleaving embryos.

alleles:

allele	origin	discoverer	synonym
<i>snk</i> ¹	EMS	Rice	<i>mel(3)4, snk</i> ⁰⁷³
<i>snk</i> ²	EMS		<i>snk</i> ²²⁹
<i>snk</i> ³	EMS		<i>snk</i> ²³³
<i>snk</i> ⁴	EMS		<i>snk</i> ^{rm4}

cytology: Placed in 87D6-13 on the basis of the molecular observation that the right breakpoint of *Df(3R)ry36* interrupts the gene and it is to the right of the molecular deficiency associated with *ry*⁵⁰⁶. *Df(3R)ry36* is not detectable cytologically, but it includes *ry* but not *Hsc2* or *pic*.

molecular biology: Gene identified by finding a DNA segment capable of rescuing *snk* by germ-line transformation. This 13.5 kb segment begins and ends in the loci of *ry* and *Hsc2*, respectively. Northern blots of embryonic RNA probed with a 2.4 kb *EcoRI* internal fragment from the above segment reveal a 1.65 kb transcript; a slightly smaller pupal transcript that differs from the embryonic transcript at its N and C termini is recognized by the same probe. Transcription is from right to left, and the cDNA sequence predicts a protein of 430 residues. The conceptual sequence reveals homology to several known serine proteases over its 246 C-terminal amino acids; may also have a Ca²⁺ binding site near its amino-terminus (DeLotto and Spierer, 1986, Nature (London) 323: 688-92).

***snl*: sonless**

location: 1-56.3.

discoverer: Paré, 1964.

references: Colaianne and Bell, 1970, Genetics 65: 619-25.

Colaianne and Bell, 1972, Genetics 72: 293-96.

Colaianne and Bell, 1972, DIS 48: 20.

phenotype: Maternal-effect recessive lethal or semilethal. *snl/snl* females crossed to normal males produce fewer than 10% of the expected number of sons. Lethality can be rescued by a paternally derived *snl*⁺ allele as demonstrated by the survival of exceptional sons but not daughters (i.e., *snl*^{+/0} but not *snl/snl/Y*) of the above mating. Lethality occurs during embryonic or early larval stages. *snl*^{+/+} females crossed to *snl/Y* males produce normal offspring in a 1:1 sex ratio. Crosses between *snl* males and females are nearly sterile, producing extreme sex ratios favoring females among surviving offspring. Data indicate that homozygosity for *tra* enhances and for *dsx* reduces the lethality of sons of *snl* mothers.

other information: Complements both *r* and *fu*, nearby genes that also act as maternal-effect lethals.

sno: strawberry notch

location: 1-41.8 (based on mapping of *sno*⁴).

synonym: *g-l: glossy like*.

references: Lefevre and Peterson, 1972, DIS 48: 126-27.

Lefevre and Wright, 1976, Genetics 83: s44-45.

phenotype: Males and females homozygous for viable alleles have notched wings, thickened, Confluens-like wing veins with deltas at the junctions of the longitudinal veins and the margins, extra hairs on thorax and wings, shortened tarsal segments, and roughened, shiny bright, somewhat mottled eyes, closely resembling *fa*⁸. All macrochaetae thin and delicate. Phenotype almost completely suppressed by euchromatic duplications of the Notch locus, e.g., *Dp(1;2)51b = Dp(1;2)3C1-2;3D6-7;52E*. Insertions into heterochromatin, e.g., *w*⁺*Y*, are less effective in suppression. *fa sno* males and *fa sno/+ sno* females have exaggerated phenotypes and are semi-lethal; *spl sno* less extreme. *sno*¹ does not survive when raised at 29° or in *Rpl1215^{Ubl}/+* genotypes; exhibits slight dominance in *trans* heterozygotes with *Rpl1215^{Ubl}* (Mortin and Lefevre, 1981, Chromosoma 82: 237-47).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>sno</i> ¹	EMS	Lefevre, 71e	<i>sno</i> ^{71e}	2, 3, 5	heat-sensitive lethal;
<i>sno</i> ²	EMS	Lefevre		2	<i>Tp(1;3)C92</i>
<i>sno</i> ^{3β}	EMS	Mayoh	<i>l(1)HM16</i>	4	cold-sensitive lethal
<i>sno</i> ^{4β}	EMS	Mayoh	<i>l(1)HM23</i>	4	cold-sensitive lethal
<i>sno</i> ⁵	EMS	Lefevre, 75b	<i>sno</i> ^{75b}	3	heat-sensitive
<i>sno</i> ^{6β}	EMS	Falke	<i>l(1)TW2</i>	1	cold-sensitive lethal
<i>sno</i> ⁷	EMS	Lefevre, 76a	<i>sno</i> ^{76a}	5	
<i>sno</i> ⁸	EMS	Lefevre	<i>l sno</i> ^{DC800}	5	lethal; lethal in heterozygotes with <i>Rpl1215^{Ubl}</i>
<i>sno</i> ⁹	ENU	Scott		6	lethal
<i>sno</i> ¹⁰	ENU	Scott		6	lethal

^α 1 = Falke and Wright, 1975, Genetics 81: 655-82; 2 = Lefevre and Peterson, 1972, DIS 48: 126-27; 3 = Lefevre and Wright, 1976, Genetics 83: s44-45; 4 = Mayoh and Suzuki, 1973, Can. J. Genet. Cytol. 15: 237-54; 5 = Mortin and Lefevre, 1981, Chromosoma 82: 237-47; 6 = Scott, 1987, Ph.D. thesis, University of California, San Diego.

^β Further phenotypic description below.

cytology: Placed in 11D9-10 based on its inclusion in *Df(1)N12 = Df(1)11D1-2;11F1-2* and the breakpoint in 11D of *Tp(1;3)C92 = Tp(1;3)6E1-2;11D9-10*.

sno³

phenotype: No survivors at 17°; short fine bristles at 25°. Females become sterile when held at 17°.

sno⁴

phenotype: No survivors at 17°; normal at 25°. Females become sterile when held at 17°.

sno⁶

phenotype: Survivors of development at 17° have, in addition to the phenotype described above, reduced or absent ocelli, dark head, small trident, and underdeveloped legs; phenotype normal when raised at 25°. Females become sterile when held at 17°.

snp: snap

location: 3-50.

references: Glover.

snRNA: see under RNA

sno: stonewall

location: 3-{41}.

references: Yu, Berg, and Spradling.

cytology: Located in 70D-E.

so: sine oculis (J.C. Hall)

location: 2-57.1.

origin: Spontaneous.

discoverer: Milani, 1939.

references: 1941, DIS 14: 52.

Buzzatti-Traverso, 1946, DIS 20: 63.

Milani, 1946, Boll. Soc. Ita. Biol. Sper. 23: 111-13.

1951, DIS 25: 79.

1951, Rend. Ist. Lombardo Sci. Lettere, Ser. 3.

Engelmann and Honneger, 1966, Z. Naturforsch. 22B: 1-2.

Hofbauer and Campos-Ortega, 1976, Wilhelm Roux's Arch. Dev. Biol. 179: 275-89.

Fischbach, 1983, Dev. Biol. 95: 1-18.

Fischbach and Lyly-Hünerberg, 1983, Cell Tiss. Res. 231: 551-63.

Helfrich and Engelmann, 1983, Physiol. Entomol. 8: 257-72.

Fischbach and Technau, 1984, Dev. Biol. 104: 219-39.

Helfrich, 1986, J. Neurogenet. 3: 321-43.

Dushay, Rosbash, and Hall, 1989, J. Biol. Rhythms 4: 1-27.

phenotype: Ocelli always absent; eyes usually reduced to small groups of ommatidia, and occasionally missing; eye field sometimes in form of an eye stalk protruding from head with an irregular arrangement of ommatidia; heavy ommatidial disruption with many receptor cells missing. Optic lobes reduced in size, and many flies have no lamina. The reduced volume of adult optic lobes is due to accentuated degeneration of precursor neurons that occurs to a certain degree in normal pupal development (Fischbach, 1983); the increased severity in the mutant includes degeneration of axons in second optic chiasma (Fischbach and Technau, 1984); *sol* enhances this kind of degeneration, but acts on a separate set of precursors for columnar visual system neurons--as confirmed by anatomical analysis of *sol*; *so* double mutant, which ends up with tiny, rudimentary optic lobes (Fischbach and Technau, 1984); *sol*; *so* also leads to a central brain that is smaller than normal due to missing afferents from visual system (Fischbach and Technau, 1984); more specifically, there is a reduction in number of axons in anterior optic track in *so*, and combining *so* with *sol* causes a further reduction, but again, these two genes act independently on separate subsets of such axons (Fischbach and Lyly-Hünerberg, 1983). Histological studies reveal that the eye-antenna disc in third-instar larvae appears normal until differentiation begins, at which time cell death is observed (Hofbauer and Campos-Ortega). More extreme at elevated temperatures; lethal at 30°; temperature-sensitive period for eye defect in third instar. Survival sensitive to elevated temperature at all developmental stages (Ransom, 1980, DIS 55: 126). Mosaic studies demonstrate that *so* acts in developing eye tissue

and that the resulting reduction in retinal innervation leads to death of cells in the lamina and breakdown of medulla and lobula-complex neuropil (Fischback and Technau). Nonphototactic (Benzer, 1967, Proc. Nat. Acad. Sci. USA 58: 1112-19) and visual orientation almost absent (Bülthoff, 1982, DIS 58: 31). Studies of circadian rhythms in *so* show eclosion to be normally periodic (Engelmann and Honneger, 1966); adult activity rhythms are robust, in that *so*, even when thoroughly eyeless, responds to light:dark cues such that it entrains to these conditions (is periodically active vs. inactive, and anticipates the environmental transitions) and subsequently free-runs with obvious circadian periodicities in constant darkness (Helfrich and Engelmann, 1983; Dushay, Rosbash, and Hall, 1989); however, these behavioral rhythms are frequently aberrant, e.g., with "split" active components appearing after several days of free-run and with dual periodicities extractable from the locomotor data (Helfrich, 1986); nearly all adults are dual-period when *so* combined with *sol* (Helfrich, 1986).

allele	origin	discoverer	synonym	ref ^α	comments
<i>so</i> ¹	spont.	Milani, 1939		2	
<i>so</i> ⁺²	spont.	Milani, 1939		2	weaker derivative of <i>so</i> ¹
<i>so</i> ^D	ENU	Sakonju, 1982	<i>Drl</i>	1	gain-of-function allele ^β
<i>so</i> ^{Drv1}		Ashburner			T(2;3)43B1-3;86E14-20
<i>so</i> ^{Drv2}		Ashburner			T(2;3)43B3-8;91A3-8

^α 1 = Craymer, 1984, DIS 60: 235; 2 = Milani, 1941, DIS 14: 52.
^β More complete description below.

cytology: Placed in 43B1-2 based on the cytology of *so*^D revertants (Ashburner).

so^D

phenotype: Heterozygote has very small glazed eyes; more extreme than *Dr*^{mio/+}; *so*^{D/+} has a very clear phenotype, but weaker than that of *Dr*^{mio/+} (i.e., about the size of *Gla*+, but not glazed). Heterozygote has excellent viability; homozygous lethal.

Sod: Superoxide dismutase

location: 3-32.5 [based on 151 *h-gv* recombinants (Jelnes, 1971, Hereditas 67: 291-93)]. Mapped to 34.6 by Finerty using closer markers.

synonym: *To*, *Tetrazolium oxidase*; *cSOD*.

references: Franklin and Chew, 1971, DIS 47: 38.

Lee, Ayala, Misra, 1981, J. Biol. Chem. 256: 8506-09.

Lee, Misra, Ayala, 1981, Proc. Nat. Acad. Sci. USA, 78: 7052-55.

phenotype: The structural gene for Cu, Zn superoxide dismutase [Superoxide: superoxide oxidoreductase; SOD (EC 1.15.1.1)], a homodimer of 15,000 subunit molecular weight that contains two Cu⁺⁺ and two Zn⁺⁺ per molecule. Enzyme catalyzes the dismutation of the superoxide anion, O₂⁻, to H₂O₂, which in turn is converted into H₂O by catalase and peroxidases. Enzyme purified by Lee, Ayala, and Misra (J. Biol. Chem. 256: 8506-09); shows homology to homologous mammalian enzymes but does not crossreact with anti-bovine-erythrocyte-SOD antibodies; specific activity 1.5 times that of other species. Amino acid sequence determined by Lee, Friedman, and Ayala (1985, Arch. Biochem. Biophys. 241: 577-89); 151 amino acid resi-

dues with molecular weight 15,750. Enzyme levels show little variation during development; slight rise in activity during adulthood (Graf and Ayala, 1986, Biochem. Genet. 24: 153-68).

alleles: Two electrophoretic variants, *Sod*^F and *Sod*^S, whose relative frequencies vary greatly among natural populations. *Sod*^S alleles differ from *Sod*^F alleles in having a lysine in place of asparagine at residue 96 (Lee and Ayala, 1985, FEBS Lett. 179: 115-19); fast alleles from California and Tunisia differ at two additional residues, tentatively California alleles have a serine and a glutamine or glutamic acid, where Tunisian alleles have a histidine and a proline. Slow forms of SOD have specific activity 2-3 times that of fast forms and they are more thermolabile (Lee, Misra, and Ayala, 1981). A low activity allele, *Sod*^{CAI} isolated from a natural population in California exhibits 3.5% of normal CRM level; low level maps to *Sod* (Graf and Ayala). In addition to these naturally occurring alleles there is a single ethyl methanesulfonate-induced null allele, *Sod*^{nl} (isolated as *l-108*; Campbell, Hilliker, and Phillips, 1986, Genetics 112: 205-15).

cytology: Placed in 68A8-9 by *in situ* hybridization (Kirkland and Phillips, 1987, Gene 61: 415-19).

molecular biology: Gene cloned and sequenced; cDNA clones have 458 base pairs of open reading frame, encoding 153 amino acids; N-terminal methionine and C-terminal valine do not appear in the mature polypeptide (Seto, Hayashi, and Tener, 1987, Nucleic Acids Res. 15: 5483). Genomic sequence contains a 725 base-pair intron between codons 21 and 22, which corresponds to the position of the first intron in the human SOD gene (Seto, Hayashi, and Tener, 1987, Nucleic Acids Res. 15: 10601).

Sod^{nl}

origin: Ethyl methanesulfonate-induced derivative of *Sod*^F.

references: Phillips, Campbell, Michaud, Charbonneau, and Hilliker, 1989, Proc. Nat. Acad. Sci. USA 86: 2761-65.

phenotype: Originally recovered as a lethal mutation; homozygotes die in the process of eclosion; rare eclosing adults are completely sterile, are devoid of SOD activity and die within 2-3 days; however, some derived sublines show higher adult survival. Surviving homozygous males are sterile and homozygous females produce few if any offspring. Reduced life span and fertility attributed to reduced capacity of embryos, larvae, and pupae to protect developing preimaginal cells from O₂⁻-initiated cytotoxic damage. Homozygotes hypersensitive to the O₂⁻-radical-generating compound, paraquat (1,1'-dimethyl-4,4'-bipyridinium dichloride) and copper ions.

soft brown: see *sb*

sog: short gastrulation

location: 1-53.

references: Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307. Zusman, Sweeton, and Wieschaus, 1988, Dev. Biol. 129: 417-27 (fig.).

phenotype: Embryonic lethal with weak ventralized phenotype. Invagination and subsequent closing of the posterior midgut and anterior midgut delayed; germ-band

extension incomplete; dorsal-most cells fail to assume normal amnioserosal fate; they are abnormally thick and fall into deep dorsal folds at the time of germ-band extension. Mosaic studies indicate that *sog* expression required only ventrally for normal development. No discernable maternal effect.

alleles: Six ethyl methanesulfonate-induced alleles, one of which is temperature sensitive, recorded by Wieschaus *et al.*; one isolated as *sog-92.31* designated *sog*¹ by Zisman *et al.* Allele *sog*^{M4} here designated *sog*²; *XM* designated *sog*³. The remaining alleles designated *sog*⁴ through *sog*⁶; identity of t.s. allele unknown. *sog*⁷ ethyl methanesulfonate induced by Eberl and Hilliker (1988, Genetics 118: 109-20) as *EH628*.

cytology: Placed in 13D1-E7 based on its location between the breakpoints of *T(1;Y)B28 = T(1;Y)13D;YS* and *T(1;Y)W23 = T(1;Y)13E1-7;YL*.

***sol*: small optic lobes (J.C. Hall)**

location: 1- {65} (Fischbach and Heisenberg, 1981, and Helfrich, 1986, quote more distal and more proximal meiotic map positions, respectively); Fischbach and Lyly-Hünerberg mapped *sol* proximal to *car*.

origin: Induced by ethyl methanesulfonate.

synonym: *PC79*; *w5000*.

references: Markow and Merriam, 1977, Behav. Genet. 7: 447-55.

Fischbach and Heisenberg, 1981, PNAS 78: 1105-09.

Bülthoff, 1982a, DIS 57: 31.

Bülthoff, 1982b, Biol. Cybernet. 45: 63-70.

Fischbach and Lyly-Hünerberg, 1983, Cell Tissue Res. 231: 551-63.

Helfrich and Engelmann, 1983, Physiol. Entomol. 8: 257-72.

Fischbach and Technau, 1984, Dev. Biol. 104: 219-39.

Helfrich, 1986, J. Neurogenet. 3: 321-43.

Coombe, 1986, J. Comp. Physiol. 159: 655-65.

Miklos, Kelly, Coombe, Leeds, and Lefevre, 1987, J. Neurogenet. 4: 1-19.

phenotype: Medulla, lobula, and lobula-plate optic ganglia reduced in volume and cell number (anatomical criteria on which several of the mutations, including the most studied allele *sol*¹, were isolated by Heisenberg and Böhl, 1979, Z. Naturforsch. 34: 143-147); lamina seems unaffected; degree of reduction in the three more proximal visual-system ganglia is allele dependent; after isogenization the severity ranking of nine alleles was as follows: *sol*² = *sol*³ (ca. 50% normal volume) > *sol*⁶ = *sol*⁹ = *sol*¹⁶ > *sol*¹ = *sol*⁴ = *sol*⁵ = *sol*⁸ (ca. 30% normal volume). Three *sol* mutants isolated on basis of fast phototaxis; Markow and Merriam (1977) showed that one such allele, *sol*⁴, causes flies to be anomalously photo positive and highly geonegative in maze tests. Anatomically, *sol* mutations cause specific cell types in medulla to be missing (Fischbach and Heisenberg, 1981); stratifications in outer medulla are missing; in general, however, mutant optic lobes are grossly well structured, and there are no disorders in the optic chiasma; special classes of transmedullary columnar neurons as well as intramedullary cells are absent; numbers of columns in the visual ganglia are normal, but numbers of neurons per column are reduced; certain neurons called T1 cells are present in each column in *sol*¹, as usual; these reductions in cell numbers are caused by cell-type-specific degen-

eration of presumptive optic lobe neurons during pupation, with no degeneration apparent in neuropiles of these ganglia (Fischbach and Technau, 1984); the number of axons severely reduced in anterior optic track, and the combining of *so* with *sol*¹ showed that these two mutations act independently on nearly exclusive subsets of these axons (Fischbach and Lyly-Hünerberg, 1983). Mosaic study showed that aberrant morphology of visual ganglia is autonomous in these optic lobes (Fischbach and Technau, 1984); adult eye and lamina optic lobe appear normal. In combination with *rol* and *mnb* mutations, *sol* causes diminished amplitudes of light-on and light-off transient spikes in electroretinogram (Coombe, 1986); visual fixation behavior notably defective (Fischbach and Heisenberg, 1981) [e.g., in the walking mode, fixation behavior is actually reversed in all *sol* alleles (Fischbach)] as, to a lesser degree, are landing responses and "figure/ground" discrimination; on the other hand, optomotor *yaw* response is nearly normal (Fischbach and Heisenberg, 1981); orientation to spots in multiple Y-maze quite subnormal (Bülthoff, 1982a,b). Shock-avoidance learning of *sol*¹ (Heisenberg, Borst, Wagner, and Byers, 1985, J. Neurogenet. 2: 1-30) and color discrimination in *sol*¹, *sol*², and *sol*³ (Fischbach) are normal; there are, however, deficits in visual plasticity (Götz, 1983, Dtsch. Zool. Ges. Gustav Fischer Verlag, Stuttgart, pp. 83-99) and in the flexibility that wild types can exhibit in optomotor flight control tests (Götz, 1985, Biol. Chem. Hoppe Seyler 366: 116-17). Circadian rhythms of adult locomotor activity basically normal (Helfrich and Engelmann, 1983; Helfrich, 1986), but when *sol*¹ combined with *so*, all flies tested showed complex periodicities, with a given behavioral record having one component at approximately 21 and another at approximately 26 h (Helfrich, 1986).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>sol</i> ¹	EMS		<i>KS58</i>	4	histology
<i>sol</i> ²	EMS		<i>KS84</i>	4	histology
<i>sol</i> ³	EMS		<i>KS160</i>	4	histology
<i>sol</i> ⁴	EMS		<i>EE111</i>	1	phototaxis
<i>sol</i> ⁵	EMS		<i>KS91</i>	1	phototaxis
<i>sol</i> ⁶	EMS		<i>PC79</i>	1	phototaxis
<i>sol</i> ⁷	EMS		<i>542</i>	3	histology
<i>sol</i> ⁸	EMS		<i>648</i>	3	histology
<i>sol</i> ⁹	EMS		<i>2303</i>	3	histology
<i>sol</i> ¹⁰	EMS		<i>3056</i>	3	histology
<i>sol</i> ¹¹	EMS		<i>3447</i>	3	histology
<i>sol</i> ¹²	EMS		<i>3575</i>	3	histology
<i>sol</i> ¹³	EMS		<i>3596</i>	3	histology
<i>sol</i> ¹⁴	EMS		<i>3819</i>	3	histology
<i>sol</i> ¹⁵	EMS		<i>w5000</i>	3	histology
<i>sol</i> ¹⁶	EMS	Bülthoff	<i>nofE</i> ^{B12}	2	behavior
<i>sol</i> ¹⁷		Heisenberg	<i>1048</i>		

^α 1 = Benzer and Merriam; 2 = Bülthoff, 1982, DIS 58: 31; 3 = Fischbach and Heisenberg, 1981, Proc. Nat. Acad. Sci. USA 78: 1105-09; 4 = Heisenberg and Böhl, 1979, Z. Naturforsch. 34: 143-47.

^β Criterion used in selection of mutant.

cytology: Tentatively placed in 19F4, along with *sgA*, these two genetically separable mutations being flanked by *l(1)19Fe* and *l(1)19Ff*; this determination (Miklos *et al.*, 1987) was based the behavioral defects and anatomical abnormalities of *sol* in heterozygous combination with *Df(1)GA104*, but not by *Df(1)16-129*, located distally or *Df(1)JA117*, located proximally; none of the three

has cytologically determined breakpoints.

molecular biology: Maps within the distal 15 kb of a 40 kb interval defined by the proximal breakpoint of *Df(1)16-129* and the distal breakpoint of *Df(1)JA117*. This region is further subdivided by a breakpoint of a deficiency, *Df(1)2/19B*, which separates *sol* and *slgA*. Only one transcription unit has been detected within this 15 kb segment by Northern blotting (should contain both *l(1)19Fe* and *sol*); a 5.3 kb cDNA complementary to the one transcript has been isolated and sequenced (Delaney, Hayward, Fischbach, and Miklos, unpublished). The conceptual translation product is *ca.* 35% identical to human or chicken calcium-activated neutral protease (calpain); sequence data also reveal an opa repeat and cysteine repeats of the zinc-finger type. Spatial expression studies so far reveal *sol* transcript to be expressed in a few cells (probably glia) within the embryonic CNS (F. Barleben and K. F. Fischbach, unpublished), although there are no apparent anatomical defects in mutant embryos or larvae.

other information: In behavioral tests, *sol*¹ was complemented by alleles of *l(1)19Ff* and also by mutations at the nearby *slgA*, *uncl*, and *stm* loci (Miklos *et al.*, 1987).

***som: sombre**

location: 1-40.8.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 75.

phenotype: Pigmentation of body and eyes dark and dull. Wings occasionally divergent or blistered. Good viability and fertility. RK2.

other information: One allele induced by CB. 1414.

sonless: see *snl*

sp: speck

location: 2-107.0.

references: Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 128 (fig.).

Morgan, Bridges, and Sturtevant, 1925, Bibliogr. Genet. 2: 211 (fig.), 236.

phenotype: Axils of wings have black specks. Body color dark. In pupa, region of anal papilla is dark (Waddington). Displays a marked decrease in the amount of the A2 component of phenoloxidase; levels restored to normal in presence of *su(s)*² (Warner, Grell, and Jacobsen, 1975, Biochem. Genet. 13: 353-56). RK1.

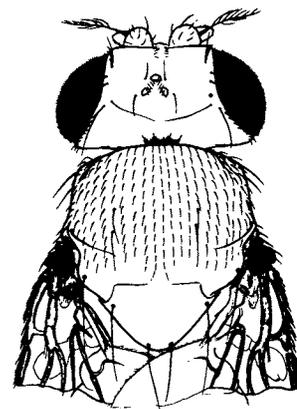
alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>sp</i> ¹	spont	Morgan, 10c	<i>ol-2: olive-2</i>	1, 2, 5	412 and <i>roo</i> inserted in region
<i>sp</i> ²		Bridges, 25f			stronger than <i>sp</i> ¹
* <i>sp</i> ^{S61}	spont	Shuman, 61c		4	like <i>sp</i> ¹
* <i>sp</i> ^u	UV	Meyer, 52d		3	weaker than <i>sp</i> ¹

^α 1 = Morgan and Bridges, 1919, Carnegie Inst. Wash. Publ. No. 278: 128 (fig.); 2 = Morgan, Bridges, and Sturtevant, 1925, Bibliogr. Genet. 2: 211 (fig.), 236; 3 = Meyer, 1955, DIS 29: 74; 4 = Meyer, 1963, DIS 37: 51; 5 = Searles and Voelker, 1985, Proc. Nat. Acad. Sci. USA 83: 404-08.

cytology: Placed in 60B13-60C5 on the basis of its inclusion in the 2R^{PX}^D element of *T(1;2)Bld = T(1;2)1C3-4;60B12-13* and *Df(2R)Px = Df(2R)60B8-10;69D1-2* but

not in *Df(2R)Px*² = *Df(2R)60C5-6;60D9-10* [Bridges, 1937, Cytologia (Tokyo), Fujii Jub., Vol. 2: 745-55].



sp: speck

From Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 129.

sp: see *sd*^{sp}

Sp: Sternopleural

location: 2-22.0.

origin: Spontaneous.

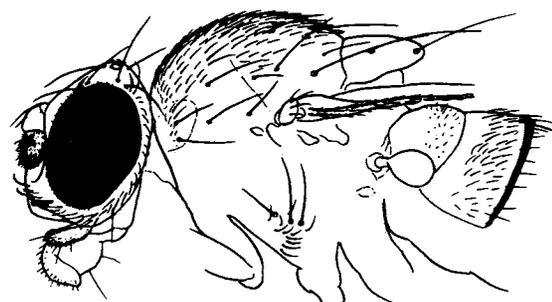
discoverer: M. (Mann) Lesley.

synonym: *Br: Bristled.*

references: 1923, Genetics 8: 27-36.

phenotype: Sternopleural bristles increased in number. At 19°, wild type; at 25°, overlaps wild type; at 28°-30°, no overlap. Apparently does not affect sternopleural bristles on metathoracic segment converted by *bx* to a mesothoracic segment (Waddington, 1939, Growth Suppl. 1, pp. 37-44). Homozygous lethal. RK2.

cytology: Placed in salivary chromosome region 27C1 to 28C1 (E. H. Grell). Salivary chromosomes apparently normal (Morgan, Bridges, and Schultz, 1937, Year Book - Carnegie Inst. Washington 36: 301).



Sp: Sternopleural

Edith M. Wallace, unpublished.

sp-w: see *w*^{sp}

spa: sparkling

location: 4- [probably most distal visible locus on chromosome 4 (Abrahamson, Herskowitz, and Muller, 1956, Genetics 41: 410-19)].

origin: Spontaneous.

discoverer: L. V. Morgan, 34k6.

references: 1941, DIS 14: 52.

1947, Genetics 32: 200-19.

Hochman, 1971, Genetics 67: 235-52.

Hochman, 1974, Cold Spring Harbor Symp. Quant. Biol. 38: 581-89.

phenotype: Eyes rough in varying degrees and somewhat bulging. Affected by genetic modifiers. More extreme at 17°-19° than at 22°-25°. Heterochromatin and sex affect expression so that $X/0 > X/X > X/Y > X/X/Y$; also enhanced by *Df(2R)M41A10*; *spa* haplo-4s have an exaggerated phenotype. RK2.

cytology: Placed in 102D-F on the basis of the absence of *spa*⁺ from the 4^P2L^D element of *T(2;4)b = T(2;4)25E;102C15-D1* (E. B. Lewis). Observations on its further location conflict. Fahmy restricts its location to 102D on the basis of its inclusion in *Df(4)M^{62e} = Df(4)101E;102D13-E1*, whereas Hochman places it between 102E2 and 102F10 on the basis of its inclusion in *Df(4)11 = Df(4)102E2-10;102F2-10*.

spa^A

references: Craymer, 1980, DIS 55: 197-200.

phenotype: Dominant rough-eye allele; more extreme than *spa^{Cat}*. *spa^A/spa^{pol}* shows extreme poliart phenotype; *spa^A/spa^{Cat}* lethal. RK1A.

cytology: Associated with *T(3;4)Ubx^A*, an extremely complex rearrangement with one breakpoint in 102E-F.

spa^{Cat}: sparkling-Cataract

origin: X ray induced.

discoverer: Belgovskiy, 1936.

synonym: *Cat*.

references: 1937, DIS 8: 7.

Morgan, 1941, DIS 14: 52.

phenotype: Posterior third or half of eye of heterozygote rough; facets irregular and fused. Homozygous lethal. Stocks vary in expression, presumably because of genetic modifiers. X/X and $X/0$ flies that are *spa^{Cat}/spa* show the bulging eyes and roughening of *spa* and the posterior fused facets of *spa^{Cat}*; $X/X/Y$ and X/Y flies have only the *spa^{Cat}* phenotype. *spa^{Cat}/spa^{pol}* has fusion of facets over entire surface of eye and roughness in posterior region of eye. *spa^{Cat}/4-sim* is wild type. *spa^{Cat}/+* more extreme than *spa^{Cat}/+/+* (Davis, 1969, Genetics 61: 577-94). RK2.

other information: *spa^{Cat}* behaves as a deficiency in that it fails to complement *l(4)102EFa*, *l(4)102EFb*, *l(4)102EFc*, and *l(4)102EFd*; complements *sv* and *l(4)102EFe*.

spa^{e(lz)}: sparkling-enhancer of lozenge

origin: Spontaneous.

discoverer: H. A. Bender, 65b23.

phenotype: Homozygote wild type in absence of *lz*; eyes strongly roughened in presence of heterozygous *lz³*, *lz³⁴*, *lz³⁶*, or *lz^D*. Slight eye roughening when both *spa^{e(lz)}* and a *lz* allele are heterozygous. *spa^{e(lz)}/spa^{pol}* and *spa^{e(lz)}/spa^{p65}* have very rough eyes but normal tarsal claws and spermathecae. RK3.

spa^{p61}: sparkling-poliart type

origin: Spontaneous.

discoverer: Sturtevant, 1961.

phenotype: Eyes small, rough, and glazed. More extreme than *spa^{pol}* or *spa^{p65}*. Nonpigmented tarsal claws. RK1.

spa^{p65}

origin: Spontaneous.

discoverer: H. A. Bender, 65j11.

phenotype: Eyes somewhat reduced in size, rough and partially glazed. More extreme than *spa^{pol}* but less so than *spa^{p61}*. Tarsal claws unpigmented and possibly reduced; reminiscent of certain lozenge mutants. Pulvilli and accessory female reproductive structures appear normal. Heterozygote with *spa^{pol}* and *spa^{p61}* has affected tarsal claws as well as rough eyes. Heterozygote with *spa* has slightly roughened eyes at 25° but markedly roughened eyes at 18°; female somewhat more extreme than male. Viability and fertility good. RK1.

spa^{pol}: sparkling-poliart

origin: Spontaneous.

discoverer: Hadorn, 51a.

synonym: *pol*.

references: Rickenbacher, 1953, DIS 27: 59.

1954, Z. Indukt. Abstamm. Vererbungslehre 86: 62-68 (fig.).

phenotype: Eyes rather small; surface smooth and glassy.

During second day of pupal life, retinula cells withdraw from other cells of eye disk. SEM studies show irregular disposition and morphology of ommatidial hairs as well as numerous necrotic pits over surface of eye [Oster and Crang, 1972, Trans. Am. Microsc. Soc. 91: 600-02 (fig.); Strum-Tegethoff and Dicke, 1974, Theor. Appl. Genet. 44: 762-65]. ERG absent [Grossfield, Handbook of Genetics (King, ed.). Plenum, New York, pp. 679-702]. *spa^{pol}/spa^{Cat}* has extreme phenotype; *spa^{pol}/spa* slightly more extreme than *spa* (Sturtevant, 1961, DIS 35: 47). Homozygote has excellent viability and fertility. RK1.

spade: see *spd*

spaghetti squash: see *sqh*

spalt: see *sal*

sparkling: see *spa*

sparse arista: see *crm^{sa}*

sparge hairs: see *sph*

spastic: see *sps*

spätzle: see *spz*

spc: see *gra*

spd: spade

location: 2-21.9 [to the left of *Sp* (E. H. Grell)].

origin: Spontaneous.

discoverer: Bridges, 30d15.

phenotype: Wings short and broad, pointed at tip, and warped at base. Effect on wing shape arises from excessive contraction of epithelium from inflated stage onward (Waddington, 1940, J. Genet. 41: 75-139). Overlaps wild type in existing stock. RK3.

cytology: Placed in 27D based on its inclusion in

Df(2L)spd = Df(2L)27D-E;28C (E. H. Grell) and location distal to *T(Y;2)A171 = T(Y;2)h3;27D* breakpoint (Kotar-ski, Pickert, and MacIntyre, 1983, Genetics 105: 371-86).

spd^{1g}: spade-flag

origin: Spontaneous.

discoverer: Doane, 60f14.

synonym: *fg*.

references: 1960, DIS 34: 49.
1961, DIS 35: 45-46.

phenotype: Wings about two-thirds the length and three-fourths the width of wild type, held tentlike over abdomen. Alulae absent or vestigial; proximal posterior wing margins often irregular with tendency to fold under about vein L4. Venation usually normal with occasional blistering. *spd^{1g}/spd* has phenotype varying from slight shortening of wings to a shape midway between the two homozygotes. Excellent viability and fertility. RK1.

spe: see *lz^s*

αSpec: alpha Spectrin

location: 3- {1.5}.

references: Dubreuil, Byers, Branton, Goldstein, and Kiehart, 1987, J. Cell Biol. 105: 2095-2102.

Byers, Dubreuil, Branton, Kiehart, and Goldstein, 1987, J. Cell Biol. 105: 2103-10.

phenotype: Structural gene for alpha spectrin, a 234 kd polypeptide that forms an elongated heterodimer with beta spectrin to form an important cytoskeletal component. Product crossreacts with antibodies against mammalian spectrins; can be shown to bind to beta spectrin subunits.

cytology: Placed in 62B1-7 by *in situ* hybridization to a single polytene site.

molecular biology: cDNA sequences isolated by antibody screening of an expression library prepared from Drosophila adult head polyadenylated RNA. Northern blots reveal a single homologous 8 kb messenger RNA. Derived probes bind to a single polytene region, suggesting the presence of a single Drosophila alpha spectrin gene. cDNA clones indicate *αSpec* contains a single open reading frame that encodes a polypeptide 2,415 residues long of molecular weight 278,364 daltons. Conceptual sequence indicates two stretches of nine 106-residue repeats separated by a short non-repetitive segment; the repeats are defined by a consensus sequence of 54 residues, 50 of which are shared by chicken *α* spectrin. Drosophila *α* spectrin is 63% identical with chicken brain *α* spectrin (Dubreuil, Byers, Sillman, Bar-Zvi, Goldstein, and Branton, 1989, J. Cell Biol. 109: 2197-2205).

βSpec: beta Spectrin

location: 1-{57}.

references: Byers, Hussain-Chishti, Dubreuil, Branton, and Goldstein, 1989, J. Cell Biol. 109: 1633-41.

phenotype: Structural gene for beta spectrin; The subunit of beta spectrin associates with the subunit of alpha spectrin, forming heterodimers which associate to form tetramers, presumably the most important functional state for spectrin.

cytology: Located in 16C1-4 by *in situ* hybridization to the salivaries (Byers *et al.*, 1989). A single polytene site was detected.

molecular biology: Drosophila *βSpec* cDNA clones were isolated from cDNA expression library using chicken alpha spectrin as a probe. These cDNAs express polypeptides that bind to alpha spectrin. Antibodies from fusion proteins expressed by these sequences react with Drosophila spectrin. Nucleotide and predicted amino acid sequences for the 5' end of spectrin determined; the amino acid sequence predicted for this region shows 62% identity to the deduced sequence for the erythrocyte beta spectrin in man. A single mRNA of about 8.0 kb was detected on Northern blots of poly A+ RNA from Drosophila heads (Byers *et al.*, 1989).

***speck*:** see *sp*

***spectacled*:** see *lz^s*

***Spectrin*:** see *αSpec* and *βSpec*

***sperm amotile*:** see *sam*

***spermatheca*:** see *spt*

spg: sponge

location: 3-95.

discoverer: Rice.

synonym: *early-D*; *mat3(6)*.

references: Rice and Garen, 1975, Dev. Biol. 43: 277-86 (fig.).

Rickoll and Counce, 1981, Wilhelm Roux's Arch. Dev. Biol. 190: 245-51 (fig.).

phenotype: Maternal-effect lethal; embryos produced by homozygous females produce pole cells, but syncytial blastoderm fails to cellularize completely; cells form at the anterior and posterior ends of the embryo but are separated by a noncellular region comprising 70% of the embryo surface. Abortive gastrulation signified by formation of posterior midgut rudiment.

alleles: Five ethyl methanesulfonate-induced alleles; *spg¹* through *spg⁵*, isolated as *145*, *242*, *805*, *842*, and *rm6*, respectively.

sph: sparse hairs

location: 1- {65}.

references: Schalet and Lefevre, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 16, pp. 847-902.

Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31.

phenotype: Thoracic hairs sparse, eye roughening variable, wings extended and margins incised. This phenotype observed in flies heterozygous for *Df(1)17-87*, *Df(1)22*, and *sph¹*, *sph³*, or *sph⁴* (Schalet and Lefevre). No effect on central or peripheral nervous system in *sph⁵* or *sph⁶*; both alleles cell lethal in female germline clones (Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-52).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>sph¹</i>	X ray		<i>l(1)4P1</i>	5, 7, 8, 9	
<i>sph²</i>	EMS	Lifschytz	<i>l(1)M147</i>	4	on <i>y[±] Ymal[±]</i>
<i>sph³</i>	EMS	Lifschytz	<i>l(1)R-9-5</i>	3, 7, 8	
<i>sph⁴</i>	X ray	Lifschytz	<i>l(1)X4</i>	2, 3, 7, 8	
<i>sph⁵</i>	EMS	Lefevre	<i>l(1)VE829</i>	1, 5	L/P
<i>sph⁶</i>	spont	Schalet	<i>sph^{SI}</i> <i>l(1)7-96</i>	5, 6	L

^α 1 = Lefevre and Watkins, 1986, *Genetics* 113: 869-95; 2 = Lifschytz and Falk, 1968, *Mut. Res.* 6: 235-44; 3 = Lifschytz and Falk, 1969, *Mut. Res.* 8: 147-55; 4 = Lifschytz and Jakobovitz, 1978, *Mol. Gen. Genet.* 161: 275-84; 5 = Perrimon, Smouse, and Miklos, 1989, *Genetics* 121: 313-31; 6 = Schalet, 1986, *Mutat. Res.* 163: 115-44; 7 = Schalet and Lefevre, 1973, *Chromosoma* 44: 183-202; 8 = Schalet and Lefevre, 1976, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.), Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902; 9 = Schalet and Singer, 1971, *DIS* 46: 131-32.

cytology: Placed in 20E by Lefevre (1981, *Genetics* 99: 461-80).

spi: spitz

location: 2-54.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 267-82 (fig.).

Tearle and Nüsslein-Volhard, 1987, *DIS* 66: 209-26.

Mayer and Nüsslein-Volhard, 1988, *Genes Dev.* 2: 1496-1511 (fig.).

phenotype: Embryonic lethal. Normal allele required for normal development of blastoderm cells just lateral to the ventral mesodermal precursors. Denticle bands narrower than normal; first row often missing; reversal of polarity between anterior and posterior rows not seen; irregular fusion in midline of adjacent denticle bands. Keilin's organs missing or strongly reduced. Labrum, antennal-maxillary complex and labial sense organ reduced in size; right and left halves of head skeleton fused to produce pointed appearance. Tail region normal. Right and left halves of ventral ganglia of the central nervous system closer together than normal resulting in shorter commissures. The cells in the CNS that stain with anti-*eve*⁺ antibodies are closer together in *spi* than in wild type. *spi*⁺ required in the female germ line; *spi* pole cells transplanted into normal embryos produce no progeny.

alleles: Three ethyl methanesulfonate-induced alleles, *spi*¹, *spi*², and *spi*³, isolated as *IIA*, *IIT*, and **IIIA*.

cytology: Placed in 37E2-38A1; in the region common to *Df(2L)E55 = Df(2L)37D2-E1;37F5-38A1* and *Df(2L)TW9 = Df(2L)37E2-F4;38A6-C1*.

molecular biology: spitz region cloned from 37F [Rutledge, Zhang, Perrimon, Bier, and Y. Jan, 1990 (Asi-lomar)].

spindle: see *spn*

spineless: see *ss*

spiny legs: see *sple*

spir: spire (T. Schüpbach)

location: 2- {54.5}.

origin: Induced by ethyl methanesulfonate.

references: Manseau and Schüpbach, 1989, *Genes Dev.* 3: 1437-52.

phenotype: Maternal-effect lethal; homozygous females lay eggs which sometimes (5-10%) have a "peak" (spire) of dorsal appendage material sitting over the anterior end of the egg, instead of two distinct dorsal appendages. Such eggs are similar to eggs formed by the female-sterile mutation *fs(1)K10*, but the extent of dorsal appendage material on *spir* eggs is much more variable than that of *fs(1)K10* eggs. Mutant females produce embryos lacking polar granules, pole cells, and normal abdominal segmentation. In combination with *Bic-D*,

however, abdominal segmentation does develop in the anterior half of the embryo; improper localization of abdominal determinants also indicated by the lack of posterior localization of *vasa* protein. Cellularization of the blastoderm irregularly defective with nuclei of different sizes and densities. Resemble embryos formed by other grandchildless-knirps-like mutations, such as *vasa* or *tudor*, but in addition, some of the embryos from spire females appear also to be dorsalized.

alleles: Twelve alleles, presumably induced by ethyl methanesulfonate; *spir*¹ to *spir*⁶ isolated as *RP*, *HP*, *HJ*, *QF*, *O3*, and *41*, respectively.

cytology: Placed in 38A6-C10, based on its inclusion in *Df(2L)pr21 = Df(2L)37E3-F1;38C6-10* but not *Df(2L)TW50 = Df(2L)36E4-F1;38A6-7*.

spiracles: see *gra*

spire: see *spi*

spi: see under *N*

Spl: Splayed

location: 3-51.6.

references: Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, *Genetics* 71: 157-84.

Tasaka and Suzuki, 1973, *Genetics* 74: 509-20.

Suzuki, Kaufman, and Falk, 1976, *Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.), Academic Press, London, New York, San Francisco, Vol. 1a, pp. 208-51.

phenotype: Legs extended; movements awkward; some melanization of leg joints. Originally described as the phenotype of a heterozygous deficiency for region 81F-82A by Lindsley *et al.*; subsequently a temperature-sensitive lethal isolated by Tasaka and Suzuki that mapped to the region and which produced the same phenotype, presumably in homozygotes reared at permissive temperatures.

alleles: Only one mutant allele, *Spl*^{ts}.

cytology: Placed in 81F-82A based on the phenotype of heterozygotes for the segmental deficiency produced from *T(Y;3)J139 = T(Y;3)80-81* and *T(Y;3)J17 = T(Y;3)82A*.

splay wing: see *splw*

splc: see *tor*^{11D}

sple: spiny legs

location: 2-55.3 [between *pk* and *pwn* (Grau and Simpson, 1987, *Dev. Biol.* 122: 186-200)].

origin: Spontaneous.

references: Goldschmidt, 1945, *Univ. Calif. (Berkeley) Publ. Zool.* 49: 503-4, 521.

Gubb and Garcia-Bellido, 1982, *J. Embryol. Exp. Morph.* 68: 37-57.

Held, Duarte, and Devakhshanian, 1986, *Roux's Arch. Dev. Biol.* 195: 145-57 (fig.).

phenotype: Polarity of chaetae and trichomes on legs irregular; relations between bracts and bristles disrupted. High incidence of ectopic tarsal joints with inverted polarity, especially in tarsae 3 and 4; incomplete intersegmental membranes between tarsal segments, especially between segments 3 and 4; no extra sensilla companion-

mia despite extra joints. Chaetae on abdominal tergites turned toward midline instead of pointing posteriorly as in wild type; polarity of bristles on sternites disrupted as well.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>sple</i>¹	spont	Goldschmidt		1, 2	
<i>sple</i>²	X ray	García-Bellido	<i>sple</i> ^{78a}	1	also mutant for <i>pk</i>
<i>sple</i>³	EMS		<i>sple</i> ^{bor}	3	described below

^α 1 = Gubb and García-Bellido, 1982, J. Embryol. Exp. Morph. 68: 37-57; 2 = Held, Duarte, and Derakhshanian, 1986, Roux's Arch. Dev. Biol. 195: 145-57; 3 = Sharma, Chitnis, and Shyngle, 1985, DIS 61: 216 (fig.).

cytology: Placed in 42C1-43C7 based on its inclusion in the region common to *Df(2R)pk78r = Df(2R)43A1;43C7* and *Df(2R)pk78s = Df(2R)42C1-7;43F5-8* (Ashburner, Angel, Detweiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91).

***sple*³**

synonym: Bristle orientation reversed.

phenotype: A low-temperature-sensitive allele. Legs of flies raised at 19° highly condensed with incomplete joints, haphazard bristle pattern, occasional increase in number of bristle rows, and absence of some prominent markers. Flies unable to walk and soon get stuck in food. In *sple*³ flies raised at 28° 53% of legs, though apparently normal, display abnormalities such as swollen second, third, and fourth tarsal segments, abnormal tarsal joints and reversals in bristle orientation in the middle of every segment; such flies able to walk and breed. About 30% of legs show phenotype intermediate between the two phenotypes described above. Mutant phenotype expressed in the homeotic legs of *Antp* and *ss*^a; also expressed in mitotic clones.

spliced: see *tor*^{IID}

split: see *spl* under **N**

split thorax: see **sp_x**

Plotched: see **Sl**

***splw*: splay wing**

location: 1-58.6.

origin: Induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 75.

phenotype: Wings shortened and usually slightly divergent. Eyes small and occasionally rough and deformed. Body size reduced slightly. Emergence delayed. Male sterile; viability about 10% wild type. RK3.

other information: One allele induced by CB. 1246.

***spn-A*: spindle A**

location: 3-96.

references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Maternal-effect lethal. Egg shape affected; in extreme cases dorsal appendages are lacking and eggs have little or no dorsal-ventral polarity; some eggs have one fused dorsal appendage. Low fecundity; eggs often slightly collapsed.

alleles: Four alleles designated as *spn-A*¹ through *spn-A*⁴ isolated as 003, 050, 057, and 215, respectively.

***spn-B*: spindle B**

location: 3-52.

references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Maternal-effect lethal. Similar to *spn-A* but normal eggs also recovered; eggs always unfertilized and abnormal eggs often long.

alleles: Three alleles designated as *spn-B*¹ through *spn-B*³ isolated as 056, 153, and 225, respectively.

cytology: Placed in 88A10-C3 based on its inclusion in *Df(3R)red-P93 = Df(3R)88A10-B1;88C2-3*. However, not in 88B1-4 based on its not being uncovered by *Df(3R)red-52P = Df(3R)88A12-B1;88B4-5*.

***spn-C*: spindle C**

location: 3-17.

references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Maternal-effect lethal. Similar to *spn-A*.

alleles: Three alleles designated as *spn-C*¹ through *spn-C*³ isolated as 094, 422, and 660, respectively.

***spn-D*: spindle D**

location: 3-91.

references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Maternal-effect lethal. Similar to *spn-A*; embryos sometimes hatch.

alleles: Two alleles, *spn-D*¹, a weak allele isolated as number 150, and a strong allele with isolation number 349.

***spn-E*: spindle E**

location: 3-62.

references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Maternal-effect lethal. Similar to *spn-A*.

alleles: Two alleles, *spn-E*¹ and *spn-E*², isolated as 616 and 653.

***spn-F*: spindle F**

location: 3-100.

references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Maternal-effect lethal. Similar to *spn-A*.

alleles: *spn-F*¹, the only allele, isolated as 234.

***spo*: spook**

location: 3-19.

references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984,

Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

phenotype: Embryonic lethal. No differentiation of cuticle and mouthparts.

alleles: *spo*¹ and *spo*² isolated as 4G9 and 7J.

****spot*: spot**

location: 3- (not located).

discoverer: Hersh, 34h15.

references: 1935, DIS 4: 14.

phenotype: Dark spot appears below eye on posterior margin of head. Expression variable. RK3.

spotted white: see *w^{sp}*

spotty: see *stt*

spotty-tergum: see *stt²*

***spr*: spread wings**

location: 3- [right arm associated with *In(3R)P*].

origin: Spontaneous.

discoverer: Bridges, 36c16.

phenotype: Wings held out at wide angle. Both sexes sterile. RK3A.

****Spr*: Spread**

location: 3- (rearrangement).

origin: X ray induced.

discoverer: Oliver, 32k21.

references: 1935, DIS 4: 15.

phenotype: Wings held outstretched perpendicular to body axis, droop in older fly. Homozygous lethal. Heterozygote viability somewhat low. Female fertile; male quite infertile. RK2A.

cytology: Associated with *In(3L)Spr*; breakpoints unknown.

****sprd*: spread**

location: 3-65.

origin: Spontaneous in *In(3R)C*.

discoverer: Dexter, 13k.

synonym: *sd* (preoccupied).

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 105.

phenotype: Wings spread at right angles to body. RK2A.

other information: Probably separable from *In(3R)C* = *In(3R)92D1-E1;100F2-3*.

Spread: see *Spr*

spread wings: see *spr*

spreadex: see *sdx*

spready: see *eg^{spy}*

***sps*: spastic**

location: 2-63.6.

origin: Ultraviolet induced.

discoverer: Edmondson and Meyer, 49d.

references: 1951, DIS 25: 73.

phenotype: Pupal and postpupal lethal. Fly that emerges from pupal case unable to walk or fly. Spastic contraction and jerking of leg and wing muscles. Fly becomes overturned and stuck; survives less than 24 hr; sterile. Muscles so relaxed in etherized fly that mutant indistinguishable from normal fly. RK3.

***spt*: spermatheca**

location: 2-63.3.

origin: Spontaneous.

discoverer: Hadorn, 43e.

references: Hadorn and Graber, 1944, Rev. Suisse Zool. 51: 418-23.

Graber, 1949, Z. Indukt. Abstamm. Vererbungsl. 83: 106-35 (fig.).

phenotype: At 28°, female has two spermathecae but ducts partly fused; at 25°, only one enlarged spermatheca on one duct; at 18°, a duct with three branches, each bearing

a spermatheca. Temperature-sensitive period in third larval instar. Female fertility not greatly affected. RK3.

spt: see *stt²*

****spw*: spur wing**

location: 3- (right arm).

origin: Spontaneous.

references: Wallbrunn, 1942, DIS 16: 54.

phenotype: Wings vary from normal to large fan-shaped structures with extra veins; often a spur-shaped lobe from costal margin. Penetrance better in female and in old cultures. RK3.

***spx*: split thorax**

location: 1-22.6.

origin: X ray induced.

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 91-92.

phenotype: In extreme manifestation, thorax split into two segments by longitudinal furrow; abdominal tergites also split along mid-dorsal line. Eyes deformed. In least abnormal fly, always a hairless stripe along the dorsal midline of thorax. Wings often slightly divergent. Occasionally, one or both palpi abnormal in position or structure. Viability and fertility rather low in male, very low in female. RK3.

other information: One allele each induced by CB. 2511 and CB. 3007. Two alleles induced by CB. 1528.

spx: see *sdx*

spy: see *eg^{spy}*

***spz*: spätzle**

location: 3-92.

references: Anderson and Nüsslein-Volhard, 1984, Nature (London) 311: 223-27.

1986, Symp. Soc. Dev. Biol. 44: 177-94.

Seifert, Müller-Holtkamp, Marcey, and Jäckle, 1987, Roux's Arch. Dev. Biol. 196: 78-82 (fig.).

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Maternal-effect lethal. Embryos dorsalized, germline dependent. Phenotypic rescue achieved by injection of cytoplasm or poly(A)⁺RNA from wild-type embryos into young embryos produced by *spz* females; cytoplasm more effective than RNA (Seifert *et al.*). Temperature-sensitive period of *spz³* extends from oogenesis through fertilization until about the time of pole-cell formation. Strong alleles amorphic by deficiency testing. Pole-cell-transplantation studies indicate that *spz* acts in germ line and not soma of mother (Seifert *et al.*).

alleles: Four ethyl methanesulfonate-induced alleles: *spz¹* (weak), *spz²* (strong), *spz³* (temperature sensitive), and *spz⁴* (strong), originally isolated as 145, 197, 670, and *rm7*, respectively.

cytology: Placed in 97D15-E4 based on its inclusion in *Df(3R)9Q-RX1* (breakpoints not given) but not *Df(3R)ro80b* = *Df(3R)97D1;97D15* or *Df(3R)D605* (breakpoints not given).

****sq*: square**

location: 2-8.4.

discoverer: Bridges, 17h17.

phenotype: Wings truncated with squarish or oblique tip. Overlaps wild type. Viability erratic. RK3.

Sq: Squat*location:** 2-38.**origin:** Spontaneous.**discoverer:** Bridges, 15k29.**references:** Bridges and Morgan, 1929, Carnegie Inst. Washington Publ. No. 278: 283-84 (fig.).**phenotype:** Wings short, broad, blunt, arched, and less transparent than normal. Thorax and head short and broad. Legs short and weak. Overlaps wild type. Homozygous lethal. RK3.**sqh: spaghetti squash (D. P. Kiehart)****location:** 1-14.**origin:** P element insertion.**references:** Chang, Edwards, and Kiehart, in prep.; Karess, Kulkarni, and Aguilera, unpublished.**phenotype:** The structural gene for cytoplasmic myosin light chain (Chang *et al.*) In the homozygous *sqh*¹ mutant, the normally diploid larval tissues (*e.g.*, brain, imaginal disc, and gonad) possess numerous polyploid cells resulting from an intermittent failure of cytokinesis. Repeated rounds of apparently normal mitosis without cytokinesis produce cells containing hundreds of chromosomes. The mutant is a late larval-early pupal lethal with an extended larval period (10-14 days), during which the brain grows to enormous size. Imaginal discs are small and poorly formed (Karess).**alleles:** *sqh*¹ (isolation number 569) was found among a collection of larval and pupal lethals generated by Denzell and Johnson. Probably a hypomorph.**cytology:** Located in 5D5-6 by *in situ* hybridization with cytoplasmic-myosin-regulatory light chain and P element probes. Uncovered by *Df(1)5D1,2;5E*.**molecular biology:** The gene was cloned independently by P-element tagging (Karess *et al.*) and oligonucleotides corresponding to partial peptide sequence of purified cytoplasmic myosin light chain (Chang *et al.*). The 2.1 kb transcription unit contains two introns, of 1 kb and 73 bp. It expresses transcript(s) of approximately 1.1 kb at all developmental stages. Sequenced cDNAs encode a 174 amino acid protein of 19.9 kd, which is more similar to vertebrate smooth muscle regulatory light chains (80% identity) than to the *Drosophila* muscle homolog *Mlc2*. *sqh*¹ has an 800 bp internally deleted P element inserted in the 5' untranslated region of the transcription unit, 48 bp from the first codon of the open reading frame.**sr: stripe****location:** 3-62.0.**references:** Craymer, 1980, DIS 55: 197-200.

Costello and Wyman, 1986, Dev. Biol. 118: 247-58 (fig.).

phenotype: Trident pattern on thorax replaced by broad light gray stripe at 25°; stripe is dark at 18° or in the presence of heterozygous or homozygous *b* or *e*. Homozygotes are flightless (Levine and Wyman, 1973, Proc. Nat. Acad. Sci. USA 70: 1050-54) and show a reduction in size of a specific indirect flight muscle, the dorsal longitudinal muscle (DLM). The DLM is absent from *sr/Df(3)sr* adults. Early development of the indirect flight muscles is normal, but in the thirty-five-hour pupa the DLM begins to degenerate leading to its absence in adults (Costello and Wyman).**alleles:** All alleles except *sr*¹ are homozygous lethal, and all but *sr*¹ and *sr*^{61j} are associated with chromosome

rearrangements.

allele	origin	discoverer	ref ^α	comments
<i>sr</i> ¹		Bridges, 22b62		
<i>*sr</i> ^{3.2}	X ray	Alexander	1	<i>ln(3R)90D1-E1;93B-E</i>
<i>*sr</i> ^{4.2}	X ray	Alexander	1	<i>T(2;3)30C;90C-96</i>
<i>sr</i> ^{61j}	X ray	Puro, 1961	3	<i>sr</i> ¹ / <i>sr</i> ^{61j} overlaps wild type; homozygous lethal
<i>*sr</i> ^{100.23}	X ray	Alexander	4	<i>T(Y;3)90E2-3</i>
<i>*sr</i> ^{100.312}	X ray	Alexander	4	<i>T(2;3)40-41;90D2-E1</i>
<i>*sr</i> ^{300.240}	X ray	Alexander	4	<i>Tp(3;3)75C;89E;92A</i>

^α 1 = Alexander, 1960, Genetics 45: 1019-22; 2 = Bridges and Morgan, 1923, Carnegie Inst. Wash. Publ. No. 327: 244; 3 = Puro, 1982, DIS 58: 205-08; 4 = Ward and Alexander, 1957, Genetics 42: 42-54.**cytology:** Placed in 90D2-F7 based on its inclusion in the region common to *Df(3R)sr100.394 = Df(3R)90C2-7;90F3-7* and *Df(3R)sr300.101 = Df(3R)90D2-4;91A6-8*.***srb: smaller body****location:** 1-62.0.**origin:** Induced by S-mustard (CB. 1735).**discoverer:** Fahmy, 1960.**synonym:** *sby-62: small body 62*.**references:** 1964, DIS 39: 58.**phenotype:** Body size slightly reduced. Bristles finer. Both sexes viable. Female fertility low. RK3.**cytology:** Not included in deficiency for 18A4-18B8 formed by combining left end of *ln(1)y*⁴ = *ln(1)1A8-B1;18A3-4* with right end of *ln(1)sc*⁹ = *ln(1)1B2-3;18B8-9*, although *sby* (1-60.8) is included (Norton).**Src1: Src proto oncogene sequence****location:** 3- {15}.**origin:** Isolated from genomic library using v-src probe.**synonym:** *Dsrc*.**references:** Shilo and Weinberg, 1981, Proc. Nat. Acad. Sci. USA 78: 67789-92.

Simon, Kornberg, and Bishop, 1983, Nature (London) 302: 837-39.

Lev, Leibovitz, Segev, and Shilo, 1984, Mol. Cell. Biol. 4: 482-84.

Simon, Drees, Kornberg, and Bishop, 1985, Cell 42: 831-40.

phenotype: Considered to be the *Drosophila* sequence homologous to mammalian c-src, based both on its origin and amino acid sequence as inferred from its nucleotide sequence. The polypeptide product as yet uncharacterized, but presumed to be a protein kinase; *Drosophila* extracts do exhibit tyrosine kinase activity (Simon *et al.*, 1983). In the region of the polypeptide responsible for kinase and transforming activity, *Drosophila* amino acid sequence 54% homologous with that from v-src, 100 base pairs beginning with the tryosine-416 codon 62% homologous with *Drosophila Abl* (Hoffman, Fresco, Hoffman-Falk, and Shilo, 1983, Cell 35: 393-401). *Src* transcripts abundant in early embryos and in adult ovaries. Level declines in later embryos and is low in males and ovariectomized females; inferred to be a maternally acting gene whose product is required for early embryogenesis (Wadsworth, Madhavan, and Bilodeau-Wentworth, 1985, Nucleic Acids Res. 13: 2153-70).**cytology:** Located in 64B by *in situ* hybridization (Simon *et al.*, 1983).**molecular biology:** Gene cloned and sequenced; concep-

tual sequence indicates a 62-kd C-SRC protein with 40% homology with vertebrate C-SRC protein (Simon *et al.*, 1985). Developmental Northern blots reveal homologous transcripts of 3.0, 4.4, and 4.8 kb [3.5, 5.0, and 5.5 kb according to Simon *et al.* (1985)]; the 3.0 and 4.4 kb transcripts are abundant in unfertilized eggs and early embryos; disappear in larval and pupal stages, reappearing in adults. 4.8 kb transcript undetectable in unfertilized eggs; starts accumulating 2-5 h after fertilization and is expressed continuously throughout development (Lev and Segev, 1986, *Biochim. Biophys. Acta* 867: 144-51); similar developmental profiles noted by Simon *et al.* (1985). After first eight h of development, *Src* RNA accumulates almost exclusively in neural tissue and differentiating smooth muscle (Simon *et al.*, 1985).

Src2

location: 2- [22].

origin: Isolated from genomic library using v-src probe.

synonym: *src4*, *Dsrc28C*.

references: Simon, Kornberg, and Bishop, 1983, *Nature* (London) 302: 837-39.

Wadsworth, Madhavan, and Bilodeau-Wentworth, 1985, *Nucleic Acids Res.* 13: 2153-70.

Gregory, Kammermeyer, Vincent, and Wadsworth, 1987, *Mol. Cell. Biol.* 7: 2119-27.

Vincent, Gregory, and Wadsworth, 1989, *Genes Dev.* 3: 334-47.

phenotype: *Src* transcripts abundant in early embryos and in adult ovaries; also found in imaginal disks. Level declines in later embryos and is low in males and ovariectomized females. Inferred to be maternally acting gene whose product is required for early embryogenesis (Wadsworth *et al.*). Monoclonal antibodies detect two protein products of *Src2*, a doublet of 66 kd and one of 55 kd; the 55 kd polypeptide appears to initiate at the third methionine codon of the sequence; a faint 95 kd band appears to track the 66 kd band in developmental Northern blots. Monoclonal antibodies that differentiate between the two protein products show that they are differentially regulated. The 66 kd polypeptide first appears at the periphery of cells of the cellular blastoderm; this staining is transiently resolved into 13 or 14 segmental stripes plus uncharacterized staining in the head region in the fully extended germ band; no 66 kd protein detectable after 12 h of development. The 55 kd polypeptide first appears at 4-6 h and is widely distributed in the central and peripheral nervous systems. Larvae transformed with constructs that produce either the 66 or the 55 kd products under *Hsp70* promoter action display cell-surface localization of the 66 kd protein and cytoplasmic localization of the 55 kd protein in third-instar salivary glands.

cytology: Placed in 28C (Simon *et al.*) by *in situ* hybridization.

molecular biology: cDNA sequence reveals a single open reading frame capable of encoding a 590 amino-acid 66 kd polypeptide. Comparison with genomic sequence indicates presence of at least 6 introns. Conceptual amino-acid sequence reveals considerable homology to C-SRC over 70% of its length; 54% identity between C-SRC residues 270 and 470; amino-terminal 140 amino acids unrelated to C-SRC sequence; *Src2* contains a 58 amino-acid N-terminal extension lacking in C-SRC N-

terminal third of polypeptide has a 27-amino-acid stretch with 74% glycine, including eight contiguous glycine residues.

***srd: small round**

location: 1-0.6.

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1955.

references: 1959, DIS 33: 92.

phenotype: Fly small with slightly dark, rounder, small eyes. One or both postvertical bristles frequently missing. Both sexes viable and fertile. RK3.

Srf: Surf wings

location: 2-66.8.

origin: Spontaneous.

references: Kang and Park, 1971, DIS 46: 41 (fig.).

phenotype: In heterozygotes distal half of wings upturned about 40° from normal wing axis as in *Si* or *j*, but usually not divergent. Penetrance at 25° is 96% in females and 84% in males. Homozygous lethal; heterozygotes fully viable and fertile.

sro: shroud

location: 3-100.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95.

Roark, Mahoney, Graham, and Lengyel, 1985, *Dev. Biol.* 109: 476-88.

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Embryonic lethal. No differentiation of cuticle and mouthparts.

alleles: Two ethyl methanesulfonate-induced alleles, *sro*¹ and *sro*², originally isolated as 8A and 11C.

cytology: Placed in 99A-100A; covered by *Y^{P3}DB172* from *T(Y;3)YL;95A+In(3R)93B-C;99A* but not by *Y^{P3}DA113* from *T(Y;3)Xhy⁺;100A*.

srp: serpent

location: 3-58.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95.

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Embryonic lethal; germ-band shortening incomplete.

alleles: Three ethyl methanesulfonate-induced alleles, *srp*¹, *srp*², and *srp*³, isolated as 288, 6G, and 9L.

cytology: Placed in 88A11-89B4 based on its being deleted by *Df(3R)sbd105 = Df(3R)88F9-89A1* but not by *Df(3R)Po4 = Df(3R)87F7-89A1;89A11-13* nor by *Df(3R)sbd45 = Df(3R)89B4;89B10* (Hughes, Nelson, Yanuk, and Szauter).

srw: shrew

location: 3-15±.

origin: Induced by ethyl methanesulfonate.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95.

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Embryonic lethal; embryos partially ventralized.

alleles: One isolated as 10K.

Sry: Serendipity

location: 3- {101}.

references: Vincent, Colot, and Rosbash, 1985, *J. Mol. Biol.* 186: 149-66.
1986, *Dev. Biol.* 118: 480-87.
Schweisguth, Lepesant, and Vincent, 1990, *Genes and Dev.* 4: 922-93.

phenotype: The *Sry* region comprises three independently transcribed genes arranged in tandem, transcribed in the same direction, and separated by less than a kilobase. These are, from left to right, *Sry-β*, *Sry-α*, and *Sry-δ*.

cytology: Placed in 99D4-8 by *in situ* hybridization (Roark, Mahoney, Graham, and Lengyel, 1985, *Dev. Biol.* 109: 476-88).

molecular biology: Genomic clones of *Sry* region isolated independently by Vaslet, O'Conner, and Izquierdo (1980, *Nature (London)* 285: 674-76) and Roark *et al.* Sequence determined by Vincent *et al.* (1985). Five mRNA's are detected on Northern blots; three correspond to *Sry-α*, -β, and -δ and two correspond to β-α and α-δ read-through transcripts, which contain the spacer sequence between the respective genes. *Sry-α* transcripts detected in all nuclei and cells, except pole cells, during syncytial and cellular blastoderm; disappear during gastrulation (James and Vincent, 1986, *Dev. Biol.* 118: 474-79). Deficiency homozygotes display cellularization defects resulting in multinuclear cells at blastoderm. The molecular biology of the individual genes described below. α, β, and δ are transcribed off of the same strand, but each in a different reading frame.

Sry-α

molecular biology: The middle gene in the Serendipity region. Transcription begins 183 base pairs downstream from the 3' terminus of the *Sry-β* transcript and terminates 331 base pairs upstream from the initiation of transcription of *Sry-δ*; transcript size is 1.9 kb. The conceptual amino acid sequence contains 530 amino acids and 58 kd molecular weight. Expressed exclusively at the blastoderm stage of development; mostly associated with invaginating membranes at cellularization.

Sry-β

molecular biology: The left-most gene in the Serendipity region. The transcript size is 1.0 kb; the conceptual amino-acid sequence indicates a polypeptide of 351 amino acids with a molecular weight of 41,000. The carboxy-terminal portion of the *Sry-β* sequence appears to contain six tandemly arranged repeats of 28 or 29 amino acids, each of which contains two invariant cysteine and two highly conserved histidine residues; this segment of the gene shows high identity to the corresponding region of *Sry-δ* (87/172 residue identity). *Sry-β*. mRNA abundant in oocyte; maternally inherited.

Sry-δ

alleles: Four recessive lethal alleles, *Sry-δ¹¹*, *Sry-δ¹²*, *Sry-δ¹³*, and *Sry-δ¹⁴* isolated by Kongsuwan, Vincent, Lengyel, and Merriam (1986 Asilomar meeting).

molecular biology: The right-most gene in the Serendipity region. The transcript size is 1.5 kb; the conceptual amino-acid sequence indicates a polypeptide of 430 amino acids and molecular weight of 50,000. The identity to *Sry-β* in the region of the six repeated motifs has been discussed. Expression as detected by Northern blots

occurs throughout development, being high in oocytes and embryos and reduced during the rest of the life cycle.

Sryc: Serendipity cognate

location: 3- {99}.

synonym: *sry-hl*.

references: Vincent, Kejzlarová-Lepesant, Segalat, Yancostas, and Lepesant, 1988, *Mol. Cell. Biol.* 8: 4459-68.

phenotype: Structural gene for a protein of 868 amino acids with eight TFIIIA-like fingers. Developmental Northern blots reveal abundant transcript in adult females but not males and during the first four hours of embryonic development; low levels present in later embryos, larvae, and pupae. *In situ* hybridization to tissue sections shows that transcripts produced in nurse cells and beginning at stage 10 are transported to and accumulate in oocytes.

molecular biology: Gene recovered from genomic library by low-stringency hybridization with both *Sry-β* and *Sry-δ*. Probes derived from genomic clone detect a single mRNA of 3.2 kb. Genomic sequence comprises three exons and two introns, one between nucleotides 1263 and 1325 and the other between nucleotides 2528 and 2639. Three kilobases of genomic clone sequenced; conceptual amino-acid sequence contains 868 amino acids with calculated molecular weight of 96,000. The polypeptide contains two proline-rich domains in residues 141-213 and 593-669 and eight tandemly repeated 28- to 29-residue DNA-binding finger motifs in the central basic portion of the molecule; the first intron is in the loop of the third finger; an 11-residue polyalanine repeat resides 28 residues carboxy to the finger domain; also contains five potential glycosylation sites, three between residues 97 and 130. The 5' untranslated region contains five AUG codons, all followed shortly by in-frame stop codons.

cytology: Placed in 98E-F by *in situ* hybridization.

ss: spineless

location: 3-58.5.

references: Bownes, Bournias-Vardiabasis, and Spare, 1979, *Mol. Gen. Genet.* 174: 67-74.
Struhl, 1982, *Genetics* 102: 737-49.

phenotype: Mutations at the spineless locus display three different phenotypes: (1) The spineless phenotype is characterized by the reduction in size of all bristles; (2) the aristapedia phenotype corresponds to the transformation of distal antennal segments, specifically the arista and the distal portion of the third antennal segment, into distal mesothoracic leg segments, *i.e.*, tarsal segments; and (3) leg-segment fusion manifested as fusion of tarsal segments on all eight legs. Homeotic tissue does not conform to developmental compartment boundaries; therefore, *ss* does not qualify as a selector gene (Struhl). Expression of these phenotypes varies among alleles. Some alleles show only the spineless phenotype. Aristapedia alleles, symbolized *ss^a*, vary in expression; weak alleles show a swelling of the third antennal joint and rudimentary tarsal transformation of the base of the arista; as expression becomes more extreme, more tarsal joints are formed until in the most extreme alleles four tarsal segments and terminal claws are formed. Tarsal fusions are characteristic of extreme aristapedia alleles.

alleles:

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discoverer	synonym	ref ^α	comments
ss¹	spont	Bridges, 14a3		3, 6, 13	
*ss^{37b}	spont	Poulson, 37b		6, 15	
ss^a	spont	Balkaschina, 1926		1, 2, 6, 7, 11, 22	
ss^{a40a}	spont	Buzzati-Traverso, 40a2		2, 4, 6, 11, 23	cold sensitive
*ss^{a41l}	spont	Neel, 41i30		6, 14	
*ss^{a44a}	spont	Buzzati-Traverso, 44a17		5, 6	weak derivative of <i>ss^{a40a}</i>
*ss^{a52g}	spont	Meyer, 52g		6, 10	
*ss^{a53e}	spont	Piternick, 1953		6	
*ss^{a63c}	spont	Merriam, Piternick, 63c		6	
ss^{aB}	spont	Bridges, 38a11		2, 6, 11, 18, 22, 23	weak allele; cold sensitive
ss^{aC1}	EMS		<i>ss^{aCam}</i>	2, 12	
ss^{aC2}	EMS	Struhl		17	
ss^{aC3}	EMS	Struhl		17	weak allele
ss^{aC4}	EMS	Struhl		17	
ss^{aC5}	EMS	Struhl		17	strong allele
ss^{aC6}	EMS	Struhl		17	
ss^{aC7}	EMS	Struhl		17	
ss^{aC8}	EMS	Struhl		17	
ss^{aC9}	EMS	Struhl		17	
ss^{aC10}	EMS	Struhl		17	strong allele
ss^{aC11}	EMS	Struhl		17	strong allele
ss^{aC12}	EMS	Struhl		17	
ss^{aC13}	EMS	Struhl		17	
ss^{aC14}	EMS	Struhl		17	
ss^{aC15}	EMS	Struhl		17	deficiency
ss^{aC16}	EMS	Struhl		17	strong allele
*ss^{aD}	X ray	Puro, 1962			dominant
*ss^{aF}	spont	von Finck, 1937		6, 20, 19	
ss^{aH}	spont	Hannah-Alava		8	
ss^{ak}	spont			2, 11	
ss^{aP88}	X ray	Lewis			<i>In(3LR)61A1-2;89C2-4</i> ; strong allele
ss^{aSp}	spont	Spencer, 36d15		6, 16, 22	
ss^{asnB}				23	
ss^{aUCI}	EMS	Vyse, James		2, 21	
ss^{ax}	spont	Hexter		2, 9, 11	
ss^{iso53}	spont	Piternick, 1953		6	
ss^v	X ray	Lewis		6	<i>T(1;3)20;89B;100F</i>

^α 1 = Balkaschina, 1929, Arch. Entwicklungsmech. Organ. 115: 448-63 (fig.); 2 = Bownes, Bournias-Vardiabasis, and Spare, 1979, Mol. Gen. Genet. 174: 67-74; 3 = Bridges and Morgan, 1923, Carnegie Inst. Wash. Publ. No. 327: 109 (fig.); 4 = Buzzati-Traverso, 1940, DIS 13: 49; 5 = Buzzati-Traverso, 1949, DIS 23: 57; 6 = Carnegie Publication 627; 7 = Gehring and Schubiger, 1975, J. Embryol. Exp. Morph. 33: 459-69; 8 = Goldschmidt, 1951, Pan-Pacific Ent. 27: 1-11; 9 = Hexter, Lozner, and Bunn, 1967, Genetics 56: 565; 10 = Meyer, 1952, DIS 26: 67; 11 = Mglinetz, 1974, Genetika 10(#1): 92-98; 12 = Morata and Lawrence, 1979, Dev. Biol. 70: 355-71; 13 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 211 (fig.), 236; 14 = Neel, 1942, Genetics 27: 530; 15 = Poulson and King, 1948, DIS 22: 55; 16 = Spencer, 1937, DIS 7: 5; 17 = Struhl, 1982, Genetics 102: 737-49; 18 = Villee, 1943, J. Exp. Zool. 93: 75-98 (fig.); 19 = Vogt, 1946, Biol. Zentralbl. 65: 238-54; 20 = von Finck, 1942, Biol. Zentralbl. 62: 379-400; 21 = Vyse and James, 1972, DIS 49: 39; 22 = Waddington, 1939, Growth Suppl. 1: 37-44; 23 = Waddington and Clayton, 1952, J. Genet. 51: 123-29.

cytology: Placed in 89C1-2 (Lewis, 1963, Am. Zoologist 3: 33-56).

other information: *ss* and *ss^a* to the left of both *ss^{a40a}* and *ss^{ax}* (Hexter, Lozner, and Bunn, 1967, Genetics 56: 565).

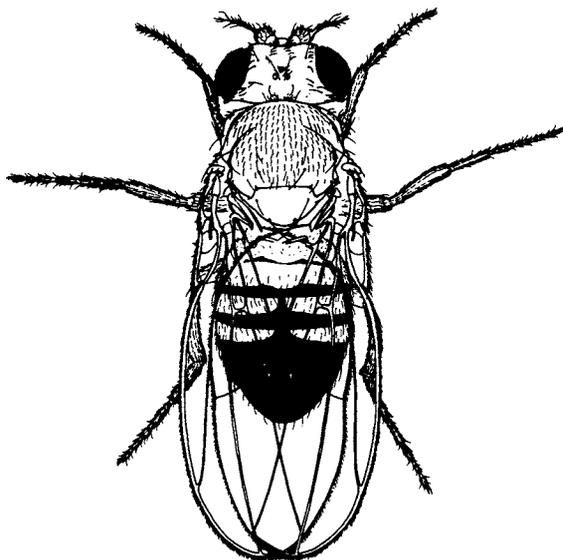
ss¹

phenotype: Bristles only a little larger than hairs; dorso-centrals least reduced; postscutellars erect. No effect on legs or arista. Growth of bristles slows during development [Lees and Waddington, 1943, Proc. Roy. Soc. (London), Ser. B 131: 87-110]. Dominant to aristapedia phenotype of all *ss^a* alleles. RK1.

ss^a: spineless-aristapedia

phenotype: Antennae and arista tarsuslike; mean number of tarsal segments = 4.0; incidence of claws = 97.1% (García-Bellido, 1968, Genetics 59: 487-99); classified as an intermediate allele by Struhl. Third joint of antenna like parts of a tarsal row but with broad, flat, plate-like lobes below. Tarsal segments of legs display intermediate level of fusion (Struhl). Bristles like those of a medium to slight Minute. Frequent extra dorsocentral bristles. Transformed tissue is leg tissue in every attri-

bute tested. Transformed tarsi elicit behavioral response similar to that of normal legs when exposed to sugar solutions (Deak, 1976, Nature 260: 252-54). Regions of arista converted into tarsi not affected by mutants affecting arista (*e.g.*, *th* and *al*) but are affected by those operating on tarsi (*e.g.*, *ff*, *d*, *app*, and *ey*) [Waddington, 1939, Growth, Suppl. 1, pp. 37-44; Braun, 1940, Genetics 25: 143-49; Mglinetz and Ivanov, 1975, Genetika 11(#11): 27-33; 1976, Genetika 12(#12): 87-94]. Dissociated cells from *ss^a* antennal disks aggregate with dissociated leg-disk cells but not with those from wild-type antennal disks (García-Bellido). Antennal disks from *ss^a* larvae give rise to leg-like structures when transplanted into wild-type hosts as do both duplicated and regenerated antennal disks formed from eye-antenna-disk fragments (Gehring and Schubiger, 1975, J. Embryol. Exp. Morph. 33: 459-69); when disks are pretreated with colchicine, the developing structures are more arista-like (Vogt, 1947, Experientia 3: 156-59). Homozygous clones of *ss^a* tissue produce antennal leg tissue when induced before (Roberts, 1964, Genetics 49: 593-98) but not after (Postlethwait and Girton, 1974, Genetics 76: 767-74) mid-third instar. Large clones conform to



ss^a: spineless-aristapedia

From Bridges and Brehme, 1944, Carnegie Inst. Washington Publ. No. 552: 179.

anterior and posterior compartments comparable to those induced in normal mesothoracic legs [Morata and Lawrence, 1979, Dev. Biol. 70: 355-71 (fig.)]. No maternal effect; temperature independent.

ss^{a40a}

phenotype: Cold sensitive; phenotype normal in flies raised at 29°; fully penetrant at 17° [mean number of tarsal segments = 2.8; incidence of tarsal claws = 1.3% as recorded by García-Bellido (1968, Genetics 59: 487-99) probably in flies raised at 25°]. Phenocritical stage in first half of third instar [Grigliatti and Suzuki, 1971, Proc. Nat. Acad. Sci. USA 68: 1307-11 (fig.)]. Shifts from 17° to 29° early in third instar restrict transformation to base of arista; distal extent of transformation increases as shift is effected later in development (Grigliatti and Suzuki). Opposite response to temperature for antennal transformation but not tarsal fusion reported by Mglinetz (1977, Genetika 13: 70-75). Developmental compartments demonstrated in antennal legs [Morata and Lawrence, 1979, Dev. Biol. 70: 355-71 (fig.)].

ss^{aB}: spineless-aristapedia of Bridges

phenotype: Bristles of female like a slight Minute, especially postscutellars. At 25°, arista inconspicuously thickened at base, plumed or threadlike for rest of extent. At 18° penetrance is 7% and expression weak; at 14°, ss^{aB} enhanced and resembles ss^a; al causes reduction of ss^{aB} arista at 25° but has no effect on transformed arista at 14° (Villee, 1943, J. Exp. Zool. 93: 75-98). Legs frequently have lumps at second joint of tarsi; more pro-

nounced in male and result in doubling of sex combs, which are strung along first and second fused joints. Eyes a little flattened. Except at low temperatures, all characters slight and may overlap wild type. ss^{aB}/ss¹ has slight Minute phenotype but wild-type legs and arista. ss^{aB}/ss^{asp} like ss^{asp} with large tarsal arista. RK2. Struhl (1982, Genetics 102: 737-49) and Posakony.

***ss^{aH} (A. Hannah Alava)**

phenotype: Homozygous viable and fertile. Displays enlargement of base of arista. Increased number of sex-comb teeth on basitarsus of first leg in males (12-38 with mean of 24 teeth per leg).

***ss^{asnB} (A. Hannah Alava)**

phenotype: Description incomplete. Extreme fusion of tarsal segments; basitarsus of male first leg has 20-50 sex-comb teeth (mean = 40); sex-comb teeth not uncommon on second leg.

ss^A: see Antp^R

ss^{Ar}: see Antp^{LC}

ss^{C1}

phenotype: Reported to be homozygous lethal and lethal in combination with Df(3R)bxd100 (Bownes, Bournias-Vardiabasis, and Spare, 1979, Mol. Gen. Genet. 174: 67-74); however both of these combinations viable in the hands of

***ss^{iso53}: spineless-isoallele**

phenotype: Homozygote is wild type. ss^{iso53}/ss^a, ss^{iso53}/ss^{a63c}, and ss^{iso53}/ss^{a53e} have thickened proximal segments of arista, like ss^{aB}. RK3.

ss^V: spineless-variegated

phenotype: Variegates for spineless character but completely mutant for aristapedia. Male sterile. RK2A.

st: scarlet

location: 3-44.0.

phenotype: Eyes bright vermilion, darkening with age. Ocelli colorless, even in old fly; a reliable trait for classifying st se. Eyes of bw; st white. Eye color autonomous in larval optic disks transplanted into wild-type hosts (Beadle and Ephrussi, 1936, Genetics 21: 230). Larval Malpighian tubes pale yellow (Beadle, 1937, Genetics 22: 587-611). Phenocritical period, as indicated by temperature-shift experiments utilizing st¹², is 24-48 h after pupariation at 25° (Howells, 1973). Uptake of kynurenine by Malpighian tubules and eye disks severely deficient (Sullivan and Sullivan, 1975, Biochem. Genet. 13: 603-13). 3-hydroxykynurenine accumulates only in pupae; virtually undetectable in larvae and adults (Howells and Ryall, 1976, Biochem. Genet. 14: 1077-90). Activity of phenoxazinone synthetase, which is found in pigment granules and which catalyzes the bimolecular condensation of 3-hydroxykynurenine in the production of brown eye pigment, xanthommatin, markedly reduced in st (Phillips, Forrest, and Kulkarni, 1973, Genetics 73: 45-56). RK1.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>st</i> ¹	spont	Richards, 16K18		3, 8, 9	7.6 kb insert in -1.7 to -0.8 kb
<i>st</i> ²	UV	Meyer, 54i	<i>st</i> ⁵⁴ⁱ	7	
<i>st</i> ³	MR	Green	<i>st</i> ^{82c3}		
<i>st</i> ⁴	spont	Tearle	<i>st</i> ^{82k}		
<i>st</i> ⁵	γ ray	Velissariou		1	<i>In(3L)73A2-3;80C</i>
* <i>st</i> ⁶	X ray		<i>st</i> ^{100.126}	10	<i>T(Y;3)73A2-3</i>
* <i>st</i> ⁷	X ray		<i>st</i> ^{100.359}	10	<i>T(2;3)21C3-5;73A2-3;98F2-4</i>
<i>st</i> ⁸	EMS	Howells	<i>st</i> ⁷⁴¹	5	
<i>st</i> ⁹	EMS	Howells	<i>st</i> ⁷⁵¹	5	severe hypomorph ^γ
<i>st</i> ¹⁰	EMS	Howells	<i>st</i> ⁷⁵²	5	moderate hypomorph ^γ
<i>st</i> ¹¹	EMS	Howells	<i>st</i> ⁷⁵³	5	
<i>st</i> ¹²	EMS	Howells	<i>st</i> ⁷⁵⁴	5	temperature sensitive
<i>st</i> ¹³	EMS	Howells	<i>st</i> ⁷⁵⁵	5	moderate hypomorph
<i>st</i> ¹⁴	X ray	Tearle	<i>st</i> ²⁴¹	9	restriction map normal
<i>st</i> ¹⁵	X ray	Tearle	<i>st</i> ^{a24}	9	restriction map normal
<i>st</i> ¹⁶	X ray	Tearle	<i>st</i> ^{a27}	9	<i>In(3LR)73A3-4;87D13-14</i>
<i>st</i> ¹⁷	X ray	Tearle	<i>st</i> ^{a31}	9	
<i>st</i> ¹⁸	X ray	Tearle	<i>st</i> ^{b22}	9	restriction map normal
<i>st</i> ¹⁹	X ray	Tearle	<i>st</i> ⁸²²	9	restriction map normal
<i>st</i> ²⁰	X ray	Tearle	<i>st</i> ⁸²⁰²	9	restriction map normal
<i>st</i> ²¹	X ray	Kennison	<i>st</i> ^{JK1}	9	
<i>st</i> ²²	EMS	Kennison	<i>st</i> ^{JK2}		
<i>st</i> ²³	EMS	Kennison	<i>st</i> ^{T1}		
<i>st</i> ²⁴	spont	Kennison	<i>st</i> ^{JK3}		in <i>TM3</i>
<i>st</i> ²⁵	X ray		<i>st</i> ^{k21}	9	<i>In(3)73A3-4;80-81</i>
<i>st</i> ²⁶	X ray		<i>st</i> ^{L15}	9	
<i>st</i> ²⁷	X ray	Marsh	<i>st</i> ^{LM2}	6	
<i>st</i> ²⁸	X ray	Marsh	<i>st</i> ^{LM10}	6	
<i>st</i> ²⁹	X ray	Marsh	<i>st</i> ^{LM54}	6	
<i>st</i> ³⁰	X ray		<i>st</i> ^{SS34}	2	
<i>st</i> ³¹	EMS		<i>st</i> ^{T1}		
<i>st</i> ^{sp}	spont	Bridges, 36b19		4, 9	5.2 kb insert in -3.0 to -2.1 kb

^α 1 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-90; 2 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95; 3 = Bridges, and Morgan, 1923, Carnegie Inst. Wash. Publ. 327: 172 (fig.); 4 = Carnegie Inst. Wash. Publication 627; 5 = Howells, 1979, Biochem. Genet. 17: 149-58; 6 = Marsh and Mock, 1985, DIS 61: 214; 7 = Meyer, 1954, DIS 28: 77; 8 = Richards, 1918, Biol. Bull. 35: 199-206; 9 = Tearle, Belote, McKeown, Baker, and Howells, 1989, Genetics 122: 595-606; 10 = Ward and Alexander, 1957, Genetics 42: 42-54.

^β Molecular coordinates based on Tearle *et al.*; origin defined as the *HindIII* site nearest to the left breakpoint of *In(3LR)st-a27 = In(3LR)73A3-4;87D13-14*; polarity undefined.

^γ Severe hypomorphs have yellow eyes in combination with *bw* and approximately 10% wild-type levels of xanthommatin; moderate hypomorphs have orange eyes in combination with *bw* and 25-45% wild-type levels of xanthommatin.

cytology: Placed in 73A3-4 by deficiency mapping (Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-90); restricted to distal two-thirds of the 73A3-4 doublet by Tearle *et al.* on the basis of the *st*⁺ phenotype of *Df(3L)tra*, which removes the proximal edge of the doublet.

molecular biology: The region of the gene has been cloned, restriction mapped, and partially sequenced; coordinates established from origin defined as *HindIII* site nearest to left breakpoint of *In(3LR)st-a27* (Tearle, Belote, McKeown, Baker, and Howells, 1989, Genetics 122: 595-606); transformation with a segment extending from -7.3 to +1.4 rescues *st* phenotype. Restriction site polymorphisms in vicinity identified. Northern blots probed with riboprobes made from restriction fragments in region identify a 2.3 kb polyadenylated mRNA transcribed from a genomic sequence containing a large intron in the N-terminal half of the gene. Transcript present with substantially lower abundance than *w* transcript, and level independent of genotype at *w* locus. Developmental Northern blots show the presence of transcript throughout development, but level about ten-fold higher during early to mid pupa. A segment of the sequence between -678 and -354 shows homology with a segment of the *w* gene; sequence determined by Tearle *et*

al. includes one of the putative ATP-binding sites of *w*, where 72% homology is observed.

***st*¹²**

phenotype: Scarlet eyes at 29°; normal eye color at 18°; xanthommatin levels 9% and 72% of wild type respectively (Howells, 1979, Biochem. Genet. 17: 149-58).

***st*^{sp}: scarlet-spotted**

phenotype: Eyes scarlet with facets and groups of facets that appear wild type. Darkening spreads in old fly. Not a variegated position effect. *st*^{sp}/*st* like *st*^{sp}. Larval Malpighian tubules pale yellow and classifiable (Brehme and Demerec, 1942, Growth 6: 351-56). RK2.

cytology: Salivary chromosomes appear normal.

****St*: Stumpy**

location: 1-55.5.

origin: X ray induced.

discoverer: Muller, 2612.

references: 1935, DIS 3: 30.

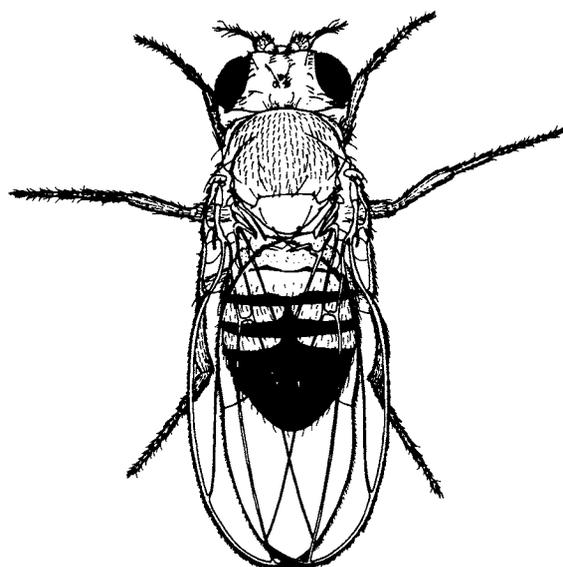
phenotype: Wings and abdomen short. Bristles Minute. Eyes rough. Male lethal. RK2.

***St*-*SD*: Stabilizer of Segregation Distorter**

references: Sandler and Hiraizumi, 1960, Genetics 45: 1269-87.

Miklos, 1972, Genetics 70: 405-18.

phenotype: Increases k value of *SD* chromosomes and by so doing decreases the male-to-male variability of k. Located to the right end of *2R* and affects k either in coupling or repulsion to *Sd*. Decreases fecundity and temperature response of *SD/+* males (Hartl, Hiraizumi, and Crow, 1967, Proc. Nat. Acad. Sci. USA 58: 2240-45; Hikawa, 1971, Jpn. J. Genet. 46: 75-82). Existence inferred from behavior of various recombinant *SD* chromosomes. Probit analysis by Miklos (1972) indicates that the phenotype results not from a single locus, but from the cumulative effects of several weak enhancers; variability in k values in *SD/+* genotypes lacking *St-SD* shown algebraically to be the consequence of reduced k values [see also Hartl, 1976, Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 615-66].



sta: stubarista

From Bridges and Brehme, 1944, Carnegie Inst. Washington Publ. No. 552: 180.

sta: stubarista

location: 1-0.3.

phenotype: Third joints of antennae short, blunt, free of hairs, and yellowish. Arista bases thickened, axes sometimes short, and branches irregular. All bristles and hairs extremely short and sparse. Eyes rotated on head slightly so that the long axis is vertical. Homozygous *sta*¹ females unable to oviposit; stage 14 oocytes appear normal (King, 1970, Ovarian Development in *Drosophila*, Academic Press, New York). Lethal allele (*sta*²) in combination with variegating translocation (*T(1;2)dor*^{var7}) produces incised wing margins, small rough eyes with missing facets, and missing head macrochaetae [Demakova and Belyaeva, 1988, DIS 67: 21 (fig.)]. RK2A.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>sta</i> ¹	X ray	Oliver, 32122		3	<i>Tp(1;3)1E1-2;2B3-4;89B21-C4</i>
<i>sta</i> ²	EMS		<i>sta</i> ¹³	1,2	lethal allele

allele	origin	discoverer	synonym	ref ^α	comments
<i>sta</i> ³	EMS		<i>sta</i> ^{13b}	1,2	lethal allele

^α 1 = Aizenzon and Belyaeva, 1982, DIS 58: 3-7; 2 = Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 185-90; 3 = Oliver, 1935, DIS 4: 15.

cytology: Placed in region between 1E1 and 2B4 on the basis of its association with *Df(1)sta = Df(1)1E1-2;2B3-4*.

sta^P: see *crm*

Sta: Stigmata

location: 3-rearranged.

origin: X ray induced on *T(2;3)P10*.

discoverer: Lewis, 1978.

references: Craymer, 1984, DIS 60: 234-36.

phenotype: Pigment absent from corners and mid-anterior edge of notum. Wings held out from body, becoming more extreme with age.

cytology: Associated with *In(3LR)Sta = In(3LR)79D;94A*.

Stabilizer of Segregation Distorter: see **St-SD**

stall: see *stl*

stambh: see *stm*

stand still: see *stil*

standby: see *fil*⁸

Star: see **S**

stardust: see *sdt*

staroid: see *std*

stau: staufen (T. Schüpbach)

location: 2-83.5.

references: Schüpbach and Wieschaus, 1986, Roux's Arch. Dev. Biol. 195: 302-17.

Nüsslein-Volhard, Frohnhofer, and Lehman, 1987, Science 238: 1675-81.

Schüpbach and Wieschaus, 1989, Genetics 121: 110-17.

phenotype: Maternal-effect lethal. Embryos from homozygous mothers exhibit a so-called "grandchildless-knirps" phenotype; all eggs lack polar granules and no pole cells are formed; most embryos show variable deletions of abdominal segments, whereby segment A4 is deleted most frequently; larger deletions may delete segments A2 through A7; in extreme cases, anterior parts of segment A1 become fused to posterior parts of segment A8, but telson elements are always present and relatively normal. In addition, embryos show deletions of the anterior-most head structures and the cephalic furrow is shifted anteriorly at gastrulation. Analysis of germline clones indicates that the mutation is germline autonomous (Schüpbach and Wieschaus, 1986, Dev. Biol. 113: 443-48).

alleles:

allele	origin	synonym	comments
<i>stau</i> ¹	EMS	<i>HL</i>	weak allele
<i>stau</i> ²	EMS	<i>D3</i>	strong allele
<i>stau</i> ³	EMS	<i>C8</i>	temperature sensitive
<i>stau</i> ⁴	EMS	<i>G2</i>	weak allele

cytology: Placed in 54F6-55C1, since uncovered by

Df(2R)Pcl7B = *Df(2R)54E8-F1;55B9-C1* and
Df(2R)Pcl11B = *Df(2R)54F6-55A1;55C1-3*.

stb: see *sbd*

***stb: short bristle**

location: 1-14.6.
origin: Induced by L-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).
discoverer: Fahmy, 1955.
references: 1958, DIS 32: 75.
phenotype: Short, thin bristles. Viability and fertility good. RK2.

std: staroid

location: 2-56.5.
origin: Spontaneous.
discoverer: E. M. Wallace, 31c26.
phenotype: Eyes small, oval, and very rough. Bristles short. Wings slender, dusky, and warped; marginal veins irregular; gap in L4; L5 short. Body dwarfed. Thorax has dark streak. Male sterile; female semisterile. Viability variable. At 19°, eye character remains but other abnormalities disappear. RK2.

std: see *Bd*^{rl}

Ste: Stellate

location: 1-45.7.
references: Meyer, Hess, and Beerman, 1961, Chromosoma 12: 676-716.
 Hardy, 1980, DIS 55: 54-55.
 Lovett, Kaufman, and Mahowald, 1980, Eur. J. Cell Biol. 22: 49.
 Lovett, 1983, PhD Thesis, Indiana University, Bloomington, Indiana.
 Hardy, Lindsley, Livak, Lewis, Sivertsen, Joslyn, Edwards, and Bonaccorsi, 1984, Genetics 107: 591-610.
 Livak, 1984, Genetics 107: 611-34.
 Livak, 1990, Genetics Submitted.

phenotype: This locus is responsible for the appearance of crystals in the nuclei and cytoplasm of primary spermatocytes of *XO* males. Enzymatic treatments indicate that these crystals are proteinaceous in nature (Meyer *et al.*). The suppression of crystal formation by the *Y* chromosome is attributable to a sequence designated *Su(Ste)*.

alleles: Two forms of *Ste*, designated *Ste* and *Ste*⁺, can be distinguished; *Ste*⁺/*0* males produce needle-shaped crystals that are longer than cell diameters and thus curve to conform to cellular boundaries, whereas *Ste/0* males produce star-shaped aggregates of shorter crystals.

cytology: Placed in 12F1-2 by *in situ* hybridization (Lovett *et al.*).

molecular biology: The testes of *XO* males produce an abundant 750-base poly-A⁺ transcript that is rare in *XY* testes; transcript also not found in male carcasses or in females. *XO*-cDNA-detected genomic clones were isolated by Lovett; Livak showed them to comprise tandem repeats of a 1250 base-pair sequence and estimates that there are 200 copies on *Ste*-bearing *X* chromosomes and low copy numbers in *Ste*⁺-bearing *Xs*; the *X* of *D. simulans* apparently lacks the sequence altogether. Sequence determinations by Livak (1990) reveals two major classes of Stellate repeats which differ by 150 base pairs in the 3' end of the gene. No TATA element associated with the

Ste promoter. The cDNA sequence reveals the presence of two introns, which are more efficiently spliced in *XO* than *XY* testes; the conceptual amino acid sequence suggests a 19,500-dalton protein that shares homology with the β subunit of casein kinase II (Henikoff). The fact that the *Ste* sequence is as similar to the bovine casein kinase sequence as it is to the *Drosophila* sequence suggests that the *Ste* and casein kinase sequences are probably not of recent common origin. A single *Ste* repeat sequence transposed into autosomal locations is regulated by the *Y* chromosome in the same way as are the endogenous sequences.

Sternopleural: see Sp

stf: stormfront

location: 1-35.
origin: Induced by ethyl methanesulfonate.
references: Eberl and Hilliker, 1988, Genetics 118: 109-20.

phenotype: Sex-linked-recessive lethal. Hemizygous embryos exhibit failure of head involution and occasional defects in the ventral-denticle-belt pattern or the failure of germ-band retraction.

cytology: Placed in 10A7-11 based on its location between the proximal breakpoints of *Df(1)v-L3* = *Df(1)9F10;10A7-8* and *Df(1)9E1;10A11;56A*.

stg: string

location: 3-99.
references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.
 Edgar and O'Farrell, 1989, Cell 57: 177-87.

phenotype: Homozygous embryonic lethal; denticle bands reduced. First 13 nuclear divisions of zygote proceed on schedule; division 14 permanently arrested in G2 in homozygotes for strong alleles; no evidence of nuclear-envelope breakdown or chromosome condensation; division 14 severely impaired in weak alleles. DNA synthesis in arrested embryos confined to the polyploid amnioserosal cells. Gastrulation proceeds on schedule in *stg* embryos with but 5,000 cells, and markers ordinarily expressed in normal embryos with 50,000 cells also expressed in *stg* embryos. The only defect seems to be in the initiation of the first mitotic division that is under zygotic control. Clones of homozygous cells induced early don't survive; few late clones produced.

alleles:

allele	origin	synonym	ref ^α	comments
stg 1	EMS	<i>stg</i> ^{4B}	2, 4	temperature sensitive
stg 2	EMS	<i>stg</i> ^{7B}	2, 4	strong allele
stg 3	EMS	<i>stg</i> ^{7L}	2, 4	strong allele
stg 4	EMS	<i>stg</i> ^{7M}	4	weak allele
stg 5	EMS	<i>stg</i> ^{8A}	2, 4	strong allele
stg 6	EMS	<i>stg</i> ^{9A}	2, 4	weak allele
stg 7	EMS	<i>stg</i> ^{9K}	2, 4	weakest allele
stg 8	EMS	<i>stg</i> ^{13D}	4	weak allele
stg 9	EMS	<i>stg</i> ^{11B}		
stg 10	EMS	<i>stg</i> ¹⁰		
stg 11	P	<i>l(3)1D3</i>	3	strong allele γ
stg 12	P	<i>l(3)3A1</i>	3	β, γ
stg 13	P	<i>l(3)neo61</i>	1	β
stg 14	P	<i>l(3)neo62</i>	1	β, γ

- α 1 = Cooley, Kelley, and Spradling, 1988, *Science* 239: 1121-28; 2 = Jan (unpublished); 3 = Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95; 4 = Edgar and O'Farrell, 1989, *Cell* 57: 177-87.
- β These alleles are homozygous lethal and do not display a *stg* phenotype; however, they fail to complement completely strong *stg* alleles.
- γ P insert detected in *EcoRI* fragment 5' to start of transcription.

cytology: Placed in 99A by *in situ* hybridization. Covered by 3^P*Y*^D from *T(Y;3)B172 = T(Y;3)YL;93B-C;95A;99A*, but not from *T(Y;3)B152 = T(Y;3)h3;98F*.

molecular biology: Region isolated by transposon tagging and plasmid rescue; 33 kb of flanking DNA cloned and restriction mapped. Probing of Northern blots reveals two transcripts of 2.8 and 3.0 kb, which are abundant during nuclear divisions 1-13 and 14-16 but not in post-division-16 cells. *stg*¹¹ has no detectable transcript during postblastoderm development. Conceptual amino-acid sequence indicates a protein with 479 amino acids, the C-terminal 187 amino acids of which exhibit 34% identity with *cdc25*, a mitotic initiator gene of *S. pombe*. The sequence has no introns. *In situ* hybridization to embryos reveals an uniform distribution of maternal transcript present throughout the first thirteen nuclear divisions; during the first 20-30 m of interphase 14 the maternal message is rapidly degraded and zygotic transcription begins 25-30 m before cells enter mitosis 14. Zygotic expression is not observed in strong *stg* mutants (e.g., *stg*¹¹). During gastrulation the spatio-temporal pattern of *stg* expression anticipates rather exactly that of mitosis.

***sth: small thin**

location: 1-3.7.

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1956.

references: 1959, *DIS* 33: 92.

phenotype: Fly small, has short thin bristles. Eyes frequently deformed and rough. Wing shape and position slightly atypical. Male ecloses late but is viable and fertile. Female sterile. RK3.

sticking: see *stk*

stiff chaetae: see *sfc*

Stigmata: see *Sta*

stil: stand still (T. Schüpbach)

location: 2-63.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus.

phenotype: Female sterile; homozygous females contain few developing egg chambers in their ovaries, which may contain abnormal numbers of nurse cells and usually degenerate before yolk uptake begins.

alleles: *stil*¹ and *stil*² recovered as WE and PS respectively.

cytology: Placed in 48D-49E, since uncovered by *Df(2R)vg135 = Df(2R)48D-E;49D-E*.

stk: sticking

location: 3- {47}.

references: Cavener, Otteson, and Kaufman, 1986, *Genetics* 114: 111-23.

phenotype: Undefined prepupariation effective lethal

phase; *stk/T(2;3)Ta*¹ fails to complete eclosion.

alleles:

allele	origin	discoverer	synonym
<i>stk</i> ¹	EMS	Cavener	<i>l(2)g2</i>
<i>stk</i> ²	EMS	Cavener	<i>l(2)g5</i>
<i>stk</i> ³	EMS	R. Lewis	<i>l(2)r13</i>

cytology: Located in 84B1-C2 based on its inclusion in *Df(3R)Scx2 = Df(3R)84A4-5;84C1-2* but not *Df(3R)Win3 = Df(3R)84A4-5;84B1-2*. Also, not complemented by *T(2;3)Ta*¹ = *T(2;3)51E1-2;84B1-2*.

stl: stall (T. Schüpbach)

location: 2-102.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus.

phenotype: Female-sterile; homozygous females often contain ovarian tumors; ovarioles filled with small undifferentiated cells, as well as egg chambers containing variable numbers of nurse cells and degenerating material. Occasional normal egg chambers take up yolk, but form abnormal, collapsing eggs which remain unfertilized.

alleles: Four alleles, *stl*¹, *stl*², *stl*³, and *stl*⁴ - isolated as *WU, AWK, PA, and PH*.

stm: stambh

Three ethyl methanesulfonate-induced temperature-sensitive paralytic mutations at different loci on chromosome 2. Paralysis dependent on time and temperature of exposure; the recovery time is proportional to the severity of the paralyzing exposure.

references: Shyngle and Sharma, 1985, *Indian J. Exp. Biol.* 23: 235-40.

locus	location	comments
<i>stm-A</i>	2-56.8	paralyzed at 35°; rapid paralysis at 39°; larval paralysis; complements <i>nap</i> ¹⁵
<i>stm-B</i>	2-97.6	paralyzed at 37°; slow paralysis at 39°; no larval paralysis
<i>stm-C</i>	2-59.4	paralyzed at 35°; rapid paralysis at 39°; no larval paralysis

stm-A

phenotype: Two-hour heat shocks applied throughout development show 0-8 h and 56-140 h to be highly sensitive, with no survival; 8-56 h and after 140 h show survival with normal phenotype. Early temperature shock leads to blastoderm arrest with no gastrulation. Embryos produced by homozygous *stm-A* mothers are exceedingly sensitive to a two-hour exposure to 36°; leads to complete lethality from 0-3 h irrespective of paternal contribution.

stn: stoned (J.C. Hall)

location: 1-66.3 (Grigliatti *et al.*, 1973) or 68.5 (Homyk and Pye, 1988).

references: Grigliatti, Hall, Rosenbluth, and Suzuki, 1973, *Mol. Gen. Genet.* 120: 107-14.

Kelly, 1983a, *Cell. Mol. Neurobiol.* 3: 127-41.

1983b, *Cell. Mol. Neurobiol.* 3: 143-49.

Miklos, Kelly, Coombe, Leeds, and Lefevre, 1987, *J. Neurogenet.* 4: 1-19.

Homyk and Pye, 1988, *J. Neurogenet.* 5: 37-48.

Perrimon, Smouse, and Miklos, 1989, *Genetics* 121: 313-31.

phenotype: Exists as temperature-sensitive behavioral,

temperature-sensitive

lethal, and unconditionally lethal alleles. Severe behavioral debilitation is quickly induced by shift of adult *stn*⁶ and *stn*⁷ from 25° to 29°; abnormalities at high temperature include uncoordinated leg and wing movements, with complete paralysis never occurring (e.g., legs still move); *stn*⁶ causes some debilitation at 22° (slow movements, occasionally falling over); whereas *stn*⁷ is more nearly normal at low temperatures, and can even walk with difficulty at 29°; at permissive temperatures, *stn* (allele unspecified) causes unusual jump response to light-off stimulus, which is more pronounced in combination with *w* (Kelly, 1983b); associated with this abnormal jumping is an increase in amplitude of light-off transient spike of electroretinogram (ERG); in tests of light-adapted mutant, the jump response, as monitored by recordings from indirect flight muscles, habituates with increasing frequencies of light-off stimulation (Kelly, 1983b); combining *stn* with *tan*, which by itself leads to decreased amplitude of ERG on and off transients, leads to loss of anomalous jumping and partial restoration of light-off spike. In biochemical experiments, *stn* (allele unspecified) found to cause accentuation of *in vivo* phosphorylation of a protein from adult head synaptosomal fractions that is modified in this way by a cAMP-dependent protein kinase, with such phosphorylation being enhanced by exposure of flies to light, prior to extraction (Kelly, 1983a). A protein, which has same molecular weight as the one noted above, found to be phosphorylated *in vitro* by a fly-derived Ca²⁺-calmodulin-dependent protein kinase; *stn* causes increased levels of *in vivo* phosphorylation of this material (Kelly, 1983b). The temperature-sensitive lethal allele, *stn*, leads to brief paralysis, followed by sporadic movements, as a result of mechanical shocking (Homyk and Sheppard, 1977, *Genetics* 87: 95-104); other abnormalities include abnormally short jump and flight distances, apparent absence of on and off transients in ERG, and lethality when raised at 29°. Effects of three of the lethal alleles, *stn*¹, *stn*¹¹, and *stn*¹⁴, were examined during development for effects on gross anatomy of CNS or PNS; no obvious abnormalities were observed (Perrimon *et al.*, 1989).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>stn</i> ¹	X ray		<i>l(1)8P1</i>	10, 11, 12	
<i>stn</i> ²	EMS	Lifschytz	<i>l(1)M143</i>	8	on <i>y</i> ⁺ <i>Ymal</i> ⁺
<i>stn</i> ³	EMS	Lifschytz	<i>l(1)R-9-10</i>	7, 12	
<i>stn</i> ⁴	EMS	Lifschytz	<i>l(1)R-9-15</i>	7	
<i>stn</i> ⁵	X ray	Lifschytz	<i>l(1)X3</i>	6, 7, 8, 10, 11	
<i>stn</i> ⁶			<i>stn</i> ^{ts1}	1	temperature-sensitive behavioral mutant
<i>stn</i> ⁷			<i>stn</i> ^{ts2}	1	temperature-sensitive behavioral mutant
<i>stn</i> ⁸	X ray	Lefevre	<i>l(1)C88</i>	4	
<i>stn</i> ⁹	X ray	Lefevre	<i>l(1)HC121</i>	4	
<i>stn</i> ¹⁰	EMS	Lefevre	<i>l(1)VA228</i>	5	
<i>stn</i> ¹¹	EMS	Lefevre	<i>l(1)VE720</i>	5	
<i>stn</i> ¹²	spont	Schalet	<i>l(1)I3-120</i>	9	
<i>stn</i> ¹³	P	Gergen	<i>l(1)30A</i>	13	
<i>stn</i> ¹⁴	P	Gergen	<i>l(1)PH1</i>	13	
<i>stn</i> ¹⁵	EMS	Sheppard	<i>stnC</i>	2, 3	temperature-sensitive lethal allele

^α 1 = Grigliatti, Hall, Rosenbluth, and Suzuki, 1973, *Mol. Gen. Genet.* 120: 107-14; 2 = Homyk and Pye, 1989, *J. Neurogenet.* 5: 37-48; 3 = Homyk and Sheppard, 1977, *Genetics* 87: 95-104; 4 = Lefevre, 1981, *Genetics* 99: 461-80; 5 = Lefevre and Watkins, 1986, *Genetics* 113: 869-95; 6 = Lifschytz and Falk, 1968, *Mut. Res.* 6: 235-44; 7 = Lifschytz and Falk, 1969, *Mut. Res.* 8: 147-55; 8 = Lifschytz and Yakobovitz, 1978, *Mol. Gen. Genet.* 161: 275-84; 9 = Schalet, 1986, *Mutat. Res.* 163: 115-44. 10 = Schalet and Lefevre, 1973, *Chromosoma* 44: 183-90; 11 = Schalet and Lefevre, 1976, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.), Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902; 12 = Schalet and Singer, 1971, *DIS* 46: 131-32; 13 = Zushman, Coulter, and Gergen, 1985, *DIS* 61: 217-18.

cytology: Tentatively placed near bands 20B-C (Miklos *et al.*, 1987); by deficiency mapping placed between *l(1)20Bb* and *l(1)20Ca*. Also maps between right breakpoints of *Df(1)17-466* and *Df(1)17-439*; however, the cytology so close to the chromocenter is intractable and no observed breakpoints are given.

***sto: stocky**

location: 1-29.8.

origin: Induced by triethylenemelamine (CB. 1246).

discoverer: Fahmy, 1953.

references: 1958, *DIS* 32: 75.

phenotype: Fly short and stocky. Wings short but normal in width. Eyes large and pear shaped. Bristles slightly shorter than normal. Male sterile; viability about 50% normal. RK2.

other information: One allele induced by CB. 1528.

***sto^{tpw}: stocky-tapered wings**

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).

discoverer: Fahmy, 1954.

synonym: *tpw*.

references: 1958, *DIS* 32: 76-77.

phenotype: Wings slightly shortened and broadened with tip pointed at L3 rather than being smoothly rounded. Eyes small and oval. Slightly dusky thorax. Both sexes viable; female rather infertile. RK2.

stoned: see *stn*

stonewall: see *snw*

stop: stopped (T. Schüpbach)

location: 2-76.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus.

phenotype: Female sterile; homozygous females often have underdeveloped ovaries that seem to lack germ cells altogether. In some females a small number of developing egg chambers are found that never develop beyond the first few stages of oogenesis.

alleles: Two alleles, *stop*⁷ and *stop*² isolated as *W5* and *W6*.

stormfront: see *stf*

***stp: silver tips**

location: 1-46.1.

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1956.

references: 1959, *DIS* 33: 92.

phenotype: Fly slightly smaller than normal. Bristles thin, weak, and most are unpigmented; hairs unaffected. Male

sterile; viability low. RK3.

Stp: see *Mhc*¹⁵

***Stp-1: Strapped in chromosome 1**

location: 1-50.6 (not allelic with *sd*).

origin: Spontaneous.

discoverer: Hannah.

references: 1950, Genetics 35: 669.

phenotype: Expression limited to male. About 15% of *Stp-1*; *Stp-2*/+ males show some scalloping of wing margins. Most *Stp-1*; *Stp-2*/*Stp-2* males have some degree of scalloping, varying from a small nick to vestigial-like wings. Modified by both genetic and environmental factors. Without *Stp-2*, *Stp-1* has no effect. RK3.

***Stp-2: Strapped in chromosome 2**

location: 2- (right arm between *c* and *sp*).

origin: Spontaneous.

discoverer: Hannah.

references: 1950, Genetics 35: 669.

phenotype: Fifteen percent of *Stp-1*; *Stp-2*/+ and most *Stp-1*; *Stp-2*/*Stp-2* males show incising of wing margin. *Stp-2*/*Stp-2*/+ and *Stp-2*/*Stp-2*/*Stp-2* intersexes show scalloping in the presence or absence of *Stp-1*. RK3.

str: see *tkv*

str(2)350: see *mfs(2)350*

***Str: Stretched wings**

location: 2-67.

discoverer: Tanaka, 34a12.

references: 1937, DIS 8: 11.

phenotype: Wings divergent. Homozygous lethal. RK2.

str-R: see *Rst(1)str*

straight abdomen: see *sab*

stranded-at-second: see *sas*

Strapped: see *Stp*

straw: see *stw*

strawberry: see *fa*^{swb} under *N*

strawberry notch: see *sno*

Streak: see *Sk*

streaked sterna: see *sts*

streakex: see *stx*

stress-sensitive: see *ses*

Stretched wings: see *Str*

string: see *stg*

stripe: see *sr*

sts: streaked sterna

location: 1-60.3.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).

discoverer: Fahmy, 1954.

references: 1959, DIS 33: 92.

phenotype: Small fly with light body color. Brown areas on abdominal sternites often form two longitudinal lines. Eclosion delayed. Viability and fertility low. RK3.

***stt: spotty**

location: 1-34.3.

origin: Induced by *p*-N,N-di-(2-chloroethyl)amino-phenylethylamine (CB. 3034).

discoverer: Fahmy, 1955.

references: 1959, DIS 33: 92.

phenotype: Fly small. Wings slightly deformed. Small dark spots on anterior abdominal segments. In extreme cases, tergites broken and abnormally rejoined, and hairs deranged. Eyes rather small. Male sterile; viability about 50% wild type. RK2.

***stt²**

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1956.

synonym: *spt*: *spotty-tergum*.

references: 1959, DIS 33: 91.

phenotype: Fly small; wings wrinkled or pleated. Darkly pigmented spots dispersed over abdomen, particularly on fourth tergite. Tergites occasionally ridged or broken. Bristles long and straggly. Male sterile; viability about 30% normal. RK2.

other information: Allelism inferred from similarity in phenotype and genetic location at 34.1.

***stu: small tumeroid**

location: 1-20.4.

origin: Induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1954.

references: 1959, DIS 33: 92.

phenotype: Fly small, frequently has small melanotic pseudotumors. Viability 5% wild type. Male fertile. RK3.

stubarista: see *sta*

*stubarista-P*³²: see *crm*

Stubble: see *Sb*

Stubble-recessive: see *sbd*

stubbloid: see *sbd*

Stubby: see *Sy*

Stubby-30: see *Bl*³⁰

Stubby-311 19: see *Bl*³¹¹

stubs: see *sbs*

stuck: see *sk*

stuck up: see *Mhc*¹⁵

Stumpy: see *St*

stw: straw

location: 2-55.1 [0.03 unit to the right of *rl* (Tano, 1966, Japan J. Genet. 41: 299-308); between *rl* and *ap*^{blt} (Sturtevant, 1949, DIS 23: 98)].

phenotype: Hair color yellowish, especially on legs. Bristles pale at tips. Heterozygous deficiency for *stw* produces paling of body color. RK2.

alleles:

allele	origin	discoverer	synonym	ref ^α
<i>stw</i> ¹	spont	Bridges, 17f11		2
<i>stw</i> ²	spont	Bridges, 21g	<i>swy</i>	2
<i>stw</i> ³	X ray	Serebrovsky, 1930		
* <i>stw</i> ⁴		Mather, 37k30		
<i>stw</i> ⁵	UV	Meyer, 51d		1
<i>stw</i> ⁶	UV	Meyer, 51e		1
* <i>stw</i> ⁷	UV	Meyer, 51f		1
* <i>stw</i> ^D	spont	Kiil, 38k28		3

^α 1 = Meyer and Edmondson, 1951, DIS 25: 73; 2 = Morgan, Bridges, and Sturtevant, 1925, Bibliogr. Genet. 2: 237; 3 = Mossige, 1939, DIS 12: 47.

cytology: Placed in 41B or C on the basis of the pale body color of heterozygotes for the deficiency from 41B3 through 42A2 formed by combining left end of *In(2R)Cy* = *In(2R)42A2-3;58A4-B1* with right end of *In(2R)bw^{VDel}* = *In(2R)41B2-C1;59E2-4* and inclusion of *stw* in several cytologically invisible deficiencies at base of 2R, e.g., *Df(2R)M41A* (Schultz).

***stw*²**

phenotype: Hairs pale yellow; bristles brownish with yellow tips. Wings pale yellow and somewhat thin and warped. Slightly more extreme than *stw*. Larval mouth parts straw colored at basal prongs and classifiable with difficulty in third-instar larvae (Brehme, 1941, Proc. Nat. Acad. Sci. USA 27: 254-61). RK2.

***stw*³**

phenotype: Hairs, bristles, wings, and wing veins straw yellow. Body yellowish with pronounced dark trident. Tyrosinase formed in adult (Horowitz). Wings thin and buckled. Hairs on wing cells incompletely chitinized (Waddington, 1941, Proc. Zool. Soc., Ser. A 111: 173-80). Puparium noticeably lighter than wild type. Larval mouth parts straw colored at basal prongs; classifiable in living larva (Brehme, 1941, Proc. Nat. Acad. Sci. USA 27: 254-61). RK2.

other information: Waddington found that irradiation of *stw*³ homozygote 2 days before eclosion produces reverse mutations that appear as single wild-type wing hairs (1940, Nature 146: 335).

****stw*⁴**

phenotype: Body pale yellow. Legs almost colorless. Wings colorless, thin, and fragile. Black areas of abdomen still black but heavily sprinkled with pale spots. Larval mouth parts normal (Brehme, 1941, Proc. Nat. Acad. Sci. USA 27: 254-61). RK2.

***stw*⁵**

phenotype: Activation of the phenoloxidase at the time of melanization reduced in *stw*⁵ homozygotes compared to wild type (Mitchell, 1966, J. Insect. Physiol. 12: 755-65). Electroretinogram normal (Hotta and Benzer, 1969, Nature 222: 354-56). Semilethal or associated with a closely linked semilethal. RK2.

***stw*⁶**

phenotype: Like *stw*. Viability low. RK2.

****stw*⁷**

phenotype: Bristles yellowish. Wing color pale, often overlaps wild type. Eclosion delayed. Poor viability. RK2.

****stw*^D: straw-Dominant**

phenotype: Body and bristles of homozygote light yellow; wings thin, buckled, and curled. In heterozygote, wings less abnormal; body and bristles wild type. *stw*^D/*stw*³ like *stw*³. *stw*^D/*Df(2R)M41A* has exaggerated *stw* phenotype. RK1.

****stx*: streakex**

location: 1- (rearrangement).

origin: X ray induced.

discoverer: Muller, 26k30.

references: 1935, DIS 3: 30.

phenotype: Dark streak down dorsal midline of thorax. Semilethal. RK3A.

cytology: Associated with *In(1)stx*; in the left end but breakpoints unknown.

*su*¹⁸: see *qs*²

su-: see *su*()

Su-: see *Su*()

Su-Pm: see *Su(bw^{V1})*

su^{s-pr}: see *In(3R)su(pr)*

Su-x4: see *Su(sph)*

****su*(b): suppressor of black**

location: 1-0.1.

origin: Spontaneous.

discoverer: Plough, 23j28.

references: 1927, Proc. Intern. Congr. Genet., 5th., Vol. 2: 1193-1200.

Sherald, 1981, Mol. Gen. Genet. 183: 102-06.

phenotype: Suppresses *b* so that body color is only slightly darker than wild type. No dominant effect. Causes the level of β-alanine, which is ordinarily reduced, to return to normal in *b/b* flies; *su*(b)⁶ in *b*⁺ genotypes leads to 60% increase above normal β-alanine levels. *su*(b)⁶ flies are unable to survive on 0.2 M β-alanine; apparently β-alanine ingested by females prevents *su*(b) embryos from hatching or the unhatched embryos from melanizing (Campbell and Sherald, 1987, DIS 66: 34). Also effects a return to normal of the fine structure of the puparial case in *su*(b)⁶; *b/b* individuals.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>su</i> (b) ¹	spont	Plough, 23j28		1	frequently reverts fertility reduced
<i>su</i> (b) ²	EMS	Sherald	<i>su</i> (b) ¹¹	2	complements <i>su</i> (b) ⁴
<i>su</i> (b) ³	EMS	Sherald	<i>su</i> (b) ¹²	2	reisolate of <i>su</i> (b) ² ?
<i>su</i> (b) ⁴	EMS	Sherald	<i>su</i> (b) ¹³	2	complements <i>su</i> (b) ²
<i>su</i> (b) ⁵	EMS	Sherald	<i>su</i> (b) ¹⁴	2	reisolate of <i>su</i> (b) ² ?
<i>su</i> (b) ⁶	EMS	Sherald	<i>su</i> (b) ³¹	2	non complementing
<i>su</i> (b) ⁷	EMS	Kolbak	<i>su</i> (b) ^{DK} ?	3	
<i>su</i> (b) ⁸	EMS	Kolbak		3	
<i>su</i> (b) ⁹	EMS	Kolbak		3	
<i>su</i> (b) ¹⁰	EMS	Kolbak		3	
<i>su</i> (b) ¹¹	EMS	Kolbak		3	
<i>su</i> (b) ¹²	EMS	Kolbak		3	

^α 1 = Plough, 1927, Proc. Intern. Congr. Genet., 5th., Vol. 2: 1193-1200; 2 = Sherald; 1981, Mol. Gen. Genet. 183: 102-06; 3 = Sϕndergaard and Kolbak, 1987, DIS 66: 134.

cytology: Placed in 1B4-9 based on its being covered by the terminal deficiency, *Df(1)260-1 = Df(1)IA4-6* but not by *Df(1)yT4-12 = Df(1)IA7-9* (Sherald and Voelker,

1985, DIS 61: 155).

***Su(b)*: Suppressor of black**

location: 1-55.5 [$1-2 \times 10^{-4}$ units distal to the distal end of the *r* locus; possibly a regulatory sequence (Søndergaard and Kolbak, 1987, DIS 66: 134)].

references: Pedersen, 1982, Carlsberg Res. Commun. 47: 391-400.

phenotype: A semidominant suppressor of *b*; homozygotes and hemizygotes in combination with *b* are slightly darker than wild type, whereas heterozygotes are intermediate between *b* and wild type in cuticle reflectance. *Su(b)* by itself has no visible phenotype. Although supplementation of the medium with 6-azathymine, an inhibitor of dihydrouracil dehydrogenase, results in a dark body color in wild-type flies, it does not darken the wild-type coloration of *Su(b)*; *b*. The double-mutant combination, *Su(b) r*, does not suppress *b* (Bahn and Søndergaard, 1983, Hereditas 99: 309-10), whereas *su(r) Su(b)*; *b* flies have enhanced black cuticle. Unlike *su(b)*; *b*, the combination *Su(b)*; *b* is not sensitive to dietary β -alanine (Campbell and Sberald, 1987, DIS 66: 34).

cytology: Placed in 15A1 based on its virtual inseparability from *r*.

****su(B)*: suppressor of Bar**

location: 2-94.

origin: Spontaneous.

discoverer: Steinberg, 361.

synonym: *m(B)*: modifier of Bar.

references: 1937, DIS 7: 20.

1937, DIS 8: 11.

1939, DIS 12: 49.

1940, Collecting Net 15: 173.

1941, Genetics 26: 325-46, 440-51.

phenotype: When homozygous, increases number of eye facets from about 75 to 220 in *B* male and to 140 in *B/B* female. Affects all *B* effects but not *ey*² or wild type. RK2.

****su(B)2*: suppressor of Bar in chromosome 2**

location: 2-46 or -60 (7 units from *Tft*).

origin: Spontaneous.

discoverer: Gans.

phenotype: *su(B)2/su(B)2* causes *B/+* female to appear wild type. RK2.

****su(B)4*: suppressor of Bar in chromosome 4**

location: 4- (not located).

origin: Spontaneous.

discoverer: Brehme, 39k.

synonym: *m(B)4*: modifier of Bar in chromosome 4.

references: 1942, DIS 16: 47.

phenotype: Facet number in eyes of *B* male increased, approaching that of *B/+* female. Effect increases with age of culture. *B/B* and *B/+* female not affected. RK3.

***Su(bw^{V1})*: Suppressor of brown-Variegated**

location: 2-105.2.

origin: Spontaneous.

discoverer: Kadel, 59b17.

synonym: *Su-Pm*: Suppressor of Plum.

references: 1959, DIS 33: 95.

phenotype: *Su(bw^{V1})/bw^{V1}* has wild-type eye color with peppering of dark spots instead of the more or less uniform brown of *bw^{V1}/+*. Effect on various *bw^V* chromo-

somes varies from none for some to complete suppression for others. Pteridines of heads of *bw^D/bw^{V1}* heterozygotes increased to 28% of normal from 2.5% for *bw^D/+* heterozygotes (Henikoff and Dreesen). Homozygous viable. RK2.

cytology: No gross aberration (Lindsley). *In situ* hybridization using *bw* cDNA probe shows a tandem duplication of the sequence, possibly including 59E2-F1 (Dreesen and Henikoff).

other information: *Su(bw^{V1})* is a tandem duplication of *bw⁺* and flanking sequences based on wild-type phenotype of *bw/bw Su(bw^{V1})* and *bw^S/bw^S Su(bw^{V1})* (Kadel and Wright). Homozygous *Su(bw^{V1})* female produces 0.3% reversions associated with crossing over in a manner analogous to reversions of *B*.

***su(Cbx)*: suppressor of Contrabithorax**

location: 1-30.

origin: Spontaneous.

discoverer: E. B. Lewis.

references: 1955, Am. Naturalist 89: 73-89.

phenotype: Almost completely suppressed *Cbx*; wings made virtually normal and segmental transformations strongly reduced. Without effect on the incidence of ether-induced *bx* phenocopies (Capdevila and García-Bellido, 1981, Wilhelm Roux's Arch. Dev. Biol. 190: 339-50). RK2.

cytology: Placed in 7F1-8C6 based on its inclusion in *Df(1)KA14* (Lefevre).

***Su(crc)*: Suppressor of cryptocephal (J.C. Sparrow)**

location: 2-54.7.

origin: Induced by ethyl methanesulfonate in *l(2)crc cn* chromosome.

discoverer: Sparrow, 1977.

references: Sparrow, J.C., 1981, Genet. Res. 38: 297-314.

phenotype: A dominant suppressor of *l(2)crc* lethality. Expression is temperature sensitive in *Su(crc) l(2)crc/l(2)crc* heterozygotes and *Su(crc) l(2)crc* hemizygotes with a temperature-sensitive period beginning before pupariation and ending at the time of pupation. The effective lethal period for the suppressor genotypes at 30°C is at the end of the pupal period, not during prepupal stages as is the case for *l(2)crc* homozygotes.

cytology: Located between 38A7-B1 and 39C2-D1 but separable by deficiency mapping from *l(2)crc* which has been localized to 39C2-D1 and therefore *Su(crc)* is not a revertant of *l(2)crc*.

su(Cy)

location: 3- not mapped.

origin: Natural population.

phenotype: Homozygotes without discernable phenotype except in the presence of *Cy*, in which case the curly-wing phenotype is suppressed.

alleles: Two independent isolations of chromosomes with this effect (Meyer and Temin, 1965, DIS 40: 62 and Kosuda, 1971, Jpn. J. Genet. 46: 41-52).

***Su(Cy)*: Suppressor of Curly**

location: 2- (not located).

origin: Spontaneous in *In(2LR)bw^{V1}*.

discoverer: Thompson, 61e.

references: 1963, DIS 38: 28.

phenotype: *Su(Cy)/Cy* has wild-type wings. RK3.

other information: Separable from *In(2LR)bw^{V1}*.

Su(da): see *Sxl^{MI}*

****su(dx)*: suppressor of *deltex***

location: 1-5.

discoverer: Bridges, 35c26.

synonym: *su^{X-dx}*: suppressor in X chromosome of *deltex*.

phenotype: Reduces phenotype of and imparts male fertility to *dxst*. RK2.

***su(dp^{ov})*: suppressor of *dumpy-oblique vortex* (T. Kjaer)**

location: 1- (proximal half of chromosome).

references: Kjaer, 1986, DIS 63: 162.

phenotype: Suppresses phenotype of *dp^{ov1}* to normal or nearly normal; also suppresses *dp^{ov-DG37}* and *dp^{cm2}* but does not suppress *dp^{vz2}* or *dp^{olv}*. Viability and fertility good.

alleles: Three non-complementing, ethyl-methane-sulfonate-induced alleles.

allele	synonym	comments
<i>su(dp^{ov})¹</i>	<i>su(dp^{ov})^{TK8-84}</i>	suppresses oblique and vortex
<i>su(dp^{ov})²</i>	<i>su(dp^{ov})^{TK25-84}</i>	suppresses oblique only
<i>su(dp^{ov})³</i>	<i>su(dp^{ov})^{TK26-84}</i>	suppresses oblique only

****Su(dx)*: Suppressor of *deltex***

location: 2- (not located).

origin: Spontaneous.

discoverer: Bridges, 31a3.

references: Morgan, Bridges, and Schultz, 1931, Year Book - Carnegie Inst. Washington 30: 410.

phenotype: *Su(dx)*+ reduces *dxst* to a slight but recognizable, fully fertile phenotype. *Su(dx)/Su(dx)* converts *dxst* to nearly wild type. RK3.

Su(dx)²

origin: Spontaneous.

discoverer: Bridges, 31f1.

references: Morgan, Bridges, and Schultz, 1931, Year Book - Carnegie Inst. Washington 30: 410.

phenotype: Less effective than *Su(dx)* as a suppressor of *dx*. RK3.

other information: Found in *dx* stock, as was *Su(dx)*, along with *ed*. *Su(dx)²* may simply be *ed Su(dx)* or it may be of independent origin. Allelism inferred from phenotype alone.

Su(en)28: see *Dp(2;3)Su-en*

Su(er)*: Suppressor of *erupt

location: 2- (near *cn*).

origin: Present in many stocks.

discoverer: Glass, 1941.

references: 1944, Genetics 29: 436-46. 1957, Science 126: 683-89.

phenotype: Only effect is suppression of *er*. Semidominant. Exposure to 1000 r of X rays from shortly after

fertilization [8 min, according to Glass (1957), but not until 6 hr, according to Hildreth, 1968, Proc. Nat. Acad. Sci. USA 58: 1924-29] to middle of second larval instar inhibits *Su(er)*, and *er* is then manifested in about 98% of flies. Tryptophan fed to larvae has similar effect. Some related compounds have a lesser effect; kynurenine and indole acetic acid have little or no effect. RK3.

su(f)*: suppressor of *forked

location: 1-65.9 (adjacent to and to the left of *bb*).

references: Schalet and Lefevre, 1974, Chromosoma 44: 183-202.

Schalet and Lefevre, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902.

phenotype: *su(f)* mutations are found with several levels of reduced expression, and some if not all mutant alleles are sensitive to temperature. The weakest exhibit no phenotype when raised at 25° other than modification of the expression of specific alleles at other loci; increasingly reduced expression leads to a specific phenotype, which variably includes Minute-like bristles, rough eyes with some anterior indentation, reduced or absent ocelli, missing ocellar and other head and thoracic bristles, irregular acrostichal rows, excessive melanization, especially on head, and some crippling of legs; also wings may be blistered, broader, with extra veins and may be held upward and outward (Grell, CP627; Schalet, 1968, DIS 44: 125). The most severely affected alleles are lethal, the lethal period becoming earlier with increasing severity. At least one instance of interallelic complementation has been reported by an allele which in surviving adults exhibits pale yellow thread-like chaetae. Viable alleles act as allele-specific but locus-nonspecific modifiers. The locus was recognized by the nearly wild-type bristle phenotype of *f su(f)*; some bristles slightly shortened or twisted at tips. Autonomous in gynandromorphs. *f* alleles fall into two classes: suppressible (*f¹*, *f⁴*, and *f⁵*) and insuppressible (*f³* and *f^{3N}*) (Green, 1959, Heredity 13: 303-15). Suppressible alleles are spontaneous and contain insertions of transposable sequences; such alleles are also frequently modified by mutations in other modifier genes. For example, among alleles with gypsy inserts, *su(f)* suppresses *ct^k*, *lz¹*, *f¹*, *f⁵*, *bx^{34e}*, but not *y²*, *Hw¹*, *sc¹*, or *ct⁶*; all are suppressed by *su(Hw)*. In addition, *su(f)* enhances the spontaneous mutants *w^a* (*copia*) and *lz³⁷*, but not *w^e*, *lz³⁴*, *lz^k*, *s¹*, or *v¹* (412); all of these are affected by one or more other suppressor genotypes (Rutledge, Mortin, Schwarz, Thierry-Mieg, and Meselson, 1988, Genetics 119: 391-97). Does not suppress the effect of *f* on the phenotype of *dvr*, i.e. crumpled wings (Lee, 1974, Aust. J. Biol. Sci. 27: 305-7).

alleles: Allele specific features are detailed at the end of the entry.

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>su(f)</i> ¹	X ray	Whittinghill, 37g4	<i>su</i> ^W (<i>f</i>)	22, 25, 30, 31, 32	viable allele; restriction map normal
<i>su(f)</i> ²	DES	Lifschytz	<i>l(1)3DES</i>	13, 22, 25, 29	~500 bp insert in -3.5 to -3.3
<i>su(f)</i> ³	DES	Lifschytz	<i>l(1)D13</i>	13, 16, 22	restriction map normal
<i>su(f)</i> ⁴	EMS	Lifschytz	<i>l(1)R-9-18</i> <i>su(f)</i> ^{pb}	13, 16, 17, 23, 25, 26	pale-bristle allele; <i>ts</i> lethal; restriction map normal
<i>su(f)</i> ⁵	X ray	Schalet	<i>su(f)</i> ^{x1}		~150 bp deletion at +2.2 to +4.3
<i>su(f)</i> ⁶	X ray	Lifschytz	<i>l(1)X2</i>	12, 16, 22, 23	-1 kb deletion at -0.3 to +1.3
<i>su(f)</i> ⁷	X ray	Schalet	<i>su(f)</i> ^{x3}		restriction map normal
<i>su(f)</i> ⁸	EMS	Wright	<i>su(f)</i> ^{ts678}	2, 3, 10, 27	<i>ts</i> lethal; restriction map normal
<i>su(f)</i> ⁹	EMS	Voss	<i>su(f)</i> ^v	28, 29	viable derivative of <i>su(f)</i> ²
<i>su(f)</i> ¹⁰	EMS	Lifschytz	<i>l(1)M171</i>	15	on y ⁺ <i>Ymal</i> ⁺
<i>su(f)</i> ¹¹	EMS	Lifschytz	<i>l(1)M168</i>	15	on y ⁺ <i>Ymal</i> ⁺ ; pale-bristle allele?
<i>su(f)</i> ¹²	EMS	Russell	<i>l(1)ts726</i>	1, 4, 6, 7, 18, 19, 20	<i>ts</i> lethal
<i>su(f)</i> ¹³	EMS	Jürgens	<i>l(1)maq^{ts}</i>	8, 9, 10	<i>ts</i> lethal; restriction map normal
<i>su(f)</i> ¹⁴	EMS	Wilson	<i>su(f)</i> ^{ts76a}	33	<i>ts</i> lethal; restriction map normal
<i>su(f)</i> ¹⁵	X ray	Lefevre	<i>l(1)GA46</i>	14	
<i>su(f)</i> ¹⁶	X ray	Lefevre	<i>l(1)GA130</i>	14	
<i>su(f)</i> ¹⁷	X ray	Lefevre	<i>l(1)HA16</i>	14	
<i>su(f)</i> ¹⁸	X ray	Lefevre	<i>l(1)HC148</i>	14	
<i>su(f)</i> ¹⁹	X ray	Lefevre	<i>l(1)L26</i>	14	8 kb deletion in -3.4 to +6.4
<i>su(f)</i> ²⁰	X ray	Lefevre	<i>l(1)N125</i>	14	
<i>su(f)</i> ²²	EMS	Lefevre	<i>l(1)VE738</i>	11, 14	
<i>su(f)</i> ²³	P		<i>su(f)</i> ^{hd37}	5	viable allele
<i>su(f)</i> ²⁴	EMS	Rutledge	<i>su(f)</i> ^{br}	21	viable allele
<i>su(f)</i> ²⁵	spont	Schalet	<i>su(f)</i> ^{S1}		~200 bp insert in 0 to +2.1
<i>su(f)</i> ²⁶	spont	Schalet	<i>su(f)</i> ^{S2}	16, 17, 24	4.7 kb <i>Doc</i> element insert at +3.1
<i>su(f)</i> ²⁷	P	Simmons	<i>l(1)19-158</i> <i>su(f)</i> ^{MS97}	5	1.1 kb <i>P</i> element insert at +0.2
<i>su(f)</i> ²⁸	P	Simmons	<i>su(f)</i> ^{MS252}	5	1.1 kb <i>P</i> element insert at +0.1

^α 1 = Clark and Russell, 1977, Dev. Biol. 57: 160-73; 2 = Dudick, Wright, and Brothers, 1974, Genetics 76: 487-510; 3 = Fekete and Lambertsson, 1980, Hereditas 93: 169-76; 4 = Girton, 1981, Dev. Biol. 84: 164-72; 5 = Girton, Langner, and Cejka, 1986, Roux's Arch. Dev. Biol. 195: 334-37; 6 = Girton and Russell, 1980, Dev. Biol. 77: 1-21; 7 = Girton and Russell, 1981, Dev. Biol. 85: 55-64; 8 = Jürgens and Gateff, 1979, Wilhelm Roux's Arch. Dev. Biol. 186: 1-27; 9 = Klose, Gateff, Emmerich, and Beikirch, 1980, Wilhelm Roux's Arch. Dev. Biol. 189: 57-67; 10 = Lambertson, 1975, Mol. Gen. Genet. 139: 145-56; 11 = Lefevre, 1981, Genetics 99: 461-80; 12 = Lifschytz and Falk, 1968, Mut. Res. 6: 235-44; 13 = Lifschytz and Falk, 1969, Mut. Res. 8: 147-55; 14 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 15 = Lifschytz and Yakobovitz, 1978, Mol. Gen. Genet. 161: 275-84; 16 = Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31; 17 = Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-521; 18 = Postlethwait, 1978, Wilhelm Roux's Arch. Dev. Biol. 185: 37-57; 19 = Russell, 1974, Dev. Biol. 40: 24-39; 20 = Russell, Girton, and Morgan, 1977, Wilhelm Roux's Arch. Dev. Biol. 183: 41-59; 21 = Rutledge, Mortin, Schwarz, Thierry-Mieg, and Meselson, 1988, Genetics 119: 391-97; 22 = Schalet, 1968, DIS 43: 125; 23 = Schalet, 1972, DIS 49: 37, 64; 24 = Schalet, 1986, Mut. Res. 163: 115-144; 25 = Schalet and Lefevre, 1973, Chromosoma 44: 183-202; 26 = Schalet and Lefevre, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902; 27 = Suzuki, Kaufman, and Falk, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 207-63; 28 = Voss, 1971, DIS 46: 55; 29 = Voss and Falk, 1973, Mutat. Res. 20: 221-34; 30 = Whittinghill, 1937, DIS 8: 11, 13; 31 = Whittinghill, 1938, Genetics 23: 305; 32 = Whittinghill, 1942, DIS 16: 70; 33 = Wilson, 1980, J. Embryol. Exp. Morph. 55: 243.

^β Coordinates are from O'Hare; the origin is at a *saII* site close to the insertion of the *P* element used to clone the gene. Positive values are toward the centromere.

cytology: Placed in 20E-F by Lefevre (Schalet and Lefevre, 1973, Chromosoma 44: 183-202); polytene chromosomes in this region are refractory to analysis; however, it is generally accepted that *su(f)* is the most proximally located euchromatic gene on the *X* chromosome.

molecular biology: Region cloned by O'Hare by means of transposon tagging using the *P*-element insert in *su(f)*²⁸. 50 kb (-32 kb to +19 kb) restriction mapped; coordinate 0 designated as *saII* site 1.1 kb to the left of the *P* insert; positive values are to the right, toward the centromere. The region comprises single-copy DNA interspersed with repeated sequences. Repeated DNA found from -32.0 to -29.4, from -23.4 to -19.9, from -10.2 to -7.8, from -7.4 to -6.6, from -5.0 to -2.0, and from +4.9 to +19.0. Three major RNAs identified by RNA blotting and cDNA analysis; they are transcribed from the interval -0.3 to +3.7; their sizes are 1.3 kb comprising 3 exons, and 2.6 kb and 2.9 kb, both containing the same eight exons, including the three from the smaller RNA; they predict two proteins. DNA lesions detected so far in *su(f)* mutations confined to the region from -5.0 to +5.0 kb (O'Hare). Transformation with -2.2 to +4.3 kb rescues lethality of *su(f)*², *su(f)*⁵, *su(f)*⁶, *su(f)*¹⁹, and *su(f)*²⁶ at

25°; also covers suppression of forked-bristle phenotype in *f su(f)*¹ (Simonelig and O'Hare).

*su(f)*¹

phenotype: Wild type in appearance with normal viability and fertility when raised at 25°; however, both *su(f)* flies raised at 29-30° and *su(f)/Df(1)su(f)* flies raised at 25° display the Minute-like syndrome. The same phenotype is seen in combinations with *su(f)*² and *su(f)*⁶ raised at 29°. *su(f)/Df(1)su(f)* is lethal when raised at 29°; viability also temperature sensitive in combination with *su(f)*³ and *su(f)*⁵ (Schalet and Lefevre, 1976). *su(f)* enhances *w^a*; *w^a su(f)* eyes nearly white at 25° and white at 18° but apricot in flies raised at 29°; suppresses *lz*¹ and enhances *lz*³⁷ at all temperatures. Increases the accumulation of *gypsy* transcript, suggesting that the wild-type allele represses *gypsy* (Parkhurst and Corces, 1986, Mol. Cell. Biol. 6: 2271-74); protein binding to a negative regulatory sequence of *gypsy* is reduced in nuclear extracts from *su(f)* pupae compared with those from wild type, again suggesting *gypsy* repression by *su(f)*⁺ (Mazo, Mizrokhi, Karavanov, Sedkov, Krichevskaha, and Ilyin, 1989, EMBO J. 8: 903-11).

***su(f)*²**

phenotype: Homozygotes lethal at the larval stage; homozygous germ-line clones don't survive; no effect on development of peripheral or central nervous systems (Perrimon, Smouse, and Miklos, 1989, *Genetics* 121: 313-31). Survives and suppresses *f* in combination with *su(f)*¹ at 25°, and exhibits *M*-like syndrome in flies raised at 18°. *w^a* displays dilute apricot pigmentation in *su(f)*¹/*su(f)*² flies raised at 25° but white eyes when raised at 18° (Schalet and Lefevre, 1976). *su(f)*² shown to be proximal to *su(f)*¹ by recombination (Schalet).

***su(f)*³**

references: Schalet, 1972, *DIS* 49: 36-37.

phenotype: Homozygous lethal. Fails to complement lethality of either *su(f)*³ or *su(f)*⁵. In combination with *su(f)*¹ shows the *M*-like syndrome at 25° and is lethal at 29°.

***su(f)*⁴**

references: Schalet, 1972, *DIS* 49: 36-37.

phenotype: May be the only pale-bristle allele. One other allele of this type was found by Dale Grace as a sex-lined lethal (Schalet). Recovered originally as a sex-linked recessive lethal, but fully viable, though weak, and suppresses *f* when raised at 18°. Survivors exhibit pale-yellow, thread-like bristles, darker pigmentation dorsoanteriorly on thorax, and curled or wrinkled wings. Lethality at late pupal stage; homozygous germ-line clones show no maternal effect; also, no zygotic effects on development of peripheral or central nervous systems (Perrimon, Smouse, and Miklos, 1989, *Genetics* 121: 313-31). Interactions of *su(f)*⁴ with other *su(f)* alleles are as follows: *su(f)*¹, same as *su(f)*¹ homozygotes at all temperatures; *su(f)*², complementation for viability and bristle color and slight complementation for suppression of *f*; *su(f)*³, lethal at the time of puparium formation at 25°, fully viable with some chaetae of all individuals showing the pale-bristle phenotype and the texture of the wings appearing abnormal at 18°; *su(f)*⁵, fully viable and normal at 18°, variable viability with a broad streak of dark pigment on thorax, which sometimes is concentrated at dorsal anterior region, and wings which may extend upward and outward at 25°, lethal prior to puparium formation at 29°; *su(f)*⁶, fully viable and normal at 18° and 25° but exhibiting the *M*-like syndrome at 29°; *Df(1)su(f)*, lethal at the time of puparium formation at 25° and just prior to eclosion at 18°. Enhances *w^a*; *w^a su(f)* eyes white in flies raised at 25° and dilute apricot at 18°; also enhances *lz*³⁷ at 18°.

***su(f)*⁶**

phenotype: Homozygotes lethal at the larval stage; homozygous germ-line clones don't survive; no effect on development of peripheral or central nervous systems (Perrimon, Smouse, and Miklos, 1989, *Genetics* 121: 313-31). Lethal with *su(f)*³ at 25° and with *su(f)*⁴ at 29°. In combination with *su(f)*¹, displays the *M*-like syndrome at 25° and lethal at 29°.

***su(f)*⁸**

references: Dudick, Wright, and Brothers, 1974, *Genetics* 76: 487-510.

phenotype: The first allele recovered specifically as a temperature-sensitive lethal; completely lethal at 29°; suppresses *f* at 25° but not at 18°. Temperature-sensitive

period for lethality from 50 to 140 h after oviposition, for *f* suppression coincident with bristle differentiation. Shift up to 30° before end of the second instar causes failure to pupariate; full-sized third instar larvae produced, which live 10-14 days; salivary-gland-secretion proteins specifically reduced or absent in these larvae, although the associated chromosome puffs appear normally; other proteins unaffected (Hansson, Linderuth, and Lambertsson, 1982, *Wilhelm Roux's Arch. Dev. Biol.* 190: 308-12); shift up prior to 70 h leads to little or no accumulation of *Sgs* transcripts as detected in Northern blots probed with sequences from *Sgs*³, *Sgs*⁴, *Sgs*⁷, and *Sgs*⁸, whereas a 48-h pulse beginning at 75 h is without effect on transcription or translation of *Sgs* genes (Hansson and Lambertsson, 1983, *Mol. Gen. Genet.* 192: 395-40). Shift up to 30° in early third instar blocks the increase in ecdysterone titer normally occurring at the end of L3; ecdysterone supplementation induces abortive pupariation and stimulates prepupal polypeptide synthesis (Hansson and Lambertsson, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 48-51); leg discs of such larvae unable to evert, either *in vivo* or *in vitro* (Fekete and Lambertsson, 1980, *Hereditas* 93: 169-76). Homozygous females raised under permissive conditions, when shifted up to 30° cease laying eggs and the ovarian oocytes degenerate; fertility recoverable after pulses of three but not eleven hours (Dudick *et al.*). Heterozygotes with *Df(1)su(f)* at 25°, *su(f)*¹ at 29°, and *su(f)*³ at 30° exhibit the *M*-like syndrome. Enhances *M(3)67C*, as indicated by reduced viability of *su(f)*⁸ versus *su(f)*⁺ sibs that are *M(3)67C/+* (Girton, Langner, and Cejka, 1986, *Roux's Arch. Dev. Biol.* 195: 334-37). Enhances *gypsy* expression, more at 25° than at 18° (Parkhurst and Corces, 1986, *Mol. Cell. Biol.* 6: 2271-74).

***su(f)*⁹**

references: Voss and Falk, 1973, *Mutat. Res.* 20: 221-34.

phenotype: Recovered as a surviving son of *su(f)*³; still suppresses *f*, but is no longer lethal. *X/Y* males survive and have small rough eyes, broad outstretched wings and irregular abdominal pigmentation; homozygous females usually lethal with escapers showing the same phenotype as the males. *XXY* females survive and resemble *XY* males. *XO* males die. *su(f)*⁹/*su(f)*³ never survives. Attributed by authors to a recessive variegated-position-effect suppressor of the lethality of *su(f)*³; locates to proximal extremity of the *X*; complements *bb*.

cytology: Salivary chromosomes normal; pseudolinkage not tested.

***su(f)*¹²**

references: Russell, 1974, *Dev. Biol.* 40: 24-39.

phenotype: Selected as a cell-autonomous, temperature-sensitive lethal. Relative survival is 85% at 22°, 75% at 25° and 1% at 29°; temperature-sensitive period from first larval instar to early pupa. Homozygous females become sterile after two days at 29°, whereas males so treated remain fertile. 23% of eggs laid at 29° fail to hatch; surviving larvae grow at subnormal rate and survive for up to twelve days, reaching the third instar; imaginal discs reduced greatly in size. 48-h pulses of 30° during late second and third instars results in considerable cell death in imaginal discs with a consequent deletion of some pattern elements and duplications of others in the head and legs; head duplications occur only in

association with deficiencies; leg duplications seen as simply and complexly branched appendages involving variable numbers of joints. Pulses applied to early pupae lead to failure of histoblast differentiation and applied later to the lack of chaetae (Russell, Girton, and Morgan, 1977, Wilhelm Roux's Arch. Dev. Biol. 183: 41-59). Extensive use made of leg duplications induced in *su(f)*¹² in investigations of pattern formation (Tiong, Girton, Hayes, and Russell, 1977, Nature 268: 435-37; Postlethwait, 1978, Wilhelm Roux's Arch. Dev. Biol. 185: 37-57; Girton and Russell, 1980, Dev. Biol. 77: 1-21; Girton, 1981, Dev. Biol. 84: 164-72; Girton and Russell, 1981, Dev. Biol. 85: 55-64). Enhances *M(3)67C*, as indicated by reduced viability of *su(f)*¹² versus *su(f)*⁺ sibs that are *M(3)67C/+* (Girton, Langner, and Cejka, 1986, Roux's Arch. Dev. Biol. 195: 334-37).

*su(f)*¹³

references: Jürgens and Gateff, 1979, Wilhelm Roux's Arch. Dev. Biol. 186: 1-25.

phenotype: A cell-autonomous, temperature-sensitive recessive allele. Phenotype similar to that of *su(f)*¹² except that trypan-blue staining provides no evidence of cell death resulting from heat shock at stages of development in which such treatment induces leg duplications. Authors postulate that *su(f)*¹² discs developmentally impaired by heat shock, giving rise to observed abnormalities. Temperature-sensitive period from late second instar until two hours into pupariation. Shifts from 22° to 29° during the first larval instar leads to inability to pupariate; shifts between 112 and 164 h arrests adult development; shifts within the first six hours after the temperature for lethality removes bristles from the tergites. Gynandromorphs and somatic clones formed normally at permissive temperatures, but are not observed in adults produced at 29°. Ecdysteroid level of larvae raised at 29° are less than one-tenth that of wild type; pupariation can be induced by ecdysterone supplementation (Klose, Gateff, Emmerich, and Beikirch, 1980, Wilhelm Roux's Arch. Dev. Biol. 189: 57-67).

*su(f)*¹⁴

references: Wilson, 1980, J. Embryol. Exp. Morphol. 55: 247-56.

phenotype: Temperature-sensitive lethal allele. Temperature-sensitive period for lethality extends from the second larval instar until twelve hours after pupariation. Shifting adult females to restrictive conditions results in the gradual abolition of oviposition; during the first day normal appearing eggs, many of which hatch, are produced, on the second day the hatchability of the eggs is reduced, and by day four the eggs are small and misshapen and lack chorions; oviposition ceases on the fifth or sixth day after shift up; at this time the ovary is deficient in stage 8-11 oocytes and lacks follicle cells indicating a breakdown in vitellogenesis. Shift back down to 25° leads to resumption of egg laying after four days. Ovarian response is autonomous in ovarian transplants. Fertility of males irreversibly impaired by shift up to 29°.

*su(f)*²³

references: Girton, Langner, and Cejka, 1986, Roux's Arch. Dev. Biol. 195: 334-37.

phenotype: Homozygotes and hemizygotes survive at all

temperatures from 18° to 29°; complements the lethality of lethal alleles. The suppression of *f* is temperature sensitive, but the sensitivity is of opposite sign from that of other temperature-sensitive alleles; *f su(f)*²³ flies raised at 18° have suppressed forked bristles, whereas those raised at 29° are forked; the temperature-sensitive period sharply confined to the short interval at which bristle development is initiated. Does not appear to enhance *M(3)67C*.

*su(f)*²⁴

phenotype: Similar to *su(f)*¹; enhancement of *w^a*, however, seen only in *su(f)*¹/*su(f)*²⁴. Closely linked to or inseparable from a variable recessive abnormality giving small misshapen eyes.

**Su(f)*: Suppressor of forked

location: 2-74.

origin: X ray induced.

discoverer: Dobzhansky, 1931.

synonym: *Su^D-f*: Suppressor of forked of Dobzhansky.

phenotype: Heterozygous *Su(f)* reduces expression of *f*; bristles blunt and wavy. Female fertility low. Homozygous lethal. RK3(A).

other information: Crossing over probably reduced.

su(fa^{swb}): suppressor of facet-strawberry

location: 1-1.96 (0.46 cm to the right of *w^a*).

origin: Spontaneous.

references: Welshons and Welshons, 1986, Genetics 113: 337-54.

phenotype: In the *cis* configuration with *fa^{swb}* it causes a facet-like rather than a glossy-like phenotype. Deficiencies for the element, when in *cis* with *fa^{swb}*, result in the complete suppression of *fa^{swb}* (Welshons and Welshons, 1985, Genetics 110: 465-77). Furthermore *In(1)78b = In(1)3A2-3;3C3-5* also suppresses *fa^{swb}* when in *cis* configuration.

cytology: Placed in 3C2-5 based on its genetic position between *w^a* and *fa^{swb}* and on the suppressive effect of *In(1)78b*.

Su(fu): Suppressor of fused

location: 3 (not mapped).

origin: Induced by ethyl methanesulfonate

references: Bussan, Limbourg-Bouchon, Mariol, Preat, and Lamour-Isnard, 1988, Roux's Arch. Dev. Biol. 197: 221-30.

phenotype: Dominant suppressor of both the wing phenotype and the maternally determined lethality of *fu* mutants.

other information: Four dominant partial suppressors observed in same experiment; not further characterized.

Su(Gl)27: Suppressor of Glued

location: 1- {64}.

origin: Induced by ethyl methanesulfonate.

references: Harte and Kankel, 1982, Genetics 101: 477-501 (fig.).

phenotype: Males carrying *Su(Gl)27* in combination with *Gl/+* are virtually wild type in phenotype, both with respect to the eye and to the optic lobe, when raised at 29°; eye slightly reduced in size with some small facets scattered throughout the eye; facets not packed together as tightly as in wild type, some smooth pigmented material separating facets from one another. The optic

lobe is very nearly normal in appearance; the lamina cell body region is thicker than in wild type and the lamina neuropil is slightly irregular in contour, but the medulla rotation and gross fiber tract abnormalities of *Gl* are missing. Phenotype less nearly normal in flies raised at 18°. Phenotypes of females not given; presumably recovered in heterozygous females and therefore dominant. *Su(Gl)/Df(1)mal10* females exhibit outheld wings; probably not allelic to *ot*, which maps to the same region, since *ot* not included in *Df(1)mal10*.

cytology: Placed in 19A5-E1 based on its inclusion in *Df(1)mal10 = Df(1)19A5-6;19E1*.

Su(Gl)57

location: 1- (not mapped).

origin: Induced by ethyl methanesulfonate.

references: Harte and Kankel, 1982, *Genetics* 101: 477-501.

phenotype: Said to reduce the severity of *Gl/+* at both 18° and 29°, but genotypes not specified.

Su(Gl)77

location: 3- (between *ru* and *h*).

origin: Induced by ethyl methanesulfonate.

references: Harte and Kankel, 1982, *Genetics* 101: 477-501 (fig.).

phenotype: Reduces the severity of *Gl* in flies raised at either 18° or 29°. The most severe allele shows more normal facet arrays in the anterior than in the posterior part of the eye. The lamina cell body layer is thicker than normal in its posterior region and the lamina neuropil is somewhat misshapen, particularly anteriorly. The medulla is abnormally rotated, its posterior edge directly apposed to the lamina, but it is more normally organized than in *Gl*. The familiar abnormal projections from the posterior lamina through the medulla are present. The second optic chiasma is also divided into several tracts. In less extreme cases, the medulla is only slightly rotated and the second optic chiasma is normal.

alleles: Although allelism not established, three independent mutations numbered 57, 102, and 160 with suppressing effects on *Gl* map between *ru* and *h* and are presumed to be allelic and are designated *Su(Gl)77¹*, *Su(Gl)77²*, and *Su(Gl)77³*, respectively. Of these *Su(Gl)77¹* is the weakest *Su(Gl)77³* is intermediate and *Su(Gl)77²* is the strongest suppressor of *Gl*.

Su(H): Suppressor of Hairless

location: 2-50.8.

synonym: *E(H); l(2)br7*.

references: Nash, 1965, *Genet. Res.* 6: 175-189.

Nash, 1970, *Genetics* 64: 471-79.

Ashburner, 1982, *Genetics* 101: 447-59.

phenotype: Homozygous lethal; hemizygotes die in the pupal stage, between head eversion and the beginning of eye pigmentation. Heterozygotes in the absence of *H* are wild type in phenotype. Suppresses *H*; *Su(H)/+*; *H/+* have 7-10 more bristles than *H/+* alone; besides affecting the bristle phenotype of *H*, all alleles tend to enhance its wing-vein phenotype of shortening L4 and L5. *Su(H)* is without effect on the lethal phenotype of homozygous *H*. One homozygous-lethal allele acts as a dominant enhancer of *H*. Most alleles are amorphic or hypomorphic; heterozygous deficiencies for the locus also suppress, and duplications enhance *H* (Nash, 1970).

Enhanced genotypes have 10-15 fewer bristles and a far more extreme loss of microchaetae on the thorax than their unenhanced counterparts. The number of bristles in *H/+* flies is inversely related to the dose of *Su(H)⁺*, with the number of bristles varying from fewer than ten with four doses to approximately 35 with one dose. Some combinations of hypomorphic alleles produce occasional escapers; survivors have distinctive phenotypes, which are described under entries for specific alleles.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>Su(H)¹</i>	spont	Plunkett, 24i		1, 2, 3, 4	
<i>Su(H)²</i>	spont	Ashburner	<i>Su(H)^{AR9}</i>	1	
<i>Su(H)³</i>	EMS	Bodmer and Walker	<i>Su(H)^{BMW4}</i>	1	hypomorphic allele
<i>Su(H)⁴</i>	EMS	Bodmer and Walker	<i>Su(H)^{BMW9}</i>	1	
<i>Su(H)⁵</i>	EMS	Harrington	<i>Su(H)^{HG3}</i>	1	
<i>Su(H)⁶</i>	EMS	Harrington	<i>Su(H)^{HG36}</i>	1	
<i>Su(H)⁷</i>	EMS	Littlewood	<i>Su(H)^{LT3}</i>	1	
<i>Su(H)⁸</i>	TEM	Ashburner	<i>Su(H)^{SF8}</i>	1	
<i>Su(H)⁹</i>	EMS	O'Donnell	<i>Su(H)^{DM9}</i>	1	
<i>Su(H)¹⁰</i>	EMS	O'Donnell	<i>Su(H)^{DM15}</i>	1	
<i>Su(H)¹¹</i>	EMS	O'Donnell	<i>Su(H)^{OK7}</i>	1	
<i>Su(H)¹²</i>	P	Shelton	<i>Su(H)^{MR1}</i>	1	
<i>Su(H)¹³</i>	P	Shelton	<i>Su(H)^{P11}</i>	1	
<i>Su(H)¹⁴</i>	P	Shelton	<i>Su(H)^{P13}</i>	1	
<i>Su(H)¹⁵</i>	P	Shelton	<i>Su(H)^{P14}</i>	1	
<i>Su(H)¹⁶</i>	EMS	Littlewood	<i>Su(H)^{S5}</i>	1	hypermorphic allele

^α 1 = Ashburner, 1982, *Genetics* 101: 447-59; 2 = Nash, 1965, *Genet. Res.* 6: 175-189; 3 = Nash, 1970, *Genetics* 64: 471-79. 4 = Plunkett, 1926, *J. Exp. Zool.* 46: 181-244.

cytology: Placed in 35B3-C1 on the basis of its inclusion in *Df(2L)AR-R1 = Df(2L)35A3-4;35B9-C1* but not *Df(2L)fn31 = Df(2L)34D3;35B3-5*.

Su(H)³

phenotype: A leaky allele; trans heterozygotes with *Su(H)¹* show 20% survival; almost viable with *Su(H)⁷*; semilethal with *Su(H)²*, *Su(H)⁴*, *Su(H)⁶*, and *Su(H)⁸*; lethal in combination with *Su(H)¹⁶*. Survivors have vestigial wings and halteres and eyes somewhat reduced and rough; bristles normal.

Su(H)⁷

phenotype: Homozygous lethal; in trans heterozygotes with other alleles produces rare escapers, which have an extreme mutant phenotype; wings and halteres similar to those of an extreme vestigial allele; eyes, although large, have rough glazed appearance and the flies are almost achaetous, having fewer than ten macrochaetae per fly; acrostichal hairs and other microchaetae reduced in number and disturbed in arrangement; tarsal claws also much reduced.

Su(H)¹⁶

phenotype: Homozygous lethal; acts as a dominant enhancer of *H*, with about the same effect as heterozygosity for a duplication for the locus.

su(Hw): suppressor of Hairy wing

location: 3-54.8.

references: Lewis, 1949, *DIS* 23: 59-60.

Klug, Bodenstein and King, 1968, *J. Exp. Zool.* 167: 151-56.

Klug, King, and Wattiaux, 1970, *J. Exp. Zool.*

174: 125-40.

Modolell, Bender, and Meselson, 1983, Proc. Nat. Acad. Sci. USA 80: 1678-82.

Rutledge, Mortin, Schwarz, Thierry-Mieg, and Meselson, 1988, Genetics 119: 391-97.

phenotype: In homozygous condition (e.g., $su(Hw)^2/su(Hw)^2$ or $su(Hw)^2/su(Hw)^7$) suppresses certain spontaneous alleles that contain *gypsy* inserts at a number of different loci, e.g., y^2 , Hw^1 , sc^1 , sc^{D2} , dm^1 , ct^6 , ct^K , lz^1 , f^1 , f^5 , f^K , B , Bxd^2 , h^1 , bx^3 , bx^{34e} , bx^{51j} , bx^{55i} , bx^K , and ci^1 ; alleles without *gypsy* inserts are not suppressed (see Lewis, 1949). Apparent exceptions to the above generalizations are sc^{D1} , which is suppressed and reportedly X ray induced and r^{SP1} and r^{SP2} , both of which show temperature-sensitive suppression but no evidence of *gypsy* insertion. With the exception of lz^{37} , which is enhanced by $su(Hw)$, alleles not known, or known not, to contain *gypsy*, but which are modified by other suppressors are unaffected by $su(Hw)$. Two alleles, ct^K and f^K , exhibit suppression in heterozygotes for either $su(Hw)$ or a deficiency for $su(Hw)$. $su(Hw)$ causes accumulation of *f* transcript in flies carrying *f* alleles that ordinarily display low levels to return to wild-type levels (Parkhurst and Corces, 1985, Cell 41: 429-37); judging from results with y^2 , this is caused by reduction of transcription from the associated *gypsy* element (Parkhurst and Corces, 1986, Mol. Cell. Biol. 6: 47-53). Females homozygous for $su(Hw)^1$, $su(Hw)^2$, $su(Hw)^3$, and $su(Hw)^4$ are sterile; females homozygous for $su(Hw)^8$ and transheterozygotes of $su(Hw)^7$ with other alleles are fertile; $su(Hw)^7/su(Hw)^2$ are fertile and, in fact, suppress the female sterility of *dm* and *lz* (Grell). In sterile combinations, vitellogenesis inhibited leading to smaller-than-normal cysts surrounded by multiple layers of follicle cells; nurse cell chromosomes remain condensed until stage 9, after which egg chambers degenerate; ovarian phenotype autonomous in transplants (Klug *et al.*, 1968, 1970). Reduced viability attributed to some alleles apparently caused by extraneous genes, since those alleles are perfectly viable in combination with $su(Hw)$ deficiencies.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
*$su(Hw)^1$	spont	Bridges, 23e4		1	
$su(Hw)^2$	spont	Lewis, 1948		2, 3, 5	1.3 kb insert
$su(Hw)^3$	EMS	Grell	$su(Hw)^{69k}$	3, 4, 5	Southern blot normal
$su(Hw)^4$	EMS	Grell	$su(Hw)^{70a}$	3, 5	
$su(Hw)^5$	γ ray	Coyne	$su(Hw)^B$	4	Molecular deletion
$su(Hw)^6$	γ ray	Coyne	$su(Hw)^C$	4	Molecular deletion
$su(Hw)^7$	spont	Grell	$su(Hw)^f$	3, 4	Southern blot normal; in <i>TM6</i>
$su(Hw)^8$	spont	Grell	$su(Hw)^{f3}$	3, 4	0.7 kb insert

^α 1 = Bridges, 1932, Proc. Int. Congr. Genet. 6th, Vol. 2: 12-14; 2 = Lewis, 1949, DIS 23: 59-60; 3 = Modolell, Bender, and Meselson, 1983, Proc. Nat. Acad. Sci. USA 80: 1678-82; 4 = Parkhurst, Harrison, Remington, Spana, Kelley, Coyne, and Corces, 1988, Genes Dev. 2: 1205-15; 5 = Rutledge, Mortin, Schwarz, Thierry-Mieg, and Meselson, 1988, Genetics 119: 391-97.

cytology: Placed in 88A12-B5 based on its inclusion in $Df(3R)red-P52 = Df(3R)88A12-B1;88B4-5$ [Lewis, 1981, Developmental Biology Using Purified Genes (Brown and Fox, eds.). Academic Press, New York, pp. 189-

208].

molecular biology: Region cloned and sequenced by Parkhurst, Harrison, Remington, Spana, Kelley, Coyne, and Corces (1988, Genes Dev. 2: 1205-15); normal allele produces a 3.3 kb message which is expressed throughout development, and is expressed in ovaries; transcription from right to left, i.e., from the tip of 3R toward the centromere. Genomic sequence comprises seven exons, separated by six introns, all but the first of which are short, in the range of 50-60 base pairs. Conceptual amino-acid sequence corresponds to a 109,000 dalton polypeptide; it contains twelve Zn finger motifs in the middle of the protein encoded by sequences contained in several of the minixons and a highly acidic region (48% Asp + Glu) between amino acids 154 and 202 immediately preceding the first Zn finger; structure suggests a transcription factor. Nuclear extracts or partially purified protein from overexpressing *E. coli* or *Drosophila* cells bind to a 367 base pair DNA sequence containing twelve copies of PyPuTTGCATACCPy from the 5' untranslated end of *gypsy* between the 5' LTR and the initial ATG (Spana, Harrison, and Corces, 1988, Genes Dev. 2: 1414-23; Mazo, Mizrokhi, Karavanov, Sedkov, Krichvskaja, and Ilyn, 1989, EMBO J. 8: 903-11); footprinting indicates protection of a 55 bp domain by $su(Hw)$ protein. Antibodies raised against the protein label 100 to 200 sites on polytene chromosomes (Spana *et al.*).

 $su(lz^{34})$: suppressor of lozenge-34

location: 3- (not located).

origin: Spontaneous.

discoverer: H. A. Bender.

references: Bender and Green, 1960, Genetics 45: 1563-66.

phenotype: lz^{34} ; $su(lz^{34})$ eyes larger, less rough, and more normal in color than lz^{34} alone. Female distinctly more fertile with $su(lz^{34})$ but still lacks parovaria and spermathecae. RK2.

***Su(M)*: Suppressor of Minute**

These are tandem duplications selected on the basis of their normal phenotype in heterozygous combination with a specific Minute. They are treated under $Dp(2;2)M^+$ and $Dp(3;3)M^+$.

su(msm1)

location: 2- (not mapped).

origin: Induced by ethyl methanesulfonate.

references: Nishida, 1980, Jpn. J. Genet. 55: 427-39.

phenotype: Most males homozygous for $su(msm1)$ and $ms(3)ml$ have small spiral-shaped testes and motile sperm in their seminal vesicles, whereas those homozygous for $ms(3)ml$ have rudimentary testes.

***Su(par)*: Suppressor of paralog**

location: 1- to the right of *v*.

references: Thierry-Mieg, 1982, Genetics 100: 209-37.

phenotype: Semidominant. In homozygous condition reduces the incidence of agametic *par/par* females from 95-100% to 51-61% and abdominal defects from 26-49% to 5-14%. No effect on fecundity at 29° or the frequency of cephalic defects caused by *par*. *par+/par Su(par)* females are 77% agametic. *Su(par)* homozygotes in the absence of *par* are normal.

Su(Pc)37D: Suppressor-of-Polycomb-37D

location: 2-{54}.

references: Kennison and Tamkun, 1988, Proc. Nat. Acad. Sci. USA 85: 8136-40.

phenotype: Suppresses *Pc*.

cytology: Located at 37D2 to 38C1.

su(pr): suppressor of purple

location: 3-95.5.

references: Schultz and Bridges, 1932, Am. Nat. 66: 323-34.

Rutledge, Mortin, Kelley, and Meselson, 1988, Genetics 119: 391-97.

phenotype: Completely suppresses *pr*¹ but not *v*¹ both of which contain an insert of transposable element *412*, and both of which are suppressed by *su(s)*. Also suppresses both *lz*³⁴ and *lz*^k; among alleles with *gypsy* inserts, *su(pr)* suppresses *ct*^K, *f*¹, and *f*⁵, enhances *Hw*¹, *ct*^K, *bx*³, and *bx*^{34e}, but has no influence on the expression of *y*², *sc*¹, or *ct*⁶. No visible mutant phenotype. Abnormal phenotypes, low viability and sterility described for *su(pr)*¹ and *su(pr)*^B extraneous to *su(pr)*, since *su(pr)*^B/*su(pr)*¹ displays normal viability and fertility.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
* <i>su(pr)</i> ¹	spont	Stem, 27C2		3, 4, 5	<i>In(3R)su(pr)</i>
<i>su(pr)</i> ²	spont	Bridges, 29a13	<i>su(pr)</i> ^B	1, 2, 3	
<i>su(pr)</i> ³	EMS?	Grell	<i>su(pr)</i> ^{e3}	2	
<i>su(pr)</i> ⁴	EMS?	Grell	<i>su(pr)</i> ^{e4}	2	

^α 1 = Bridges, 1932, Z. Indukt. Abstamm. Vererbungs. 60: 207-18; 2 = Rutledge, Mortin, Kelley, and Meselson, 1988, Genetics 119: 391-97; 3 = Schultz and Bridges, 1934, Am. Nat. 66: 323-34; 4 = Stem, 1929, Z. Indukt. Abstamm. Vererbungs. 52: 373-89; 5 = Stem, 1934, DIS 1: 35.

su(r): suppressor of rudimentary

location: 1-27.7.

references: Bahn, 1972, DIS 49: 38 & 98.

Strøman, Bahn, Nørby, and Sick, 1973, Hereditas 73: 239-46 (fig.).

Strøman, 1974, Hereditas 78: 157-68.

phenotype: Suppresses both the phenotypic effects and the female sterility of all *r* alleles; also suppresses the *r* phenocopying and lethal effects of 6-azauracil administration. Little or no dihydrouracil dehydrogenase (EC 1.3.99.11) activity. Thought to interfere with pyrimidine catabolism. Unable to degrade uracil and extremely sensitive to exogenous pyrimidine. Enhances the expression of *dp* and *net*, and causes *b* flies to have very black bodies, darker than wild type, especially along the wing veins; forms synthetic lethals in combination with *tt* and

whd.

alleles: Ten ethyl-methanesulfonate-induced alleles recorded; one by Bahn and nine by Falk and DeBoer (1980, Mol. Gen. Genet. 180: 419-24).

su(s): suppressor of sable (R.A. Voelker)

location: 1-0+ [rare crossovers occur between *y* and *su(s)*].

references: Bridges, 1919, Anat. Rec. 15: 357-58.

Schultz and Bridges, 1932, Am. Nat. 66: 323-34.

Shapard, 1960, Genetics 45: 359-76.

Baglioni, 1960, Heredity 15: 87-96.

Hayman and Maddern, 1972, DIS 49: 72.

Maddern, R.M., 1973, Ph.D. Thesis, Univ. Adelaide, Australia.

Jacobson, Grell, Yim and Gardner, 1982a, Genet. Res. 40: 19-32.

Jacobson, Yim, Grell and Wobbe, 1982b, Cell 30: 817-23.

Chang, Wisely, Huang and Voelker, 1986, Mol. Cell. Biol. 6: 1520-28.

Rutledge, Mortin, Schwarz, Thierry-Mieg and Meselson, 1988, Genetics 119: 391-97.

Searles and Voelker, 1986, Proc. Nat. Acad. Sci. USA 83: 404-08.

Voelker, Huang, Wisely, Sterling, Bainbridge and Hiraizumi, 1989, Genetics 122: 625-42.

phenotype: In hemizygous or homozygous condition suppresses certain spontaneous alleles that contain *412* insertions at several loci (e.g., *v*, *v*², *v*^k, *pr*, *pr*^{bw}, *sp*) or hybrid dysgenesis-induced alleles that contain *P* element insertions (e.g., *sn*^w, *y*^{76d28}). *s* is suppressible but does not have a *412* insertion (Searles and Voelker, 1986). *bx* is caused by a *412* insertion but is not suppressed (Voelker *et al.*, 1989). Not all *su(s)* alleles suppress all suppressible alleles (Jacobson *et al.*, 1982a). Although reported as recessive, some alleles show a slight dominance (Shapard, 1960; Baglioni, 1960). No lethal alleles recovered in a lethal saturation screen of the *su(s)* region, but a number of X-ray-, EMS- and ENU-induced alleles exhibit sterility or reduced male fertility when reared at 18°C (Voelker *et al.*, 1989). Some alleles cause a slight spreading of the wings, especially in males. Suppression of *v* and *y*^{76d28} occurs by elevating the levels of pseudo-wild-type, presumably translatable, message (Searles, Ruth, Pret, Fridell, and Ali, submitted; P. Geyer, V. Corces and M. Green, personal communication). Some alleles enhance some *gypsy*-caused mutations (Rutledge *et al.*, 1988). A duplication of *su(s)*⁺ enhances suppressible *pr* alleles (Jacobson *et al.*, 1982b).

alleles:

allele	origin	discoverer	synonym	ref	comments
* <i>su(s)</i> ¹	spont	Bridges		1	
<i>su(s)</i> ²	spont	Bridges		1, 2, 4	<i>gypsy</i> insertion in first intron associated with 425 bp deletion; <i>gypsy</i> excised leaving solo LTR in <i>su(s)</i> ² <i>w^a cv t</i> stock
<i>su(s)</i> ³	X ray	Schultz		1, 2, 12	No abnormality on Southern; enhances some <i>gypsy</i> -caused mutations
<i>su(s)</i> ⁴	spont	Stern	<i>su</i> ^{S2} _S ^{v pr}	1, 2, 4, 10	<i>rambler</i> insertion in first intron
* <i>su(s)</i> ⁵	X ray	Green	<i>su</i> ⁵⁰¹⁶ _S ^{-v}	1	
<i>su(s)</i> ⁶	spont	Shapard	<i>su(s)</i> ⁵⁰¹		
<i>su(s)</i> ⁷	spont	Green	<i>su(s)</i> ^{51c15}	1, 2, 4, 6	<i>gypsy</i> insertion in first intron
<i>su(s)</i> ⁸	spont	Green	<i>su</i> ^{51j6} _S ^{-v}	1, 2, 4	<i>gypsy</i> insertion in first intron
* <i>su(s)</i> ⁸	spont	Green	<i>su(s)</i> ^{51j}		
<i>su(s)</i> ⁹	spont	Maddern	<i>su</i> ^{52c} _S ^{-v}	1	
<i>su(s)</i> ⁹	spont	Maddern	<i>su(s)</i> ^{52c}		
* <i>su(s)</i> ¹⁰	EMS	Maddern	<i>su(s)</i> ⁶⁸ⁱ	2, 4, 5	weak, variable suppressor, good viability; <i>rover</i> insertion in first intron
<i>su(s)</i> ¹¹	EMS	Maddern	<i>su(s)</i> ¹²⁷⁰		
* <i>su(s)</i> ¹¹	EMS	Maddern	<i>su(s)</i> ^{68h}	5	strong suppressor, reduced viability
* <i>su(s)</i> ¹²	EMS	Maddern	<i>su(s)</i> ^{68j10}	5	very strong suppressor, very low viability
* <i>su(s)</i> ¹³	EMS	Maddern	<i>su(s)</i> ^{68j21}	5	very strong suppressor, low viability
* <i>su(s)</i> ¹⁴	EMS	Maddern	<i>su(s)</i> ^{68j23}	5	weak, variable suppressor, good viability
* <i>su(s)</i> ¹⁵	EMS	Maddern	<i>su(s)</i> ^{68j25}	5	medium suppressor, low viability
* <i>su(s)</i> ¹⁶	EMS	Maddern	<i>su(s)</i> ⁶⁸¹⁽¹⁾	5	strong suppressor, low viability
* <i>su(s)</i> ¹⁷	EMS	Maddern	<i>su(s)</i> ⁶⁸¹⁽²⁾	5	strong suppressor, low viability
* <i>su(s)</i> ¹⁸	EMS	Maddern	<i>su(s)</i> ⁶⁸¹⁽³⁾	5	strong suppressor, very low viability
* <i>su(s)</i> ¹⁹	EMS	Maddern	<i>su(s)</i> ^{69f11}	5	weak, variable suppressor
* <i>su(s)</i> ²⁰	EMS	Maddern	<i>su(s)</i> ^{69f14}	5	very strong suppressor
* <i>su(s)</i> ²¹	X ray	Maddern	<i>su(s)</i> ^{69f19}	5	strong suppressor
* <i>su(s)</i> ²²	X ray	Maddern	<i>su(s)</i> ^{69g7}	5	strong suppressor, good viability
* <i>su(s)</i> ²³	X ray	Maddern	<i>su(s)</i> ^{69g11}	5	strong suppressor, good viability
* <i>su(s)</i> ²⁴	X ray	Maddern	<i>su(s)</i> ^{69g12}	5	strong suppressor, good viability
* <i>su(s)</i> ²⁵	X ray	Maddern	<i>su(s)</i> ⁶⁹¹⁽¹⁾	5	weak suppressor, reduced viability
* <i>su(s)</i> ²⁶	X ray	Maddern	<i>su(s)</i> ⁶⁹¹⁽²⁾	5	strong suppressor, good viability
* <i>su(s)</i> ²⁷	X ray	Maddern	<i>su(s)</i> ⁶⁹¹⁽³⁾	5	strong suppressor, good viability
* <i>su(s)</i> ²⁸	DNA trans-formation	Fox	<i>su(s)</i> ⁶⁹¹⁽⁴⁾	5	weak, variable suppressor, good viability
<i>su(s)</i> ²⁸	DNA trans-formation	Fox	<i>su(s)</i> ^{e5.6}	8, 10	<i>Delta 88</i> insertion in first intron; does not suppress <i>sp</i>
<i>su(s)</i> ²⁹	DNA trans-formation	Germeraad	<i>su(s)</i> ⁶⁶	9	<i>17.6</i> insertion in first intron
<i>su(s)</i> ³⁰	EMS	Grigliatti	<i>su(s)</i> ^{J1}	7	normal Southern
<i>su(s)</i> ³¹	EMS	Grigliatti	<i>su(s)</i> ^{J8}	7	normal Southern
<i>su(s)</i> ³²	EMS	Grigliatti	<i>su(s)</i> ^{J18}	7	normal Southern
<i>su(s)</i> ³³	EMS	Grigliatti	<i>su(s)</i> ^{J24}	7	normal Southern
<i>su(s)</i> ³⁴	EMS	Grigliatti	<i>su(s)</i> ^{J30}	7	normal Southern
<i>su(s)</i> ³⁵	X ray	Grell	<i>su(s)</i> ^{X1}	10	normal Southern, cs male fertility
<i>su(s)</i> ³⁶	X ray	Grell	<i>su(s)</i> ^{X4}	10	normal Southern
<i>su(s)</i> ³⁷	EMS	Grell	<i>su(s)</i> ^{e1}	10	normal Southern, cs male fertility
<i>su(s)</i> ³⁸	EMS	Grell	<i>su(s)</i> ^{e6}	10	normal Southern, poor viability
<i>su(s)</i> ³⁹	HD	Voelker	<i>su(s)</i> ^{W20}	2, 4	<i>P</i> element insertion in untranslated leader
<i>su(s)</i> ⁴⁰	spont	Carpenter	<i>su(s)</i> ^{ab}	2, 4, 10	<i>HMS Beagle</i> insertion in first intron; does not suppress <i>sp</i>
<i>su(s)</i> ⁴¹	spont	Voelker	<i>su(s)</i> ^{83f1}	2, 4	<i>roamer</i> insertion in first intron
<i>su(s)</i> ⁴²	HD	Green	<i>su(s)</i> ^{83f24(8)}	2, 4	<i>P</i> element insertion in untranslated leader, induced in <i>R(1)2</i>
<i>su(s)</i> ⁴³	HD	Green	<i>su(s)</i> ^{83f24(10)}	2, 4	<i>P</i> element insertion in first intron, induced in <i>R(1)2</i>
<i>su(s)</i> ⁴⁴	HD	Green	<i>su(s)</i> ^{83g2(15)}	2, 4	<i>P</i> element insertion in untranslated leader, induced in <i>R(1)2</i>
<i>su(s)</i> ⁴⁵	HD	Green	<i>su(s)</i> ^{83g2(22)}	2, 4	<i>P</i> element insertion in untranslated leader, induced in <i>R(1)2</i>
<i>su(s)</i> ⁴⁶	HD	Green	<i>su(s)</i> ^{83g2(10)}	13	induced in <i>R(1)2</i>
<i>su(s)</i> ⁴⁷	spont	Voelker	<i>su(s)</i> ^{84a}	4	<i>springer</i> insertion in first intron
<i>su(s)</i> ⁴⁸	ENU	Voelker	<i>su(s)</i> ^{A17}	3	normal Southern
<i>su(s)</i> ⁴⁹	ENU	Voelker	<i>su(s)</i> ^{A18}	3	normal Southern, cs male fertility
<i>su(s)</i> ⁵⁰	ENU	Voelker	<i>su(s)</i> ^{A65}	3	normal Southern
<i>su(s)</i> ⁵¹	ENU(spont?)	Voelker	<i>su(s)</i> ^{A66}	3, 4	<i>roamer</i> insertion in first intron
<i>su(s)</i> ⁵²	ENU	Voelker	<i>su(s)</i> ^{A67}	3	normal Southern, cs male fertility
<i>su(s)</i> ⁵³	ENU	Voelker	<i>su(s)</i> ^{A68}	3	normal Southern
<i>su(s)</i> ⁵⁴	ENU	Voelker	<i>su(s)</i> ^{A107}	3	normal Southern, cs male fertility
<i>su(s)</i> ⁵⁵	ENU	Voelker	<i>su(s)</i> ^{B2}	3	normal Southern, induced in <i>Dp(1;3)E1</i>
<i>su(s)</i> ⁵⁶	ENU	Voelker	<i>su(s)</i> ^{B6}	3	normal Southern, cs male fertility, induced in <i>Dp(1;3)E1</i>
<i>su(s)</i> ⁵⁷	ENU	Voelker	<i>su(s)</i> ^{B7}	3	normal Southern, cs male fertility, induced in <i>Dp(1;3)E1</i>
* <i>su(s)</i> ⁵⁸	ENU	Voelker	<i>su(s)</i> ^{B8}	3	induced in <i>Dp(1;3)E1</i>
<i>su(s)</i> ⁵⁹	ENU	Voelker	<i>su(s)</i> ^{B13}	3	normal Southern, cs male fertility, induced in

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allele	origin	discoverer	synonym	ref	comments
su(s) 60	ENU	Voelker	<i>su(s) B14</i>	3	<i>Dp(1;3)E1</i> normal Southern, cs male fertility, induced in
su(s) 61	ENU	Voelker	<i>su(s) B15</i>	3	<i>Dp(1;3)E1</i> normal Southern, cs male fertility, induced in
su(s) 62	ENU	Voelker	<i>su(s) B16</i>	3	<i>Dp(1;3)E1</i> normal Southern, cs male fertility, induced in
su(s) 63	ENU	Voelker	<i>su(s) B17</i>	3	<i>Dp(1;3)E1</i> normal Southern, cs male fertility, induced in
su(s) 64	ENU	Voelker	<i>su(s) B18</i>	3	<i>Dp(1;3)E1</i> normal Southern, induced in <i>Dp(1;3)E1</i>
su(s) 65	ENU	Voelker	<i>su(s) B22</i>	3	normal Southern, cs male fertility, induced in
*su(s) 66	ENU	Voelker	<i>su(s) B25</i>	3	<i>Dp(1;3)E1</i> induced in <i>Dp(1;3)E1</i>
su(s) 67	ENU	Voelker	<i>su(s) B26</i>	3	normal Southern, cs male fertility, induced in
su(s) 68*	ENU	Voelker	<i>su(s) B27</i>	3	<i>Dp(1;3)E1</i> induced in <i>Dp(1;3)E1</i>
su(s) 69	EMS	Voelker	<i>su(s) C1</i>	3	normal Southern, cs male fertility, induced in
su(s) 70	EMS	Voelker	<i>su(s) C2</i>	3	<i>Dp(1;3)E1</i> normal Southern, cs male fertility, induced in
su(s) 71	spont	Simmons	<i>su(s) sn-w</i>	11	<i>Dp(1;3)E1</i> unidentified insertion; suppresses some <i>P</i> element-caused <i>sn</i> alleles
su(s) 72	EMS	Rutledge	<i>su(s) 44</i>	12	enhances some <i>gypsy</i> -caused mutations
su(s) 73	γ ray	Voelker	<i>su(s) 87k1</i>	13	induced in <i>Dp(1;3)E1</i>
su(s) 74	γ ray	Voelker	<i>su(s) 88a1</i>	13	induced in <i>Dp(1;3)E1</i>
su(s) 75	γ ray	Voelker	<i>su(s) 88a2</i>	13	induced in <i>Dp(1;3)E1</i>
su(s) 76	γ ray	Voelker	<i>su(s) 88a3</i>	13	induced in <i>Dp(1;3)E1</i>
su(s) 77	γ ray	K. White	<i>su(s) g1</i>	14	induced in <i>Dp(1;3)E1</i>
su(s) 78	γ ray	K. White	<i>su(s) g2</i>	14	
su(s) 79	γ ray	K. White	<i>su(s) g3</i>	14	
su(s) 80	γ ray	K. White	<i>su(s) g4</i>	14	
su(s) 81	γ ray	Voelker	<i>su(s) 89a1</i>	13	induced in <i>Dp(1;3)E1</i>
su(s) 82	γ ray	Voelker	<i>su(s) 89a2</i>	13	induced in <i>Dp(1;3)E1</i>
su(s) 83	γ ray	Voelker	<i>su(s) 89a3</i>	13	induced in <i>Dp(1;3)E1</i>
su(s) 84	γ ray	Voelker	<i>su(s) 89a4</i>	13	induced in <i>Dp(1;3)E1</i>
su(s) 85	γ ray	Voelker	<i>su(s) 89a5</i>	13	induced in <i>Dp(1;3)E1</i>
su(s) 86	γ ray	Voelker	<i>su(s) 89a6</i>	13	induced in <i>Dp(1;3)E1</i>
su(s) 87	γ ray	Voelker	<i>su(s) 89b1</i>	13	induced in <i>Dp(1;3)E1</i>
su(s) 88	γ ray	Voelker	<i>su(s) 89b2</i>	13	induced in <i>Dp(1;3)E1</i>
su(s) 89	γ ray	Voelker	<i>su(s) 89b3</i>	13	induced in <i>Dp(1;3)E1</i>
su(s) 90	γ ray	Voelker	<i>su(s) 89b4</i>	13	induced in <i>Dp(1;3)E1</i>
su(s) 91	γ ray	Voelker	<i>su(s) 89b5</i>	13	induced in <i>Dp(1;3)E1</i>
su(s) 92	γ ray	Voelker	<i>su(s) 89b6</i>	13	induced in <i>Dp(1;3)E1</i>
su(s) 93	γ ray	Voelker	<i>su(s) 89b7</i>	13	induced in <i>Dp(1;3)E1</i>
su(s) 94	HD	Green	<i>su(s) 89g1</i>	15	induced in <i>Dp(1;3)E1</i>
su(s) 95	HD	Green	<i>su(s) 89g2</i>	15	<i>P</i> element insertion; suppresses <i>y</i> ^{76d28}
su(s) 96	γ ray	Voelker	<i>su(s) f2</i>	13	<i>P</i> element insertion; suppresses <i>y</i> ^{76d28}
su(s) 97	γ ray	Voelker	<i>su(s) f3</i>	13	
su(s) 98	γ ray	Voelker	<i>su(s) f4</i>	13	
su(s) 99	γ ray	Voelker	<i>su(s) f15</i>	13	
su(s) 100	γ ray	Voelker	<i>su(s) f25</i>	13	
su(s) 101	γ ray	Voelker	<i>su(s) f35</i>	13	
su(s) 102	γ ray	Voelker	<i>su(s) f39</i>	13	
su(s) 103	γ ray	Voelker	<i>su(s) f42</i>	13	
su(s) 104	γ ray	Voelker	<i>su(s) f43</i>	13	
su(s) 105	γ ray	Voelker	<i>su(s) f49</i>	13	
su(s) 106	γ ray	Voelker	<i>su(s) f54</i>	13	
su(s) 107	γ ray	Voelker	<i>su(s) f57</i>	13	
su(s) 108	γ ray	Voelker	<i>su(s) f59</i>	13	
su(s) 109	γ ray	Voelker	<i>su(s) f61</i>	13	
su(s) 110	γ ray	Voelker	<i>su(s) f66</i>	13	
su(s) 111	γ ray	Voelker	<i>su(s) f67</i>	13	
su(s) 112	γ ray	Voelker	<i>su(s) f68</i>	13	
su(s) 113	γ ray	Voelker	<i>su(s) f71</i>	13	
su(s) 114	γ ray	Voelker	<i>su(s) f72</i>	13	
su(s) 115	γ ray	Voelker	<i>su(s) f76</i>	13	
su(s) 116	γ ray	Voelker	<i>su(s) f77</i>	13	
su(s) 117	γ ray	Voelker	<i>su(s) f80</i>	13	
su(s) 118	γ ray	Voelker	<i>su(s) f83</i>	13	
su(s) 119	γ ray	Voelker	<i>su(s) f85</i>	13	
su(s) 120	γ ray	Voelker	<i>su(s) m5</i>	13	induced in <i>Dp(1;Y)y</i> ⁺ <i>sc</i>
su(s) 121	γ ray	Voelker	<i>su(s) m18</i>	13	induced in <i>Dp(1;Y)y</i> ⁺ <i>sc</i>
su(s) 122	γ ray	Voelker	<i>su(s) m27</i>	13	induced in <i>Dp(1;Y)y</i> ⁺ <i>sc</i>
su(s) 123	γ ray	Voelker	<i>su(s) m44</i>	13	induced in <i>Dp(1;Y)y</i> ⁺ <i>sc</i>
su(s) 124	γ ray	Voelker	<i>su(s) m48</i>	13	induced in <i>Dp(1;Y)y</i> ⁺ <i>sc</i>
su(s) 125	γ ray	Voelker	<i>su(s) m72</i>	13	induced in <i>Dp(1;Y)y</i> ⁺ <i>sc</i>

allele	origin	discoverer	synonym	ref	comments
su(s) 126	γ ray	Voelker	<i>su(s) m77</i>	13	induced in <i>Dp(1;Y)y⁺ sc</i>
su(s) 127	γ ray	Voelker	<i>su(s) m78</i>	13	induced in <i>Dp(1;Y)y⁺ sc</i>
su(s) 128	γ ray	Voelker	<i>su(s) m80</i>	13	induced in <i>Dp(1;Y)y⁺ sc</i>
su(s) 129	γ ray	Voelker	<i>su(s) m90</i>	13	induced in <i>Dp(1;Y)y⁺ sc</i>
su(s) 130	γ ray	Voelker	<i>su(s) m94</i>	13	induced in <i>Dp(1;Y)y⁺ sc</i>
su(s) 131	γ ray	Voelker	<i>su(s) m100</i>	13	induced in <i>Dp(1;Y)y⁺ sc</i>
su(s) 132	γ ray	Voelker	<i>su(s) m103</i>	13	induced in <i>Dp(1;Y)y⁺ sc</i>
su(s) 133	γ ray	Voelker	<i>su(s) m105</i>	13	induced in <i>Dp(1;Y)y⁺ sc</i>

^α 1 = CP627; 2 = Chang, Wisely, Huang and Voelker, 1986, Mol. Cell. Biol. 6: 1520-28; 3 = Voelker, Huang, Wisely, Sterling, Bainbridge and Hiraizumi, 1989, Genetics 122: 625-42; 4 = Voelker, Graves, Gibson and Eisenberg (in preparation for Genetics); 5 = Maddern, 1973, Ph.D. Thesis, Department of Genetics, University of Adelaide; 6 = Shapard, 1960, Genetics 45: 359-76; 7 = Grigliatti, personal communication; 8 = Fox, 1977, Molecular Genetic Modification of Eukaryotes, (Rubenstein *et al.*, eds.), pp. 101-31; Fox and Yoon, 1970, Proc. Nat. Acad. Sci. USA 67: 1608-15; 9 = Germeraad, 1975, Genetics 80: 534-35; 1976, Nature 262: 229-31; 10 = Jacobson, Grell, Yim and Gardner, 1982, Genet. Res. 40: 19-32; 11 = Simmons, unpublished data; 12 = Rutledge, Mortin, Schwarz, Thierry-Mieg and Meselson, 1988, Genetics 119: 391-97; 13 = Voelker, unpublished data; 14 = White, unpublished data; 15 = Green, unpublished data.

cytology: Placed in 1B10-1C1 (Hayman and Maddern, 1972; Chang *et al.*, 1986).

molecular biology: An 8 kb region that rescues *su(s)* mutations when reintroduced by *P* element-mediated transformation of embryos was sequenced (R. Voelker, W. Gibson, J. Graves, J. Sterling and M. Eisenberg, in preparation). A 7.8 kb primary transcript that is produced throughout the life cycle is transcribed telomere to centromere. It is processed to a ~5 kb mature message by splicing out five introns that range in size from 60 to 2053 nucleotides, the latter occurring within but near the 3' end of a ~500 nucleotide nontranslated leader. All eleven spontaneous mutations examined contain mobile element insertions within the 2053 nt intron. One dysgenesis-induced allele contains a *P* element insertion within that same intron, whereas four other alleles contain *P* element insertions within the nontranslated leader 5' to the large intron (R. Voelker, J. Graves, W. Gibson and M. Eisenberg, in preparation). The conceptual protein is a ~145,000 dalton polypeptide that contains an *opa*-like sequence and a highly charged region similar to regions of some RNA-binding/splicing proteins. The protein is located primarily in the nucleus.

Su(S): Suppressor of Star

location: 2-3; based on cytological location between *shr* (2-2.3) and *dpp* (2-4.0).

origin: Synthetic.

discoverer: Curry, 37b.

references: Morgan, Bridges, and Schultz, 1937, Year Book - Carnegie Inst. Washington 36: 301. Lewis, 1945, Genetics 30: 154.

phenotype: *Su(S)/S* and *Su(S)/+* wild type. RK2A.

cytology: Associated with the deficiency for 22D1 to 22E1 or the deficiency for 33F to 34A9, or both, derived by combining the left end of *In(2L)Cy = In(2L)22D1-2;33F5-34A1* and the right end of *In(2L)t = In(2L)22D3-E1;34A8-9*. According to Lewis (1945), the region between 22D1 and 22E1 is more likely responsible.

***Su(sc): Suppressor of scute**

location: 3-59.

discoverer: Payne.

synonym: *sc-Inh-3: scute Inhibitor on chromosome 3; Ext-sct-3*.

references: 1921, Genetics 5: 501-42.

Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 158.

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 225, 235.

phenotype: Tends to restore bristles removed by *sc* in *Su(sc)/+* heterozygotes. RK3.

su(SD): suppressor of Segregation Distortion

location: 1-49.5 [53/74 of the distance from *g* to *sd* (Waddle, Owens, and Petty)].

references: Sandler, 1962, Am. Nat. 96: 161-65.

Sandler and Rosenfeld, 1962, Can. J. Genet. Cytol. 4: 453-57.

Katakao, 1967, Jpn. J. Genet. 42: 327-33.

Waddle and Oster, 1977, DIS 52: 5.

phenotype: Several *X* chromosomes that reduce the *k* value in *SD/+* males from nearly 1.0 to a value closer to 0.5. Not clear that the various *X*-linked suppressors of distortion are related.

Su(sph): Suppressor of sparse hairs

location: 2-24 or 25.

synonym: *Su-x4*.

references: Voss and Falk, 1973, Mutat. Res. 20: 221-34.

phenotype: Allele specific suppressor of *sph*. *Su(sph)* permits survival of *sph*⁴ but not *sph*⁷ males; both homozygous and heterozygous *Su(sph)* allow 50 to 60% control survival. *sph*⁴ females are rescued by homozygous but not heterozygous *Su(sph)* to nearly 20% of control.

alleles: One spontaneous allele [*Su(sph)*¹] and one induced by X rays [*Su(sph)*²].

cytology: Polytene chromosomes normal.

***Su(ss): Suppressor of spineless**

location: 3-61 (between *bx* and *sr*).

origin: Spontaneous.

discoverer: Bridges, 22g15.

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 236.

phenotype: *Su(ss)/+* converts *ss/ss* to wild type except for reduced and erect posterior scutellars. Homozygous lethal. RK2.

Su(ss)²

origin: Spontaneous.

discoverer: E. B. Lewis, 1947.

references: 1950, DIS 24: 59.

phenotype: Homozygous or heterozygous *Su(ss)*² causes *ss* to have long bristles that are only slightly thin, like a mild Minute; however, the posterior scutellars remain greatly reduced as in un-suppressed *ss*. Temperature-sensitive allele; suppression more extreme at 29° than at

17°; temperature-sensitive period from 5-34 h after onset of puparium formation (Mglinetz, Ivanov, and Kostina, 1978, *Ontogenez* 9: 136-41). RK2.

Su(ss)³

origin: Spontaneous.

discoverer: Hexter, 1950.

references: 1953, *DIS* 27: 55-56.

phenotype: *ss Su(ss)³* homozygote wild type for all bristles; *ss Su(ss)³/ss* + intermediate between *ss* and wild type. *ss Su(ss)³/ss bx Su(ss)²* is wild type. RK2.

Su(Ste): Suppressor of Stellate

location: Y-between *kl2* and *kl1*.

references: Meyer, Hess, and Beerman, 1961, *Chromosoma* 12: 676-716.

Hardy and Kennison, 1980, *DIS* 55: 55.

Gatti, M. and S. Pimpinelli, 1983, *Chromosoma* 88: 349-373.

Hardy, Lindsley, Livak, Lewis, Sivertsen, Joslyn, Edwards, and Bonaccorsi, 1984, *Genetics* 107: 591-610.

Livak, 1984, *Genetics* 107: 611-634.

Livak, 1990, *Genetics* 124: 303-16.

phenotype: Designates the region of the Y chromosome whose presence decreases both abundance and splicing of the X-linked *Ste* transcripts. *Ste* males deficient for *Su(Ste)* display abundant star-shaped aggregates of needle-shaped crystals in the nuclei and cytoplasm of their primary spermatocytes; their spermatids contain micronuclei and nebenkerne of nonuniform size; and they are sterile. *Ste*⁺ males deficient for *Su(Ste)* have one or more long needle-shaped crystals in their primary spermatocytes and micronuclei and irregular nebenkerne in their spermatids; these males are fertile and display irregular disjunction as follows: (1) both the sex chromosomes and the large autosomes undergo nondisjunction, (2) the fourth chromosomes disjoin regularly, (3) sex chromosome nondisjunction is more frequent in cells in which the second or third chromosomes nondisjoin than in cells in which autosomal disjunction is regular, (4) in doubly exceptional cells, the sex chromosomes tend to segregate to the opposite pole from the autosomes, and (5) there is meiotic drive; *i.e.*, reciprocal meiotic products are not recovered with equal frequencies, complements with fewer chromosomes being recovered more frequently than those with more chromosomes. Two smaller component deficiencies of the *Su(Ste)* deficiency display a normal meiotic phenotype in *Ste*⁺ males and low levels of meiotic nondisjunction in *Ste* males.

cytology: Placed primarily in the Hoechst-dull region h11 of the Y chromosome, between the Y breakpoints of *T(1;Y)P7 = T(1;Y)h33;h11* and *T(1;Y)E15 = T(1;Y)h26-29;h12-13*.

molecular biology: Comprises approximately 80 copies of a sequence that is partly homologous to the *Ste* sequences on the X chromosome; actually the number of repeats varies over a three-fold range in Y's from natural populations (Lyckegaard and Clark, 1989, *Proc. Nat. Acad. Sci. USA* 86: 1944-48). The repeat length, however, is 2.6-2.8 kb compared to 1.15-1.25 for *Ste*. The majority of these sequences lie proximal to the breakpoint of *T(1;Y)P7 = T(1;Y)h33;h11*; and distal to that of *T(1;Y)E15 = T(1;Y)h26;h11*; all *Su(Ste)* sequences lie distal to the breakpoint of *T(1;Y)E15 = T(1;Y)h26-29;h12-13*. Dele-

tions for most or all of these sequences results in abundant spliced *Ste* RNA in the testes, compared the less abundant and largely unspliced transcripts detectable in XY males. Results from partial deletions more nearly resemble those from XY than from XO testes. Sequences of two *Su(Ste)* repeats show segments homologous to *Ste* and totally unrelated segments; the homologous regions show a number of single nucleotide substitutions, including one that alters a splice-acceptor site; the sequence also contains an insert of a variable number of AAC repeats.

Su(stn): Suppressor of stoned

location: 1-22 (between *sn* and *oc*).

references: Kelly, Hannan, and Petrovich, 1987, *J. Neurogenet.* 4: 144-45.

phenotype: Suppresses both the behavioral and the viability effects of *stn*.

su(t): suppressor of tan

location: 3-26.

origin: Spontaneous.

discoverer: Bridges, 22k2.

phenotype: Converts *t* to wild type. RK3.

su(tu-bw): suppressor of tumor with brown

location: 3- (not located but probably in 3L).

origin: Naturally occurring allele.

discoverer: Glass, 1941.

references: Glass and Plaine, 1952, *Proc. Nat. Acad. Sci. USA* 38: 697-705.

Glass, 1954, *DIS* 28: 74.

Burnet and Sang, 1964, *Genetics* 49: 223-35, 599-610.

phenotype: Reduces incidence of melanotic masses in *tu-bw* homozygote from 85-100% in *su(tu-bw)/+* to 5-10% in *su(tu-bw)* homozygote. Suboptimal ratios of pentose nucleotides, cholesterol deficiency, or excess L-tryptophan in the larval diet, as well as X irradiation of embryos, increase incidence of melanotic masses in *tu-bw*; *su(tu-bw)* homozygote. Glass and colleagues attribute this to an effect on *su(tu-bw)*, whereas Burnet and Sang believe the reaction controlled by *tu-bw* is affected. Does not suppress *tu-48* (Burnett, 1966, *DIS* 41: 161). RK3.

Su(var): Suppressor of variegation

A series of dominant suppressors of variegation, mostly selected on the basis of their ability to suppress the mottling of *In(1)w^{md}*; where they have been tested, these mutations suppress variegated type position effects involving other loci as well. Two groups have performed extensive mutagenesis experiments, but there has apparently been no exchange of material nor any attempt to determine allelic relationships between the two samples of mutants. The Canadian group has designated its mutants as a *Su(var)200* series for second-chromosome mutations and a *Su(var)300* series for third-chromosome mutations. The German group uses *Su-var(3)1*, *etc.* to designate a series of suppressors on the third chromosome; we reconcile these terminologies but keep them separate by altering the second nomenclature to *Su(var)3-1*, *etc.* Despite the fact that some mutants from the two groups map to virtually identical positions, we choose to list them separately until appropriate complementation tests are performed. Most of these suppressors appear to be loss of function mutations and are frequently associated

with deficiencies; in addition variegation suppression is often produced by heterozygous deficiencies for their loci. Of these, several have been shown to enhance variegation when duplicated (Tartof, Bishop, Jones, Hobbs, and Locke, 1989, *Dev. Genet. (Amsterdam)* 10: 162-76).

locus	genetic location	cytological location	ref ^α	comments ^β
<i>Su(var)</i>	3-41.3	70	10,11	viable
<i>Su(var)2-1</i>	2-40.5	31A2-C1	1,4,14	butyrate sensitive; female sterile; 12 alleles
<i>Su(var)2-2</i>	2-79.8		14	lethal
<i>Su(var)2-3</i>	2-29.7		14	lethal
<i>Su(var)2-4</i> ^γ	2-7.6	23A3-D4	7,14	lethal
<i>Su(var)2-5</i>	2-31.1	28F2-29A1	14	lethal; 6 alleles; allelic to <i>Su(var)205</i> ; triplo-enhancer
<i>Su(var)2-7</i>	2-38.2		14	viable; fertile
<i>Su(var)2-8</i>	2-13.0	24F7-25A1	7,12,14	lethal
<i>Su(var)2-9</i>	2.44.8		14	female sterile
<i>Su(var)2-10</i>	2-57.6		8,14	
<i>Su(var)2-11</i>	2-45.7		14	female sterile
<i>Su(var)2-12</i>	2-37.0		14	lethal
<i>Su(var)2-13</i>	2-38.8	31A2-C1	14	viable; fertile
<i>Su(var)2-37</i>			8	
<i>Su(var)2-57</i>			8	
<i>Su(var)2-142</i>			8	
<i>Su(var)2-144</i>			8	
<i>Su(var)2-147</i>			8	
<i>Su(var)201</i>	2-40.1		9	lethal
<i>Su(var)205</i>	2-28.9	29A	2, 9	lethal; allelic to <i>Su(var)2-5</i> ; triplo-enhancer
<i>Su(var)206</i>	2-51.3		9	lethal
<i>Su(var)207</i>	2-32.0		9	lethal
<i>Su(var)209</i>	2-35.4		9	viable
<i>Su(var)210</i>	2-32.6		9	lethal
<i>Su(var)213</i>	2-32.9		9	lethal
<i>Su(var)215</i>	2-32.9		9	lethal
<i>Su(var)3-1</i>	3-31.4		5,14	viable; fertile; 6 alleles
<i>Su(var)3-2</i>	3-43.8		5,14	viable; fertile; 3 alleles
<i>Su(var)3-3</i>	3-46.6		5,14	semilethal; female sterile; butyrate sensitive; 26 alleles
<i>Su(var)3-4</i>	3-47.7	84D13-E2	5,14	lethal; 6 alleles
<i>Su(var)3-5</i>	3-{50}	86B	5	butyrate sensitive; semilethal; sterile; <i>T(2;3)58B;86B</i>
<i>Su(var)3-6</i>	3-51.1	87B4-7	5,6,14	lethal; 3 alleles
<i>Su(var)3-7</i>	3-{53}	87E4-5	3,5,6,14	lethal; <i>T(Y;3)87E1-2</i> ; triplo-enhancer
<i>Su(var)3-8</i>	3-53.5		5,14	lethal; 2 alleles
<i>Su(var)3-9</i>	3-56.4		5,14	viable; fertile; 4 alleles; triplo-enhancer
<i>Su(var)3-10</i>	3-61.7		5,14	lethal; 2 alleles
<i>Su(var)3-11</i>	3-76.4	94D-95A3	5	Minute
<i>Su(var)3-12</i>	3-100.2	100F3-5	5	Minute
<i>Su(var)3-13</i>	3-{53}	86F4-7	5,6,14	lethal
<i>Su(var)3-14</i>	3-{50}	86C-D	5,6,14	lethal
<i>Su(var)302</i>	3-62.4		9	viable
<i>Su(var)306</i>	3-61.1		9	viable
<i>Su(var)309</i>	3-56.4	88D	5, 9, 13	viable
<i>Su(var)314</i>	3-60.8		9	viable
<i>Su(var)316</i>	3-47.4		9	viable
<i>Su(var)319</i>	3-48.6		9	viable
<i>Su(var)320</i>	3-59.9		9	viable
<i>Su(var)323</i>	3-47.3		9	viable
<i>Su(var)325</i>	3-53.3		9	viable
<i>Su(var)326</i>	3-left end		9	lethal
<i>Su(var)327</i>	3-55.5		9	viable
<i>Su(var)328</i>	3-left end		9	lethal
<i>Su(var)330</i>	3-54.7		9	viable

^α 1 = Dorn, Heymann, Lindigkeit, and Reuter, 1986, *Chromosoma* 93: 398-403; 2 = James and Elgin, 1986, *Mol. Cell. Biol.* 6: 3862-72; 3 = Henikoff, 1979, *Genetics* 93: 105-15; 4 = Reuter, Dorn, and Hoffmann, 1982, *Mol. Gen. Genet.* 188: 480-85; 5 = Reuter, Dorn, Wustmann, Friede, and Rauh, 1986, *Mol. Gen. Genet.* 202: 481-87; 6 = Reuter, Gausz, Gyurkovics, Friede, Bang, Spierer, Hall, and Spierer, 1987, *Mol. Gen. Genet.* 210: 429-36; 7 = Reuter and Szidonya, 1983, *Chromosoma* 88: 277-85; 8 = Reuter and Wolff, 1981,

Mol. Gen. Genet. 182: 516-19; 9 = Sinclair, Mottus, and Grigliatti, 1983, *Mol. Gen. Genet.* 191: 326-33; 10 = Spofford, 1967, *Genetics* 57: 751-56; 11 = Spofford, 1969, *Genetics* 62: 555-71; 12 = Szidonya and Reuter, 1988, *Genet. Res.* 51: 197-208; 13 = Tartof, Bishop, Jones, Hobbs, and Locke, 1989, *Dev. Genet.* 10: 162-76; 14 = Wustmann, Szidonya, Taubert, and Reuter, 1989, *Mol. Gen. Genet.* 217: 520-27.

^β Phenotypes listed refer to homozygotes.
^γ Synonym: *Su(var)5401*.

other information: In addition to the tabulated mutations, deficiencies for 31C-32A, 38C6-D1, 47E3-48A6, 86C-D, and 99B-D and duplications for 26B, 33C-34A, 57D-58A, and 99C-D shown to suppress variegation of *In(1)w^{m4}* (Wustmann, Szidonya, Taubert, and Reuter, 1989, *Mol. Gen. Genet.* 217: 520-27).

phenotype: Reduces variegated mutant effect (sometimes completely) of *w*, *rst*, *fa*, *spl*, *nd*, and *dm* in *Dp(1;3)N²⁶⁴⁻⁵⁸*. Also reduces *w* variegation of *In(1)w^{m4}*, *rst* variegation of *In(1)rst³*, and *sc* variegation of *In(1)sc⁸*. Enhances *sc* variegation of *In(1)sc⁴* and *y* variegation of *In(1)y^{3P}*. Semidominant; heterozygote less suppressed than homozygote. Shows maternal effect; *Su(var)/+* offspring of *Su(var)/Su(var)* more nearly normal than *Su(var)/+* offspring of *Su(var)/+* mothers. Homozygote fertility slightly reduced. Viability excellent. RK2.

Su(var)2-1

phenotype: *Su(var)2-1/+* but not *Su(var)2-1/+* display nearly normal amounts of eye pigment in *In(1)w^{m4}* flies. Homozygous viable; females sterile; do not produce eggs. Survival reduced by the presence of the Y chromosome or by administration of sodium n-butyrate, an inhibitor of histone deacetylation (Reuter, Dorn, and Hoffmann, 1982, *Mol. Gen. Genet.* 188: 480-85). Exhibits significant hyperacetylation of histone H4 and increased accessibility of chromatin to endogenous nuclease, suggesting a defect in chromosome condensation (Dorn, Heymann, Lindigkeit, and Reuter, 1986, *Chromosoma* 93: 398-403).

alleles: Five alleles defining two complementation groups.

allele	origin	comments
<i>Su(var)2-1¹</i>	spont	noncomplementing
<i>Su(var)2-1²</i>	EMS	complements <i>Su(var)2-1³</i>
<i>Su(var)2-1³</i>	X ray	complements <i>Su(var)2-1²</i> and <i>Su(var)2-1⁴</i>
<i>Su(var)2-1⁴</i>	EMS	complements <i>Su(var)2-1³</i>
<i>Su(var)2-1⁵</i>	X ray	noncomplementing

Su(var)205

origin: Induced by ethyl methanesulfonate.

synonym: *E(var)29A*.

phenotype: Encodes a nonhistone chromosomal protein, HP1, which by immunofluorescent staining of polytene chromosomes with a monoclonal antibody, is localized to α , β , and intercalary heterochromatin as well as chromosome 4 (James and Elgin, 1986, *Mol. Cell. Biol.* 6: 3862-72).

cytology: Placed in 29A by *in situ* hybridization.

molecular biology: Sequence isolated from an expression library; hybridizes to a broad band in polytene subdivision 29A. In Northern blots the sequence recognizes an abundant 1.2 kb RNA; furthermore an aberrant transcript is seen in *Su(var)205*-bearing flies; attributable to missplicing caused by G → A transition at the first nucleotide

of the last intron (Eissenberg, James, Foster-Hartnett, Hartnett, Ngan, and Elgin, 1990, Proc. Nat. Acad. Sci. USA, 87: 9923-27). *Su(var)2-5*⁴, an allele of *Su(var)205*, shown to contain a nonsense mutation in the gene encoding HP1 (Eissenberg, Hartnett, and Reuter). The conceptual amino acid sequence specifies a 18,101 dalton hydrophilic polypeptide; basic-to-acidic amino acid ratio of 1.2; has a run of six glutamic acid residues in a row in a putative α -helical stretch. No apparent similarity to published amino acid sequences (James and Elgin).

other information: Duplications for the locus cause enhancement of variegation (Tartof, Bishop, Jones, Hobbs, and Locke, 1989, Dev. Genet. (Amsterdam) 10: 162-76).

Su(var)210

origin: Induced by ethyl methanesulfonate.

alleles: Three alleles by complementation testing of lethal mutations: *Su(var)210*¹, *Su(var)210*², and *Su(var)210*³ recovered as *Su(var)210*, *Su(var)214*, and *Su(var)216*, respectively.

Su(var)3-1

alleles: Six ethyl methanesulfonate-induced alleles, *Su(var)2-1*¹ to *Su(var)2-1*⁶.

Su(var)3-2

alleles: Three alleles: *Su(var)3-2*¹ and *Su(var)3-2*² induced by ethyl methanesulfonate; *Su(var)3-2*³ by X rays.

Su(var)3-3

location: 3-46.6 (on 3L based on its absence from induced *C(3R)RM* derivatives of *Su(var)3-3*-bearing chromosomes).

phenotype: Homozygotes strongly semilethal in males; weakly so in females; homozygous females sterile; produce no eggs. Homozygous males have spread-wing phenotype and reduced fertility. Survival sensitive to the presence of the Y chromosome as well as to the presence of 0.1 M butyrate in the medium.

alleles: Twenty-five alleles: *Su(var)3-3*¹³, *Su(var)3-3*¹⁸, and *Su(var)3-3*¹⁹ induced by X rays; the remainder by ethyl methanesulfonate.

Su(var)3-4

alleles: Six alleles: *Su(var)3-4*¹, and *Su(var)3-4*⁶ induced by X rays; the remainder by ethyl methanesulfonate.

Su(var)3-5

origin: X ray induced.

phenotype: Homozygotes strongly semilethal in males; weakly so in females; homozygous females sterile; produce no eggs. Homozygous males have reduced fertility. Survival sensitive to the presence of the Y chromosome as well as to the presence of 0.1 M butyrate in the medium.

Su(var)3-6

alleles: Three alleles: *Su(var)3-6*¹ ethyl methanesulfonate induced with normal cytology, *Su(var)3-6*² X ray induced, associated with *T(Y;3)15 = T(Y;3)87B5-7*, and *Su(var)3-6*³ X ray induced, associated with *Df(3R)Su-var6 = Df(3R)87B1-2;87D9-11*. *Su(var)3-6*¹ shows some reduction in viability in trans heterozygotes with some alleles of *l(3)87Bg*; in one case a clear maternal

effect is noted.

cytology: Placed in 87B5-10 based on its inclusion in *Df(3R)E079 = Df(3R)86F1-2;87B8-10* but not *Df(3R)TE45 = Df(3R)86F1-2;87B5-6*.

Su(var)3-7

origin: X ray induced.

cytology: Identified by virtue of the variegation suppression of *T(Y;3)409 = T(Y;3)87E2-5*. Located between *Ace* and *l(3)87Ee* as indicated by its survival in combination with *Df(3R)ry1607 = Df(3R)87D4-6;87E1-2* but not *Df(3R)N42 = Df(3R)?;87E3-4* or *Df(3R)N78 = Df(3R)?;87E3-4*, the latter two deficiencies themselves suppressing variegation. Corresponds to the dominant suppressor function identified by Henikoff (1979, Genetics 93: 105-15) by deficiency analysis. Duplication for the locus causes enhancement of variegation (Wustmann, Szidonya, Taubert, and Reuter, 1989, Mol. Gen. Genet. 217: 520-27).

alleles: Complements all lethals in the region except *Ace*⁴¹; *Ace*⁴¹ has a weak dominant variegation-suppression effect.

molecular biology: Genomic clone identified by transformation (Reuter, Giarre, Farah, Gausz, Spierer, and Spierer, 1990, Nature 344: 219-23). The sequence located in region 59-63 on chromosomal walk of Bender, Spierer, and Hogness (1983, J. Mol. Biol. 186: 17-33). cDNA clones isolated with transforming genomic clone sequenced; conceptual polypeptide has 932 amino acids with five widely separated zinc fingers, from 40-107 residues apart in the N-terminal half of the protein. The regions between and around the fingers are rich in acidic residues.

Su(var)3-8

phenotype: Homozygous lethal. One allele temperature sensitive; dies at 25° but not at 18°; homozygous flies raised at 18°, nevertheless, exhibit some suppression of position-effect variegation. Homozygous females sterile; lay eggs that fail to hatch.

alleles: Two ethyl methanesulfonate-induced alleles: *Su(var)3-8*² temperature sensitive.

Su(var)3-9

alleles: Thirteen alleles: *Su(var)3-9*⁷ induced by X rays; the remainder by ethyl methanesulfonate.

other information: Duplications for the locus cause enhancement of variegation (Tartof, Bishop, Jones, Hobbs, and Locke, 1989, Dev. Genet. (Amsterdam) 10: 162-76).

Su(var)3-11

origin: X ray induced.

cytology: Identified by virtue of its association with *Df(3R)M95A = Df(3R)94D-95A3*. *M(3)95A*² does not suppress variegation, however.

Su(var)3-12

origin: X ray induced.

cytology: Identified by virtue of its association with *Df(3R)MSu2*, which is deficient in the region 100F3-5.

Su(var)3-13

origin: Induced by ethyl methanesulfonate.

phenotype: Heterozygote shows weak suppression of variegation; homozygote lethal.

alleles: Fails to complement alleles of *l(3)86Fc*; however, *l(3)86Fc*⁴ and *l(3)86Fc*⁵ fail to suppress variegation.

cytology: Placed in 86F4-7 based on its location between *Df(3R)TE7 = Df(3R)86F1-2;86F4-5* and *Df(3R)E229 = Df(3R)86F6-7;87B1-2*.

Su(var)3-14

cytology: Gene inferred from the suppression of variegation seen in *Df(3R)cu = Df(3R)86C1-2;86D8* heterozygotes; no suppression observed in *Df(3R)M86D = Df(3R)86D1-2;86D4* heterozygotes.

su(ve): suppressor of veinlet

location: 3 -0.1 (0.1 unit to the left of *ru*).

origin: Spontaneous.

discoverer: Curry, 37a.

phenotype: At 19°, suppression of *ve* is complete except tip of L2 occasionally missing. At 25°, suppression only partial with some overlap into range of unsuppressed *ve*. At 19°, *su(ve)/+* partially suppresses *ve*. RK2.

cytology: Not included in *Df(3L)D = Df(3L)61E2-F1;61A4-6* from *T(Y;2;3)D*; therefore, probably located in 61A-E.

su(vg): suppressor of vestigial

location: 3-98.

origin: Spontaneous.

references: David, Javellot, and Tazet, 1970, DIS 45: 33.

phenotype: Temperature-sensitive recessive suppressor of *vg*. Suppression partial at 25°; wings and halteres intermediate in size between *vg* and +. Wings, halteres, and positions of scutellar bristles nearly normal in flies reared at 28° and above. Flies reared below 20° resemble *vg* alone. Slight effect of *su(vg)/f* on *vg* expression at 28°. *vg; su(vg)* lethal above 30° and below 13°; 30% mortality at 25°.

su(w^a): suppressor of apricot

location: 1-0.1.

references: Levis, O'Hare, and Rubin, 1984, Cell 38: 471-81.

Zachar, Davison, Garza, and Bingham, 1985, Genetics 111: 495-515.

Zachar, Garza, Chou, Goland, and Bingham, 1987, Mol. Cell. Biol. 7: 2498-2505.

Rutledge, Mortin, Schwarz, Thierry-Mieg, and Meselson, 1988, Genetics 119: 391-97.

phenotype: Produces a darker, brownish eye color in *w^a* flies. Suppresses *w^a*, *w^{am}*, *w^{a59k1}*, and *w^{a59k9}* but not *w^{a2}*, *w^{a3}*, *w^{a4}*, *w^{aR84h}*, *w^{sl0}* or any other *w* allele tested by Green (1959, Heredity 13: 303-15). Enhances *w^{a60a5}* and *w^M*. Seems to act on *w^a* by promoting transcription through the *copia* element that is inserted into the second intron in the same orientation as that of *w*; the level of a read-through transcript is elevated in *su(w^a) w^a* compared to *w^a*. *su(w^a)* is without effect on the phenotype of *w^{hd81b11}*, which has *copia* inserted at another position and in the opposite direction (Zachar *et al.*, 1985). *su(w^a)* also enhances the expression of a subset of alleles with *gypsy* inserts that are also enhanced by *su(s)* and suppressed by *su(f)* and *e(w^e)*; it also enhances *lz³⁴* and *lz^k* (Rutledge *et al.*).

alleles: No *bona fide* amorphic alleles reported; however, *Df(1)sta*, which is deficient for *su(w^a)* survives (Rayle

and Hoar, 1969, DIS 44: 94).

allele	origin	discoverer	synonym	ref ^α	comments
<i>su(w^a)</i> ¹	X ray	Schultz, 1941			
<i>*su(w^a)</i> ²	X ray	Schultz, 1944			possibly
<i>su(w^a)</i> ³	spont	Green	<i>su(w^a)^G</i>	1	<i>In(1)1D;1E</i> in <i>In(1)sc</i> ⁸ , <i>y</i> ^{31d} <i>w^a</i>
<i>su(w^a)</i> ⁴	EMS	Rayle		3	
<i>su(w^a)</i> ⁵	EMS	Rayle		3	
<i>su(w^a)</i> ⁶	EMS	Rayle		3	
<i>su(w^a)</i> ⁷	EMS		<i>su(w^a)^{A12}</i>	5	
<i>su(w^a)</i> ⁸	EMS		<i>su(w^a)^{D5}</i>	5	
<i>su(w^a)</i> ⁹	EMS		<i>su(w^a)^{DM17}</i>	5	
<i>su(w^a)</i> ¹⁰	EMS		<i>su(w^a)^{SD10}</i>	5	
<i>su(w^a)</i> ¹¹	X ray		<i>su(w^a)^{γ44}</i>	5	
<i>su(w^a)</i> ¹²	X ray		<i>su(w^a)^{γ107}</i>	5	
<i>su(w^a)</i> ¹³	P		<i>su(w^a)^{hd7}</i>	5	
<i>su(w^a)</i> ¹⁴	EMS		<i>su(w^a)^{hdM8}</i>	5	
<i>su(w^a)</i> ¹⁵		Birchler	<i>su(w^a)⁸⁴ⁱ</i>	2	
<i>su(w^a)</i> ¹⁶		Rendahl	<i>su(w^a)^{85g}</i>	2	
<i>su(w^a)</i> ¹⁷		Jaffe	<i>su(w^a)^{BJ}</i>	2	
<i>su(w^a)</i> ¹⁸	EMS		<i>su(w^a)²⁰</i>	4	

^α 1 = Green, 1954, DIS 28: 74; 2 = Mount, Green, Rubin, 1988, Genetics 118: 221-34; 3 = Rayle, 1972, Genetics 71: s50; 4 = Rutledge, Mortin, Schwarz, Thierry-Mieg, and Meselson, 1988, Genetics 119: 391-97; 5 = Zachar, Garza, Chou, Goland, and Bingham, 1987, Mol. Cell. Biol. 7: 2498-2505.

cytology: Placed in 1E1-2 by Rayle (1972, Genetics 71: s50); transposed to chromosome 3 by *Tp(1;3)sta = Tp(1;3)1D3-1E1;2A;89B21-C4*.

molecular biology: Gene isolated by transposon tagging. Identity of the *su(w^a)* transcription unit confirmed by transformation with a 6.2 kb fragment. Three overlapping mature transcripts of 3.5, 4.6, and 5.2 kb produced from the 6.2 kb region, the two larger ones drastically reduced in *su(w^a)* animals. The transcripts produced at approximately the same level at all developmental stages, in all body parts, and in cultured *Drosophila* cells (Zachar *et al.*, 1987).

Su(w^a)B1

location: 1- (to the right of *w*).

origin: Recovered from hybrid-dysgenic cross.

references: Birchler, 1984, Genetics 107: s12.

phenotype: Heterozygotes, homozygotes, and hemizygotes display increased levels of pigment in *w^a* eyes.

Su(w^a)B2

location: 2- (Just proximal to *c*).

origin: Recovered from hybrid dysgenic cross.

references: Birchler, 1984, Genetics 107: s12.

phenotype: Homozygous lethal. In heterozygotes causes an approximately two-fold increase in pigmentation of *w* alleles with lesions in the coding region. Without effect on four alleles that are not dosage compensated or on *w^{sp}* alleles, whose lesions are 5' to the *w* transcription unit. Duplications for the locus reduce pigmentation of *w* alleles.

Su(w^{ch}): Suppressor of cherry

location: 2-4.0.

discoverer: Lewis.

references: Rasmusen, 1970, Hereditas 65: 83-96.

Rasmusen and Ljung, 1973, Hereditas 73: 71-84.

phenotype: Homozygous lethal; in heterozygotes leads to increased levels of eye pigmentation in *w^{ch}*, *w^e*, and *w^a*; heterozygotes exhibit increased courtship activity leading

to mating advantage over wild type (Connally, Burnet, and Sewell, 1969, *Evolution* 23: 548-59).

su(w^h): suppressor of white honey

location: 3- (not mapped).

references: Lee, 1972, *DIS* 48: 18-19.

phenotype: In homozygous *su(w^h)* flies pigmentation of *w^h* increased to a bright cherry color; *w^a* and *w^{a4}* show slight suppression. Without effect on *w*, *w^a*, *w^{aM}*, *w^{bl}*, *w^c*, *w^{cf}*, *w^{col}*, *w^e*, *wⁱ*, *w^{sat}*, and *w^t*.

su(w^{sp}): suppressor of white-spotted

location: 1-0.16 (based on 19 *y-w* recombinants).

discoverer: Gelbart.

references: Chapman and Bingham, 1985, *DIS* 61: 48-49.

Davison, Chapman, Wedeen, and Bingham, 1985, *Genetics* 110: 479-94.

phenotype: Results in nearly wild-type pigmentation of *w^{sp1}*, *w^{sp2}*, *w^{sp3}*, and *w^{sp4}*, but not *w^{sp81d5}*. No phenotypic effect seen on eight other tested *w* alleles. The lesions of the spotted alleles map 500 to 1000 base pairs 5' to the *w* transcription unit and are postulated to affect an enhancer sequence. The normal allele of *su(w^{sp})* is postulated to encode a repressor of this enhancing function; the combination of the repressor and defective enhancer produces the spotted phenotype; the mutant allele of *su(w^{sp})*, lacking repressor function, results in the derepression of the defective enhancers at the time of eye-pigment deposition, leading to increased pigmentation. *w^{sp81d5}*, a molecular deletion extending more 3' than the other *w^{sp}* alleles is postulated to be insensitive to the presence or absence of repression owing to loss of the site of interaction between the *su(w^{sp})* gene product and the *w* locus. Adult transcription of all *w* alleles, including *w⁺*, markedly increased by *su(w^{sp})*, apparently after pigment deposition, since with the exception of *w^{sp}* alleles, eye color does not respond to *su(w^{sp})*. The latter observation implies a second site of repressor binding specific to adult transcription.

Su-x4: see *Su(sph)*

***Su(y^{3P}): Suppressor of yellow-3 of Patterson**

location: 3-90.

origin: X ray induced.

discoverer: Parker, 48h.

synonym: *su-y^{31e}*.

references: 1950, *DIS* 24: 62.

phenotype: *Su(y^{3P})/+* suppresses *y^{3P}* to about normal color, except that wings remain yellowish. *y^{3P}*; *Su(y^{3P})/Su(y^{3P})* is darker than wild type but wings remain yellow. May be suppression of variegation since extra Y chromosomes also suppress *y^{3P}*. No effect on *y*, *y²*, *y^{2S}*, *y^{3d}*, *y⁴*, *y^{35a}*, or *y^{id}*. Homozygote has low viability and fertility; occasionally, wings held out from body. RK2.

Su(z)2: Suppressor 2 of zeste

location: 2-67 (distal to *vg*; also distal to *Psc*).

references: Kalisch and Rasmuson, 1974, *Hereditas* 78: 97-104.

Persson, 1976, *Hereditas* 82: 111-20.

Wu, Jones, Lasko, and Gelbart, 1989, *Mol. Gen. Genet.* 218: 559-64.

Adler, Charlton, and Brunk, 1989, *Dev. Genet. (Amsterdam)* 10: 249-60.

phenotype: Dominant suppressor of the yellow eye color associated with the *z¹* mutation. Can be detected in homozygous *z¹* females and in males. Lethal in homozygous or hemizygous condition and in *trans*-allelic heterozygotes. Viability reduced in *trans* heterozygotes in some *Psc/Su(z)2* combinations, lethal in others. *Psc* and *Su(z)2* are both members of the *Su(z)2* complex.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>Su(z)2¹</i>	EMS	Rasmuson	<i>Su(z)2²</i>	3, 4, 5, 6	zeste→red eyes
<i>Su(z)2^{1.b7}</i>	X ray	Wu		1	revertant of <i>Su(z)2¹</i> ; 2 kb deletion at 5' end of transcription unit
<i>Su(z)2^{1.b8}</i>	X ray	Wu		1	revertant of <i>Su(z)2¹</i>
<i>Su(z)2⁴</i>	X ray	Gelbart	<i>Su(z)45</i>	2, 4, 5, 6	zeste→orange-maroon
<i>Su(z)2⁵</i>	γ ray	Wu		5, 6	eyes zeste→yellow-orange eyes

^α 1 = Adler, Charlton, and Brunk, 1989, *Dev. Genet.* 10: 249-60; 2 = Gelbart, 1971, Doctoral dissertation, University of Wisconsin, Madison; 3 = Kalisch and Rasmuson, 1974, *Hereditas* 78: 97-104; 4 = Persson, 1976, *Hereditas* 82: 111-20; 5 = Wu, 1984, Doctoral dissertation, Harvard University, Cambridge; 6 = Wu, Jones, Lasko, and Gelbart, 1989, *Mol. Gen. Genet.* 218: 559-64.

cytology: Placed in 49E-F on the basis of its inclusion in *Df(2R)vg-B = Df(2R)49D3-4:49F15-50A2* and its exclusion from *Df(2R)vg-C = Df(2R)49B2-3:49E7-F1* and *Df(2R)vg-D = Df(2R)49C1-2:49E4-5* (Wu).

molecular biology: Reported to be distal to and transcribed from left to right, divergently from *Psc* (Adler *et al.*).

Su(z)301: see *E(z)*

Su(z)302: see *Scm*

sub: subito (T. Schüpbach)

location: 2-86.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1989, *Genetics* 121: 101-17.

phenotype: Maternal-effect lethal or female sterile; homozygous females lay eggs which show no visible signs of development when observed under transmitted light in a stereo microscope. These eggs are defective in fertilization or very early embryonic development.

alleles: Two alleles, *sub¹* and *sub²*, isolated as *PF* and *HM*.

cytology: Placed in 55A-F, since uncovered by *Df(2R)PC4 = Df(2R)55A:55F*.

other information: *sub²* females form rare syncytial-blastoderm embryos.

Succinic dehydrogenase: see Sdh

Sucr: Sucrase

location: 2- {37}.

references: Oliver and Williamson, 1979, *Biochem. Genet.* 17: 987-907.

phenotype: Thought to be the structural gene for sucrase [EC 3.2.1.26], a 100 kilodalton protein.

alleles: Thought to exist in two forms by isoelectric focusing.

cytology: Placed in 31C-F by changes in enzyme levels

associated with segmental aneuploids.

sunburst: see *p^{snb}*

***sup: superwith**

location: 3- (not located).

discoverer: Morgan, 10k.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 35.

phenotype: Trident pattern on thorax dark. RK3.

supact: suppressor activated

location: 1-0.0 (near *mul*; separable from *sc*).

origin: Spontaneous.

references: Lee, 1970, Austr. J. Biol. Sci. 23: 645-55.

phenotype: Recessive; bristle and eye abnormalities produced by over 95% of females that are homozygous for *su(Hw)*². Abnormalities include irregular pattern of missing bristles (scutellars, dorsocentrals, vertical, post-verticals, and ocellars) and small granulated eyes. Restricted to females; not influenced by gene dosage or the presence of the Y chromosome, since neither XY nor X0 males homozygous for *su(Hw)*² are affected, nor are males with two doses of the *supact* region at the tip of the X. No effect of *supact* on flies raised at 30°; females but not males raised at 18° show enhanced eye effect, but no bristle effect.

super sex combs: see *sxc*

Superabdominal: see *Sab* under *BXC*

Super-Bar: see *B^{S3i}*

superwith: see *sup*

suppressor: see *su()*

Suppressor: see *Su()*

suppressor activated: see *supact*

Surf wings: see *Srf*

***sv: shaven**

location: 4-3.0 [diplo-4 triploids (Sturtevant, 1951, Proc. Nat. Acad. Sci. USA 37: 405-7)].

origin: Spontaneous.

discoverer: Bridges, 20k14.

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 235 (fig.).

Bridges, 1935, Biol. Zh. 4: 401-20.

phenotype: Bristles reduced, somewhat variably. Trichogen irregularly displaced and usually partly converted to socket (Lees and Waddington, 1942, DIS 16: 70). *sv/sv/sv* triplo-4 nearly normal. *sv* haplo-4 extreme shaven (Schultz, 1935, Am. Naturalist 69: 30-54). Expression depends on temperature: excellent at 19°, overlaps wild type at 25°, and entirely wild type at 30°. RK2.

cytology: Placed in region between 102E2 and 102F10 on the basis of its inclusion in *Df(4)11 = Df(4)102E2-10;102F2-10*.

*sv*²: see *svⁿ*

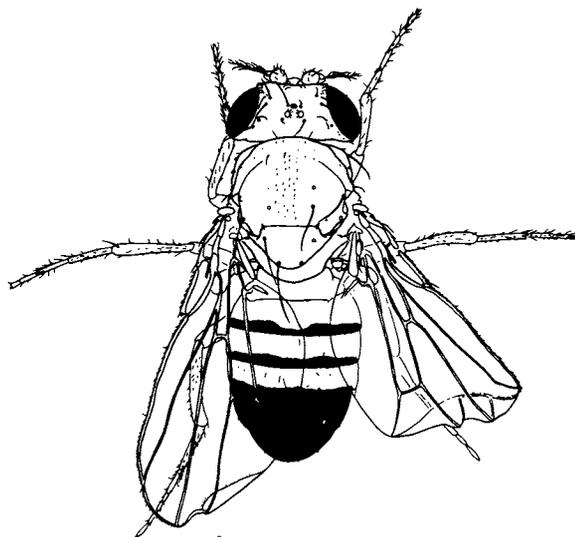
sv^{35a}

discoverer: Ives, 35a18.

references: 1935, DIS 4: 11.

phenotype: Resembles *svⁿ* more than *sv*. Bristles fre-

quently reduced to stumps. RK2.



sv^{de}: shaven-depilate

Edith M. Wallace, unpublished.

sv^{de}: shaven-depilate

origin: Spontaneous.

discoverer: E.M. Wallace, 37a24.

phenotype: More extreme than *svⁿ*. Thorax denuded over large areas. Phenotype more severe than *H*²; bristleless sockets found on adult integumentary derivatives of all imaginal discs; nearly 100% penetrance on thorax. Shafts, where present, often bent, twisted, or forked; up to 97% of bristle organs on wing costa and distal leg segments fail to produce normal shafts. Bracts present when bristle normally formed; otherwise bracts missing (Tobler, Rothenbuhler, and Nöthiger, 1973, Experientia 29: 370-71). Both sexes sterile. RK2.

svⁿ: shaven-naked

discoverer: Mohr, 31j13.

synonym: *sv*².

references: 1933, Hereditas 17: 317-22 (fig.).

phenotype: Extremely short bristles. Viability excellent. Trichogen irregularly displaced, becoming more or less converted into tormogen [Lees and Waddington, 1943, Proc. Roy. Soc. (London), Ser. B, 131: 87-110 (fig.)]. Polarity of microchaetae in vicinity of double sockets disrupted such that they form a whorl around socket [Toney and Thompson, 1980, Experientia 36: 644-45 (fig.)]. In triplo-4 *svⁿ/svⁿ/svⁿ*, the phenotype is more nearly normal than in diplo-4. RK1.

other information: Selective advantage for triplo-4 in stocks of *svⁿ* results in accumulation.

svb: shaven baby

location: 1-9.8.

references: Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307.

Gergen and Wieschaus, 1985, Dev. Biol. 109: 321-35 (fig.).

phenotype: Homozygous lethal. Many fewer denticles than wild type; remaining denticles small and with

characteristic morphology. Denticles arranged in belts in the abdominal segments; absent from thoracic segments. Cell autonomous in mosaics; useful in studying embryonic mosaics.

alleles:

allele	origin	synonym	ref ^α	comments
svb¹	EMS	<i>svb^{YD39}</i>	3	polyphasic, <i>ovo</i> ⁻
svb²	EMS	<i>svb^{YP17b}</i>	2, 3	polyphasic, <i>ovo</i> ⁺ , no maternal effect
svb³	EMS	<i>svb^{EH587}</i>	1	polyphasic, <i>ovo</i> ⁻ <i>hnt</i> ⁺

^α 1 = Eberl and Hilliker, 1988, Genetics 118: 109-20; 2 = Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-52; 3 = Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307.

cytology: Placed in 4E1-2 along with *ovo* based on the simultaneous loss of both functions in many *ovo*^D reversions. Included in *Df(1)bi-DL5 = Df(1)3C7-12;4E1-2* and *Df(1)bi-D2 = Df(1)4B6-C1;4D7-E1*, both of which retain *ovo*⁺ (Oliver, Perrimon, and Mahowald, Genes Dev. 1: 913-23).

svp: seven up (Y. Hiromi)

location: 3- {52}.

references: Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowitz, 1981, Genetics 98: 775-89.

Mlodzik, Hiromi, Weber, Goodman, and Rubin, 1990, Cell 60: 211-24.

phenotype: Originally identified as recessive lethal mutations; independently identified by an enhancer-trap screen to be a gene expressed in a subset of neuroblasts in the embryonic CNS and also in a subset of photoreceptor-cell precursors in the developing compound eye. Mutant alleles die as embryos with normal cuticular morphology, but have alterations in numbers of *eve*-positive neurons in some neuroblast lineages. *svp*⁻ clones in the eye produce abnormal ommatidia where photoreceptors R1, R3, R4, and R6 are transformed toward another photoreceptor R7. In addition, *svp*⁻ ommatidia usually contain one to two extra photoreceptor cells. Mosaic studies show that transformation towards R7 is due to cell autonomous requirement in R1, R3, R4, and R6, whereas production of extra cell(s) is a secondary phenotype caused by lack of *svp*⁺ function in other cells, most likely in R3 and R4. Formation of some, but not all, R7-like cells in *svp*⁻ ommatidia is dependent on *sev*⁺ function.

alleles: Of eight ethyl methanesulfonate-induced alleles listed by Gausz *et al.*, only two are now existent (*svp*¹ and *svp*²); *svp*³ has an enhancer trap *P*-element insertion at the *svp* locus and expresses β-galactosidase in a pattern resembling the *svp* transcription pattern.

allele	synonym	comments
svp¹	<i>l(3)ck16^{e22}</i>	embryonic lethal
svp²	<i>l(3)ck16^{e300}</i>	embryonic lethal
svp³	<i>svp^{H162}</i>	embryonic lethal

cytology: Placed in 87B4-6 based on its inclusion in the region of overlap of *Df(3R)T45 = Df(3R)86E;87B5-6* and *Df(3R)kar-H10 = Df(3R)87B4;87D7-8*.

molecular biology: Walking from the enhancer trap *P*-element insert, a transcription unit was identified whose expression pattern is indistinguishable from the β-galactosidase expression pattern of the *P*-insertion allele. The conceptual amino-acid sequence from a *svp* cDNA

clone shows that *svp* is a member of the steroid receptor gene superfamily, and is likely to be the *Drosophila* homologue of the human transcription factor COUP.

svr: silver

location: 1-0.0.

discoverer: Bridges, 23g23.

synonym: *slv*.

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 235.

Morgan, 1940, DIS 13: 51.

phenotype: Color of legs, wings, veins, and integument pale and silvery. Bristles and trident pattern on thorax dark. Tyrosinase formed in adult (Horowitz). Wings of all males and some females pointed. Viability fair. Larval mouth parts normal in color. RK2.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
svr¹		Bridges, 23g23			
svr²	EMS	Lim	<i>svr⁰¹⁶</i>		
svr³	EMS	Lim	<i>svr¹²¹</i>		
svr⁴	MMS	Lim	<i>svr^{M9}</i>		
*svr⁵	MMS	Lim	<i>svr^{M14}</i>		
*svr⁶	X ray	Lefevre	<i>l(1)C235</i>	6	
svr⁷	X ray	Lefevre	<i>l(1)RA61</i>	6	
svr⁸	EMS	Lefevre	<i>l(1)DC765</i>	7, 8	larval; no maternal effect
svr⁹	EMS	White			
svr¹⁰	EMS	White			
svr¹¹	EMS	White			
svr¹²	EMS	White			
svr¹³	EMS	White			
svr¹⁴	EMS	White			
svr¹⁵	EMS	White			
svr¹⁶	EMS	White			
svr¹⁷	ENU	Voelker	<i>l(1)A41</i>		
svr¹⁸	ENU	Voelker	<i>l(1)A71</i>		
svr¹⁹	ENU	Voelker	<i>l(1)A106</i>		
svr²⁰	ENU	Voelker	<i>l(1)A122</i>		
*svr^{poi}	spont	Goldschmidt, 1934	<i>poi</i>	1, 2, 3, 5	pointed wings
*svr^{poi-bl}	spont	Goldschmidt		1, 3	pointed blistered wings
*svr^{poi-Ca}	spont	Goldschmidt		1, 3	pointed from Canton S
svr^{poi-dish}	spont	Goldschmidt		1, 3, 5	dishevelled microchaetae
*svr^{poi-h}	spont	Goldschmidt		1	
*svr^{poi-ll}	spont	Goldschmidt		1, 4	lanceolate wings
*svr^{poi-s}	spont	Goldschmidt		1, 3	wing tips
*svr^{poi-si}	spont	Goldschmidt		1, 3	pointed, narrow
*svr^{poi-sq}	spont	Goldschmidt		1, 3	pointed, blistered wings
*svr^{Poi}	spont	Goldschmidt		1, 4	pointed wings, dominant
*svr^{Poi-s}	spont	Goldschmidt		1, 4	semidominant
svr^{ts1}	EMS	White			
svr^{ts2}	ENU	Voelker	<i>l(1)A95</i>		
svr^{ts3}	ENU	Voelker	<i>l(1)A104</i>		

^α 1 = CP 627; 2 = Goldschmidt, 1944, DIS 18: 42; 3 = Goldschmidt, 1945, Univ. Calif. (Berkeley) Publ. Zool. 49: 291-550; 4 = Goldschmidt, 1947, J. Exp. Zool. 104: 197-222; 5 = Goldschmidt and Hanah, 1944, Proc. Nat. Acad. Sci. USA 30: 299-301; 6 = Lefevre, 1981, Genetics 99: 461-80; 7 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 8 = Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-52.

cytology: Locus placed at 1B5-6 (Demerec, Kaufmann, Fano, Sutton, and Sansome, 1942, Year Book - Carnegie

Inst. Washington 41: 191).

other information: Pointed alleles studied by Goldschmidt enhanced by *a^{ba}*, *svr^{poi}* and *svr^{poi-dish}* reported to suppress *s* and *sp*.

***svs: shortened veins**

location: 1-24.6.

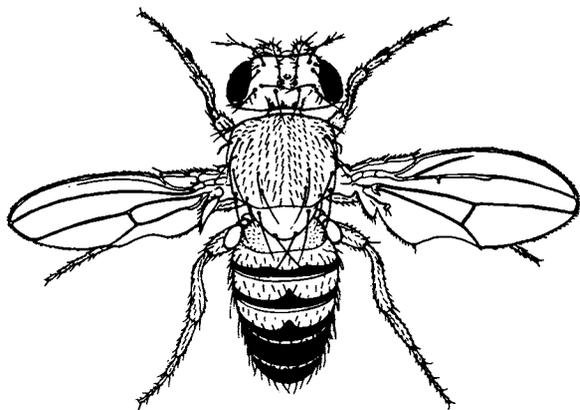
origin: Induced by ethyl methanesulfonate (CB. 1528).

discoverer: Fahmy, 1956.

synonym: *shv* (preoccupied).

references: 1959, DIS 33: 90.

phenotype: Wings highly abnormal, varying from small stubs to almost full size with inner margin cut away. Vein L4 often shortened and posterior crossvein absent. Eyes small and deformed. Male fertile; viability about 50% wild type. Female sterile. RK2.



sw: short wing

From Eker, 1935, J. Genet. 30: 357-68.

sw: short wing

location: 1-64.0 (to the left of *mel*).

discoverer: Eker, 32a12.

references: 1935, J. Genet. 30: 357-68 (fig.).

1939, J. Genet. 38: 201-27.

phenotype: Above 23°, most *sw¹* flies have spread and incised wings with irregular veins; eyes reduced and roughened. Male expression more extreme than female. 15% of males eclose at 25°; show wing and eye abnormalities (Schalet, 1969, DIS 43: 128); above 31°, *sw* is lethal. At 17°, most flies are wild type; at 14°, all are wild type. *+Df(1)mal10* and *+sw* exhibit short-wing phenotype in presence of *Rp11215^{Ubl}* (Mortin and Lefevre, 1981, Chromosoma 82: 237-47). RK2 at 28°.

alleles: Either this is a complex locus with *sw⁴* being the only noncomplementing allele or *sw⁴* is a double mutant and there are two loci. Numerous lethal alleles at this locus recovered from line containing a *P* element in 19C (Simmons, Raymond, Johnson, and Fahey, 1984, Genetics 106: 85-94).

allele	origin	discoverer	synonym	ref ^α	comments
<i>sw¹</i>		Eker, 32a12		1, 2	
<i>sw²</i>	X ray	Lefevre	<i>l(1)GA80</i>	4	lethal with <i>sw¹</i>
<i>sw³</i>	X ray	Lefevre	<i>l(1)HC274</i>	4	complements <i>sw¹</i>
<i>sw⁴</i>	X ray	Lefevre	<i>l(1)HC326</i>	4	noncomplementing
<i>sw⁵</i>	EMS	Lefevre	<i>l(1)DA648</i>	5	lethal with <i>sw¹</i>
<i>sw⁶</i>	EMS	Lefevre	<i>l(1)VA172</i>	5	lethal with <i>sw¹</i>
<i>sw⁷</i>	EMS	Lefevre	<i>l(1)VA214</i>	5	lethal with <i>sw¹</i>
<i>sw⁸</i>	EMS	Baldwin	<i>l(1)LB11</i>	6	

allele	origin	discoverer	synonym	ref ^α	comments
<i>sw⁹</i>	EMS	Lefevre	<i>l(1)EF401</i>	5	complements <i>sw¹</i>
<i>sw¹⁰</i>	EMS	Lefevre	<i>l(1)VA125</i>	5	complements <i>sw¹</i>
<i>sw¹¹</i>	EMS	Lefevre	<i>l(1)VA138</i>	5	complements <i>sw¹</i>
<i>sw¹²</i>	EMS	Lefevre	<i>l(1)VA271</i>	5	complements <i>sw¹</i>
<i>sw¹³</i>	EMS	Lefevre	<i>l(1)VA310</i>	5	complements <i>sw¹</i>
<i>sw¹⁴</i>	neutron	Muñoz	<i>l(1)17-234</i>	6	<i>sw¹⁴/sw¹</i> wild type at 25°; <i>sw</i> at 29° early pupal lethal with <i>sw¹</i>
<i>sw¹⁵</i>	spont	Schalet	<i>sw¹-SIM</i>		
<i>*sw¹⁶</i>	spont	Lee	"short wing"	3	wings with curled edges, broad tips, broken veins; female sterile; allelism tentative

^α 1 = Eker, 1935, J. Genet. 30: 357-68 (fig.); 2 = Eker, 1939, J. Genet. 38: 210-27; 3 = Lee, 1972, DIS 48: 18; 4 = Lefevre, 1981, Genetics 99: 461-80; 5 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 6 = Schalet and Lefevre, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.), Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902.

cytology: Placed in 19B3-C4 based on its inclusion in *Df(1)T2-4A = Df(1)19B3;19C4*.

sw^y: see *stw²*

swa: see *crm^{swa}*

swa: swallow

location: 1-15.9 (between *rux* and *shf*).

synonym: *sww*.

references: Zalokar, Audit, and Erk, 1975, Dev. Biol. 47: 419-32.

Fronhöfer and Nüsslein-Volhard, 1987, Genes Dev. 1: 880-90.

phenotype: Maternal-effect lethal; homozygous females sterile. Some blastoderms irregularly populated, with some areas on surface devoid of nuclei, others display asynchronous nuclear divisions and nuclei of non uniform size. Gastrulation occurs before full complement of nuclei achieved; mitosis continues into gastrulation. Developmental arrest at time of first muscular activity. In *swa²* development normal but abnormal larval mouth parts prevent feeding. Mosaic studies with *swa¹* indicate that the gene functions in germ line (Perrimon and Gans, 1981, Dev. Biol. 100: 365-73). Morphology of developing embryos shows variable temperature-sensitive failure of head-segment development and abdominal segmentation defects. Anterior defects more severe at 29°, and posterior ones cold sensitive, strongly enhanced at 18°. *ftz* expression pattern abnormal; number of stripes varies from three (PS 2, 4, & 6) at 18° to seven at 29°. Unlike wild-type cytoplasm, anterior cytoplasm from embryos of *swa* mothers ineffective in rescuing *bcd* phenotype; also embryos from *swa* females resistant to the *bcd*-phenocopying effects of removing anterior cytoplasm (Fronhöfer and Nüsslein-Volhard).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>swa¹</i>	EMS	Gans	<i>fs(1)1497</i>	1, 5	strong allele
<i>swa²</i>	EMS	Gans	<i>fs(1)1502</i>	1, 5	weak allele
<i>swa³</i>	EMS	Mohler	<i>fs(1)11-999</i>	2, 3	strong allele
<i>swa⁴</i>	EMS	Mohler	<i>fs(1)13C82</i>	2, 3	strong allele
<i>swa⁵</i>	EMS	Stephenson	<i>fs(1)T573</i>	3	weak allele
<i>swa⁶</i>	EMS		<i>fs(1)384</i>	4	

^α 1 = Gans, Audit, and Masson, 1975, *Genetics* 81: 683-704; 2 = Mohler, 1977, *Genetics* 85: 259-72; 3 = Mohler and Carroll, 1984, *DIS* 60: 236-41; 4 = Stephenson and Mahowald, 1987, *Dev. Biol.* 124: 1-8; 5 = Zalokar, Audit, and Erk, 1975, *Dev. Biol.* 47: 419-32.

cytology: Placed in 5E6-7 based on *in situ* hybridization to a normal X chromosome but not to *Df(1)JF5 = Df(1)5E5-6;5E7-8* (Stephenson and Mahowald).

molecular biology: Gene cloned and restriction mapped; identity confirmed by rescue of *swa* by transformation. Detects a 2.1 kb transcript on Northern blots whose expression is restricted to the nurse cells. The message is uniformly distributed in the mature oocyte, becoming more concentrated peripherally at blastoderm formation, and disappearing by the time of gastrulation. The anterior localization of *bcd*⁺ product disrupted in the oocytes of homozygous *swa* females (Stephenson, Chao, and Fackenthal, 1988, *Genes Dev.* 2: 1655-65).

swarthy: see *swy*

swb: see *fa*^{swb} listed under *N*

swi: see *hfw*

swiss cheese: see *sws*

swollen antenna: see *crm*^{swa}

sws: swiss cheese (J.C. Hall)

location: 1-22.

origin: Induced by ethyl methanesulfonate.

discoverer: Heisenberg.

phenotype: Isolated in anatomical screen (*cf.* Heisenberg and Böhl, 1979, *Z. Naturforsch.* 34: 143-47); many Swiss-cheese-like holes seen in brain sections of adults (similar to phenotype observed in dying *drd* flies).

alleles: Three alleles, *sws*¹, *sws*², and *sws*³, with isolation numbers *HK151*, *KS43*, and *RH7*, respectively.

other information: Phenotype has become less extreme, apparently due to accumulation of modifiers (Heisenberg).

***swy: swarthy**

location: 1-42.5.

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1956.

references: 1959, *DIS* 33: 92.

phenotype: Body color slightly dark; darkened scutellum particularly noticeable. Eyes brownish (best detected immediately after eclosion) and occasionally misshapen. *swy/s* is wild type. Viability about 50% wild type. Both sexes fertile. RK2.

sx: sexcombless

location: 1- (rearrangement).

origin: X ray induced.

discoverer: Muller, 261.

references: Mukherjee, 1965, *Genetics* 51: 285-304 (fig.). Reinhard and Sánchez, 1982, *Wilhelm Roux's Arch. Dev. Biol.* 191: 264-69 (fig.).

phenotype: Number of teeth in primary sex comb reduced from the normal 10 to 1. Bristles intermediate between normal bristles and sex-comb teeth also appear in sex-comb area. Bristle pattern of *sx* male basitarsus feminized in other respects. *sx/+* reduces the mean number of

sex-comb teeth in *tra/tra* female from 11.37 to 3.7. Sex-comb development autonomous in mosaic from either chromosome loss or somatic crossing over in *tra/tra* female (Mukherjee and Stern, 1965, *Z. Indukt. Abstamm. Vererbungsl.* 96: 36-48). Reduces number of teeth in secondary sex comb of *en/en* male and in primary sex comb of *ey*^{D/+} male. Homozygous females generated from homozygous *mei-332* mothers have normal leg chaetotaxy (Reinhard and Sánchez). Male sterile owing to imperfect development of internal duct system; testes often remain unattached to ducts, and are therefore ellipsoidal, but contain fully developed sperm (Stern, 1941, *J. Exp. Zool.* 87: 113-58). External genitalia also greatly modified. Size, shape, and arrangement of teeth on clasper varies; occasionally more than one penial apparatus (Mukherjee). Structures frequently missing and usually displays bilateral asymmetry; also extra and missing bristles. Homozygous females characteristically lack vulva; thorn bristles on vaginal plate often present as a double row; internal genitalia absent; ovaries small but normal in shape; anal plates reduced or absent (Reinhard and Sánchez). RK2A.

cytology: Associated with *In(1)sx = In(1)11D4-6;11E2-6;14B8-9;15E2-4* (Mukherjee, 1963, *DIS* 38: 62).

sxc: super sex combs (P.W. Ingham)

location: 2-55.3 (0.8 unit to the right of *pr*).

references: Ingham, 1984, *Cell* 37: 815-23.

phenotype: Homozygous adults exhibit homeotic transformation of antenna to first leg, of second and third legs to first leg, of wing to haltere, and of abdominal segments A1, A4, and A5 to A2, A5, and A6, respectively. Homozygotes derived from homozygous female germ line exhibit transformation of larval thoracic and abdominal segments toward A8.

alleles:

allele	origin	discoverer	comments
sxc ¹	EMS	Ingham	
sxc ²	X ray	Ingham	<i>T(2;3)41C1-2;98C;99A</i>
sxc ³	EMS	Ingham	
sxc ⁴	EMS	Ingham	
sxc ⁵	EMS	Ingham	

cytology: Placed in 41C1-2 based on the breakpoint of *T(2;3)sxc*² and on its inclusion in *Df(2R)M41A4*, which, although cytologically invisible, is deficient for *M(2)41A*, at the base of 2R.

Sxl: Sex lethal (T.W. Cline)

location: 1-19.2.

synonym: *F1: Female lethal; Su(da)*.

references: Muller and Zimmering, 1960, *Genetics* 45: 1001-02.

Zimmering and Muller, 1961, *DIS* 35: 103-04.

Marshall and Whittle, 1978, *Genet. Res.* 32: 103-11.

Cline, 1978, *Genetics* 90: 683-97.

1979a, *Dev. Biol.* 72: 266-75.

1979b, *Genetics* 93: 681-701.

1980, *Genetics* 96: 903-26.

Lucchesi and Skripsky, 1981, *Chromosoma* 88: 217-27.

Skripsky and Lucchesi, 1982, *Dev. Biol.* 94: 153-62.

Uenoyama, 1982, *Jpn. J. Genet.* 59: 335-48.

Sánchez and Nöthiger, 1982, *Wilhelm Roux's Arch.* 191: 211-14.

Cline, 1983, *Dev. Biol.* 95: 260-74.

- Sánchez and Nöthiger, 1983, EMBO J. 2: 485-91.
 Cline, 1984, Genetics 107: 231-77.
 Maine, Salz, Cline, and Schedl, 1985a, Cell 43: 521-29.
 Maine, Salz, Schedl, and Cline, 1985b, CSHSQB 50: 595-604.
 Schüpbach, 1985, Genetics 109: 529-48.
 Cline, 1986, Genetics 113: 641-63; corrigendum 114: 345.
 Salz, Cline, and Schedl, 1987, Genetics 117: 221-31.
 Gergen, 1987, Genetics 117: 477-85.
 Bell, Maine, Schedl, and Cline, 1988, Cell 55: 1037-46.
 Cline, 1988, Genetics 119: 829-62.
 Oliver, Perrimon, and Mahowald, 1988, Genetics 120: 159-71.
 Steinmann-Zwicky, 1988, EMBO J. 7: 3889-98.
 Steinmann-Zwicky, Schmid, and Nöthiger, 1989, Cell 57: 157-66.
 Salz, Maine, Keyes, Samuels, Cline, and Schedl, 1989, Genes Dev. 3: 708-19.
 Steinmann-Zwicky, Schmid, and Nöthiger, 1989, Cell 57: 157-66.
 Tompkins and McRobert, 1989, Genetics 123: 535-41.

phenotype: *Sxl*⁺ is a switch gene that acts throughout development to control all aspects of sexual dimorphism. Its products are required for female and must be absent for male development. Uniquely among sex-determination genes, after responding early in development to the primary sex-determination signal (the X:A ratio), *Sxl* maintains its own activity state as well as that of the downstream genes with which it interacts. It is required in a cell-autonomous fashion for both germ-line and somatic female development. It controls dosage compensation in females by suppressing hyperactivation of X-linked genes. Mutations of *Sxl* fall into two general classes: (1) recessive loss-of-function alleles that are deleterious to homozygous females, but viable and without phenotypic consequences in males, and (2) dominant gain-of-function alleles that behave as constitutive mutations, dominant and deleterious in males but without adverse effect in females, either heterozygous or homozygous. The variety of functions of the *Sxl* gene can be affected differentially by mutations, accounting in part for the complex complementation pattern observed for the large array of diverse mutant alleles. It is important to be aware that phenotypic parameters of mutant alleles and allele combinations can be very sensitive to culture conditions and genetic background. A number of positive regulators of *Sxl* are known, including the genes *da*, *fs(1)A1621*, *sis-a*, and *sis-b*. The female-specific lethal or sterile effects of mutations in these genes are suppressed by gain-of-function *Sxl* alleles. Throughout all but the very earliest period of development, female-specific expression of *Sxl* is known to be achieved by female-specific splicing of mRNA. The translation products from these female-spliced RNAs appear to help maintain the female-specific (productive) RNA processing mode which generates them, thereby establishing a positive feedback loop that maintains the female state throughout development.

alleles: The current convention for this gene is that alleles specifically disrupting female development (generally recessive loss of function) are designated *Sxl*^f, followed by a number, whereas those specifically disrupting male development (generally dominant gain of function) are

designated *Sxl*^M, followed by a number. In cases where a single lesion might have both characters, the *Sxl*^M designation would prevail. *P* in the designation indicates that the allele (and sometimes the stock as well) is likely to harbor *P*-element sequences. For new alleles selected as changes in the functioning of pre-existing mutant alleles, the original allele designation is followed by a *d* (for "derivative"), then a number. If and when such derivatives are shown to carry more than one lesion within the gene, the mutant designation will change to reflect the presence and order on the chromosome of the multiple lesions, individual mutations being separated by commas. For the most part, alleles in common use before these conventions were adopted were renamed only if the changes were relatively minor and self evident.

allele	origin	synonym	ref ^α	comments
<i>Sxl</i> ^{f1}	spont	<i>Fl</i>	4, 5, 9, 12, 17, 21, 23, 24, 25, 26, 28, 29	canonical amorph
<i>Sxl</i> ^{f2}	spont	<i>Fl</i> ^s , <i>Sxl</i> ^{fs}	11, 12, 14, 16	hypomorph, escapers female but sterile; 5 kb insert present fully viable; female sterile
<i>Sxl</i> ^{f4}	EMS	<i>fs(1)M106</i> ¹	15, 18, 19	fully viable; female sterile
<i>Sxl</i> ^{f5}	EMS	<i>fs(1)M106</i> ²	15, 18	fully viable; female sterile
<i>Sxl</i> ^{f6}	EMS	<i>In(1)Sxl-af</i>	12	null; associated with 6F; breakpoint
<i>Sxl</i> ^{f7,M1}	γ ray	<i>Sxl</i> ^{fm7,M1}	5, 6, 7, 19, 23, 25	male-viable, female-semilethal, double-mutant derivative of <i>Sxl</i> ^{M1} ; escaper females masculinized
<i>Sxl</i> ^{f9}	EMS		6, 13, 19	hypomorph; provides later functions best
<i>Sxl</i> ^{f2593}	EMS	<i>Sxl</i> ²⁵⁹³	5, 13, 19	hypomorph; ts for variety of functions; escapers masculinized to variable degree
<i>Sxl</i> ^{fhv1}	spont		3, 6, 11, 12	hypomorph; females viable and fertile; ca 6 kb insert present
<i>Sxl</i> ^{fLS}	spont		6, 12, 13, 19, 21, 22	hypomorph; provides early functions best; gypsy insert present
<i>Sxl</i> ^{fP3G2}	HD	<i>Sxl</i> ^{f3G2}	19, 20	male-viable null deletion of ca 22 kb from <i>Sxl</i> ^{fPb}
<i>Sxl</i> ^{fP6C2}	HD	<i>Sxl</i> ^{f6C2}	19	male-viable null deletion from <i>Sxl</i> ^{fPb}
<i>Sxl</i> ^{fP7A1}	HD	<i>Sxl</i> ^{f7A1}	19	male-viable hypomorph; intragenic deletion within <i>Sxl</i> ^{fPb}
<i>Sxl</i> ^{fP7AV}	HD	<i>Sxl</i> ^{f7AV}	19	male-viable hypomorph; intragenic deletion within <i>Sxl</i> ^{fPb}
<i>Sxl</i> ^{fP7B0}	HD	<i>Sxl</i> ^{f7B0}	19, 20, 27	male-viable null; deletion of >50 kb; deletion from <i>Sxl</i> ^{fPb}
<i>Sxl</i> ^{fP7C2}	HD	<i>Sxl</i> ^{f7C2}	19	male-viable null; deletion from <i>Sxl</i> ^{fPb}
<i>Sxl</i> ^{fPb}	spont		10, 12, 13, 19	hypomorph due to <i>P</i> element insertion; mosaic intersex female clones
<i>Sxl</i> ^{fPED1}	HD	<i>Sxl</i> ^{fED1}	19, 20	male-viable null; deletion of >28 kb; deletion from <i>Sxl</i> ^{fPb}
<i>Sxl</i> ^{fPED2}	HD	<i>Sxl</i> ^{fED2}	19	male-viable null;

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allele	origin	synonym	ref ^α	comments
<i>Sxl^{fPODH}</i>	HD	<i>Sxl^{fODH}</i>	19	deletion from <i>Sxl^{fPb}</i> male-viable hypo- morph; intragenic deletion within <i>Sxl^{fPb}</i>
<i>Sxl^{fPRC1}</i>	HD	<i>Sxl^{fRC1}</i>	19	<i>Sxl</i> hypomorph; <i>l(1)7Aa</i> deletion extending proximally from <i>Sxl^{fPb}</i>
<i>Sxl^{fPRJ2}</i>	HD	<i>Sxl^{fRJ2}</i>	19	<i>Sxl</i> hypomorph; <i>l(1)7Aa</i> deletion extending proximally from <i>Sxl^{fPb}</i>
<i>Sxl^{fPRL2}</i>	HD	<i>Sxl^{fRL2}</i>	19	<i>Sxl</i> hypomorph; <i>l(1)7Aa</i> deletion extending proximally from <i>Sxl^{fPb}</i>
<i>Sxl^{M1}</i>	spont		1, 2, 3, 4, 5, 9, 10, 11, 26	partially constitutive due to <i>roo</i> insert near male-specific exon
<i>Sxl^{M1,f3}</i>	γ ray	<i>Sxl^{M1,fm3}</i>	5, 25	male-viable, female- lethal, double- mutant derivative of <i>Sxl^{M1}</i> ; escaper females
<i>Sxl^{M1,fPa}</i>	HD		12, 13	double mutant; apparent null due to <i>P</i> -element insertion into <i>Sxl^{M1}</i>
<i>Sxl^{M1,fPa-ra}</i>	HD		8, 13, 27	complex partial revertant of <i>Sxl^{M1,fPa}</i> com- plementing <i>Sxl^{f9}</i> ; male ts; female cs return of wild-type function by partial deletion of <i>Sxl^{M1}</i> insert
<i>Sxl^{M1-vdl}</i>	spont		12	similar to <i>Sxl^{M1}</i> at molecular level
<i>Sxl^{M2}</i>	spont		12	<i>ca</i> 5 kb <i>hobo</i> ^β insert near male- specific exon 3
<i>Sxl^{M3}</i>	spont		12	<i>ca</i> 2 kb insert near male-specific exon 3
<i>Sxl^{M4}</i>	spont		12	similar to <i>Sxl^{M3}</i> at molecular level
<i>Sxl^{M5}</i>	spont		12	

^α 1 = Cline, 1978, *Genetics* 90: 683-97; 2 = Cline, 1979, *Dev. Biol.* 72: 266-75; 3 = Cline, 1980, *Genetics* 96: 903-26; 4 = Cline, 1983, *Dev. Biol.* 95: 260-74; 5 = Cline, 1984, *Genetics* 107: 231-77; 6 = Cline, 1986, *Genetics* 113: 641-63, corrigendum 114: 345; 7 = Cline, 1988, *Genetics* 119: 829-62; 8 = Cline, 1989, in *Evolutionary Mechanisms in Sex Determination* (Wachtel, ed.), CRC Press, pp. 23-36; 9 = Gergen, 1987, *Genetics* 117: 477-85; 10 = Golubovskiy, 1983, *DIS* 59: 42-43; 11 = Lucchesi and Skrip-sky, 1981, *Chromosoma* 88: 217-27; 12 = Maine, Salz, Cline, and Schedl, 1985, *Cell* 43: 521-29; 13 = Maine, Salz, Schedl, Cline, 1985, *Cold Spring Harbor Symp. Quant. Biol.* 50: 595-604; 14 = Marshall and Whittle, 1978, *Genet. Res.* 32: 103-11; 15 = Mohler and Carroll, 1984, *DIS* 60: 236-41; 16 = Muller and Zimmering, 1960, *Genetics* 45: 1001-02; 17 = Oliver, Perrimon, and Mahowald, 1988, *Genetics* 120: 159-71; 18 = Perrimon, Mohler, Engstrom, and Mahowald, 1986, *Genetics* 113: 695-712; 19 = Salz, Cline, and Schedl, 1987, *Genetics* 117: 221-31; 20 = Salz, Maine, Keyes, Samuels, Cline, and Schedl, 1989, *Genes Dev.* 3: 708-19; 21 = Sánchez and Nöthiger, 1982, *Wilhelm Roux's Arch. Dev. Biol.* 191: 211-14; 22 = Sánchez and Nöthiger, 1983, *EMBO J.* 2: 485-91; 23 = Schüpbach, 1985, *Genetics* 109: 529-48; 24 = Skrip-sky and Lucchesi, 1982, *Dev. Biol.* 94: 153-62; 25 = Steinmann-Zwicky, 1988, *EMBO J.* 7: 3889-98; 26 = Steinmann-Zwicky, Schmid, and Nöthiger, 1989, *Cell*

57: 157-66; 27 = Tompkins and McRobert, 1989, *Genetics* 123: 335-41; 28 = Uenoyama, 1982, *Jpn. J. Genet.* 59: 335-48; 29 = Zimmering and Muller, 1961, *DIS* 35: 103-04.
^β Shown to be a *hobo* element (Bernstein, 1989) rather than 297 as originally reported.

cytology: Placed in 6F4-7B3 (Nicklas and Cline, 1983, *Genetics* 103: 617-31) based on its inclusion in *Df(1)Sxl-ra = Df(1)6F4-5;7B1-3* (Lim).

molecular biology: The genomic region cloned by transposon tagging from *Sxl^{fPb}*; *ca* 50 kb walk restriction mapped and mutant alleles characterized over a 26 kb region between coordinates -13 and +13 (coordinate 0 corresponds to the site of insertion of the *P* element in *Sxl^{fPb}*) (Maine *et al.*, 1985; Salz *et al.*, 1989). Northern blot analysis reveals ten different *Sxl* transcripts derived from a 23 kb region, coordinates +1 to -22. All have closely related structures and are transcribed in the same (increasing negative coordinate) direction (Salz *et al.*, 1989). Three transcripts of 4.0, 3.1, and 1.7 kb appear transiently in blastoderm embryos. All others appear in later embryogenesis and persist throughout the remainder of development. Adult females have four *Sxl* transcripts of 4.2, 3.3, 3.3, and 1.9 kb; one 3.3 and the 1.9 kb species are reduced or absent in adults lacking a functional germ line. Adult males produce transcripts of 4.4, 3.6, and 2.0 kb. Two cDNAs, one from adult males and one from adult females derived from the 5' 15 kb of the *Sxl* transcription unit were sequenced (Bell *et al.*, 1988); the correspondence to the mRNAs is not known. The amino acid sequence inferred from the female cDNA predicts a 354 amino-acid protein of 39,000 daltons; the sequence indicates a basic protein which by hydrophathy analysis has characteristics of a soluble protein; it contains duplicated domains of 74 residues each, one at 126-199 and the other at 212-285, which exhibit 35% identity and show sequence similarity to known RNA-binding proteins (Bell *et al.*). Eight exons have been identified so far, with translation initiation likely from exon 2; exon 3 is found only in the three male specific transcripts, which otherwise correspond closely to three female-specific transcripts. Males are the sex in which genetic analysis shows *Sxl* to be nonfunctional. This exon contains two in-frame stop codons that would abort translation early on. Thus for most of development the on/off regulation of *Sxl* involves alternative modes of RNA processing. Considered in light of the genetic evidence for positive autoregulation of *Sxl* independent of the primary sex-determination signals (Cline, 1984, 1989), it would appear that *Sxl* maintains the female developmental state by controlling its own RNA splicing (Bell *et al.*). The predicted amino-acid sequence of *Sxl* products is consistent with this idea. Since *Sxl* is known to be required for sex-specific splicing of more developmentally specialized genes such as *tra* (Nagoshi, McKeown, Burtis, Belote, and Baker, 1988, *Cell* 53: 229-36), it appears that the biochemical activities of *Sxl* that maintain the sexually determined state may be the same as those that direct sexual differentiation through the regulation of genes downstream. Characteristics of the transcription pattern very early in development suggest that a different mode of regulation operates during this period when the gene is first responding to the primary sex-determination signal to set the sexual fate of cells (Salz *et al.*, 1989). Genetic (Salz *et al.*, 1987) and molecular (Salz *et al.*, 1989) stu-

dies suggest that the promoter operating during this early period is different from that which functions throughout the remainder of development, when sex-specific activity is determined by RNA splicing.

***Sxl*^{f1}**

phenotype: Homozygous females invariably die as embryos but hemizygous males are fully viable and fertile. In most wild-type genetic backgrounds, heterozygous females exhibit normal viability and fertility, although occasionally display morphological defects characteristic of early cell death; however, can be dominant semilethal for females in some wild-type genetic backgrounds and under suboptimal growth conditions. In doubly heterozygous combination with otherwise recessive mutations in positive regulators of *Sxl*, this allele can behave as a dominant: heterozygote viability is reduced for daughters of *dal*⁺ females, as well as for females that are also heterozygotes for either *sis-a*, *sis-b* or *fs(1)A1621*. In some such doubly heterozygous situations, escaper females may be incompletely masculinized (mosaic intersex). Homozygosity for mutations in the autosomal male-specific lethal loci does not suppress recessive *Sxl*^{f1} lethality, but it does partially masculinize *Sxl*^{f1}/+ females (generating mosaic intersexes) and suppresses cell-death-related morphological defects. Homozygous moribund embryos show sex-specific alterations in the phenotypic expression of hypomorphic X-linked alleles such as *run*²⁵, a reflection of upsets in dosage compensation (female hyperactivation). Depending on the time of induction, *Sxl*^{f1}/*Sxl*^{f1} clones induced in *Sxl*^{f1}/+ females can be phenotypically male and reduced in size. 2X:3A animals homozygous or heterozygous for *Sxl*^{f1} are viable but masculinized. In genetic mosaics and chimeras, *Sxl*^{f1} homozygous germ cells develop abnormally and fail to generate functional gametes. In some situations, the mutant female tissue displays masculine traits. *Sxl*^{f1} rescues males from the otherwise lethal effects of a simultaneous duplication of *sis-a*⁺ and *sis-b*⁺.

***Sxl*^{f2}**

phenotype: Homozygous females are either inviable or very poorly viable, depending on genetic background. Escapers are invariably sterile but otherwise display no obvious sexual abnormalities. Complements *Sxl*^{f2593}. Homozygotes defective in dosage compensation as indicated by hyperincorporation of uridine by their polytene chromosomes. Allele fails to support oogenesis in germ-line clones induced by mitotic recombination.

***Sxl*^{f3}**

phenotype: A hypomorphic allele selected as an intragenic suppressor of *Sxl*^{M1} male lethality; maps 0.0065 cM to the right of *Sxl*^{M1}. Only characterized in *cis* combination with *Sxl*^{M1}. The double mutant is fully viable in males and poorly viable in homozygous females, with escapers being phenotypically male and sterile. Hemizygous females are lethal. Partially complements *Sxl*^{f2593}, generating true intersexes. Partially complements *Sxl*^{f7,M1} with escapers phenotypically male and sterile. Fully complements *Sxl*^{fhv1}. By itself, double mutant fails to bypass maternal *da*⁺ requirement for activation, but can complement *Sxl*^{f7,M1} in this regard. Double heterozygote with *fs(1)A1621* is fertile.

***Sxl*^{f7}**

phenotype: A hypomorphic allele selected as an intragenic suppressor of *Sxl*^{M1} male lethality; maps 0.0099 cM to the left of *Sxl*^{M1}. Only characterized in *cis* combination with *Sxl*^{M1}. The double mutant is male viable and semiviable in homozygous females. Escaper females are phenotypically male and sterile. Hemizygous females are inviable. Double heterozygote with *fs(1)A1621* is sterile, like *Sxl*^{f1} but unlike *Sxl*^{M1,f3}. The double-mutant allele retains some ability to rescue daughters from the otherwise lethal maternal effect of *da*; however, lowering maternal *da*⁺ activity appears to decrease *Sxl*^{f7,M1} functioning, consistent with other evidence that the parental allele, *Sxl*^{M1}, is not fully constitutive. In the absence of a wild-type *Sxl* allele, *Sxl*^{f7,M1} daughters that survive the *da* maternal effect are phenotypically male and sterile; in contrast, the addition to this genotype of a wild-type *Sxl* allele in *trans* renders survivors phenotypically female, but still sterile with masculinized gonads. The latter genotype of female is fertile provided mothers carry at least one *da*⁺ allele. The ability of *Sxl*^{f7,M1} to rescue daughters is greatly enhanced by mutations in the autosomal, male-specific-lethal loci, genes involved in hyperactivation of X-linked genes in males. The basis for this enhancement is related to the ability of these same mutations to enhance the survival of *Sxl*^{f7,M1} hemizygous females. Although *Sxl*^{f7,M1} was used to demonstrate the ability of *Sxl* gene products to activate *Sxl*⁺ alleles in *trans*, it can be inferred that this allele is far below wild type in this activity. *Sxl*^{f7,M1} is a dominant suppressor of *sis-a* female-specific lethality, generating sterile females remarkably similar to those described above rescued from the *da* maternal effect. Unlike *Sxl*^{M1,f3}, fails to complement *Sxl*^{f2593}, yet partially complements *Sxl*^{M1,f3} and *Sxl*^{fPb}, generating sterile phenotypic males. Allele supports oogenesis in homozygous mutant germ-line clones induced by mitotic recombination. In males, mutant allele suppresses the otherwise lethal effect of a duplication of region 3C2-5A2; addition of *Sxl*⁺ to this aneuploid genotype generates mosaic intersexes indicating that the positive autoregulatory activity of *Sxl* products can bypass the X/A signal. Double heterozygote with *fs(1)A1621* is sterile (like *Sxl*^{f1} and unlike *Sxl*^{M1,f3}).

***Sxl*^{f9}**

phenotype: A lethal hypomorphic allele defective in some very early steps in the sex-determination process, but which has no adverse effect on the growth or sexual development of homozygous mutant diplo-X clones induced by mitotic recombination. Rare escapers at 18° are phenotypically female; nevertheless, it has a dominant masculinizing effect on the phenotype of triploid intersexes (2X:3A) and interacts in a dominant-lethal fashion with mutations in *da* or *sis-a*, both early acting positive regulators of *Sxl*. Fully complements *Sxl*^{fPR} class (partial deletions of *Sxl* information that impair later functions of the gene more than earlier). Complements *Sxl*^{M1,fPa-ra}.

***Sxl*^{f2593}**

phenotype: A hypomorphic allele that is temperature sensitive for most *Sxl* functions. Perhaps most notable for the fact that homozygote viability can be quite high, with the females developing as true intersexes (their specific

grade of intersexuality depends on temperature). Lethal over a deficiency, a null allele, or *Sxl*^{*f7,M1*} at any temperature; at permissive temperatures, weakly complements *Sxl*^{*M1,f3*} and hypomorphic alleles of the *Sxl*^{*fPR*} class, generating (true) intersexual escapers; complementation better with *Sxl*^{*fPb*}, generating sterile females; fully complements *Sxl*^{*f2*}, *Sxl*^{*f9*}, and *Sxl*^{*fsv1*}.

***Sxl*^{*fsv1*}**

phenotype: A subliminal allele, viable and fertile as homozygous females, but with greatly reduced viability in *trans* to nulls. Polytene chromosomes of *Sxl*^{*fsv1*}/*Sxl*^{*f1*} larvae that survive to third instar hyperincorporate uridine, revealing female dosage compensation upsets. Mutation of *mle* appears to partially masculinize this heteroallelic combination and may slightly increase viability under some conditions. *Sxl*^{*fsv1*} homozygotes and heterozygotes display an increased requirement for maternal *da*⁺ activity, suggestive of defects in early *Sxl* regulation.

***Sxl*^{*fLS*}**

phenotype: A lethal hypomorphic allele that is able to initiate female development, but is defective in its ability to maintain the female developmental commitment and/or to elicit female sexual differentiation. It is masculinizing in homozygous mutant somatic clones induced by mitotic recombination, and it causes the tissue in such clones to grow poorly; nevertheless, it has no dominant effect on the sexual phenotype of triploid intersexes, nor does it interact in a dominant fashion with mutations in *da* or *sis-a*, both early acting positive regulators of *Sxl*. Fully complements *Sxl*^{*f9*}, which appears to have a very different set of defects.

***Sxl*^{*f7BO*}**

phenotype: Female-lethal null allele that appears to be deleted for the entire *Sxl* transcription unit. Males are fully viable, fertile, and display normal male sexual behavior.

***Sxl*^{*fPa*}**

phenotype: A hybrid-dysgenesis-induced apparent null allele selected as an intragenic suppressor of *Sxl*^{*M1*} male lethality. Only characterized in *cis* combination with *Sxl*^{*M1*}. A *P*-element insertion 5' to the site of the DNA insertion in *Sxl*^{*M1*} but still within the region of *Sxl* transcribed at all stages. Revertible.

***Sxl*^{*fPa-ra*}**

phenotype: A hybrid-dysgenesis-induced derivative of *Sxl*^{*M1,fPa*} selected for having regained the ability to complement *Sxl*^{*f9*}. This complex allele disrupts both male and female development, with the magnitude of the effects in either sex depending on culture temperature in a reciprocal fashion: high temperature is more permissive for females and less permissive (more feminizing) for males. Intersexual males show little male sexual behavior and stimulate courtship from other males. Dominant male-lethal effects are greatly enhanced by the presence of a duplication of *Sxl*⁺ in *trans*; male escapers with both alleles exhibit an unusual dorsalization of the abdomen, their sternites being variably transformed into tergites.

***Sxl*^{*fPb*}**

phenotype: A *P*-insertion-induced lethal hypomorphic allele with the unusual distinction of displaying a mosaic intersex phenotype in homozygous mutant diplo-*X* clones induced by mitotic recombination; hence, appears to be defective in the cellular maintenance of the female sexual commitment. Under dysgenic conditions, can mutate further to less extreme or to more extreme condition. Partially complements *Sxl*^{*f7,M1*}, generating masculinized individuals; partially complements *Sxl*^{*f2593*}, generating sterile females; fully complements *Sxl*^{*f9*}.

***Sxl*^{*M1*}**

phenotype: Unconditionally lethal to males, even in the presence of a *Sxl*⁺ duplication. Retains normal level of female function as evidenced by full viability and fertility of homozygous and hemizygous mutant females. Recovered by virtue of ability to bypass the normal requirement by females for maternally supplied *da*⁺ product, a positive regulator of *Sxl*⁺; however, bypass is incomplete at higher temperatures. Phenotype in both sexes results from expression of *Sxl*⁺ female sex determination and dosage compensation functions largely (though not completely) independently of the normal controls. This is shown by the observation that induction of mutations in *cis* that suppress dominant, male-specific lethality is invariably associated with a corresponding reduction in *Sxl*⁺ female-specific activities and the dominant *da* maternal-effect bypass phenotype. *Sxl*^{*M1*} is lethal to most gynandromorphs by the pharate-adult stage, disrupting the development of their haplo-*X* tissue in a cell-autonomous fashion; mutant haplo-*X* tissue in gynandromorphs is often, but not always, feminized. This variable penetrance of the sex transformation suggests a residual level of control by the *X/A* balance. *Sxl*^{*M1*} feminizes triploid intersexes, killing them as pharate adults, while suppressing *B* and *Hw* alleles in a fashion consistent with expectations for constitutive expression of normal female dosage-compensation functions. Analysis of effects on the dosage compensation of the very early acting segmentation gene, *run*, suggests that constitutive expression of female functions is not observed prior to the time when the later *Sxl* promoter is required and RNA processing control is known to be operating. Since *run* dosage compensation during this period does require functioning of maternal *da*⁺, zygotic *Sxl*⁺, and the *X/A* balance, the ability of *Sxl*^{*M1*} to bypass these controls during later stages of development would seem to indicate that the effect of the mutant lesion it carries is on *Sxl*-RNA splicing, a process that these other *Sxl*⁺ regulators may only affect indirectly. The position of the *Sxl*^{*M1*} mutant lesion in the vicinity of the male-specific exon is suggestive in this connection. Variable expressivity of this mutant allele may underlie two additional observations: (1) *Sxl*^{*M1*} male lethality can be suppressed by *fs(1)A1621*, yet *fs(1)A1621* female sterility can be suppressed by *Sxl*^{*M1*}, and (2) transplants of *Sxl*^{*M1*}/*Y* and *Sxl*^{*M1*}/+ germ cells show that although the allele does not appear to interfere with spermatogenesis in testes, it blocks the otherwise masculinizing effect of testicular somatic tissue on diplo-*X* (female) germ cells.

sy: see *os*^{*s*}

***Sy: Stubby**

location: 1- or 2- (rearrangement).

discoverer: Ives, 34j31.

phenotype: Bristles short and thick, especially humerals and notopleurals. Male sterile. RK2.

cytology: Associated with *T(1;2)Sy*; breakpoints unknown, but break in *X* is genetically at the right end.

*Sy*³⁰ : see *Bl*³⁰

*Sy*³¹¹¹⁹ : see *Bl*³¹¹

Syb: Synaptobrevin

location: 2- {60}.

origin: Isolated from a *Drosophila* cDNA library using a bovine cDNA probe.

references: Südhoff, Baumert, Perin, and Jahn, 1989, *Neuron* 2: 1475-81.

phenotype: Encodes a 20 kd polypeptide that is recognized by antibodies to rat synaptobrevin. Synaptobrevin is an intrinsic membrane protein of small synaptic vesicles from mammalian brain; it is highly homologous to *Torpedo* VAMP-1. Signal from *Drosophila* heads exceeds that from rat brain suggesting that it is an abundant protein in flies.

cytology: Placed in 46E-F by *in situ* hybridization to polytene chromosomes.

molecular biology: The isolated cDNA clone contained an 0.8 kb insert whose conceptual amino acid sequence

defines a polypeptide of 152 amino acids with a calculated molecular weight of 16,585. The polypeptide contains a 47-amino-acid N-terminal sequence that is rich in asparagine (26%) and shows little homology to the N-terminal sequences of mammals and *Torpedo*; the next 63 amino acids are highly conserved and display 75% identity in all three species; this region is followed by a putative transmembrane domain of 20 amino acids which display 60% identity with bovine synaptobrevin; finally the C-terminal domain, which is thought to be intravesicular comprises 22 amino acids in *Drosophila*, but only two in bovine and *Torpedo* polypeptides. Northern blots of *Drosophila* head RNA reveals mRNAs of 0.85 and 0.9 kb, much shorter than the 2.2 kb observed in mammals.

***syn: syndrome**

location: 3-14.7.

origin: γ ray induced.

discoverer: Wallbrunn, 61i21.

references: 1964, *DIS* 39: 58.

phenotype: Eyes of male translucent brown, of female slightly darker than normal. Wings of male held at right angle to body, of female held out at about 45°. Viability low. Both sexes sterile. RK2.

Synaptobrevin: see **Syb**

syndrome: see **syn**

t: tan (J.C. Hall)

location: 1-27.5.

- references:** McEwen, 1918, *J. Exp. Zool.* 25: 49-106.
 Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 237.
 Demerec, Kaufman, Fano, Sutton, and Sansome, 1942, *Year Book-Carnegie Inst. Washington* 41: 191.
 Benzer, 1967, *Proc. Nat. Acad. Sci. USA* 58: 1112-19.
 Pak, Grossfield, and White, 1969, *Nature (London)* 222: 351-54.
 Hotta and Benzer, 1969, *Nature (London)* 222: 354-56.
 Heisenberg, 1971, *J. Exp. Biol.* 55: 85-100.
 Konopka, 1972, *Nature (London)* 239: 281-82.
 Heisenberg and Götz, 1975, *J. Comp. Physiol.* 98: 217-41.
 Heisenberg and Buchner, 1977, *J. Comp. Physiol.* 117: 127-62.
 Wright, 1987, *Adv. Genet.* 24: 127-222.

phenotype: Body color varies from light tan to almost wild-type. Easiest to identify by light tan antennae; male easier than female. Larval mouthparts in mutant alleles, *t*¹, *t*², and *t*³ lighter than normal at basal prongs; classifiable with difficulty in larvae (Brehme, 1941, *Proc. Nat. Acad. Sci. USA* 27: 254-61). Neurological defect first observed in *t*¹ by McEwen (1918), who reported that the mutant flies behaved as if they were nonphototactic in his test apparatus (tube with light at one end); Benzer (1967) and Pak *et al.*, (1969), using a similar but more elaborate apparatus and EMS-induced tan alleles, reached the same conclusion. In a Y-maze test for phototaxis, however, tan mutants are sensitive to light (Hadler, 1984, *Biol. Bull.* 126: 264-73). In the electroretinogram (ERG) tracing obtained when one electrode is inserted in the first optic ganglion (lamina) below the ommatidia, the on- and off-transients of tan flies are reduced or missing, but the other ERG components are present, indicating that the mutant is not blind although it appears behaviorally blind in some tests. Mosaic studies (Hotta and Benzer, 1970, *Proc. Nat. Acad. Sci. USA* 67: 1156-63) indicate that the ERG phenotype of tan is cell autonomous. The optomotor responses of mutants to horizontal and vertical movements are abnormal in that light intensity thresholds for responses are raised, the extent depending on the allele (Heisenberg, 1971, *DIS* 46: 68; Heisenberg and Götz, 1975; Heisenberg and Buchner, 1977). No walking optomotor responses are detected, but there can be positive responses in flying optomotor tests if the rotating vertical stripes are relatively wide.

The enzyme tyrosinase, which converts tyrosine to 3,4-dihydroxyphenylalanine (DOPA), is formed in tan adults (Horowitz). Dopamine, an important component of catecholamine metabolism (involved in both melanization of cuticle and synthesis of neurotransmitters), has been found in reduced amounts (about 60% of the wild-type concentration) in *t*¹ and *t*⁵ (Konopka, 1972). The dopamine was detected only in late pupae that show some pigmentation and in adult flies. One of the enzymes involved in the formation of dopamine, β-alanyldopamine hydrolase (BAH), shows no activity in *t/t* and *t/Y* mutants and reduced activity in *t/+* heterozygotes as compared to wild-type (Black, Pentz, and Wright). This dosage effect implicates tan as the structural gene for BAH (Wright, 1987).

The mutant tan is defective in courtship behaviors, the

males showing aberrant visually-mediated responses to moving female flies (Cook, 1980, *Biol. Cybernet.* 37: 41-51; Tompkins, Gross, Hall, Gailey, and Siegel, 1982, *Behav. Genet.* 12: 295-307).

alleles: No complementation for body color or phototaxis between *t*¹ and *t*⁵, *t*⁶, *t*⁸, *t*⁹, and *t*¹⁰. Deficiencies listed in rearrangement section.

allele	origin	discoverer	synonym	ref ^α	comments
<i>t</i> ¹	spont	Bridges, 14g16		2, 6-15	nonphototactic; dopamine < <i>t</i> ⁺ ; ERG abnormal
<i>t</i> ²	spont	Bridges, 19d5		3, 13	darker than <i>t</i> ¹
<i>t</i> ³	spont	Bridges, 31e11		3	lighter than <i>t</i> ¹ ; tan spot on abdomen
<i>t</i> ⁴	spont	Bridges, 33c14			weak <i>t</i> in color
<i>t</i> ⁵		Konopka			dopamine < <i>t</i> ⁺ ; ERG abnormal
<i>t</i> ⁶	EMS	Homyk, Sheppard	<i>t</i> ¹⁰¹	8	flight abnormal; ERG abnormal
<i>t</i> ⁷	EMS	Heisenberg	<i>opm8</i>	5	weak optomotor response; ERG abnormal
<i>t</i> ⁸	EMS	Heisenberg	<i>opm24</i>	7	ERG abnormal
<i>t</i> ⁹	EMS	Benzer	<i>PC13</i>	14	ERG abnormal
<i>t</i> ¹⁰	EMS	Pak	<i>x-7</i>	1, 4, 14, 15	nonphototactic; ERG abnormal

^α 1 = Alawi and Pak, 1971, *Science* 172: 1055-57; 2 = Benzer, 1967, *Proc. Nat. Acad. Sci. USA* 58: 1112-19; 3 = Brehme, 1941, *Proc. Nat. Acad. Sci. USA* 27: 254-61; 4 = Grossfield and Pak, 1971, *DIS* 47: 59; 5 = Heisenberg, 1971, *DIS* 46: 68; 6 = Heisenberg, 1971, *J. Exp. Biol.* 55: 85-100; 7 = Heisenberg and Buchner, 1977, *J. Comp. Phys.* 117: 127-62; 8 = Homyk, 1977, *Genetics* 87: 105-28; 9 = Hotta and Benzer, 1969, *Nature (London)* 222: 354-56; 10 = Hotta and Benzer, 1970, *Proc. Nat. Acad. Sci. USA* 67: 1156-63; 11 = Konopka, 1972, *Nature (London)* 239: 281-82; 12 = McEwen, 1918, *J. Exp. Zool.* 25: 49-106; 13 = Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 237; 14 = Pak, 1975, *Handbook of Genetics* (King, ed.), Plenum Press, New York and London, Vol. 3, pp. 703-33; 15 = Pak, Grossfield, and White, 1969, *Nature (London)* 222: 351-54.

^β Legs not retracted in flight (Homyk and Sheppard, 1977, *Genetics* 87: 95-104).

cytology: Placed in region 8C2-D1 (Demerec *et al.*, 1942) on the basis of its inclusion in *Df(1)t282-1 = Df(1)8C2-3;8C14-D1*.

other information: *t* suppresses jump response of *stn*^{ts} (Kelly, 1983, *Cell Molec. Neurobiol.* 3: 143-49).

*T*²: see *dp*^{olv2}

*T*¹: see *Tp1*

T-cp1: T-complex 1

location: 3- {76}.

origin: Molecular cloning.

references: Ursic and Ganetzky, 1988, *Gene* 88: 267-74.

phenotype: Codes for a cDNA from *Drosophila melanogaster* that shares 66% sequence identity with the mouse *Tcp-1* gene encoding *TCP1* (t-complex polypeptide 1).

cytology: Placed in 94B1-2 by *in situ* hybridization to salivaries.

molecular biology: Gene cloned and sequenced. The predicted protein is made up of 557 amino acids and is 72% identical to the mouse polypeptide. The yeast *Saccharomyces cerevisiae* also carries a genomic fragment that can be detected by the *Drosophila T-cp1* cDNA.

ta: tapered (J.C. Hall)**location:** 2-56.6.**origin:** Ultraviolet induced.**discoverer:** Edmondson and Meyer, 49c.**references:** 1949, DIS 23: 61.

Wood and Butterworth, 1972, DIS 49: 67-68.

phenotype: Wings narrow and pointed, somewhat longer than normal. Veins close together. Viability good. Female fertility low; male sterile. Males show abnormal courtship pattern, *i.e.* vague interest in circling and approaching female with no interest in touching. Periodic wing vibrations at 90° and proboscis movements occur, but females not receptive to mutant males. 20% of *ta/ta* males had non-motile spermatozoa. Frequently, accessory glands on one or both sides were atrophied; testes often atrophied or show club-like structures on the apical ends (Wood and Butterworth, 1972). RK2.*ta*: see *tar***Ta: Thickened arista****location:** 3- {47}.**synonym:** *Ta^L*: *Thickened arista-Lethal* (used by Cavener *et al.*, 1986b, for *Ta¹*).**origin:** γ ray induced (*Ta¹*).**references:** Kaufman, 1978, Genetics 90: 579-96.

Kaufman, Lewis, and Wakimoto, 1980, Genetics 94: 115-33.

Lewis, Kaufman, Denell, and Talerico, 1980, Genetics 95: 367-81.

Cavener, Corbett, Cox, and Whetten, 1986a, EMBO J. 5: 2939-48.

Cavener, Otteson, and Kaufman, 1986b, Genetics 114: 111-23.

phenotype: Semidominant homeotic mutant that transforms arista segments into tarsal segments. Both *Ta¹* and *Ta²* are homozygous lethal; lethality of *Ta²* may be due to its association with a Minute. The heteroallelic combination *Ta¹/Ta²* is viable and shows transformation of the whole arista and the fourth and fifth antennal segments into a segmented tarsus with a claw at the distal end; little or no transformation of the third antennal segment; males show extreme reduction in the number of sex-comb teeth on the first leg, as in *Df(3R)Scr* (Kaufman *et al.*, 1980). *Ta¹/+* and *Ta²/+* flies have a less extreme antennal phenotype without the reduction in the number of sex-comb teeth.**alleles:** Two mutant alleles have been identified: *Ta¹*, associated with *T(2;3)Ta*, and *Ta²*, which is cytologically normal. These alleles are partially complementing. Both are viable (but mutant) over *Df(3R)Scr* = *Df(3R)84A1-2;84B1-2*; *Ta²* is also viable over *Df(3R)Antp17* = *Df(3R)84B1-2;84D11-12*.**cytology:** Placed in 84C1-2 on the basis of molecular data for the breakpoint of *T(2;3)Ta* = *T(2;3)51E1-2;84C1-2* which lies between the proximal breakpoint of *Df(3R)dsx2M* = *Df(3R)84C1-2;84E1-2* and the distal breakpoint of *Df(3R)Scx2* = *Df(3R)84A4-5;84C1-2*.**molecular biology:** Distal (84C1-2) breakpoint of *T(2;3)Ta* located at about +34 kb [in the 15 kb overlap of *Df(3R)Scx2* and *Df(3R)dsx2M* (Baker and Wolfner, 1988, Genes Dev. 2: 477-89; 0 point = Hind III site in α Tub84B; "+" values to the right, "-" values to the left)].**Tab:** see **BXC***Tac*: see *Pin^{Tac}***tailless:** see **tll****tailup:** see **tup****tammo:** see **tmo****tan:** see **t****tapered:** see **ta****tar: tarry****location:** 1-27.3 (0.4 unit from *lz*, probably to the left).**origin:** Found among progeny of deuteron-irradiated male.**discoverer:** Hildreth, 51i.**synonym:** *ta* (preoccupied).**references:** 1953, DIS 27: 56.**phenotype:** Expression ranges from small black spots on distal end of femora or proximal end of tibiae to cases in which the tibiae, femora, and bases of coxae are encapsulated in a dark, brownish-black, glossy covering. Legs weak. Some overlap wild type. Viability reduced. RK2.**other information:** Possibly an allele of *me* (1-29.0).*Tarhos*: see *Fs(2)Sz9**Tarnished*: see *bw^{V3}***tarry:** see **tar****tarsi irregular:** see **ti****tastebblind:** see **tbl*****taw: tawny****location:** 1-41.1.**origin:** Induced by D-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3026).**discoverer:** Fahmy, 1955.**references:** 1958, DIS 32: 75-76.**phenotype:** Head and thorax slightly dark; abdomen pale. Wings usually scooped or tips curved. Female tergites often narrow, serrated, or broken. Viability and fertility good. RK3.**taxi:** see **tx*****tb: tiny bristle****location:** 1-35.8.**discoverer:** Bridges, 16a4.**references:** 1919, J. Gen. Physiol. 1: 645-56.**phenotype:** All bristles short and fine; wings somewhat short. Female fertility low. RK2.*tb*: see *tbr***Tb: Tubby****location:** 3-90.6.**synonym:** *Tu*.**references:** Auerbach, 1943, DIS 17: 49.

Lindsley, 1973, DIS 50: 21.

Craymer, 1980, DIS 55: 197-200.

phenotype: Dominant; larvae, pupae, and adults shorter and thicker than wild-type in both heterozygotes and homozygotes (not separable). Larvae distinguishable on basis of reduced length and tortuous tracheal trunks. Classification reliable in larvae and pupae but not adults. Both sexes viable and fertile.**alleles:** Two alleles, *Tb*, induced by chemicals, presumably nitrogen mustard (Auerbach, 1943) and **Tb²*, induced by

γ rays (Lindsley, 1973).

cytology: Salivary chromosomes normal (Lindsley, 1973).

tb-53: see *mc*

****tbd*: tiny bristleoid**

location: 1-25.

origin: Spontaneous.

discoverer: Curry, 37g23.

phenotype: Bristles short and thin, like a medium Minute. Fly somewhat smaller than wild type. Good viability and fertility. RK2.

cytology: Locus between 7C5 and 8C1 (Demerec, Kaufmann, Fano, Sutton, and Sansome, 1942, Year Book – Carnegie Inst. Washington 41: 191). Further restricted to 8A1-C1 on the basis of its genetic location to the right of *oc* at 8A1-2 (Hinton and Welshons, 1955, DIS 29: 125-26).

***tbl*: tastblind (J.C. Hall)**

location: 2- or 3-.

origin: Induced by ethyl methanesulfonate.

references: Isono and Kikuchi, 1974, Jpn. J. Genet. 49: 113-24.

Tanimura, Isono, and Kikuchi, 1978, Jpn. J. Genet. 53: 71-73.

phenotype: Electrophysiological response of labellar hair sensory structures to D-glucose, maltose, and sucrose reduced compared to wild type; no response to D-mannose, D-xylose, and lactose. Response to D-fructose normal.

***tbr*: tracheae broken**

location: 3- (not located).

origin: Spontaneous.

discoverer: Slatis.

synonym: *tb* (preoccupied).

references: 1959, Genetics 44: 536.

phenotype: Main tracheal trunks of larva have interruptions. Penetrance 17% at 16°, 5% at 25°. Does not seem to affect viability. RK3.

***tbs*: thin bristles**

location: 2-81.6.

origin: Spontaneous.

synonym: *Ho*.

references: Mostashfi and Koliantz, 1970, DIS 48: 104.

phenotype: Thin and somewhat short bristles.

***tc*: tiny chaetae**

location: 1-51.6.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007)

discoverer: Fahmy, 1954.

references: 1958, DIS 32: 76.

phenotype: Bristles extremely short and fine. Eclosion delayed. Viability and fertility good. RK1.

cytology: Located in 13F since included in *Df(1)sd72b* = *Df(1)13F1;14B1* but not in *Dp(1;4)r⁺* = *Dp(1;4)13F10;16A1-2* (Lefevre).

***Tcp*: Third chromosome cold-sensitive paralytic (J.C. Hall)**

location: 3-66.0.

origin: Induced by ethyl methanesulfonate.

synonym: *TCP*.

references: Sønndergaard, 1980, Heredity 92: 335-40.

phenotype: *Tcp/+* mutants kept at 25° show an unsteady gait upon mechanical agitation, the more extreme alleles falling over and having difficulty in righting themselves. Surviving heteroallelic combinations have motor problems even when undisturbed. All heterozygotes reversibly paralyzed within 18 sec at 12° (more variable from 12° to 19°). Homozygotes and heteroallelic combinations lethal for the most part; survivors more sensitive to cold than *Tcp/+*. During development, heterozygous embryos, but not larvae, are killed by exposure to temperatures less than 13°. In extracts from mutant adults, assays of a mitochondrial enzyme show shifts in Arrhenius activation energy.

alleles: Eleven alleles designated *Tcp*¹ to *Tcp*¹¹. Most alleles are recessive lethals. *Tcp*² appears to be a hypomorph; *Tcp*⁹ survives as a homozygote, but is lethal in many heteroallelic combinations.

***Tcr*: Third chromosome resistant**

location: 3-39.6.

origin: Induced by ethyl methanesulfonate.

discoverer: Bishop, Sherald, and Wright.

synonym: *l(3)Tcr*.

references: Wright, 1987, Adv. Genet. 24: 127-222.

phenotype: *Tcr/+* heterozygotes resistant to dietary α methyl dopa. Homozygous lethal. Heteroallelic combination *Tcr*⁴⁰/*Tcr*⁴⁵ partially lethal, 48.3% dying as late embryos, these unhatched larvae having underpigmented mouthparts like lethal *Ddc/Ddc* embryos; only 7.5% go through pupation and manage to eclose, but these adults die soon. Both pharate and hatched adults have cuticular abnormalities involving the eyes (rough, often small, with black, necrotic spots), ocelli (often missing), wings (notched), scutellum (reduced), bristles (missing or *M*-like), sex combs (reduced), and abdomen (grayish). *Tcr/+* heterozygotes suppress the lethality of *amd* heteroallelic combinations; *Tcr* heteroallelic combinations are not rescued genetically by *amd/+*, but show increased embryonic mortality as a maternal effect of *amd*.

alleles: Two partially complementing alleles described, *Tcr*⁴⁰ and *Tcr*⁴⁵.

****tdd*: tiddler**

location: 1-0.0 (0/871 crossovers with *sc*).

origin: Induced by ethyl methanesulfonate (CB. 1528).

discoverer: Fahmy, 1956.

references: 1958, DIS 32: 76.

phenotype: Body small. Viability and fertility good. RK3.

****te*: tenerchaetae**

location: 1-5.6.

origin: Induced by triethylenemelamine (CB. 1246).

discoverer: Fahmy, 1952.

references: 1958, DIS 32: 76.

phenotype: Bristles short and fine. Eyes dark and glistening. Wings frequently small, deformed in various ways. Eclosion delayed. Male viability, but not fertility, good. Female infertile. RK3.

technical knockout: see *tko*

ted: trapped

location: 3- [47].

references: Cavener, Otteson, and Kaufman, 1986, *Genetics* 114: 111-23.

phenotype: Homozygous lethal; homozygotes attempt to eclose but cannot break the operculum seams. Rescue of fully viable and fertile adults can be effected by dissection from the puparium.

alleles: Five mutant alleles have been described.

allele	origin	discoverer	synonym
<i>ted</i> ¹	EMS	Grigliatti	<i>l(3)4.15</i>
<i>ted</i> ²	EMS	Grigliatti	<i>l(3)5.12</i>
<i>ted</i> ³	EMS	Cavener	<i>l(3)g8</i>
<i>ted</i> ⁴	EMS	R. Lewis	<i>l(3)r5</i>
<i>ted</i> ⁵	EMS	R. Lewis	<i>l(3)r12</i>

cytology: Placed in 84C6-8 since in the region of overlap between the distal breakpoint of *In(3R)Antp*^{73b} = *In(3R)84B1-2;84C5-6* and the proximal breakpoint of *Df(3R)dsx-M29 = Df(3R)84C8-D1;84F6-7*.

other information: The rescuable mutant phenotype is the same as that observed in *Gla*ⁿ mutants (Cavener and MacIntyre, 1983, *Proc. Nat. Acad. Sci. USA* 80: 6286-88).

Tegula: see under *shv* in *dpp* entry

Tekele: see *Fs(2)Sz10*

telegraph: see *tg*

telescope: see *ts*

temperature induced paralysis A: see *tipA*

Temporal protein 1: see *Tp1*

***ten: tenuis chaetae**

location: 1-43.9.

origin: Induced by D-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3026).

discoverer: Fahmy, 1953.

references: 1958, *DIS* 32: 76.

phenotype: Bristles short and thin. Body small. Expression more extreme in female. Eclosion slightly delayed. Viability and fertility good. RK3.

tender little chaetae: see *tlc*

tenerchaetae: see *te*

tent: see *tnt*

tenuis chaetae: see *ten*

ter: terraced

location: 2-36.

origin: Spontaneous (*ter*^U appeared as cluster in mutable *sc z w*^{zmz} stock).

discoverer: Bridges, 29c12.

references: Kalisch, 1970, *Mol. Gen. Genet.* 107: 321-35. 1980, *DIS* 55: 206-07.

phenotype: Eye abnormality varies from tiny facetless nick in anterior eye rim plus a horizontal seam of irregular facets to a large indentation in the anterior eye margin. Large tuft of bristles often found in facetless eye area. Penetrance in *ter*^U about 94% at 16-25°. Viability good, but *ter*^U/*ter*^U homozygotes show lower hatching rate than *ter*^U/+ heterozygotes.

alleles: Two alleles of similar phenotype described: **ter* (Bridges) and *ter*^U, *terraced of Umeå* (Kalisch).

Term: Terminus

location: 3- [45].

synonym: *Bsg75C; ter; hid(?)*.

references: Roark, Mahoney, Graham, and Lengyel, 1985, *Dev. Biol.* 109: 476-88.

Baldarelli, Mahoney, Salas, Gustavson, Boyer, Chang, Roark, and Lengyel, 1988, *Dev. Biol.* 125: 85-95.

phenotype: Blastoderm-specific locus whose function may involve binding to the DNA (see **molecular biology** section). In the syncytial blastoderm stage, *Term* RNA is distributed uniformly throughout the embryo. In late cellular blastoderm, it is concentrated at the posterior pole. At gastrulation, the RNA is found in the invagination that gives rise to the posterior midgut and proctodeum, the region that will form the ventral furrow, and the antero-dorsal neurogenic region, but it disappears by the end of the period of germ band extension.

cytology: Placed in 75C1-2 on the basis of *in situ* hybridization to the salivary chromosomes at 75C (Roark *et al.*, 1985) and inclusion in *Df(3L)W10 = Df(3L)75A6-10;75C2-4* (Baldarelli *et al.*, 1988).

molecular biology: Gene cloned (Roark *et al.*). A 5.8 kb. *EcoRI* fragment subcloned and restriction mapped and the nucleotide sequence of the *Term* coding region determined (Baldarelli *et al.*, 1988). There are two transcription units, α and σ , which appear to have identical coding regions and to be oriented in opposing directions with 3' ends adjacent. The longest open reading frame begins at the methionine codon at position 155 and extends without interruption to the tyrosine codon at 1436; it is believed to be the ORF that encodes the *Term* protein. The RNA contains 150 nucleotides of untranslated sequences at its 5' end. A potential regulatory region between positions -387 and -431 contains two identical inverted repeats. The protein predicted from the nucleotide sequences has a TFIIIA-like finger which may bind to the DNA (Baldarelli *et al.*, 1988).

terraced: see *ter*

tet: tetraltera

location: 2- [located on 3 by Vilee (1942); location later revised by Goldschmidt (1952, 1953), who considered *tet* to be multifactorial with the main factor on 2 and enhancing factors on X and 3; placed on 2 by E.B. Lewis and García-Bellido].

origin: Spontaneous.

discoverer: Goldschmidt, 34l.

references: 1940, *Material Basis of Evolution*, Yale University Press, p. 325 (fig.).

Vilee, 1942, *Univ. Calif. Publ. Zool.* 49: 125-84.

Goldschmidt, Hannah, and Pitternick, 1951, *Univ. Calif. Publ. Zool.* 55: 67-294.

Goldschmidt, 1952, *J. Exp. Zool.* 119: 405-60.

1953, *J. Exp. Zool.* 123: 79-114.

James and Bryant, 1981, *Dev. Biol.* 85: 39-54.

phenotype: Wings reduced; may have tendency to be haltere-like. Duplications of the notum as well as the costa and ventral hinge of the wing accompanied by deficiencies for the rest of the wing; outgrowths from wing formed by duplications of the costa; no evidence for wing to haltere transformation observed by James and

Bryant (1981). Little cell death in third larval instar. Expression variable; overlaps wild type; enhanced by *os*^S (E.B. Lewis). Penetrance temperature-sensitive (0-1% at 29°, 35% at 15°). RK3.

alleles: *tet* (Goldschmidt; Vilee); *tet*³ (García-Bellido), **tet*^{Bd} (Goldschmidt; Piternick).

tetanic: see *tta*

tetraltera: see *tet*

tetrapter: see *ttr*

tetrodotoxin-sensitive: see *ttx*

Tevel: see *Fs(3)Sz21*

***tf: trefoil**

location: 2-55 (between 50 and 60).

discoverer: Morgan, 13k.

references: Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 244 (fig.).

phenotype: Scutellum darkened. Base of trident pattern and back of head have extra areas of dark pigmentation. Classification uncertain. RK3.

tfd: two-faced (J.C. Hall)

location: Complex; major components on 3.

origin: Spontaneous.

references: Lipschitz and Kankel, 1985, Dev. Biol. 107: 1-12.

phenotype: Flies show variable expression of eye-antennal abnormalities on the dorsal head cuticle, ranging from cuticular deformities with missing bristles and ocelli to duplications of the antennae (frequent), eyes, and labial palps (infrequent). The extra antenna is usually a perfect copy of the normal one, but the extra eye is much smaller, with irregular facets. In the late third instar, larvae often have extra tissue in their eye-antennal disks; in adults there is everted but not externalized disc tissue in the head. Nerves from extra antennae were traced with silver staining and Golgi and tannic acid and found to frequently connect to and make normal projections in the brain; no connections between the extra eyes and the brain were found. The infrequent palp duplications were not examined in detail.

other information: The *tfd* phenotype originally appeared with low frequency (about 1%) in a *In(3LR)DcxF, ru h D Sb sr e^S/Gl Ubx e⁴* stock, was separated from the *DcxF* chromosome and selected for until penetrance was almost complete and 20-40% of the flies showed strong *tdf* expression. Both penetrance and expression decreased rapidly when selection was relaxed except when *tdf* enhancer(s) had accumulated in the stock.

***tft: tufts**

location: 2-102 (between *px* and *bw*).

origin: γ ray induced.

discoverer: R. M. Valencia, 1959.

references: 1959, DIS 33: 99-100.

phenotype: Sternopleural bristles form a dense tuft. Fully penetrant at 20°, poorly so at 25°. RK2.

Tft: Tufted

location: 2-53.6; 1.2 cM to left of *Bl* (Tokunaga, 1967).

origin: X ray induced.

discoverer: Ritterhouse, 52f25.

references: 1952, DIS 56: 68-69.

Arnheim, 1967, Genetics 56: 253-63.

Tokunaga, 1967, DIS 42: 40.

Wright, Hodgetts, and Sherald, 1976, Genetics 84: 267-85.

Ghysen and Richelle, 1979, Dev. Biol. 70: 438-52.

phenotype: Dominant mutation characterized by an increased number of bristles in the postalar, dorsocentral, and scutellar regions; tufts of bristles formed on mesothorax in both homo- and heterozygotes; bristles shorter in homozygotes. Extra bristles located dorsal to halteres at junction of thorax and abdomen. Posterior part of mesonotum appears wider than normal. Homo- and heterozygous females have a greatly reduced scutellum; scutoscutellar suture almost absent; heterozygous males have a nearly normal scutellum (Arnheim, 1967). Small to moderate amounts of fluid tend to remain between the epithelial layers of the wing. Penetrance of extra-bristle character 100%. *Tft* not suppressed by *Df(1)sc19 = Df(1)1A1;1B4-5* but suppressed by *Df(1)260-1 = Df(1)1A1;1B4-6* (García-Bellido, communicated to Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, Cell 40: 327-38). Cell autonomous in mosaics (Arnheim, 1967). Viability and fertility low.

alleles: Single *Tft* allele and a viable point mutant revertant, *Tft*^{rv54} (Wright *et al.*, 1976).

cytology: Probably located in 37A3-6 since reverted to *Tft*⁺ by *In(2L)47 = In(2L)37A2-B1;38A6-C1* (Wright *et al.*, 1976). Uncovered by *Df(2L)TW3 = Df(2L)36F7-37A1;37B2-8* and *Df(2L)TW50 = Df(2L)36E4-F1;38A6-7*.

other information: Extra bristles often very close together; underlying neurons make functional contacts with the CNS (Ghysen and Richelle, 1979). Innervated bristles also found in the metanotum, which has no bristles in wild-type flies (García-Bellido and Deak).

tg: telegraph

location: 2-0.0.

discoverer: Bridges, 16c27.

references: Morgan, Bridges and Sturtevant, 1925, Bibliog. Genet. 2: 237.

Stern and Bridges, 1926, Genetics 11: 507 (fig.), 508-10.

Carlson, 1966, Ohio J. Sci. 66: 340-46.

1970, Ohio J. Sci. 70: 365-71.

Thompson, 1973, DIS 50: 59.

phenotype: Vein L2 has one or more gaps or thin sections. Postscutellar bristles erect or misdirected. Overlaps wild type. RK3.

alleles: Two alleles: *tg* of Bridges and *tg*^C of Carlson (1966, 1970).

Tg: see under *shv* in *dpp* entry

th: thread

location: 3-43.2.

references: Ward and Alexander, 1957, Genetics 42: 42-54.

Korge, 1972, DIS 48: 20.

Mglinetz and Ivanov, 1975, Genetika (Moscow) 11: 88-96.

Ashburner, Richards, and Velissariou, 1980, DIS 55: 196.

phenotype: Aristae threadlike, without side branches. Mutation affects development of tarsal claws at 29°

(Mglinetz and Ivanov, 1975). Deficiency for *th* homozygous lethal (Ward and Alexander, 1957; Korge, 1972). Mutant males and females less successful in mating than wild-type (Burnet, Connolly, and Dennis, 1971, Anim. Behav. 19: 409-15).

alleles: Mutants and rearrangements (other than deficiencies) are described in the following table.

allele	origin	discoverer	ref ^α	cytology
<i>th</i>	spont	Bridges, 22j31	3	
<i>th</i> 3β	γ ray	Velissariou	1	
* <i>th</i> 293γ	X ray	Alexander	3	<i>In(3L)72A2-B1;76A4-B1;79A4-B1</i>
<i>th</i> SS107	X ray		2	
<i>th</i> SS108	X ray	Smith	2	<i>In(3L)63F3-5;72A3-4 + In(3L)68F5;73F</i>

^α 1 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91; 2 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95; 3 = Ward and Alexander, 1957, Genetics 42: 42-54.

β Induced together with *Df(3L)st3 = Df(3L)72E5-F1;73A4-5*.

γ Homozygous lethal.

cytology: Placed between 72A2 and 72C1-2 (Korge, 1972) since uncovered by *Df(3L)th701 = Df(3L)72A2;72D* and *Df(3L)th70kl = Df(3L)71C3-4;72C1-2*; probably in 72B1 according to Ashburner *et al.*, 1980.

Th: Tyrosine-3-hydroxylase (K. White)

location: 3-{21}.

origin: Isolated as a cDNA clone using homology to rat tyrosine hydroxylase (TH).

references: Neckameyer and Quinn, 1989, Neuron 2: 1167-75.

cytology: Placed in 65B by *in situ* hybridization to the salivaries.

molecular biology: Gene cloned and nucleotide sequence of the *Drosophila* cDNA and deduced amino acid sequence of the protein obtained (Neckameyer and Quinn, 1989). A 3.2 kb clone isolated from an adult *Drosophila* head library was analyzed. There is a single open reading frame that encodes a 58 kd protein that is almost 50% identical to rat tyrosine hydroxylase. The protein is specifically recognized by an antibody made against bovine TH. The *Drosophila* gene is expressed in head tissue and first instar larvae. Transcripts of 3.2 and 3.65 kd were detected, the latter appearing to be adult-specific.

other information: The *Drosophila melanogaster* mutant pale has been genetically mapped to the same region (65B) as the rat TH-homologous *Drosophila* cDNA clones, suggesting that *ple* may be a TH mutation. The ability of *Th* DNA to rescue *ple* mutants, however, has not been tested.

***tha: thin arched**

location: 1-27.8.

origin: Induced by S-2-chloroethylcysteine (CB. 1592).

discoverer: Fahmy, 1957.

references: 1959, DIS 33: 93.

phenotype: Fly small, has short thin bristles. Wings arched over abdomen or drooping at sides. Viability and fertility low. RK3.

***thb: thin bristle**

location: 1-48.0.

origin: Induced by triethylenemelamine (CB. 1246).

discoverer: Fahmy, 1951.

references: 1958, DIS 32: 76.

phenotype: Bristles thin, short in female. Occasionally, vibrissae abnormal and eyes rough. Vein L5 sometimes faint or missing beyond posterior crossvein. Viability and fertility good in male and reduced in female. RK2.

thg: throng

location: 2-{0}.

references: Shamanski and Orr-Weaver, 1989.

Genetics: Maternal effect mutant.

cytology: Located at 21B.

thi: thick head

location: 2-72.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1983, DIS 59: 158-60.

1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Homozygous lethal in embryo. Broad head. In combination with *Pc*-like mutants, abdominal transformations occur.

alleles: Two alleles, *thi*¹ and *thi*² isolated as *HM* and *HN*.

thic: thickened aristae

location: 1- (to right of *f*).

origin: Induced by ethyl methanesulfonate.

references: Homyk and Sheppard, 1977, Genetics 87: 95-104.

Homyk, 1977, Genetics 87: 105-28.

phenotype: Aristae and sex combs thickened; dark specks in eye. Lethal at 29°.

thick: see *tk*

thick head: see *thi*

thick legs: see *thl*

thick legs-darker: see *thl-d*

thick vein: see *thv*

thick vein delta: see *thvd*

thick veins: see *tkv*

thickened arista: see *thic*

Thickened arista: see *Ta*

thickened veins: see *thiv*

thickoid: see *tkd*

thickset: see *tht*

thin: see *tn*

thin arched: see *tha*

thin bristle: see *thb*

thin bristles: see *tbs*

thin macros: see *thm*

Third chromosome cold-sensitive paralytic: see *Tcp*

Third chromosome resistant: see *Tcr*

thiv: thickened veins

location: 2-71.4.
origin: Nuclear polyhedrosis virus of *Galleria melonella*.
references: Shandala, 1985, Tsitol. Genet. 19: 179-83.
phenotype: Wing veins thickened. Maximum penetrance at 16°, minimum at 28°; TSP for penetrance between second and third larval instars.

***thl: thick legs**

location: 1-60.7.
origin: Induced by D-p -N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3026).
discoverer: Fahmy, 1955.
references: 1959, DIS 33: 93.
phenotype: Legs short and swollen, particularly posterior pair; swelling most pronounced in tibial and tarsal regions. Wings small and broad, divergent or slightly upheld. Body color slightly dusky and eye color a bit brownish. Male fertile; viability about 20% wild type. RK3.
other information: One allele each induced by CB. 1506 and CB. 1528.

***thl-d:** see **dkl*

***thm: thin macros**

location: 1-48.9.
origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).
references: Fahmy, 1958, DIS 32: 76.
phenotype: Bristles slightly shorter and thinner than normal. Viability and fertility good. RK2.

thorny: see *tny*

thr: three rows

location: 2-86.
origin: Induced by ethyl methanesulfonate.
references: Nüsslein-Volhard, Wieschaus, and Kluding, 1983, DIS 59: 158-60.
 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.
 Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.
phenotype: Larval denticles sparse and arranged in few rows, all pointing posteriorly. Denticles larger than normal. Apparently no mitoses after the first post-blastoderm mitosis, resulting in embryos with fewer and larger cells than normal. TSP 4-9 h. Homozygous lethal in embryo.
alleles:

allele	synonym	comments
<i>thr</i> ¹	<i>thr</i> ^{IB}	
<i>thr</i> ²	<i>thr</i> ^{IL}	
<i>thr</i> ³	<i>thr</i> ^{IIV}	temperature-sensitive
<i>thr</i> ⁴	<i>thr</i> ^{BH}	hybrid dysgenic
<i>*thr</i> ⁵		
<i>*thr</i> ⁶		
<i>*thr</i> ⁷		
<i>*thr</i> ⁸		

cytology: Placed in 55A-F.

thread: see *th*

thread bristle: see *trb*

three rows: see *thr*

***tht: thickset**

location: 1-42.1.
origin: Induced by DL-p -N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007)
discoverer: Fahmy, 1953.
references: 1959, DIS 33: 93.
phenotype: Fly reduced in size, more in length than breadth, giving a stocky appearance. Eye shape slightly altered, has a few deranged facets. Viability about 10% wild type. Male fertile. RK3.

thv: thick vein

location: 1-49.7.
origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).
discoverer: Fahmy, 1956.
references: 1958, DIS 32: 76.
phenotype: Veins thick, especially at junction of L1 and L2. Wings short and broad; marginal hairs irregular. Eyes small and dark. Body color rather pale. Eclosion delayed. Male viable and fertile. Female fertility sub-normal. RK2.

***thvd: thick vein delta**

location: 1-55.2.
origin: Induced by L-p -N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).
discoverer: Fahmy, 1955.
synonym: *dtv: delta vein*.
references: 1958, DIS 32: 69-70.
phenotype: Wings slightly short and broad; has extra venation, especially around L2, which usually ends in a delta. Anal plates and genital arch deformed; genital region protruding. Male fertile but viability about 50% normal. RK3.

***ti: tarsi irregular**

location: 2-55.9.
origin: Spontaneous.
discoverer: Ives, 38k5.
references: 1942, DIS 16: 48.
phenotype: Third and fourth tarsal segments more or less fused and swollen. Eyes slightly rough. Viability sub-normal. RK2.

tiddler: see *tdd*

tilt: see *tt*

tiny: see *ty*

tiny bristle: see *tb*

tiny bristle 2: see *tyb2*

tiny bristleoid: see *tbd*

tiny chaetae: see *tc*

tiny eggs: see *tne*

tiny ovaries: see *tov*

tiny wing: see *tyw*

tinylike: see *tyl*

tipA: temperature induced paralysis A

(J.C. Hall)

location: 2-44.0.**origin:** Induced by ethyl methanesulfonate.**references:** Kulkarni and Padhye, 1982, Genet. Res. 40: 191-99.**phenotype:** Becomes paralyzed rapidly at 38°C; time required for recovery (after return to 23°) increases quickly with duration of exposure to high temperature.**alleles:** *tipA*¹ and *tipA*², the latter of which apparently leads to more rapid heat-induced paralysis and a longer recovery time.**TipB (J.C. Hall)****location:** 2-109.9.**origin:** Induced by ethyl methanesulfonate.**references:** Kulkarni and Padhye, 1982, Genet. Res. 40: 191-99.**phenotype:** Homozygous lethal; heterozygotes become rapidly paralyzed at 38°C and recover fairly quickly when temperature is lowered.**tipC (J.C. Hall)****location:** 2-35.3.**origin:** Induced by ethyl methanesulfonate.**references:** Kulkarni and Padhye, 1982, Genet. Res. 40: 191-99.**phenotype:** Becomes paralyzed gradually at high temperatures (the higher the temperature, the faster the paralysis); recovery from paralysis, after temperature lowered, is somewhat slow, and time required for this increases with longer heat exposure.**tipD (J.C. Hall)****location:** 2-110.**origin:** Induced by ethyl methanesulfonate.**references:** Kulkarni and Padhye, 1982, Genet. Res. 40: 191-99.**phenotype:** Becomes paralyzed rapidly at 38°C; recovers fairly quickly when temperature is lowered.**other information:** Complemented lethality associated with *TipB*. *tipD/TipB* adults exhibit same heat-induced paralysis kinetics as does *TipB/+*; these mutations probably define separate genes in spite of similar map locations.**tipE (J.C. Hall)****location:** 3-13.5.**origin:** Induced by ethyl methanesulfonate.**references:** Kulkarni and Padhye, 1982, Genet. Res. 40: 191-99.

Ganetzky, 1986, J. Neurogenet. 3: 19-31.

Jackson, Wilson, and Hall, 1986, J. Neurogenet. 3: 1-17.

phenotype: Mutant becomes paralyzed rapidly at 38°; recovers within seconds when temperature is lowered. No apparent increase in recovery times with longer exposure to high temperature (Jackson *et al.*, 1986). When in a double mutant combination with *para*^{ts1}, *tipE* results in action potential failures in recordings from larval motor neurons at 33°, a temperature at which either *para*^{ts1} or *tipE* alone seems to have normal nerve conduction (Ganetzky, 1986). Adult action potential failures in *nap*^{ts}; *tipE* double mutants occur at a lower temperature (31°) than in wild type (Ganetzky, 1986); flies of this double-mutant genotype exhibit poorer viability and become paralyzed at lower temperatures than eithermutant alone. The *para*^{ts1}; *tipE* double mutants also survive poorly, showing unconditional lethality in combinations with certain *para* alleles; In *para/+*; *tipE/tipE* mutants, *para* alleles become semi-dominant for heat-induced paralysis (Ganetzky, 1986).Biochemically, *tipE* leads to reduced levels of saxitoxin binding activity (not temperature-dependent) in adult head extracts, but the dissociation constants for such binding is normal (Jackson *et al.*, 1986). The mutant is slightly resistant to veratrine and shows enhanced tetrodotoxin sensitivity (Jackson *et al.*, 1986). Sodium current density in cultured embryonic neurons is about half that recorded using "tight-seal" techniques from wild-type cells (O'Dowd and Aldrich, 1988, J. Neurosci. 8: 3633-43).When rather lengthy heat treatments are given to *sei*^{ts}, the fairly long recovery times are partially suppressed by *tipE*. Partial suppression of *Sh*-induced leg shaking is also effected by *tipE* (Jackson *et al.*, 1986).**cytology:** Located in 64A6-64B12 (L.M. Hall and colleagues) since included in *Df(3L)HR277* = *Df(3R)63B12-C1;64B12* but not in *Df(3L)HR298* = *Df(3L)63B6;64A6*.**tipF (J.C. Hall)****location:** 3-15.2.**origin:** Induced by ethyl methanesulfonate.**references:** Kulkarni and Padhye, 1982, Genet. Res. 40: 191-99.**phenotype:** Kinetics of paralysis at a series of elevated temperatures like that associated with *tipC*, though *tipF* requires substantially longer times to recover after temperature lowered.**other information:** Complements *tipE*.**tk: thick****location:** 2-55.3.**discoverer:** Guthrie, 24k.**references:** 1925, Am. Naturalist 59: 479-80.**phenotype:** Legs and especially tarsi thick. Wings somewhat short and broad, has slight *px*-like effect. According to Waddington [1942, Proc. Zool. Soc. London Ser. A., 111: 181-88 (fig.)], these effects result from inadequate contraction of the legs and whole pupa after inflation period. RK2.**cytology:** Placed in region between 42A2 and 42B1 on the basis of its inclusion in inverted segment of *In(2R)Cy* = *In(2R)42A2-3;58A4-B1* as well as in *Df(2R)M41A-vg11* = *Df(2R)40F-41A1;42A19-B1* (Morgan, Schultz, Bridges, and Curry, 1939, Carnegie Inst. Wash. Year Book 38: 273-77).**tkd: thickoid****location:** 2-40 (30 to 50).**discoverer:** Bridges, 33d25.**phenotype:** Fly large and thickset, has thick legs. Wings blunt at tip. Eyes large and slightly rough. Male genitalia sometimes rotated. Fertile; viability about 50% wild type. RK3.**tko: technical knockout (J.C. Hall)****location:** 1-1.0 (.006 to left of *z* according to Judd *et al.*, 1972).**references:** Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56.

Shannon, Kaufman, Shen, and Judd, 1972, Genetics

72: 615-38.

Lim and Snyder, 1974, *Genet. Res.* 24: 1-10.

Liu and Lim, 1975, *Genetics* 79: 601-11.

Ganetsky and Wu, 1982, *Genetics* 100: 597-614.

Burg and Wu, 1987, *Neurosci. Abstr.* 13: 619.

Royden, Pirota, and Jan, 1987, *Cell* 51: 165-73.

Perrimon, Engstrom, and Mahowald, 1989, *Genetics* 121: 333-52.

phenotype: Most *tko* alleles are lethal in all genetic combinations. In homo- and hemizygotes of the viable allele *tko*^{25t}, however, mechanically shocked adults fall over and are briefly immobilized; after recovery, there is a refractory period during which sensitivity to further such shocks is reduced. *tko*^{25t} is considered a semi-lethal by Judd, 1972, who finds that survivors exhibit a fine bristle phenotype. This allele is also a heat-sensitive paralytic (Burg and Wu, 1987). Its aberrant behavior is suppressed by *nap*^{ts} when the latter is kept at its permissive temperature (Ganetsky and Wu, 1982). When heterozygous with deficiencies for the locus, most *tko* alleles are lethal.

alleles: One viable and nine lethal alleles have been described.

allele	origin	synonym	ref ^α	comments
<i>tko</i> ¹	EMS	<i>l(1)EC229</i>	5	lethal
<i>tko</i> ²	EMS	<i>l(1)VA256</i>	5	lethal
<i>tko</i> ³	EMS	<i>l(1)VE691</i>	5	lethal
<i>tko</i> ^{4β}		<i>l(1)64k11</i>	2	lethal
<i>tko</i> ^{15p}	NNG		4	lethal
<i>tko</i> ^{25t}	NNG		1, 3, 4, 8	quasi-viable and behaviorally defective; female hemizygotes lethal
<i>tko</i> ^{e75}	EMS		6	lethal
<i>tko</i> ^{k11γ}	dimethyl sulfoxide + X rays		4, 9	larval lethal
<i>tko</i> ^{m78}	MMS		7	lethal
<i>tko</i> ^{m100}	MMS		7	lethal

^α 1 = Burg and Wu, 1987, *Neurosci. Abstr.* 13: 619; 2 = García-Bellido and Robbins, 1983, *Genetics* 103: 235-47; 3 = Ganetsky and Wu, 1982, *Genetics* 100: 597-614; 4 = Judd, Shen, and Kaufman, 1972, *Genetics* 71: 139-56; 5 = Lefevre and Watkins, 1986, *Genetics* 113: 869-95; 6 = Lim and Snyder, 1974, *Genet. Res.* 24: 1-10; 7 = Liu and Lim, 1975, *Genetics* 79: 601-11; 8 = Royden, Pirota, and Jan, 1987, *Cell* 51: 165-73; 9 = Shannon, Kaufman, Shen, and Judd, 1972, *Genetics* 72: 615-38.

^β Discovered by Geer.

^γ Discovered by Alexander (Judd *et al.*, 1972).

cytology: Placed in 3A2 (Judd *et al.*, 1972) based on uncoverage of lethality by *Df(1)w-rJ1 = Df(1)3A1-2;3C2-3*, *Df(1)X12 = Df(1)2F5-3A1;3B5-3C1*, and *Df(1)62g18 = Df(1)3A1-2;3A4-5*, and complementation by *Df(1)64c4 = Df(1)3A3-4;3C2-3*.

molecular biology: *tko* region cloned, originally by microdissection (Mariani, Pirrotta, and Manet, 1985, *EMBO J.* 4: 2045-52) and identified molecularly by transformation rescue. A 3.1 kb *Xho*I/*Bam*HI fragment covers lethality caused by *tko*^{k11} and behavioral defects of *tko*^{25t} (Royden *et al.*, 1987). This genomic DNA fragment encodes one detectable transcript of 0.68 kb. Predicted protein shows considerable sequence similarity to ribosomal protein S12 from *Euglena gracilis* chloroplasts and *E. coli* (Royden *et al.*, 1987).

Tkr: Tyrosine kinase related

location: 2- {107}.

origin: Induced by ethyl methanesulfonate.

synonym: *dTKR* (Haller *et al.*, 1987); *g2* (Côte *et al.*, 1987).

references: Côte, Preiss, Haller, Schuh, Kienlin, Seifert, and Jäckle, 1987, *EMBO J.* 6: 2793-2801.

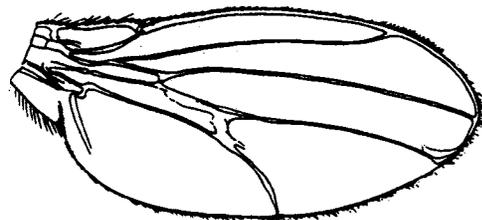
Haller, Côte, Brönnner, and Jäckle, 1987, *Genes Dev.* 1: 862-67.

phenotype: No mutant phenotype identified as yet. Transcripts initially expressed in the syncytial blastoderm stage (Haller *et al.*, 1987; Côte *et al.*, 1987), accumulating temporarily at dorsal-lateral positions in the embryo. At the end of blastoderm, the transcripts accumulate unevenly and are restricted to the dorsal region along 20-85% of the egg length. During gastrulation they are found in the dorsal region of the cephalic furrow and cover the neurogenic ectoderm in the procephalic region, disappearing from anterior to posterior during the extended germband stage. Later *Tkr* is expressed anew in the developing nervous system, including the brain, but disappears completely at the end of embryogenesis (Haller *et al.*, 1987).

cytology: Located in 60F1 on the basis of *in situ* hybridization. Also, *Tkr* included in *Df(2R)IIX62 = Df(2R)60E9-10;60F1-2* (Côte *et al.*, 1987).

molecular biology: To determine the molecular structure of the *Tkr* gene, the cDNA clone, the corresponding genomic DNA, and the genomic DNA upstream of the 5' end of the cDNA were sequenced and the amino acids predicted. There is a single open reading frame of three exons (a 5' exon of 1357 bp, a small exon of 132 bp and a 3' exon of 1895 bp separated by two introns of 2500 and 3800 bp, respectively) which would encode a 753-amino-acid polypeptide. A single major transcript (4 kb long) is indicated by northern blot analysis (Côte *et al.*, 1987).

other information: Weak homology has been demonstrated between the putative *Tkr* protein and various serine, threonine, and tyrosine kinases and related oncogenes. The presence of putative ATP-binding and tyrosine autophosphorylation sites in the *tkr* sequence also suggests a tyrosine kinase function (Haller *et al.*, 1987).



tkv: thick veins

Edith M. Wallace, unpublished.

tkv: thick veins

location: 2-16.

references: Kotarski, Pickert, and MacIntyre, 1983, *Genetics* 105: 371-86.

Reuter and Szidonya, 1983, *Chromosoma*, 88: 277-85.

Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.
 Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-69.
 Szidonya and Reuter, 1988a, DIS. 67: 77-79.
 1988b, Genet. Res. 51: 197-208.

phenotype: Two types of mutants have been described, recessive lethals and visibles (viable as adults). Some of the recessive lethals die as embryos, lacking dorsal hypoderm (Nüsslein-Volhard *et al.*, 1984). The wing veins of the viable mutants are thickened and branched in the region of crossveins, near end of L2, and elsewhere. Sometimes there is a blister near the posterior crossvein in female flies; L4 sometimes shortened, especially in females. Expression more extreme at 19° than at higher temperatures and in females than in males. Some heteroallelic combinations are lethal; others are viable with thick veins and thoracic abnormalities (Szidonya and Reuter, 1988b).

alleles: Lethal and viable alleles are listed in the following table. Deficiencies for *tkv* are described in the rearrangement section.

alleles	origin	discoverer	synonym	ref ^α	comments
<i>tkv</i> ¹	spont	Nichols-Skoog, 33b25			homozygous viable with thick veins
<i>tkv</i> ²	spont	Bridges, 34e20			homozygous viable with thick veins
<i>tkv</i> ³	EMS	Szidonya	<i>tkv</i> ^{Sz3}	1	homozygous viable with thick veins; <i>Tp(2;3)25A2-3;25D5-E1;69C</i>
<i>tkv</i> ^{4β}	EMS	Szidonya	<i>tkv</i> ^{a12}	1, 2	homozygous lethal
<i>tkv</i> ⁵	EMS	Szidonya	<i>tkv</i> ^{a33}	1, 2	homozygous lethal
<i>tkv</i> ^{6β}	EMS	Szidonya	<i>tkv</i> ^{Sz15}	1, 2	homozygous lethal; hemizygous viable with thick veins;
<i>tkv</i> ^{7γ}	EMS	Nüsslein-Volhard	<i>str</i> ¹⁰	3	homozygous lethal
<i>tkv</i> ^{8γ}	EMS	Nüsslein-Volhard	<i>str</i> ^{11B}	3	homozygous lethal

^α 1 = Szidonya and Reuter, 1988a, DIS 67: 77-79; 2 = Szidonya and Reuter, 1988b, Genet. Res. 51: 197-208; 3 = Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-69.

^β Viable with strong *tkv* phenotype over *Tp(2;3)tkv*³ (Szidonya and Reuter, 1988b).

^γ Allelism with *tkv* indicated by location, failure to complement lethal and viable *tkv* alleles, and thick vein and abnormal thorax phenotype over *Tp(2;3)tkv*³ (Szidonya and Reuter, 1988b).

cytology: Located in 25D5-E1 (Szidonya and Reuter, 1988a, 1988b); in the region of overlap of *Df(2L)tkv-Sz3* = *Df(2L)25A2-3;25D5-E1* and *Df(2L)cl-h2* = *Df(2L)25D5-6;26A7-8*.

Tl: Toll

location: 3-91.

discoverer: Wieschaus and Nüsslein-Volhard.

synonym: *Fs(3)Tl* (dominant allele); *mel(3)9*, *mel(3)10* (recessive alleles).

references: Campos-Ortega, 1983, Wilhelm Roux's Arch. Dev. Biol. 192: 317-26.

Anderson and Nüsslein-Volhard, 1984, Nature 311: 223-27.

Anderson, Jürgens, and Nüsslein-Volhard, 1985a, Cell 42: 779-89.

Anderson, Bokla, and Nüsslein-Volhard, 1985b, Cell 42: 791-98.

Anderson and Nüsslein-Volhard, 1986, Symp. Soc. Dev. Biol. 44: 177-94.

Anderson, 1987, Trends Genet. 3: 91-97.

Carroll, Winslow, Trombly, and Scott, 1987, Develop-

ment 99: 327-32.

Gerttula, Jin, and Anderson, 1988, Genetics 119: 123-33.

Hashimoto, Hudson, and Anderson, 1988, Cell 52: 269-79.

Erdélyi and Szabad, 1989, Genetics 122: 111-27.

phenotype: Maternal expression of the Toll gene is required for the normal production and distribution of positional information in the embryo (Anderson *et al.*, 1985); zygotic expression is required to maintain viability in early larvae (Gerttula *et al.*, 1988). Toll mutants and deficiencies occurring in the mother result in lethal abnormalities in the pattern of gastrulation and the differentiation of cuticular structures in the offspring. When null alleles and deficiencies are homozygous in the zygote, delayed development and early lethality result.

Females heterozygous for dominant Toll alleles are sterile, their lethal embryos being partially ventralized regardless of their genotype. Dorsventral polarity is present; a furrow is formed in the midventral region, but the lateral cephalic fold is shifted to the dorsal side and the normal dorsal folds are missing. The cuticle lacks dorsal hairs, filzkörper, spiracles, head sensory organs, and a head skeleton; there are patches of denticles extending around the entire dorsoventral circumference of the embryo (Anderson *et al.*, 1985a). The ventral nervous system is also expanded (Campos-Ortega, 1983). Embryos produced by females hemizygous for some dominant alleles (*Tl*¹/*Df*; *Tl*³/*Df*) are ventralized, but the embryos of other hemizygotes (*Tl*²/*Df*; *Tl*⁴/*Df*) are dorsalized, all cells behaving at gastrulation and in differentiation like wild-type dorsal cells. In embryos derived from *Tll*+ females, virtually the entire ectoderm capable of neurogenesis in response to absence of *Dl* function (Campos-Ortega, 1983, Wilhelm Roux's Arch. Dev. Biol. 192: 317-26).

Whereas females heterozygous for recessive alleles of *Tl* are fertile, homozygous *Tl*-recessive females are viable but sterile, their lethal embryos lacking dorsoventral polarity and forming no ventral furrow at gastrulation. In most recessive alleles (*Tl*^{r5}, *Tl*^{r6}, *Tl*^{r7}), the embryos are partially dorsalized with laterally derived structures (Anderson *et al.*, 1985a); for example, *Tl*^{r6} embryos differentiate dorsal hairs, filzkörper, and ventral denticle bands of nearly normal width, but lack mesoderm (Anderson and Nüsslein-Volhard, 1986). In one allele (*Tl*^{r4}), however, embryos have no dorsal hairs and show rings of denticles as in *Tl*^D embryos (Anderson *et al.*, 1985a). Hemizygotes for the Toll-recessives resemble the corresponding homozygotes in phenotype.

A number of Toll alleles were obtained as reversions of the Toll-dominant phenotype (see table). When crossed to wild-type males, females heterozygous for a null-type reversion are fully fertile; however, when crossed to males who are also heterozygous for a Toll null, these females produce *Tl*-homozygotes who are zygotic lethals, dying as early larvae and producing no Toll transcript. Heteroallelic combinations of reversions such as *Tl*^{r1}/*Tl*^{r2} produce sterile females with lethal dorsalized embryos. Females carrying combinations of certain reversions and Toll-dominant (or Toll-recessive) alleles produce embryos with phenotypes like those of Toll-dominant (or Toll-recessive) hemizygotes. Most of the reversions, when in *trans* to deficiencies, result in females with dorsalized embryos, but a few hemizygous

reversion females (Tl^{rv21} , Tl^{rv22} , Tl^{rv23}) produce ventralized embryos (Hashimoto *et al.*, 1988).

The lethal embryos of $Df(3R)Tl-X/Df(3R)ro-XB3$ (null) females (Hashimoto *et al.*, 1988), are completely dorsalized, never making ventral furrows, filzkörper, or denticles; their germ bands fail to extend; no Toll transcript is produced in these embryos except when contributed by wild-type fathers (Gerttula *et al.*, 1988). The 97D1-2 breakpoint of the Toll deficiency $Df(3R)Tl-X$ maps within the 6.0 kb *EcoRI* fragment of a Toll clone (Hashimoto *et al.*, 1988). Injection of wild-type cytoplasm into embryos of Toll-deficient females restores the wild-type dorsoventral pattern, the site of the injection determining the midventral part of the pattern (Anderson *et al.*, 1985b); (also see **molecular biology** section).

alleles: Dominant, recessive, and revertant Toll alleles are listed in the following table. Six HD-induced revertants are mentioned but not named (Hashimoto, Hudson, and Anderson, 1988, Cell 52: 269-279). Deficiencies are listed in the rearrangement section.

allele	origin	allele	ref ^α	comments	cytology
Tl^1	EMS		2,5	dominant	
Tl^2	EMS	Tl^{5B}	1,2,5	dominant	
Tl^3	EMS	Tl^{9Q}	1,2,5,6	dominant	
$Tl^{4β}$	EMS	Tl^{84C}	2,5	dominant	
$Tl^{5γ}$	HD	$Tl^{9QRPBR14}$	4,6	dominant	
Tl^6	EMS	Tl^{2b}	4	semidominant	
Tl^7	EMS	Tl^{3c}	4	dominant	
Tl^8	EMS	Tl^{10b}	4	dominant	
Tl^9	EMS	Tl^{18a}	4	dominant	
Tl^{10}	EMS	Tl^{20i}	4	semidominant	
$Tl^{r1δe}$	EMS	Tl^{26}	2,5	recessive	
$Tl^{r2ε}$	EMS	Tl^{r444}	2,5	recessive	
$Tl^{r3ε}$	EMS	Tl^{r632}	2,3,5	recessive	
$Tl^{r4ζ}$	EMS	Tl^{rm9}	2,4	recessive	
Tl^{r5}	EMS	Tl^{LB1}	4,5	recessive	
Tl^{r6}	EMS	Tl^{rB1}	4,5	recessive	
Tl^{r7}	EMS	Tl^{rB2}	4,5	recessive	
Tl^{rv1}	X ray	Tl^{RXA}	2	revertant	
Tl^{rv2}	X ray	Tl^{RXD}	2	revertant	
Tl^{rv3}	X ray	Tl^{RXE}	2	revertant	$T(2,3)40-41;97D$
Tl^{rv4}	X ray	Tl^{RXH}	2,5	null revertant	
Tl^{rv5}	X ray	Tl^{RXJ}	2	revertant	$In(3R)97D;98F$
Tl^{rv6}	X ray	Tl^{RXZ}	2	revertant	$In(3R)97D;99E-F$
Tl^{rv7}	EMS	Tl^{5BREF}	2	null revertant	
Tl^{rv8}	EMS	Tl^{5BREH}	2	null revertant	
Tl^{rv9}	EMS	Tl^{5BREJ}	2	null revertant	
Tl^{rv10}	EMS	Tl^{5BREL}	2	null revertant	
Tl^{rv11}	EMS	Tl^{5BREM}	2	null revertant	
Tl^{rv12}	EMS	Tl^{5BREN}	2	null revertant	$In(3R)97D;99B$
Tl^{rv13}	EMS	Tl^{5BREQ}	1,2,5	null revertant	
Tl^{rv14}	EMS	Tl^{5BRER}	2	null revertant	
Tl^{rv15}	EMS	Tl^{5BRES}	2	null revertant	
Tl^{rv16}	EMS	Tl^{5BREV}	1,2,5	null revertant	
Tl^{rv17}	EMS	Tl^{5BREW}	2	null revertant	$In(3R)97A;97D + Df(3R)97B;97D$
$Tl^{rv18η}$	X ray	Tl^{5BRXV}	2,5	null revertant	$In(3R)74A;97D$
Tl^{rv19}	EMS	Tl^{9QRE}	1,2,5	null revertant	$In(3R)97D;98C-D$
Tl^{rv20}	X ray	Tl^{84CRXB}	2	revertant	
$Tl^{rv21θ}$	HD	Tl^{9QRPA}	6	null revertant	
$Tl^{rv22θ}$	HD	Tl^{9QRPB}	6	null revertant	
$Tl^{rv23θ}$	HD	Tl^{9QRPL}	6	null revertant	
Tl^{rv24}		Tl^{9QRPS}	5	null revertant	
Tl^{rv25}		Tl^{9QRPU}	5	null revertant	

^α 1 = Anderson, Bokla, and Nüsslein-Volhard, 1985, Cell 42: 791-98; 2 = Anderson, Jürgens, and Nüsslein-Volhard, 1985, Cell 42: 779-89; 3 = Carroll, Winslow, Trombly, and Scott, 1987, Development 99: 327-32; 4 = Erdélyi and Szabad, 1989, Genetics 122: 111-27; 5 = Gerttula, Jin, and Anderson, 1988, Genetics 119: 123-33; 6 = Hashimoto, Hudson, and Anderson, 1988, Cell 52: 269-79.

- β Pattern normalized by extra copy of wild-type allele. All $Tl^4/++$ embryos make dorsal hairs and one quarter of these embryos make filzkörper.
- γ Derivative of Tl^{rv22} that has restored the dominant Tl^3 phenotype; apparently associated with precise excision of the P element (Hashimoto *et al.*, 1988).
- δ Isolated on the basis of a leaky dominant dorsalized phenotype.
- ε Temperature sensitive for the maternal effect, showing stronger dorsalization of the embryonic pattern at 29° than at 18°; also temperature sensitive for viability. TSP for the maternal effect begins slightly before pole cell formation and ends in midsyncytial blastoderm in the offspring of Tl^{r7} females. TSP for zygotic viability begins late in embryogenesis and extends into the second larval instar in Tl^{r5} and Tl^{r6} mutants.
- ζ Discovered by T. Rice, who named it *mel(3)9*. Unique lateralized pattern. Cuticular phenotype of embryos resemble that of Toll-dominant embryos, but denticles finer and less heavily pigmented and body shape often an elongated tube.
- η 97D breakpoint located in the 6.0 kb *EcoRI* fragment of the Toll clone $Tp1$ (Hashimoto *et al.*, 1988).
- θ All HD-induced revertants are derivatives of Tl^3 .

cytology: Placed in 97D1-2 since uncovered by $Df(3R)Tl-X = Df(3R)97B;97D1-2$ and $Df(3R)ro-XB3 = Df(3R)97D1-2;97D9$.

molecular biology: Toll has been cloned using P element tagging and the nucleotides have been sequenced (Hashimoto *et al.*, 1988). The Toll clone $Tp1$ contains 15 kb of DNA flanking the P element insertion. Nucleotide sequencing reveals one long open reading frame, with the initiation codon at nucleotide 575 and the termination codon at 3866; it could encode a protein of 1097 amino acids [thought to be a transmembrane protein made up of a cytoplasmic domain and an extracytoplasmic domain containing multiple copies of a 24-amino-acid leucine-rich sequence in two blocks (Hashimoto *et al.*, 1988)]. This mRNA is transcribed (Hashimoto *et al.*, 1988) and also zygotically (Gerttula *et al.*, 1988); no transcript is found when Tl^- females are mated to Tl^- males, but the 5.3 kb mRNA appears in the embryo when either or both parents carry Tl^+ . A 5.3 kb hybrid-selected RNA abundant in young embryos (and also in older embryos and pupae) rescues the Tl^- mutant phenotype when injected at a posterior ventral site; a wild-type gastrulation pattern and ventral and lateral cuticular structures are produced in these injected Tl^- embryos. The sequence of cDNAs indicates that *Tl* encodes an integral membrane protein with a cytoplasmic and an extracytoplasmic domain, the latter containing 15 repeats of a 24 amino acid leucine-rich sequence occurring in both human and yeast membrane proteins (Hashimoto *et al.*, 1988) and the former showing striking homology to human interleukin-1 receptor (Gay and Kieth, 1991, Nature 351: 355-56).

other information: Females carrying the dominant allele Tl^3 , when combined with the mutants *gd*, *ndl*, *pip*, *snk*, or *ea*, produce embryos that are lateralized like embryos derived from Tl^{r8} females; these embryos lack dorsal-most and ventralmost pattern elements, and have rings of denticles (Anderson *et al.*, 1985a). Some alleles of *ea* increase the probability that the temperature-sensitive alleles Tl^{r5} , Tl^{r6} , and Tl^{r7} will survive. An interaction has been reported between the recessive allele Tl^{r7} and *dpp* (Irish and Gelbart, 1987, Genes and Development 1: 868-79). Double mutants of Tl^3 and *dl* produce embryos that are completely dorsalized and indistinguishable from the embryos of *dl* homozygotes. Females carrying Tl^2 or Tl^4 in combination with *gd*, *ndl*, or *dl* also produce dorsalized embryos.

tlc: tender little chaetae**location:** 1- {0.5}.**phenotype:** Short thin bristles.**alleles:** Four alleles induced.

allele	origin	discoverer	synonym	comments
<i>tlc</i> ¹ α	EMS	Lefevre	<i>l(1)DA654</i>	lethal
<i>tlc</i> ² α	EMS	Lefevre	<i>l(1)VDM30</i>	viable
<i>tlc</i> ³	EMS	Lefevre	$\Omega 247$	viable
<i>tlc</i> ⁴	spont	Schalet	<i>l(1)11-76-1</i>	lethal

 ^{α} References: Lefevre and Watkins, 1986, *Genetics* 113: 869-95.**cytology:** Placed in 2B15 (Lefevre).***tld: tolloid*****location:** 3-85.**origin:** Induced by ethyl methanesulfonate.**references:** Jürgens, Kluding, Nüsslein-Volhard, and Wieschaus, 1983, *DIS* 59: 157-58.Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95.**phenotype:** Embryonic lethal. Partially ventralized. Cephalic furrow shifted dorsally; defects in germband extension. Denticle belts extended laterally.**alleles:**

allele	synonym	comments	
<i>tld</i> ¹	<i>tld</i> ^{5H}	weak	
<i>tld</i> ²	<i>tld</i> ^{6B}		
<i>tld</i> ³	<i>tld</i> ^{6P4}	temperature-sensitive	
<i>tld</i> ⁴	<i>tld</i> ^{6P7}		
<i>tld</i> ⁵	<i>tld</i> ^{7H}		
<i>tld</i> ⁶	<i>tld</i> ^{7M}		
<i>tld</i> ⁷	<i>tld</i> ^{7O}		
<i>tld</i> ⁸	<i>tld</i> ^{8L}		
<i>tld</i> ⁹	<i>tld</i> ^{9B}		
<i>tld</i> ¹⁰	<i>tld</i> ^{9D}		
<i>tld</i> ¹¹	<i>tld</i> ^{9K}		
<i>tld</i> ¹²	<i>tld</i> ^{9Q1}		
<i>tld</i> ¹³	<i>tld</i> ^{9Q7}		
<i>tld</i> ¹⁴	<i>tld</i> ^{10E}		strong
<i>tld</i> ¹⁵	<i>tld</i> ^{10F}		
<i>tld</i> ¹⁶	<i>tld</i> ^{FF}		
<i>tld</i> ¹⁷	<i>tld</i> ^T		

cytology: Located in 96B-D based on segmental aneuploidy of translocations (Jürgens *et al.*, 1984).***tll: tailless (J. A. Lengyel)*****location:** 3-102.**references:** Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95.Strecker, Kongsuwan, Lengyel, and Merriam, 1986, *Dev. Biol.* 113: 64-76.Mahoney and Lengyel, 1987, *Dev. Biol.* 122: 464-70.Strecker, Merriam, and Lengyel, 1988, *Development* 102: 721-34.Pignoni, Baldarelli, Steingrimsson, Diaz, Patapoutian, Merriam, and Lengyel, 1990, *Cell* 62: 151-63.**phenotype:** Recessive; zygotic lethal with pattern deletions in anterior and posterior of embryo (but body of normal length due to increase in length of non-deleted pattern elements). Anteriorly, dorsal portion of cephalopharyngeal skeleton is defective (dorsal arms shortened, dorsal bridge unfused), dorsal pouch shortened and sclerotized, much of brain missing. Posteriorly, eighth abdominal segment and telson, Malpighian tubules, hindgut and

much of posterior midgut missing.

alleles: The alleles can be arranged in a series as follows: *tll*¹⁴⁹ > *tll*¹ > *tll*^a = *tll*¹²⁹ > *tll*² > *tll*^{le3}, where *tll*¹⁴⁹ has almost the amorphic phenotype, and *tll*^{le3} is missing only part of the anal pads and part of the hindgut (Strecker *et al.*, 1988; Pignoni *et al.*, 1990; Pignoni and Lengyel, unpublished). The extant *tll* alleles and rearrangements (other than deficiencies) are listed in the following table.

allele	origin	ref ^{α}	comments
<i>tll</i> ¹ β	EMS	1-3	leaky
<i>tll</i> ²	spont	2-4	<i>1n(3R)85F10-86A1;100A6-B1</i>
<i>tll</i> ^a	X ray	2, 3	
<i>tll</i> ¹²⁹	X ray	5	
<i>tll</i> ¹⁴⁹	X ray	4	
<i>tll</i> ^{le3}	EMS	5	

 ^{α} 1 = Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95; 2 = Strecker, Kongsuwan, Lengyel, and Merriam, 1986, *Dev. Biol.* 113: 64-76; 3 = Strecker, Merriam, and Lengyel, 1988, *Development* 102: 721-34; 4 = Pignoni, Baldarelli, Steingrimsson, Diaz, Patapoutian, Merriam, and Lengyel, 1990, *Cell* 62: 151-63; 5 = Merriam, unpublished. ^{β} Originally named *tll*^{L10}.**cytology:** Placed between 100A5-6 and 100B1-2 on the basis of *in situ* hybridization of the cloned *tll* gene and mapping of *tll* into the synthetic deficiency produced by combining *3^{PY}D* of *T(Y;3)A113 = T(Y;3)100A* with *Dp(3;1)150P = Dp(3;1)20F;100B1-2* (Strecker *et al.*, 1988; Pignoni *et al.*, 1990).**molecular biology:** Gene cloned and sequenced (Pignoni *et al.*, 1990). The 2.0 kb *tll* mRNA is maximally expressed in the blastoderm stage embryo. Initial activation of *tll* transcription is in two mirror image symmetrical caps at the poles of the embryo; this expression resolves into a posterior cap and an anterodorsal stripe, consistent with the position on the blastoderm fate map of anlagen which give rise to structures deleted in *tll* embryos. The *tll* gene is also expressed in cells which appear to be neuroblasts of the brain and sensillum precursors of the peripheral nervous system. The conceptual *tll* protein has significant similarity to both the DNA-binding and ligand-binding domains of the members of the steroid receptor superfamily (Pignoni *et al.*, 1990). Transcription of the *tll* gene during early embryogenesis is apparently activated via a phosphorylation cascade containing the membrane-bound, tyrosine kinase receptor-like *tor* gene product and the serine-threonine kinase *phl* gene product. The *tll* gene has a positive effect on gene expression in that it is required for appearance of the seventh stripe of expression of at least two pair-rule genes, *h* and *ftz* (Mahoney and Lengyel, 1987) and for specific domains of expression of *cad*, *hb*, and *fkf* (Mlodzik and Gehring, 1987, *Development* 101: 421-35; Schröder, Tautz, Seifert, and Jäckle, 1988, *EMBO J.* 7: 2881-87; Weigel, Jürgens, Klingler, and Jäckle, 1990, *Science* 248: 495-98). The *tll* gene also has negative effects on gene expression; thus *kni* expression expands posteriorly in *tll* embryos [Pankratz, Hock, Seifert, and Jäckle, 1989, *Nature (London)* 341: 337-40] and *tll* is required for the repression of *Kr* and *ftz* expression that occurs in *tor* gain-of-function embryos [Klingler, Erdélyi, Szabad, and Nüsslein-Volhard, 1988, *Nature (London)* 335: 295-77; Strecker, Halsell, Fisher, and Lipshitz,

1989, Science 243: 1062-66].

Tm1: Tropomyosin 1 (J.C. Hall)

location: 3- {55}. Closely linked to *Tm2*.

origin: Naturally occurring.

synonym: *mTm1*; *cTm*(isoform).

references: Bautch, Storti, Mischke, and Pardue, 1982, J. Mol. Biol. 162: 231-50.

Basi, Boardman, and Storti, 1984, Mol. Cell. Biol. 4: 2828-36.

Karlik, Mahaffey, Coutu, and Fyrberg, 1984, Cell 37: 469-81.

Basi and Storti, 1986, J. Biol. Chem. 261: 817-27.

Karlik and Fyrberg, 1986, Mol. Cell Biol. 6: 1965-73.

Tansey, Mikus, Dumoulin, and Storti, 1987, EMBO J. 6: 1375-85.

phenotype: Structural gene for tropomyosin, a 34,000-dalton protein that acts as a regulator of motility in muscle and nonmuscle cells. At least five tropomyosin isoforms are encoded by *Tm1*, some in embryos (during myogenesis) and myogenic cell culture and others in the myofibrils of adult indirect flight muscles and leg muscles.

cytology: Located in 88F4-5 by *in situ* hybridization with cloned DNA (Bautch *et al.*, 1982). *Tm1* proximal to *Tm2*.

molecular biology: Gene cloned (Bautch *et al.*, 1982; Karlik *et al.*, 1984). The constant proximal coding region (codons 1-257) and the variable distal coding region (codons 258-284) have been restriction-mapped and sequenced (Basi *et al.*, 1984; Karlik *et al.*, 1984; Karlik and Fyrberg, 1986). Transcripts of 2.0, 2.3 and 2.8 kb accumulate in the cytoplasm of early embryos, while transcripts of 1.3, 1.4, 1.6, 1.7, 1.8, and 1.9 kb accumulate in muscles of later embryos and adults, the 1.7 and 1.9 kb transcripts being restricted to the indirect flight muscle. The proximal coding region at the 5' end occupies about 18 kb and is made up of five exons and four introns (according to Basi *et al.*, 1984) or five to nine exons and up to eight introns (according to Karlik and Fyrberg, 1986). The distal coding region at the 3' end includes four variable exons and three introns, the first exon coding for cytoplasmic tropomyosin, and the second and third for heavy tropomyosins found in the indirect flight muscles (Karlik and Fyrberg, 1986). As a result of the variable splicing at least five developmentally-regulated isoforms are specified.

Tropomyosin in the cytoplasmic (cytoskeletal) form of early embryos and Kc cells (a form originally believed to be encoded by a separate gene) has been found to be an alternatively spliced product of *Tm1* (Hanke, Lepinske and Storti, 1987, J. Biol. Chem. 262: 17370-73; Hanke and Storti, 1988, Mol. Cell Biol. 8: 3591-3602).

Tm2: Tropomyosin 2 (J.C. Hall)

location: 3- {55} (closely linked to *Tm1*).

origin: Naturally occurring and induced by ethyl methanesulfonate.

synonym: *mTm1*; *lfm(3)3*.

references: Mogami and Hotta, 1981, Mol. Gen. Genet. 183: 407-17.

Bautch, Storti, Mischke, and Pardue, 1982, J. Mol. Biol. 162: 231-50.

Basi, Boardman, and Storti, 1984, Mol. Cell Biol. 4: 2828-36.

Basi and Storti, 1986, J. Biol. Chem. 261: 817-27.

Karlik and Fyrberg, 1986, Mol. Cell Biol. 6: 1965-73.

Fyrberg and Karlik, 1987, Mol. Cell Biol. 7: 2977-80.

Tansey, Mikus, Dumoulin, and Storti, 1987, EMBO J. 6: 1375-85.

phenotype: Structural gene for tropomyosin that is active in particular muscle lineages in late embryos and adult flies (Basi *et al.*, 1984). A dominant flightless mutant *lfm(3)3* was induced by Mogami and Hotta (1981); this mutant, which was unable to jump or fly, turned out to be a *Tm2* allele, *Tm2*³. Although arranged in sarcomeres, the myofibrils of *Tm2*³ are structurally weak (Karlik and Fyrberg, 1986). The flightless phenotype can be rescued by *P*-element mediated transformation with two copies of the wild-type allele of *Tm2* (Fyrberg and Karlik, 1987); the ability to jump can be restored with one copy of *Tm2*⁺ (Tansey *et al.*, 1987).

alleles: One mutant allele *Tm2*³ [= *lfm(3)3*].

cytology: Located in 88F2-3 by *in situ* hybridization with cloned DNA (Bautch *et al.*, 1982). *Tm2* distal to *Tm1*.

molecular biology: Gene cloned (Bautch *et al.*, 1982). The constant proximal coding region (codons 1-257) includes two exons and occupies 833 nucleotides that are interrupted by a single 62-nucleotide intron between codons 198 and 199 (Karlik and Fyrberg, 1986). The variable distal coding region (codons 258 to 284) includes two alternate exons with large untranslated regions. The proximal coding region of *Tm2* is 1/20th the size of the corresponding *Tm1* coding region, presumably due to deletion of introns in *Tm2* and duplication of exons in *Tm1* (Karlik and Fyrberg, 1986). The complete nucleotide sequence of *Tm2* has been given by Basi and Storti (1986).

In the flightless mutant *Tm2*³, pupae lack the 1.7 and 1.9 kb indirect-flight-muscle transcripts, but accumulate the 1.3 and 1.6 kb transcripts (Karlik and Fyrberg, 1986; Tansey *et al.*, 1987). There is an 8.8 kb insertion of middle-repetitive copia-like DNA in the mutant, interrupting the exon believed to be involved in the synthesis of the indirect flight muscle isoform (Tansey *et al.*, 1987).

***tmc: tonomacrochaetae**

location: 1-17.5.

origin: Induced by D-*p*-N,N-di-(2-chloroethyl)aminophenylalanine (CB. 3026).

discoverer: Fahmy, 1955.

references: 1958, DIS 32: 76.

phenotype: Bristles thin. Abdomen underpigmented, especially in female. Eclosion slightly delayed. Viability and fertility good. RK2.

***tms: tumorous**

location: 1-58.7.

origin: Induced by L-*p*-N,N-di-(2-chloroethyl)aminophenylalanine (CB. 3025).

discoverer: Fahmy, 1954.

references: 1959, DIS 33: 93.

phenotype: Many small, diffuse tumors. Fly slightly small. Both sexes viable and fertile. RK3.

tn: thin

location: 2-85.6 (between *c* and *px*).

origin: Spontaneous.

synonym: *l(2)tn*.

references: Ball, Ball, and Sparrow, 1985, *Dev. Genet.* (Amsterdam) 6: 77-92.

phenotype: Fully penetrant recessive lethal (no homozygous adults recovered). Hatching occurs late and second/third instar moult is delayed. Third instar larvae are long, thin, and rather slow-moving; histological examination shows abnormal muscle structure. Puparium also long and thin. Pupal development of homozygotes incomplete. Since mutants are unable to perform movements necessary for pupation, they die prior to eclosion.

cytology: Placed within 55F. Gene lies between the transposing elements *TE55DE* (=TE178) and *TE56B* (=TE124) and is not lethal over *Df(2R)PC4 = Df(2R)55A;55F*.

***tne: tiny eggs**

location: 3-91.

origin: Induced by ethyl methanesulfonate.

discoverer: Anderson.

phenotype: Female sterile. Very small eggs.

alleles: Four alleles induced.

cytology: Located in 97D9-15; it is uncovered by *Df(3R)ro80b = Df(3R)97D1;97D15* but not by *Df(3R)roXB3 = Df(3R)97D1-2;97D9*.

***tnt: tent**

location: 1-18.0.

origin: X ray induced.

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 93.

phenotype: Wings droop to variable extent. Bristles thin. Fly small. Male sterile. RK2.

***tny: thorny**

location: 1-33.5.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).

discoverer: Fahmy, 1954.

references: 1959, DIS 33: 93.

phenotype: Fly grossly deformed, extremely inviable. Eyes small, very rough, and dull red. Thoracic bristles very short. Wings abnormal, spread, incompletely expanded. Male sterile. RK2.

To: see *Sod*

Told: see *Fs(2)Sz11*

Toll: see *Tl*

tolloid: see *tld*

tom: see *ix*²

Tomaj: see *Fs(3)Sz22*

tomboy: see *ix*²

***ton: tonochaetae**

location: 1-60.1.

origin: Induced by 1:4-dimethanesulfonylbut-2-yne (CB. 2058).

discoverer: Fahmy, 1951.

references: 1958, DIS 32: 76.

phenotype: Bristles short and thin. Eyes large, have deranged facets. Wings short, have incised inner margins and abnormal venation. Variable expression of eye and wing effects. Eclosion slightly delayed. Male infertile;

viability about 50% wild type. Female sterile. RK2.

other information: One allele induced by CB. 1506.

Tonuz: see *Fs(3)Sz23*

tonomacrochaetae: see *tmc*

top: see *Egfr*

Top2: Topoisomerase 2

location: 2- {53} (very close to *Sd*).

references: Hsieh and Brutlag, 1980, *Cell* 21: 115-25.

Sander and Hsieh, 1983, *J. Biol. Chem.* 258: 8421-28.

Shelton, Osheroff, and Brutlag, 1983, *J. Biol. Chem.* 258: 9530-35.

Sander, Nolan, and Hsieh, 1984, *Proc. Nat. Acad. Sci. USA* 81: 6938-42.

Berrios, Osheroff, and Fisher, 1985, *Proc. Nat. Acad. Sci. USA* 82: 4142-46.

Udvardy, Schedl, Sander, and Hsieh, 1985, *Cell* 40: 933-41.

Wang, 1985, *Ann. Rev. Biochem.* 54: 665-97.

Heller, Shelton, Dietrich, Elgin, and Brutlag, 1986, *J. Biol. Chem.* 261: 8063-69.

Nolan, Lee, Wyckoff, and Hsieh, 1986, *Proc. Nat. Acad. Sci. USA* 83: 3664-68.

Hsieh, Lee, Nolan, and Wyckoff, 1987, *NCI Monographs* 4: 7-10.

Wyckoff, Natalie, Nolan, Lee, and Hsieh, 1988, *J. Mol. Biol.* 205: 1-13.

phenotype: *Top2* is an essential gene that encodes the large subunit of type II DNA topoisomerase, an enzyme believed to play an important role in the condensation, decondensation, and segregation of chromosomes. The enzyme is a major component of the nuclear matrix of *Drosophila* cells (Berrios *et al.*, 1985) and is distributed along polytene chromosomes paralleling the distribution of the DNA (Heller *et al.*, 1986). It is believed to act by passing a DNA segment through a transient double-stranded break in another segment. Major cleavage sites for type II topoisomerase have been found in nontranscribed spacer segments and in the 5' and 3' ends of *Hsp70* and the histone genes (Udvardy *et al.*, 1985). When prepared from embryos, the purified enzyme is made up of a major polypeptide encoded by *Top2* of 166,000 daltons, with binding sites for both DNA and ATP, and, in addition, smaller polypeptides of 30,000-40,000 and 132,000-145,000 daltons (Sander and Hsieh, 1983; Shelton *et al.*, 1983; Heller *et al.*, 1986). Protein kinase activity is associated with *Drosophila* topoisomerase II (Sander *et al.*, 1984; Ackerman, Glover, and Osheroff, 1985, *Proc. Nat. Acad. Sci. USA* 82: 3164-68).

cytology: Located at 37D2-6 (Nolan *et al.*, 1986; Hsieh *et al.*, 1987) or at 36E1-2 (Philip, Heller, and Brutlag) by *in situ* hybridization to the salivary chromosomes. It is a single-copy gene.

molecular biology: *Top2* has been cloned and the DNA sequenced (Nolan *et al.*, 1986; Hsieh *et al.*, 1987; Wyckoff *et al.*, 1988). The gene is 5.1 kb in length, as would be expected for a gene encoding topoisomerase II (MW about 170,000) and is divided into five exons of approximately 230, 600, 400, 3100, and 700 nucleotides (Nolan *et al.*, 1986) and four introns of 933, 57, 66, and 81 nucleotides (Wyckoff *et al.*, 1988). The direction of transcription is thought to be from centromere toward

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telomere (Hsieh). The predicted protein is 1447 amino acids in length with a molecular weight of 164,424. Drosophila topoisomerase II shows significant sequence identity with the type II topoisomerases of *B. subtilis*, *E. coli*, bacteriophage T4, and two yeasts. The overall sequence identity between Drosophila and yeast is about 46% (Wyckoff and Hsieh, 1988, Nat. Acad. Sci. USA 85: 6272-76). cDNA sequences encoding Drosophila topoisomerase II, when under the transcriptional control of a yeast promoter, were able to complement the lethality of temperature-sensitive or null yeast mutants (provided the Drosophila genes were continually expressed by growth of yeast cells in galactose media) (Wyckoff and Hsieh, 1988).

tor: torso

location: 2- {57}.

references: Schüpbach and Wieschaus, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 302-17.

Nüsslein-Volhard, Frohnhöfer, and Lehmann, 1987, Science 238: 1675-81.

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-69.

Klingler, Erdélyi, Szabad, and Nüsslein-Volhard, 1988, Nature (London) 335: 295-77.

Casanova and Struhl, 1989, Genes Dev. 3: 2025-38.

Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

Sprenger, Stevens and Nüsslein-Volhard, 1989, Nature (London) 338: 478-83.

Strecker, Halsell, Fisher, and Lipschitz, 1989, Science 243: 1062-66.

phenotype: Maternal-effect lethal; embryos from homozygous mothers show alterations in the anterior-posterior pattern. Hypoactivity (loss-of-function) mutant embryos lack anteriormost head structures (labrum, dorsal bridges) as well as structures posterior to the seventh abdominal segment. Hyperactivity (gain of function) mutant embryos, on the other hand, show segment defects in the middle of the embryos, but may have enlarged terminal structures (Klingler *et al.*, 1988; Strecker *et al.*, 1989). A large number of revertants have been obtained from dominant or semi-dominant hypermorphic alleles.

During cellularization at the blastoderm stage, hypoactivity mutant embryos show a "pole hole" phenotype. A funnel of yolk-free cytoplasm with a small number of nuclei (between 10 and 20) is formed at the posterior pole, extending from the egg periphery to the inner yolk mass. At gastrulation the cephalic furrow is shifted toward the anterior and the germband extends all the way to the posterior end. Analysis of germline clones indicates that the torso mutant is germline autonomous (Schüpbach and Wieschaus, 1986, Dev. Biol. 113: 443-48).

alleles: Unless indicated otherwise, alleles are assumed to be recessive. *rv* in superscript = revertant.

allele	origin	discoverer	synonym	comments
<i>tor</i> ¹	EMS	Wieschaus, Nüsslein-Volhard	<i>tor</i> ^{WK}	
<i>tor</i> ²	EMS	Schüpbach, Wieschaus	<i>tor</i> ^{HH}	
<i>tor</i> ³	EMS	Schüpbach, Wieschaus	<i>tor</i> ^{HM}	
<i>tor</i> ⁴	EMS	Schüpbach, Wieschaus	<i>tor</i> ^{PM}	
<i>tor</i> ⁵	EMS	Schüpbach, Wieschaus	<i>tor</i> ^{QA}	

allele	origin	discoverer	synonym	comments
<i>tor</i> ⁶	EMS	Schüpbach, Wieschaus	<i>tor</i> ^{QK2}	5 kb insertion
<i>tor</i> ⁷	EMS	Schüpbach, Wieschaus	<i>tor</i> ^{Q4}	
<i>tor</i> ⁸	EMS	Schüpbach, Wieschaus	<i>tor</i> ^{QL}	
<i>tor</i> ⁹	EMS	Schüpbach, Wieschaus	<i>tor</i> ^{RI}	
<i>tor</i> ¹⁰	EMS	Mohler	<i>tor</i> ^{JM}	
<i>tor</i> ^{11D}	EMS	Schüpbach	<i>tor</i> ^{spic} , <i>tor</i> ^{RL3} , <i>tor</i> ^{spic} , <i>tor</i> ^{RL3} , <i>tor</i> ^{Y9}	semidominant; heat-sensitive
<i>tor</i> ^{12D}	EMS	Klingler	<i>tor</i> ^{Y9}	semidominant
<i>tor</i> ^{13D}	EMS	Szabad	<i>tor</i> ⁴⁰²¹	dominant
<i>tor</i> ¹⁴	HD	Schüpbach	<i>tor</i> ^{TC17}	2.9 kb insertion
<i>tor</i> ^{rv1}	EMS	Klingler	<i>tor</i> ^{re1}	
<i>tor</i> ^{rv2}	EMS	Klingler	<i>tor</i> ^{re2}	
<i>tor</i> ^{rv3}	EMS	Klingler	<i>tor</i> ^{AL1}	
<i>tor</i> ^{rv4}	EMS	Klingler	<i>tor</i> ^{AL2}	
<i>tor</i> ^{rv5}	EMS	Klingler	<i>tor</i> ^{AL3}	
<i>tor</i> ^{rv6}	EMS	Klingler	<i>tor</i> ^{AL4}	
<i>tor</i> ^{rv7}	EMS	Klingler	<i>tor</i> ^{AL5}	
<i>tor</i> ^{rv8}	EMS	Klingler	<i>tor</i> ^{AL6}	
<i>tor</i> ^{rv9}	EMS	Klingler	<i>tor</i> ^{AL7}	
<i>tor</i> ^{rv10}	EMS	Klingler	<i>tor</i> ^{AL8}	
<i>tor</i> ^{rv11}	EMS	Klingler	<i>tor</i> ^{AL9}	
<i>tor</i> ^{rv12}	EMS	Klingler	<i>tor</i> ^{AL10}	
<i>tor</i> ^{rv13}	EMS	Klingler	<i>tor</i> ^{AL11}	
<i>tor</i> ^{rv14}	EMS	Klingler	<i>tor</i> ^{AL12}	
<i>tor</i> ^{rv15}	EMS	Klingler	<i>tor</i> ^{AL13}	
<i>tor</i> ^{rv16}	EMS	Klingler	<i>tor</i> ^{AL14}	
<i>tor</i> ^{rv17}	EMS	Klingler	<i>tor</i> ^{AL15}	
<i>tor</i> ^{rv18}	EMS	Klingler	<i>tor</i> ^{AL16}	
<i>tor</i> ^{rv19}	EMS	Klingler	<i>tor</i> ^{AL17}	
<i>tor</i> ^{rv20}	EMS	Klingler	<i>tor</i> ^{AL18}	
<i>tor</i> ^{rv21}	EMS	Klingler	<i>tor</i> ^{AY1}	
<i>tor</i> ^{rv22}	EMS	Klingler	<i>tor</i> ^{AY2}	
<i>tor</i> ^{rv23}	EMS	Klingler	<i>tor</i> ^{AY3}	
<i>tor</i> ^{rv24}	EMS	Klingler	<i>tor</i> ^{AY4}	
<i>tor</i> ^{rv25}	EMS	Klingler	<i>tor</i> ^{AY5}	
<i>tor</i> ^{rv26}	EMS	Klingler	<i>tor</i> ^{AY6}	
<i>tor</i> ^{rv27}	EMS	Klingler	<i>tor</i> ^{AY7}	
<i>tor</i> ^{rv28}	EMS	Klingler	<i>tor</i> ^{AY8}	
<i>tor</i> ^{rv29}	EMS	Klingler	<i>tor</i> ^{AY9}	
<i>tor</i> ^{rv30}	EMS	Klingler	<i>tor</i> ^{AY10}	
<i>tor</i> ^{rv31}	EMS	Klingler	<i>tor</i> ^{AY11}	
<i>tor</i> ^{rv32}	EMS	Klingler	<i>tor</i> ^{AY12}	
<i>tor</i> ^{rv33}	EMS	Klingler	<i>tor</i> ^{AY13}	
<i>tor</i> ^{rv34}	EMS	Klingler	<i>tor</i> ^{AY14}	
<i>tor</i> ^{rv35}	EMS	Klingler	<i>tor</i> ^{AY15}	
<i>tor</i> ^{rv36}	EMS	Klingler	<i>tor</i> ^{AY16}	
<i>tor</i> ^{rv37}	EMS	Klingler	<i>tor</i> ^{AY17}	
<i>tor</i> ^{rv38}	EMS	Klingler	<i>tor</i> ^{AY18}	
<i>tor</i> ^{rv39}	EMS	Klingler	<i>tor</i> ^{AY19}	
<i>tor</i> ^{rv40}	EMS	Klingler	<i>tor</i> ^{AY20}	
<i>tor</i> ^{rv41}	EMS	Klingler	<i>tor</i> ^{AY21}	
<i>tor</i> ^{rv42}	EMS	Klingler	<i>tor</i> ^{AY22}	
<i>tor</i> ^{rv43}	EMS	Klingler	<i>tor</i> ^{AY23}	
<i>tor</i> ^{rv44}	EMS	Klingler	<i>tor</i> ^{AY24}	
<i>tor</i> ^{rv45}	EMS	Klingler	<i>tor</i> ^{AY25}	
<i>tor</i> ^{rv46}	EMS	Klingler	<i>tor</i> ^{AY26}	
<i>tor</i> ^{rv47}	EMS	Klingler	<i>tor</i> ^{AY27}	
<i>tor</i> ^{rv48}	EMS	Klingler	<i>tor</i> ^{AY28}	
<i>tor</i> ^{rv49}	EMS	Klingler	<i>tor</i> ^{AY29}	
<i>tor</i> ^{rv50}	EMS	Klingler	<i>tor</i> ^{AY30}	
<i>tor</i> ^{rv51}	EMS	Klingler	<i>tor</i> ^{AY31}	
<i>tor</i> ^{rv52}	EMS	Klingler	<i>tor</i> ^{AY32}	
<i>tor</i> ^{rv53}	EMS	Klingler	<i>tor</i> ^{AY33}	
<i>tor</i> ^{rv54}	EMS	Klingler	<i>tor</i> ^{AY34}	
<i>tor</i> ^{rv55}	EMS	Klingler	<i>tor</i> ^{AY35}	
<i>tor</i> ^{rv56}	EMS	Klingler	<i>tor</i> ^{AY36}	
<i>tor</i> ^{rv57}	EMS	Klingler	<i>tor</i> ^{AY37}	
<i>tor</i> ^{rv58}	EMS	Klingler	<i>tor</i> ^{AY38}	
<i>tor</i> ^{rv59}	EMS	Klingler	<i>tor</i> ^{AY39}	
<i>tor</i> ^{rv60}	EMS	Klingler	<i>tor</i> ^{AY40}	
<i>tor</i> ^{rv61}	EMS	Klingler	<i>tor</i> ^{AY41}	

allele	origin	discoverer	synonym	comments
<i>tor^{rv62}</i>	EMS	Klingler	<i>tor^{rx2}</i>	
<i>tor^{rv63}</i>	EMS	Klingler	<i>tor^{rx8}</i>	
<i>tor^{rv64}</i>	EMS	Klingler	<i>tor^{rx9}</i>	
<i>tor^{rv65} α</i>	EMS	Klingler	<i>tor^{YRE16}</i>	
<i>tor^{rv66} β</i>	EMS	Klingler	<i>tor^{XR1}</i>	9.5 kb deletion
<i>tor^{rv67} γ</i>	EMS	Klingler	<i>tor^{XY83}</i>	0.5 kb insertion

α Revertant of *tor^{12D}* (Klingler *et al.*, 1988).

β Revertant of *tor^{11D}* (Sprenger *et al.*, 1989).

γ Revertant of *tor^{12D}* (Sprenger *et al.*, 1989).

cytology: Placed in 43C5-E7 by deficiency mapping (Schüpbach and Wieschaus, 1989) and 43E by *in situ* hybridization (Casanova and Struhl, 1989; Sprenger *et al.*, 1989).

molecular biology: Gene cloned from DNA isolated by *P*-element transposon tagging from *tor¹⁴* (= *tor^{TC17}*), an allele carrying a *P* insert (Sprenger *et al.*, 1989). Two clones hybridized to *tor⁺* salivary chromosomes at 43E5-11. 35 other *tor* mutants were analyzed and three of them (as well as *tor¹⁴*) showed restriction map alterations in the cloned region.

Rescue of the *tor¹* mutant was accomplished by germ-line transformation with *P* elements using a 12 kb *EcoRI* fragment carrying *tor⁺* (Sprenger *et al.*, 1989).

A transcript of 3.6 kb was demonstrated by Northern blot analysis in *tor⁺* mRNA (transcript not shown in *tor^{rv66}* mRNA). This transcript was also demonstrated by *in situ* hybridization of *tor⁺* mRNA to tissue sections of wild-type ovaries; a high level of evenly-distributed transcript was found in nurse cells and oocytes as well as in 0-4 hour-old embryos. The transcript level was much reduced in older embryos, but some could be detected throughout development. A high level of transcript expression was found in adult females, but not in males.

The genomic and cDNA's of torso have been sequenced (Sprenger *et al.*, 1989) and the amino acid sequence of the protein predicted. The 3.6 kb transcript includes at least 13 introns, their lengths varying from 54 to 152 kb. The cDNA sequence includes a single open reading frame of 2,936 bp, 2,768 bp of which encode a putative protein of 923 amino acids. Although the amino-terminal half of the torso protein shows no significant identity to any published sequences, the rest of the protein shows significant amino-acid identity to growth-factor receptor tyrosine kinases of other organisms and includes an intermediate hydrophobic region of 22 bp that resembles in structure the transmembrane domain of the receptor protein kinases (Sprenger *et al.*, 1989). The *tor* protein is associated with the surface membrane in early embryos and is distributed everywhere along the cell surface (Casanova and Struhl, 1989), although its activity is localized at both poles. Different levels of active *tor* protein are able to specify distinct parts of the terminal pattern.

other information: A single dose of *itl¹* from a *tor^{11D}/tor^{11D}* mother can partially rescue the *tor^{11D}* mutant effect in the embryo (loss of abdominal segments); complete rescue may occur when the embryo is homozygous for *itl¹*, receiving the gene from both parents (Strecker *et al.*, 1989). Injection of *tor⁺* cytoplasm from early cleavage embryos can partially rescue *tor* loss-of-function mutants.

fz expression is reduced or lost in strong gain-of-

function mutants (Klinger *et al.*, 1988; Strecker *et al.*, 1989).

phl mutations have been found to be epistatic over *tor* gain-of-function alleles (Nüsslein-Volhard *et al.*).

***torp: torpid* (J.C. Hall)**

location: 1-15.5.

origin: Induced by ethyl methanesulfonate.

references: Singh and Siddiqi, 1981, Mol. Gen. Genet. 181: 400-02.

phenotype: Adults reversibly paralyzed at 35° or higher. Time of onset of paralysis increases as temperature is lowered (down to 35°); recovery period on return to room temperature is directly proportional to length of high-temperature paralysis time. Flies seem normal after development at 29° or below; larvae unaffected behaviorally at temperatures up to 39°. After anterior nerve stimulation, responses of flight muscles are blocked in all-or-none fashion at about 35°. Mosaic experiments suggest that heat-induced paralysis of individual legs is due to neural defects, possibly in thoracic ganglia.

torpedo: see *Egfr*

torpid: see *torp*

torso: see *tor*

torsolike: see *tsl*

tov: tiny ovaries

location: 3- (unmapped).

origin: Induced by ethyl methanesulfonate.

discoverer: Nüsslein-Volhard.

references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-69.

phenotype: Female sterile; no eggs laid; ovaries rudimentary; may be agametic.

alleles:

allele	synonym
<i>tov 1</i>	<i>tov 012</i>
<i>tov 2</i>	<i>tov 155</i>
<i>tov 3</i>	<i>tov 267</i>
<i>tov 4</i>	<i>tov 345</i>
<i>tov 5</i>	<i>tov 404</i>

Tp1: Temporal protein 1

location: 2- {54}.

synonym: *Tl*.

references: Fruscoloni, Al-Atia, and Jacobs-Lorena, 1983, Proc. Nat. Acad. Sci. USA 80: 3359-63.

phenotype: Present during oogenesis and later embryogenesis, gene codes for small, acidic protein (TI).

cytology: Located in 39C-D by *in situ* hybridization.

molecular biology: mRNAs (prepared from egg chambers or embryos) translated in a cell-free system; found to be associated with polysomes in eggs and 18 hr embryos, but not in early embryos. Cloned probe with cDNA complementary to *Tp1* mRNA hybridized to salivaries.

Tpi: Triosephosphate isomerase

location: 3-101.3 (between *Acph-1* and *bv*).

references: Voelker, Langley, Leigh-Brown, and Ohnishi, 1978, DIS 53: 200.

Voelker, Ohnishi, and Langley, 1979, Biochem. Genet. 17: 769-83.

Voelker, Langley, Leigh-Brown, Ohnishi, Dickson,

Montgomery, and Smith, 1980, Proc. Nat. Acad. Sci. USA 77: 1091-95.

phenotype: Structural gene for the glycolytic enzyme triphosphate isomerase [TPI (EC5.3.1.1)] found in muscle. Heterozygote contains the parental isozymes plus a hybrid isozyme.

alleles: Two electrophoretic alleles, *Tpi*⁴, the most common and slower allele, and *Tpi*⁶, the fast allele (Voelker *et al.*, 1979).

cytology: Located in 99B-E since included in the duplication segregant of *Tp(3;Y)L127 = Tp(3;Y)99B;99E*.

Tpl: Triplo-lethal

location: 3-47.4 (Roehrdanz and Lucchesi, 1980); to the left of *Ki* [no *ri-Tpl* crossovers among 59 *ri Ki* recombinants (Dorer and Christensen)].

origin: Segmental aneuploidy, using the Y-autosome translocations *T(Y;3)L132* and *T(Y;3)A109* (Lindsley *et al.*, 1972).

references: Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84.

Denell, 1974, DIS 51: 124.

1976, Genetics 84: 193-210.

Keppy and Denell, 1979, Genetics 91: 421-41.

Lucchesi and Roehrdanz, 1979, Genetics 91: s71.

Roehrdanz and Lucchesi, 1979, Genetics 91: s105.

1980, Genetics 95: 355-66.

1981, Dev. Genet. (Amsterdam) 2: 147-58.

Kennison and Russell, 1987, Genetics 116: 75-86.

Dorer and Christensen, 1989, Genetics 122: 397-401.

phenotype: Unique dosage-sensitive locus at 83D-E; lethal when present in either one (*Tpl/Df*) or three doses (*Tpl/Dp*) in an otherwise diploid individual. These individuals do not survive to the adult stage, but a few larvae with three doses of 83D-E develop to the third instar. The surviving larvae are also hyperploid for the X chromosome (as in 3X;2A metafemales); they can be produced in genotypes duplicated for 7C and 7D-E (Roehrdanz and Lucchesi, 1979, 1981). Flies with a deficiency for 83D-E in one 3 and a duplication for the region in the other (two doses in all) are viable (Denell, 1976). Crosses of these *Df/Dp* flies to wild-type (*Tpl*-normal) mates fail to produce viable adults (Keppy and Denell, 1979). When wild-type flies or flies bearing a duplication and a deficiency for 83D-E (*i.e.*, viable *Df/Dp* stocks) are treated with EMS or γ -rays (Keppy and Denell, 1979; Roehrdanz and Lucchesi, 1980), new deficiencies and mutations that are viable over *Tpl* duplications but lethal over *Tpl* deficiencies and *Tpl*-normal chromosomes are produced. No function in measuring the X/A ratio has been observed in *Tpl*; it does not interact with the sex-determining genes *Sxl* and *da* (Christiansen and Lucchesi, 1988, DIS 67: 15).

alleles: Cytologically normal reversions to the wild-type *Tpl* phenotype at 83D-E were obtained by mutagen-treatment of *Df Tpl/Dp Tpl* flies. Complementation tests confirm the allelism of these mutants; they are viable over *Tpl*-duplicated and *Tpl*-normal chromosomes, but lethal over *Tpl* deficiencies; they are homozygous lethal. *Tpl* deficiencies (viable over *Tpl* duplications; lethal over *Tpl*-deficient and *Tpl*-normal chromosomes) are described in the section on chromosome rearrangements.

Tpl reversions are listed in the following table:

revertant	origin	synonym	ref ^{α}
<i>Tpl</i> ^{rv1}	EMS	10d77-6	2
<i>Tpl</i> ^{rv2}	HCHO	18i77	2
<i>Tpl</i> ^{rv3} β	EMS	<i>tpl</i> ¹⁰	1, 3
<i>Tpl</i> ^{rv4} β	EMS	<i>tpl</i> ¹⁷	1, 3
<i>Tpl</i> ^{rv5} β	EMS	<i>tpl</i> ³⁸	1, 3

^{α} 1 = Dorer and Christensen, 1989, Genetics 122: 397-401; 2 = Keppy and Denell, 1979, Genetics 91: 421-41; 3 = Roehrdanz and Lucchesi, 1980, Genetics 95: 355-66.

^{β} Causes an increase in recombination between flanking markers of 6.5-10.5 times, while recombination in other adjacent regions is not changed.

cytology: Located in 83D5-E1 since included in *Df(3R)Tpl4 = Df(3R)83D4-5;83E1-2* and *Df(3R)Tpl7 = Df(3R)83D4-5;83E1-2* (Keppy and Denell, 1979).

Tpn: Troponin

location: 2- {67}.

discoverer: French and Pardue.

phenotype: Structural gene for troponin-C.

cytology: Placed in 49D3-50A3 based on the deletion of the cloned sequence in *Df(2R)vg = Df(2R)49D3-4;49F15-50A2-3*.

tpw: see *sto*^{*tpw*}

*tr261: triangle 261

location: 3- (not located).

origin: Spontaneous.

discoverer: Spencer.

references: 1934, DIS 1: 35.

1935, Am. Naturalist 69: 222-38.

phenotype: Small extra crossvein between marginal vein and L2, near their juncture. Variable; overlaps wild type. RK3.

tra: transformer

(M. McKeown and J.M. Belote)

location: 3-45 (between *st* and *cp*).

origin: Spontaneous.

references: Sturtevant, 1945, Genetics 30: 297-99.

Brown and King, 1961, Genetics 46: 143-56.

Marsh and Wieschaus, 1978, Nature 272: 249-51.

Belote, McKeown, Andrew, Scott, Wolfner, and Baker, 1985, Cold Spring Harbor Symp. 50: 605-14.

Butler, Pirrotta, Irminger-Finger, and Nöthinger, 1986, EMBO J. 5: 3607-3613.

McKeown, Belote, and Baker, 1987, Cell 48: 489-499.

McKeown, Belote, and Boggs, 1988, Cell 53: 887-95.

Belote, McKeown, Boggs, Ohkawa, and Sosnowski, 1989, Dev. Genet. 10: 143-54.

phenotype: XX flies homozygous for *tra* transformed into sterile males with fully developed sex combs, male-colored abdomen, male abdominal tergites and plates, external and internal male genitalia. Mate readily with females. Testes rudimentary, without sperm, and with ovarian nurse-cell-like cells [Brown and King, 1961]. Testes reduced in size, but of normal color and shape. Transformed female slightly larger than normal male, developmental rate about that of female. *X/X/Y; tra/tra* also sterile. *tra* not required in male since *X/Y; tra/tra* flies are normal males. *X/X/X* and *X/X/Y; tra/tra* like diploid, *i.e.* male in phenotype, but with larger wing cells as expected of triploids. Normal testis anlagen tran-

splanted into *tra* female becomes attached to duct apparatus and produces sperm. Not needed for female germ cell development since *X/X*, *tra/tra* pole cells transplanted into a wild-type female embryo give rise to progeny of both sexes [Marsh and Wieschaus, 1978]. Cell autonomous in mitotic clones [Baker and Ridge, 1980, Genetics 94: 383-423].

alleles:

allele	origin	discoverer	synonym
<i>tra</i> ¹ α	spont	Sturtevant	<i>tra</i>
<i>tra</i> ² β	spont	Carpenter	<i>tra</i> ^{AC}
<i>tra</i> ³ γ	EMS	Roost	<i>tra</i> ^{Z4}
<i>tra</i> ⁴ β	EMS	Hoffmann	<i>tra</i> ^{V1}
<i>tra</i> ⁵ β	EMS	Hoffmann	<i>tra</i> ^{V2}
<i>tra</i> ⁶ α	HD	Jacquenoud	<i>tra</i> ^{Z5}

α *tra*¹ and *tra*⁶ result from ~1 kb deletions which remove most or all of the *tra* locus.

β *tra*², *tra*⁴ and *tra*⁵ are not detectably rearranged.

γ *tra*³ contains an insertion of ~200 bases.

cytology: Placed in salivary chromosome region 73A8-9 as a result of its exclusion from *Df(3L)st-E5* and *Df(3L)st-7P* and its inclusion in *Df(3L)st-E52* and *Df(3L)st-g24* [Belote *et al.*, 1985; Butler *et al.*, 1986; McKeown *et al.*, 1987].

molecular biology: *tra* has been cloned [Belote *et al.*, 1985; Butler *et al.*, 1986; McKeown *et al.*, 1987] and shown to lie approximately 85 kb to the right of *st*, at position +85 in the map of McKeown *et al.* or position -1 in the map of Butler *et al.* The immediately adjacent gene on the distal side is *l(3)73Ah* [Boggs, Gregor, Idriss, Belote, and McKeown, 1987, Cell 50: 739-747]. The gene immediately to the proximal side has not been identified mutationally. Nucleotide sequences have been obtained for the genomic region around *tra*, for the non-sex-specific cDNA, and for the female-specific cDNA; the sequence of the putative female-specific *tra* protein has been inferred (Boggs *et al.*, 1987). The *tra* transcription unit is just over 1050 nucleotides long. Transcription is from proximal to distal. There are two size classes of *tra* RNA. One is 0.9 kb and is female-specific and the other is 1.1 kb and is present in both sexes. Both of these overlap the 3' end of *l(3)73Ah* by about 70 bases. These two classes of RNA differ as a result of the use of an alternative splice acceptor for the first intervening sequence. The 0.9 kb RNA has a single long open reading frame and is capable of supplying essentially all *tra*⁺ function while the 1.1 kb RNA has no long open reading frame and is dispensable in both males and females [Boggs *et al.*, 1987; McKeown *et al.*, 1988]. Sex-specific splicing, but not transcription, is dependent upon *Sxl*⁺. There is no requirement for *tra*⁺, *tra2*⁺ or *dsx*⁺ for transcription or sex-specific splicing of *tra* [McKeown *et al.*, 1988; Nagoshi, McKeown, Burtis, Belote, and Baker, 1988, Cell 53: 229-36]. Expression of the 0.9 kb RNA causes XY flies to develop as somatic females. This transformation is independent of *Sxl*⁺ and dependent upon *tra2*⁺, *dsx*⁺ and *ix*⁺ [McKeown *et al.*, 1988]. Molecular studies of *dsx* show that lack of *tra*⁺ expression results in *dsx* being expressed in its male mode [Nagoshi *et al.*, 1988], while gain of *tra*⁺ expression results in *dsx* being expressed in its female mode [McKeown *et al.*, 1988]. This suggests that, in a formal genetic sense, the regulatory hierarchy controlling sex is

organized in the following order: *X*: A ratio > *Sxl* > *tra* > *tra2* > *dsx* \geq *ix* > terminal differentiation (Belote *et al.*, 1989).

tra^D: see *dsx*^D

***tra2*: transformer 2**

location: 2-70, between *Bl* and *L* (Watanabe, 1975).

references: Watanabe, 1975, Jpn. J. Genet. 50: 269-71.

Fujihara, Kawabe, and Oishi, 1978, J. Hered. 6: 229-36.

Baker and Ridge, 1980, Genetics 94: 383-423.

Belote and Baker, 1981, Genetics 97: s9.

Ota, Fukunaga, Kawabe, and Oishi, 1981, Genetics 99: 429-41.

Baker and Belote, 1983, Annu. Rev. Genet. 17: 345-83.

Belote and Baker, 1983, Dev. Biol. 95: 512-17.

Belote, Handler, Wolfner, Livak, and Baker, 1985, Cell 40: 339-48.

Butler, Pirrota, Irminger-Finger, and Nöthiger, 1986, EMBO. J. 5: 3607-13.

Belote and Baker, 1987, Proc. Nat. Acad. Sci. USA 84: 8026-30.

Amrein, Gorman, and Nöthiger, 1988, Cell 55: 1025-35.

Goralski, Edström, and Baker, 1989, Cell 56: 1011-18.

Mattox, Palmer, and Baker, 1990, Genes Dev. 4: 789-805.

phenotype: One role of *tra2*⁺ (like *tra*⁺) is to regulate sex determination by directing *dsx*⁺ in such a way that female primordia are expressed and male primordia repressed in chromosomal females. In addition, *tra2*⁺ serves as a regulator of spermiogenesis and copulation; as a result, functional sperm are produced by chromosomal males and transmitted to the females. Null or amorphic *tra2* mutations, however, transform chromosomal females into flies that are phenotypically male in regard to external cuticular morphology, pigment pattern, internal genital ducts, and mating behavior. Their gonads are much reduced and lack sperm and they are not affected by *mle* (Fujihara *et al.*, 1978). *tra2* mutations in chromosomal males produce normal looking adult males showing normal sexual behavior, but the sperm are amotile (Watanabe, 1975; Belote and Baker, 1981). Some *tra2* mutants are temperature-sensitive; homozygotes become phenotypic males when reared at 29°, but phenotypic females when reared at 16° (Belote and Baker, 1983). When *X/X; tra2^{ts2}* homozygotes are shifted to the female-specifying temperature during or before the third instar, no development of the male accessory glands occurs (Chapman and Wolfner, 1988, Dev. Biol. 126: 195-202). In *X/X; tra2^{ts2}* homozygotes reared throughout development at the permissive temperature of 16°, yolk polypeptide synthesis occurs as in *X/X; tra2^{ts2}/+* controls; in *X/X; tra2^{ts2}* homozygotes raised and kept at 29°, however, no synthesis of yolk polypeptides can be detected (Belote *et al.*, 1985). Temperature shift experiments with this temperature-sensitive allele show that *tra2*⁺ function must be present in the adult for the initiation and maintenance of yolk polypeptide synthesis. This control over YP on the part of *tra2*⁺ was shown to be at the level of transcription (Kraus, Lee, Lis, and Wolfner, 1988, Mol. Cell Biol. 8: 4756-64). *tra2^{ts}* homozygous females do not always maintain male courtship behavior at 29°, but transformed females hemizygous for *tra2* [*tra2^{ts1}/Df(2R)trix*] court in a reliably male fashion (Belote and Baker, 1987). Temperature-

shift experiments indicate that the TSP for induction of male courtship starts in the last half of the pupal period and ends before the end of pupation.

alleles:

allele	origin	ref ^α	comments
tra2	spont	2-4, 8, 10, 11	null ^β
tra2^{ad}		6	strong allele
tra2^B	EMS	3	amorphic ^γ
tra2^{OTF}	spont	3, 8-10	leaky ^δ
tra2^P	HD	1	<i>tra2^P/tra2^P</i> females sterile, males fertile ^ε
tra2^{Pd1}	HD	1	<i>tra2^{Pd1}/tra2^{Pd1}</i> females male-like intersexes, males normal and fertile ^ζ
tra2^{Pd2}	HD	1	<i>tra2^{Pd2}/tra2^{Pd2}</i> lethal ^η
tra2^{ts1}	EMS	2-4, 7	temperature-sensitive
tra2^{ts2}	EMS	2, 3, 5	temperature-sensitive

- ^α 1 = Amrein, Gorman, and Nöthiger, 1988, Cell 55: 1025-35; 2 = Belote and Baker, 1982, Proc. Nat. Acad. Sci. USA 79: 1568-72; 3 = Belote and Baker, 1983, Dev. Biol. 95: 512-17; 4 = Belote and Baker, 1987, Proc. Nat. Acad. Sci. USA 84: 8026-30; 5 = Belote, Handler, Wolfner, Livak, and Baker, 1985, Cell 40: 339-48; 6 = Dotti and Nöthiger, unpublished; 7 = Epper and Bryant, 1983, Dev. Biol. 100: 294-307; 8 = Fujihara, Kawabe, and Oishi, 1978, J. Hered. 69: 229-36; 9 = Oishi and Ota, 1982, DIS 58: 121; 10 = Ota, Fukunaga, Kawabe, and Oishi, 1981, Genetics 99: 429-41; 11 = Watanabe, 1975, Jpn. J. Genet. 50: 269-71.
- ^β No yolk-protein precursors in males or females (Ota *et al.*, 1981).
- ^γ *tra2^B/tra2^B* identical to *tra2^B/tra2* or to *tra2^B/Df(2R)LA* (Baker and Ridge, 1980, Genetics 94: 383-423).
- ^δ Ovaries rudimentary (Fujihara *et al.*, 1978).
- ^ε Mutant can be reverted to wild type or a more extreme mutant. In homozygous females, tergite 6 partially pigmented, vaginal bristles long, sex combs not malelike. *tra2^P/tra2* females intersexes.
- ^ζ *tra2^{Pd1}/tra2* females pseudomales, *tra2^{Pd1}/tra2* males normal and fertile.
- ^η *tra2^{Pd2}/tra2* females pseudomales.

cytology: Located in 51B4-6 since gene deleted by *Df(2R)trix = Df(2R)51A1-2;51B6* but not by *Df(2R)L48 = Df(2R)51A1;51B4* (Goralski *et al.*, 1989).

molecular biology: DNA from the 51B region was cloned from wild-type and *tra2^P* flies by microdissection-microcloning and chromosome walking (Amrein *et al.*, 1988; Goralski *et al.*, 1989; Mattox *et al.*, 1990); mutants, rearrangements, and a P-element insertion were located on the molecular map of the region. Nucleotide sequences of the entire genomic DNA and of ten cDNAs have been obtained and the amino acid sequences of the corresponding proteins predicted (Amrein *et al.*, 1988; Goralski *et al.*, 1989; Mattox *et al.*, 1990). A DNA fragment of 3.9 or 4.5 kb from the *tra2* region was able to rescue (by P-element transformation) homozygous *tra2* flies, both in regard to sex determination and male fertility (Amrein *et al.*, 1988; Goralski *et al.*, 1989). Alternative splicing of *tra2* mRNA creates at least four overlapping transcript structures, each of which encodes one of four different polypeptides. Different combinations of ribonucleoprotein consensus sequences (RNP-CS) and arginine-serine rich regions are included in each polypeptide. The mature mRNAs are described in the following table:

mRNA	location in fly	exons	polypeptide size
type A	soma of males and females	1,2,4-7	264 amino acids
type B	ovary ^α	1-7	226 amino acids
type C ^β	testis	3-7	179 amino acids

mRNA	location in fly	exons	polypeptide size
type D ^γ type E ^δ	testis	3-7	136 amino acids

- ^α Some type B mRNA has been found in somatic tissues (males and females).
- ^β Intron of 232 nucleotides included in the mRNA.
- ^γ Found in low abundance.
- ^δ Not characterized..

tra2 proteins show similarities to certain known RNA-binding proteins. A *tra2-lacZ* fusion protein has been localized by antibody stains to the nuclei of salivary gland and fat body cells in *Drosophila melanogaster* (Mattox *et al.*, 1990). Molecular studies show that the mutants *tra2^{OTF}*, *tra2^P*, and *tra2^{Pd1}*, which are not null alleles, carry insertions of about 1.7 kb and presumably map in the 5' flanking region of the gene; cytological and molecular studies indicate that the mutant *tra2^{Pd2}* is associated with an inversion and is accompanied by a deletion (Amrein *et al.*, 1988). The *tra2* product regulates the splicing pattern of the primary transcript of the "downstream" gene *dsx* in females [Baker, 1989, Nature (London) 340: 521-524; Goralski *et al.*, 1990; Mattox *et al.*, 1990]. *tra2* activity in somatic tissues is believed to be regulated by means of a post-translational sex-specific interaction with the protein product of the *tra* gene (Mattox *et al.*, 1990).

tracheae broken: see *tbr*

trachealess: see *trh*

Tramtrack: see *Ttk*

Transcription factor: see *Trf*

transformer: see *tra*

transformer 2: see *tra2*

transient receptor potential: see *trp*

translucent: see *trl*

trapped: see *ted*

trb: thread bristle

location: 1-36.3.

origin: Induced by D-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3026).

discoverer: Fahmy, 1955.

references: 1959, DIS 33: 93.

phenotype: Bristles short and very thin. Hairs small and sparse. Wings more rounded at tips, margins often incised; veins slightly thickened. Trident pattern slightly darker than wild type. Male viable and fertile; female sterile. RK2.

other information: One allele induced by CB. 3026.

trc: tricorner

location: 3-46 (to left of *Pc* at 47.1).

references: Ferrus, 1976, Ph.D. Thesis, Univ. Autonoma de Madrid.

Struhl, 1981, Nature 293: 36-41.

Gubb and Garcia-Bellido, 1982, J. Embryol. Exp. Morph. 68: 37-57.

Vinson and Adler, 1987a, DIS 66: 150.

1987b, Nature (London) 329: 549-51.

phenotype: Mutant wing and notum have rosettes of three or more short trichomes instead of single long hairs as in wild type. Useful as cell marker. Mutant cell autonomous in mitotic cells in a wild-type background, but not in a Minute background (Vinson and Adler, 1987a).

trd: tridenticle

location: 2-.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1983, DIS 59: 158-60.

phenotype: Larval denticles thickset and forked. Some alleles viable.

***tre: triangle eye**

location: 1-20.2.

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 93.

phenotype: Eyes triangular with apex pointing forward. Fly large. Wings broad, blunt tipped, and slightly divergent. Male viable and fertile; female sterile. RK3.

Tre: Trehalose sensitivity (J.C. Hall)

location: 1-13.6 (linked to *cx*).

origin: Spontaneous (naturally occurring variants).

references: Tanimura, Isono, Takimura, and Shimada, 1982, J. Comp. Physiol. 147: 433-37.

Tanimura, Isono, and Yamamoto, 1988, Genetics 119: 399-406.

phenotype: Locus determining differences in sensitivity to the taste of the disaccharide trehalose in behaviorally-based (fluid intake) tests (no differences in sensitivity to glucose, fructose, or sucrose). The *Tre*⁺ allele results in high trehalose sensitivity, the *Tre* allele in low trehalose sensitivity. Females with half the normal dose of a given *Tre*⁺ allele are half as sensitive to trehalose, but flies (male or female) with twice the normal dose of *Tre*⁺ show only a slight increase in sensitivity as compared to normal flies. *Tre* is thought to be a structural gene for the trehalose specific receptor in *D. melanogaster* (Tanimura *et al.*, 1988).

alleles: Two alleles, *Tre*⁺ and *Tre*, are found in wild-type strains.

cytology: Placed by Tanimura *et al.*, 1988, between 5A10 and 5B1-3 by analyzing the sensitivity to trehalose in segmentally aneuploid flies carrying deficiencies or duplications from *T(1;Y)* translocations with breaks in the 5A-C region.

trefoil: see tf

Treh: Trehalase

location: 2-92.9.

references: Oliver, Huber, and Williamson, 1978, Biochem. Genet. 16: 927-40.

Oliver and Williamson, 1978, Can. J. Genet. Cytol. 20: 452.

Laurie-Ahlberg, Wilton, Curtsinger, and Emigh, 1982, Genetics 102: 191-206.

phenotype: Structural gene for trehalase [TRE (EC 3.2.1.28)], a hydrolase that splits the sugar trehalose into two glucose molecules. Polyacrylamide gel electrophoresis and isoelectric focusing indicates the presence of one molecular form of the enzyme in flight muscles,

hemolymph, and abdomens of adult flies. No mutant alleles obtained.

cytology: Placed in 55B-55E on the basis of dosage sensitivity determined by examination of the duplication- and deletion-bearing aneuploids.

Trehalose sensitivity: see Tre

Trf54F: Described as Ntf

Trf100D: Described as Ttk

trh: trachealess

location: 3;-1.

origin: Induced by ethyl methanesulfonate.

references: Jürgens, Kluding, Nüsslein-Volhard, and Wieschaus, 1983, DIS 59: 157-58.

Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

phenotype: Embryonic lethal. Tracheae are absent and filzkörper not elongated.

alleles: Two alleles, *trh*¹ and *trh*², isolated as *trh*^{5D} and *trh*^{7J}.

cytology: Placed in 61E-F since covered by *Y*^{P3D} segregant of *T(Y;3)A144* = *T(Y;3)Xhy*⁺;61F and by *Dp(3;Y)G130* = *Dp(3;Y)61E;66F*.

tri: trident

location: 2-55.

origin: Spontaneous.

synonym: Probably *tri*^{32k}, *tri*^{33d27}, and *b-1*^{33g18} are the same.

discoverer: Plough, 32k.

references: Plough and Ives, 1934, DIS 1: 34. 1935, Genetics 20: 42-69.

phenotype: Dark trident or streak on thorax. Scutellum and sternopleural plates also dark. Thorax often contains bubbles. Variable; overlaps wild type but also semidominant. RK3.

triangle 261: see tr261

triangle eye: see tre

tricornor: see trc

trident: see tri

tridenticle: see trd

trimmed: see fr²

Triosephosphate isomerase: see Tpi

Triplo-lethal: see Tpl

trithorax: see trx

trk: trunk (T. Schüpbach)

location: 2-36.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 302-17.

Nüsslein-Volhard, Frohnhofer, and Lehmann, 1987, Science 238: 1675-81.

Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

phenotype: Maternal-effect lethal; embryos from homozygous mothers lack anterior-most head structures and structures posterior to the seventh abdominal segment. At gastrulation cephalic furrow is shifted toward anterior and the germband extends all the way to the posterior

end. During cellularization at the blastoderm stage a funnel of yolk free cytoplasm containing a small number of nuclei (between 10 and 30) forms at the posterior pole of the embryos extending from the egg periphery to the inner yolk mass. Analysis of germline clones indicates that the mutation is germline autonomous (Schüpbach and Wieschaus, 1986, *Dev. Biol.* 113: 443-48).

alleles:

allele	synonym
<i>trk 1</i>	<i>trk RA</i>
<i>trk 2</i>	<i>trk HD</i>
<i>trk 3</i>	<i>trk HH</i>
<i>trk 4</i>	<i>trk PI</i>
<i>trk 5</i>	<i>trk PK</i>
<i>trk 6</i>	<i>trk RI</i>

cytology: Located at 31A-C (Nüsslein-Volhard *et al.*, 1987).

****trl*: translucent**

location: 2-45 or -65 (10 units from *Bl*).

origin: Spontaneous.

discoverer: Bridges, 20b17.

references: Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 238.

phenotype: Eye color translucent ruby, like *p. RK2*.

trm: see *fr*²

tRNA*:** see under ***RNA

Tropomyosin*:** see ***Tm1, Tm2

Troponin*:** see ***Tpn

***trp*: transient receptor potential (J.C. Hall)**

location: 3-97 (Manning, unpublished); 3-106 (Hardy, Orevi, and Merriam, unpublished); 3-100 (inferred from cytology).

synonym: Cosens-Manning mutant (= *trp*^{CM}).

references: Cosens and Manning, 1969, *Nature (London)* 224: 285-87.

Cosens, 1971, *J. Insect. Physiol.* 17: 285-302.

Cosens and Perry, 1972, *J. Insect. Physiol.* 18: 1773-86.

Minke, Wu, and Pak, 1975, *Nature (London)* 285: 84-87.

Minke, 1977, *Biophys. Struct. Mech.* 34: 59-63.

Hu, Reichert, and Stark, 1978, *J. Comp. Physiol.* 126: 15-24.

Reichert and Bicker, 1979, *J. Comp. Physiol.* 133: 283-90.

Minke and Armon, 1980, *Photobiol.* 32: 553-62.

Lo and Pak, 1981, *J. Gen. Physiol.* 77: 155-75.

Swanson and Cosens, 1981, *J. Insect Physiol.* 27: 215-23.

Levy, Ganguly, Ganguly, and Manning, 1982, *Dev. Biol.* 94: 451-64.

Minke, 1982, *J. Gen. Physiol.* 79: 361-85.

Chen and Stark, 1983, *J. Insect Physiol.* 29: 133-40.

Minke, 1983, *J. Comp. Physiol.* 151: 283-86.

Montell, Jones, Hafen, and Rubin, 1985, *Science* 230: 1040-43.

Wong, Hokanson, and Chang, 1985, *Invest. Ophthalm. and Vis. Sci.* 26: 243-46.

Wong, Yuh, Schaefer, Roop, and Ally, 1987, *Somat. Cell and Molec. Genet.* 13: 661-69.

Montell and Rubin, 1989, *Neuron* 2: 1313-23.

Suss, Barash, Stavenga, Stieve, Selinger, and Minke,

1989, *J. Gen. Physiol.* 94: 465-91.

Wong, Schaefer, Roop, LaMendola, Johnson-Seaton, and Shao, 1989, *Neuron* 3: 81-94.

phenotype: Mutation believed to affect an intermediate step in phototransduction, the wild-type gene apparently encoding a protein involved in an intermediate step between photoreception and opening of the light-sensitive ion channels (Minke, 1977, 1982; Minke and Armon, 1980; Montell and Rubin, 1989; Suss *et al.*, 1989). Although the mutants behave normally in dim light, they behave as though blind in bright light and there is an abnormally slow dark recovery (Cosens and Manning, 1969; Cosens, 1971). The light-evoked response of the photoreceptors, as shown in the ERG, decays to baseline during an intense, prolonged stimulus, but not during a dim or brief stimulus (Cosens and Manning, 1969; Minke *et al.*, 1975); each quantum bump, however, seems intrinsically normal in shape and amplitude (Suss *et al.*, 1989); fluoride ions, which lead to excitation and adaptation of wild-type photoreceptors in the dark when superfused onto an eye slice, did neither to mutant cells. A hydrolysis-resistant analogue of GTP, which excites wild-type photoreceptors and results in noisy depolarizations, reduced the mutant's light response.

The visual pigment in R1-6 photoreceptors is normal in young *trp* flies, but its concentration decreases with age (Minke, 1982). The rhabdomeres degenerate with age and there is accumulation of glycogen granules (Cosens and Perry, 1972). Ultimately, photoreceptor cell bodies also degenerate (Isono, unpublished). Raising the mutant flies in complete darkness prevents the degenerative changes from appearing but has no effect on the electrophysiological phenotype (Isono and Pak, unpublished). Initially, the light-evoked migration of pigment granules occurs in a normal manner (*i.e.*, toward the rhabdomeres) in *trp* mutants, but the granules move away after only five seconds of sustained light (Lo and Pak, 1981). The *trp* mutation (unlike *norpA*) does not block degeneration caused by *rdgB* (Chen and Stark, 1983, *J. Insect Physiol.* 29: 133-40); in a *rdgB;trp* genetic background, R1-6 photoreceptors are eliminated and the R7,8 photoreceptors that remain show the *trp* phenotype.

trp is expressed in the photoreceptors of the ocelli as well as in those of the compound eyes (Hu *et al.*, 1978; Montell *et al.*, 1985).

trp mutants seem to exhibit normal visually-mediated learning under the high light-intensity conditions that largely eliminate photoreceptor potentials (Reichert and Bicker, 1979).

alleles: Mutant alleles are listed in the following table:

allele	origin	discoverer	synonym	comments
<i>trp 1</i>	spont	Cosens, Manning	<i>trp</i> ^{CM}	temperature-sensitive ^α
<i>trp 2</i>	EMS	Pak	<i>trp</i> ^{P301}	
<i>trp 3</i>	EMS	Pak	<i>trp</i> ^{P302}	temperature-sensitive ^β
<i>trp 4</i>	EMS	Pak	<i>trp</i> ^{P303}	
<i>trp 5</i>	EMS	Pak	<i>trp</i> ^{P304}	
<i>trp 6</i>	EMS	Pak	<i>trp</i> ^{P310}	
<i>trp 7</i>	EMS	Pak	<i>trp</i> ^{P313}	
<i>trp 8</i>	EMS	Pak	<i>trp</i> ^{P338}	temperature-sensitive ^β
<i>trp 9</i>	EMS	Pak	<i>trp</i> ^{P343}	
<i>trp 10</i>	EMS	Hardy, Orevi, Merriam	<i>trp</i> ^{US4699}	

^α Rearing at 19° (instead of 25°) slows the rate of the decay-to-baseline of the photoreceptor potential and leads to faster initial dark recovery than occurs under non-permissive conditions. Rearing at 25°, followed by shift of adults to 19°, leads to more nearly normal behavior in bright light within four to six days (Minke, 1983).

^β Permissive temperature for normal ERG phenotype <25°; restrictive temperature ≥29°. Temperature-sensitive period limited to last 72 hours of pupal life (Wong *et al.*, 1989).

cytology: Located in 99C5-6 by *in situ* hybridization and breakpoint analysis (Montell *et al.*, 1985; Wong *et al.*, 1987, 1989). *trp* is covered by *Dp(3;Y)L127 = Dp(3;Y)99B5-6;99F3-4* and *Dp(3;1)52* [a duplication produced by translocation onto the X chromosome of a piece of the third chromosome (98F14-99A12;100F), followed by deletion of 99A9-10;99C5-6 (Frisardi and MacIntyre, 1984, *Mol. Gen. Genet.* 197: 403-13)], but is not covered by *Dp(3;1)78 = Dp(3;1)X^P;99C5-7;100F* (Pye, unpublished) and *Dp(3;1)165P = Dp(3;1)X^P;99B2-4;99C5-6* (Frisardi and MacIntyre, 1984; Wong *et al.*, 1989).

molecular biology: Cloned by isolation of DNA fragments hybridizing preferentially to head rather than body mRNA (Levy *et al.*, 1982; Montell *et al.*, 1985) and by genetic analysis and chromosomal walking (Wong *et al.*, 1987, 1989). cDNA and putative protein of *trp* sequenced (Montell and Rubin, 1989; Wong *et al.*, 1989); total length of the 13 exons is 1645 bp (Wong *et al.*, 1989). In germline transformation experiments (Montell *et al.*, 1985), a 7.1 kb genomic fragment rescued the ERG defects of *trp¹*. Montell *et al.* (1985) identified a 4.1 kb transcript which appeared during the pupal stage and was localized to the retinula cells of the compound eyes and the ocelli. This transcript encodes a 143 kd integral membrane protein of 1275 amino acids with eight putative transmembrane domains; it is not similar in sequence to any previously analyzed protein (Montell and Rubin, 1989) and is concentrated in the rhabdomeres of the photoreceptor cells. Wong *et al.* (1987, 1989) identified a somewhat larger transcript (4.5 kb) that is believed to be "eye specific" by virtue of its absence from the mutant eyeless. This transcript also encodes an unique protein of 142 kd that seems to be associated with the photoreceptor membrane. Mutants *trp¹*, *trp²*, and especially *trp⁹* (according to Montell and Rubin, 1989) show reduced amounts of the 4.1 kb transcript and completely lack the *trp* protein. The mutant *trp³* (according to Wong *et al.*, 1989) also shows a reduction in transcript level, but they find that *trp¹* and *trp⁷* show close to normal levels; *trp* protein is lacking in head extracts in all three mutants. Transformed *trp* flies show normal protein levels.

other information: The same kinds of results from applying fluoride ions or the GPT analogue to the *trp* mutant in *Drosophila* (see "phenotype") were observed in experiments on the *nss* mutant in the blowfly *Lucilia*.

Truncate 51b: see *dp^{oIM}*

trunk: see *trk*

trw: see *ap^{trw}*

***trx*: trithorax**

location: 3-54.2.

references: García-Bellido, 1977, *Amer. Zool.* 17: 613-29.

Ingham and Whittle, 1980, *Mol. Gen. Genet.* 179: 607-

14.

Capdevila and García-Bellido, 1981, *Wilhelm Roux's Arch. Dev. Biol.* 190: 339-50.

Ingham, 1981, *Wilhelm Roux's Arch. Dev. Biol.* 190: 365-69.

Duncan and Lewis, 1982, *Developmental Order: Its Origin and Regulation* (S. Subtelny, ed.). Alan R. Liss, Inc., New York, pp. 533-54).

Ingham, 1983, *Nature* 306: 591-93.

1985a, *Cold Spring Harbor Symp. Quant. Biol.* 50: 201-08.

1985b, *J. Embryol. Exp. Morph.* 89: 349-65.

Sato and Denell, 1987, *Genetics* 116: 389-98.

Mozer and Dawid, 1989, *Proc. Nat. Acad. Sci. USA* 86: 3738-42.

Shearn, 1989, *Genetics* 121: 517-25.

phenotype: The presence of *trx⁺* is required throughout embryonic and larval development for the appropriate differentiation in the adult of segments in the head, thorax, and abdomen (Ingham and Whittle, 1980; Ingham, 1981), the primary effect being in the thoracic segments. Mutants show transformations of the first and the third thoracic segments to the second thoracic segment as well as transformations in the abdomen (Mozer and Dawid, 1989). The gene seems to be involved in the positive regulation of the *BXC* and the *ANTC* (Duncan and Lewis, 1982). The viable mutant combinations *trx¹/trx¹* and *trx^D/+* show variable segmental transformations in adults, as do heterozygous deficiencies [*Df(3R)red-P52/+*, for example]. The frequency of homeotic transformations in adults and, to some extent, in larvae of such genotypes varies inversely with the dosage of the *BXC* (Duncan and Lewis, 1982; Sato and Denell, 1987). A similar dosage effect has been proposed for the *ANTC* (Sato and Denell, 1987). When the mutant allele or deficiency is maternal in origin, the frequency of transformations is higher in adults (but not in larvae). The alleles *trx²*, *trx³*, and *trx^D* are larval or pupal lethals as homozygotes, trans-heterozygotes, or deficiency heterozygotes, and may show weak homeotic transformations in larvae or in homozygous clones in adults (Capdevila and García-Bellido, 1981; Ingham, 1981, 1983, 1985b).

alleles: Alleles that show a recessive or dominant homeotic phenotype and fail to complement each other have been identified. All pairwise combinations of *trx^{B14}*, *trx^{B16}* - *trx^{B18}*, and *trx^{E1}* - *trx^{E13}* are inviable except for rare survivors of the genotypes *trx^{E11}/trx^{B16}*, *trx^{E11}/trx^{B17}*, and *trx^{B16}/trx^{B17}*. Deficiencies are listed in the rearrangement section under *Df(3R)red*.

allele	origin	discoverer	synonym	ref ^α	comments
<i>trx¹</i>	spont	Ingham (1980)		3-6	recessive; viable in homozygotes
<i>trx²</i>	EMS	Ingham (1981)		4, 5	recessive; lethal in homozygotes or with <i>trx³</i> or <i>trx^D</i>
<i>trx³</i>	EMS	Ingham (1981)		4, 5	recessive; lethal in homozygotes or with <i>trx²</i> or <i>trx^D</i>
<i>trx^{B14}</i>	γ rays	Kennison, Tamkun		7	dominant; suppresses extra sex combs in <i>Pc⁴/+</i>
<i>trx^{B16β}</i>	HD	Kennison		8	1.5 kb insertion into 3 kb fragment at DNA map site 29-32
<i>trx^{B17}</i>	HD	Kennison		8	1 kb insertion into

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allele	origin	discoverer	synonym	ref ^α	comments
					distal part of 5.4 kb fragment at DNA map site 21-26.5
<i>trx</i> ^{B18β}	HD	Kennison		8	2.5 kb insertion into 3 kb fragment at DNA map site 29-32
<i>trx</i> ^{Dγδ}	spont	Lewis (1968)	<i>Rg-bx</i> <i>l(3)bx^v</i> <i>trx^L</i>	1, 2, 3	dominant; lethal in homozygotes or with <i>trx¹</i> , <i>trx²</i> , or <i>trx³</i>
<i>trx</i> ^{E1}	γ rays	Kennison, Tamkun		7	dominant; suppresses extra sex combs in <i>Pc⁴/+</i>
<i>trx</i> ^{E2}	γ rays	Kennison, Tamkun		7	dominant; suppresses extra sex combs in <i>Pc⁴/+</i>
<i>trx</i> ^{E3}	EMS	Kennison, Tamkun		7	dominant; suppresses antenna to leg trans- formation in <i>Antp^{Ns}</i> ; deletion of 100 bp at map site 16
<i>trx</i> ^{E4}	EMS	Kennison			
<i>trx</i> ^{E5}	EMS	Kennison			
<i>trx</i> ^{E6}	EMS	Kennison, Tamkun		7	dominant; suppresses extra sex combs in <i>Pc⁴/+</i>
<i>trx</i> ^{E7}	EMS	Kennison, Tamkun		7	dominant; suppresses extra sex combs in <i>Pc⁴/+</i>
<i>trx</i> ^{E8}	EMS	Kennison, Tamkun		7	dominant; suppresses extra sex combs in <i>Pc⁴/+</i>
<i>trx</i> ^{E9}	EMS	Kennison, Tamkun		7	dominant; suppresses extra sex combs in <i>Pc⁴/+</i>
<i>trx</i> ^{E10}	EMS	Kennison, Tamkun		7	dominant; suppresses extra sex combs in <i>Pc⁴/+</i>
<i>trx</i> ^{E11}	EMS	Kennison, Tamkun		7	dominant; suppresses extra sex combs in <i>Pc⁴/+</i>
<i>trx</i> ^{E12}	EMS	Kennison, Tamkun		7	dominant; suppresses extra sex combs in <i>Pc⁴/+</i>
<i>trx</i> ^{E13}	γ rays	Kennison, Tamkun		7	dominant; suppresses extra sex combs in <i>Pc⁴/+</i>
<i>trx</i> ^{P2}	cloning	Ingham		8	0.9 kb <i>P</i> -element insertion in 1.35 kb fragment at DNA map site 33-34.5
<i>trx</i> ^{rib7ε}	HD	Karpen	<i>trx^Prib7,ry+</i>	8	21 kb <i>P</i> -element insertion in 1.35 kb fragment at DNA map site 33-34.5
<i>trx</i> ^{rv4}	HD	Ingham			4.5 doublet band lost; wild-type 9 kb band restored

^α 1 = Capdevila and García-Bellido, 1981, Wilhelm Roux's Arch. Dev. Biol. 190: 339-50; 2 = García-Bellido, 1977, Am. Zool. 17: 613-29; 3 = Ingham, 1980, DIS 55: 63-64; 4 = Ingham, 1981, Wilhelm Roux's Arch. Dev. Biol. 190: 365-69; 5 = Ingham, 1985, J. Embryol. Exper. Morph. 89: 349-65; 6 = Ingham and Whittle, 1980, Mol. Gen. Genet. 179: 607-14; 7 = Kennison and Tamkun, 1988, Proc. Nat. Acad. Sci. USA 85: 8136-40; 8 = Moser and Dawid, 1989, Proc. Nat. Acad. Sci. USA 86: 3738-42.

^β Breakpoint in long intron (Moser and Dawid, 1989).

^γ Slightly reduced pigmentation of fifth abdominal tergite in heterozygotes (Lewis, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 155-72).

^δ Cited by Capdevila and García-Bellido, 1981.

^ε Carries transposon *P*[*rib7/ry⁺*] with one *Drosophila melanogaster* ribosomal transcription unit and *ry⁺* marker at 88B (Karpen, Schaeffer, and Laird, 1988, Genes Dev. 2: 1745-63); fails to complement *trx* (Moser and Dawid, 1989).

cytology: Placed in 88A12-B5 since uncovered by *Df(3R)red-P52 = Df(3R)88A12-B1;88B4-5*.

molecular biology: The gene was cloned by *P*-element transposon tagging. Five insertion mutants were located within a region of 10 kb; reversion of one of these mutations resulted in excision of the *P*-element insertion (Moser and Dawid, 1989). Two major mRNAs of 12 and 15 kb, transcribed in a distal to proximal direction, are present. The 12-kb mRNA is most abundant in embryos of 0-6 hours, but it also appears in larvae and pupae. The 15 kb mRNA is most abundant in larvae, in pupae eight to nine days after oviposition, and in male and female adults; it also appears in 3- to 6-hr embryos. A 10 kb transcript was seen (but rarely) in 1- to 3-hr embryos and also in adult females; it may be maternally derived. Early embryos show an uniform distribution of transcript, but later (14-15 hours after fertilization) the ventral nerve cord has a higher concentration than other parts (Moser and Dawid, 1989).

other information: 22-52% of double heterozygotes involving a null allele of *Ash-1* and the *trx* deficiency, *Df(3R)red-P93*, show partial transformations of halteres to wings and/or partial transformations of third (and sometimes first) legs to second legs, whereas in single heterozygotes no transformations are shown (Shearn, 1989). Heterozygosis for null alleles of *trx* suppresses the extra sex combs phenotype of *+Df(3L)Pc-MK*, and increases penetrance of the maternal-effect homeotic phenotype of *fs(1)h*. *trx* function seems to be necessary for optimal expression of *Scr* (Sato, 1988, Roux's Arch. Dev. Biol. 197: 435-40).

***trx*¹**

phenotype: Flies homozygous for *trx*¹ show a variety of partial homeotic transformations [ventral prothorax and metathorax to mesothorax and second to seventh abdominal segments to first abdominal segment (Ingham and Whittle, 1980)]. Penetrance of the transformation phenotype is stronger in hemizygotes than homozygotes and increases as the temperature is raised from 18° to 25°C. At the higher temperature, the penetrance of the mutant offspring of *trx*¹/*trx*¹ females is almost 100%, while mutant offspring of *trx*¹/*+* females show only about 50% penetrance. The temperature-sensitive period occurs prior to hatching. Ingham (1980) noted the following abnormalities in extreme *trx*¹ mutants: (1) Extra bristles between humerus and coxa and on the distal tibia of the first leg; (2) Similar changes on the third leg; (3) Loss or reduction of transverse bristle rows, and, in males, decrease in number of sex comb teeth on the first leg; (4) Replacement of halter disk derivatives by wing blade, notal, and scutellar structures; (5) Rotated genitalia and abnormal tergite pigmentation in male flies. 75% of heterozygotes with *Df(3R)red* or with the lethal allele *trx*³ are lethal, either as larvae or pupae (Ingham, 1981); the heterozygotes that survive show cuticular transformations of the ventral prothorax and the metathorax, an extra mesonotum developing posterior to the normal one, and anteriorly-directed abdominal transformations (Ingham, 1985a).

***trx*²**

phenotype: The recessive embryonic lethal *trx*² fails to complement *trx*¹, *trx*³, or *trx*^D, either for the transformation phenotype or for lethality. Trans-heterozygotes

of *trx*² with another allele or with a deficiency for the locus show weak expression of the *trx* homeotic phenotype and about 40-50% pupal lethality. Since the lethal mutations are cell viable, *trx*²/*trx*³ clones have been induced by mitotic recombination in *trx*²/*trx*³; *Dp(3;1)kar*⁵¹, *trx*⁺ flies (Ingham, 1981, 1985b). The clones produced showed transformations of the antenna, eye, head capsule, and proboscis, bristle abnormalities in the legs, vein and bristle abnormalities in the wings, and transformations of halter to wing tissue and of genital to thoracic tissue; mutant clones in abdominal segments one to seven were not found (Ingham, 1985b).

*trx*³

phenotype: Like *trx*². About 75% of the *trx*¹/*trx*³ trans-heterozygotes are lethals, larval and pupal (Ingham, 1981).

trx^D

phenotype: Adult *trx*^D/+ flies are viable and characterized by their "bithorax variegated" phenotype (Lewis). These mutants show no prothoracic transformations, but do show patchy transformations of halter into wing and third leg into second leg (as in *bx* and *pbx*) and variable transformations of posterior abdominal segments into more anterior ones (Capdevila and García-Bellido, 1981; Duncan and Lewis, 1982). In homozygotes, deficiency heterozygotes, or trans-heterozygotes over *trx*¹, *trx*², or *trx*³, the *trx*^D allele is lethal or semilethal in larvae or pupae; in clones it is cell viable. Transformed *trx*^D/*trx*^D clones were found in the head region (but not in the thorax or abdomen) by Capdevila and García-Bellido (1981), while transformed *trx*²/*trx*^D clones were found in both head and thorax (but not in the abdomen) by Ingham (1985b).

Try: Trypsin

location: 2- [60].

references: Davis, 1985, Genetics 110: s12.

phenotype: Structural gene for enzyme trypsin.

cytology: Placed in 47D-47F.

**ts: telescope*

location: 2-68.

discoverer: Bridges, 15127.

references: Bridges and Morgan, 1919, Carnegie Inst. Wash. Publ. No. 278: 291 (fig.).

phenotype: Abdominal segments somewhat drawn out. Wings drooping and divergent. Overlaps wild type. RK3.

ts398: see *l(1)IIA*f

tsg: twisted gastrulation

location: 1-36.8.

origin: Induced by ethyl methanesulfonate.

references: Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307. Zusman and Wieschaus, 1985, Dev. Biol. 111: 359-71. Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-52.

phenotype: Gene expressed 1.5-3.5 hours after oviposition. Mutants are embryonic lethals and show abnormal gastrulation, with deep dorsal folds resulting in temporary blockage of germband extension. Later, dorsal

folds released, but posterior midgut abnormal in position, dorsal cells very thick, and cephalic folds very deep (Zusman and Wieschaus, 1985). At the end of embryonic development, *tsg* cuticle shows head defects and condensed, retracted posterior spiracles. Ventral nervous system split posteriorly. No particular cell must be wild type for survival in *tsg*//+ mosaics, but wild-type cells on dorsal side are most effective in rescue (Zusman and Wieschaus, 1985).

alleles: Four *tsg* alleles have been described.

allele	origin	synonym	ref ^α
<i>tsg</i> ¹	EMS	<i>tsg</i> ^{B8}	2, 3
<i>tsg</i> ²	EMS	<i>tsg</i> ^{N9}	2, 3
<i>tsg</i> ³	EMS		2
<i>tsg</i> ⁴	EMS	<i>tsg</i> ^{RF32}	1

^α 1 = Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-52; 2 = Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307; 3 = Zusman and Wieschaus, 1985, Dev. Biol. 111: 359-71.

cytology: Placed in 11A1-8 since uncovered by *Df(1)KA10* = *Df(1)11A1;11A7-8* (Wieschaus *et al.*, 1984); also placed in 11A2-3 (Perrimon *et al.*, 1989).

molecular biology: The region containing *tsg* has been cloned and the nucleotide sequence determined (Konrad and Marsh, 1989). A transcript has been found in early embryos.

tsl: torsolike

location: 3-71.

origin: Induced by ethyl methanesulfonate.

references: Frohnhöfer and Nüsslein-Volhard.

Nüsslein-Volhard, Frohnhöfer, and Lehmann, 1987, Science 238: 1675-81.

phenotype: Maternal-effect lethal. Anterior- and posteriormost structures (labrum, dorsal bridge, telson, eighth and part of seventh abdominal segments) deleted in embryos produced by homozygous mothers. *tsl* pole cells transplanted into wild-type hosts produce normal progeny, whereas the reciprocal transplant produces *tsl* embryos.

alleles:

allele	synonym	comments
<i>tsl</i> ¹	<i>tsl</i> ⁰³⁵	
<i>tsl</i> ²	<i>tsl</i> ¹³⁵	temperature-sensitive
<i>tsl</i> ³	<i>tsl</i> ¹⁴⁶	
<i>tsl</i> ⁴	<i>tsl</i> ⁶⁹¹	
<i>tsl</i> ⁵	<i>tsl</i> ¹⁷⁴	
<i>tsl</i> ⁷	<i>tsl</i> ^{MK}	

cytology: Placed in 93F6-14; uncovered by *Df(3R)e-F4* = *Df(3R)93C3-6;93F11-14*, but not by *Df(3R)e-D7* = *Df(3R)93C3-6;93F6-8*.

tt: tilt

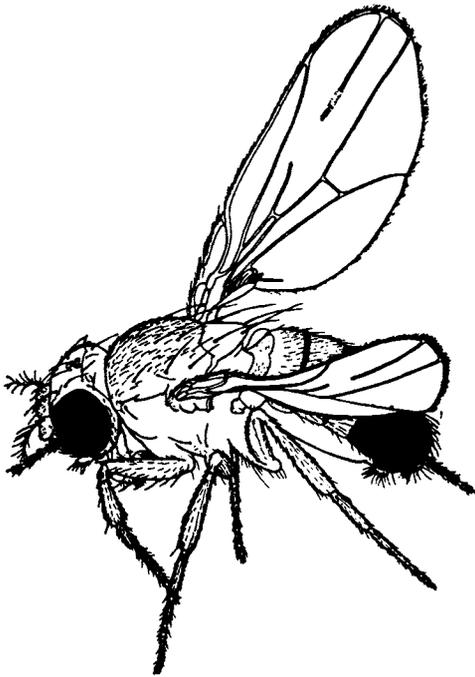
location: 3-40.0.

discoverer: Bridges, 15h29.

references: Bridges and Morgan, 1923, Carnegie Inst. Wash. Publ. No. 327: 134 (fig.). Mossige, 1938, Hereditas 24: 115.

phenotype: Wings spread, elevated, and warped in a compound curve. Vein L3 shows gap. Eye color may be slightly dilute. Developmentally, L3 originally complete but central section disappears during contraction period (Waddington, 1940, J. Genet. 41: 75-139). Synthetic

lethal in presence of *su(r)* (Strømten, 1974, Hereditas 78: 157-68). RK2.



tt: tilt

From Bridges and Morgan, 1923, Carnegie Inst. Wash. Publ. No. 327: 135.

tta: tetanic

location: 1-.

references: Ferrús, Llamazares, de la Pompa, and Yuste, 1987, *J. Neurogenet.* 4: 125-26.

phenotype: Viable, but shows tetanization under anaesthesia. Unable to learn. *tta* males with two doses of the Shaker gene complex are lethal.

Ttk: Tramtrack

location: 3-{102}.

references: Harrison and Travers, 1990, *EMBO J.* 9: 207-16.

phenotype: Gene encodes a zinc-finger protein binding to a number of sites involved in the transcriptional control of *fushi-tarazu* in the Antennapedia complex.

cytology: Located in 100D by *in situ* hybridization to the salivary chromosomes.

molecular biology: *Ttk* clones from a *Drosophila melanogaster* embryonic cDNA library contain a protein associated with binding site #16 in the *ftz* promoter region. At least three additional *Ttk* binding sites are believed to be present in the *ftz* promoter [Harrison and Travers, 1988, *Nucleic Acids Res.* 16: 11403-16; Topol, Wiederrecht and Parker, 1987, *Genetic Regulation of Development* (Loomis, ed.). Alan R. Liss, New York, pp. 3-12]. Nucleotide and putative amino acid sequences were obtained from the *Ttk* cDNA (Harrison and Travers, 1990). The translated protein has a predicted molecular weight of 69 kd. Two zinc-finger DNA-binding motifs (Cys-Cys His-His class) occur within the 327 amino acids believed to make up the *Ttk* binding domains; the zinc fingers show significant homology to the consensus finger motif and to the fingers of the protein products of

Kr and *hb* (Rosenberg, Schroder, Preiss, Kienlin, Côté, Riede, and Jäckle, 1986, *Nature* 319: 336-39; Tautz, Lehmann, Schnurch, Schuh, Seifert, Kienlin, Jones, and Jäckle, 1987, *Nature* 327: 383-89). The *Ttk* protein also contains PEST sequences found in proteins with short half-lives, as well as a serine and alanine-rich region at the C terminus [as in the *hb* and *inv* gene products (Coleman, Poole, Weir, Soeller, and Kornberg, 1987, *Genes Dev.* 1: 19-28]. *Ttk* RNA (probably maternal in origin) can be detected in embryos before stage 3; this RNA has almost disappeared by the start of *ftz* expression in stage 4 and is completely absent by the time the characteristic seven *ftz* stripes develop in the embryo. *Ttk* zygotic expression can be detected in stage 7 in the anterior mid-gut primordia and pole cells and later in the posterior mid-gut primordia. At stage 9, when *ftz* stripes are no longer present, *Ttk* RNA forms a pattern of about 14 stripes in the ectoderm and mesoderm of the extended germ-band. These stripes later fuse, forming a "tram-track" pattern around the germ-band; at stage 13, the *Ttk* RNA is only found in the developing epidermis and gut. Larvae and adults show very weak expression of the zygotic transcript.

other information: *Trf100D*: Transcription factor in 100D is a more informative designation.

****ttr: tetrapter***

location: 3-51.3.

discoverer: Tshetverikov, 25b.

references: Astaurov, 1929, Wilhelm Roux's *Arch. Entwicklungsmech. Org.* 115: 424-47.

1930, *Z. Indukt. Abstamm. Vererbungsl.* 55: 183-262.

Timoféeff-Ressovsky, 1934, *Z. Indukt. Abstamm. Vererbungsl.* 67: 248 (fig.).

Villee, 1942, *Univ. Calif. (Berkeley) Publ. Zool.* 49: 180-81.

phenotype: Like *bx*. Halteres tend to become winglike. Most flies wild type but may have, in place of a haltere, an organ one-half the size of a normal wing with veins, bristles, and sense organs. RK3.

***ttx: tetrodotoxin-sensitive* (J.C. Hall)**

location: 3-45.

origin: Spontaneous.

discoverer: Kelly, L.M. Hall.

references: Gitschier, Strichartz, and Hall, 1980, *Biochim. Biophys. Acta* 595: 291-303.

phenotype: Adults and larvae highly sensitive to the sodium channel blocker tetrodotoxin (TTX) by feeding; mutant adults are especially sensitive.

alleles: One mutant allele, *ttx*¹, causing accentuated TTX sensitivity.

tu: tumor

General term used to denote genes that lead to formation of single or multiple melanotic masses, either free or attached to internal organs in the abdomen or thorax. In most cases, these tumors (called "pseudotumors" by Barigozzi, 1968) appear in third instar larvae and may persist throughout pupal and adult life. They are formed by aggregation of hemocytes ("plasmatocytes") that have been transformed into flattened disk-like cells ("lamellocytes") which encapsulate other tissues (Rizki, 1957, *J. Morphol.* 100: 437-58; Rizki and Rizki, 1974, *J. Invert. Path.* 24: 37-40). Just before pupation, these masses of

mutant	location	origin	synonym	discoverer	ref ^α	comment
*tu-1a	2-	spont		Payne	60	in combination with <i>tu-1b</i> , produces melanotic tumors in 20% of larvae and 14% of adults; larval lethal in 57-81%
*tu-1b	3-	spont		Payne	60	in combination with <i>tu-1a</i> , produces melanotic tumors in 20% of larvae and 14% of adults; larval lethal in 57-81%
*tu-2	2-	spont		Payne	60	melanotic tumors in 20-100% of larvae; lethal in 41%
tu-36e		spont		Skoog, 36e15	56	low penetrance
tu-47		spont			56	low penetrance
tu-48	2-29.5	spont	<i>tu-48a</i>	Ghelelovitch	15, 23, 24, 56	melanotic tumors in abdomen of 3rd instar larvae; penetrance 96%; modifiers on X and 4 (?)
tu-48^{49h}	2-	spont	<i>tu-49h</i>	Brcic	7, 11, 13	penetrance 50-60%; fertility and viability good; modifier on 3
tu-48^{A2}	2-(25.8-28.5)	spont	<i>tu-A2</i>	Barigozzi	1-3, 21	low penetrance
tu-48j	2-46	spont		Herskowitz	11, 27, 28	melanotic tumors in 50% of larvae and in adult (abdomen)
tu-49k	2-(75-100)	spont		Oftedal	38, 39	black hemocyte aggregations in late larvae; modifiers on X and 2
tu-53	1-41	neutrons	<i>tu-53l</i>	Darrow, 53l20	32	small melanotic tumors in 15-20% of adults; wing abnormalities; egg hatch 65% of normal and delayed; lethality 50-70%
tu-54e		spont		Haddox, 54e25	12	small melanotic tumors in 0.44% of adults under tergites 1 and 2
*tu-59h	2-	spont		Oshima	40	Small melanotic tumors in late larvae and adults
*tu-be	3-25	spont	<i>*be-3</i>	Stark, 16k	57-59	melanotic tumors in larvae and adults; nonlethal; modifiers
tu-bw	2-(80.5-84)	spont	<i>mt^A</i>	Morgan, 1922	8, 9, 11, 21, 26, 37, 52, 55	numerous melanotic tumors in larval hemocoel and in adults; penetrance 59-86%; suppressed by <i>su(tu-bw)</i> , affected by diet
tu-bw^{36a}	2-	spont	<i>tu-36a</i>	Bridges, 36a16	11, 14, 50, 51	melanotic tumors in late larvae; penetrance 5-8%
tu-bw^{50d}	2-		<i>tu-50d</i>		11, 14, 56	penetrance 12-27%; enhancer on X
tu-bw^{50j}	2-	spont	<i>tu-50j</i>	Mittler, 1950	34-36	penetrance 5-10%; melanotic spots under ventral abdominal surface; mutant viable and fertile
tu-bw^{55g}	2-	spont	<i>tu-55g</i>	Jacobs	21, 30	melanotic tumors in larvae and adults; almost 100% penetrance; usually in abdomen but may be in thorax or head (rare)
tu-bw^{B3}	2-(85-90)	spont	<i>tu-B3</i>	Barigozzi; Di Pasquale	2, 3, 15, 21, 41, 56	suppressed by <i>su(tu-bw)</i> ; penetrance 90%
tu-bw^e	2-88.0	spont	<i>e¹¹ tu</i>		22, 25, 31	melanotic tumors in posterior of larvae and abdomen and thorax of adults
tu-C4	3-(52-53)	spont		Barigozzi	2, 5, 6	incomplete dominance; modifiers probably on 2
tu-D	2-	spont		Barigozzi	2	
tu-e144	2-	spont	<i>e144</i>	Barigozzi	2, 4	
tu-K	2-	spont		Sang, Burnet	8, 9, 16, 53-55	small black nodules in hemocoel or with fat body in late larvae; penetrance low but can be increased by dietary changes
tu-mt	3-		<i>mt</i>		33	melanotic tumors in abdomen in 0-100% of larvae
tu-mwh					2, 17	pupal lethal; 88% tumorous; lymph gland melanotic mass
tu-pb	2-3-(62-70.7)	spont			18, 19, 20	melanotic tumors at base of proboscis
*tu-R	1-	spont		Rosenberg	29	penetrance 40%; lethality 50%; bristles short
tu-So^c		spont			2, 42	melanotic masses in 33% of larvae and 5% of adults; semilethal
tu-Sz^{ts β}	1-34.3	EMS		Suzuki	45, 46	melanotic tumors in caudal

mutant	location	origin	synonym	discoverer	ref ^α	comment
<i>tu-W</i>	2-66.2	dibenzanthracene		Gowen	8, 9, 43, 44, 47, 56, 61	fat bodies of larvae at 26°; none at 18° melanotic tumors in caudal fat bodies; penetrance incomplete though raised to 100% by selection
<i>tu-W^g</i>	2-		<i>tu-g</i>		11, 13, 14, 56, 62	melanotic tumors in caudal fat bodies in late larvae; penetrance 50-100%
<i>tu-wps</i>					10, 11, 14	penetrance up to 17%

^α 1 = Barigozzi, 1962, Atti Assoc. Genet. Ital. 7: 9-76; 2 = Barigozzi, 1968, Nat. Canc. Inst. Monogr. 31: 277-90; 3 = Barigozzi and Di Pasquale, 1956, Rend. Inst. Lomb. Sci. Lett. 90: 484-509; 4 = Barigozzi, Castiglione, and Di Pasquale, 1960, Heredity 14: 151-62; 5 = Belt, 1971, J. Insect. Physiol. 17: 1217-23; 6 = Belt and Burnet, 1972, Genet. Res. 20: 115-35; 7 = Brncic, 1950, DIS 24: 57; 8 = Bryant and Sang, 1968, Nature (London) 220: 393-94; 9 = Bryant and Sang, 1969, Genetics 62: 321-36; 10 = Burdette, 1951, Acta Unio. Int. Cancrum 7: 670-74; 11 = Burdette, 1951, DIS 25: 101-02; 12 = Burdette, 1954, DIS 28: 73; 13 = Burdette, 1959, Univ. Texas Publ. 5914: 57-68; 14 = Burdette and Carver, 1970, DIS 45: 151; 15 = Burnet, 1966, DIS 41: 161; 16 = Burnet and Sang, 1964, Genetics 49: 599-610; 17 = Castiglione, 1958, DIS 32: 118; 18 = Di Pasquale and Cavolina, 1983, DIS 59: 31-33; 19 = Di Pasquale and Cavolina, 1984, DIS 60: 83-84, 84-86; 20 = Di Pasquale, Cavolina, Romano, and Ribaudo, 1987, DIS 66: 47-48; 21 = Erk and Sang, 1966, DIS 41: 95; 22 = Friedman, Harnly, and Goldsmith, 1951, Cancer Res. 11: 904-11; 23 = Ghelelovitch, 1950, C.R. Hebd. Seanc. Acad. Sci., Paris 230: 1002-04; 24 = Ghelelovitch, 1958, Biologie Med. (Paris) 47: 711-810; 25 = Glassman, 1956, DIS 30: 116; 26 = Hartung, 1950, J. Hered. 41: 269-72; 27 = Herskowitz, 1949, DIS 23: 57; 28 = Herskowitz and Burdette, 1951, J. Exp. Zool. 117: 499-521; 29 = Hinton, 1957, DIS 31: 83; 30 = Jacobs, Bowman, and Walliser, 1958, DIS 32: 130; 31 = Kaplan, 1955, Trans. N.Y. Acad. Sci. 17: 289-93; 32 = King, 1955, DIS 29: 73; 33 = Mampell, 1967, Genetica 37: 449-65; 34 = Mittler, 1951, DIS 25: 74; 35 = Mittler, 1952, Science 115: 271-72; 36 = Mittler, 1952, Science 116: 657-59; 37 = Morgan, 1938, DIS 9: 108; 38 = Oftedal, 1951, DIS 25: 122-23; 39 = Oftedal, 1953, Z. Indukt. Abstamm. Vererbungsl. 85: 408-22; 40 = Oshima, 1959, DIS 33: 99; 41 = Perotti, Bairati, and Bairati, 1968, J. Invert. Path. 10: 122-38; 42 = Raimondi, 1956, DIS 30: 147; 43 = Rizki and Rizki, 1974, Experientia 30: 543-46; 44 = Rizki and Rizki, 1974, J. Invert. Path. 24: 37-40; 45 = Rizki and Rizki, 1979, Genetics 91: s103-104; 46 = Rizki and Rizki, 1980, Wilhelm Roux's Arch. Dev. Biol. 189: 197-206; 47 = Rizki and Rizki, 1981, J. Hered. 72: 78-80; 48 = Rizki, Rizki, and Bellotti, 1985, Mol. Gen. Genet. 201: 7-13; 49 = Rizki, Rizki, Bebbington, and Roberts, 1983, Wilhelm Roux's Arch. Dev. Biol. 192: 1-7; 50 = Russell, 1940, J. Exp. Zool. 84: 363-79; 51 = Russell, 1942, Genetics 27: 612-18; 52 = Sang, 1969, Nat. Cancer Inst. Monogr. 31: 291-301; 53 = Sang and Burnet, 1963, Genetics 48: 235-53; 54 = Sang and Burnet, 1964, Genetics 49: 223-35; 55 = Sang and Burnet, 1967, Genetics 56: 743-54; 56 = Sparrow, 1974, Genet. Res. 23: 13-21; 57 = Stark, 1919, Proc. Nat. Acad. Sci. USA 5: 573-80; 58 = Stark, 1935, DIS 4: 62; 59 = Stark and Bridges, 1926, Genetics 11: 249-66; 60 = Wilson, 1924, Genetics 9: 343-62; 61 = Wilson, King, and Lowry, 1955, Growth 19: 215-44; 62 = Yamazaki and Ohnishi, 1968, Genetics 59: 237-43.

^β cytology: Located in 10A9 to 10B8 since included in *Df(1)KA7 = Df(1)10A9;10F6-7* but not in *Df(1)N71 = Df(1)10B2-8;10D3-8*.

tumor cells become melanized. Some of the melanotic tumor mutants are larval lethals [for example, eight lethals in the *Ddc* region described by Wright, 1987 (Adv. Genet. 24: 127-222)]; a few of the melanotic lethals are malignant, such as the *mbn* mutants of Gateff (1974, DIS 51: 21; 1977, DIS 52: 4) and the mutant *Tum* (Hanratty and Ryerse, 1981, Dev. Biol. 83: 238-49). In many cases, however, the melanotic tumors are benign and do not affect adult viability. Most of the genes designated *tu* are recessive and act in combination with modifying factors. The encapsulation and subsequent melanization (described for *tu-bw*, *tu-bw^{B3}*, *tu-C4*, *tu-K*, *tu-Sz¹⁵* and *tu-W*) seem to be a reaction to developmental abnormalities in the mutants. General references for the *tu* mutants include:

- CP627.
- Sparrow, 1978, The Genetics and Biology of *Drosophila* (Ashburner and Wright, eds.). Academic Press, London, New York, San Francisco, Vol. 2b, pp. 277-313.

The preceding table describes the mutants and their alleles.

Tu: Turned-up wing

- location:** 1-59.
- origin:** X ray induced.
- discoverer:** Muller, 46i19.
- references:** Muller and Valencia, 1947, DIS 21: 70.
- phenotype:** Wings curled; somewhat wrinkled in longitudinal direction. Heterozygous viability good; homozygote also viable. RK1.

Tu: see *Tb*

tul: see *tuhl*

TU36B: see Cyt-b

tub: tube

- location:** 3-47.
- origin:** Induced by ethyl methanesulfonate.
- references:** Anderson and Nüsslein-Volhard, 1984, Nature (London) 311: 223-27.
Carroll, Winslow, Twombly, and Scott, 1987, Development 99: 327-32.
Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.
- phenotype:** Maternal effect embryonic lethal. Embryos can be rescued with wild-type cytoplasm and RNA; however, none hatched (Anderson and Nüsslein-Volhard, 1984). Dorsalization observed in the pattern of *ftz* stripes in *tub* embryos (Carroll *et al.*, 1987).
- alleles:** *tub¹* and *tub²* recovered as *tub¹¹⁸* and *tub²³⁸*.

Tub: Tubulin

Tubulin proteins are found in a wide variety of species from unicellular organisms to man; their biochemical and molecular structure is highly conserved. The α - and β -subunits from different organisms can be combined *in vitro* into hybrid microtubule structures and there is a high level of primary amino acid sequence identity in the proteins (Sánchez, Natzle, Cleveland, Kirschner, and McCarthy, 1980, Cell 22: 845-54; Raff, 1984, J. Cell Biol. 99: 1-10).

In *Drosophila melanogaster*, two multigene families, each made up of four members, code for α - and β -tubulins, each tubulin subunit being a 55,000 dalton polypeptide. Tubulins are the main structural components of microtubules in mitotic and meiotic spindles, cilia, flagella, neural processes, and the cytoskeleton; nontubulin proteins (MAPS or microtubule-associated proteins) are involved along with tubulins in the forma-

tion of specialized microtubules (Theurkauf, Baum, Bo, and Wensink, 1986, Proc. Nat. Acad. Sci. USA 83: 8477-81; Rudolph, Kimble, Hoyle, Subler, and Raff, 1987, Mol. Cell Biol. 7: 2231-42). The tubulin genes in each multigene family are dispersed in the second and/or third chromosomes rather than arranged in clusters.

(1) α -Tubulin: Codes for the α -subunit; message of about 2000 bp.

(2) β -Tubulin: Codes for the β -subunit; message of about 1800 bp.

Variants of both α - and β -subunits have been detected. The following table summarizes current information in regard to these genes in *Drosophila melanogaster*.

locus	synonym	location	cytology	comments
α Tub67C	$\alpha 4t$	3-[29]	67C4-6	maternal
α Tub84B	$\alpha 1t$	3-47.8	84B3-6	ubiquitous
α Tub84D	$\alpha 3t$	3-48	84D4-8	ubiquitous; except in adult male
α Tub85E	$\alpha 2t$	3-49	85E6-10	expressed from 7 hr through adult
β Tub56D	$\beta 1t; \beta 4t$	2-[87]	56D	maternal; ubiquitous
β Tub60D	$\beta 3t$	2-[107]	60D	embryonic; pupal
β Tub85D	$\beta 2t$	3-48.5	85D4-7	testis-specific
β Tub97EF	$\beta 4t; \beta 1t$	3-[92]	97E-F	ubiquitous

α Tub67C ($\alpha 4t$)

references: Sánchez Natzle, Cleveland, Kirschner, and McCarthy, 1980, Cell 22: 845-54.

Kalfayan and Wensink, 1981, Cell 24: 97-106.

Kalfayan, Loewenberg, and Wensink, 1982, Cold Spring Harbor Symp. Quant. Biol. 46: 185-90.

Kalfayan and Wensink, 1982, Cell 29: 91-98.

Baum, Livneh, and Wensink, 1983, Nucleic Acids Res. 11: 5569-87.

Natzle and McCarthy, 1984, Dev. Biol. 104: 187-98.

Theurkauf, Baum, Bo, and Wensink, 1986, Proc. Nat. Acad. Sci. USA 83: 8477-81.

Matthews, Miller, and Kaufman, 1989, Dev. Biol. 132: 45-61.

Rees, Kaufman, and Matthews.

phenotype: A structural gene for α -tubulin. It is transcribed into mRNA that is maternal in origin and is synthesized in the nurse cells; the transcript accumulates in 0- to 3-hr embryos and adult ovaries (Kalfayan and Wensink, 1982; Matthews *et al.*, 1989). The protein is clearly different from the other α -tubulins of *Drosophila melanogaster* as well as from those of other animal species. A stable pool of $\alpha 67C$ tubulin is found in ovaries, unfertilized eggs, and embryos, but synthesis of the protein probably only occurs in the ovary (Matthews *et al.*, 1989).

allele	origin	discoverer	comments
α Tub67C ¹	EMS	Matthews	antimorph
α Tub67C ²	EMS	Rees	hypomorph
α Tub67C ³	X-ray	Rees	hypomorph
α Tub67C ⁴	DEB	Rees	hypomorph

cytology: Located in 67C4-6 by *in situ* hybridization (Sánchez *et al.*, 1980; Kalfayan and Wensink, 1981; Mischke and Pardue, 1982, J. Mol. Biol. 156: 449-66; Natzle and McCarthy, 1984); included in *Df(3L)AC1 = Df(3L)67A5;67D9-13* (Carpenter). Single copy present (Kalfayan and Wensink, 1981).

molecular biology: Gene cloned (Kalfayan and Wensink,

1981, 1982; Natzle and McCarthy, 1984), nucleotide sequences determined, and transcript maps formulated (Theurkauf *et al.*, 1986). α Tub67C tubulin differs markedly from the other three α -tubulins although it is similar in length and shares many sequences with the others; it ends with a carboxyl-terminal phenylalanine residue instead of the tyrosine characteristic of the other tubulins and differs from the α -tubulin of α Tub84B at 149 of the 450 residues (Theurkauf *et al.*, 1986). The 3' end of α Tub67C is complementary to RNA bands on gels of 1.50, 1.70, 1.90, and 1.95 kb (Kalfayan and Wensink, 1982). A transcript of 1.5 kb was detected in early embryos and adult females (Matthews *et al.*, 1989).

α Tub84B ($\alpha 1t$)

references: Sánchez, Natzle, Cleveland, Kirschner, and McCarthy, 1980, Cell 22: 845-54.

Kalfayan and Wensink, 1981, Cell 24: 97-106.

Kalfayan, Loewenberg, and Wensink, 1982, Cold Spring Harbor Symp. Quant. Biol. 46: 185-90.

Kalfayan and Wensink, 1982, Cell 29: 91-98.

Mischke and Pardue, 1982, J. Mol. Biol. 156: 449-66.

Baum, Livneh, and Wensink, 1983, Nucleic Acids Res. 11: 5569-87.

Mischke and Pardue, 1983, J. Submicrosc. Cytol. 15: 367-70.

Natzle and McCarthy, 1984, Dev. Biol. 104: 187-98.

Cavener, Otteson, and Kaufman, 1986, Genetics 114: 111-23.

Theurkauf, Baum, Bo, and Wensink, 1986, Proc. Nat. Acad. Sci. USA 83: 8477-81.

Matthews and Kaufman, 1987, Dev. Biol. 119: 100-14.

Matthews, Miller, and Kaufman, 1989, Dev. Biol. 132: 45-61.

phenotype: A structural gene for α -tubulin; present in one copy per haploid genome; transcribed into mRNA in the oocyte and the embryo, reaching a maximum concentration 6-9 hr after the egg is laid (Kalfayan and Wensink, 1982). A variety of post-translational modifications of α Tub84b protein found in all tissues, some of which are tissue-specific (Matthews *et al.*, 1989). The gene appears to be constitutively expressed, its functions being common to most cells; the resulting tubulins are very similar to those of other animal species (Theurkauf *et al.*, 1986). Mutations in α Tub84B have been induced as lethals over *Df(3R)Scx2*. Alleles α Tub84B¹ and α Tub84B³ produce proteins that are electrophoretic variants of the wild-type tubulin. Based on the extent of lethality and sterility found in interallelic heterozygotes and in heterozygotes over wild type, the severity of α Tub84B alleles can be ordered as follows: α Tub84B⁶ < α Tub84B¹ < α Tub84B² < α Tub84B⁴ < α Tub84B⁵ = α Tub84B³ (Matthews and Kaufman, 1987). Maternal-effect as well as zygotic lethality is shown by the more severe alleles. In the less severe hypomorphic alleles, heterozygotes die late in pupal life or early in adult life, the adults showing head, thoracic, or abdominal defects, bristle abnormalities, leg tremors, and sterility (Matthews and Kaufman, 1987).

alleles: Seven alleles induced by ethyl methanesulfonate.

allele	discoverer	synonym	ref ^α	comments
α Tub84B ¹	Denell	/(3)d10	1, 2	hypomorph
α Tub84B ²	Grigliatti	/(3)5.10	1, 2	hypomorph
α Tub84B ³	Cavener	/(3)g3	1, 2	null

allele	discoverer	synonym	ref ^α	comments
<i>αTub84B</i> ⁴	Matthews		2	hypomorph
<i>αTub84B</i> ⁵	Matthews		2	null
<i>αTub84B</i> ⁶	Matthews		2	hypomorph
<i>αTub84B</i> ⁷	Kemphues	<i>αTub84B^{nc33}</i>	3	hypomorph

^α 1 = Cavener, Otteson, and Kaufman, 1986, Genetics 114: 111-23;
 2 = Matthews and Kaufman, 1987, Dev. Biol. 119: 100-14.
 3 = Hayes, Deuring, Robertson, Prout, and Fuller, 1989, Mol. Cell Biol. 9: 875-84.

cytology: Located in 84B3-6 by *in situ* hybridization (Sánchez *et al.*, 1980; Kalfayan and Wensink, 1981; Mischke and Pardue, 1982; Natzle and McCarthy, 1984); cytologically located in 84B since included in *Df(3R)Scx2 = Df(3R)84A4-5;84C1-2* (Matthews and Kaufman, 1987), but not in *Df(3R)Win3 = Df(3R)84A4-5;84B1-2* (Cavener *et al.*, 1986) or *Df(3R)dsx2M = Df(3R)84C1-2;84D14* (B. Baker). The strongest hybridization among the α -tubulins is observed at the 84B site; cytological data suggest the presence of two copies of *αTub84B* per haploid genome (Mischke and Pardue, 1982, 1983), the cloned DNA fragments hybridizing to salivaries and producing two rows of silver grains at 84B3-6.

molecular biology: Gene cloned (Kalfayan and Wensink, 1981, 1982; Mischke and Pardue, 1982; Natzle and McCarthy, 1984), nucleotide sequences determined, and transcript maps formulated (Theurkauf *et al.*, 1986). *αTub84B* and *αTub84D* show 92% nucleotide sequence identity within the protein coding region, the tubulins they encode differing by two substitutions, isoleucine-170 and cysteine II in the 84B tubulin being replaced by valine residues in the 84D tubulin. These *αTub* genes also have blocks of identical sequences outside the coding region (between nucleotides +27 and +147 of *αTub84B* and nucleotides +354 and +503 of *αTub84D*). The tubulin encoded by *αTub85E* differs from the *αTub84B* polypeptide at 21 of the 450 residues, while the tubulin encoded by *αTub67C* differs from the polypeptide of *αTub84B* at 149 residues. The *αTub84B* tubulin, like most of the other α -tubulins except the *αTub67C* protein, ends with a tyrosine residue (Theurkauf *et al.*, 1986). The 3' end of *αTub84B* is complementary to an RNA band on a gel of approximately 1.8 kb (Kalfayan and Wensink, 1982).

***αTub84D* (*α3t*)**

references: Sánchez, Natzle, Cleveland, Kirschner, and McCarthy, 1980, Cell 22: 845-54.
 Kalfayan and Wensink, 1981, Cell 24: 97-106.
 Kalfayan, Loewenberg, and Wensink, 1982, Cold Spring Harbor Symp. Quant. Biol. 46: 185-90.
 Kalfayan and Wensink, 1982, Cell 29: 91-98.
 Mischke and Pardue, 1982, J. Mol. Biol. 156: 449-66.
 Baum, Livneh, and Wensink, 1983, Nucleic Acids Res. 11: 5569-87.
 Mischke and Pardue, 1983, J. Submicrosc. Cytol. 15: 367-70.
 Natzle and McCarthy, 1984, Dev. Biol. 104: 187-98.
 Fuller, 1986, Symp. Soc. Dev. Biol. 44: 19-41.
 Theurkauf, Baum, Bo, and Wensink, 1986, Proc. Nat. Acad. Sci. USA 83: 8477-81.
 Matthews and Kaufman, 1987, Dev. Biol. 119: 100-14.
 Matthews, Miller, and Kaufman, 1989, Dev. Biol.

132: 45-61.

synonym: *ms(3)nc33* (located in 84D).

phenotype: A structural gene for α -tubulin. Coordinately expressed with *αTub84B* at most developmental stages with the exception of the adult stage where no 84D mRNA can be detected in males (Matthews *et al.*, 1989).

alleles: *αTub84D^{nc33}* [= *ms(3)nc33*] induced by ethyl methanesulfonate, fails to complement certain β *Tub* mutant alleles for male fertility and is male sterile when homozygous (Fuller, 1986).

cytology: Located in 84D4-8 by *in situ* hybridization (Sánchez *et al.*, 1980; Kalfayan and Wensink, 1981; Mischke and Pardue, 1982; Natzle and McCarthy, 1984); cytologically located in 84C-D since included in *Df(3R)dsx2D = Df(3R)84C1-3;84D14* (B. Baker), but not in *Df(3R)Scx2 = Df(3R)84A4-5;84C1-2* (Matthews and Kaufman, 1987). Single copy present (Kalfayan and Wensink, 1981).

molecular biology: Gene cloned (Kalfayan and Wensink, 1981, 1982; Mischke and Pardue, 1982; Natzle and McCarthy, 1984), nucleotide sequences determined, and transcript maps formulated (Theurkauf *et al.*, 1986). The 3' end of *αTub84D* is complementary to an RNA band on a gel of approximately 1.95 kb (Kalfayan and Wensink, 1982; Matthews *et al.*, 1989). The transcript was found in both ovary and carcass RNA.

***αTub85E* (*α2t*)**

references: Sánchez, Natzle, Cleveland, Kirschner, and McCarthy, 1980, Cell 22: 845-54.
 Kalfayan and Wensink, 1981, Cell 24: 97-106.
 Kalfayan, Loewenberg, and Wensink, 1982, Cold Spring Harbor Symp. Quant. Biol. 46: 185-90.
 Kalfayan and Wensink, 1982, Cell 29: 91-98.
 Mischke and Pardue, 1982, J. Mol. Biol. 156: 449-66.
 Baum, Livneh, and Wensink, 1983, Nucleic Acids Res. 11: 5569-87.
 Mischke and Pardue, 1983, J. Submicrosc. Cytol. 15: 367-70.
 Natzle and McCarthy, 1984, Dev. Biol. 104: 187-98.
 Theurkauf, Baum, Bo, and Wensink, 1986, Proc. Nat. Acad. Sci. USA 83: 8477-81.
 Bo and Wensink, 1989, Development 106: 581-87.
 Matthews, Miller, and Kaufman, 1989, Dev. Biol. 132: 45-61.

phenotype: A structural gene for α -tubulin. Only α -tubulin not expressed maternally. Transcripts present from 6-8 hr through adult. Protein found in support cells of chondotonal organs, developing muscles, and somatic component of the testis (Matthews, Miller, and Kaufman).

cytology: Located in 85E6-10 by *in situ* hybridization (Sánchez *et al.*, 1980; Kalfayan and Wensink, 1981; Mischke and Pardue, 1982; Natzle and McCarthy, 1984). Single copy present (Kalfayan and Wensink, 1981).

molecular biology: Gene cloned (Kalfayan and Wensink, 1981, 1982; Mischke and Pardue, 1982; Natzle and McCarthy, 1984), nucleotide sequences determined, and transcript maps formulated (Theurkauf *et al.*, 1986). An *αTub85E*- β galactosidase fusion gene was expressed in chondotonal organs (sensory organs in the peripheral nervous system) and in the testes (Bo and Wensink, 1989). Chordotonal expression occurs in males and females from late embryonic to early pupal stages. The 3' end of *αTub85E* is complementary to bands on gels of approxi-

mately 1.65 kb in pupae and adult males and 1.8 kb in larvae according to Kalfayan and Wensink (1982); the 1.8 kb transcript was not found by Natzle and McCarthy (1984) or Matthews *et al.* (1989), who report a single 1.55 kb band.

β Tub56D ($\beta 1t$)

references: Raff, Fuller, Kaufman, Kempfues, Rudolph, and Raff, 1982, *Cell* 28: 33-40.

Bialojan, Falkenburg, and Renkawitz-Pohl, 1984, *EMBO J.* 3: 2543-48.

Natzle and McCarthy, 1984, *Dev. Biol.* 104: 187-98.

Raff, 1984, *J. Cell Biol.* 99: 1-10.

Michiels, Falkenburg, Müller, Hinz, Otto, Bellmann, Glätzer, Brand, Bialojan, and Renkawitz-Pohl, 1987, *Chromosoma* 95: 387-95.

Rudolph, Kimble, Hoyle, Subler, and Raff, 1987, *Mol. Cell. Biol.* 7: 2231-42.

Gasch, Hinz, Leiss, and Renkawitz-Pohl, 1988, *Mol. Gen. Genet.* 211: 8-16.

phenotype: A structural gene for β -tubulin. Its mRNA is maternally stored in the oocytes and expressed in the nurse cells and in early embryos (Natzle and McCarthy, 1984; Gasch *et al.*, 1988); at this stage, the transcripts are evenly distributed throughout the embryo, but later in development they are concentrated in the primordia of the nervous system and their derivatives (the supraoesophageal ganglion and ventral nerve cord) and in the visceral mesoderm. Since β Tub56D is expressed throughout development and in all adult tissues (Rudolph *et al.*, 1987), β Tub56D tubulin is probably involved in all the microtubular structures necessary for cell division and cell shape (Raff *et al.*, 1982; Bialojan *et al.*, 1984). It is the major β -tubulin of adult flies in all structures except the testis.

cytology: Located in 56C (Bialojan *et al.*, 1984) or in 56D (Natzle and McCarthy, 1984) by *in situ* hybridization.

molecular biology: Gene cloned (Bialojan *et al.*, 1984; Natzle and McCarthy, 1984); transcript of 1.8 kb present in all developmental stages (Raff, 1984); maximal expression in 6-9 hr embryos (Bialojan *et al.*, 1984). Complete nucleotide sequences and predicted amino acid sequences of the protein-coding regions have been determined (Michiels *et al.*, 1987; Gasch *et al.*, 1988). A single intron of 2.6 kb is found between codons for amino acids 19 and 20. The 5' untranslated region is encoded in the same exon as the first 19 amino acids. There is a typical TATA box promoter element at -27. Transcription starts at an adenosine residue as in most eukaryotic genes, the initiation site being 110 bases upstream of the translation initiation codon (Gasch *et al.*, 1988). The 3' untranslated region of β Tub56D mRNA is not identical to the 3' region of β Tub85D mRNA, but the protein coding regions show 95% identity at the amino acid level; there are 25 differences in amino acid sequence between these two tubulins (Michiels *et al.*, 1987).

β Tub60D ($\beta 3t$)

references: Sánchez, Natzle, Cleveland, Kirschner, and McCarthy, 1980, *Cell* 22: 845-54.

Raff, Fuller, Kaufman, Kempfues, Rudolph, and Raff, 1982, *Cell* 28: 33-40.

Bialojan, Falkenburg, and Renkawitz-Pohl, 1984, *EMBO J.* 3: 2543-48.

Natzle and McCarthy, 1984, *Dev. Biol.* 104: 187-98.

Raff, 1984, *J. Cell Biol.* 99: 1-10.

Rudolph, Kimble, Hoyle, Subler, and Raff, 1987, *Mol. Cell. Biol.* 7: 2231-42.

Gasch, Hinz, Leiss, and Renkawitz-Pohl, 1988, *Mol. Gen. Genet.* 211: 8-16.

Leiss, Hinz, Gasch, Mertz, and Renkawitz-Pohl, 1988, *Development* 104: 525-31.

Gasch, Hinz, and Renkawitz-Pohl, 1989, *Proc. Nat. Acad. Sci. USA* 86: 3215-18.

Kimble, Incardona, and Raff, 1989, *Dev. Biol.* 131: 415-29.

Kimble, Dettman, and Raff, 1990, *Genetics* 126: 991-1105.

phenotype: A structural gene for β -tubulin. The mRNA first appears in the mesoderm during mid-embryogenesis at about the time when synthesis of β -tubulin begins and later disappears from the embryo when synthesis of the tubulin ends; the transcript reappears in the mesoderm during the pupal period (Raff *et al.*, 1982; Natzle and McCarthy, 1984; Raff, 1984; Rudolph *et al.*, 1987; Gasch *et al.*, 1988; Kimble *et al.*, 1989). During embryogenesis, β Tub60C is only expressed in developing muscles; in pupae, it is expressed in adult muscles, imaginal discs, wing blades, optic lobes, ovaries and testes, but ceases (except in ovaries and testes) in adults. As compared to the tubulin encoded by β Tub56D, the product of β Tub60C is but a minor component of the total tubulin pool (Rudolph *et al.*, 1987). When synthesis of the β Tub60C tubulin begins in the embryo, α -tubulin synthesis is increased (Raff *et al.*, 1982).

alleles: Five EMS-induced alleles (Kimble *et al.*, 1990).

allele	synonym	comments	α	β
α Tub60D ¹	$\beta 3t^1$	intermediate hypomorph		
α Tub60D ²	$\beta 3t^2$	strong hypomorph		
α Tub60D ³	$\beta 3t^3$	strong hypomorph		
α Tub60D ⁴	$\beta 3t^4$	weak hypomorph		
α Tub60D ⁵	$\beta 3t^5$	intermediate hypomorph		

α Effect on viability: $\beta 3t^2 > \beta 3t^3 > \beta 3t^4 > \beta t^1 > \beta 3t^5$.

β Effect on fertility: $\beta 3t^3 > \beta t^2 > \beta 3t^4 > \beta 3t^1 > \beta t^3$.

cytology: Located in 60D; also located in 60A-B (Sánchez *et al.*, 1980), 60B (Bialojan *et al.*, 1984), or 60C (Natzle and McCarthy, 1984) by *in situ* hybridization; placed in 60C6-8 by other molecular methods (see Raff, 1984).

molecular biology: Gene cloned (Bialojan *et al.*, 1984; Natzle and McCarthy, 1984); 2.3-2.5 kb transcript found in mid-embryogenesis and during pupation (Bialojan *et al.*, 1984; Raff, 1984). The transcription initiation sites are identical in both of these developmental periods (Gasch *et al.*, 1988). Gene structure, nucleotide sequences, and predicted amino acid sequences of the protein-coding regions have been determined (Rudolph *et al.*, 1987; Gasch *et al.*, 1988). The tubulin encoded by β Tub60C is longer and slightly more basic than that encoded by β Tub56D, and contains 454 amino acids (Rudolph *et al.*, 1987). The first intron occurs between the codons for amino acids 19 and 20 and is about 4.5 kb long; the second intron occurs after the first nucleotide in the codon for amino acid 56 and is only 62 bp long; the third intron occurs between the codons for amino acids 137 and 138 and is about 56 bp long. Six amino acids not found in any other β -tubulin are positioned immediately after the 3' junction of the second intron in a highly variable region. The first and third introns occur in rela-

tively conserved regions of the protein coding sequence (Rudolph *et al.*, 1987). Upstream sequences of $\beta Tub56D$, $\beta Tub60C$, and $\beta Tub85D$ are highly divergent, with the exception of the sequence common to $\beta Tub60C$ and $\beta Tub85D$ promoters (Gasch *et al.*, 1988). Upstream of -1.2 kb (relative to the transcription start site) are regulatory elements required for expression of $\beta Tub60C$ in the somatic muscles, the pharyngeal muscles, and the dorsal vessel (Gasch *et al.*, 1989). The first intron carries an enhancer element necessary for $\beta Tub60C$ expression in the visceral muscles.

$\beta Tub85D$ ($\beta 2t$)

- references:** Kempfues, Raff, Kaufman, and Raff, 1979, Proc. Nat. Acad. Sci. USA 76: 3991-95.
 Kempfues, Raff, Raff, and Kaufman, 1980, Cell 21: 445-51.
 Sánchez, Natzle, Cleveland, Kirschner, and McCarthy, 1980, Cell 22: 845-54.
 Kempfues, Kaufman, Raff, and Raff, 1982, Cell 31: 655-70.
 Kempfues, Raff, and Kaufman, 1983, Genetics 105: 345-56.
 Bialojan, Falkenburg, and Renkawitz-Pohl, 1984, EMBO J. 3: 2543-48.
 Natzle and McCarthy, 1984, Dev. Biol. 104: 187-98.
 Fuller, 1986, Symp. Soc. Dev. Biol. 44: 19-41.
 Fuller, Caulton, Hutchens, Kaufman, and Raff, 1987, J. Cell Biol. 104: 385-94.
 Michiels, Falkenburg, Müller, Hinz, Otto, Bellmann, Glätzer, Brand, Bialojan, and Renkawitz-Pohl, 1987, Chromosoma 95: 387-95.
 Rudolph, Kimble, Hoyle, Subler, and Raff, 1987, Mol. Cell. Biol. 7: 2231-42.
 Fuller, Caulton, Hutchens, Kaufman, and Raff, 1988, J. Cell Biol. 107: 141-52.
 Gasch, Hinz, Leiss, and Renkawitz-Pohl, 1988, Mol. Gen. Genet. 211: 8-16.

phenotype: A structural gene for β -tubulin. It is transcribed into mRNA that is testis-specific; the mRNA is translated into a β -tubulin subunit that is involved in the formation of microtubules in the sperm tail axoneme, the cytoplasmic microtubules, and the meiotic spindle (Kempfues *et al.*, 1982; Fuller *et al.*, 1988). Only the microtubules of the mitotic spindle are not affected by a null mutation in $\beta Tub85D$. The first mutant discovered, $\beta Tub85D^D$ (Kempfues *et al.*, 1979, 1980, 1982, 1983), codes in males for an electrophoretic variant of β_2 -tubulin that causes disruption of microtubule function in all stages of spermatogenesis (beginning with meiosis) and shows abnormal spindle formation, abnormal chromosome movement, and no cytokinesis. This phenotype is expressed in males in both homo- and heterozygotes; mutants heterozygous over wild type contain both wild-type and mutant β_2 -tubulins; mutants over a deficiency for the locus contain only mutant β_2 -tubulin. Severity of effect on meiosis is as follows: $\beta Tub85D^D/\beta Tub85D^D > \beta Tub85D^D/+ > \beta Tub85D^D/+$, the first two genotypes being sterile and the last weakly fertile. All females are fertile. Chromosome replication and condensation appear normal. Recessive male-sterile mutations have also been induced, two of them in $\beta Tub85D^D$ chromosomes and the rest in $\beta Tub85D^+$ chromosomes. Testes of flies homozygous for the recessives $\beta Tub85D^3$,

$\beta Tub85D^4$, $\beta Tub85D^{Drv1}$, and $\beta Tub85D^{Drv2}$ synthesize, but later degrade, both α -tubulin and β -tubulin and show abnormalities in meiotic divisions, nuclear shaping, and formation of the flagellar axoneme (Kempfues *et al.*, 1982, 1983; Fuller, 1986). The most extreme recessive allele, $\beta Tub85D^n$, is male sterile but female fertile when homozygous (Fuller *et al.*, 1988); heterozygotes raised at 25° are male fertile, but those raised at 18° are male sterile. Recessive alleles $\beta Tub85D^6 - \beta Tub85D^{10}$ seem to accumulate normal amounts of β -tubulin but the β -tubulin subunits are defective. $\beta Tub85D^6$, $\beta Tub85D^7$, and $\beta Tub85D^8$ cause different defects in spermatogenesis. $\beta Tub85D^8$ is unable to form normal closed microtubules (Fuller *et al.*, 1987); in homozygous males it is defective in meiosis, nuclear shaping, and flagellar elongation. This allele is semi-dominant; heterozygous males with one normal and one abnormal tubulin subunit, form some functional sperm. Transheterozygotes between *ms(3)nc* (second site non-complementing) mutations and certain $\beta Tub85D$ alleles are male sterile even if wild-type copies of both genes are present (Fuller, 1986); a deletion of a *ms(3)nc* mutation in a heterozygote over $\beta Tub85D^n$, however, is fertile in males.

alleles:

allele	origin	synonym	ref ^α	comments
$\beta Tub85D^3$	EMS	$\beta 2t^3$; $T\beta 2^3$	1, 4, 5	male fertile in heterozygote, sterile in homozygote
$\beta Tub85D^4$	EMS	$\beta 2t^4$; $T\beta 2^4$	1, 4, 5	same phenotype as above
$\beta Tub85D^6$	EMS	$\beta 2t^6$; $T\beta 2^6$	1, 3	same phenotype as above
$\beta Tub85D^7$	EMS	$\beta 2t^7$; $T\beta 2^7$	1, 3	same phenotype as above
$\beta Tub85D^8$	EMS	$\beta 2t^8$; $T\beta 2^8$	1, 2, 3, 8	same phenotype as above
$\beta Tub85D^9$	EMS	$\beta 2t^9$; $T\beta 2^9$	9	same phenotype as above β
$\beta Tub85D^{10}$	EMS	$\beta 2t^{10}$; $T\beta 2^{10}$	9	same phenotype as above γ
$\beta Tub85D^{11}$	EMS	$\beta 2t^{11}$; $T\beta 2^{11}$	9	same phenotype as above γ
$\beta Tub85D^D$	EMS	<i>ms(3)KK^D</i> ; $\beta 2t^D$; $T\beta 2^D$	1, 4-7	male sterile in homo- and heterozygote
$\beta Tub85D^{Drv1}$	EMS	$\beta 2t^{D+R1}$; $T\beta 2^{D+R1}$	1, 4, 5	male fertile in heterozygote, sterile in homozygote
$\beta Tub85D^{Drv2}$	EMS	$\beta 2t^{D+R2}$; $T\beta 2^{D+R2}$	1, 4, 5	same phenotype as above
$\beta Tub85D^n$	EMS	$\beta 2t^n$; $T\beta 2^n$	1	null allele; no protein synthesized

^α 1 = Fuller, 1986, Symp. Soc. Dev. Biol. 44: 19-41; 2 = Fuller, Caulton, Hutchens, Kaufman, and Raff, 1987, J. Cell Biol. 104: 385-94; 3 = Fuller, Caulton, Hutchens, Kaufman, and Raff, 1988, J. Cell Biol. 107: 141-52; 4 = Kempfues, Kaufman, Raff, and Raff, 1982, Cell 31: 655-70; 5 = Kempfues, Raff, and Kaufman, 1983, Genetics 105: 345-56; 6 = Kempfues, Raff, Kaufman, and Raff, 1979, Proc. Nat. Acad. Sci. USA 76: 3991-95; 7 = Kempfues, Raff, Raff, and Kaufman, 1980, Cell 21: 445-51; 8 = Rudolph, Kimble, Hoyle, Subler, and Raff, 1987, Mol. Cell. Biol. 7: 2231-42; 9 = Regan and Fuller, 1988, Genes and Development 2: 82-92.
^β Fertile in transheterozygotes with *hay^{nc2}*.
^γ Sterile in transheterozygotes with *hay^{nc2}*.

cytology: Placed in 85D by *in situ* hybridization of cloned DNA (Sánchez *et al.*, 1980; Bialojan *et al.*, 1984; Natzle and McCarthy, 1984); placed in 85D7-11 by deficiency testing since in the 84F1-85E deletion of *In(3R)Msc^LAnp^{BR}* but not in *Df(3R)p46 = Df(3R)84D4-6;85D6* or *Df(3R)by10 = Df(3R)85D8-12;85E7-F1* (Kempfues *et al.*, 1983).

molecular biology: Gene cloned (Bialojan *et al.*, 1984; Natzle and McCarthy, 1984); clone contains one complete β -tubulin gene and the 3' end of another tubulin-like sequence; 2.0-2.2 kb transcript specifically expressed in spermatogenesis (Bialojan *et al.*, 1984; Raff, 1984).

Complete nucleotide sequences and predicted amino acid sequences of the protein-coding regions have been determined (Rudolph *et al.*, 1987; Gasch *et al.*, 1988). The proteins encoded by β Tub85D and β Tub60C show 87% overall identity in amino acid sequence; the β Tub85D tubulin contains 446 amino acids while the β Tub60C tubulin contains 454 amino acids. The β Tub85D gene has a single intron of 61 bases and the mRNA contains a 5' untranslated region of 175 bases. The first exon comprises this 5' region, the translation initiation codon ATG, and codons up to amino acid 131; the second exon includes the codons for amino acids from 132 to the stop codon. Transcription starts at an adenosine residue. The sequence for the mutant β Tub85D⁸ is identical to that of the wild-type in both intron and protein-coding regions, except that at nucleotide 862 there is a single nucleotide substitution resulting in a change from glu to lys at amino acid residue 288 (Rudolph *et al.*, 1987). *P*-element mediated germline transformation using the β Tub85D-*lacZ* fusion gene resulted in β Tub85D expression in the testis only (Michiels, Gasch, Kaltschmidt, and Renkawitz-Pohl, 1989, EMBO J. 8: 1559-65). 53 bp of upstream sequences are necessary for correct testis expression.

β Tub97EF (β 4t)

references: Natzle and McCarthy, 1984, Dev. Biol. 104: 187-98.

Raff, 1984, J. Cell Biol. 99: 1-10.

Gasch, Hinz, Leiss, and Renkawitz-Pohl, 1988, Mol. Gen. Genet. 211: 8-16.

phenotype: A structural gene for β -tubulin. It is transcribed into mRNA that occurs ubiquitously throughout development. β Tub97EF is expressed coordinately with β Tub56D, but the β Tub97EF transcripts are much less prevalent. They occur at highest concentration during the first half of embryogenesis and the first and second larval instars (Natzle and McCarthy, 1984). No protein variant has been described (Gasch *et al.*, 1988).

cytology: Located in 97E-F by *in situ* hybridization (Natzle and McCarthy, 1984).

molecular biology: Gene cloned (Natzle and McCarthy, 1984); 1.8 kb transcript obtained (Raff, 1984).

Tubby: see *Tb*

tube: see *tub*

Tubulin: see *Tub*

tud: tudor (*T. Schüpbach*)

location: 2- {97}.

origin: Induced by ethyl methanesulfonate.

discoverer: Wieschaus and Nüsslein-Volhard.

references: Boswell and Mahowald, 1985, Cell 43: 97-104.

Degelmann, Hardy, Perrimon, and Mahowald, 1986, Dev. Biol. 115: 479-89.

Schüpbach and Wieschaus, 1986, Roux's Arch. Dev. Biol. 195: 302-17.

Nüsslein-Volhard, Frohnhöfer, and Lehmann, 1987, Science 238: 1675-81.

O'Donnell, Boswell, Reynolds, and Mackay, 1989, Genetics 121: 273-80.

Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

phenotype: Maternal-effect mutant; embryos from homozygous mothers exhibit a so-called "grandchildless-knirps"

phenotype: all eggs lack polar granules and no pole cells are formed; most of the embryos show variable deletions of abdominal segments, whereby segment A4 is deleted most frequently; larger deletions may include segments A2 through A7; in extreme cases anterior parts of segment A1 become fused to posterior part of segment A8, but telson elements are always present and relatively normal. Around 30% of all embryos survive and grow into sterile adults. Analysis of germline clones indicates that the mutation is germline autonomous (Schüpbach and Wieschaus, 1986, Dev. Biol. 113: 443-48).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments ^β
<i>tud</i> ¹	EMS	Wieschaus Nüsslein-Volhard	<i>tud</i> ^{WC}	1, 2, 3	
<i>tud</i> ²	EMS	Boswell		1	
<i>tud</i> ³	EMS	Boswell		1	
<i>tud</i> ⁴	EMS	Boswell		1	
<i>tud</i> ⁵	EMS	Boswell		1	
<i>tud</i> ⁶	EMS		<i>tud</i> ^{B46}	2, 3	<i>ln(2R)B46 = ln(2R)53D2-E1;57B6-D11</i>

^α 1 = Boswell and Mahowald, 1985, Cell 43: 97-104; 2 = Schüpbach and Wieschaus, 1986, Roux's Arch. Dev. Biol. 195: 302-17; 3 = Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

^β Eight alleles, *l(2)57Ce1-l(2)57Ce8*, complement both lethal and grandchildless phenotypes of *tud* (O'Donnell *et al.*, 1989).

cytology: Located in 57C7-9 by O'Donnell *et al.*, 1989, since not complemented by *Df(2R)PF1 = Df(2R)57C5;57D1* but complemented by deficiencies with distal break proximal to 57C6-7.

molecular biology: Gene has been cloned and found to encode a 8.0 kb transcript which is expressed most strongly in early embryos and in pupae (Golumbeski, O'Rourke, and Boswell, 1989, New Orleans Drosophila meeting).

tuf: tufted

location: 2-59 (Ashburner).

references: Sturtevant, 1948, DIS 22: 56.

Nüsslein-Volhard and Wieschaus, 1980, Nature (London) 287: 795-801.

Nüsslein-Volhard, Wieschaus, and Kluding, 1983, DIS 59: 158-60.

1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

Carroll and Scott, 1986, Cell 45: 113-26.

Nakano, Guerrero, Hidalgo, Taylor, Whittle, and Ingham, 1989, Nature (London) 341: 508-13.

synonym: *ptc*, *patched*.

phenotype: The *tuf* gene is involved in patterning within segments in *Drosophila*. The viable first-identified mutant has a small tuft of hairs between eyes and antennae and shows basal twinning of the anterior halves of wings; it overlaps wild type. *tuf*^{T(2;3)dp} has an extreme form of this mutant phenotype. Other mutants are embryonic lethals of the segment-polarity type. There is a mirror-image duplication of segment boundaries and adjacent cuticle of all segments with deletion of the remainder of the segment. Defect visible during extended-germ-band stage (6 hr) (Nüsslein-Volhard and Wieschaus, 1980). Normal number of denticle bands; duplicated region of embryo includes some naked cuticle anterior to denticle bands. Pattern of neurons underlying affected epidermal region is altered (Patel, Schafer, Goodman, and Holmgren, 1989, Genes Dev. 3: 890-

904). This mutant has no effect on the spatial expression of the "pair-rule" mutant *ftz* (Carroll and Scott, 1986). *tuf* embryos cultured in vivo produced derivatives of the eye-antennal and thoracic discs, the latter being abnormal in morphology and in *en* expression (Simcox, Roberts, Hersperger, Gribbon, Shearn, and Whittle, 1989, Development 107: 715-22).

alleles: One viable allele and numerous lethal alleles reported.

allele	origin	synonym	ref ^α	comments
<i>tuf</i> ¹	spont		3	viable
<i>tuf</i> ^{2β}	EMS		5	homozygous lethal; extreme allele
<i>tuf</i> ³	EMS	<i>ptc</i> ^{6C}	4	homozygous lethal
<i>tuf</i> ⁴	EMS	<i>ptc</i> ^{6P}	4	homozygous lethal
<i>tuf</i> ⁵	EMS	<i>ptc</i> ^{7M}	4	homozygous lethal
<i>tuf</i> ⁶	EMS	<i>ptc</i> ^{8H}	4	homozygous lethal
<i>tuf</i> ⁷	EMS	<i>ptc</i> ^{9B}	4	homozygous lethal
<i>tuf</i> ⁸	EMS	<i>ptc</i> ^{1F}	4	homozygous lethal; temperature-sensitive
<i>tuf</i> ⁹	EMS	<i>ptc</i> ^{1N}	1,4	homozygous lethal
<i>tuf</i> ¹⁰	EMS	<i>ptc</i> ^{11B}	4	homozygous lethal
<i>tuf</i> ¹¹	EMS	<i>ptc</i> ^{11C2}	4	homozygous lethal
<i>tuf</i> ¹²	EMS	<i>ptc</i> ^{11C8}	4	homozygous lethal
<i>tuf</i> ¹³	EMS	<i>ptc</i> ^{11E}	4	homozygous lethal
<i>tuf</i> ¹⁴	EMS	<i>ptc</i> ^{11R}	4	homozygous lethal; weak
<i>tuf</i> ¹⁵	EMS	<i>ptc</i> ^{11U}	4	homozygous lethal; weak
<i>tuf</i> ¹⁶	EMS	<i>ptc</i> ^{11W}	4	homozygous lethal
<i>tuf</i> ¹⁷	EMS	<i>ptc</i> ^{11X}	4	homozygous lethal
<i>tuf</i> ¹⁸	HD	<i>ptc</i> ^{P78}	2	homozygous lethal
<i>tuf</i> ¹⁹	HD		2	homozygous lethal
<i>tuf</i> ²⁰	HD		2	homozygous lethal
<i>tuf</i> ²¹	HD		2	homozygous lethal
<i>tuf</i> ²²	HD		2	homozygous lethal
<i>tuf</i> ²³	HD		2	homozygous lethal
<i>tuf</i> ²⁴	HD		2	homozygous lethal
<i>tuf</i> ²⁵	HD		2	homozygous lethal
<i>tuf</i> ²⁶	X ray	<i>ptc</i> ^{RX67}	1	homozygous lethal

^α 1 = Hooper and Scott, 1989, Cell 59: 751-65; 2 = Nakano, Hidalgo, Taylor, Whittle, and Ingham, 1989, Nature (London) 341: 508-13; 3 = Sturtevant, 1948, DIS 22: 56; 4 = Tearle and Nüsslein-Vollhard, 1987, DIS 66: 209-26; 5 = Whittle, 1980, DIS 55: 211.

^β Wings of *tuf*¹/*tuf*² grossly foreshortened and shaped like a ping-pong paddle; these flies also show duplications and triplications of anterior wing structures, lack costal bristles, and have more head abnormalities than *tuf*¹ homozygotes.

cytology: *tuf* (= *ptc*) placed in 44D3-4 by *in situ* hybridization to P-element-free polytene chromosomes (Nakano *et al.*, 1989). Located in 44B-E based on uncoverage by the deficiency segregant of *Tp(2;3)eve*^{1.18} but not by the deficiency segregant of *Tp(2;3)eve*^{2.28} (Nüsslein-Vollard *et al.*, 1984; Tearle and Nüsslein-Vollard, 1987, DIS 66: 209-26); located in 44D1-2 to 44E1-4 since included between the centromere-proximal breaks of *Df(2R)P14TE* (a synthetic deficiency) and *Df(2R)44CE* (Hooper and Scott, 1989).

molecular biology: Gene cloned; transcription unit of about 17-30 kb encodes a 5.8 to 7 kb transcript (Hooper and Scott, 1989, Cell 59: 751-65; Nakano *et al.*, 1989). At blastoderm, *tuf* transcribed uniformly except in pole cells and A-D region; later expressed as 14 and then 28 stripes. Expression in larvae occurs in the imaginal disks. Nucleotide sequence of *tuf* and predicted amino acid sequences obtained (Nakano *et al.*, 1989). There is a single open reading frame of 4152 bases (Hooper and Scott, 1989). Predicted protein is large (1286 amino acids) with at least seven putative transmembrane α helices.

Tufted: see *Tft*

tufts: see *tft*

tuh1: tumorous head in chromosome 1

location: 1-65.3 (Woolf).

origin: Spontaneous.

discoverer: Griffen.

references: Gardner, 1949, DIS 23: 57.

Gardner and Woolf, 1949, Genetics 34: 573-85.

Newby, 1949, J. Morphol. 85: 177-95.

Newby and Thelander, 1950, DIS 24: 89-90.

Gardner, 1959, Genetics 44: 471-81.

Woolf, 1966, Genetics 53: 295-302.

1968, Genetics 60: 111-21.

Gardner, 1970, Adv. Genet. 15: 115-46.

Postlethwait, Bryant, and Schubiger, 1972, Dev. Biol. 29: 337-42.

Pyati, 1976, Mol. Gen. Genet. 146: 189-90.

Bournias-Vardiabasis and Bownes, 1978, J. Embryol. Exp. Morphol. 44: 227-41.

Bournias-Vardiabasis and Bownes, 1979, Wilhelm Roux's Arch. Dev. Biol. 186: 87-90.

Kuhn, Züst, and Illmensee, 1979, Mol. Gen. Genet. 168: 117-24.

Woolf and Passage, 1980, Mol. Gen. Genet. 178: 423-27.

Kuhn, Woods, and Andrew, 1981, Genetics 99: 99-107.

Kuhn and Packert, 1988a, Dev. Biol. 125: 8-18.

1988b, Genetics 118: 103-07.

phenotype: Maternal effect gene with no phenotypic expression of its own; presence of gene indicated by difference in results obtained from reciprocal crosses between phenotypically tumorous head and inbred wild-type flies, the mutant phenotype only appearing in the offspring when the mother comes from the *tuh* stock (Gardner, 1959). There seems to be an interaction involving maternal effect substances between *tuh1* (or *tuh1*⁺) and *iab9*^{tuh3}, an allele of the most distal gene in the *BXC*; the homoeotic effects of homo- or heterozygous *iab9*^{tuh3} flies are strongly enhanced by the *tuh1* allele. One type of abnormality in tumorous head mutants involves homoeotic changes, deficiencies, and duplications in the eye, antenna, or rostralhant regions that transform parts of these structures into tergite-like, leg-like, or genital-like growths in the adult (Postlethwait *et al.*, 1972). In the mutant larvae, patches of aldehyde oxidase positive tissue can be demonstrated within the aldehyde oxidase negative eye discs, where, presumably, transformed tissue will occur (Kuhn *et al.*, 1979). Penetrance of these head abnormalities is increased by high temperature during oogenesis and early embryogenesis (Gardner, 1970; Bournias-Vardiabasis and Bownes, 1979) and also by the presence of *E(tuh1)* an enhancer of the mutant phenotype on 3R (Kuhn and Dorgan, 1974, Genetics 77: s37; Kuhn and Packert, 1988a). Modifiers on the first, second, and third chromosomes in certain lines likewise increase the maternal effect of *tuh1* (Gardner, 1970; Woolf and Passage, 1980). The other type of abnormality occurring in tumorous head stocks involves genital disc defects that result in missing or undeveloped testes and associated organs, both in males and in transformed females (Gardner and Woolf, 1949; Woolf, 1966, 1968; Kuhn *et al.*, 1981). Viability of stocks showing head abnormalities is about 70%; lethal-

ity may occur in the egg, larval, or pupal periods (Bournias-Vardiabasis and Bownes, 1978).

alleles:

allele	synonym	ref ^α	phenotype with <i>iab9^{tuh3} β</i>
tuh1	<i>tuh1^h</i> , <i>tu-1</i>	1, 2 3, 4	homeotic leg, tergite, or genital tissue in eye-antenna region (penetrance up to 90%)
tuh1⁺	<i>tuh1^g</i>	2, 3 5, 6	defects in male genital disc or complete absence of disc (penetrance up to 60%)

^α 1 = Gardner and Woolf, 1949, *Genetics* 34: 573-85; 2 = Kuhn and Packert, 1988a, *Dev. Biol.* 125: 8-18; 3 = Kuhn and Packert, 1988b, *Genetics* 118: 103-07; 4 = Kuhn, Züst, and Illmensee, 1979, *Mol. Gen. Genet.* 168: 117-24; 5 = Woolf, 1966, *Genetics* 53: 295-302; 6 = Woolf, 1968, *Genetics* 60: 111-21.

^β Effect with *tuh1* semidominant; effect with *tuh1⁺* recessive (Kuhn and Packert, 1988a and 1988b).

cytology: Located in 20A1-2 since uncovered by *Df(1)JC4* = *Df(1)20A1-2;20E-F* and *Df(1)mal6* = *Df(1)19C3;20A2-3*, but not by *Df(1)Q539* = *Df(1)19E6;19F6-20A1* (Pyati, 1976).

other information: A third chromosome EMS-induced mutant *II27*, when homozygous or when heterozygous over *iab9^{tuh3}*, produces head abnormalities involving genital and abdominal transformations under the influence of the *tuh1* maternal effect (Bownes, Roberts, Demster, and Bournias-Vardiabasis, 1981, *Mol. Gen. Genet.* 183: 158-62; Kuhn and Packert, 1988a); may be an allele of *iab9^{tuh3}*.

tuh3: see *iab9^{tuh3}*

Tul: Turneduplike

location: 1-50 (between *g* and *f*).

origin: Spontaneous.

references: Muller, 1965, *DIS* 40: 35.

phenotype: Like *Tu*. Wing tips of heterozygote turned up slightly but definitely not twisted. Male and homozygous female more extreme with wrinkled wings sometimes held somewhat apart; viable and fertile. RK2.

Tum: Tumorous

location: 1-35.8.

origin: Induced by ethyl methanesulfonate.

references: Corwin and Hanratty, 1976, *Mol. Gen. Genet.* 144: 345-47.

Hanratty and Ryerse, 1981, *Dev. Biol.* 83: 238-49.

phenotype: Dominant tumorous gene that is a temperature-sensitive lethal at 29° in hemizygous males and homozygous females; about two-thirds of the hemizygous males survive at 18° and one-quarter of these have melanotic tumors. Males raised at 26° and heterozygous females raised at 29° survive to adulthood, but show melanotic masses in the abdominal cavity or small black specks in the legs, wings, or thorax. Mutant larvae kept at 29° show enlargement of the lymph glands in the late second- or early third-instar larvae, but no melanotic masses. By mid third-instar, the lymph glands are large and diffuse and the gastric caeca have become encapsulated and melanized. By late third-instar, the larvae have melanotic masses in the body cavity, lack lymph glands, and have reduced, encapsulated and melanized gastric caeca as well as encapsulated and melanized muscles and fat bodies. These mutants do not survive beyond the late third-instar or the early pupal stage. When lymph glands from *Tum* larvae are injected into adult female hosts, transplantable neoplasms are produced. Melanization, at

first associated with the leg joints and later with the head, thorax, and abdomen, takes place; also abdominal bloating. The lymph glands become melanotic and the abdomen is filled with encapsulated masses before the premature death of the injected individuals. Injection of *Tum* tissue other than lymph glands fails to produce these effects. The melanotic neoplasms can be transplanted into a succession of hosts in which they produce the same abnormalities. The neoplastic cells resemble hemocytes; some cell lines are melanotic and others are unpigmented, but in both types, the tissue, when transplanted, grows rapidly in the hosts and kills them.

other information: Shown to be allelic to *hop* too late to be included in the *hop* entry (Dearoff).

tumor: see *tu*

tumor head 63: see *ey^{tu}*

tumorous: see *tms*

Tumorous: see *Tum*

tumorous head in chromosome 1: see *tuh1*

tup: tailup

location: 2-54.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 267-82.

Tearle and Nüsslein-Volhard, 1987, *DIS* 66: 209-69.

phenotype: Embryonic lethal. Head broad. Germband shortening apparently ceases early, resulting in the posterior-most three segments remaining on dorsal side of the embryo.

alleles: *tup¹* and *tup²*, isolated as *tup^{III B}* and *tup^{III e}*.

tur: turnip (J.C. Hall)

location: 1- (proximal to *f*, distal to *car*).

origin: Induced by ethyl methanesulfonate.

references: Aceves-Piña and Quinn, 1979, *Science* 206: 93-96.

Quinn, Sziber, and Booker, 1979, *Nature* 277: 212-114.

Booker and Quinn, 1981, *Proc. Nat. Acad. Sci. USA* 78: 3940-44.

Duerr and Quinn, 1982, *Proc. Nat. Acad. Sci. USA* 79: 3645-50.

Tully and Quinn, 1985, *J. Comp. Physiol.* 157: 253-77.

Tully, 1987, *Trends Neurosci.* 10: 330-35.

1989, *Adaptation, Learning, and Affect* (Madden, Matthisse, and Barchas, eds.). Raven, New York, pp. 1989.

phenotype: Homozygotes or hemizygotes are blocked or impaired in learning, with respect to certain of the conditioning tests used on groups of flies or larvae or on individual adults, e.g. tests involving olfactory "shock-avoidance" learning (Acevas-Piña and Quinn, 1979; Quinn *et al.*, 1979), leg-shock/leg-lift learning (Booker and Quinn, 1981); "courtship conditioning" (Gailey, Jackson, and Siegal, 1982, *Genetics* 102: 771-82; 1984, *Genetics* 106: 613-23), and habituation to sugar stimuli when applied to tarsus (Duerr and Quinn, 1982). When the original shock-odor testing system is modified by redesigning the choice chamber, homozygous and hemizygous mutants show about 60% of the control wild-type values when tested just post-training (Tully and Quinn, 1985; Tully, 1987), but the mutant shows very rapid

short-term and slower long-term memory losses. *tur*+/ heterozygous females show seemingly normal levels of shock-odor conditioned behavior when tested immediately after training, but very rapid memory losses (Quinn *et al.*, 1979). Homozygous *tur* flies are said to have a deficiency in (and possibly absence of) protein kinase C (PKC) (Smith, Choi, Tully, and Quinn, 1986, Soc. Neurosci. Abstr. 12: 399), and *tur* flies are almost totally deficient in phosphorylation of a 76 kd head membrane protein which is a major substrate for PKC (Choi, Smith, Marler, and Quinn, 1989), but the lowered PKC levels and abnormal learning do not map to the same cytogenetic location (see **cytology**).

cytology: *Df(1)JA27 = Df(1)18A5;18D1-2*, which uncovers the PKC deficiency and the non-phosphorylation of head membrane protein phenotypes, did not uncover any deficit in classical conditioning in one series of tests (Tully and Quinn, 1985; Tully, 1989). Another series, however, revealed 15% reduction in learning scores for classical conditioning tests and 70% reduction in scores for shock-avoidance tests in *tur/Df(1)JA27* females (Choi *et al.*, submitted). Other proximal X deletions [*Df(1)N19*, *Df(1)mal8*, *Df(1)16-3-22*, and *Df(1)DCB1-35B*] have not uncovered any classical conditioning deficits.

Turned-up wing: see *Tu*

Turneduplike: see *Tul*

turnip: see *tur*

tw: twisted

location: 1-0.4 (*tw*); 1-0.1 (*tw*³).

references: Demerec, Kaufmann, Fano, Sutton, and Samson, 1942, Year Book - Carnegie Inst. Washington 41: 191.

Davis, 1975, Genetics 80: s25.

1980, DIS 55: 29-31, 31-33.

Kramers, Schalet, Paradi, and Huiser-Hoogteyling, 1983, Mutat. Res. 107: 187-201.

Lefevre and Watkins, 1986, Genetics 113: 869-95.

phenotype: Abdomens of males and females twisted about 30° clockwise, as viewed from the posterior. Body tends to be dwarfed. Viability reduced; hatching delayed. Males usually fertile. 1% nondisjunction for homologs in males (Davis, 1975, 1980).

alleles: 13 alleles are listed in the following table. Phenotypes of *tw*² and *tw*³ are given in more detail in subsequent sections.

allele	origin	discoverer	synonym	ref ^α	viability
<i>tw</i> ¹	X ray	Demerec		1, 3	60%
<i>tw</i> ²	spont	Mohr			50%
<i>tw</i> ³	EMS	Davis	<i>tricky dicky</i>	2, 3	
<i>tw</i> ⁴	EMS	Lefevre	<i>l(1)DAM7</i>		low
<i>tw</i> ⁵	EMS	Lefevre	<i>l(1)VAM201</i>		low
<i>tw</i> ⁶	ENU	Voelker	<i>l(1)A23</i>		low
<i>tw</i> ⁷	ENU	Voelker	<i>l(1)B9</i>		low
<i>tw</i> ⁸	ENU	Voelker	<i>l(1)B19</i>		low
<i>tw</i> ⁹	ENU	Voelker	<i>l(1)B32</i>		low
<i>tw</i> ¹⁰	ENU	Voelker	<i>l(1)B39</i>		low
<i>tw</i> ¹¹	ENU	Voelker	<i>l(1)B44</i>		low
<i>tw</i> ¹²	ENU	Voelker	<i>l(1)B47</i>		low
<i>tw</i> ¹³	EMS	Voelker	<i>l(1)C6</i>		low

^α 1 = CP627; 2 = Davis, 1975, Genetics 80: s25; 3 = Davis, 1980, DIS 55: 29-31, 31-33.

cytology: Located at 1C5-1D4.

tw²

phenotype: Abdomens of males and females twisted 30°-60° clockwise, as viewed from the posterior; more extreme than *tw*. Male genitalia often twisted counter-clockwise. Viability reduced. Males usually fertile. *tw*²/*tw* flies resemble *tw*²/*tw*².

tw³

phenotype: Abdomens of males and females twisted up to 90°, as viewed from the posterior. Male genitalia and anal plate often twisted clockwise or counter-clockwise (misalignment up to 180°). Males and females often sterile, although male genitalia appear normal and sperm are motile. 1% nondisjunction for homologues in males (Davis, 1975, 1980).

***twg: twisted genitals**

location: 1-48.1.

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 93-94.

phenotype: External genitalia abnormally positioned on extreme tip of abdomen. Tergites often notched at mid-dorsal line. Eyes large, abnormally shaped, and slightly rough. Wings vary from almost normal to small, deformed structures with very abnormal venation. Bristles frequently waved or bent. Male viability and fertility subnormal. RK2.

twi: twist

location: 2-100.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1983, DIS 59: 158-60.

Simpson, 1983, Genetics 105: 615-32.

Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

Carroll, Winslow, Twombly, and Scott, 1987, Development 99: 327-32.

Thisse, Stoetzel, El Messal, and Perrin-Schmitt, 1987, Genes and Development 1: 709-15.

Thisse, Stoetzel, Gorostiza-Thisse, and Perrin-Schmidt, 1988, EMBO J. 7: 2175-83.

phenotype: The wild-type allele of *twi* is involved in the establishment of germ layers. Mutants are embryonic lethals (zygotic), partially dorsalized, and without mesodermal differentiation. A normal blastoderm is formed; at gastrulation, no ventral furrow is visible, but the endoderm invaginates, a cephalic furrow is formed, and the germband elongated. The embryo is twisted or coiled in the egg case, often with posterior side up. There are few mesodermally derived internal tissues. Some embryos fail to make a properly differentiated cuticle, although 40-100% make normal ectodermal derivatives (Simpson, 1983). There is no maternal effect in germline chimeras. *twi*+/ heterozygous embryos have delayed ventral furrow formation. The TSP of the *twi* gene is around gastrulation (Thisse *et al.*, 1987).

alleles:

allele	synonym	comments
<i>twi</i> ¹	<i>twi</i> ^{1D}	
<i>twi</i> ²	<i>twi</i> ^{11E}	
<i>twi</i> ³	<i>twi</i> ^{11H}	
<i>twi</i> ⁴	<i>twi</i> ^{D5}	

allele	synonym	comments
<i>twi</i> ⁵	<i>twi</i> ^{OS}	
<i>twi</i> ⁶	<i>twi</i> ^{X10}	
<i>twi</i> ⁷	<i>twi</i> ^{ey63}	
<i>twi</i> ⁸	<i>twi</i> ^{IG}	temperature-sensitive

cytology: Placed in 59C3-D2; uncovered by *Df(2R)twi* = *Df(2R)59C3-4;59D1-2*.

molecular biology: Region containing *twi* cloned (Thisse, El Messal and Perrin-Schmidt, 1987, Nucleic Acids Research 15: 3439-53) and the nucleotide sequence obtained (Thisse *et al.*, 1988). The sequence is a simple transcription unit, with an unique 5' end and a 120 bp intron near its 3' end. The mature twist mRNA is 1878 bp long excluding the poly(A) tail, and contains one large open reading frame that generates a protein of 490 amino acids that is present at the anterior and posterior poles and in the midventral region at the cellular blastoderm stage. The *twi* protein is localized in the nucleus (Thisse *et al.*, 1988). Translation apparently occurs just after the transcription process.

other information: Mutations in *dl*, *pll*, *ea*, or *T1* abolish the expression of *twi*. At least one dose of *dl*⁺ in females is necessary for transcription of *twi* (Thisse *et al.*, 1987). *twi* shows extensive identity to a pair of *myc*-related polypeptides whose dimerized products bind to a sequence in the immunoglobulin kappa chain enhancer; the identical regions have the potential to form two amphipathic helices separated by an intervening loop (Murre, McCaw, and Baltimore, 1989, Cell 56: 777-83).

twirl: see *twi*

twirled tips: see *twi*

twist: see *twi*

twisted: see *tw*

twisted bristles roughened eye: see *twr*

twisted gastrulation: see *tsg*

twisted genitals: see *twg*

twl: *twirl*

location: 2-63.5.

origin: Ultraviolet induced.

discoverer: Meyer, 54d.

references: 1955, DIS 29: 74-75.

phenotype: Wings strongly curled. Good viability; easy to classify. RK2.

other information: Possibly an allele of *upw* (2-62).

***Two-b: Two bristles**

location: 3-58.3.

origin: Spontaneous.

discoverer: Bridges, 16b22.

references: Bridges and Morgan, 1923, Carnegie Inst. Wash. Publ. No. 327: 155.

phenotype: Two postvertical bristles always and two anterior dorsocentrals usually absent. Heterozygote viability excellent. Homozygous lethal. RK1.

two-faced: see *tfd*

twr: twisted bristles roughened eye

location: 3-{47}.

references: Merrill, Diederich, Turner, and Kaufman, 1989, Dev. Biol. 135: 376-91.

phenotype: Most alleles lethal; few escapers display twisted bristles and rough eyes; survivors female sterile.

alleles:

allele	origin	discoverer	synonym
<i>twr</i> ¹	EMS	Wakimoto	<i>Efw5</i>
<i>twr</i> ²	EMS	Wakimoto	<i>Efw9</i>
<i>twr</i> ³	EMS	Wakimoto	<i>Efw27</i>
<i>twr</i> ⁴	EMS	Kaufman	<i>k1</i>
<i>twr</i> ⁵	EMS	Kaufman	<i>k4</i>
<i>twr</i> ⁶	EMS	Fornili	<i>f17</i>
<i>twr</i> ⁷	EMS	Fornili	<i>f37</i>
<i>twr</i> ⁸	EMS	Fornili	<i>f66</i>
<i>twr</i> ⁹	EMS	Fornili	<i>f68</i>
<i>twr</i> ¹⁰	EMS	Fornili	<i>f70</i>
<i>twr</i> ¹¹	EMS	Lambert	<i>c33</i>

cytology: Placed in 84A1-6 based on its inclusion in *Df(3R)Scr* = *Df(3R)84A1-2;84B1-2* but not in *Dr(3R)Antp17* = *Df(3R)84A6;84D13-14*.

***twi: twirled tips**

location: 1-37.1.

origin: Induced by 1:4-dimethanesulfonylbut-2-yne (CB. 2058).

discoverer: Fahmy, 1951.

references: 1959, DIS 33: 94.

phenotype: Wings completely or partially unexpanded; tips frequently twisted. Male inviable, dies shortly after eclosion, and does not breed. RK3.

tx: taxi (J.C. Hall)

location: 3-91.

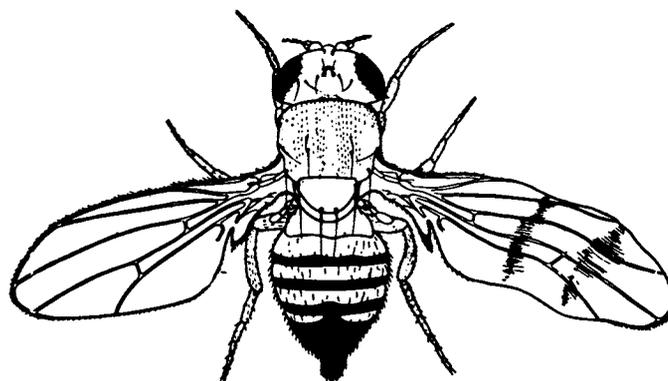
origin: Spontaneous.

phenotype: Wings held out at about 75° from body axis, often arched or wavy, somewhat narrow and dusky. Unable to fly because of shape and posture of wings. RK2.

alleles:

allele	discoverer	ref ^α
<i>tx</i> ¹	Collins, 24j30	1, 2
<i>tx</i> ^{52j}	Tsukamoto, 52j	3

^α 1 = Chiarodo, Reing, and Saranchak, 1971, J. Exp. Zool. 178: 325-30; 2 = Collins, 1928, Am. Nat. 62: 127-36; 3 = Tsukamoto, 1956, DIS 30: 79.



tx: taxi

From Collins, 1928, Am. Naturalist 62: 127-36.

ty: tiny

location: 1-44.5.

discoverer: Bridges, 25k1.

references: King and Koch, 1963, Quart. J. Microscop. Sci. 104: 297-320.

King, 1970, Ovarian Development in *Drosophila melanogaster*, Acad. Press, New York.

King and Mohler, 1975, Handbook of Genetics (R.C. King, ed.), Plenum Press, New York and London, Vol. 3, pp. 757-91.

Perrimon and Gans, 1983, Dev. Biol. 100: 365-73.

phenotype: Bristles small. Body small. Eclosion delayed. Viability excellent. Female sterile; eggs rarely found and only in a few ovarioles (Perrimon and Gans, 1983). Yolk formation in oocytes inhibited [King and Burnett, 1957, Growth 21: 263-80 (fig.)]. Follicular cells form abnormal derivatives of endoplasmic reticulum and migrate abnormally or form excess of normal endoplasmic reticulum derivative [King and Vanoucek, 1960, Growth 24: 333-38; Falk and King, 1964, Growth 28: 291-324 (fig.)]. *ty* ovaries in *ty*⁺ host develop autonomously (King and Bodenstein, 1965, Z. Naturforsch. 20B: 292-97). RK2. The heteroallelic combination *ty/ty*¹⁵ exhibits the visible phenotypes described for *ty* homozygotes (Schalet, 1986, Mutat. Res. 163: 115-44), and, in addition, may show a reduction in thoracic hairs and abnormally-shaped rough eyes; viability severely reduced in some cultures.

alleles: A, spontaneous lethal, *l(1)4-103*, was found to be allelic to *ty* (Schalet, 1986).

cytology: Located in 12A6-12D3.

***tyb2: tiny bristle 2**

location: 1-19.5.

origin: Spontaneous.

discoverer: Neel, 41i9.

references: 1942, DIS 16: 52.

phenotype: Bristles small and thin. Viability and fertility good. RK1.

tyl: tinylike

location: 1-36.

origin: X ray induced in *In(1)dl-49*.

discoverer: Oliver, 28k4.

references: 1935, DIS 3: 28.

1942, DIS 16: 53.

phenotype: Bristles short, fine, and stubblelike. Eclosion delayed. Both sexes viable and fertile. RK2A.

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
<i>tyl</i> ¹	X ray	Oliver		1, 2	
<i>tyl</i> ²	EMS	Voelker	<i>l(1)A8</i>	3	
<i>tyl</i> ³	ENU	Voelker	<i>l(1)M14</i>	3	leaky

^α 1 = Oliver, 1935, DIS 3: 28; 2 = Oliver, 1942, DIS 16: 53; 3 = Voelker, Wisely, Huang, and Gyurkovics, 1985, Mol. Gen. Genet. 201: 437-45.

cytology: Located in 10C3-5 since included in the transposed region of *Tp(1;1)^{N48} = Tp(1;1)9F;10C3-5;20*, but not in *Df(1)GA112 = Df(1)10A11-B1;10C2*.

tyr1: tyrosine 1 (J.C. Hall)

location: 2-54.5 (Huntly, 1978, Ph.D. Thesis, University of Virginia); inseparable so far from *pr*.

origin: Spontaneous.

discoverer: H.W. and H.S. Lewis.

synonym: α : *alpha*; *tyrosinase-1*.

references: 1960, DIS 34: 51.

H.W. and H.S. Lewis, 1961, Proc. Nat. Acad. Sci. USA 47: 78-86.

H.W. Lewis, 1962, Biol. Bull. 123: 464.

H.W. and H.S. Lewis, 1963, Ann. N.Y. Acad. Sci. 100: 827-39.

Rizki, Rizki, and Bellotti, 1985, Mol. Gen. Genet. 201: 7-13.

Wright, 1987a, Results and Problems in Cell Differentiation (Hennig, ed.), Springer-Verlag, Berlin, Heidelberg, Vol. 14, pp. 95-119.

1987b, Adv. Genet. 24: 127-222.

Pentz, Black, and Wright, 1990, Biochem. Genet. 28: 151-71.

phenotype: *tyr1* originally thought to be a structural gene for phenol oxidase (Lewis and Lewis, 1963); the mutant reported to be heat stable relative to wild type, but this differential heat stability in the original mutant strain has not been confirmed (Wright, 1987b). Rizki *et al.* suggest that *tyr1* acts as a regulator of phenol oxidase activity. Mutant homozygotes have about 10% of the phenol oxidase activity of most wild-type strains. 50 h mutant pupae lack activity for the proenzyme PHOX and have detectable but reduced amounts of the diphenol oxidase components A1, A2, and A3 as compared to Samarkand wild-type pupae (Wright, 1987b; Warner, Grell, and Jacobson, 1974, Biochem. Genet. 11: 359-65); result confirmed by Pentz, Black, and Wright, 1987, who also showed that activator preparations from *tyr1* were as efficient as those from wild type (Wright, 1987b). Homozygous *tyr1* adults slightly underpigmented; less so than *qs* which has more phenol oxidase activity. Males (normal or *tyr1*) have three times higher dopamine levels than females (normal or mutant); the mutants (males or females) have 70% normal dopamine levels [Burnell and Daly, 1982, Advances in Genetics, Development, and Evolution of *Drosophila* (Lakovaara, ed.), Plenum Press, New York, pp. 361-70)]. Homozygous viable and fertile. Hemolymph of *tyr1* mutant larvae will turn black but hemolymph from mutant adults will not.

alleles: Only one mutant allele identified so far.

cytology: Placed in salivary chromosome region 38B3 to 38B6 based on lack of recombinant with *pr* (Pentz *et al.*, 1990); uncovered by *Df(2L)TW150 = Df(2L)37F5-38A1;38B2-C1* which also uncovers *pr* (Huntley, 1978).

Tyr2: Tyrosine 2 (J.C. Hall)

location: 2-66.3 (between *cn* and *L*; Homyk and Pye, 1989).

origin: Spontaneous in *In(2L)Cy* and *In(2R)Cy* or induced by ethyl methanesulfonate.

discoverer: H.W. and H.S. Lewis.

synonym: β : *beta*; *Tyrosinase-2*.

references: H.W. Lewis, 1962, Biol. Bull. 123: 464.

H.W. and H.S. Lewis, 1963, Ann. N.Y. Acad. Sci. 100: 827-39.

Homyk and Pye, 1989, J. Neurogenet. 5: 37-58.

phenotype: *Tyr2/+* heterozygotes show about 50% of the

diphenol oxidase activity of wild-type strains; modifiers involved in this dominant effect. *Tyr2*^{P208}, isolated as a recessive light-on/light-off transient-minus mutant by Pak, also affects electroretinograms when in heteroallelic combination with the original *Tyr2* (Homyk and Pye, 1988). *Tyr2* homozygotes also show this ERG defect.

alleles: Two alleles, *Tyr2* and *Tyr2*^{P208} (the latter not characterized biochemically).

Tyr3: Tyrosine 3

location: 3- (right arm)

synonym: *Tyrosinase-3*.

references: H.W. Lewis, 1962, Biol. Bull. 123: 464.

H.W. and H.S. Lewis, 1963, Ann. N.Y. Acad. Sci. 100: 827-39.

phenotype: *Tyr3* heterozygotes show 35% of the diphenol oxidase activity of wild-type strains; modifiers involved in this dominant effect.

tyrosinase-1 : see *tyr1*

Tyrosinase-2 : see *Tyr2*

Tyrosinase-3 : see *Tyr3*

tyrosine 1 : see *tyr1*

Tyrosine 2 : see *Tyr2*

Tyrosine 3 : see *Tyr3*

Tyrosine hydroxylase : see *Th*

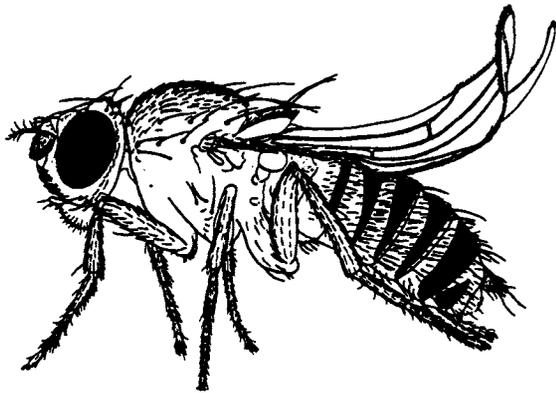
Tyrosine kinase related : see *Tkr*

****tyw*: tiny wing**

location: 3-0.

discoverer: Bridges, 18c9.

phenotype: Wings small. Postscutellars divergent, curling upward and forward. Extra bristles on head and thorax. Viability 60% wild type. RK3.



U: Upturned

Edith M. Wallace, unpublished.

U: Upturned

location: 2-70 (based on *U^{H20}*, whose allelism is uncertain). Wings upturned like those of *Cy* but dark and waxy. Postscutellars crossed as in *cu*. Body color darker than normal. Eyes mottled with light flecks. RK2A.

alleles:

allele	origin	discoverer	ref ^α	comments
<i>U¹</i>	X ray	Ball, 32a27	1, 2	homozygous lethal
<i>U^{H20}</i>		Tanaka, 35a6	2, 3	homozygous viable

^α 1 = Ball, 1935, DIS 3: 17; 2 = Bridges, 1938, DIS 9: 108; 3 = Tanaka, 1937, DIS 8: 11.

cytology: *U¹* associated with *In(2LR)U = In(2LR)40F;53A* (Bridges and Li, 1935, Year Book - Carnegie Inst. Washington 35: 293; Bridges, 1938, DIS 9: 57).

u-shaped: see *ush*

U1: see *snRNA*

U2: see *snRNA*

U3: see *snRNA*

U4: see *snRNA*

U5: see *snRNA*

U6: see *snRNA*

u-shaped: see *ush*

Uab: see *BXC*

Ual: see *ftz^{Ual}*

UB3-D: see *Ubi-m*

UB597

location: 1-55.9.

origin: Dysgenic cross of Berlin wild-type (M-cytype) females with Harwich (P-cytype) males (Campos-Ortega).

references: Fischbach, Houbé, Boschert, Barleben, and

Gschwander,

1987, J. Neurogenet. 4: 128-29 (abstract).

phenotype: Eye mutant. When flies reared in normal light-dark cycle, rhabdomeres are about 40% normal length, not extending down to the basal membrane. Rhabdomeres R7 and R8 are split into two symmetrical halves in nearly all of the ommatidia. The proximal part of the eye is filled with pigment cells. No degeneration of lamina or medulla is observed. When flies are reared in the dark, both rhabdomeres and lamina show the beginnings of disintegration and the pigment cells are swelled.

UB883

location: 1- (between *y* and *cy*).

origin: Dysgenic cross of Berlin wild-type (M-cytype) females with Harwich (P-cytype) males (Campos-Ortega).

references: Fischbach, Houbé, Boschert, Barleben, and Gschwander, 1987, J. Neurogenet. 4: 128-29 (abstract).

phenotype: Eye mutant. Inner optic chiasma in disorder. Bundles of fibers connecting medulla and lobula plate penetrate neuropil of lobula.

Ubi: Ubiquitin

The ubiquitous and highly conserved protein ubiquitin binds to certain proteins within cells, marking them for degradation by various enzymes, and is later released after these proteins have been degraded. The proteins include histone H2A, H2B, and actin. Ubiquitin genes are found in *Drosophila melanogaster* as well as in many eukaryotes from yeast to man. The proteins encoded by these genes may be polypeptides that are later processed into monomeric units or they may be hybrid proteins, each of which is made up of a single unit fused to a nonhomologous tail sequence. Both protein types have been identified in yeast, man, and *Drosophila melanogaster*.

Ubi-m: Ubiquitin-monomeric

location: 3-.

synonym: *UB3-D*.

references: Lee, Simon, and Lis, 1988, Mol. Cell Biol. 8: 4727-35.

phenotype: Structural gene for ubiquitin (Lee *et al.*, 1988). *Ubi-m* codes for a fusion protein whose single ubiquitin sequence is attached to a tail polypeptide with 65% identity to the yeast ubiquitin tail protein and 82% identity to the human ubiquitin tail protein. Ubiquitin-monomeric is not inducible by heat shock.

molecular biology: Gene cloned and nucleotide and deduced amino acids sequences obtained (Lee *et al.*, 1988). The fusion protein is made up of a 76-amino-acid ubiquitin monomer (identical to that of *Ubi-p*) and an 80-amino-acid basic tail polypeptide. *Ubi-m* encodes a 0.9 kb mRNA whose expression is not detectably increased by heat shock.

Ubi-p: Ubiquitin-polymeric

location: 3- {same map position as an early ecdysone-induced puff; see Ashburner, 1982, Developmental Studies on Giant Chromosomes (Berman, ed.). Springer-Verlag, Berlin, pp. 101-51}.

synonym: *polyubiquitin*.

references: Lis, Neckameyer, Dubensky, and Costlow,

1981, *Gene* 15: 67-80.

Izquierdo, Arribas, Galcerán, Burke, and Cabrera, 1984, *Biochim. Biophys. Acta* 783: 114-21.

Simon, Sutton, and Lis, 1985, *Chromosoma* 93: 26-30.

Arribas, Sampedro, and Izquierdo, 1986, *Biochim. Biophys. Acta* 868: 119-27.

Lee, Simon, and Lis, 1988, *Mol. Cell Biol.* 8: 4727-35.

phenotype: Structural gene for ubiquitin (Arribas *et al.*, 1988); structure and expression described by Lee *et al.* (1988). Threefold increase in level of gene by heat shock. An *Ubi-p-lacZ* fusion gene introduced by germ line transformation has been expressed at high levels at all life stages tested (embryonic, larval, pupal, and adult).

cytology: *Ubi-p* has been localized to 63F, previously identified as the early ecdysone and heat-shock puff region (Ashburner, 1972; Lis *et al.*, 1981; Izquierdo *et al.*, 1984; Simon *et al.*, 1985). In the Canton-S strain, the gene contains 18 repeats of a 228-base pair ubiquitin-encoding unit, all arranged in tandem (Lee *et al.*, 1988).

molecular biology: Intact *Ubi-p* genes have been cloned. The nucleotide sequences of several monomeric and dimeric repeats have been independently determined (Arribas *et al.*, 1986; Lee *et al.*, 1988). These sequences varied, especially at the third position of the codon. The predicted amino acid sequences of the repeats, however, were identical to those of five of the nine ubiquitin-encoding repeats previously reported. The *Ubi-p* gene encodes a major 4.4 kb mRNA in all tested *in vivo* and *in vitro Drosophila melanogaster* cells; the expression of this mRNA is increased by heat shock. The nucleotide sequences of *Ubi-m* and *Ubi-p* are about 83% identical.

Ubi: see *RpII215^{Ubi}*

Ubx: see *BXC*

uex: unextended

location: 2-55.

references: Maeda, 1962, *DIS* 36: 39.

Hilliker, 1976, *Genetics* 83: 765-82.

Maeda, 1984, *Jpn. J. Genet.* 59: 249-57.

phenotype: Wings incompletely expanded as in a newly emerged fly, about one-half normal length, and frequently inflated. Tibiae and tarsi of third legs irregularly shortened and gnarled. Posterior scutellars convergent. The rare *uex²-uex⁷* hemizygous survivors may also have etched tergites and small bodies.

alleles: Original allele found by Maeda, 5813, has not been lost (Maeda, 1984); the six hemizygous late-pupal lethals induced by Hilliker are inferred to be allelic to *uex¹* on the basis of map position and phenotype.

allele	origin	synonym	ref ^α	comments
<i>uex¹</i>	spont	<i>unexpanded</i>	2, 3	low male viability
<i>uex²</i>	EMS	<i>l(2)EMS34-07</i>	1	late lethal, few escapers
<i>uex³</i>	EMS	<i>l(2)EMS45-01</i>	1	late lethal, few escapers
<i>uex⁴</i>	EMS	<i>l(2)EMS45-17</i>	1	late lethal, few escapers
<i>uex⁵</i>	EMS	<i>l(2)EMS45-37</i>	1	late lethal, few escapers
<i>uex⁶</i>	EMS	<i>l(2)EMS45-40</i>	1	late lethal, few escapers
<i>uex⁷</i>	EMS	<i>l(2)EMS45-73</i>	1	late lethal, few escapers

^α 1 = Hilliker, 1976, *Genetics* 83: 765-82; 2 = Maeda, 1962, *DIS* 36: 39. 3 = Maeda, 1984, *Jpn. J. Genet.* 59: 249-57.

cytology: Located in 41A; included in *Df(2R)A''* but not in *Df(2R)A*.

Uf: Unfolded

location: 2- (to the left of *b*).

origin: X ray induced.

discoverer: Belgovsky, 36c29.

phenotype: Wings spread in homozygote and heterozygote. Viability and fertility good. RK3.

Ugra: see *Fs(2)Sz12*

Ultrabar: see *BB*

Ultrabithorax: see *BXC*

ultraspiracle: see *usp*

un: uneven

location: 1-54.4.

references: Mohr, 1927, *Nyt Mag. Naturv.* 65: 266.

Bateman, 1951, *DIS* 25: 78.

Krivshenko, 1956, *DIS* 30: 75.

Garen and Kankel, 1983, *Dev. Biol.* 96: 445-66.

phenotype: Eyes smaller than normal, surface rough. Hemi- and homozygotes have retinas with slight to moderate disorganization of the ommatidial array, with occasional fused or aberrant ommatidia and missing or condensed rhabdomeres (Garen and Kankel, 1983). There is some variation in the structure of the individual neurons of the optic lobe. Within the retina, *un* behaves in a nonautonomous way as indicated by analysis of genetic mosaics.

alleles: The three surviving and three lost alleles are described in the following table:

allele	origin	discoverer	ref ^α	comments
<i>un¹</i>	spont	Mohr, 25a14	2, 4	RK1
<i>*un^{3β}</i>	X ray	Demerec, 28f30		wing margins frayed, eyes like <i>un¹</i> ; RK1
<i>un⁴</i>	X ray	Dubinín, 1928		less extreme, more viable than <i>un¹</i> or <i>un³</i> ; RK2
<i>un^C</i>		Craymer	2	
<i>*un^{Kγ}</i>	spont	Krivshenko, 56b9	3	eyes small, bulging, rough; scutellum long, narrow, with thin, deformed bristles; good viability, fertility; RK1
<i>*un^{Pδ}</i>	32 p	Bateman	1	eyes like <i>un¹</i> ; RK2

^α 1 = Bateman, 1951, *DIS* 25: 78; 2 = Garen and Kankel, 1983, *Dev. Biol.* 96: 445-66; 3 = Krivshenko, 1956, *DIS* 30: 75; 4 = Mohr, 1927, *Nyt Mag. Naturv.* 65: 266.

^β Synonym: *ro-63*.

^γ Cytology: Salivary chromosomes appear normal.

^δ Other information: Allelism inferred from phenotype and genetic location.

cytology: Located in *14D1;15A4* since included in *Df(1)r-D1 = Df(1)14D1-2;15D1-2* but not covered by *Dp(1;3)f⁺71b = Df(1;3)15A4;16C2-3;80-81* (Schalet, 1986, *Mutat. Res.* 163: 115-44).

unc: uncoordinated

location: 1-65.9 (reduced from Fahmy's value of 68.9 to fit on map).

references: Fahmy, 1960, *DIS* 34: 49.

Schalet, 1972, *DIS* 49: 36-37, 64-66.

Schalet and Lefevre, 1973, *Chromosoma* 44: 183-202.

Schalet and Lefevre, 1976, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902.

Miklos, Healy, Pain, Howells, and Russell, 1984, *Chro-*

mosoma 89: 218-27.

Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31.

phenotype: Fly unable to walk because of lack of coordination in moving legs. Wings held up and frequently curled at tips. Death usually takes place after eclosion.

alleles: *unc*¹, the original allele induced by Fahmy, has been lost. 22 mutants identified as male lethals or semi-lethals were found to be allelic to *unc*¹.

allele	origin	discoverer	synonym ^α	ref ^β	comments
<i>*unc</i> ¹	CB. 3025	Fahmy, 1954		1	dies shortly after eclosion; RK3
<i>unc</i> ^{2γ}	neutrons	Muñoz	<i>l(1)16-3-212</i>	6, 7, 9	
<i>unc</i> ³	EMS	Baldwin	<i>l(1)LB17</i>	8, 9	
<i>*unc</i> ⁴	HT	Kaplan	<i>l(1)LV7</i>	9, 11	
<i>unc</i> ⁵	EMS	Lifschytz	<i>l(1)M169</i>	5	on y ⁺ <i>Ymal</i> ⁺
<i>unc</i> ⁶	EMS	Lifschytz	<i>l(1)R-9-31</i>	4	
<i>unc</i> ⁷	EMS	Lifschytz	<i>l(1)R-10-1</i>	4, 8, 10	
<i>unc</i> ⁸	EMS	Lifschytz	<i>l(1)W1</i>	4, 8, 10	also <i>run</i> ⁸
<i>unc</i> ⁹	EMS	Lifschytz	<i>l(1)W5</i>	4, 8, 10	
<i>unc</i> ¹⁰	X ray	Lefevre	<i>l(1)GE230</i>	2	
<i>unc</i> ¹¹	X ray	Lefevre	<i>l(1)GE250</i>	2	
<i>unc</i> ¹²	X ray	Lefevre	<i>l(1)HA28</i>	2	
<i>unc</i> ¹³	X ray	Lefevre	<i>l(1)HF329</i>	2	
<i>unc</i> ¹⁴	X ray	Lefevre	<i>l(1)JA69</i>	2	<i>T(1;A)19F3;h?</i>
<i>unc</i> ¹⁵	X ray	Lefevre	<i>l(1)RC60</i>	2	
<i>unc</i> ¹⁶	X ray	Lefevre	<i>l(1)RF7</i>	2	
<i>unc</i> ¹⁷	X ray	Lefevre	<i>l(1)S86</i>	2	
<i>unc</i> ^{18γ}	EMS	Lefevre	<i>l(1)DC803</i>	3, 7	
<i>unc</i> ¹⁹	EMS	Lefevre	<i>l(1)VA288</i>	3	<i>ln(1)17E-F;19E</i>
<i>unc</i> ²⁰	EMS	Lefevre	<i>l(1)VE715</i>	3	<i>Tp(1;2)19E;27A</i>
<i>unc</i> ²¹	EMS	Lefevre	<i>l(1)VE797</i>	3	
<i>unc</i> ²²	EMS	Lefevre	<i>l(1)VE799</i>	3	
<i>unc</i> ²³	EMS	Lefevre	<i>l(1)VE824</i>	3	
<i>unc</i> ^{24γ}	X ray	Schalet	<i>l(1)27E2</i>	7	

^α Synonym for *unc* locus = *l(1)19Ef*.
^β 1 = Fahmy, 1960, DIS 34: 49; 2 = Lefevre, 1981, Genetics 99: 461-80; 3 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 4 = Lifschytz and Falk, 1968, Mut. Res. 8: 147-55; 5 = Lifschytz and Jakobovitz, 1978, Mol. Gen. Genet. 161: 275-84; 6 = Miklos, Healy, Pain, Howells, and Russell, 1984, Chromosoma 89: 218-27; 7 = Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31; 8 = Schalet, 1972, DIS 49: 36-37, 64-66; 9 = Schalet and Lefevre, 1973, Chromosoma 44: 183-202; 10 = Schalet and Lefevre, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.), Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902; 11 = Schalet and Singer, 1971, DIS 46: 131-32.

^γ Lethal phase during pupation or after emergence; *unc* gene not maternally required (Perrimon, Smouse, and Miklos, 1989).

cytology: Placed in 19E7-F1 (Schalet and Lefevre, 1973, 1976; Miklos *et al.*, 1984) since included in *Df(1)Q539* = *Df(1)19E6;19F6-20A1* but not in *Df(1)DCB1-35b* = *Df(1)19F1-2;20E-F* or *Df(1)A118* = *Df(1)19E4-5;19E7-8*.

molecular biology: A cloned entry point into the transition zone between the euchromatic and heterochromatic regions of the X chromosome was mapped to the near vicinity of *unc* (Miklos *et al.*, 1984).

uncl: uncoordinatedlike

location: 1- {66} (between *wap* and *fog*).

references: Lifschytz and Falk, 1968, Mut. Res. 6: 235-44.

Lifschytz and Falk, 1969, Mut. Res. 8: 147-55.

Lifschytz and Jakobovitz, 1978, Mol. Gen. Genet. 161: 275-84.

Schalet, 1972, DIS 49: 36-37, 64-66.

Schalet and Lefevre, 1973, Chromosoma 44: 183-202.

Schalet and Lefevre, 1976, The Genetics and Biology of

Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 848-902.

Lefevre and Watkins, 1986, Genetics 113: 869-95.

Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31.

phenotype: Most flies die before eclosion, but the ones that hatch have uncoordinated leg movements and soon die. Females that are hemizygous have the same abnormal phenotype as homozygotes. Viability of various allele heterozygotes is only 1-12%.

alleles:

allele	origin	discoverer	synonym ^α	ref ^β	comments
<i>uncl</i> ¹	X ray	Lifschytz	<i>l(1)B83</i>	3, 4, 6-8	
<i>uncl</i> ²			<i>l(1)J14</i>		
<i>uncl</i> ³	EMS	Lifschytz	<i>l(1)M82</i>	5	on y ⁺ <i>Ymal</i> ⁺
<i>uncl</i> ⁴	EMS	Lifschytz	<i>l(1)Q456</i>	4, 6-8	
<i>uncl</i> ⁵	EMS	Lifschytz	<i>l(1)R-10-10</i>	4, 6-9	pupal lethal
<i>*uncl</i> ⁶	X ray	Lefevre	<i>l(1)HC175</i>	1	
<i>uncl</i> ⁷	X ray	Lefevre	<i>l(1)RF7</i>	1	
<i>uncl</i> ⁸	EMS	Lefevre	<i>l(1)DF905</i>	2	
<i>uncl</i> ⁹	EMS	Lefevre	<i>l(1)GA137</i>	2	
<i>uncl</i> ¹⁰	EMS	Lefevre	<i>l(1)VA228</i>	2, 6	early larval lethal
<i>uncl</i> ¹¹	spont	Schalet	<i>l(1)16-66</i>	10	

^α Synonym for *uncl* locus = *l(1)20Ae*.

^β 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 3 = Lifschytz and Falk, 1968, Mutat. Res. 6: 235-44; 4 = Lifschytz and Falk, 1969, Mut. Res. 8: 147-55; 5 = Lifschytz and Jakobovitz, 1978, Mol. Gen. Genet. 161: 275-84; 6 = Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31; 7 = Schalet, 1972, DIS 49: 36-37, 64-66; 8 = Schalet and Lefevre, 1973, Chromosoma 44: 183-202; 9 = Schalet and Lefevre, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.), Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902. 10 = Schalet, 1986, Mutat. Res. 163: 115-44.

cytology: Placed in 20A4-5 (Schalet and Lefevre, 1976; Lefevre and Watkins, 1986); since included in *Df(1)17-439* = *Df(1)20A;20B* (Schalet and Lefevre, 1976) and in *Df(1)GA33* (Lefevre), which also includes *unc* (Perrimon *et al.*, 1989), but not in *Df(1)17-257* = *Df(1)19F3;20A1-2* or *Df(1)16-3-22* = *Df(1)19D1;20A2*.

uncoordinated: see *unc*

uncoordinatedlike: see *uncl*

undersized: see *us*

uneven: see *un*

Uneven wing: see *Bg*²

unexpanded: see *unp*

unexpanded irregular: see *unr*

unextended: see *uex*

unfolded: see *uf*

unp: unexpanded

location: 1-63.1.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).

discoverer: Fahmy, 1954.

references: 1959, DIS 33: 94.

phenotype: Wings always unexpanded, frequently droop.

Two symmetrical grooves occur on the pronotum immediately anterior to wing base. Postscutellar bristles often crossed. Eclosion delayed. Male fertile; viability

about 10% normal. Female extremely inviable. RK3.
alleles: One allele each induced by CB. 1356 and X rays.

unpaired: see *upd*

****unr:* unexpanded irregular**

location: 1-52.3.

origin: Induced by 2-chloroethyl methanesulfonate (CB. 1506).

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 94.

phenotype: Wings usually unexpanded to some degree; if expanded, they are short, broad, and slightly drooping or divergent. Fertility reduced in both sexes. RK3.

Uo:* see *Uro

***up:* upheld (J.C. Hall)**

location: 1-41.0 [between *s* and *g* (Deak *et al.*, 1982)].

references: Fahmy, 1958, DIS 32: 77.

Hotta and Benzer, 1972, *Nature* (London) 240: 527-35.

Grigliatti, Hall, Rosenbluth, and Suzuki, 1973, *Mol. Gen. Genet.* 120: 107-14.

Deak, 1977, *J. Embryol. Exp. Morphol.* 40: 35-63.

Fekete and Szidonya, 1979, *Acta Biol. Acad. Sci. Hung.* 30: 47-57.

Homyk, Szidonya, and Suzuki, 1980, *Mol. Gen. Genet.* 177: 553-65.

Vasudev, Krishnamurthy, and Gayathri, 1980, DIS 55: 211.

Mogami, Nonomura, and Hotta, 1981, *Jpn. J. Genet.* 56: 51-65.

Deak, Bellamy, Bienz, Dubuis, Fenner, Gollin, Rähmi, Ramp, Reinhardt, and Cotton, 1982, *J. Embryol. Exp. Morphol.* 69: 61-81.

Hall, 1982, *Quart. Rev. Biophys.* 15: 223-479.

Homyk and Emerson, 1988, *Genetics* 119: 105-21.

phenotype: Wings held upright and mutants unable to jump or fly when homo- or hemizygous. The indirect flight muscles of the thorax are abnormal, with many mitochondria in the muscle fiber envelopes but with defective myofibrils (Hotta and Benzer, 1972; Deak, 1977); electron microscope and electrophoresis studies indicate that these muscles lack internal structure in the Z-bands and Z-band proteins (Fekete and Szidonya, 1979; Mogami *et al.*, 1981). Although wings are held in normal position, wild-type heterozygotes of certain mutant alleles are unable to jump or fly; their muscles contain half the normal amount of Z-band proteins. Fate maps of the behavioral and morphological foci of *up* and *up*² indicate that the gene has its site of action in the presumptive musculature of the ventral mesoderm (Hotta and Benzer, 1972; Deak, 1977). Viability and fertility of *up* is good. RK1.

alleles: *up* alleles are listed in the following table. *int*³ may be an *up* allele since the two genes are close together and do not complement each other, but *int*³ is not listed in the table because its homozygotes show a different pattern of muscle abnormalities than do *up* homozygotes (Deak *et al.*, 1982; Homyk and Emerson, 1988).

allele	origin	ref ^α	wing phen.		able to fly?	
			<i>up/up</i>	<i>up/+</i>	<i>up/up</i>	<i>up/+</i>
<i>up</i> ^{1β}	CB. 3007	1-3, 6	abnormal	normal	-	-
<i>up</i> ^{2γ}	EMS	1, 2, 7-10	abnormal	normal	-	-
<i>up</i> ³	EMS	2, 7	abnormal	normal	-	-
<i>up</i> ^{101δ}	EMS	4, 7, 8	abnormal	normal	-	+

allele	origin	ref ^α	wing phen.		able to fly?	
			<i>up/up</i>	<i>up/+</i>	<i>up/up</i>	<i>up/+</i>
<i>up</i> ^{wg}	amino-naphthol-sulfonic acid	11	abnormal		-	-
<i>up</i> ^{whuε}	EMS	4, 5, 7, 8	3 normal: 1 abnormal	normal	-	+/-
<i>up</i> ^x	EMS	7	abnormal	normal	-	+

^α 1 = Deak, 1977, *J. Embryol. Exp. Morphol.* 40: 35-63; 2 = Deak, Bellamy, Bienz, Dubuis, Fenner, Gollin, Rähmi, Ramp, Reinhardt, and Cotton, 1982, *J. Embryol. Exp. Morphol.* 69: 61-81; 3 = Fahmy, 1958, DIS 32: 77; 4 = Fekete and Szidonya, 1979, *Acta Biol. Acad. Sci. Hung.* 30: 47-57; 5 = Grigliatti, Hall, Rosenbluth, and Suzuki, 1973, *Mol. Gen. Genet.* 120: 107-14; 6 = Hall, 1982, *Quart. Rev. Biophys.* 15: 223-479; 7 = Homyk and Emerson, 1988, *Genetics* 119: 105-21; 8 = Homyk, Szidonya, and Suzuki, 1980, *Mol. Gen. Genet.* 177: 553-65; 9 = Hotta and Benzer, 1972, *Nature* (London) 240: 527-35; 10 = Mogami, Nonomura, and Hotta, 1981, *Jpn. J. Genet.* 56: 51-65; 11 = Vasudev, Krishnamurthy, and Gayathri, 1980, DIS 55: 211.

^β All mutant flies raised at 29° hold their wings in a vertical position, while 60% of those raised at 18° hold their wings in a ventrolateral position (Deak, 1977); all flies are flightless and have abnormal muscles regardless of temperature. *up*¹ shows more muscle abnormalities than *up*².

^γ **Synonym:** *wupB*. Flight muscles lack cross-striations (Hotta and Benzer, 1972). "jump" muscle absent or very small. Extracts of indirect flight muscles do not show a 54 kd polypeptide that may be a Z-band component (Deak *et al.*, 1982). Shows electron-dense structures instead of normal Z-bands (Deak *et al.*, 1982).

^δ **Synonym:** *wupB*¹⁰¹. Although allele previously reported as semi-dominant for flightlessness (Homyk *et al.*, 1980), heterozygotes found to be able to hop and fly about as well as wild type (Fekete and Szidonya, 1979; Homyk and Emerson, 1988).

^ε Semidominant for flightlessness (Homyk and Emerson, 1988).

cytology: Located in 12A1-7; between the proximal end of *Df(1)C246 = Df(1)11D-E;12A1-2* and the distal end of *Df(1)HA92 = Df(1)12A6-7;12D3* (Deak *et al.*, 1982), a region thought to be haplolethal since it is not uncovered by known deficiencies (Stewart and Merriam, 1973, DIS 50: 167-70). Although *up*^{2/+} females show the dominant flightless phenotype, *up*^{2/+/+} females carrying a duplication for 11E-12A can hop and fly as well as wild type (Homyk and Emerson, 1988).

other information: Two alleles, *up*¹⁰¹ and *up*^x, that show a wild-type flight phenotype as heterozygotes, are flightless when also heterozygous for the wing mutants *hdp* (*hdp-a* and *hdp-b* not distinguished) or *rsd* (Homyk and Emerson, 1988). *up*^{2/+;rsd/+} are not only flight-defective, but about 1/3 of them have abnormal wing positions (Deak *et al.*, 1982). *up*^{101;Tm2/+;int³;Tm2/+}, *up*^{x;Tm2/+}, *up*^{101;Mhc⁵/+}, and *up*^{101;Mhc¹⁶/+} are lethal. Double heterozygotes involving *up*¹⁰¹, *int*³, or *up*^x and *Mhc*⁵ cannot fly and have abnormal wing postures. These alleles over *hdp-a*² show the same phenotype.

***upd:* unpaired**

location: 1-58.7.

origin: Induced by ethyl methanesulfonate.

references: Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 296-307.

Carroll and Scott, 1986, *Cell* 45: 113-26.

Gergen and Wieschaus, 1986, *Wilhelm Roux's Arch. Dev. Biol.* 195: 49-62.

Wieschaus and Noell, 1986, *Wilhelm Roux's Arch. Dev. Biol.* 195: 63-73.

Perrimon, Engstrom, and Mahowald, 1989, *Genetics* 121: 333-52.

phenotype: Embryonic lethal. Mutants show variable larval cuticular phenotype with defects predominantly in the mesothorax and the fifth abdominal segment (Nüsslein-Volhard *et al.*, 1984; Gergen and Wieschaus, 1986); also some head defects and fourth, sixth, seventh, and eighth abdominal segment defects. Heterozygotes show 88-93% viability (Wieschaus and Noell, 1986).

alleles:

allele	synonym	ref ^α	comments
upd¹	<i>upd^{C43}</i>	1,3	T2 and A5 defects; no effect on <i>ftz</i>
upd²	<i>upd^{M5}</i>	3	
upd³	<i>upd^{YC43}</i>	2	T2, A5, A8 denticle belts deleted; A6 and A7 denticle belts fused; abnormal posterior spiracles
upd⁴	<i>upd^{YM55}</i>	2	T2 defect; rudimentary filzkörper and posterior spiracles; A6 and A7 denticle belts fused; A8 denticle belt reduced

^α 1 = Carroll and Scott, 1986, Cell 45: 113-26; 2 = Gergen and Wieschaus, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 49-62; 3 = Wieschaus and Noell, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 63-73.

cytology: Located in 17A-B (Perrimon *et al.*, 1989).

other information: Determined to be allelic to *os* too late to be included in the *os* entry (Perrimon).

upheld: see *up*

upi: see *Pu*

***ups: upright scutellars**

location: 1-40.8.

origin: Spontaneous.

discoverer: Fahmy, 1955.

references: 1958, DIS 32: 77.

phenotype: Posterior scutellar bristles held vertically. Fly small. Eyes dull, small, and abnormally shaped. Wings short and folded. Male sterile; viability about 20% normal. RK2.

upt: upturned bristles

location: 2- (between *dp* and *pr*).

origin: Spontaneous.

discoverer: Bryan, 63f.

references: Whittinghill and Clancy, 1967, DIS 42: 37.

phenotype: Dorsocentral and/or anterior scutellar bristles curled upwards. Increase in penetrance as temperature raised (40% at 21°; 60% at 25°). Variable expression.

Upturned: see *U*

upturned bristles: see *upt*

***upw: upward**

location: 2-62.

discoverer: Bridges, 33k21.

phenotype: Wings turned up at tips. More extreme at higher temperatures. Veins sometimes have lumps. RK3.

uq: see *br^{uq}*

Urate oxidase: see *Uro*

urd: urdur (J.A. Kennison)

location: 3-53.

origin: Induced by ethyl methanesulfonate.

discoverer: Kennison, 1983.

references: Kennison and Tamkun, 1988, Proc. Nat. Acad.

Sci. USA 85: 8136-40.

phenotype: Isolated as a dominant suppressor of *Pc* mutations. Also suppresses *Pcl* and *Msc* alleles. Recessive larval lethal.

alleles: *urd²* induced by ethyl methanesulfonate.

cytology: Placed in 87F12-88A1 based on the cytology of *Df(3R)urd*, which lacks several bands in 87F; also its exclusion from *Df(3R)I26c = Df(3R)87D14-E1;87F11-12* and its inclusion within *Df(3R)red31 = Df(3R)87F12-14;88C1-3* and *Df(3R)ry85 = Df(3R)87B15-C1;87F15-88A1*.

Uro: Urate oxidase

location: 2- {22}.

synonym: *Uo*.

references: Friedman and Johnson, 1977, Science 197: 477-79.

Friedman and Barker, 1982, Insect Biochem. 12: 563-70.

Kral, Johnson, Wing, and Friedman, 1982, Dev. Genet. (Amsterdam) 3: 213-15.

phenotype: Structural gene for urate oxidase [UO (E.C. 1.7.3.3)], the enzyme involved in the oxidation of uric acid to allantoin. Enzyme confined to the Malpighian tubules and only expressed in third-instar larvae and adults. Rapid decline in urate oxidase activity in late third-instar larvae attributed to the accumulation of 20-hydroxyecdysone for puparium formation; *ecd* larvae, when kept at 29°, fail to accumulate 20-hydroxyecdysone and, unless fed 20-hydroxyecdysone, retain high urate oxidase activity (Kral *et al.*, 1982).

cytology: Located in 28C by *in situ* hybridization.

molecular biology: Gene cloned (Kral, Johnson, Burnett, and Friedman, 1986, Gene 45: 131-37). Highest concentration of mRNA in third instar larvae; decline in enzyme activity accompanied by disappearance of mRNA. Sequence and expression pattern described by Walbrath, Burnett, and Friedman (1990, Mol. Cell. Biol. 10: 5114-27).

***us: undersized**

location: 1-52.5.

origin: X ray induced.

discoverer: Fahmy, 1956.

references: 1959, DIS 33: 94.

phenotype: Body small. Viable and fertile. RK3.

alleles: One allele each induced by CB.1506 and CB.1528; two by X rays.

ush: u-shaped

location: 2-0.1.

origin: Induced by ethyl methanesulfonate.

synonym: *l(2)19*.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

phenotype: Embryonic lethal. No shortening of the germ band. Lateral fusion of anterior and posterior hypoderm.

alleles: *ush¹* and *ush²*, both strong alleles, recovered as *19* and *Ila*.

cytology: Located in 21C since uncovered by *Df(2L)al = Df(2L)21B8-C1;21C8-D1*.

usp: ultraspiracle (A. E. Oro)

location: 1- {0.5}.

discoverer: Lefevre.

synonym: *XR2C* (Oro *et al.*, 1990); *Cfl* (Shea *et al.*, 1990).

references: Perrimon, Engstrom, and Mahowald, 1984,

Dev. Biol. 105: 404-14.

1985, Genetics 111: 23-41.

Perrimon and Mahowald, 1986, Symp. Soc. Dev. Biol. 44: 221-35.

Henrich, Sliter, Lubahn, MacIntyre, and Gilbert, 1990, Nucleic Acids Res. 18: 4143-48.

Oro, McKeown, and Evans, 1990, Nature 347: 298-301.

Shea, King, Conboy, Mariani, and Kafatos, 1990, Genes Dev. 4: 1128-40.

phenotype: Mutants are recessive lethals. *usp/usp* or *usp/γ* progeny of *usp/+* mothers die during the first larval instar or in the molt to the second instar. Those that die during the molt to the second instar sometimes have incompletely molted the first instar set of larval spiracles and thus have two sets of spiracles (Perrimon *et al.*, 1985; Oro, McKeown and Evans, in prep.). *usp/Y* embryos derived from *usp/usp* germ cells die just prior to or just after hatching with an oval scar on the ventral surface of the posterior eighth abdominal or ninth abdominal segment. The spiracles of these animals appear normal as does the rest of the cuticle and the ventral nervous system. Paternally supplied *usp⁺* completely rescues this phenotype and allows survival to adulthood (Perrimon *et al.*, 1985; Oro, McKeown, and Evans, in prep.). Use of a conditional expression system for rescue of the first/second instar lethal phase results in survival into the third instar and early pupal periods, with no animals surviving beyond pupal stage P4 (Oro, McKeown, and Evans, in prep.). *usp/0//usp⁺* gynandromorphs do not survive suggesting that the gene is required in multiple parts of the body and not just in the terminal regions. γ -ray induced *usp/usp* clones survive in the female germline, abdomen, thorax and head, showing that *usp* is not a general cell lethal. Clones in the head are associated with defects in rhabdomere and ommatidial morphology (Oro, McKeown, and Evans, in prep.). Expression of a *usp* cDNA at high levels throughout development rescues the *usp⁻* phenotype and has no deleterious effect on *usp⁺*

animals, suggesting that any necessary spatial or temporal regulation of *usp* action occurs by regulation of some other factor such as a ligand.

alleles:

allele	origin	synonym	ref ^α	comments
<i>usp¹</i>	X ray	<i>l(1)HF346</i>	1	<i>T(1;4)2C1-2;101</i>
<i>usp²</i>	X ray	<i>l(1)KA21</i>	1, 2	<i>Tr(3;1)2C9;66B;67E</i>
<i>usp³</i>	EMS	<i>l(1)VE653</i>	1-3	
<i>usp⁴</i>	EMS	<i>l(1)VE849</i>	1-3	

^α 1 = Lefevre; 2 = Oro, McKeown, and Evans, 1990, Nature 347: 298-301; 3 = Perrimon, Engstrom, and Mahowald, 1985, Genetics 111: 23-41.

cytology: Placed in 2C1-2D1 since covered by *Dp(1;3)w^{vc0}* = *Dp(1;3)2B17-C1;3C5-6* but not by *Dp(1;Y)w⁺303* = *Dp(1;Y)2D1-2;3D3-4* (Perrimon *et al.*, 1970).

molecular biology: Cloning and nucleotide and putative amino acid sequencing of the 2C region indicates that the protein product of *usp* (= *XR2C*) is a homologue of the human retinoid X receptor, (Oro *et al.*, 1990). Factors with the same putative DNA-binding domain as *usp* have been cloned and sequenced by Shea *et al.* (1990) and Henrich *et al.* (1990). *usp* produces a single 2.4-kb transcript with higher levels in adults and embryos than in larvae. The colinearity of the cDNA and the genomic DNA indicates that there are no introns. In the mutant *usp²*, the coding region has been disrupted by insertion of a 66B-67E fragment from the third chromosome. The structure of the cloned breakpoints of this transposition [*Tr(3;1)usp⁴*] has been determined. The lethal mutants *usp²*, *usp³*, and *usp⁴* are rescued by germ-line transformation with an 8-kb *EcoR1* fragment carrying the *XR2C* transcription unit, the transformed flies being as viable as wild-type (*usp⁺*) flies (Oro *et al.*, 1990).

Uw: see *Bg²*

v: vermilion

location: 1-33.0.

discoverer: Morgan, 10k.

references: Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. No. 237: 27 (fig.).

Schultz and Bridges, 1932, Am. Nat. 66: 323-32.

Sturtevant, 1932, Proc. Intern. Congr. Genet., 6th, Vol. 1: 304-07.

Offerman, 1935, DIS 3: 28.

Beadle and Ephrussi, 1936, Proc. Nat. Acad. Sci. USA 22: 536-40.

Sturtevant and Beadle, 1939, An Introduction to Genetics, W.B. Saunders Co., Philadelphia, p. 64 (fig.).

Tatum and Beadle, 1939, Biol. Bull. 77: 415-22.

Brehme and Demerec, 1942, Growth 6: 351-56.

Green, 1952, Proc. Nat. Acad. Sci. USA 38: 300-05.

Baglioni, 1959, Nature (London) 184: 1084-85.

Green, 1959, Genetics 34: 564-72.

Baglioni, 1960, Heredity 15: 87-96.

Shapard, 1960, Genetics 45: 359-76.

Kaufman, 1962, Genetics 47: 807-17.

Rizki, 1963, J. Cell Biol. 16: 513-320.

Marzluf, 1965, Genetics 52: 503-12.

Rizki and Rizki, 1968, Genetics 59: 477-85.

Tartof, 1969, Genetics 62: 781-95.

Baillie and Chovnick, 1971, Mol. Gen. Genet. 112: 341-53.

Jacobson, Grell, Yim, and Gardner, 1982, Genet. Res. 40: 19-32.

Searles and Voelker, 1986, Proc. Nat. Acad. Sci. USA 83: 404-08.

Walker, Howells, and Tearle, 1986, Mol. Gen. Genet. 202: 102-07.

Pastink, Vreeken, Nivard, Searles, and Vogel, 1989, Genetics 123: 123-29.

phenotype: The v^+ gene, expressed in the eyes, fat body, and Malpighian tubules of the wild type (Nissani, 1975, Genet. Res. 26: 63-72), is believed to code for the enzyme tryptophane oxidase (also known as tryptophane pyrrolase)(EC 1.3.11.11), a 150,000 dalton protein that catalyzes the conversion of tryptophane into N-formylkynurenine. The eye color of v mutants is bright scarlet owing to absence of brown ommochrome; ocelli are colorless. Flies with the mutant combination $y;w$ have white eyes. The eye color is wild type in genetically v eyes of gynandromorphs mosaic for wild type and v tissue (Sturtevant, 1932), indicating the non-autonomous nature of the vermilion gene. v eye disks develop wild-type pigmentation when transplanted into wild-type larvae (Beadle and Ephrussi, 1936). yw nuclei from preblastoderm stages implanted into the posterior end of a fertilized $v;bw$ egg can produce a mosaic fly with brown eyes (Zalokar, 1973, Dev. Biol. 32: 189-93). The diffusible v^+ hormone of Beadle and Ephrussi involved in mosaic and transplantation experiments has been identified as kynurenine (Butenandt, Weidel, and Becker, 1940, Naturwissenschaften 28: 63-64). Activity of the inducible enzyme tryptophane oxidase is absent in v mutants (Baglioni, 1959, 1960). As a result, nonprotein tryptophane is accumulated in vermilion flies (Green, 1959) rather than converted into N-formylkynurenine and then into formic acid and kynurenine. In mutant larvae tryptophane in the fat body is not converted into kynurenine (Rizki, 1963; Rizki and Rizki, 1968). Certain v alleles

(v^l , v^2 , and v^k) are suppressed by mutations at the $su(s)$ locus; these mutants show wild-type eye color, fail to accumulate nonprotein tryptophane, and partially restore tryptophane oxidase activity in spite of the mutation at v (Schultz and Bridges, 1932; Green, 1952; Baglioni, 1960; Shapard, 1960; Kaufman, 1962; Marzluf, 1965; Tartof, 1969; Jacobson *et al.*, 1982); other v alleles (v^{36f} , v^{48a} , v^{51a} , v^{51b} , v^{51c} , and v^{E1}) show no change in the mutant eye color with these $su(s)$ alleles and little or no increase in tryptophane oxidase activity (small increase observed in $su(s) v^{36f}$ flies). Some brown pigment is formed under conditions of partial starvation in suppressed v mutants (Tatum and Beadle, 1938; Shapard, 1960), but starvation has no effect on unsuppressed v alleles.

alleles: The following table lists wild-type and mutant alleles. The revertants v^{+37} [Germeraad, 1976, Nature (London) 262: 229-31] and v^{36f+} (Green) are not described in the table. v deficiencies are listed as rearrangements.

allele	discoverer	origin	ref ^α	cytology
v^1	Morgan, 10k	spont	1-6, 17-19, 21, 22, 25, 27, 30, 32-39, 40-42, 44, 46, 47, 51, 52, 53	
v^{05}	Fomina	EMS	49, 50, 52, 53	
v^{06}	Fomina	EMS	49, 50, 52, 53	
v^2	Plunkett, 24g	spont	8, 19, 33, 35-38, 46, 53	
v^{2B27}	Belyaeva	EMS	50, 53	
v^{2B37}	Belyaeva	EMS	50, 53	
v^{2B54}	Belyaeva	EMS	53	
v^{2B157}	Belyaeva	EMS	50, 53	
v^{2B160}	Belyaeva	EMS	50, 53	
v^{2B162}	Belyaeva	EMS	50, 53	
v^{2B165}	Belyaeva	EMS	50, 53	
v^{2B195}	Belyaeva	EMS	50, 53	
v^{2B206}	Belyaeva	EMS	50, 53	
v^{2B207}	Belyaeva	EMS	50, 53	
v^{2B236}	Belyaeva	EMS	50, 53	
v^{2B237}	Belyaeva	EMS	50, 53	
v^8	Geer	EMS	17, 45	
v^9	Geer	EMS	17, 45	
v^{10}	Geer	EMS	17, 45	
v^{24}	Loker	EMS	9, 16	
v^{36f}	Williams, 36f	spont	8, 18-20, 21, 33, 35, 37, 41, 43, 45, 46, 52, 53	
v^{48a}	Fox, 48a7	X ray	7, 11, 19, 33, 34, 41, 46, 53	
v^{51a}	Green	X ray	19	
v^{51b}	Green	spont	19	
v^{51c}	Green	X ray	19, 33, 46, 53	
v^{51g}	Edmondson, 51g	U-V	26	
v^{61j}	Goodwins		50, 53	
v^{65c}			6	
v^{71P}		spont	37, 46	
v^{74k}	Lim	EMS	50, 53	
v^{79j}	Najera	spont	29a	
v^{80d}	Najera	spont	29b	
v^{81d}	Najera	spont	29, 29a	
v^{125}		EMS	52	
v^{127}	Geer	EMS	17, 45	
v^{162}		EMS	51	
v^{166}		EMS	51	
v^{210}	Geer	EMS	17, 45	
v^{216}	Geer	EMS	17, 45	
v^{217}	Geer	EMS	17, 45	
v^{219}	Geer	EMS	17, 45	
v^{221}	Geer	EMS	17, 45	
v^{267-4}	Hoover, 35i	X ray		T(1;2)11A7-8;36
v^{+63i}	Lefevre		11, 17, 22, 24, 49, 50, 54	Dp(1;2)9E1; 10A11;56A
v^{+65b}	Lefevre		11, 17, 22,	Tp(1;2)10A1;

allele	discoverer	origin	ref ^α	cytology	allele	discoverer	origin	ref ^α	cytology
			24, 45, 54	11A7-8;40-41	v G57	Pokholkova	EMS	49, 50, 52, 53	
v +74c	Lefevre		11, 28	<i>Tp(1;3)9E3-4;</i> <i>11B12;80-81</i>	v G64	Pokholkova	EMS	49, 50, 52, 53	
v +75d	Lefevre		11, 17, 22, 45	<i>Tp(1;2)9A2;</i> <i>10C2;40-41</i>	v G73	Pokholkova	EMS	49, 50, 52, 53	
v +	Fox, Yoon	DNA	12-15		v G90	Pokholkova	EMS	49, 50, 52, 53	
v A	Anderson		1-3, 23, 48-50,	<i>T(1;3)10A1-2;</i> <i>93B7-10</i>	v G92	Belyaeva	EMS	51	
			52, 54		v G99	Belyaeva	EMS	51	
v AE111	Belyaeva	EMS	49, 50, 52		v G100	Pokholkova	EMS	49, 50, 52, 53	
v AM1	Pokholkova	EMS	49, 50, 52, 53		v G117	Pokholkova	EMS	49, 50, 52, 53	
v B1	Bgatov	X ray	50, 53		v G118		EMS	53	
v B84		X ray	53		v G119		EMS	53	
v B85		X ray	53		v G121	Pokholkova	EMS	49, 50, 52, 53	
v B86		X ray	53		v G126	Pokholkova	EMS	49, 50, 52, 53	
v B126		X ray	53		v G130	Belyaeva	EMS	51	
v B127		X ray	53		v H2a	Kidwell	HD	37	
v B150		X ray	53		v IE109	Belyaeva	EMS	51	
v B152		X ray	53		v J9	Khudyakov	EMS	49, 50, 52, 53	
v B153		X ray	53		v J20	Belyaeva	EMS	51	
v B154		X ray	53		v J25	Khudyakov	EMS	49, 50, 52, 53	
v BN	Zhimulev	EMS	49, 50, 52, 53		v J26	Khudyakov	EMS	50	
v DK		EMS	49, 50, 52, 53		v J27	Khudyakov	EMS	50, 53	
v dpG1	Zhimulev	EMS	50, 52, 53		v J28	Khudyakov	EMS	50, 53	
v dpZ1	Baritcheva	EMS	49, 50, 52, 53		v J29	Khudyakov	EMS	50, 53	
v dpZ2	Baritcheva	EMS	49, 50, 52, 53		v k		X ray	6, 33, 35, 37, 38, 41, 46	
v dpZ7	Baritcheva	EMS	49, 50, 52, 53		v L1	Lefevre	X ray	12, 17, 24, 50, 54	<i>T(1;3)3E1-2;</i> <i>90C9-11</i> <i>+ Df(1)10A1-2;</i> <i>10A4-5</i>
v E1	Schalet	EMS	6, 35		v L8	Lefevre	X ray	22	<i>T(1;2)9D3-4;</i> <i>10A1-2;25E-F</i> <i>+ T(1;3)3E5;62D</i>
v E37	Belyaeva	EMS	49, 50, 51, 53		v L13	Lefevre	X ray	24	<i>T(1;3)9B13-14;</i> <i>10A1-2;64F</i>
v E57	Belyaeva	EMS	49, 50, 51, 52, 53		v M4	Kochneva	X ray	50, 53	
v E63	Belyaeva	EMS	49, 50, 51, 52, 53		v M8	Kochneva	X ray	50, 53	
v E70	Belyaeva	EMS	49, 50, 51, 52, 53		v N48	Schalet	Neutron	45	<i>Tp(1;1)9F;</i> <i>10C3-5;20</i>
v E73	Belyaeva	EMS	51		v NK	Zhimulev	EMS	49, 50, 52, 53	
v E76	Belyaeva	EMS	49, 50, 52, 53		v NN	Zhimulev	EMS	49, 50, 52, 53	
v E78	Belyaeva	EMS	49, 50, 51, 52, 53		v Of	Offerman	X ray	31, 53	<i>In(1)dl49</i>
v E82	Belyaeva	EMS	49, 50, 52, 53		v OS	Zhimulev	EMS	49, 50, 52, 53	
v E84	Belyaeva	EMS	49, 50, 52, 53		v P30		X ray	53	
v E107	Belyaeva	EMS	49, 50, 52, 53		v P31		X ray	53	
v E110	Belyaeva	EMS	49, 50, 52, 53		v P32		X ray	53	
v E115	Belyaeva	EMS	51		v P33		X ray	53	
v E118	Belyaeva	EMS	49, 50, 52, 53		v P34		X ray	53	
v E119	Belyaeva	EMS	49, 50, 52, 53		v P35		X ray	53	
v E124	Belyaeva	EMS	49, 50, 52, 53		v P36		X ray	53	
v E128	Belyaeva	EMS	51		v s15-1	Golubovsky		50, 53	
v E129	Belyaeva	EMS	49, 50, 52		v s16	Golubovsky	spont	50, 51, 53	
v E143	Belyaeva	EMS	51		v tm1			50, 53	
v E146	Belyaeva	EMS	49, 50, 53		v tm2			50, 53	
v E147	Belyaeva	EMS	51		v ts		EMS	10	
v E154	Belyaeva	EMS	49, 50, 52, 53		v u β	Pastink	ENU	31a	
v E158	Belyaeva	EMS	49, 50, 52, 53						
v E160	Belyaeva	EMS	49, 50, 52, 53						
v E184	Belyaeva	EMS	49, 50, 52, 53						
v E195	Belyaeva	EMS	49, 50, 53						
v ESB	Belyaeva	EMS	49, 50, 53						
v F1	Pokholkova	EMS	53						
v F2	Pokholkova	EMS	53						
v F3	Pokholkova	EMS	53						
v F4	Pokholkova	EMS	53						
v F5	Pokholkova	EMS	53						
v F6	Pokholkova	EMS	53						
v F7	Pokholkova	EMS	53						
v F8	Pokholkova	EMS	53						
v F9	Pokholkova	EMS	53						
v F10	Pokholkova	EMS	53						
v F11	Pokholkova	EMS	53						
v F12	Pokholkova	EMS	53						
v F13	Pokholkova	EMS	53						
v F14	Pokholkova	EMS	53						
v F15	Pokholkova	EMS	53						
v F16	Pokholkova	EMS	53						
v F17	Pokholkova	EMS	53						
v F303	Pokholkova	EMS	50, 53						
v F308	Pokholkova	EMS	50, 53						
v F364	Pokholkova	EMS	50, 53						
v G50	Belyaeva	EMS	51, 52						

^α 1 = Anderson, 1925, Papers Mich. Acad. Sci. 5: 355-66; 2 = Anderson, 1926, Papers Mich. Acad. Sci. 7: 273-78; 3 = Anderson, 1929, Z. Indukt. Abstamm. Vererbungsl. 51: 397-411; 4 = Baglioni, 1959, Nature (London) 184: 1084-85; 5 = Baglioni, 1960, Heredity 15: 87-96; 6 = Baillie and Chovnick, 1971, Mol. Gen. Genet. 112: 341-53; 7 = Barish and Fox, 1956, Genetics 41: 45-57; 8 = Brehme and Demerec, 1942, Growth 6: 351-56; 9 = Busson, Gans, Komitopoulou, and Masson, 1983, Genetics 105: 309-325; 10 = Camfield and Suzuki, 1973, Genetics 74: s37; 10a = CP627; 11 = Craymer and Roy, 1980, DIS 55: 200-04; 12 = Fox, 1948, DIS 22: 53; 13 = Fox, 1949, Genetics 34: 647-64; 14 = Fox and Valencia, 1973, Genetics 74: s83; 15 = Fox and Yoon, 1970, Proc. Nat. Acad. Sci. USA 67: 1608-16; 16 = Gans, Audit, and Masson, 1975, Genetics 81: 683-704; 17 = Geer, Lischwe, and Murphy, 1983, J. Exp. Zool. 225: 107-18; 18 = Green, 1949, Genetics 34: 564-72; 19 = Green, 1952, Proc. Nat. Acad. Sci. USA 38: 300-05; 20 = Green, 1954, Proc. Nat. Acad. Sci. USA 40: 92-99; 21 = Kaufman, 1962, Genetics, 47: 807-17; 22 = Lefevre, 1969, Genetics 63: 589-600; 23 = Lefevre, 1970, DIS 45: 39; 24 = Lefevre, 1971, Genetics 67: 497-513; 25 = Marzluf, 1965, Genetics 52: 503-12; 26 = Meyer

and Edmondson, 1951, DIS 25: 74; 27 = Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. No. 237: 27 (fig.); 28 = Mortin and Lefevre, 1981, Chromosoma 82: 237-47; 29 = Najera, 1984, DIS 60: 241-42; 29a = Najera, 1985, DIS 61: 215; 29b = Najera, 1986, DIS 63: 167; 30 = Nissani, 1975, Genet. Res. 26: 63-72; 31 = Offerman, 1935, DIS 3: 28; 31a = Pastink, Vreeken, Nivard, Searles, and Vogel, 1989, Genetics 123: 123-29; 32 = Rizki and Rizki, 1963, J. Cell Biol. 17: 87-89; 33 = Rizki and Rizki, 1968, Genetics 59: 477-85; 34 = Rizki, Soliman, Rizki, Friedman, and Healy, 1970, Genetics 64: 459-69; 35 = Schalet, 1971, DIS 46: 135; 36 = Schultz and Bridges, 1932, Am. Nat. 66: 323-32; 37 = Searles and Voelker, 1986, Proc. Nat. Acad. Sci. USA 83: 404-08; 38 = Shapard, 1954, Genetics 39: 992-93; 39 = Sturtevant, 1932, Proc. Intern. Congr. Genet. 6th, Vol. 1: 304-07; 40 = Sturtevant and Beadle, 1939, An Introduction to Genetics, W.B. Saunders Co., Philadelphia, p. 64; 41 = Tartof, 1969, Genetics 62: 781-95; 42 = Tatum and Beadle, 1939, Biol. Bull. 77: 415-22; 43 = Tobler, Bowman, and Simmons, 1971, Biochem. Genet. 5: 111-17; 44 = Voelker, Chang, Huang, and Wisely, 1984, Genetics 107: s111-12; 45 = Voelker, Wisely, Huang, and Gyurkovics, 1985, Mol. Gen. Genet. 201: 437-45; 46 = Walker, Howells, and Tearle, 1986, Mol. Gen. Genet. 202: 102-07; 47 = Wehner, Gartenmann, and Jungi, 1969, J. Insect Physiol. 15: 815-23; 48 = Zhimulev, Belyaeva, Khudyakov, and Pokholkova, 1980, DIS 55: 211; 49 = Zhimulev, Belyaeva, Pokholkova, Kochneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1981, DIS 56: 192-96; 50 = Zhimulev, Belyaeva, Pokholkova, Kochneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1982, DIS 58: 210-14; 51 = Zhimulev, Belyaeva, Semeshin, Bgatov, and Baritcheva, 1980, Genetika 16: 1404-24; 52 = Zhimulev, Pokholkova, Bgatov, Semeshin, and Belyaeva, 1981, Chromosoma, 82: 25-40; 53 = Zhimulev, Pokholkova, Bgatov, Umbetova, and Belyaeva, 1987, DIS 66: 194-9; 54 = Zhimulev, Semeshin, and Belyaeva, 1981, Chromosoma, 82: 9-23.

β Twenty-five alleles; see table in molecular biology.

cytology: Left part of the 10A1-2 band between the X breakpoint of $T(1;Y)BI49 = T(1;Y)10A1-2:YL$ and the proximal break of $Df(1)v-L4 = Df(1)9F5-6;10A1-2$ (Green, 1954, Proc. Nat. Acad. Sci. USA 40: 92-99; Lefevre, 1969, Genetics 63: 589-60; Zhimulev, Belyaeva, Pokholkova, Kochneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1982, DIS 58: 210-14). Also located in 10A by *in situ* hybridization (Searles and Voelker, 1986).

molecular biology: Gene cloned using the v^{H2a} allele with a P element insertion (Searles and Voelker, 1986); v^+ allele also cloned. A 1.4 kb transcript is present in v^+ RNA. Mutations that disrupt the wild-type expression of v are grouped within approximately 2 kb of DNA. Five mutant alleles previously located on the genetic map by fine structure mapping (Schalet, 1971, DIS 46: 135-36; Baillie and Chovnick, 1971, Mol. Gen. Genet. 112: 341-53) have been located on the molecular map of v (Searles and Voelker, 1986). The suppressible alleles v^1 , v^2 , and v^k , inseparable by recombination, appear to be identical insertions of the transposable element 412. v^{36f} , mapping about 0.7 kb to the right of v^1 , is a *roo* insertion. v^{48a} , which lies to the left of v^{36f} on the molecular map, is a 200-bp deletion in the same restriction fragment as v^1 . Base-pair changes were observed in 25 ENU mutants that have been cloned and sequenced (Pastink *et al.*, 1989). The following table summarizes the mRNA and amino acid changes in these mutant alleles:

allele	position	mRNA change	amino acid change
v^{u101}	624	GC → AT	gly → ser
v^{u103}	365	GC → AT	trp → UGA
v^{u106}	58	GC → AT	splice

allele	position	mRNA change	amino acid change
$v^{u107\alpha}$	988	AT → GC	his → arg
	1053	AT → GC	ile → val
v^{u108}	1014	GC → AT	arg → cys
v^{u111}	1430	GC → AT	asp → asn
v^{u113}	372	GC → AT	gln → UAG
v^{u114}	838	GC → AT	trp → UAG
v^{u150}	1331	GC → AT	ser → asn
v^{u151}	1048	AT → CG	ile → ser
v^{u152}	468	GC → AT	val → met
$v^{u153\alpha}$	912	AT → TA	lys → UAG
	936	GC → CG	asp → his
$v^{u155\alpha}$	585	GC → AT	asp → asn
v^{u156}	224	GC → TA	gln → his
v^{u158}	1322	GC → AT	ser → phe
v^{u159}	285	GC → AT	his → tyr
v^{u161}	207	AT → TA	lys → UAA
v^{u164}	1126	GC → AT	ser → leu
v^{u166}	1316	AT → GC	leu → pro
v^{u167}	1322	GC → AT	ser → phe
v^{u168}	796	AT → GC	leu → pro
v^{u171}	1017	GC → AT	arg → trp
v^{u174}	1117	AT → GC	asp → gly
v^{u177}	351	GC → AT	splice
v^{u185}	1122	GC → AT	asp → asn

α Two base-pair changes.

other information: Hereditary reversion of the eye color of v mutants to that of wild-type flies by treatment of permeable eggs with DNA (Fox and Yoon, 1970, Proc. Nat. Acad. Sci. USA 67: 1608-15) has been described. Later, v^I was reverted by microinjection of wild-type DNA (Germeraad, 1976).

***Va: Venae abnormeis**

location: 2- (not located).

discoverer: Timoféeff-Ressovsky.

references: 1927, Wilhelm Roux's Arch. Entwicklungsmech. Organ. 109: 70-109 (fig.).

Roelofs, 1937, Genetica 19: 518-36.

phenotype: Veins irregularly branched or interrupted. Heterozygote overlaps wild type in 50% of flies. RK3.

***vac: vacuolated**

location: 1-58.5.

origin: Induced by D-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3026).

discoverer: Fahmy, 1955.

references: 1958, DIS 32: 77.

phenotype: Wings blistered; character varies from small vacuole to involvement of entire wing. At least one wing affected in 95% of flies. Viability and fertility good. RK2.

cytology: Located in 19E7-8.

vacuolar pendunculi: see *vap*

Vacuolar medulla: see *Vam*

vacuolated: see *vac*

Vaja: see *Fs(2)Sz13*

valois: see *vls*

Vam: Vacuolar medulla (J. C. Hall)

location: 1-50.6 (between v and f).

origin: Induced by ethyl methanesulfonate.

references: Heisenberg and Bohl, 1978, Z. Naturforsch. 34: 143-47.

Coombe, 1984, J. Comp. Physiol. 155: 661-72.

1986, *J. Comp. Physiol.* 159: 655-65.

Coombe and Heisenberg, 1986, *J. Neurogenet.* 3: 135-58.

phenotype: Mutants show many vacuoles in the distal part of the medulla. Appearance of vacuoles age-dependent, first appearing in homo- and hemizygotes half an hour after eclosion and occurring in 100% of these mutants after one hour. In older flies, vacuoles are often visible in the lamina and lobula, and occasionally in the central brain. Lamina monopolar neurons L1 and L2 start to degenerate at eclosion and soon afterwards electroretinogram transients disappear. All hemi- or homozygous mutant flies appear nearly blind in tests of movement detection (optomotor response to vertical or horizontal movement and landing response lost). Fixation to a broad stripe has higher light intensity threshold in *Vam* than in wild type (Coombe, 1984). These behavioral defects, but not the anatomical aberrations, have full penetrance (Coombe and Heisenberg, 1986). In heterozygotes, vacuoles make their first appearance in the distal medulla about six days after eclosion and the heterozygous flies show much less lamina degeneration than the homozygotes, the anatomical defects being semi-dominant. Mosaic analysis (Coombe and Heisenberg, 1986) showed the vacuolization to be independent of eye genotype and the degeneration to be sometimes unilateral. Fate mapping leads to a ventral (blastoderm) focus.

alleles: Only one mutant allele, isolation number KS74, which is semi-dominant.

cytology: Probably located to the right of 13A5 since not uncovered by *Df(1)KA9 = Df(1)12E1;13A5*.

vao: varied outspread

location: 1- {64} (between *lf* and *unc*). Symbol originally used by Fahmy for a mutant (now lost) associated with *In(1)vao* (see CP627); now used for the phenotype shown by *Df(1)A118/Df(1)Q539* females lacking 19E7.

discoverer: Fahmy, 1953 (mutant); Schalet and Finnerty, 1970 (deficiency).

references: Fahmy, 1959, *DIS* 33: 94.

Schalet and Finnerty, 1970, *DIS* 45: 77.

Schalet, 1972, *DIS* 49: 36-37.

Schalet and Lefevre, 1973, *Chromosoma* 44: 183-202.

Schalet and Lefevre, 1976, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 848-902.

Miklos, Healy, Pain, Howells, and Russell, 1984, *Chromosoma* 89: 218-27.

Perrimon, Smouse, and Miklos, 1989, *Genetics* 121: 313-31.

phenotype: Wings outspread. Eye color mottled brown. Male sterile. Viability poor.

alleles: No point mutations defined yet (Perrimon *et al.*, 1989).

cytology: Placed in 19E7 since the heterozygous combination of the overlapping deletions *Df(1)A118 = Df(1)19E4-5;19E7-8* and *Df(1)Q539 = Df(1)19E6;19F6-20A1* reveals the *vao* phenotype (Schalet and Finnerty, 1970; Schalet, 1972; Miklos *et al.*, 1984). Flies with the heterozygous combination of *Df(1)B57 = Df(1)19E1-2;19F1* and *Df(1)Q539* show the *vao* wing and eye abnormalities (plus the *unc* phenotype) at 24° - 25°, but the flies have normal eyes at 17° - 18°

(Schalet, 1972).

vap: vacuolar pedunculi (J. C. Hall)

location: 1-54.2.

origin: Induced by ethyl methanesulfonate.

references: Heisenberg, 1980, *Development and Neurobiology of Drosophila* (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York and London, pp. 373-90.

phenotype: Isolated as a neuro-anatomical mutant (Heisenberg and Böhl, 1979, *Z. Naturforsch.* 34: 143-47). Mutant shows vacuolar spaces at a certain depth along the pedunculi of the mushroom bodies as if extrinsic cells are undergoing degeneration. Intrinsic fibers appear continuous. Viability of mutants reduced (Heisenberg).

alleles: One allele, isolation number KS67.

Var^{34k22}: see *bw*^{34k}

Varas: see *Fs(3)Sz24*

variable size and shape: see *vss*

varied outspread: see *vao*

varnished: see *vr*

vas: vasa (T. Schüpbach)

location: 2- {51}.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1986, *Wilhelm Roux's Arch. Dev. Biol.* 195: 302-17.

Nüsslein-Volhard, Frohnhofer, and Lehmann, 1987, *Science* 238: 1675-81.

Hay, Jan, and Jan, 1988, *Cell* 55: 577-87.

Lasko and Ashburner, 1988, *Nature (London)* 335: 611-17.

Schüpbach and Wieschaus, 1989, *Genetics* 121: 101-17.

phenotype: Maternal-effect lethal. Embryos from homozygous mothers exhibit a so-called "grandchildless-knirps" phenotype: all eggs lack polar granules and no pole cells are formed; most of the embryos show large deletions of abdominal segments, whereby anterior parts of segment A1 become fused to posterior parts of segment A8. Telson elements are always present and relatively normal. Eggs have abnormal shape. Analysis of germline clones indicates that the mutation is germline autonomous (Schüpbach and Wieschaus, 1986, *Dev. Biol.* 113: 443-448). Homozygous *vasa* males cannot be distinguished from wild-type males in viability and fertility.

alleles: Seven (*vasa*¹ through *vasa*⁷); isolated as *PD*, *B5*, *D1*, *O11*, *O14*, *Q6* and *Q7*.

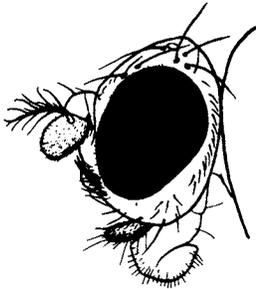
cytology: Placed in 35C1-2 on the basis of molecular information (Lasko and Ashburner, 1988); uncovered by *Df(2L)TE35BC-29 = Df(2L)35A3;35C1*, but not by *Df(2L)64j = Df(2L)34D1-2;35B9-C1*, which deletes sequences distal to *vasa*, or by *Df(2L)TE35D-4 [Df(2L)TE116-GW4]*, which deletes sequences proximal to the gene.

molecular biology: Gene cloned and the cDNA sequenced (Lasko and Ashburner, 1988; Hay *et al.*, 1988); relatively complex, with seven exons and five small introns between 36 and 70 nucleotides in length and one large intron of 3,700 nucleotides. The major transcript is 2.0 kb long and is found in the female germ line and early

embryos only; it codes for a protein of 650 amino acids. The distribution of this protein is generalized before division, but increases at posterior pole as division proceeds (Ashburner). The predicted amino acid sequence is very similar to that of the murine translation initiation factor eIF-4A and the human nuclear antigen p68 (Lasko and Ashburner, 1988). The amino-terminal region carries five tandem heptad repeats (Hay *et al.*, 1988).

vb: vibrissae

location: 1-49.3 (Bridges); 1-54.8 (Waddle, Monk, and Williams).
discoverer: Bridges, 25122.
phenotype: Vibrissae form tufts of bristles beneath eyes. Overlaps wild type. RK2.



vb: vibrissae

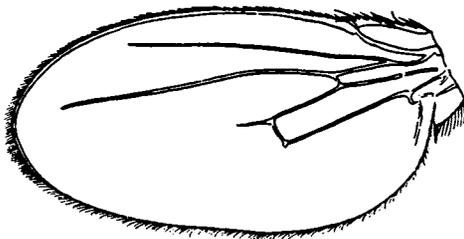
From Bridges and Brehme, 1944, Carnegie Inst. Washington Publ. No. 552: 212.

vb²

origin: X ray induced.
discoverer: Muller, 261.
other information: Associated with *In(1)sx* (Craymer, 1980, DIS 55: 197-200).

***Vc: Vortice**

location: Autosomal.
origin: Spontaneous.
discoverer: Smith, 37c20.
references: Novitski, 1937, DIS 8: 10.
phenotype: Enhances *dpldp* to give phenotype like *hy*. Homozygous lethal. RK3.



ve: veinlet

From Duncan, 1935, Am. Naturalist 69: 94-96.

ve: veinlet (E. Bier)

location: 3-0.2; to the right of *ru* (Roberts and Evans-Roberts, 1979, Genetics 93: 663-79; Robertson and Rivera, 1972, DIS 48: 21).

synonym: *rho: rhomboid*.

references: Duncan, 1935, Am. Nat. 69: 94-96.
 Waddington, 1939, Proc. Nat. Acad. Sci. USA 25: 305.
 1940, J. Genet. 41: 75-139.
 Bertschmann 1955, DIS 29: 69-70.
 Thompson, 1976, Genetics 81: 387-402.
 Thompson and Thoday, 1976, Genetics 83: s76.
 Thompson, 1977, DIS 52: 76.
 Spivey and Thompson, 1984, Genetics 107: s102.
 Jürgens, Weischaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol 193: 283-95.
 Mayer and Nüsslein-Volhard, 1988, Genes Dev. 2: 1496-1511.
 Bier, Jan, and Jan, 1990, Genes Dev 4: 190-203.
 Díaz-Benjumea and García-Bellido, 1990, Wilhelm Roux's Arch. Dev. Biol 198: 336-54.

phenotype: Viable alleles exhibit wing venation defects; strong alleles are embryonic lethal. In flies homozygous for viable alleles the L3, L4, and L5 veins do not reach the wing margins (Duncan; Waddington). Developmentally, veins appear complete in prepupa but distal tips are obliterated during the contraction period (Waddington, 1939, 1940). The shortened-vein phenotype is suppressed by *px* (Waddington), *net*, and *su(ve)*, and is enhanced by *vn*, *H*, *Ax*, *ci*, *tg²*, and *ri* (Waddington; Díaz-Benjumea and García-Bellido, 1990, Wilhelm Roux's Arch. Dev. Biol 198: 336-54.). Vein-specific modifiers, such as *gp*, (Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 208) or *PL(2)L4a* (Thompson, 1976), interact with the effect of *ve* on L4. The L5 vein seldom extends beyond the posterior crossvein. *ve²* is a stronger allele, in which the L2 is also affected (Bertschmann); L2 vein occasionally complete (Thompson, 1976), but other veins do not overlap wild type. Distribution of sense organs (campaniform sensilla and bristles) on L3 is shifted proximally in *ve* (Spivey and Thompson) When a *ve* stock is selected for shortened veins, the F1 produced by mating wild-type males to mutant females show terminal gaps in L5 (Thompson and Thoday, 1976). *ve/ve/+* intersexes are veinlet, whereas *ve/ve/+* triploids are normal, according to Pipkin. Interestingly flies heterozygous for *ve* and strong embryonic lethal alleles display less severe veinlet phenotypes than *ve* homozygotes (Bier *et al.*; Díaz-Benjumea and García-Bellido); furthermore, *ve^{1/ve⁵}* flies appear wild type (Bier, unpublished). Homozygous *ve⁵ embryos exhibit three major types of defects: (1) Dorsoventral defects: Embryos exhibit a deletion of epithelial cells derived from a ventrolateral strip of the blastoderm fate map (i.e., loss of mediolateral cuticular denticles and sensory structures). Other phenotypes resulting from blastoderm patterning defects include failure to complete dorsal closure and development of an abnormal pointed head skeleton (Jürgens *et al.*; Mayer and Nüsslein-Volhard). (2) Midline defects: Mesectodermal cells giving rise to glia and unpaired neurons are abnormal or fail to form. Late developmental consequences include a narrower CNS and pathfinding abnormalities (Mayer and Nüsslein-Volhard). (3) Peripheral-nervous-system defects: Two stretch receptor organs (lateral abdominal chordotonal organs) fail to form in lethal *ve* mutants. The primary chordotonal-organ-precursor cells are likely to be affected since the four progeny sensory-organ cells derived from that precursor cell*

are missing as a group (Bier *et al.*). Other late embryonic defects include loss of longitudinal body-wall muscles, ventrally displaced muscle-attachment sites (Bier *et al.*), and loss of the first row of denticles in abdominal segments (Mayer and Nüsslein-Volhard).

alleles:

allele	origin	discoverer	synonym	ref ^α	comments
ve¹	spont	Duncan, 34a		4	viable; wing veins do not reach margins
ve²	spont	Bertschmann		4	viable; like <i>ve¹</i> but stronger
ve³		Waddle		6	L4 longer than in <i>ve¹</i> ; <i>ve¹/ve³</i> distinguishable from <i>ve¹</i> or <i>ve²</i>
ve⁴	EMS	Jürgens	<i>rho^{7M43}</i>	5	strong allele; embryonic lethal
ve⁵	<i>P-lacw</i> insertion	Bier	<i>rho^{lac1}</i> <i>rho^{6-63a}</i>	2	weak allele; embryonic lethal
ve⁶ β		Bier	<i>rho^{del1}</i> <i>rho^{rev37}</i>	2	strong allele; embryonic lethal
ve⁷ γ		Díaz-Benjumea	<i>ve^{M1}</i>	3	lethal
ve⁸ γ		Díaz-Benjumea	<i>ve^{M2}</i>	3	lethal
ve⁹ γ		Díaz-Benjumea	<i>ve^{M3}</i>	3	lethal
ve¹⁰ γ		Díaz-Benjumea	<i>ve^{M4}</i>	3	lethal
ve¹¹ γ		Díaz-Benjumea	<i>ve^{M5}</i>	3	lethal

^α 1 = Bertschmann, 1955, DIS 29: 69-70; 2 = Bier, Jan, and Jan, 1990, Genes and Dev 4: 190-203; 3 = Díaz-Benjumea and García-Bellido, 1990, Roux's Arch. Dev. Biol 198: 336-54; 4 = Duncan, 1935, Am. Nat. 69: 94-96; 5 = Mayer and Nüsslein-Volhard, 1988, Genes Dev. 2: 1496-1511; 6 = Waddle and Oster, 1973, DIS 50: 23.

β Transposase induced deletion of *P-lacw* from *ve⁴*.

γ EMS or X rays?

cytology: 62A by *in situ* hybridization with a *rho* probe (Bier *et al.*). Also placed in 61B-62D interval on the basis of *ve*'s being covered by *Dp(3;Y)H141 = Dp(3;Y)61B;62D* (Jürgens *et al.*).

molecular biology: Genomic clones have been obtained by plasmid rescue of *P-lacw* from *ve⁴* flies (Bier *et al.*). Sequence analysis of cDNA clones corresponding to a 2.5 kb mRNA indicates the mature mRNA is comprised of a 5' noncoding exon and two coding exons. An open reading frame encodes a predicted membrane protein of 355 amino acids. The pattern of *ve* transcription, as monitored by whole mount tissue *in situ* hybridization, correlates well with the spatial requirement for *ve* function: (1) Blastoderm expression includes two longitudinal strips of cells corresponding in location to the domain of the fate map dependent on *ve* function for proper dorso-ventral patterning. Expression in the cells corresponding to head and dorsal cells may also coincide with cells involved in head skeleton formation and dorsal closure. (2) As ventral furrow formation proceeds expression becomes limited to a single row of mesectodermal cells which give rise to the glial and neuronal cells along the ventral midline which are affected in lethal mutant embryos. (3) *ve* is expressed in a single cell per hemisegment at the germband extended stage, which is likely to be the precursor for the chordotonal organs missing in lethal mutants. When the germ band is nearly retracted, *ve* is expressed in rows of cells at the anterior margin of each body segment. This expression may correlate with loss of the first row of denticles in lethal mutants.

vein: see *vn*

Vein: see *Vn*

vein off: see *iab^{vno}* under *BXC*

veinlet: see *ve*

veins longitudinally shortened: see *vli*

***Vel: Velvet**

location: 1- or 3- (rearrangement).

discoverer: Patterson, 1933.

phenotype: Hairs on eyes conspicuous. RK3A.

cytology: Associated with *T(1;3)Vel*; breakpoints unknown.

***ven: venation**

location: 3- [right arm associated with *In(3R)P*].

origin: Spontaneous.

discoverer: Bridges, 33g18.

references: 1937, DIS 7: 17.

Bridges and Bridges, 1938, Genetics 23: 111-14.

phenotype: Veins irregularly thickened and branched, especially L3 and crossveins. Eyes bulging and bright. Bristles gnarled. Body small. Often sterile. RK3A.

Ven: see *Fs(3)Sz31*

Venae abnormeis: see *Va*

venation: see *ven*

Vencellin: see *Fs(3)Sz31*

ventral nervous system defective: see *vnd*

ventral veins lacking: see *vvl*

venula: see *vnl*

vermillion: see *v*

verthandi: see *vtd*

vertical wing: see *vtw*

verticals: see *vt*

***ves: vestigium**

location: 1-1.4.

origin: Induced by L-p-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 77.

phenotype: Wings abnormal, vary from small and curved to almost normal with cut-away inner margins. Eyes slightly rough and abnormally shaped. Male infertile; viability about 50% normal. RK2.

alleles: One allele induced by CB. 3025.

vesiculated: see *vs*

vestar: see *vst*

vestigial: see *vg*

vestigium: see *ves*

vg: vestigial

location: 2-67.0.

references: Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 150.

Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet.

2: 59, 232.

Chen, 1929, *J. Morphol.* 47: 135-99.

Mohr, 1932, *Proc. Int. Congr. Genet.*, 6th, Vol. 1: 190-212.

Goldschmidt, 1935, *Biol. Zentralbl.* 55: 535-54.

Auerbach, 1936, *Trans Roy. Soc. Edinburgh* 58: 787-815.

Harnly, 1936, *J. Exp. Zool.* 74: 41-59.

Goldschmidt, 1937, *Univ. Calif. Publ. Zool.* 41: 277-82.

Morgan, Bridges, and Schultz, 1938, *Year Book - Carnegie Inst. Washington* 37: 305-06.

Waddington, 1939, *Proc. Nat. Acad. Sci. USA* 25: 299-307.

Green and Oliver, 1940, *Genetics* 25: 584-92.

Waddington, 1940, *J. Genet.* 41: 75-139.

Green, 1946, *Genetics* 31: 1-20.

Waddington, 1953, *J. Genet.* 51: 243-58.

Fristrom, 1968, *J. Cell Biol.* 39: 488-91.

1969, *Mol. Gen. Genet.* 103: 363-79.

David, Javellot, and Touzet, 1970, *DIS* 45: 33.

Bryant and Girton, 1980, *Development and Neurobiology of Drosophila* (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York and London, pp. 109-27.

Carson, Ferriola and Schuchman, 1980, *DIS* 55: 23-24.

Girton and Bryant, 1980, *Dev. Biol.* 77: 233-43.

Bownes and Roberts, 1981a, *J. Embryol. Exp. Morphol.* 65 (Suppl.): 49-76.

1981b, *Differentiation* 18: 89-96.

James and Bryant, 1981, *Dev. Biol.* 85: 39-84.

Simpson, Lawrence, and Maschat, 1981, *Dev. Biol.* 84: 206-11.

O'Brochta and Bryant, 1983, *Wilhelm Roux's Arch. Dev. Biol.* 192: 285-94.

Lasko and Pardue, 1988, *Genetics* 120: 495-502.

Williams and Bell, 1988, *EMBO J.* 7: 1355-63.

Williams, Pappu, and Bell, 1988a, *Mol. Cell. Biol.* 8: 1489-97.

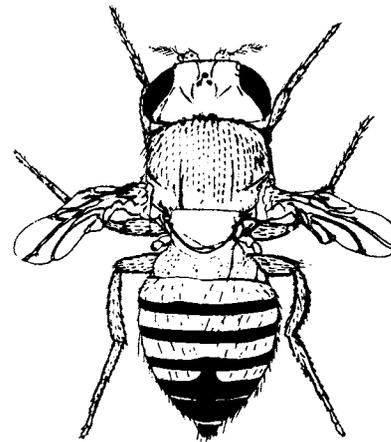
1988b, *Mol. Gen. Genet.* 212: 370-74.

Williams, Atkin, and Bell, 1990, *Mol. Gen. Genet.* 221: 8-16.

Williams, Scott, Atkin, Brooks, Russell, and Bell, 1990, *Genetics* 25: 833-44.

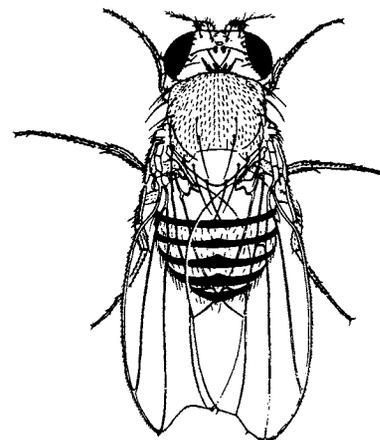
phenotype: The vestigial locus seems to be mainly involved in the development of the wing margin. The mutants are recessive viable (with or without a visible phenotype), recessive lethal, or dominant (with a visible phenotype over wild type or a *vg* allele); some alleles complement each other; others show pleiotropic effects or homeosis (Bownes and Roberts, 1981). In the classical *vg* mutants, the wings of homozygotes are reduced to vestiges and usually held at right angles to the body, the wing veins still visible. Some mutants have narrow, nicked, or scalloped wings. Halteres may be reduced or absent. Postscutellar bristles are frequently held erect. Viability is somewhat reduced; null mutants are sterile. Temperatures of 29° or greater appreciably increase wing size (Harnly, 1936, *Genetics* 21: 84-103; Stanley, 1935, *J. Exp. Zool.* 69: 459-95). A suppressor of *vg* on the third chromosome, *su(vg)*, results in an almost normal phenotype at 28°, an intermediate *vg* phenotype at 25°, and a strong *vg* phenotype (in wings and especially halteres) under 20° (David, *et al.*, 1970). *vg/+* heterozygotes with certain Minutes show scalloping of the wings (Green and Oliver, 1940; Simpson *et al.*, 1981). *vg/vg/+*

has scalloped wings more often than *vg/+* (Green, 1946). Final size of larva is smaller than in wild type and pupation occurs slightly later. Wing disks of late larva are also somewhat smaller than in wild type (Auerbach, 1936), as are haltere disks (Chen, 1929). Goldschmidt (1935, 1937) claimed that wings are more or less fully formed and subsequently eroded by degeneration during pupation. Waddington (1939, 1940) found no evidence of erosion and concluded that the effect of the gene occurs during the larval period and involves reduction in size of prospective wing area and shift in position of line along which wing area is folded out from the imaginal disk. Fristrom (1968, 1969), however, using both light and electron microscopy, found numerous degenerating cells in the presumptive wing blade region of the *vg* wing disks, as did Bryant and Girton (1980), Bownes and Roberts (1981a, 1981b), James and Bryant (1981), and O'Brochta and Bryant (1983). Duplications of the mesonotum along with deficiencies of wing disk material occur in a small percentage of *vg* mutants (Girton and Bryant, 1980; James and Bryant, 1981).



vg¹: vestigial

From Bridges and Morgan, 1919, *Carnegie Inst. Wash. Publ.* No. 278: 148.



vg^{no}: vestigial-notched

From Mohr, 1932, *Proc. Intern. Congr. Genet.* 6th Vol. 1: 190-212.

alleles: Mutant *vg* alleles are listed in the following tables. *vg* deficiencies are described in the rearrangement section.

Table I

symbol	name	origin	discoverer	synonym	ref ^α	comments
<i>vg</i> ¹	vestigial	spont	Morgan, 101	<i>vg</i> ^{BG}	2, 5, 7, 8, 38-40, 45-47	wings, halteres reduced; small wing disks
* <i>vg</i> ²		spont	Ward, 1920		43	wings, halteres absent or reduced to knobs; low viability; females and males (usually) sterile
* <i>vg</i> ¹¹		X ray	Ruch, 1931		26	wings, halteres smaller than in <i>vg</i> ¹ ; some wing nicks in <i>vg</i> ¹¹ /+; fertile
<i>vg</i> ¹²		γ ray	Lasko	<i>vg</i> ^{82h10}	18, 45-46	like <i>vg</i> ¹ ; <i>vg</i> ¹² /Df inviable
<i>vg</i> ¹⁷		EMS	Lasko	<i>vg</i> ^{82E28}	18	like <i>vg</i> ¹
<i>vg</i> ¹⁸		γ ray	Lasko	<i>vg</i> ^{82h11}	18, 45	like <i>vg</i> ¹
<i>vg</i> ²¹		HD			45-48	cytotype-dependent allele; homozygote normal; weak <i>vg</i> over <i>vg</i> ¹
* <i>vg</i> ^{33k}		spont	Ives, 33k30		30, 31	like <i>vg</i> ^{No1}
<i>vg</i> ³⁴		EMS	Lasko	<i>vg</i> ^{82h15}	18	like <i>vg</i> ¹
* <i>vg</i> ^{37g}		spont	Poulson, 37g		32, 33	weak allele; slight nick in wing; P higher in male
* <i>vg</i> ^{40b}		spont	Ives, 40b		15	like <i>vg</i> ¹ ; <i>vg</i> ¹ /+ semidominant for <i>vg</i>
* <i>vg</i> ^{40c}		spont	Buzzati-Traverso, 40c20		6	like <i>vg</i> ¹
* <i>vg</i> ^{48a}		spont	King, 48a1		34	15% of homozygotes have wing nicks; halteres and post-scutellars normal; <i>vg</i> ¹ / <i>vg</i> ^{31h25} show wing scallops
<i>vg</i> ^{51h25}		spont like <i>vg</i> ¹ but wings larger	Ives, 51h25		16, 39, 45	
<i>vg</i> ^{67d1} - <i>vg</i> ^{85f3}						see Table II
<i>vg</i> ^{72a}		spont			41	like <i>vg</i> ¹
<i>vg</i> ^{79f}		spont	Ashburner		1	like <i>vg</i> ¹
<i>vg</i> ¹⁶⁸					45	recessive lethal
* <i>vg</i> ^a	vestigial- antlered	spont	Morgan, 12j		25	wings long, but narrow and scalloped; halteres and post-scutellars normal; wings straplike and smaller in females female sterile; no eggs
* <i>vg</i> ^{Cli}	vestigial- Clipped	spont	Robertson, Reeves, 1947		3	
* <i>vg</i> ^{dn}	vestigial- double notch	spont (from <i>vg</i> ¹)	Nolte, 1942		29	two notches on wing; halteres, postscutellars normal; <i>vg</i> ^{dn} / <i>vg</i> ¹ has straplike wings
<i>vg</i> ^{E7}		EMS			7	normal as homozygote; <i>vg</i> ^{E7} / <i>vg</i> ¹ straplike wings
* <i>vg</i> ^h	vestigial- hemithorax	spont (from <i>vg</i> ¹)	Ludwig, 1936		19, 20, 37	20% show hemithorax; most mutants like <i>vg</i> ¹ , 10-20% like <i>vg</i> ^{nw}
* <i>vg</i> ^{hR}	vestigial- hemithorax of Reck	spont (from <i>vg</i> ¹)	Reck, 1937		35	like <i>vg</i> ^h ; <i>In(2R)49C1-2;50C1-2</i>
<i>vg</i> ^{ms}	vestigial- more sensitive		Nakashina- Tanaka, 1967		28	small wings at 25°, normal at 27-30°
* <i>vg</i> ⁿ	vestigial- nick	spont	Bridges, 15h7		25	homozygote normal; <i>vg</i> ⁿ / <i>vg</i> ¹ shows nicks in 85% of females, 45% of males
<i>vg</i> ^{nG}	vestigial- nick of Green	spont (from <i>vg</i> ¹)	Green, 40j26		11, 12	homozygote normal (occasional wing nicks); enhanced by Minutes; <i>vg</i> ^{nG} / <i>vg</i> ¹ has scalloped wings
<i>vg</i> ⁿⁱ	vestigial- nick of Green	spont (from <i>vg</i> ¹)	Mohr, 1926		10, 12, 24, 38, 45	homozygote normal; enhanced by Minutes; 1/4 of <i>vg</i> ⁿⁱ / <i>vg</i> ¹ show wings nicks
<i>vg</i> ⁿⁱ²		spont	Mohler, 55b9		23	homozygote normal; nicks or scalloping in heterozygotes over <i>vg</i> ¹ , <i>vg</i> ^{nw} , <i>vg</i> ^{nwl}
<i>vg</i> ^{no}	vestigial- notched	spont (from <i>vg</i> ¹)	Mohr, 1926		24	homozygote notched (45%); <i>vg</i> ^{no} /+ normal; <i>vg</i> ^{no} / <i>vg</i> ¹ has ragged wings (100%)
* <i>vg</i> ^{No1}	vestigial- Notch	heat- treatment of parents	Swigert, 31c	<i>vg</i> ^{31c}	30, 31	homozygote shows vestiges of wings; postscutellars erect; viability 50%; female sterile; terminal nicks in <i>vg</i> ^{No1} /+ like <i>vg</i> ^{No1} ; <i>vg</i> ^{No2} /+ enhanced by Minutes; lethal over <i>vg</i> ^{nw}
<i>vg</i> ^{No2}		spont	Plough, 31j	<i>vg</i> ^{31j}	7, 12, 30, 31, 38, 40, 45	
<i>vg</i> ^{np}	vestigial- nipped	spont	Wallace, 38a5		7, 10, 38, 39, 42, 45, 46	terminal incisions in wings; overlaps wild type at 25°, not at 19°. Wing buds show scalloping.
<i>vg</i> ^{nw}	vestigial- no wing	spont	Morgan, 1924		7, 8, 24, 27, 38, 40, 45, 46	tiny knobbed wings and halteres, erect postscutellars; low viability; female usually sterile; occasional wing nicks in <i>vg</i> ^{np} /+; also shortened L2 vein in <i>vg</i> ^{np} /+; small wing buds
* <i>vg</i> ^{nwl}	vestigial-	spont	Mohler, 55b9		23	homozygous lethal; <i>vg</i> ^{nwl} / <i>vg</i> ^{nw} like <i>vg</i> ^{nw} ,

THE GENOME OF *DROSOPHILA MELANOGASTER*

symbol	name	origin	discoverer	synonym	ref ^α	comments
<i>vg^P</i>	no wing lethal vestigial-pennant	spont (from <i>vg^I</i>)	Kerr, 30a15		13, 14	sterile; <i>vg^{nwl}/+</i> has wing nicks (50%) homozygotes usually normal; <i>vg^P/vg^I</i> has narrow straplike wings, larger in male (temperature-sensitive)
* <i>vg^{PK}</i>	vestigial-pennant of Kutschera	spont (from <i>vg^I</i>)	Kutschera, 1955		21, 22	homozygote normal (17°-28°); <i>vg^{PK}/vg^I</i> has small wings at 17°, straplike wings at 25°, normal at 30°.
* <i>vg^{R1}</i>		γ ray	Lasko		18	like <i>vg^I</i>
* <i>vg^S</i>	vestigial-strap	spont (from <i>vg^I</i>)	Morgan, 12d		25	wings narrow, straplike, full length, held at right angles; halteres reduced; postscutellars normal.
* <i>vg^{S2}</i>		spont	Williams, 56c		44	wings incised, narrow, of variable length.
<i>vg^{Uβ}</i>	vestigial-Ultra	γ ray	Ives, 55131		17, 39 45, 46	homozygous lethal; dominant; <i>vg^U/+</i> has much reduced wings and halteres; <i>vg^U/vg^I</i> has tiny wings, no halteres
<i>vg^{Wγ}</i>	vestigial-wingless	X ray	Shukla		4, 36 45, 46 49	homozygous lethal; dominant with homeotic effect; <i>vg^W/+</i> is wingless or has tiny buds, no halteres (or one); female fertile and viable; <i>In(2R)47F15-48A12;49E4-5</i> (Ashburner)
<i>vg^X</i>		γ ray	Erk, Podraza, 1986		9	wings, halteres tiny knobs (sometimes halteres absent); postscutellars erect; defects in legs, abdominal tergites, notum; female sterile; notching or scalloping in heterozygotes.

^α 1 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91; 2 = Auerbach, 1936, Trans R. Soc. Edinburgh 58: 787-815; 3 = Beatty, 1949, Proc. R. Soc. Edinburgh, B 63: 249-70; 4 = Bownes and Roberts, 1981, J. Embryol. Exper. Morphol. 65 (Suppl.): 49-7; 5 = Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 150; 6 = Buzzati-Traverso, 1940, DIS 13: 49; 7 = Carlson, Ferriola, and Schuchman, 1980, DIS 55: 23; 8 = Chen, 1929, J. Morphol. 47: 135-99; 9 = Erk and Podraza, 1986, DIS 63: 161; 10 = Giesel, 1984, Genetics 107: s37; 11 = Green, 1941, DIS 14: 39; 12 = Green, 1946, Genetics 31: 1-20; 13 = Harnly, 1935, DIS 4: 14; 14 = Harnly, 1936, J. Exp. Zool. 74: 41-59; 15 = Ives, 1941, DIS 14: 39; 16 = Ives, 1952, DIS 26: 65; 17 = Ives, 1956, DIS 30: 72-73; 18 = Lasko (personal communication); 18a = Lasko and Pardue, 1988, Genetics 120: 495-502; 19 = Ludwig, 1936, Verh. Dtsch. Zool. Ges. 38, Zool. Anz. Suppl. 9: 21-73; 20 = Ludwig, 1937, DIS 7: 18; 21 = Mainx, 1956, DIS 30: 77; 22 = Mainx, 1957, Z. Indukt. Abstamm. Vererbungsl. 88: 286-88; 23 = Mohler, 1959, DIS 33: 98; 24 = Mohr, 1932, Proc. Int. Congr. Genet., 6th, Vol. 1: 190-212; 25 = Morgan and Bridges, 1919, Carnegie Inst. Washington Publ. No. 278: 211; 26 = Morgan, Bridges, and Schultz, 1938, Year Book - Carnegie Inst. Washington 37: 305-06; 27 = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 59, 232; 28 = Nakashima-Tanaka, 1967, Genetica 38: 447-58, 459-70; 29 = Nolte, 1944, DIS 18: 44; 30 = Plough and Ives, 1934, DIS 1: 32-33; 31 = Plough and Ives, 1935, Genetics 20: 42-69; 32 = Poulson, 1938, DIS 10: 55; 33 = Poulson '939, DIS 12: 49; 34 = Poulson and King, 1948, DIS 22: 55; 35 = Reck, 1937, DIS 8: 10; 36 = Schukla, 1980, DIS 55: 210; 37 = Schultz, 1938, Wilhelm Rou Arch. Entwicklungsmech. Org. 138: 69-102.; 38 = Silber, 1980, Genetica 54: 91-99; 39 = Silber and Becker, 1981, Genetica 55: 217-20; 40 = Silber and Goux, 1977, Arch. Zool. Exp. Gen. 118: 471-80; 41 = Thompson and Purcell, 1972, DIS 48: 16; 42 = Waddington, 1940, J. Genet. 41: 75-139; 43 = Ward, 1923, Genetics 8: 286-300; 44 = Williams, 1956, DIS 30: 80; 45 = Williams and Bell, 1988, EMBO J. 7: 1355-63. 46 = Williams, Atkin, and Bell, 1990, Mol. Gen. Genet. 221: 8-16. 47 = Williams, Pappu, and Bell, 1988, Mol. Cell. Biol. 8: 1489-97. 48 = Williams, Pappu, and Bell, 1988, Mol. Gen. Genet. 212: 370-74. 49 = Williams, Scott, Atkin, Brooks, Russell, and Bell, 1990, Genetics 25: 833-44.

^β Associated with *In(2R)vg^U* = *In(2R)49C1-2;50C1-2* (Ratty and Lindsley, 1964, DIS 38: 30). Survivors of *vg^U/Df(2R)vg* (50%) show extreme mutant phenotype. Partial revertant of *vg^U* reported by Silber and Lemeunier (1981).

^γ Associated with *In(2R)vg^W* = *In(2R)47F15-48A12;49E4-5* (Ashburner).

Table II

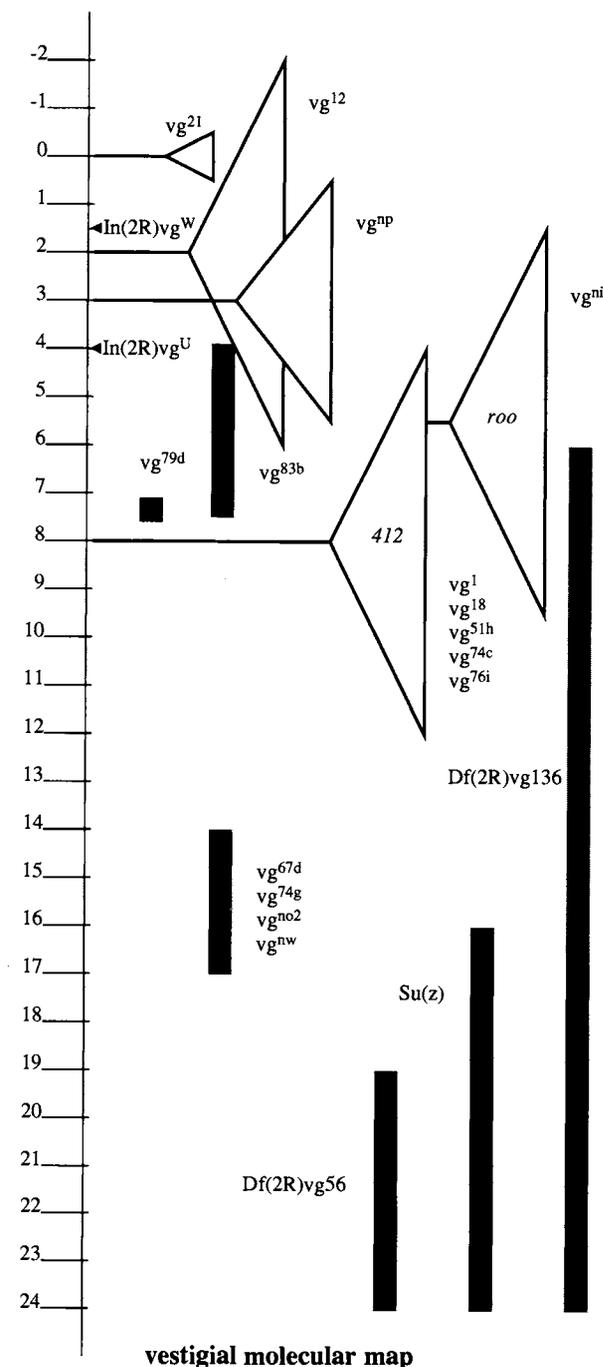
allele ^α	origin	Phenotype of homozygote ^β					cytology
		wing phen.	halteres	bristles	viab.	fert.	
<i>vg^{67d1}</i>	γ ray				-		<i>T(2;3)49C2-D2;93E-F1</i>
<i>vg^{67d2}</i>	βγ						
<i>vg^{71k2}</i>	γ ray	<i>vg^{nw}</i>	-	-	+	-	
* <i>vg^{72a1}</i>	γ ray						
<i>vg^{74b1}</i>	γ ray						<i>In(2R)44C2-5;49D2-E1</i>
<i>vg^{74b2}</i>	C+γ ray						<i>In(2LR)37F-38A1;49D2-E1</i>
* <i>vg^{74c1}</i>	C+γ ray	<i>vg^{nw}</i>	-	-	+	+	
<i>vg^{74c4}</i>	C+γ ray	<i>vg^{nw}</i>	-	-	+	-	
<i>vg^{74c5}</i>	γ ray	<i>vg^{nw}</i>	-	-	+	-	<i>In(2LR)22A5-B1;49D2-E</i>
<i>vg^{74c5}</i>	γ ray	<i>vg^S</i>	+/-	+	+	+	
<i>vg^{74c6}</i>	γ ray	<i>vg^{nw}</i>	-	-	+	-	
<i>vg^{74c7}</i>	γ ray	<i>vg^{nw}</i>	-	-	+	-	
<i>vg^{76d1}</i>	C+γ ray	<i>vg</i>	-	-	+	+	
<i>vg^{76d2}</i>	C+γ ray	<i>vg</i>	-	-	+	+	
<i>vg^{76f}</i>	γ ray	<i>vg</i>	-	-	+	+	<i>T(Y;2)58B+Dp(2R)58B-D</i>
<i>vg⁷⁶ⁱ¹</i>	AD+γ ray						<i>T(2;3)49D2-E1;84E2-3</i>
<i>vg⁷⁶ⁱ²</i>	AD+γ ray	<i>vg</i>	-	-	+	+	
<i>vg^{76j1}</i>	AD+γ ray	<i>vg</i>	-	-	+	+	
<i>vg^{76k2}</i>	AD+γ ray						<i>Tp(2;2)49D;60B;60A;50F;49E;60C</i>
<i>vg^{77a4}</i>	AD+γ ray						
<i>vg^{77d1}</i>	AD+γ ray	<i>vg^{np}</i>	+	+	+	-	
<i>vg^{77d2}</i>	X ray						<i>In(2LR)25C-D;49D2-E1</i>
<i>vg^{77d2}</i>	X ray	<i>vg^S</i>	-	-	+	-	<i>Tp(2;2)49B4;49B5-12;49D2-E</i>
<i>vg^{78a1}</i>	γ ray	<i>vg^{no}</i>	+	+/-	+	+	<i>In(2R)41D-E;49D3-E1</i>
<i>vg^{78a2}</i>	γ ray	<i>vg</i>	-	-	+	-	<i>In(2R)49D3-E;56E</i>

allele α	origin	Phenotype of homozygote β				fert.	cytology
		wing phen.	halteres	bristles	viab.		
vg 78b1	γ ray	vg ^{nw}	-	-	+	-	
vg 78b2	γ ray	vg ^{nw}	-	-	+	+	
vg 78b3	γ ray	vg	-	-	+	+	
vg 78b4	NaF+ γ ray		-	-	-	-	T(2;3)49D2-3;49E7-F1;80C
vg 78f2	NaF+ γ ray	vg	-	-	+	-	
vg 78j1	γ ray	vg ^{nw}	-	-	+	-	Tp(2;2)49D2-3;49D7-E1;50C1-6
vg 78j3	γ ray		-	-	-	-	In(2R)41D2-E1;49D2-E1
vg 78k2	NaF+ γ ray	vg	-	-	+	+	
vg 78k3	γ ray		-	-	-	-	In(2R)49D2-E1;59D4-8
vg 79b1	γ ray	vg	-	-	+	+	
vg 79b4	X ray		-	-	-	-	In(2R)41C-D;49D2-E1
vg 79b6	X ray		-	-	-	-	Tp(2;2)49D2;50C9-14;49E1
vg 79d3	X ray		-	-	-	-	In(2R)41A;49D2-E2
vg 79d4	X & γ ray	vg ^{nw}	-	-	+	-	In(2R)41E;49D2-E1
vg 79d5 γ	γ ray & neutrons	vg ^{np}	+	+	+	+	
vg 79d6	X ray	vg ^{nw}	-	-	+	-	
vg 79d7	X ray	vg ^{nw}	-	-	+	-	In(2R)41D-E;49D3-F1
vg 79f1	NaF+ γ ray		-	-	-	-	Tp(2;2)36D;53F;49E;41A;54A;41A
vg 79f2	γ ray	vg ^{nw}	-	-	+	-	In(2R)49D2-E1;49E7-F13
vg 79h1	γ ray	vg ^{nw}	-	-	+	-	
vg 79h4	γ ray		-	-	-	-	In(2LR)24D;49D1-E1
vg 79h5	γ ray	vg	-	-	+	-	In(2R)49D2-E;50A2-3
vg 79h6	γ ray	vg ^{nw}	-	-	+	-	In(2R)41E-F;49D2-E1+In(2R)42B2-3;57F-58A1
vg 79h7	γ ray	vg ^{nw}	-	-	+	-	In(2R)49D2-7;49F10-13
vg 8012	X ray	vg ^{nw}	-	-	+	-	
vg 81a	γ ray		-	-	-	-	T(2;3)49D2-E1;64B2-12
vg 81a-s	γ ray	vg ^{s2}	+/-	+/-	+	-	
vg 81b1	γ ray		-	-	-	-	In(2R)48C4-D1;49D2-E
*vg 81b2	γ ray	vg ^{nw}	-	-	+	+	
vg 81c13	γ ray	vg	-	-	+	+	
vg 81c18	γ ray	vg ^{nw}	-	-	+	-	
vg 81c28	γ ray	vg ^{nw}	-	-	+	-	In(2R)41D-E;49D3-E7
vg 81c41d	γ ray		-	-	-	-	In(2R)49C2;49F14
vg 81f	X ray	vg	-	-	+	+	
vg 81k1	γ ray	vg ^{nw}	-	-	+	-	
vg 81i18	γ ray	vg	-	-	+	-	In(2LR)36C4-D1;49D2-F1
vg 81i24	γ ray	vg	-	-	+	+	
vg 81i26	γ ray	vg ^{nw}	-	-	+	-	
vg 82c13	252 Cf	vg	-	-	+	+	
vg 82c14	252 Cf	vg ^{nw}	-	-	+	-	In(2LR)36C-D;49D2-E
vg 82c61	252 Cf		-	-	-	-	In(2LR)24E2-F1;49D2-E7
vg 83b22	γ ray	vg ^{nw}	-	-	+	-	
vg 83b24	γ ray	vg ^{nw}	-	-	+	-	
vg 83b27 γ	γ ray	vg ^{nw}	-	+	+	+	
vg 83b39	γ ray	vg ^{nw}	-	-	+	-	In(2R)49D2-E;51D2-6
vg 83c	γ ray	vg ^{np}	-	+/-	+	-	T(2;3)49D2-E;65F6-66A
vg 83c3	γ ray	vg ^{nw}	-	-	+	-	In(2R)41C-D;49D2-E1
vg 83c5	γ ray	vg ^{nw}	-	-	+	-	
vg 83c7	γ ray	vg ^{nw}	-	-	+	-	
vg 83c24	γ ray	vg ^{nw}	-	-	+	-	
vg 83c42	γ ray	vg ^s	-	-	+	-	
vg 83c43	γ ray	vg ^{nw}	-	-	+	-	In(2R)43C2-3;49D2-E
vg 83c45	γ ray	vg	-	-	+	-	
vg 83d	252 Cf	vg ^{nw}	-	-	+	-	
vg 83d4	252 Cf	vg ^{nw}	-	-	+	-	In(2R)48E2-F1;49D2-E1
vg 83f38	γ ray		-	-	-	-	In(2R)49;59D-E
vg 83f-XD	X ray	vg ^{nw}	-	-	+	-	In(2R)48E2-F1;49D2-E1
vg 83i2b	X ray		-	-	-	-	In(2R)41D-E;49D2-E1
vg 83l-s	X ray		-	-	-	-	
vg 84f γ	X ray	vg ^{nw}	-	-	+	-	
vg 84f51	X ray	vg ^{nw}	-	-	+	-	In(2R)44F2-45A1;49D2-E2
vg 84hXC	γ ray		-	-	-	-	In(2R)48E6-F1;49D2-E1
vg 84hXD	γ ray	vg ^{nw}	-	-	+	-	In(2R)41B-C;49D2-E1
vg 85b	X ray	vg ^{np}	+	+	+	+	
vg 85d1	X ray	vg ^{nw}	-	-	+	-	
vg 85d2	X ray	vg ^{nw}	-	-	+	-	T(2;3)49D2-E;84F4-6
vg 85e2	γ ray	vg ^{nw}	-	-	+	-	Tp(2;2)41B;49E;55F
vg 85e3	γ ray	vg ^{nw}	-	-	+	-	
vg 85e4	γ ray	vg ^{nw}	-	-	+	-	
vg 85f3 γ	γ ray	vg	-	-	+	+	

α Reference for all alleles: Alexandrov and Alexandrova, 1987, DIS 66: 185-87. Other information: Cf = caffeine; AD = actinomycin-D; bristles = postscutellars.

β Wings classified according to similarity to known alleles; for halteres and postscutellar bristles, "-" = absence and "+" = presence; under fertility, "-" = sterile, sex unspecified, but probably female.
 γ Other references: Williams and Bell, 1988, EMBO J. 7: 1355-63; Williams, Pappu, and Bell, 1988, Mol. Cell Biol. 8: 1489-97; Williams, Atkin, and Bell, 1990, Mol. Gen. Genet. 221: 8-16.

cytology: Placed in 49D2-E1 based on breakpoints common to rearrangements in Table II.



(modified from Williams and Bell, 1988 by D. Conner). Heavy lines represent deficiencies; hatched line represents an inverted segment accompanying *Df(1)vg136*.

molecular biology: The vestigial region has been cloned by *P* element transposon tagging (Williams and Bell, 1988). Transcript of 3.8 kb obtained (Williams, Atkin, and Bell, 1990). The proximal breakpoints of *In(2R)vg^U* and *In(2R)vg^W* were located at 49D-F by *in situ* hybridization of salivaries. Alleles placed on the molecular map of the *vg* region derived from Oregon-R are listed in the

following table:

alleles	map location (kb) α	comments
<i>vg¹</i>	+8	412 insertion (8 kb)
<i>vg¹²</i>	+3	insertion (8 kb)
<i>vg¹⁸</i>	+8	412 insertion (8 kb)
<i>vg²¹</i>	0	insert hybridizing to <i>P</i> sequences
<i>vg^{51h25}</i>	+8	412 insertion (8 kb)
<i>vg^{62d2}</i>	+14 to +17	deletion
<i>vg^{74c5}</i>	+8	412 insertion (8 kb)
<i>vg⁷⁶ⁱ²</i>	+8	412 insertion (8 kb)
<i>vg^{79d5}</i>	+7 to +8	deletion
<i>vg^{83b27}</i>	+5 to +8	deletion (3 kb)
<i>vgⁿⁱ</i>	+8 (in 412 of <i>vg¹</i>)	<i>roo</i> insertion (8 kb)
<i>vg^{No2}</i>	+14 to +17	deletion
<i>vg^{np}</i>	+4	insertion (8 kb)
<i>vg^{nw}</i>	+14 to +17	deletion (3 kb)
<i>vg^{Uβ}</i>	+4 (proximal break)	inversion
<i>vg^{Wγ}</i>	+2 (proximal break)	inversion

α "-" values to left (proximal), "+" values to right (distal); 0 coordinate assigned to insert position of *vg²¹*.
 β Fusion between *mam* and *vg* (second intron) (Williams *et al.*, 1990).
 γ Fusion between *inv* and *vg* (first intron) (Williams *et al.*, 1990).

Vi: see *M(1)1B*

Viability: see *M(1)1B*

vibrissae: see *vb*

vin: *vin*

location: 3-36.3 (not allelic to *rs²*).

origin: Spontaneous.

discoverer: Periquet, 72c.

references: Anxolabehere and Periquet, 1973, DIS 50: 21.

Akam, Roberts, Richards, and Ashburner, 1978, Cell 13: 215-25.

Ashburner, Richards, and Velissariou, 1980, DIS 55: 196.

phenotype: Eye color reddish brown, paler in young flies.

Eyes become dark brown with age. *v;vin* flies have pale orange eyes. Ocelli, adult testis sheath, and Malpighian tubules colored. Viability and fertility excellent. RK1.

alleles: Five alleles described in the following table. Deficiencies for *vin* listed in the rearrangement section.

allele	origin	discoverer	ref α	comments
<i>vin¹</i>	spont	Periquet	2	
<i>vin¹⁸</i>	X ray		1	
<i>vin¹⁰¹</i>	γ ray		1	lethal; <i>T(Y;3)87F12-14</i>
<i>vin¹²³</i>	γ ray		1	
<i>vin^M</i>	γ ray		1	

α 1 = Akam, Roberts, Richards, and Ashburner, 1978, Cell 13: 215-25; 2 = Anxolabehere and Periquet, 1973, DIS 50: 21.

cytology: Located in 68C8-68D3 (Ashburner *et al.*, 1980); included in *Df(3L)vin6 = Df(3L)68C8-11;69A4-5* and *Df(3L)vin66 = Df(3L)68A3;68D3*.

vlr: *virilizer*

location: 2-103.3.

origin: Induced by ethyl methanesulfonate.

discoverer: Schüpbach.

references: Amrein, Gorman, and Nöthinger, 1988, Cell 55: 1025-35;

Hilfiker, Amrein, and Nöthiger, 1988, Crete.

phenotype: A new undescribed gene involved in sex determination. Female sterile; may be temperature sensitive.

alleles: Six alleles, including *vir*²⁵⁰, *vir*^{H2}, *vir*^{ts}.

cytology: Placed in 59D2-8.

molecular biology: Region cloned.

Vitelline membrane: see *Vm*

***vli: veins longitudinally shortened**

location: 3- (not located).

origin: Spontaneous.

discoverer: Buchman, 1936.

references: 1937, DIS 8: 8.

phenotype: Veins L2, L4, and L5 tend to be shortened. Overlaps wild type. Semidominant. RK3.

vls: valois (T. Schüpbach)

location: 2-53.

origin: Induced by ethyl methanesulfonate.

references: Schüpbach and Wieschaus, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 302-17.

Nüsslein-Volhard, Frohnhofer, and Lehmann, 1987, Science 238: 1675-81.

Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

phenotype: Maternal-effect lethal. Embryos from homozygous mothers exhibit a so-called "grandchildless-knirps" phenotype: all eggs lack polar granules and no pole cells are formed; most of the embryos show variable deletions of abdominal segments; whereby segment A4 is deleted most frequently, larger deletions may remove segments A2 through A7; in extreme cases anterior parts of segment A1 become fused to posterior parts of segment A8, but telson elements are always present and relatively normal. In addition, 80-90% of the embryos fail to cellularize normally at the blastoderm stage and die without forming cuticle, or only fragmented pieces of cuticle. Analysis of germline clones indicates that the mutation is germline autonomous (Schüpbach and Wieschaus, 1986, Dev. Biol. 113: 443-48).

alleles: *vls*¹-*vls*⁴ isolated as *RB*, *PE*, *PG*, *HC*.

cytology: Placed in 38A6-E9 based on its inclusion in *Df(2L)TW2 = Df(2L)37D2-E1;38E6-9*, but not *Df(2L)TW50 = Df(2L)36E4-F1;38A6-7*.

Vm: Vitelline membrane

The *Vm* genes make up a multigene family that includes at least four members and is responsible for encoding the major proteins of the first layer of the *Drosophila* eggshell, the vitelline membrane. These proteins are synthesized during the later stages of oogenesis in the follicular epithelium of mature ovaries. The following table describes the four vitelline membrane genes that have been identified and described.

gene	location	synonym	ref ^α	cytology ^β	comments	
					mRNA	protein
<i>Vm26Aa</i> ^γ	2- {20}	<i>Vm23</i>	1, 3-7	26A	700-800 bp	17.5 kd
<i>Vm26Ab</i> ^{γδ}	2- {20}		3-5	26A	700-800 bp	23 kd
<i>Vm32Ea</i> ^ε	2- {44}	<i>Vm3</i>	4, 7	32E	460 bp	13 kd
<i>Vm32Ec</i> ^ε	2- {44}		2	32E	430 bp	13kd
<i>Vm34Ca</i>	2- {47}	<i>Vm2</i>	4, 7	34C	650 bp	

^α 1 = Burke, Waring, Popodi, and Minoo, 1987, Dev. Biol. 124: 441-50; 2 = Gigliotti, Graziani, De Ponti, Rafti, Manzi, Lavorgna, Gargiulo, and Malva, 1989, Dev. Genet. 10: 33-41; 3 = Higgins, Walker, Holden, and White, 1984, Dev. Biol. 105: 155-65; 4 = Mindrinos, Scherer, Garcini, Kwan, Jacobs, and Petri, 1985, EMBO J. 4: 147-53; 5 = Popodi, Minoo, Burke, and Waring, 1988, Dev. Biol. 127: 248-56; 6 = Savant and Waring, 1989, Dev. Biol. 135: 43-52; 7 = Scherer, Harris, and Petri, 1988, Dev. Biol. 130: 768-88.

^β Determined by *in situ* hybridization to the salivaries (Higgins *et al.*, 1984; Mindrinos *et al.*, 1985; Burke *et al.*, 1987; Gigliotti *et al.*, 1989). *fs(2)QJ42*, a mutant of *Vm26Ab*, was mapped cytologically to 25D-26A by using the chromosome *Df(2L)c17 = Df(2L)25D7-E1;26A7-8* (Savant and Waring, 1989).

^γ Single long open reading frame; no introns. Transcription occurs from opposite DNA strands in the two genes at 26A (Popodi *et al.*, 1988).

^δ 10- to 15-fold reduction in transcript level in the mutant *fs(2)QJ42*. This mutant can be rescued by germline transformation with *Vm26Ab*⁺ and restored to normal production of vitelline membrane protein and female fertility (Popodi *et al.*, 1988).

^ε Uninterrupted open reading frame (Gigliotti *et al.*, 1989).

molecular: The vitelline membrane genes listed in the table have been cloned and their nucleotide and predicted amino-acid sequences determined (Mindrinos *et al.*, 1985; Burke *et al.*, 1987; Popodi *et al.*, 1988; Scherer *et al.*, 1988; Gigliotti *et al.*, 1989). Clones show cross-hybridization (Mindrinos *et al.*, 1985). High abundance, ovary-specific transcripts have been obtained, but only during vitelline membrane synthesis in adult females in stages 9 and 10 of oogenesis (Higgins *et al.*, 1984; Mindrinos *et al.*, 1985; Gigliotti *et al.*, 1989). A central 114 bp conserved region (the *Vm* domain) occurs in the coding region of three and probably four of these genes (Scherer *et al.*, 1988). At the nucleic acid level, this conserved region shows the following sequence identities: 91% between *Vm26Aa* and *Vm34C*, 79% between *Vm26Ab* and *Vm34C*, and 77% between *Vm26Aa* and *Vm26Ab*. *Vm26Aa* and *Vm34C* show 100% amino acid identity in the VM domain, but *Vm26Ab* and *Vm34C* show only 86% amino acid identity (Scherer *et al.*, 1988). These three *Vm* genes as well as *Vm32E*, which contains at least a portion of the VM domain (Scherer *et al.*, 1988), encode translation products with the high proline and alanine content characteristic of vitelline membrane proteins (Mindrinos *et al.*, 1985; Gigliotti *et al.*, 1989).

vn: vein

location: 3-16.2.

origin: X ray induced.

discoverer: Puro, 1960.

references: 1982, DIS 58: 205-08.

Díaz-Benjumea, González-Gaitán, and García-Bellido, 1989, Genome 31: 612-19.

phenotype: Large section of vein L4 and anterior crossvein missing. Posterior crossvein often incomplete; gap sometimes present in L3. Male sterile, female fertile.

alleles: Six (Díaz-Benjumea *et al.*, 1989).

other information: Not tested for allelism with *Vn*, a dominant mutant of similar map position and phenotype which has been lost.

***Vn: Vein**

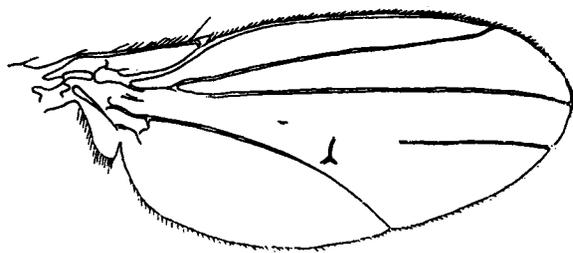
location: 3-19.6.

origin: Spontaneous.

discoverer: Mohr, 28j21.

references: 1932, Proc. Intern. Congr. Genet., 6th., Vol. 1: 190-212.

1938, Avh. Nor. Vidensk.-Akad. Oslo, Mat. Naturvi-



vn: vein

Right wing of *vn* (Puro, 1982)

densk. Kl. 4: 1-7.

Mohr and Mossige, 1942, Avh. Nor. Vidensk.-Akad. Oslo, Mat. Naturvidensk. Kl. 7: 1-51.

phenotype: Vein L4 not complete. Wings slightly spread. Fly smaller than normal. Homozygous lethal. RK2A.

cytology: Associated with *Df(3L)vn = Df(3L)64C12-D1;65D2-E1*.

***vnd*: ventral nervous system defective**

location: 1-0.0.

references: White, 1980, Dev. Biol. 80: 332-44.

Lefevre, 1981, Genetics 99: 461-80.

White, Decelles, and Enlow, 1983, Genetics 104: 433-48.

Campos, Grossman, and White, 1985, J. Neurogenet. 2: 197-218.

Mason, Voelker, Rosen, Campos, White, and Lim, 1986, DIS 63: 164-65.

Jiménez and Campos-Ortega, 1987, J. Neurogenet. 4: 179-200.

Rosen, Martin-Morris, Luo, and White, 1989, Proc. Nat. Acad. Sci. USA 86: 2478-82.

phenotype: Late embryonic lethal. Ventral nervous system disorganized and not condensed. Mutants have fewer cells in the CNS; pattern of neuronal connections damaged (Jiménez and Campos-Ortega, 1987). alleles:

allele	origin	discoverer	synonym	ref ^α
<i>vnd</i> 1	EMS	White		3
<i>vnd</i> 2	EMS	White		3
<i>vnd</i> 3	EMS	White	<i>vnd</i> 2	3
<i>vnd</i> 4	EMS	White		3
<i>vnd</i> 5	EMS	White		3
<i>vnd</i> 6	EMS	Lim	<i>l(1)EC6</i> ⁰⁰⁴	
<i>vnd</i> 7	EMS	Lim	<i>l(1)EC6</i> ²⁵	
<i>vnd</i> 8	EMS	Lim	<i>l(1)EC6</i> ⁸³	
<i>vnd</i> 9	EMS	Lim	<i>l(1)EC6</i> ⁸⁴	
<i>vnd</i> 10	EMS	Lim	<i>l(1)EC6</i> ¹⁰¹	
<i>vnd</i> 11	EMS	Lim	<i>l(1)EC6</i> ¹⁰⁵	
<i>vnd</i> 12	EMS	Lim	<i>l(1)EC6</i> ¹¹⁶	
<i>vnd</i> 13	EMS	Lim	<i>l(1)EC6</i> ¹³⁸	
<i>vnd</i> 14	EMS	Lim	<i>l(1)EC6</i> ¹⁴²	
<i>vnd</i> 15	MMS	Lim	<i>l(1)EC6</i> ^{M15}	

allele	origin	discoverer	synonym	ref ^α
<i>vnd</i> 16	MMS	Lim	<i>l(1)EC6</i> ^{M22}	
<i>vnd</i> 17	TEM	Lim	<i>l(1)EC6</i> ^{T008}	
<i>vnd</i> 18	X ray	Lefevre	<i>l(1)A131</i>	1
<i>vnd</i> 19 β	X ray	Lefevre	<i>l(1)GA100</i>	1
* <i>vnd</i> 20	X ray	Lefevre	<i>l(1)GA122</i>	1
* <i>vnd</i> 21	X ray	Lefevre	<i>l(1)HC143</i>	1
* <i>vnd</i> 22	X ray	Lefevre	<i>l(1)HC218</i>	1
<i>vnd</i> 23	X ray	Lefevre	<i>l(1)RC24</i>	1
<i>vnd</i> 24	EMS	Lefevre	<i>l(1)EA142</i>	2
<i>vnd</i> 25	EMS	Lefevre	<i>l(1)VA208</i>	2
<i>vnd</i> 26	EMS	Lefevre	<i>l(1)VE769</i>	2
<i>vnd</i> 27	spont	Schalet	<i>l(1)2-43</i>	
<i>vnd</i> 28	ENU	Voelker	<i>l(1)A22</i>	
<i>vnd</i> 29	ENU	Voelker	<i>l(1)A55</i>	

^α 1 = Lefevre, 1981, Genetics 99: 461-80; 2 = Lefevre and Watkins, 1986, Genetics 113: 869-95; 3 = White, Decelles, and Enlow, 1983, Genetics 104: 433-48.

^β *T(1;3)1B6;88A*.

cytology: Placed in 1B9 since included in *Df(1)yT8* but not in *Df(1)yT7* or *Df(1)yT9* (Mason *et al.*, 1986); not covered by *Dp(1:f)24 = Dp(1:f)1B5;19-20*.

molecular biology: The genomic DNA in which the *vnd* gene resides was cloned by breakpoint analysis (White).

****vnl*: venula**

location: 2- (not located).

origin: Spontaneous.

discoverer: Plaine, 50h.

references: 1951, DIS 25: 77.

phenotype: Extra veins between L3 and L4 largely between anterior and posterior crossveins; some also arise from L4 distal to posterior crossvein. Penetrance in male 1.3%, in female 43%. With *Sb*, penetrance is 63% in female; expressivity also increased. RK3.

Vno: see *iab6*^{Vno} under *BXC*

vo-3: see *e(dp^v)*

vortex in chromosome 3: see *e(dp^v)*

Vortice: see *Vc*

****vr*: varnished**

location: 3-44.

discoverer: Mohr, 20j22.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 237.

phenotype: Eyes small, have fused facets. Female sterile. RK2.

***vs*: vesiculated**

location: 1-16.3.

references: Mohr, 1927, Hereditas 9: 173.

Evang, 1925, Z. Indukt. Abstamm. Vererbungsl. 39: 165-83 (fig.).

Waddington, 1939, Proc. Nat. Acad. Sci. USA 25: 299-307.

phenotype: Wings warped, wrinkled, blistered, rough textured, discolored, and divergent. Phenotype may overlap wild type. Abnormalities may result from breakage of fibers that normally hold wing surfaces together during unfolding (Waddington, 1939).

alleles:

allele	origin	discoverer	ref ^α	comments
<i>vs</i> 1	spont	Mohr, 24c23	2, 7	see phenotype
* <i>vs</i> 29c	X ray	Oliver, 29c9	9	penetrance:

allele	origin	discoverer	ref ^α	comments
				100% at 25° - 95% (males) and 88% (females) at 30° β
*vs ^{52a}	p 32	King, 52a	3	viability: 40%
vs ^{61j} γδ		Mickey, 61j	5, 6	
*vs ^{64j}	X ray	Mayo, 1964	4	penetrance: 77%; one or both wings crumpled, partially expanded, may be blistered
vs ^{66a}	X ray	Becker	1	fertility, viability good
vs ^{66j}	X ray	Nilsson, Valentin	8	penetrance: 93% at 25°
vs ⁶⁷¹	EMS	Rockwell, 1967	10	penetrance: 100% at 19°, 25°; blistering variable

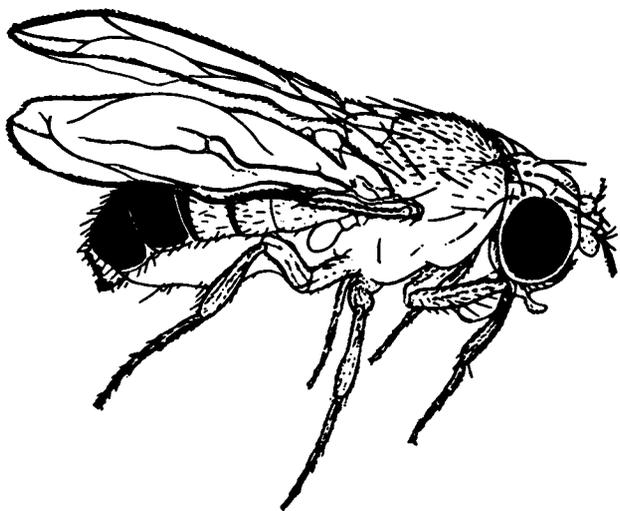
α 1 = Becker, 1968, DIS 43: 59; 2 = Evang, 1925, Z. Indukt. Abstamm. Vererbungsl. 39: 165-83 (fig.); 3 = King, 1952, DIS 26: 65; 4 = Mayo, 1966, DIS 41: 58; 5 = Mickey, 1963, DIS 38: 28; 6 = Mickey, 1964, DIS 39: 58; 7 = Mohr, 1927, Hereditas 9: 173; 8 = Nilsson and Valentin, 1968, DIS 43: 61; 9 = Oliver, 1937, DIS 7: 19; 10 = Rockwell, 1967, DIS 44: 52-53.

β Green, 1939, DIS 11: 45.

γ Found among progeny of male treated with radio frequency.

δ Synonym: *bw*^{61j}; *bubble wing 61j*; *bu-w*^{61j}.

cytology: Salivary chromosome location at about 6B1 (Lefevre).



vs: vesiculated

From Evang, 1925, Z. Indukt. Abstamm. Vererbungsl. 39: 165-83.

vss: variable size and shape

location: 3-.

origin: Induced by ethyl methanesulfonate.

discoverer: Nüsslein-Volhard.

references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

phenotype: Maternal effect female-sterile. Produces eggs of variable size and shape.

alleles: *vss*¹ and *vss*² isolated as 258 and 2675.

*vst: vestar

location: 2-4.3.

discoverer: Glass, 41i15.

references: 1944, DIS 18: 40.

phenotype: Wings small and straplike, variable. Eyes small, very rough, and somewhat glazed. Female sterile. Viability low. RK3.

vt: verticals

location: 1-2.3.

origin: Synthetic.

discoverer: Gersh.

references: 1965, Genetics 51: 477-80.

phenotype: Anterior vertical, anterior dorsocentral, and anterior scutellar bristles often missing, verticals being most likely to be affected. RK2.

cytology: Placed in 3C5-6 on the basis of the *vt* phenotype of the following genotypes: *Df(1)rst*² = *Df(1)3C3-4;3C6-7*; the heterozygote between *Df(1)rst*² and the synthetic deficiency for 3C5 and 6 produced by combining the 4^P*X*^D element of *T(1;4)w*²⁵⁸⁻¹⁸ = *T(1;4)3C4-5;101* and the *X*^{P4}^D element of *T(1;4)N*²⁶⁴⁻¹² = *T(1;4)3C6-7;101F*; and the synthetic deficiency for 3C5 and 6 produced by combining the 4^P*X*^D element of *T(1;4)w*²⁵⁸⁻¹⁸ with a recombinant between *In(1LR)l-v139* = *In(1LR)3C6-7* and the right end of a normal *X* chromosome.

other information: Not known as a point mutation.

vtd: verthandi (J.A. Kennison)

location: 3-46.

origin: Induced by ethyl methanesulfonate.

discoverer: Kennison, 1983.

references: Kennison and Tamkun, 1988, Proc. Nat. Acad. Sci. USA 85: 8136-40.

phenotype: Recessive. Suppresses extra sex combs in *Pc*^{4/+} male flies.

alleles:

allele	origin
*vtd ¹	EMS
vtd ²	EMS
vtd ³	γ ray
vtd ⁴	γ ray
vtd ⁵	γ ray

vtw: vertical wing (J.C. Hall)

location: 1-18 (between *cv* and *sn*).

origin: Induced by ethyl methanesulfonate.

discoverer: Butler.

references: Deak, 1977, J. Embryol. Exp. Morphol. 40: 35-63.

Deak, Bellamy, Bienz, Dubuis, Fenner, Gollin, Rähmi, Ramp, Reinhardt, and Cotton, 1982, J. Embryol. Exp. Morphol. 69: 61-81.

phenotype: Flightless, with a penetrance of 85-100%; cannot jump more than 2 cm. Wings held up vertically (penetrance 63-90%). In some flies, large amount of disorganized fibrillar muscle material present in the thorax; in others, most of the fibrillar muscles are absent (Deak *et al.*, 1980). Both dorsal longitudinal muscles (DLMs) and dorsoventral muscles (DVMs) are reduced; in the electron microscope, myofibrillar structure appears disorganized and Z-band structure disturbed (Deak *et al.*, 1982), although the indirect flight muscle fibers that do

form are superficially normal. The abnormal behavioral and muscular phenotypes are thought to be due to a primary defect in embryonic cells that map (in mosaic experiments) to the dorsal region of the blastoderm. Viability of mutant alleles is about 95% (Deak, 1977).

alleles: Two recessive alleles, *vtw*¹ (stronger allele) and *vtw*² (weaker allele). Penetrance of flightless phenotype 100% for *vtw*¹, 85% for *vtw*²; penetrance of vertical wing phenotype 90% for *vtw*¹, 63% for *vtw*² (Deak, 1977).

***vvl*: ventral veins lacking**

Location: 3- (rearranged).

references: Díaz-Benjumea, González-Gaitán, and García-Bellido, 1989, Genome 31: 612-19.

phenotype: Prevents differentiation of longitudinal veins 2 and 4 which form on the ventral surface of the wing; in *Hw*; *vvl* flies, extra sensillae form in the ventral surface where veins would ordinarily form.

alleles: Three.

w: white**location:** 1-1.5.**references:** Morgan, 1910, *Science* 32: 120-22.Morgan and Bridges, 1916, *Carnegie Inst. Washington Publ. No. 237*: 25, 28, 51.Muller, 1932, *Proc. Intern. Congr. Genet.* 6th, Vol. 1: 234.Muller, 1935, *J. Genet.* 30: 407-14.Beadle and Ephrussi, 1936, *Genetics* 21: 230.Sturtevant and Beadle, 1939, *An Introduction to Genetics*, W.B. Saunders Co., Philadelphia, p.64 (fig).Brehme and Demerec, 1942, *Growth* 6: 351-56.Hadorn and Mitchell, 1951, *Proc. Nat. Acad. Sci. USA* 37: 650-65.Lewis, 1952, *Proc. Nat. Acad. Sci. USA* 38: 953-61.MacKendrick and Pontecorvo, 1952, *Experientia* 8: 390-91.Lewis, 1956, *Genetics* 41: 651.Judd, 1957, *Genetics* 42: 379-80.1958, *Proc. Intern. Congr. Genet.*, 10th, Vol. 2: 137.Ziegler-Günder and Hadorn, 1958, *Z. Indukt. Abstamm. Vererbungsl.* 89: 235-45.Green, 1959a, *Heredity* 113: 303-15.1959b, *Proc. Nat. Acad. Sci. USA* 45: 549-53.1959c, *Z. Indukt. Abstamm. Vererbungsl.* 90: 375-84.Judd, 1959, *Genetics* 44: 34-42.Sullivan and Sullivan, 1975, *Biochem. Genet.* 13: 603-13.Green, 1976, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.), Academic Press, London, New York, San Francisco, Vol. 1b, pp. 929-46.Judd, 1976, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.), Academic Press, London, New York, San Francisco, Vol. 1b, pp. 767-99.Gehring and Paro, 1980, *Cell* 19: 897-904.Bingham, 1981, *Cold Spring Harbor Symp. Quant. Biol.* 45: 519-25.Bingham and Judd, 1981, *Cell* 25: 705-11.Bingham, Levis, and Rubin, 1981, *Cell* 25: 693-704.Collins and Rubin, 1982, *Cell* 30: 71-79.Goldberg, Paro, and Gehring, 1982, *EMBO J.* 1: 93-98.Levis, Bingham, and Rubin, 1982, *Proc. Nat. Acad. Sci. USA* 79: 564-68.Levis and Rubin, 1982, *Cell* 30: 543-50.Zachar and Bingham, 1982, *Cell* 30: 529-41.O'Hare, Levis, and Rubin, 1983, *Proc. Nat. Acad. Sci. USA* 80: 6917-21.Pirrota, Hadfield, and Pretorius, 1983, *EMBO J.* 2: 927-34.Fjose, Polito, Weber, and Gehring, 1984, *EMBO J.* 3: 2087-94.Gehring, Klemenz, Weber, and Kloter, 1984, *EMBO J.* 3: 2077-85.Hazelrigg, Levis, and Rubin, 1984, *Cell* 36: 469-81.Levis, O'Hare, and Rubin, 1984, *Cell* 38: 471-81.O'Hare, Murphy, Levis, and Rubin, 1984, *J. Mol. Biol.* 180: 437-55.Pirrota and Bröckl, 1984, *EMBO J.* 3: 563-68.Carbonara and Gehring, 1985, *Mol. Gen. Genet.* 199: 1-6.Chapman and Bingham, 1985, *DIS* 61: 48-50.Davison, Chapman, Wedeen, and Bingham, 1985, *Genetics* 110: 479-94.Levis, Hazelrigg, and Rubin, 1985, *EMBO J.* 4: 3487-

99.

Pirrota, Steller, and Bozzetti, 1985, *EMBO J.* 4: 3501-08.Rubin, Hazelrigg, Karess, Laski, Lavery, Levis, Rio, Spencer, and Zuker, 1985, *Cold Spring Harbor Symp. Quant. Biol.* 50: 529-33.Steller and Pirrota, 1985, *EMBO J.* 4: 3765-72.Zachar, Chapman, and Bingham, 1985, *Cold Spring Harbor Symp. Quant. Biol.* 50: 337-46.Bingham and Chapman, 1986, *EMBO J.* 5: 3343-51.Davis, Shen, and Judd, 1987, *Proc. Nat. Acad. Sci. USA* 84: 174-78.Hazelrigg, 1987, *TIG* 3: 43-47.Judd, 1987, *Structure and Function of Eukaryotic Chromosomes* (Hennig, ed.), Springer-Verlag, Berlin, Heidelberg, pp. 81-94.Mount, 1987, *Nature* (London) 325: 487.Pastink, Schalet, Vreeken, Parádi, and Eeken, 1987, *Mutat. Res.* 177: 101-15.Dreesen, Johnson, and Henikoff, 1988, *Mol. Cell Biol.* 8: 5206-15.Miyashita and Langley, 1988, *Genetics* 120: 119-212.Mount, Green, and Rubin, 1988, *Genetics* 118: 221-34.Pastink, Vreeken, and Vogel, 1988, *Mutat. Res.* 199: 47-53.Birchler and Hiebert, 1989, *Genetics* 122: 129-38.Birchler, Hiebert, and Rabinow, 1989, *Genes Dev.* 3: 73-84.Rabinow and Birchler, 1989, *EMBO J.* 8: 879-89.Tearle, Belote, McKeown, Baker, and Howells, 1989, *Genetics* 122: 595-606.Gubb, Ashburner, Roote, and Davis, 1990, *Genetics* 126: 167-76.

phenotype: The white locus is involved in the production and distribution of ommochrome (brown) and pteridine (red) pigments found in the compound eyes and ocelli of adult flies as well as the pigments in adult testis sheaths and larval Malpighian tubules; the specific function of the protein it encodes is still unknown, but it is believed to be a membrane-associated ATP-binding transport protein for pigment precursors in both the ommochrome and pteridine pathways (Sullivan and Sullivan, 1975; Mount, 1987; Dreesen *et al.*, 1988; Tearle *et al.*, 1989). w^1 was the first mutant found in *Drosophila melanogaster* (Morgan, 1910; Morgan and Bridges, 1916). Mutant alleles do not appreciably affect the viability and fertility of the flies. Extreme white alleles as well as white deficiencies remove both brown and red pigments, the w^1 allele having very little, if any, pteridine (Hadorn and Mitchell, 1951); isoxanthopterin is present in considerable quantity during pupation but is eliminated during the first three days of adult life (Hadorn, 1954, *Experientia* 10: 483-84). Hypomorphic alleles are visibly lighter in combination with w^1 than when present as homozygotes. Intermediate white alleles result in partial loss of ommochromes and pteridines; some alleles also affect the distribution of these pigments in the compound eyes (Lewis, 1956; Green, 1959a, 1959c). Although the mutants are positively phototactic, they show no optomotor responses (Kalmus, 1943, *J. Genet.* 45: 206-13). Wild-type alleles are incompletely dominant over mutant alleles, w/w^+ heterozygotes, though visibly indistinguishable from w^+/w^+ , have less red pigment (Muller, 1935; Ziegler-Günder and Hadorn, 1958; Green, 1959b). Mutant larval

disks transplanted into wild-type host develop autonomously (Beadle and Ephrussi, 1936).

Early genetic studies identified mutations separable by intralocus recombination into at least seven groups spanning 0.03 cm (Lewis, 1952; MacKendrick and Pontecorvo, 1952; Green, 1959a; Judd, 1959). Mutants occupying the centromere-proximal sites apparently play a regulatory role (Judd, 1976). Subsequent molecular analysis has localized the proximal mutations to the 5' end of the transcription unit (w^e) and the upstream flanking sequences (w^{sp}) (Judd, 1987). Mutations at the distal sites have been mapped to the protein coding exons and the introns between them. The proximally-located regulatory mutants (w^e , for example) do not show dosage compensation; they suppress the *zeste* gene, and some of them (the w^{sp} alleles) affect the distribution of the red and brown screening pigments of the eyes. Most of the distally-located structural mutants show dosage compensation, w^a/Y males having the same eye color as w^a/w^a females, and do not suppress (but may interact with) *zeste*. Green (1959a) found that w^i fails to show dosage compensation and does not suppress *zeste*; but w^h exhi-

bits both *zeste* suppression and dosage compensation. In spite of their heterogeneity, the alleles at the white locus fail to complement each other except for w^{sp} which partially complements all other w alleles except in the presence of z^a [Babu and Bhat, 1980, *Development and Neurobiology of Drosophila*, (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York and London, pp. 35-40]. Some white alleles (w^c for example) are extremely unstable (Green, 1976); w^l is slightly unstable, giving rise to w^e and w^h , mutants with darker eyes than w^l . The locus is characterized by asymmetrical recombination involving transposons; the mutants $w^{r.def}$ and $w^{r.dup}$ are the result of such exchange (Davis *et al.*, 1987). Some *P*-element white transformations show reproducible patterns of pigmentation which can be altered by the trans-acting gene *zeste* (Rubin *et al.*, 1985).

alleles: Mutant and wild-type w alleles are tabulated below. Deficiencies are described in the rearrangement section. The first table includes alleles believed to be extant, the second alleles known to be lost. See end of text for more detailed description of certain alleles.

Table I

allele	origin	discov.	synonym/ superscript name	ref ^α	comments	cytology
w¹	spont	Morgan, 10c		10, 46, 54, 56, 67, 83, 96, 112, 116, 119, 125, 130-31, 140	eyes white; ocelli, adult testis sheath, larval Malpighian tubes colorless; suppresses <i>z</i> ; com- plements <i>w^{sp} β</i>	
w^{3A3}	X ray	Parádi		153a	partially complements <i>w^{sp} γ</i>	
w⁵	X ray (from <i>w^c</i>)	Green		59	like <i>w¹</i> ; mutates to <i>w⁺, w^{dc}, wⁱ</i>	
w^{5a}	neutrons	Schalet		153a	eyes pigmented; partially complements <i>w^{sp} γ</i>	<i>T(1;3)3C;77 +</i> <i>T(1;3)1A;80 +</i> <i>1n(3L)67;77</i>
w⁸	X ray (from <i>w^c</i>)	Green		59, 66a, 162	like <i>w¹</i> ; mutates to <i>w⁺, w^c, w^{dc}, w^{di}</i>	
w^{8x1}	spont	Welshons		66a, 183	reversion of <i>1n(1)N^{66h26}</i>	abnormality in 3C1
w^{8x2}	spont	Welshons		66a, 183	reversion of <i>1n(1)N^{66h26}</i>	appears normal
w⁹	X ray (from <i>w^c</i>)	Green		59	like <i>w¹</i> ; mutates to <i>w⁺, w^e, wⁱ</i>	
w^{9A1}	X ray	Parádi		153a	semilethal; partially complements <i>w^{sp} γ</i>	coding region deletion
w^{10gA}	γ ray	Alexandrov		1-2, 4	like <i>w¹</i> ; suppresses <i>z</i>	
w^{11E4}	X ray	Gans		46	eyes white; suppresses <i>z</i>	
w^{11G3}					see <i>z^{11G3}</i>	
w^{13D1}	X ray	Parádi		153a, 153b	slowly complements <i>w^{sp} δ</i>	coding region deletion
w^{13gA}	γ ray	Alexandrov		1, 4	does not suppress <i>z</i>	
w^{15gA}	γ ray	Alexandrov		1, 4	does not suppress <i>z</i>	
w^{17D2}	X ray	Parádi		153a, 153b	eyes pigmented; complements <i>w^{sp} β</i>	coding region deletion
w^{17g}		Kalisch		93	like <i>w¹</i> ; does not suppress <i>z</i>	
w^{20C2}	X ray	Pastink		153a	slowly complements <i>w^{sp} δ</i>	
w^{21D2}	X ray	Pastink		153a	partially complements <i>w^{sp} γ</i>	
w^{25B2}	X ray	Pastink		153a	slowly complements <i>w^{sp} δ</i>	
w³⁰					see <i>w^{e2}</i>	
w^{30C}	neutrons	Schalet		153a	does not complement <i>w^{sp} ε</i>	<i>T(1;3)</i>
w^{31D1}	X ray	Parádi		153a	slowly complements <i>w^{sp} δ</i>	<i>T(1;3)3C;64B-C</i>
w^{32k}					see <i>w^{bf2}</i>	
w^{33e31}					see <i>w^{dil}</i>	
w^{33l}					see <i>w^{sat}</i>	
w^{35A2}	X ray	Parádi		153a, 153b	slowly complements <i>w^{sp} δ</i>	coding region deletion
w^{40aH1}	X ray	Valencia		182	eyes white; male lethal	<i>1n(1)1A1-C3;4C4-7;</i> <i>17B7-8;18E2-3</i>
w^{41C1}	X ray	Parádi		153a, 153b	slowly complements <i>w^{sp} δ</i>	coding region deletion
w⁴⁹	neutrons	Schalet		153a	does not complement <i>w^{sp} ε</i>	<i>T(1;2)</i>
w^{51A}	neutrons	Schalet		153a	slowly complements <i>w^{sp} δ</i>	
w^{53A}	neutrons	Schalet		153a	does not complement <i>w^{sp} ε</i>	<i>Dp(1;1)3A3;3C1</i>
w⁵⁴	neutrons	Schalet		153a	partially complements <i>w^{sp} γ</i>	
w^{57gA}	γ ray	Alexandrov		1, 4	does not suppress <i>z</i>	
w^{59gA}	γ ray	Alexandrov		1, 4	does not suppress <i>z</i>	
w⁶⁰	spont (from <i>w^a</i>)	Hollander, 1960		75	like <i>w¹</i>	
w^{60gA}	γ ray	Alexandrov		1	does not suppress <i>z</i>	
w^{61a}	spont	Hess		72	eyes white at 24°; yellowish brown at 28°	
w^{62k}	spont (from <i>wⁱ</i>)			19	<i>w⁺</i> revertant stable (unlike <i>wⁱ</i>)	
w^{64g3}	spont	Kidd, 1964		102	eyes dark carnation	
w^{65a25}	X ray	Lefevre		15, 100, 113, 176	<i>w^{65a25}/Y</i> eyes white; <i>w^{65a25}/w^{sp}</i> eyes brown;	
w^{66A}	neutrons	Schalet		153a	does not suppress <i>z</i>	
w^{66g}					does not complement <i>w^{sp} ε</i>	
w^{67a}	X ray	Lefevre		3	see <i>w^{10gA}</i>	
w^{67g}	X ray	Lefevre		3	eyes white; does not suppress <i>z</i>	
w^{68e}	EMS	Maddern, 68e4		70	eyes white;	
w^{68g}	EMS	Maddern, 68g13		70	eyes white; ocelli white	
w^{68h}	EMS	Maddern, 68h7		70	eyes white; ocelli white	
w⁶⁹	NNG	Kaufman		99	like <i>w¹</i>	
w^{69e}	spont	Neeley, 69c		144	eyes white; ocelli colorless	

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allele	origin	discov.	synonym/ superscript name	ref ^α	comments	cytology
w^{69gA}	γ ray	Alexandrov		1-2, 4	does not suppress z	
w^{70C2}	X ray	Parádi		153a	eyes pigmented; complements w ^{sp} β	
w^{71e}	spont	Whitney, 71e		185	like w ^l	
w^{73A1}	X ray	Parádi		153a, 153b	slowly complements w ^{sp} δ	coding region deletion
w^{73d}	spont	Periquet, 73d		5	like w ^l	
w^{74b}	γ ray	Alexandrov		3	eyes ecru; does not suppress z	
w^{74d50}	γ ray	Alexandrov		3	eyes white; does not suppress z	
w^{74d145}	γ ray	Alexandrov		3	eyes white; does not suppress z	
w^{74g}	EMS	Craymer, 74g7		32	like w ^l	
w^{74k}	γ ray	Alexandrov		3	does not suppress z	
w^{79b6}	γ ray	Alexandrov		3	eyes white; does not suppress z	
w^{80B2}	X ray	Parádi		153a	slowly complements w ^{sp} δ	
w^{80j1}	w ^c FB elements	Collins, Rubin		30	eyes white	
w^{80k1}	w ^c FB elements	Collins, Rubin		30	eyes white	
w^{81d}	spont	Najera		143	eyes white	
w^{81e1}	w ^c FB elements	Collins, Rubin		30	eyes white; mutable	
w^{82a3}	w ^c FB elements	Collins, Rubin		30	eyes white; stable	
w^{83B}	X ray	Parádi		153a	partially complements w ^{sp} γ	T(1;3)3C;64B-C
w^{86.1}	transposon- mediated deficiency	Judd		34, 100	eyes white; does not suppress z	
w^{86.3}	transposon- mediated duplication	Judd		34, 100	eyes white; does not suppress z	
w^{86.5}	transposon- mediated deficiency	Judd		34, 100	eyes white; does not suppress z	
w¹⁰¹	ENU	Pastink		153c	eyes pigmented	
w¹⁰²	ENU	Pastink		153c	eyes pigmented	
w¹⁰³	ENU	Pastink		153c	eyes pigmented	
w¹⁰⁶	ENU	Pastink		153c	eyes pigmented	
w¹⁰⁷	ENU	Pastink		153c	eyes white	
w¹⁰⁸	ENU	Pastink		153c	eyes pigmented	
w¹⁰⁹	ENU	Pastink		153c	eyes white	
w¹¹³	ENU	Pastink		153c	eyes white	
w¹¹⁴	ENU	Pastink		153c	eyes white	
w¹¹⁵	ENU	Pastink		153c	eyes white	
w¹¹⁶	ENU	Pastink		153c	eyes white	
w¹¹⁷	ENU	Pastink		153c	eyes pigmented	
w¹¹⁸	ENU	Pastink		153c	eyes white	
w¹¹⁹	ENU	Pastink		153c	eyes white	
w¹²⁰	ENU	Pastink		153c	eyes white	
w¹²¹	ENU	Pastink		153c	eyes pigmented	
w¹²³	ENU	Pastink		153c	eyes pigmented	
w^{123D}	X ray	Parádi		153c	semi-dominant; wing deltas; partially complements w ^{sp} γ	In(1)1A;3C; Tp(3;1)12F;64B-C;65F
w¹²⁴	ENU	Pastink		153c	eyes white	
w¹²⁶	ENU	Pastink		153c	eyes pigmented	
w¹²⁷	ENU	Pastink		153c	eyes pigmented	
w¹²⁸	ENU	Pastink		153c	eyes pigmented	
w¹³⁰	ENU	Pastink		153c	eyes white	
w¹³²	ENU	Pastink		153c	eyes pigmented	
w¹³³	ENU	Pastink		153c	eyes pigmented	
w^{133C2}	X ray	Parádi		153a	eyes pigmented; complements w ^{sp}	
w¹³⁴	ENU	Pastink		153c	eyes pigmented	
w¹³⁶	ENU	Pastink		153c	eyes white	
w¹³⁷	ENU	Pastink		153c	eyes pigmented	
w¹³⁸	ENU	Pastink		153c	eyes white	
w¹⁴¹	ENU	Pastink		153c	eyes pigmented	
w¹⁴³	ENU	Pastink		153c	eyes white	
w¹⁴⁴	ENU	Pastink		153c	eyes pigmented	
w¹⁴⁷	ENU	Pastink		153c	eyes white	

allele	origin	discov.	synonym/ superscript name	ref ^α	comments	cytology
w¹⁴⁸	ENU	Pastink		153c	eyes pigmented	
w¹⁵⁹	ENU	Pastink		153c	eyes white	
w¹⁶³	ENU	Pastink		153c	eyes white	
w¹⁶⁸	ENU	Pastink		153c	eyes white	
w^{191E}	neutrons	Schalet		153a	slowly complements <i>w^{sp} δ</i>	
w²⁵⁸⁻¹²	X ray	Demerec, 33j			like <i>w^l</i>	
w⁷⁸¹	TE?	Valadé del Rio		181	eyes white; stable	
w¹¹¹⁸	spont (from <i>w^{DZL}</i>)	R. Levis		71, 116	eyes white; deletion of part of gene	
w^{#6}	HD	Simmons, Lim		147, 165, 166, 171	eyes white; mutates to <i>w⁺</i>	
w^{#12}	HD	Simmons, Lim		147, 165, 171	eyes white; mutates to <i>w⁺</i>	
w^{+A}		Timoféeff- Ressovsky		140, 180	eyes of <i>w/w/w^{+A}</i> triploids pinkish, later maroon	
w^{+C}		Green		55, 56, 58	eyes of <i>w/w/w^{+C}</i> triploids reddish	
w^{+O}		Green		55, 56, 58	eyes of <i>w/w/w^{+O}</i> triploids maroon	
w^{+R}		Timoféeff- Ressovsky		140, 180	eyes of <i>w/w/w^{+R}</i> pinkish, later normal red	
w^{+TE}		Rasmuson			eyes <i>w⁺</i> ; unstable	
w^{+u}	spont	Gethmann		48, 62, 63	eyes <i>w⁺</i> ; mutates to <i>w⁻</i>	
w^{-r}					see <i>Df(1)w^{rJ1}</i>	
w^{-r}					see <i>Df(1)w^{rJ2}</i>	
w^{-rN}					see <i>Df(1)w^{rJ3}</i>	
w^a	spont	Huestis, 1923	<i>apr</i>	10, 17, 17a, 17b, 23, 53-54, 58, 63, 91, 93, 113, 126, 132, 135, 136, 139, 153a, 187, 189	eyes of male yellow-orange, of female lighter, yellower; variegated with <i>mw</i> ; larval Malpighian tubes colorless; suppressed by <i>su(w^a)</i> , lightened by <i>E(w^a)</i> ; does not suppress <i>z</i> ; complements <i>w^{sp} β</i> ; carries <i>copia</i>	
w^{a2}	spont	Bridges, 1929		24, 25a, 54	eyes orange, darker in male; larval Malpighian tubes colorless; does not suppress <i>z</i> ; not affected by <i>su(w^a)</i>	
w^{a3}	spont	Curry, 34g2		24, 33, 54	eyes brownish orange; larval Malpighian tubes colorless; does not suppress <i>z</i> ; not affected by <i>su(w^a)</i>	
w^{a3}					see <i>w^h</i>	
w^{a4}	spont	Nichols-Skoog, 35c12		17, 17a, 17b, 24, 54, 117	eyes of male yellow- orange; of female lighter, yellower (both paler than <i>w^a</i>); carries <i>copia</i> ; variegated with <i>mw</i> ; larval Malpighian tubes colorless; does not suppress <i>z</i> ; not affected by <i>su(w^a)</i> , but affected by <i>E(w^a)</i> eye color between <i>w^a</i> and <i>w⁺</i> ; more brown pigment; enhanced by <i>su(f)</i> ; does not suppress <i>z</i> like <i>w^a</i>	
w^{a59k13}	spont (from <i>w^a</i>)	Green, 59k13		158-59	revertant of <i>w^a</i> eye color between <i>w^a</i> and <i>w⁺</i> ; more brown pigment; enhanced by <i>su(f)</i> ; does not suppress <i>z</i> like <i>w^a</i>	
w^{a79l}	spont	Najera		143	revertant of <i>w^a</i>	
w^{a+1}	EMS	Banerjee		9	revertant of <i>w^a</i>	
w^{a+2}	EMS	Banerjee		9	revertant of <i>w^a</i>	
w^{aE}					see <i>w^e</i>	
w^{aLTR1}	HD	Zachar		189	revertant of <i>w^a</i>	
w^{aM}	spont	Mossige	<i>w^{aRM}</i>	17a, 17b, 133	eye color between <i>w^a</i> and <i>w⁺</i> ; more brown pigment; variegated with <i>mw</i> ; suppressed by <i>su(w^a)</i> ; does not suppress <i>z</i> ; lightened by <i>E(w^a)</i> ; insertion into <i>copia</i> 5' LTR	
w^{apl}	HD	Birchler		17, 17a, 17b	insertion mutant; revertant of <i>w^l</i>	

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allele	origin	discov.	synonym/ superscript name	ref ^α	comments	cytology
<i>w^{aR57j}</i> <i>w^{aR59k1}</i>	X ray	Green		27, 135-36	see * <i>w^{a57i}</i> partial revertant of <i>w^a</i> ; <i>w^{aRsLTR}</i> derivative	
<i>w^{aR79I27}</i>	spont			135-36	partial revertant of <i>w^a</i> ; insertion into <i>copia</i>	
<i>w^{aR84e7}</i> <i>w^{aR84e19}</i> <i>w^{aR84h}</i>	spont spont spont	Mossige Mossige		135-36 135-36 17a, 17b, 135-36	partial revertant of <i>w^a</i> partial revertant of <i>w^a</i> strong revertant of <i>w^a</i> ; variegated with <i>mw</i> ; affected by <i>su(f)</i> and <i>E(w^a)</i> but not <i>su(w^a)</i> see <i>w^{aM}</i>	
<i>w^{aRM}</i> <i>w^{aS}</i>	spont	Neeley, 68g		144	eyes light orange; ocelli colorless	
<i>w^{bf}</i>	spont	Safir, 15g28	<i>buff</i>	17, 24, 34, 54, 83, 113, 117, 147, 163, 169, 187	eyes light buff; somewhat lighter in males; lighter at 19° than at 25°; larval Malpighian tubes colorless	
<i>w^{bf2}</i>	X ray	Oliver, 32k16	<i>w^{32k}</i>	24, 54, 148	eyes light buff but darker than <i>w^{bf}</i> or <i>w^{bf3}</i> ; larval Malpighian tubes yellow; does not suppress <i>z</i> ; carries <i>roo</i>	
<i>w^{bf3}</i>	spont	Curry, 36k9		24	eyes buff; larval Malpighian tubes pale yellow	
<i>w^{bl}</i>	spont	Hyde, 14g10	<i>blood</i>	15a, 24, 39, 54, 58, 77, 82, 125, 155	eyes yellowish-ruby, later sepialike; female lighter than male; testis sheaths colorless; larval Malpighian tubes pale yellow at 25°; temp. sens.;	
<i>w^{bw71g}</i>	spont	Whitney, 71g	<i>brownish</i>	185	does not suppress <i>z</i> eyes dark orange over <i>w^{71e}</i> ; variegated over <i>T(1;3)w^{vco}</i> ;	
<i>w^{Bwx}</i>	spont	Mossige, 52a	<i>Brownex</i>	81, 83, 113, 133, 155, 187	male lethal like <i>bw</i> ; <i>w^{Bwx}/+</i> duller, darker than <i>w⁺</i> ; testis sheaths colorless; larval Malpighian tubes pale yellow; does not suppress <i>z</i>	
<i>w^c</i>	X ray (from <i>wⁱ</i>)	Green	<i>crimson</i>	29-30, 48, 59, 60-63, 69, 115, 149, 162	eyes crimson; <i>w^c</i> mutates to <i>w¹</i> , <i>w⁵</i> , <i>w⁸</i> , <i>w⁹</i> , <i>w⁻</i> , <i>w^{dc}</i> , <i>w^{di}</i> , <i>wⁱ</i> , <i>w⁺</i> ; complements <i>w^{sp}</i>	<i>Tp(1;3)w^c</i>
<i>w^{cf}</i>	X ray	Nicoletti, 1960	<i>coffee</i>	145, 184	eyes deep ruby, later sepialike; larval Malpighian tubes bright yellow	
<i>w^{cf2}</i>	NNG	Kaufman		98	eyes ruby red, darkening to sepia in both males and females; with <i>v</i> , eyes orange; ocelli purplish	
<i>w^{ch}</i>	spont	Safir, 12j	<i>cherry</i>	48, 54, 131, 155, 168, 187	eyes translucent pink; male lighter than female; ocelli pale; adult testis sheaths, larval Malpighian tubes colorless; suppresses <i>z</i>	
<i>w^{co}</i>	spont	Lancefield, 1917	<i>coral</i>	24, 54, 104, 125, 126, 155	eyes of male deep ruby, later garnetlike; female eyes lighter; larval Malpighian tubes pale yellow; does not suppress <i>z</i> like <i>w^{co}</i> ;	
<i>w^{co2}</i>	EMS	Farmer		44-45	suppressed by <i>bw⁺</i> like <i>w^{co}</i>	
<i>w^{co61}</i>	spont (from <i>w^l</i>)	Hollander, 1961		76		
<i>w^{col}</i>	X ray	Demerec, 33j6	<i>w²⁵⁸⁻⁸</i> <i>colored</i>	54, 105, 155	eyes brick-red to dull, brownish in young males, lighter in females; does not suppress <i>z</i> revertant of <i>w^{col}</i>	
<i>w^{col+1}</i>	EMS	Banerjee		9		

allele	origin	discov.	synonym/ superscript name	ref ^α	comments	cytology
w^{crr}	spont	Judd, 1962	<i>carrot</i>	86, 113 155, 187	eyes reddish brown; no sex difference in color;	
w^{crr2}	NNG	Kaufman		99	does not suppress z eyes orange, darkening to reddish brown in both males and females;	
w^{dc}	spont (from <i>w^c</i>)	Green	<i>dark crimson</i>	59, 63, 162	ocelli colorless eyes dark crimson; mutates to <i>w⁺</i> , <i>w^I</i> , <i>w^c</i> , <i>w^{dc}</i> , <i>wⁱ</i>	
w^{dil}	spont (from <i>w^c</i>)	Green	<i>dilute</i>	59, 63	eye color between <i>w^c</i> and <i>wⁱ</i> ; mutates to <i>w⁺</i> , <i>w^I</i> , <i>w^c</i> , <i>w^{dc}</i> , <i>wⁱ</i>	
w^{dp}	chloroethyl methane- sulfonate	Auerbach, 1957	<i>deep purple</i>	6, 51, 54, 155	eyes deep purple; no sex difference in color;	
w^{dt}	spont	Neeley, 68k	<i>dirty</i>	144	does not suppress z eyes sooty white; <i>w^{dt}</i> /w grayer than w/w; ocelli colorless	
w^{DZL}	spont	Bingham	<i>Dominant- zeste-like</i>	11, 15, 16, 30, 114-15, 118, 147, 166, 187, 188	dominant over <i>w⁺</i> ; eyes dull red brown in <i>z⁺</i> males, yellow in <i>z</i> males and in homo- and hetero- zygous females (<i>z⁺</i> or <i>z</i>) when X's synapsed	
w^e	spont (from <i>w^I</i>)	Morgan, 11h	<i>w^{aE}</i> ; <i>eosin</i>	24, 54, 58 117, 123, 131, 139, 155, 177, 187	eyes of female yellowish pink; eyes of male and <i>w^e</i> /w female lighter; adult testis sheaths and larval Malpighian tubes colorless; enhanced by <i>e(w^e)</i> ; suppresses z	
w^{e2}	spont (from <i>w^I</i>)	Hefner, 1925	<i>w³⁰</i>	24, 54, 187	similar to <i>w^e</i> but darker and with less sex difference; larval Malpighian tubes colorless; enhanced by <i>e(w^e)</i> ; suppresses z	
w^{e3}	spont (extracted from <i>w^e rb</i>)	Nolte, 1953		146, 187	eyes of female darker than <i>w^e</i> female, of male pinker than <i>w^e</i> male	
w^{e59}				153a	slowly complements <i>w^{sp} δ</i>	
w^{e+1}	EMS	Banerjee		9	revertant of <i>w^e</i>	
w^{e+2}	EMS	Banerjee		9	revertant of <i>w^e</i>	
w^{ec3}	X ray	Muller	<i>ecru</i>	54	eyes almost white	
w^{FM6}	spont (in <i>FM6</i>)	Kidd, 1964	<i>First</i>	102	eyes white	
w^{gd}	spont	Rayle, 1964	<i>multiple 6</i> <i>garnetoid</i>	161	eyes yellowish ruby, eyes brown in <i>w^{8d} su(f)</i> males at 25°; suppresses z eye color between <i>w^{bf}</i> and <i>w^{a4}</i> ; variegated with <i>mw</i> ; larval Malpighian tubes colorless;	
w^h	spont (from <i>w^I</i>)	Dunn, 34j27	<i>w^{a3}</i> ; <i>honey</i>	17, 17a, 38, 54, 91, 147, 155	enhanced by <i>e(w^e)</i> ; suppresses z like <i>w^h</i> in phenotype	
w^{h82}	spont (from <i>w^I</i>)	Kennison, 1982				
w^{hd80k17}	HD	Kidwell	<i>hybrid dysgenesis</i>	117, 147, 165	eyes white; reverts to <i>w⁺</i> on excision of <i>P</i> -element	
w^{hd81b6}	HD	Kidwell		165	eyes white; stable in <i>P</i> and <i>M</i> cytotypes;	
w^{hd81b9}	HD	Kidwell		147, 165	<i>copia</i> insertion eyes white; reverts to <i>w⁺</i> on excision of <i>P</i> -element	
w^{hd81b11}	HD	Kidwell		147, 165, 189	eyes white; stable in <i>P</i> and <i>M</i> cytotypes;	
w^{hd81b25}	HD	Kidwell		165	<i>copia</i> insertion eyes white; stable in <i>P</i> and <i>M</i> cytotypes; <i>copia</i> insertion	

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w^{hd81c2}	HD	Kidwell		165	eyes white; reverts to w ⁺ on excision of P-element	
wⁱ	spont (from w ^l)	Sturtevant, 1918	<i>ivory</i>	20-22, 24, 29, 54, 59, 91, 97, 121, 123, 137, 147, 149, 153a, 155, 166 42, 43	eyes light buff or yellowish, lighter in male; larval Malpighian tubes colorless; does not suppress z; slowly complements w ^{sp} δ like w ⁱ ; unstable	
wⁱ¹⁶		Fahmy, Fahmy		97, 147	red eyes; carries 3 kb transposable element F	
w^{i+A}	X ray (from w ⁱ)	Karess, Rubin		22, 97	eyes light orange (partial revertant of w ^l)	
w^{ip}	spont (from w ⁱ)	Bowman, Green		22	partial reversion of w ⁱ	
w^{ip2}		Bowman, Green		25b, 154, 170	brownish eyes at 20°, lighter at 25°; enhances z; z ^l w ^{IR1} males have mottled orange eyes; carries I factor	
w^{IR1}	HD	Pelisson	<i>Inducer Reactive</i>	154, 170	brown eyes at all temp; carries I factor	
w^{IR2}	HD	Pelisson		25b, 154, 170	brown eyes; carries I factor	
w^{IR3}	HD	Pelisson		154, 170	brown eyes at 20°; lighter at 25°; carries I factor	
w^{IR4}	HD	Pelisson		154, 170	brown eyes at all temp; carries I factor	
w^{IR5}	HD	Pelisson		154, 170	red-brown eyes at all temp; carries I factor	
w^{IR6}	HD	Pelisson		154, 170	eyes white	molecular deletion
w^{IR7}	HD	Pelisson		154, 170	eyes white	molecular deletion
w^{IR8}	HD	Pelisson		157	eye color normal; male has greatly reduced amount of isoxanthopterin in abdomen; z w ⁱ⁵ male has variegated eye, between z and w ⁺ in color; probably insert of foreign DNA (R. Jones)	may be intralocus duplication
w^{is}	spont	Rasmuson	<i>isoxanthopterinless</i>		eyes mottled; viable and fertile in both sexes	
w^{m4}	X ray	Muller, 1929	<i>mottled</i>	138, 164, 179	eyes strongly variegated; homozygous lethal revertant of w ^{m4}	<i>In(1)3C1-2;20F</i> (Lefevre)
w^{m4h}		Reuter, Wolff		164	eyes strongly variegated; homozygous lethal revertant of w ^{m4}	<i>In(1)3C1-2;20E-F</i>
w^{m4rv5}	X ray	Tartof		179	revertant of w ^{m4}	<i>In(1)3C;4A;20</i>
w^{m4rv6}	X ray	Tartof		179	revertant of w ^{m4}	<i>In(1)1F;3C1-2;20</i>
w^{m4rv26}	X ray	Tartof		179	revertant of w ^{m4}	<i>In(1)3C;6B;20</i>
w^{m5}	X ray	Muller, 1929		18, 138	eyes mottled; viable and fertile in both sexes	<i>T(1;4)3C3-4;</i> <i>101F1-2</i>
w^{m49a}	X ray	Lefevre, 49a7	w ^{mSp}	106-07, 160	eyes have red sectors on white background; extra Y → w ⁺ eye	<i>Tp(1;3)3A10-B1;</i> <i>3E2-3;80</i>
w^{m51b}	X ray	W.K. Baker, 51b19		47	eyes mottled for w and <i>rst</i>	<i>In(1)3C1-2;20</i>
w^{m51c}	X ray	Lefevre, 51c20		107-08	eyes of w ^{m51c} / _w variegated, male lethal like w ^{m4}	<i>Tp(1;4)3C1-2;</i> <i>3C4-7;20A;101</i>
w^{m54i}	neutrons	Mickey, 5413		127	eyes cream colored, mottled; viable, fertile in both sexes	<i>In(1)3C3-5;20F</i>
w^{m258-18}	X ray	Demerec, 33k		37	eyes, larval Malpighian tubes mottled; mottling more extreme at low temperature; heterozygous female Notch; nearly lethal	<i>T(1;4)3C4-5;101</i>
w^{m258-21}	X ray	Demerec, 1934	w ^{VD3}	80	eyes variegated; 3 lines differing in degree of variegation and parental effect; homozygous viable in female only; maternal effect on variegation	<i>T(1;3)3C2-3;</i> <i>100C3-4</i>
w^{m264-58}	X ray induced (with N ²⁶⁴⁻⁵⁸)	Demerec, 38d		7-8, 36, 73, 172-74	eyes variegated	associated with <i>Tp(1;3)N²⁶⁴⁻⁵⁸ =</i> <i>Tp(1;3)3B2-3;</i> <i>3D6-7;80D-F</i>
w^{m609e}	X ray	Patterson		64-66	eyes variegated	<i>T(1;3)3C2-3;</i> <i>100C3-4</i>

allele	origin	discov.	synonym/ superscript name	ref α	comments	cytology
w m4000	X ray	Buzzati-Traverso, 4117		26	eyes cream colored, darker in males; variegated for <i>w</i> , <i>rst</i> , <i>fa</i> , <i>co</i> ; viable and fertile in both sexes see <i>Df(1)N-63b</i>	rearrangement with break in <i>w</i>
w ma w mD3 w mJ	X ray	Dubinin Jonsson, 61i28		167 110, 112, 179	eyes variegated eyes mottled red and white	<i>T(1;4)3C:101</i> <i>T(1;4)3C2-3; 20;102C</i>
w mMc	X ray	McLean		142, 179	eye color light mottled; variegated for <i>rst</i>	<i>In(1)3C1-2;20A-C</i>
w mo	spont	Hanly		68, 186	eyes light mottled orange, later darkening; drosopterin reduced to 10% normal; <i>w mo/w</i> → dark brown eyes; viability good see <i>w m49a</i>	
w mSp w Mt w ntg w r	spont NNG spont (<i>w^a</i> partly reverted)	Morata Kaufman Muller, 1944	<i>w^M</i> ; Morata nitrosoguanidine <i>reddish</i>	101 99 135-136, 141	eyes white like <i>w^{crr2}</i> <i>w^r/w^r</i> nearly <i>w⁺</i> ; <i>w^r/w^a</i> dark maroon; believed to be <i>w^{ALTR}</i> derivative	
w r,def	intralocus exchange	Judd, 1961	<i>w^{rdf}</i> ; recombinant- deficiency	34, 84, 86-87, 91	eyes white; <i>w^{rdf}/w^{sp}</i> like <i>w^{sp}/w^{sp}</i> ; reduces recombination in <i>w</i> region; suppresses <i>z</i>	intralocus deficiency for <i>w^{ch}</i> site
w r,dup	intralocus exchange	Judd, 1961	<i>w^{rdp}</i> ; recombinant- duplication	34, 79, 84, 86-87, 91	eye color lighter than <i>w^{bf}</i> ; enhances <i>z</i> ; increases recombination in <i>w</i> locus	intralocus duplication for <i>w^{ch}</i> site
w ric	spont	Judd	<i>roo-in-copia</i>	34, 136	eyes like <i>w^a</i> ; carries <i>roo</i> in <i>copia</i> insertion	
w Rr	EMS	Pokholkova, 1976	<i>Russian red</i>	190	eyes vermilion, later almost wild type; ocelli colorless	
w rv#6 w rv#12 w rvD5 w s10	spont spont spont		<i>revertant</i> <i>revertant</i> <i>spontaneous</i>	165 165 118 54	<i>w⁺</i> revertant of <i>w^{#6}</i> <i>w⁺</i> revertant of <i>w^{#12}</i> revertant of <i>w^{DZL}</i> eyes white; not affected by <i>su(w^a)</i> ; does not suppress <i>z</i>	
w sat	spont	Bridges, 33126	<i>w³³¹</i> ; <i>satsuma</i>	24-25, 54	eyes deep ruby; larval Malpighian tubes nearly <i>w⁺</i> ; does not suppress <i>z</i>	
w sey	spont	Erickson	<i>sepia-yellow</i>	40-41	eyes lemon yellow with <i>se</i> at 25°, white at 18°	
w sp1		Showell, 1944	<i>spotted</i> ; <i>sp-w</i>	54, 57, 89, 91 117, 120, 147, 187, 188	facets yellowish to brown; eyes with fine-grained mottling; male darker than female; suppresses <i>z</i> ; suppressed by <i>su(w^{sp})</i>	
w sp2	spont	Mohler, 56c22		34, 48, 129	eyes mottled like <i>w^{sp}</i> ; <i>w^{sp2}/w</i> and <i>w^{sp}/w^e</i> uniform brown	
w sp3 w sp4 w sp55	X ray spont	Green, 59a29 LeFever	<i>sp-w⁴</i>	34, 52, 187 34, 113, 147, 187 17, 17a, 17b, 117, 187	like <i>w^{sp}</i> like <i>w^{sp}</i> eye yellow; color lightened by <i>E(w^a)</i> ; variegated with <i>mw</i> ;	
w sp81d	EMS	Green		34, 35	enhances <i>z</i> in <i>XY</i> male darker than <i>w^{sp1}</i> ; not suppressed by <i>su(w^{sp})</i>	
w sp+1 w sp+2 w sp+3 w sp+4 w spA	EMS EMS EMS EMS γ ray	Banerjee Banerjee Banerjee Banerjee Alexandrov		9 9 9 9 3	revertant of <i>w^{sp}</i> revertant of <i>w^{sp}</i> revertant of <i>w^{sp}</i> revertant of <i>w^{sp}</i> eyes darker and more speckled than <i>w^{sp2}</i> ; suppresses <i>z</i>	
w t	spont	Hyde, 14k2	<i>tinged</i>	77	eyes light pinkish; larval Malpighian tubes colorless	
w tache	from <i>w^{zm}</i>	Judd	<i>tache</i>	79	like <i>w^{zh}</i> , but red pigment incomplete around circumference of eye	

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allele	origin	discov.	synonym/ superscript name	ref ^α	comments	cytology
w^{tuh}	spont	Kuhn, Walker	<i>tumorous head</i>	103	like <i>w^l</i>	
w^{u11}		Fahmy, Fahmy	<i>unstable</i>	42, 48	eyes white; unstable	
w^{udf}	spont	Gethmann	<i>unstable deficient</i>	48	like <i>w^l</i> ; <i>w^{sp} / w^{udf}</i> eyes pale lemon yellow; male viable	
w^{vC}	X ray [in <i>R(1)2</i>]	Catcheside	<i>variegated of Catcheside</i>	74	eyes mottled; male viability zero → fair; <i>w^{vC} / +</i> females may be <i>N</i>	<i>1n(1)3C1-2; 20F + R(1)1A3-4; 20F</i>
w^{vco}		Clausen	<i>variegated cobbled</i>	111	eye mostly white with red mottling	<i>Tp(1;3)2B17-C1; 3C5-6; 77D3-5; 8I</i>
w^{VD3}					see <i>w^{m258-21}</i>	
w^{vr}	spont	Mostashfi, Kouliantz	<i>venetian red</i>	134	eyes white	
w^w	spont (from <i>w^{zmz}</i>)	Kalisch, 1980	<i>white</i>	93-94	eyes white; mutates to <i>w^{zm+}</i> , <i>w^{zl}</i> , <i>w^{zmz}</i> <i>w^{zmzz}</i> ; may revert to normal X; does not suppress <i>z</i>	<i>1n(1)3B2-C; 4B4-C1</i>
w^z	from <i>w^{zm}</i>	Judd, 1967	<i>zeste</i>	48, 88-89	white eyes; highly mutable does not suppress <i>z</i>	
w^{zh}	from <i>w^{zm}</i>	Judd, 1967	<i>w^{halo}</i>	71, 79, 88, 90, 114	"halo" eyes with <i>z</i> in homozygotes; mutable enhances <i>z</i> ;	<i>Tp(1;3)3C2-3; 3C6; 61D</i>
w^{zl}	spont (from <i>w^{zm}</i>)	Becker, 1958	<i>z^l</i>	12-13, 63, 85, 86	<i>z w^{zl}</i> female has lemon yellow eyes at 25°-30°; same female at 14° and male at 25°-30° have yellow eyes with large red spots; <i>z w^{zl}</i> male at 14° has red spots on yellow with salt-and-pepper mottling enhances <i>z</i> ; eye color mottled with <i>z</i> and <i>mw</i> ; <i>z w^{zm}</i> female like <i>z w^{zl}</i> female at 25°-30°; same female at 14°-17° has orange eyes with large red spots; <i>z w^{zm}</i> male at 25°-30° has yellow eyes with small red spots; at 14°-17°; male is <i>w⁺</i> ; carries <i>BEL</i> eyes of <i>z w^{zm}</i> males like <i>z w⁺</i> at 14-17°, duller at 25-30°; eyes of <i>z w^{zm}</i> females between <i>z w⁺</i> and <i>z w^{zm}</i>	
w^{zm}	spont (asym. exchange within <i>w</i>)	Becker, 1959	<i>z^m</i>	12-13, 17, 17a, 63, 85-86, 92, 117, 147, 187	eyes of <i>z w^{zmz}</i> males red-yellow (pepper and salt mottling) at 14-17°; males at 25-30° and all <i>z</i> females have lemon yellow eyes; mutates to <i>w^{zm}</i> , <i>w^{zm-}</i> <i>w^{zmzw}</i> , <i>w^w</i> <i>w^{zmzz}</i> , <i>w^{zmzrb}</i> <i>w^{zmzrb}</i> at 28°, <i>z w^{zmzrb}</i> females have lemon yellow eyes, males have red to red brown eyes	
w^{zm+}	from <i>w^{zm}</i> <i>w^{zmz}</i> <i>w^{zmzz}</i>	Kalisch, Becker, 1970		95	eyes of <i>z w^{zmz}</i> males red-yellow (pepper and salt mottling) at 14-17°; males at 25-30° and all <i>z</i> females have lemon yellow eyes; mutates to <i>w^{zm}</i> , <i>w^{zm-}</i> <i>w^{zmzw}</i> , <i>w^w</i> <i>w^{zmzz}</i> , <i>w^{zmzrb}</i> <i>w^{zmzrb}</i> at 28°, <i>z w^{zmzrb}</i> females have lemon yellow eyes, males have red to red brown eyes	
w^{zmz}	spont (from <i>w^{zm}</i>)	Becker, 1959		63, 94-95	eyes of <i>z w^{zmz}</i> males red-yellow (pepper and salt mottling) at 14-17°; males at 25-30° and all <i>z</i> females have lemon yellow eyes; mutates to <i>w^{zm}</i> , <i>w^{zm-}</i> <i>w^{zmzw}</i> , <i>w^w</i> <i>w^{zmzz}</i> , <i>w^{zmzrb}</i> <i>w^{zmzrb}</i> at 28°, <i>z w^{zmzrb}</i> females have lemon yellow eyes, males have red to red brown eyes	
w^{zmzrb}	from <i>w^{zmz}</i>	Kalisch, Becker, 1970		95	eyes of <i>z w^{zmz}</i> males red-yellow (pepper and salt mottling) at 14-17°; males at 25-30° and all <i>z</i> females have lemon yellow eyes; mutates to <i>w^{zm}</i> , <i>w^{zm-}</i> <i>w^{zmzw}</i> , <i>w^w</i> <i>w^{zmzz}</i> , <i>w^{zmzrb}</i> <i>w^{zmzrb}</i> at 28°, <i>z w^{zmzrb}</i> females have lemon yellow eyes, males have red to red brown eyes	
w^{zmzw}	spont (from <i>w^{zmz}</i>)	Kalisch, Becker, 1970		94-95	eyes of males and females white in genotypes <i>z w^{zmzw}</i> and <i>z⁺ w^{zmzw}</i> ;	
w^{zmzz}	spont (from <i>w^{zmz}</i>)	Judd, 1967		63, 94-95	does not suppress <i>z</i> eyes of males with <i>z</i> deep yellow at 14-17°, pale yellow at 25-30°; eyes of <i>z</i> females pale yellow at all temperatures	
w^{zs}	from <i>w^{zm}</i>	Judd, 1967		88	eyes white; suppresses <i>z</i>	

Table II

allele	origin	discov.	synonym/ superscript name	ref ^α	comments	cytology
*w 13G2 *w 48h	X ray mustard gas (from <i>w^{m4}</i>)	Gans Lindsley, 48h13		122	eyes variegated like <i>w^l</i>	<i>T(1;2)3C3-5;56F</i> see <i>In(1)w^{m4}</i>
*w 48hS *w 51a *w 62d	X ray spont spont (from <i>w^a</i>)	Schultz, 48h Redfield, 51a Mickey, 62d	<i>w 48h</i>	31 163 127	like <i>w^l</i> like <i>w^l</i> like <i>w^l</i>	
*w 64 *w 258-43 *w 258-47 *w 258-49 *w 258-50 *w 258-51 *w 258-52	spont X ray X ray X ray X ray X ray X ray	Whitten, 1964 Demerec, 38k Demerec, 39a Demerec, 39c Demerec, 39c Demerec, 39k Demerec, 40a		31 31 31 31 31 31	like <i>w^l</i> male lethal like <i>w^l</i> like <i>w^l</i> like <i>w^l</i> like <i>w^l</i> eyes white and rough	<i>*T(1;4)3C3-5;102F4-5</i> <i>*In(1)3C7-9;8E11-F1</i> (Sutton)
*w a55k *w a57i	X ray spont (from <i>w^a</i>)	Clark Green, 57i11	<i>apr 55k</i> <i>w aR57j</i>	28 158-59	like <i>w^a</i> eye color between <i>w^a</i> and <i>w⁺</i> ; more brown pigment; enhanced by <i>su(f)</i> ; does not suppress <i>z</i>	
*w a58l	spont	Green, 58l12		158-59	eye color between <i>w^a</i> and <i>w⁺</i> ; enhanced by <i>su(f)</i> ; does not suppress <i>z</i>	
*w a59k1	X ray (from <i>w^a</i>)	Green, 59k1		158-59	eye color between <i>w^a</i> and <i>w⁺</i> ; more brown pigment; enhanced by <i>su(f)</i> , suppressed by <i>su(w^a)</i> ; does not suppress <i>z</i>	
*w a59k9	X ray (from <i>w^a</i>)	Green, 59k9		158-59	eye color between <i>w^a</i> and <i>w⁺</i> ; more brown pigment; enhanced by <i>su(f)</i> , suppressed by <i>su(w^a)</i> ; does not suppress <i>z</i>	
*w a60a5	spont (from <i>w^a</i>)	Sherwood, 60a5		158-59	eye color between <i>w^a</i> and <i>w⁺</i> ; more brown pigment; enhanced by <i>su(w^a)</i> ; does not suppress <i>z</i>	
*w ch2			<i>cherry</i>	54	does not suppress <i>z</i> like <i>w^{ch}</i> except no sex difference in eye color; does not suppress <i>z</i>	
*w ch41j *w dil	spont spont	Ives, 41j9 Ives, 33e31	<i>w 33e31</i> ; <i>dilute</i>	78 156	like <i>w^{ch}</i> eyes dilute red; overlaps <i>w⁺</i>	
*w dp2	chloroethyl methane- sulfonate	Auerbach, 1957	<i>deep purple</i>	6, 51, 54	like <i>w^{dp}</i> ; does not suppress <i>z</i>	
*w ec *w ec2 *w G	spont X ray (in <i>In(1)y^G</i>)	Muller, 1918 Goldschmidt	<i>ecru</i> <i>Goldschmidt</i>	28, 137 54 49	eyes very pale buff like <i>w^{ec}</i> like <i>w^l</i>	see <i>In(1)y^G</i>
*w m1	X ray	Muller, 1927	<i>mottled</i>	50, 138	variegated for eye color and <i>N</i> ; <i>XY</i> male lethal, <i>XYY</i> male viable and sterile, with dark eye color	<i>*T(1;3)</i>
*w m2	X ray	Patterson, 1929		138	eyes mottled; male sterile	<i>*T(1;3)</i>
*w m3	X ray	Muller, 1929		138	only viable with normal <i>X</i> ; <i>w/Dp(1)f)w^{m3}</i> → mottling	<i>*Dp(1)f)3C-D;19-20</i>
*w m11 *w m52b12	X ray X ray (in <i>rst³</i>)	Panshim Ratty, 52b12		151-53 109	eyes mottled eyes variegated	<i>*T(1;4)3C3-4;101A-D</i> <i>*Tp(1;2)1E5-F1;3C3-4;</i> <i>20F;40-41</i>
*w m52b13	X ray (in <i>rst³</i>)	Ratty, 52b13		109	eyes variegated	<i>*Tp(1;4)2A2-3;</i> <i>3C3-4;20F;101</i>
*w m53a	X ray	Farnsworth, 53a4		109	eyes variegated	<i>*Tp(1;2)3B2-C1;</i> <i>3C9-D1;40-41</i>
*w m53e	neutrons	Mickey, 53e11		127	eyes variegated	<i>*T(1;2)3C3-4;20A2-3;</i> <i>58F8-59A1</i>
*w m53j	X ray (in <i>In(1)EN</i>)	Bender, 53j		14	eyes mottled with small dark brown patches	<i>*In(1)1A;3C3-5;</i> <i>20;20F;20F;</i> tentative

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allele	origin	discov.	synonym/ superscript name	ref ^α	comments	cytology
*<i>w</i> m55b	X ray [from <i>R(1)2</i>]	Bender, 55b		14	eyes mottled with large pink or white patches	* <i>In(1)1A3-4;3C-5</i> <i>20;19F-20A1;20A1-F</i> (inferred from origin)
*<i>w</i> m258-31	X ray	Demerec, 371		31	eyes cream colored, mottled; male viable	* <i>T(1;4)3C3-5;</i> <i>102F4-17</i>
*<i>w</i> m258-32	X ray	Demerec, 371		31	eyes cream colored, mottled; male viable	* <i>T(1;3)3C3-5;81</i>
*<i>w</i> m258-34	X ray	Demerec, 38b		31	eyes cream colored mottled; male viable	* <i>T(1;2)3C3-5;41A</i>
*<i>w</i> m258-36	X ray	Demerec, 38b		178	eyes cream colored mottled; male viable	* <i>Tp(1;2)3C6-7;4C2-3;</i> <i>41A-B;41F5-6</i>
*<i>w</i> m258-37	X ray	Demerec, 38j		31	eyes mottled; male viable	* <i>T(1;2)3C3-4;40-41A</i>
*<i>w</i> m258-39	X ray	Demerec, 38e		31	eyes cream colored, mottled; male viable	* <i>T(1;2)3C3-5;40E-F</i>
*<i>w</i> m258-40	X ray	Demerec, 38e		31	eyes cream colored, mottled, rough	* <i>T(1;2)3C3-5;41</i>
*<i>w</i> m258-44	X ray	Demerec, 38k		31	eyes mottled; male viable	* <i>T(1;2;3)3C3-4;</i> <i>4D2-E1;</i> <i>56E1-F1;80D</i>
*<i>w</i> m258-53	X ray	Demerec, 391		178	eyes mottled; male viable	* <i>T(1;4)3C1-2;101E-F</i> (<i>101F-102F</i> lost)
*<i>w</i> m258-54	X ray	Sutton, 40e		31	eyes cream colored, mottled; male lethal	* <i>T(1;3)3B2-C1;</i> <i>19F2-20A1;</i> <i>20E;63C7-8</i>
*<i>w</i> mA	X ray (in <i>YSX.YL</i>)	Stone		65-66	eyes variegated; male viable and fertile	* <i>T(1;4)3C2-3;101A2-3</i>
*<i>w</i> mCi	X ray	Cicak		150	eyes variegated with red and white facets	
*<i>w</i> mD1	X ray	Dubinini		167	eyes variegated	* <i>T(1;2)3B;19-20;21F</i>
*<i>w</i> mDG1		Dubinini, Goldat		167	eyes variegated	3B inserted in chromocenter
*<i>w</i> mDV4		Dubinini, Volotov		167	eyes mottled; male lethal; <i>w^{mD4}</i> /+ female <i>N</i>	* <i>T(1;4)3C3-7;</i> <i>3D;101A-D;</i> (<i>3C-3D</i> missing)
*<i>w</i> mMed		Medvedev, 1934		31	mottling on <i>w^a</i> background	<i>T(1;4)</i>
*<i>w</i> M	spont (from <i>w^a</i>)	MacKendrick, 1955	<i>of MacKendrick</i>	124, 158-59	eye color almost <i>w⁺</i> (less red pigment); <i>w^M</i> / <i>w</i> like <i>w^{co}</i> (a bit darker); viable and fertile; enhanced by <i>su(w^a)</i> and <i>su(f)</i> ; does not suppress <i>z</i>	
*<i>w</i> M59	spont	Muller, 59d	<i>of Muller</i>	128	eyes cream colored, darkening a bit with age	
*<i>w</i> P	spont	Steinberg, 37b17	<i>pearl</i>	24, 175	eyes very pale (lighter than <i>w^l</i>); larval Malpighian tubes colorless	
*<i>w</i> s1	spont	Green, 1959	<i>spontaneous</i>	54	eyes white;	
*<i>w</i> s2	spont	Green, 1959		54	does not suppress <i>z</i> eyes white;	
*<i>w</i> s3	spont	Green, 1959		54	does not suppress <i>z</i> eyes white;	
*<i>w</i> s4	spont	Green, 1959		54	does not suppress <i>z</i> eyes white;	
*<i>w</i> s5	spont	Green, 1959		54	does not suppress <i>z</i> eyes white;	
*<i>w</i> s9	spont	Green, 1959		54	does not suppress <i>z</i> eyes white;	
*<i>w</i> vD1	X ray	Demerec, 33j19	<i>variegated</i> <i>of Demerec</i>	31	eyes variegated red and white; male fertile	* <i>Dp(1;4)3C1-4;</i> <i>101A-D</i>
*<i>w</i> vD2	X ray	Demerec, 33k27		31	eyes with fine-grained variegation of cream with dark spots; female occasionally variegated for <i>rst</i>	* <i>T(1;2;4)3C4-5;18F;</i> <i>38;101A-C</i>
*<i>w</i> vD4	X ray	Demerec, 33k2		31	<i>w^{vD4}</i> /+ female mottled; <i>XY, w^{vD4}</i> male mottled, usually lethal; <i>XYY</i> male sterile	* <i>T(1;2)3D6-E1;40F</i>
*<i>w</i> X1	X ray	Green, 1959	<i>from X</i> <i>irradiation</i>	54	eyes white; does not suppress <i>z</i>	

allele	origin	discov.	synonym/ superscript name	ref ^α	comments	cytology
^w X2	X ray	Green, 1959		54	eyes white; does not suppress z	
^w X3	X ray	Green, 1959		54	eyes white; does not suppress z	
^w X4	X ray	Green, 1959		54	eyes white; does not suppress z	
^w X5	X ray	Green, 1959		54	eyes white; does not suppress z	
^w X6	X ray	Green, 1959		54	eyes white; does not suppress z	
^w X8	X ray	Green, 1959		54	eyes white; does not suppress z	
^w X16	X ray	Green, 1959		54	eyes white; suppresses z	

- ^α 1 = Alexandrov, 1971, DIS 46: 71, 72; 2 = Alexandrov, 1972, DIS 48: 88, 133; 3 = Alexandrov, 1982, DIS 58: 7-8, 9-10, 10-12; 4 = Alexandrov and Soluyanov, 1974, DIS 51: 32; 5 = Anxolabehere and Periquet, 1973, DIS 50: 21; 6 = Auerbach, 1957, DIS 31: 107-09; 7 = Baker, 1963, Am. Zool. 3: 57-69; 8 = Baker and Spofford, 1959, Univ. Texas Publ. 5914: 135-54; 9 = Banerje, Hazra, and Sen, 1978, Mutat. Res. 50: 309-15; 10 = Beadle and Ephrussi, 1936, Genetics 21: 230; 11 = Beckendorf, 1983, Nucleic Acids Res. 11: 737-51; 12 = Becker, 1959, DIS 33: 82; 13 = Becker, 1960, Genetics 45: 519-34; 14 = Bender, 1955, DIS 29: 69; 15 = Bingham, 1980, Genetics 95: 341-53; 15a = Bingham and Chapman, 1986, EMBO J. 5: 3343-51; 16 = Bingham and Zachar, 1985, Cell 40: 819-25; 17 = Birchler, 1986, Genetics 113: s47; 17a = Birchler and Hiebert, 1989, Genetics 122: 129-38; 17b = Birchler, Hiebert, and Rabinow, 1989, Genes Dev. 3: 73-84; 18 = Bolen, 1931, Am. 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- β Heteroalleles with w^{sp} have brown eyes at eclosion, complementing w^{sp} ; eyes somewhat darker after 4-5 days.
 γ Heteroalleles with w^{sp} have yellow-orange, orange, or orange-brown eyes at eclosion; mottling only on first day.
 δ Heteroalleles with w^{sp} usually have orange-brown eyes at eclosion; mottling almost gone after first day; eyes slowly darken.
 ε Heteroalleles with w^{sp} have yellow eyes at eclosion, usually with mottling; eyes become orange after 4-5 days, but not brown.

cytology: Placed in 3C2 by Schultz and also by Lefevre and Wilkins (1966, *Genetics* 53: 175-987) on the basis of rearrangement breakpoints [$In(1)w^4 = In(1)3C1-2;20F$, for example]. An electron microscope study of $In(1)z^{+64b9} = In(1)3C1-2;12B9-10$ shows a faint band to the right of 3C1 consisting of the leftmost border of 3C2 plus material from 12B9 [Sorsa, Green, and Beerman, 1973, *Nature (London) New Biol.* 245: 34-37]; this band is not seen in most preparations of normal chromosomes. Judd (1976, 1987) suggests that w^+ is located in a very small band between 3C1 and 3C2. Goldberg *et al.* (1982) showed that the 3C1-2 break of $In(1)z^{+64b9}$ is proximal to the white locus on the molecular map (about 10 kb to the right of the w^a insertion of *copia*).

molecular biology: The w^a allele was the first gene cloned by "transposon tagging", using previously cloned transposing element *copia* (Bingham *et al.*, 1981). The w^a clone was extended on both sides by chromosome walking and a segment of 14 kb was found to contain all of the white locus sequences essential to the w^+ eye-color phenotype (Levis *et al.*, 1982). The white locus was also cloned by Goldberg *et al.* (1982), using a large TE containing w^a and *rst* inserted at 87A7 next to previously cloned sequences (genes encoding *Hsp70*). Pirrotta *et al.* (1983) later cloned white by microdissection of the 3C2 region of the salivary chromosomes.

A physical map of the white locus was prepared after cleaving DNA from various mutants with restriction endonucleases (Levis *et al.*, 1982; Goldberg *et al.*, 1982); transcripts were identified, the major one being a 2.6 kb poly-(A)⁺ RNA found in embryos, larvae, pupae, and adults (O'Hare *et al.*, 1983; Pirrotta *et al.*, 1983; Fjose *et al.*, 1984; Pirrotta and Bröckl, 1984). The nucleotide sequence of more than 14 kb of white DNA and the probable structures of the introns and exons were determined (O'Hare *et al.*, 1984). The presence of at least four introns is indicated (Pirrotta and Bröckl, 1984); a 3 kb intron separates the proximal (centromere) sequences from the distal sequences (O'Hare *et al.*, 1984). DNA sequences required for normal expression of w^+ were found to be within a 9.9 kb segment of the gene, with regulatory elements in the 5' flanking region (Levis *et al.*, 1985; Pirrotta *et al.*, 1985); these regions include sequences for Malpighian tubule expression and testis sheath pigment as well as those regulating the pattern of eye pigmentation and zeste interaction.

Insertions associated with w mutations were found throughout most of the region of the 2.6 kb w^+ transcript as well as 6 kb upstream. Alleles associated with these insertions or with molecular deletions or duplications are listed on the figure and table on the following page.

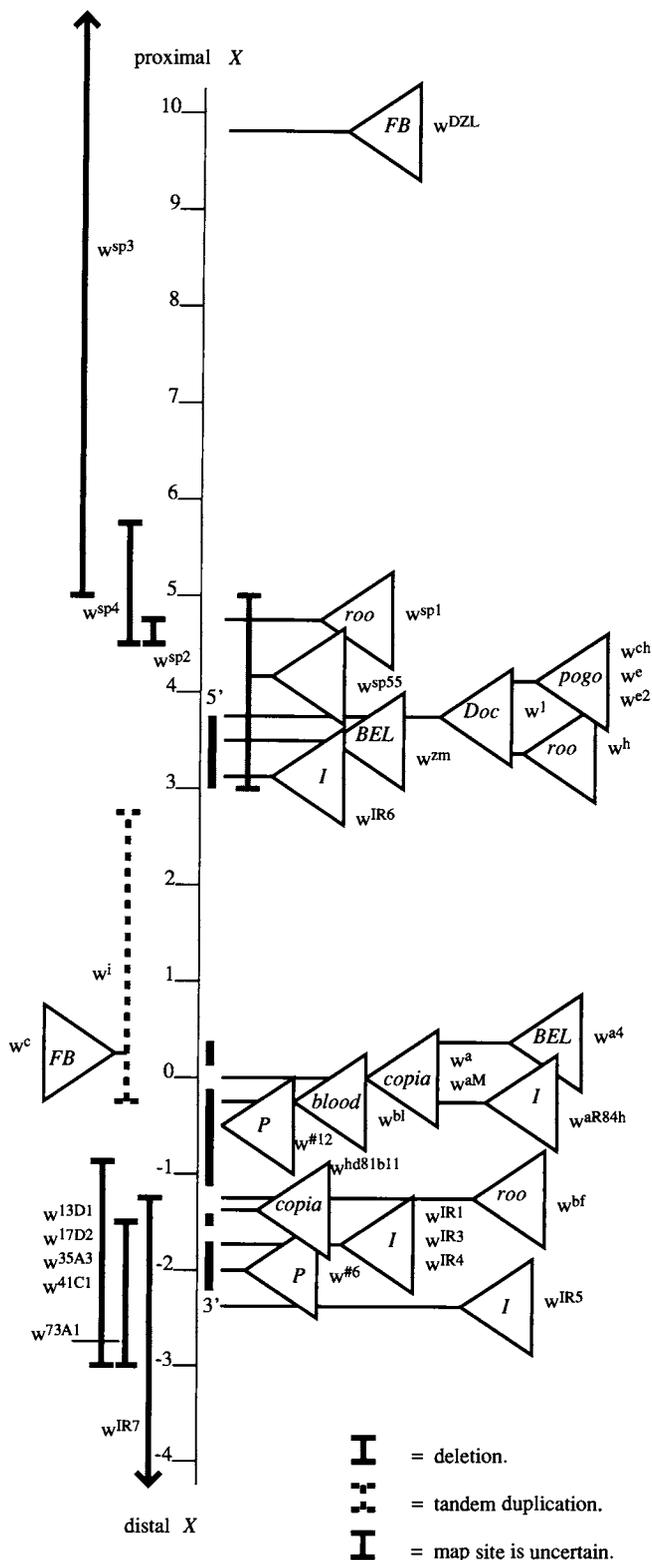
The mottler of white locus interacts with white alleles that are transposon-insertion mutants, producing a variegated eye color (Birchler *et al.*, 1989). All of the w sites involved are within the structural part of the gene.

w^+ (red-eyed) flies were produced by *P*-element-mediated germ-line transformation, a 12-14 kb segment spanning the w^+ locus (in a *P*-transposon vector) being inserted in various new chromosomal locations (Gehring *et al.*, 1984; Hazelrigg *et al.*, 1984) and with various amounts of upstream sequences (Levis *et al.*, 1984; Pirrotta *et al.*, 1985). These w^+ transformants show the same tissue-specific transcript accumulation as wild-type flies. They also show dosage compensation, but differ markedly in their interactions with *z*. Temperature shocks given at various times in development to white transformants under the control of heat shock regulatory sequences indicate the period of maximum expression of white to be in the first two days of the pupal period (Steller and Pirrotta, 1985).

Extensive amino acid similarity has been found between the putative protein products of the white and brown genes, both involved in the pteridine pathway (Dreesen *et al.*, 1988) and the protein products of the white and scarlet genes, both involved in the ommochrome pathway (Tearle *et al.*, 1989).

While the amount of detectable eye pigment variation among 64 *X* chromosome lines from three collections in North Carolina, Texas, and Fukuoka, Japan, was not large, these lines showed a very large amount of molecular variation in a 45-kb region of the white locus, a total of 109 polymorphisms being found (Miyashita and Langley, 1988).

other information: The original *TE35A* [= *TE146(Z)*] which carries two copies of w^+rst^+ in tandem order ($w^+rst^+w^+rst^+$) is prevented by certain rearrangements on the homologous chromosome from interacting with z^1 , but a derivative of *TE35A* which carries two copies of w^+rst^+ in the reversed order ($rst^+w^+w^+rst^+$) is not affected by these rearrangements in regard to its interaction with z^1 (Gubb *et al.*, 1990).



white molecular map

Modified from data supplied by O'Hare, Murphy, Levis, and Rubin, 1984, J. Mol. Biol. 184: 437-55. By D. Conner.

allele	mut. agent	size of agent	map site (kb) ^α	ref ^β
w¹	<i>Doc</i>	5 kb	+3.71	14, 16
w^{5a}	translocation break		+4.38 to +4.44	12
w^{13D1}	deletion		-3.0 to -0.7	12
w^{17D2}	deletion		-3.0 to -0.7	12
w^{25B2}	deletion		-1.5 to -0.7	12
w^{31D1}	translocation break		-3.0 to -0.7	12
w^{35A2}	deletion		-3.0 to -0.7	12
w^{41C1}	deletion		-3.0 to -0.7	12
w^{53A}	dup (3A-3C) + deletion		+2.0 to +4.8	12
w^{66A}	deletion			12
w^{73A1}	deletion		-3.0 to -1.5	12
w^{83B}	translocation break		-3.0 to -0.7	12
w¹⁰⁸	deletion		+3.2 to +4.4	13
w^{123D}	inversion break (3C) in <i>Trp(3:1)</i>		-0.7 to +0.4	12
w¹³²	insertion	10 kb	+0.4 in large intron	13
w^{#6}	<i>P</i>	1.1 kb	-2.02	11, 14
w^{#12}	<i>P</i>	1.6 kb	-0.51	11, 14
w^{aγ}	<i>copia</i> insertion in intron 2	5.0 kb	0	2, 7, 8, 10, 11, 16
w^{a4γ}	<i>BEL</i> insertion in intron 2	7.3 kb		8, 16
w^{aMγ}	insertion in <i>copia</i> 5' LTR	2.3 kb		10
w^{aR84hγ}	insertion in <i>copia</i> 3' LTR			10
w^{bfγ}	<i>roo</i>	8.7 kb	-1.13	8, 11, 16
w^{bl}	<i>blood</i>	6 kb	-0.01	1
w^c	<i>FB</i> in <i>wⁱ</i>	9 kb	+0.19	3, 11
w^{ch}	<i>pogo</i> in <i>Doc</i>	0.2 kb	+3.71	O'Hare, 16
w^{DZL}	<i>FB</i>	13 kb	+9.77	9, 11, 16
w^e	<i>pogo</i> in <i>Doc</i>	0.2 kb	+3.71	8
w^{e2}	<i>pogo</i> in <i>Doc</i>	0.2 kb	+3.71	O'Hare
w^{e59}	<i>F</i>		+3.76	O'Hare
w^{hγ}	<i>roo</i> in <i>Doc</i>	5.7 kb	+3.71	11
w^{hd80k}	<i>P</i>		-2.03	8, 11, 14
w^{hd81b11}	<i>copia</i>		-1.3	11, 14
wⁱ	dup of intron 1	2.96 kb	-0.17 to +2.80	3, 11
w^{l+A}	<i>F</i>	3 kb	+0.8	8, 11
w^{IR1}	<i>I</i>	5.4 kb	-1.45	6, 15
w^{IR2}	<i>I</i> into <i>Doc</i>	5.4 kb		6, 15
w^{IR3}	<i>I</i>	5.4 kb	-1.45	6, 15
w^{IR4}	<i>I</i>	5.4 kb	-1.45	6, 15
w^{IR5}	<i>I</i>	5.4 kb	-1.45	6, 15
w^{IR6}	<i>I</i>	5.4 kb	-2.2	6, 15
w^{IR7}	<i>I</i>	5.4 kb	+3.2	6, 15
w^{IR8}	deletion		-1.17 to outside <i>w</i>	6, 15
w^{rlc}	<i>roo</i> in <i>copia</i>		-1.29 to -3.45	4
w^{sp1δ}	<i>roo</i>	8.7 kb	+4.92	8, 11, 16
w^{sp2}	deletion in regulatory region			16
w^{sp3}	deletion		+5 to +22	16
w^{sp4}	deletion in regulatory region		+4.78 to +5.85	11, 16
w^{sp55γc}	retrotransposon insertion	5.8 kb	+3 to +5	8, 16
w^{sp81d5}	deletion			5
w^{zm}	<i>BEL</i>	6.0 kb	+3.43	8, 11, 16

^α Origin = insertion of *w^a copia*; "-" values to left (telomere) end; "+" values to right (centromere) end.

^β 1 = Bingham and Chapman, 1986, EMBO J. 5: 3343-51; 2 = Bingham and Judd, 1981, Cell 25: 705-11; 3 = Collins and Rubin, 1982, Cell 30: 71-79; 4 = Davis, Shen, and Judd, 1987, Proc. Nat. Acad. Sci. USA 84: 174-78; 5 = Davison, Chapman, Wedeen, and Bingham, 1985, Genetics 110: 479-94; 6 = Fawcett, Lister, Kellett, and Finnegan, 1986, Cell 47: 1007-15; 7 = Gehring and Paro, 1980, Cell 19: 897-904; 8 = Levis, O'Hare, and Rubin, 1984, Cell 38: 471-81;

9 = Levis and Rubin, 1982, Cell 30: 543-50; 10 = Mount, Green, and Rubin, 1988, Genetics 118: 221-34; 11 = O'Hare, Murphy, Levis, and Rubin, 1984, J. Mol. Biol. 180: 437-55; 12 = Pastink, Schalet, Vreeken, Parádi, and Eeken, 1987, Mutat. Res. 177: 101-15; 13 = Pastink, Vreeken, and Vogel, 1988, Mutat. Res. 199: 47-53; 14 = Rubin, Kidwell, and Bingham, 1982, Cell 29: 987-94; 15 = Sang, Pelisson, Bucheton, and Finnegan, 1984, EMBO J. 3: 3079-85; 16 = Zachar and Bingham, 1982, Cell 30: 529-41. Also see references in Table 1 of Judd, 1987, Structure and Function of Eukaryotic Chromosomes (Hennig, ed.). Springer-Verlag, Berlin, Heidelberg, pp. 81-94 and Birchler, Hiebert, and Rabinow, 1989, Genes Dev. 3: 73-84.

- Y Shows *mw* mottling (Birchler *et al.*, 1989). w^a suppressed by *Doa* (Rabinow and Birchler, 1989, EMBO J. 8: 879-89).
- δ In spite of the variegated pattern of spots in the eye in this mutant, the transcript is like that of w^+ in size, structure, and amount (Levis *et al.*, 1984; Pirrotta and Bröckl, 1984, EMBO J. 3: 563-68).
- ε w^+ mRNA not detected in this allele. w^{sp55} enhanced by *Doa* (Rabinow and Birchler, 1989).

w^a : white-apricot

phenotype: Placed on the genetic map of white to the right of w^{bf} and the left of w^{ch} . The amount of pigment formed by w^a is a function of gene dose: w^a/w^a female < w^a/Y male = w^a/w^a female < $w^a/w^a/w^a$ female < w^a/w^a male (Muller, 1932). A w^a optic disk transplanted into a wild-type host shows autonomous eye color development (Beadle and Ephrussi, 1936). Deficiencies and duplications for w^a can be produced as a result of nonhomologous exchanges within the white region. w^a gives rise to partial revertants, as w^r (Muller), w^{aM} (Mossige), and w^{a57i} (Green). Eye color is modified in certain mutant combinations. w^a/bw is slightly lighter than w^a . w^a/st is light pinkish yellow (Mainx, 1938) as is w^a/v . $z w^a$ is lighter than either mutant alone, only slightly darker than w^{bf} (Green, 1959a). w^a/rb and w^a/g have nearly white eyes; $w^a w^{ch}$, $w^{bf} w^a$, and w^a in combination with *su(f)* all have white eyes. *su(w^a) w^a* and *su(w^a)^G w^a* have browner eyes than w^a . The triple mutant *su(w^a) w^a su(f)* has eyes only slightly lighter than w^a (Levis *et al.*, 1984). $w^{Bwx} w^a$ is like w^a (Judd). $w^a/+$ has lighter eyes than $+/+$ in *v* homozygotes (Braver, 1953); *Tp(2;3)P* darkens w^a .

Transpositions of w^a and the neighboring gene *rst⁺* have been isolated at more than 120 sites in the genome [Ising and Ramel, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 947-54].

molecular biology: The w^a allele of the white locus has been cloned by retrieving previously cloned segments carrying a copy of the transposable element *copia* together with contiguous sequences (Bingham *et al.*, 1981; Gehring and Paro, 1980). Bingham and Judd (1981) have shown that *copia* homology is tightly linked to the site of w^a , *copia* being located at coordinate 0.0 on the physical map of the white locus (Levis *et al.*, 1982, 1984) and inserted in the small second intron (Pirrotta and Bröckl, 1984). The size of the *copia* insertion is 5 kb. The mutant gene codes for major transcripts of 1.25 and 5.7 kb (Levis *et al.*, 1984). A small amount of the normal 2.6 kb white transcript is also found in w^a mutants. *su(w^a)*, which darkens the eyes of w^a flies, results in a higher level of the 2.6 kb transcript, while *su(f)*, which decreases the amount of pigment, results in a lower level of this transcript. Partial revertants of w^a have been

sequenced and found to retain one LTR of *copia* (Carbonare and Gehring, 1985; Mount *et al.*, 1988; Zachar *et al.*, 1985).

w^{bf} : white-buff

phenotype: Occupies a recombination site between w^{Bwx} and w^a (Judd, 1959). Spontaneous reversions reported by Redfield (1952, DIS 26: 28). w^{bf} ; *st* has white eyes (Mainx, 1938, Z. Indukt. Abstamm. Vererbungsl. 75: 256-76). Eyes of $w^{bf} rb$ and $w^{bf} g$ are lighter than the eyes of w^{bf} , *rb*, or *g* (Green, 1959a).

molecular biology: w^{bf} is associated with an insertion of the transposable element *roo* (*B104*) at coordinate -1.13 on the molecular map of white within the intron between the third and fourth exons (Levis *et al.*, 1984; Zachar and Bingham, 1982).

w^{bl} : white-blood

phenotype: Located distal to w (MacKendrick and Pontecorvo, 1952) and w^e (Judd, 1958; Green, 1959a). At 19°, eye color as dark as *pn*; at 30°, as light as w^{bf} or w^i ; sensitivity greatest 40-48 hr after pupation (Ephrussi and Herold, 1945, Genetics 30: 62-70).

molecular biology: The 6 kb transposable element *blood* is inserted in this allele (Bingham and Chapman, 1986, EMBO J. 5: 3343-51).

w^{Bwx} : white-Brownex

phenotype: Located distal to w^{bf} (Judd, 1957, 1959). Reduces recombination in the *y-spl* interval. Heterozygotes between w^{Bwx} and other white alleles or deficiencies are indistinguishable in eye color from w^{Bwx}/w^{Bwx} . The double mutant $w^{Bwx} w^{col}$ is lighter than either single mutant, but w^{Bwx}/w^a and w^{Bwx}/w^{bf} are indistinguishable from w^a and w^{bf} , respectively.

molecular biology: Restriction map of w^{Bwx} resembles that of Canton-S wild type except for a deletion of 150 bp (Zachar and Bingham, 1982).

w^c : white-crimson

phenotype: Maps at the same site as w^a . Derivatives of w^c may be stable (w^+ for example) or mutable (such as w^{dc} and w^{di}) and include both point mutations and deficiencies (Green, 1967, Genetics 56: 467-82). The mutations take place in both males and females, may occur in clusters, and do not appear to involve recombination. Transpositions of a segment of the *w* gene that includes w^c to different locations on the third chromosome have been recovered and are mutable (Green, 1969, Genetics 61: 423-28; Green, 1976).

molecular biology: w^c is derived from w^i which is a tandem duplication of 2.9 kb of the white locus; it contains an *FB* element inserted in the proximal copy of the duplication, near the junction of the duplicated segments (Collins and Rubin, 1982).

w^{cf} : white-coffee

phenotype: Located near w^{Bwx} and just distal to w^a (Welshons and Nicoletti, 1963, DIS 38: 80). Females heterozygous for w^{cf} and w , w^a , w^{co} , w^{ch} , w^{bl} , w^{col} , or w^{sat} have eye color of w^{cf} homozygous females. $w^{cf}/+$ flies wild-type.

w^{ch} : white-cherry

phenotype: Occupies site proximal to w^a and distal to w^{sp} (Lewis, 1956). Eyes light in double mutant with *rb* or *g*,

white with w^a . Enhanced by P and $e(w^e)$; suppressed by $Su(w^{ch})$, making eyes brownish (Rasmuson, 1970, *Hereditas* 65: 83-96).

w^{co} : white-coral

phenotype: Located distal to w^l (MacKendrick and Pontecorvo, 1952). Enhanced by $e(w^e)$; lightens rb and g (Green, 1959a). w^{co},st has yellow eyes (Mainx, 1938).

w^{DZL} : white-Dominant-zeste-like

phenotype: w^{DZL} is located in or immediately proximal to the rightmost set of previously-defined white mutant sites (Bingham, 1981). While this mutant affects the pigmentation of the eyes, it has no effect on the color of the larval Malpighian tubules or the testis sheath of adult males. w^{DZL} shows synapsis-dependent dominance over w^+ . It is a highly mutable allele (like w^c), giving rise spontaneously to w^+ and w^- derivatives with a frequency of 0.5-1.5%. Interactions between w^{DZL} and z are summarized in the allele table. It was observed that, when carrying the wild-type allele of z , w^{DZL}/w^- females have brown eyes; with z^l , however, hemizygous w^{DZL} females have yellow eyes (Bingham, 1980, *Genetics* 95: 341-53).

molecular biology: Physical analysis of w^{DZL} shows that the mutant phenotype results from the insertion (proximal to white) of a complex transposable element made up of sequences normally found at 21D ("unique segment sequences") flanked by sequences of two FB transposons (Levis, Collins, and Rubin, 1982, *Cell* 30: 551-65; Zachar and Bingham, 1982, *Cell* 30: 529-41; Bingham and Zachar, 1985, *Cell* 40: 819-25). A transcript 1.3 kb larger than the major (2.6 kb) w^+ transcript is found in w^{DZL} adult heads and head precursor tissues; this is a composite transcript, made up of sequences from the major w^+ transcript and from the w^{DZL} unique segment (Bingham and Zachar, 1985; Zachar *et al.*, 1985). In w^{DZL} flies, a reduction in the levels of the w^+ transcript is observed in adult head and head precursor tissues, but not in abdominal tissues. Revertants of w^{DZL} show loss of most of the unique segments of the insertion with 1.9 and 6.2 kb of the insertion remaining (Levis and Rubin, 1982; Zachar and Bingham, 1982).

w^e : white-eosin

phenotype: Placed proximal to w^a (Green, 1959a). Amount of pigment formed by w^e not a function of gene dose: w^e female = w^e male < w^e/w^e male = w^e/w^e female < $w^e/w^e/w^e$ female (Muller, 1932). Mutant enhanced by P , cru , and whg as well as by $E(w^e)$. Lightens rb and g (Green, 1959a). A w^e optic disk transplanted into a wild-type host shows autonomous eye color development (Beadle and Ephrussi, 1936).

molecular biology: w^e is derived from w^l and carries an insertion of the transposable element $pogo$ within the w^l Doc insertion (O'Hare).

w^i : white-ivory

phenotype: Placed on the genetic map distal to w^l (MacKendrick, 1953, *DIS* 27: 100). w^i is unstable, reverting spontaneously to w^+ with a frequency of 5×10^{-5} in w^l/w^i females and 5×10^{-6} in w^l/Y males and $w^i/Df(1)w$ females (Lewis, 1959, *Genetics* 44: 522; Bowman, 1965, *Genetics* 52: 1069-79). The frequency of germinal reversions and of somatic reversions in larval eye tissue is increased by X rays (Lewis, 1959; Bowman and Green, 1964, *Genetics* 50: 237). No dosage com-

pensation shown by the mutant (Green 1959a). Recombination between flanking w alleles reduced in w^i , but restored in its revertants (Bowman, 1965; Bowman and Green, 1966, *Genetica* 37: 7-16).

molecular biology: Cloning and analysis of Southern blots of w^i DNA indicate the presence of a 2.9 kb duplication within the white locus (Karess and Rubin, 1982, *Cell* 30: 63-69). Revertants are produced by excision of one copy of the repeat in derivatives w^{i+B} , w^{i+C} , w^{i+D} , and w^{i+E} . w^{i+A} and the partial revertant w^{ip} are insertion mutations that are more complex, their altered phenotype apparently resulting from the insertion of new DNA into w^i and the loss of some w^i DNA.

w^m : white-mottled

phenotype: There are many w alleles that show variegated eye color. The w^m mutants most commonly used for variegation studies are w^{m4} and $w^{m264-58}$. In these alleles, extra heterochromatin partially suppresses eye mottling (Gowen and Gay, 1933, *Proc. Nat. Acad. Sci. USA* 19: 122-26; Koliantz, Hartmann-Goldstein, and Fuller, 1984, *Heredity* 52: 203-13; Koliantz and Hartmann-Goldstein, 1984, *Heredity* 53: 215-22; Baker and Spofford, 1959, *Univ. Texas Publ.* 5914: 135-54; Spofford, 1959, *Proc. Nat. Acad. Sci. USA* 45: 1003-07). In $w^{m264-58}$, variegation less (more wild-type in color) in homozygous females than in heterozygous females. Color variegation found in the testis-sheath as well as the eyes of $w^{m264-58}$ male flies (Baker, 1968, *Adv. Genet.* 14: 133-169). In some lines, less variegation when paternally inherited; in others, less variegation when maternally inherited or no parental effect. Mottling in w^{m4} and w^{m4h} is enhanced by $E(var)7$ and $E(var)c^{101}$ (Reuter and Wolff, 1981, *Mol. Gen. Genet.* 182: 516-19); mottling in w^{m4} and $w^{m264-58}$ is suppressed by $Su(var)$ (Spofford, 1962, *Genetics* 47: 986-87) and a number of other suppressor mutations (Reuter and Wolff, 1981).

molecular biology: The DNA map location of three white-mottled mutations has been determined (Tartof, Bishop, Jones, Hobbs, and Locke, 1989, *Dev. Genet.* 10: 162-76). The euchromatic breakpoint of $In(1)w^{m4}$ is at -24.5 kb, and the breakpoints of $In(1)w^{m51b}$ and $In(1)w^{mMc}$ are at about -21.3 kb (origin being the site of the $copia$ insertion in w^a).

w^{sp} : white-spotted

phenotype: Located proximal to w^{ch} and distal to w^{DZL} . w^{sp} affects that deposition of the eye pigments, resulting in a variegated phenotype, but does not affect the pigmentation of the larval Malpighian tubules. Testis pigmentation varies with different alleles, w^{sp3} males having unpigmented testes, but w^{sp1} and w^{sp2} males showing enhanced testis pigmentation (Davison *et al.*, 1985; Pirrotta, Stellar, and Bozzetti, 1985, *EMBO J.* 4: 3501-08; Judd, 1987). Partial complementation occurs between w^{sp} alleles and certain other w mutations when they are synapsed; for example, $w^{sp/w}$, $w^{sp/w^{ch}}$, and w^{sp/w^a} females have homogeneous brown eyes (Green, 1959a). The double mutants $w^a w^{sp}$ and $w^{ch} w^{sp}$ have white and pale yellow eyes, respectively. w^{sp} , when heterozygous with a deficiency for all or part of the w locus, produces a phenotype like that of w^{sp} homozygotes (Green, 1959c). In the presence of z^l , two synapsed copies of w^{sp} in *trans* (or tandemly repeated)

result in yellow-eyed females; z^1 females with one copy of w^{sp} have wild-type eye color. A specific regulator of the w^{sp} eye phenotype, $su(w^{sp})$, has been isolated as a partial revertant of w^{sp1} (Chapman and Bingham, 1985); this suppressor restores wild-type eye color to w^{sp1} , w^{sp2} , w^{sp3} , and w^{sp4} flies, but not to the w^{sp81d} mutant (Davison *et al.*, 1985).

molecular biology: The w^{sp} region has been cloned (Zachar and Bingham, 1982) and sequenced (O'Hare *et al.*, 1984); it is located 1.0-1.9 kb upstream from the 5' end of the start of w^+ transcription (Zachar and Bingham, 1982; O'Hare *et al.*, 1983). w^{sp} shows sequence homology and behavior analogous to enhancer sequences (Davison *et al.*, 1985). The lesions responsible for w^{sp1} , w^{sp2} , and w^{sp4} have been analyzed by sequencing cloned portions of these alleles; the breakpoints of w^{sp3} and w^{sp81d} have also been established by molecular methods. w^{sp1} carries *roo*; the other alleles are molecular deletions. Sensitivity to the effect of $su(w^{sp})$ on eye pigmentation is deleted by w^{sp81d} but not by w^{sp3} (Davison *et al.*, 1985).

w^{zm} : white-zeste mottled

phenotype: w^{zm} is located to the right of w^a and to the left of w^1 . It is an unstable white allele, mutating to derivatives, most of which are unstable (Judd, 1963, Proc. Int. Congr. Genet. 11th, Vol. 1: 3-4; 1964, DIS 39: 60). Since all z^+ w^{zm} males (as well as z^+ w^{zm}/z^+ w^{zm} females) have wild-type eye color, the mutant z was used as an indicator of the mutability of w^{zm} strains. Derivatives of w^{zm} (Kalisch and Becker, 1970, Mol. Gen. Genet. 107: 321-35) include w^{z1} (from the $z w^{zm}$ stock), w^{zms} (from the $z w^{zm}$ stock), and w^{zmr} , w^{zmsz} and w^{zmsw} (from the $z w^{zms}$ stock). Only w^{z1} is stable. The mutants were often recovered in clusters. w^{zms} reverts to w^{zm+} (eye color between $z w^{zm}$ and $z w^+$) and the white-eyed w^{zmsz} and w^w . Other derivatives (w^z , w^{zh} , w^{zs}) were recovered by Judd (1957; 1969, Genetics 61: s29).

molecular biology: w^{zm} carries an insertion of the transposable element *BEL* near the 5' junction of the first intron at +3.5 (O'Hare *et al.*, 1984). This insertion does not appear to alter the size of the 2.6 kb w^+ transcript in w^{zm} and $z w^{zm}$ flies (Levis *et al.*, 1984).

W: Wrinkled

location: 3-46.0.

origin: Recovered among progeny of female exposed to stratosphere.

discoverer: Jollos, 1936.

references: 1936, Natl. Geograph. Soc. Tech. Papers, Stratosphere Ser. No. 2: 153-57.

Jollos and Waletzky, 1937, DIS 8: 9.

phenotype: Homozygote viable. Wings remain small and unexpanded. Black spots on head beside proboscis or ocelli. Heterozygous female like homozygote but less extreme. Male much less extreme; wings often expanded but wrinkled, blistered, and surface finely pebbled and grayish; no overlap with wild type. Suppressed by *D* in male and nearly so in female. From prepupal stage through adult, wing bases abnormally narrow, possibly preventing flow of body fluid in sufficient quantity to expand wings [Waddington, 1940, J. Genet. 41: 75-139 (fig.)]. RK1 as dominant.

cytology: Located at 75C5-D3.

W13: see *T(1;4)A1*

*** wa : warty**

location: 1-64.4 (based on location of wa^2 ; wa said to be near *car*).

origin: Induced by P³².

discoverer: Bateman, 1950.

references: 1950, DIS 24: 56.

phenotype: Eyes rough with scattered enlarged facets.

Occasional notched wing tip. Penetrance low. Viability variable. Male infertile in proportion to degree of expression. Heterozygous female often infertile. RK3.

wa^2

origin: Induced by L-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 77.

phenotype: Eyes irregularly roughened and of varying size and shape; ommatidia deranged. Wing tips rarely notched. RK3.

alleles: Allelism inferred from phenotype and genetic location. One allele each induced by CB. 1540, CB. 3025, and X rays.

waisted: see *ws*

wap: wings-apart

location: 1- {66}.

synonym: *l(1)20Ab*.

discoverer: Lifschytz.

references: Schalet, 1972, DIS 49: 36-37, 64-66.

Schalet and Lefevre, 1973, Chromosoma 44: 183-202.

1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 846-902.

Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31.

Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-52.

phenotype: Semilethal. Most flies die shortly before eclosion. Flies which hatch have wings set slightly apart and a somewhat darker than normal thorax; may have extra crossveins. Mutant males, mutant females, and mutant/deficiency females have the same phenotype, which is more extreme at 29-30°. Viability of heteroallelic compounds usually low (2-9%).

alleles:

allele	origin	discov.	synonym	ref ^α	comments
wap¹	X ray	Lifschytz	<i>l(1)A200</i>	4, 5, 8, 9	
wap²	EMS	Lifschytz	<i>l(1)Q217</i>	5, 7, 8, 9	no maternal effect
wap³	EMS	Lifschytz	<i>l(1)Q464</i>	5, 8	
wap⁴	EMS	Lifschytz	<i>l(1)M155</i>	6	on $y^+ Ymal^+$
wap⁵	X ray	Lefevre	<i>l(1)C243</i>	3	on <i>T(1;3)20A3;75C</i>
wap⁶	X ray	Lefevre	<i>l(1)KC31</i>	3	
wap⁷	X ray	Lefevre	<i>l(1)RF13</i>	3	
wap⁸	HMS	Kramers	<i>l(1)HM33</i>	2	
wap⁹	MR	Eeken	<i>l(1)D48</i>	1, 7	maternal effect

^α 1 = Eeken, Sobels, Hyland, and Schalet, 1985, Mutat. Res. 150: 261-75. 2 = Kramers, Schalet, Paradi, and Huisser-Hoogteyling, 1983, Mut. Res. 107: 187-201; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Lifschytz and Falk, 1968, Mutat. Res. 6: 235-44; 5 = Lifschytz and Falk, 1969, Mutat. Res. 8: 147-55; 6 = Lifschytz and Yakobovitz, 1978, Mol. Gen. Genet. 161: 275-84; 7 = Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31; 8 = Schalet and Lefevre, 1973, Chromosoma 44: 183-220;

9 = Schalet and Lefevre, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902.

cytology: Placed in 20A3; included in *Df(1)DCB1-35c* but not in *Df(1)B12 = Df(1)19E1;20A1-2*.

wapl: wings-apart-like

location: 1-0.7(between *Pgd* and *pn*).

synonym: *wap; l(1)2Dd*.

references: Gvozdev, Gostimsky, Gerasimova, Dubrovskaya, and Braslavskaya, 1975, Mol. Gen. Genet. 141: 269-75.

Gvozdev, Gerasimova, Kovalev, and Ananiev, 1977, DIS 52: 67-68.

Perrimon, Engstrom and Mahowald, 1985, Genetics 111: 23-44.

phenotype: Most alleles are lethal. Semilethal escapers show wings-apart phenotype.

alleles:

allele	origin	discov.	synonym	ref ^α
<i>wapl</i> ¹ β	X ray	Lefevre	<i>l(1)A17</i>	3, 4
<i>wapl</i> ² γ	X ray	Lefevre	<i>l(1)C204</i>	3, 4
<i>wapl</i> ³ γ	X ray	Lefevre	<i>l(1)HC262</i>	3, 4
<i>wapl</i> ⁴	NMU	Gvozdev	<i>wap</i> ³	1
<i>wapl</i> ²⁵	EMS	Gvozdev		1
<i>wapl</i> ²⁹	EMS	Gvozdev		1
<i>wapl</i> ³³	EMS	Gvozdev		1
<i>wapl</i> ³⁷	EMS	Gvozdev		1
<i>wapl</i> ³⁸	EMS	Gvozdev		1
<i>wapl</i> ⁴⁰	EMS	Gvozdev		1
<i>wapl</i> ⁴⁴	EMS	Gvozdev		1
<i>wapl</i> ⁴⁶	EMS	Gvozdev		1
<i>wapl</i> ⁴⁹	EMS	Gvozdev		1
<i>wapl</i> ⁵²	EMS	Gvozdev		1
<i>wapl</i> ⁵⁶	EMS	Gvozdev		1
<i>wapl</i> ⁷³	EMS	Gvozdev		1
<i>wapl</i> ⁷⁴	EMS	Gvozdev		1
<i>wapl</i> ⁷⁹	EMS	Gvozdev		1
<i>wapl</i> ⁸⁶	EMS	Gvozdev		1
<i>wapl</i> ⁹⁵	EMS	Gvozdev		1
<i>wapl</i> ¹¹⁰	α ray	Gvozdev		1
<i>wapl</i> ¹¹³ γ	EMS	Perrimon	<i>l(1)113p</i>	4
<i>wapl</i> ^{HMS}	HMS		<i>l(1)HM404</i>	2

^α 1 = Gvozdev, Gerasimova, Kovalev, and Ananiev, 1977, DIS 52: 67-68; 2 = Kramers, Schalet, Paradi, and Huizer-Hoogteyling, 1983, Mut. Res. 107: 187-201; 3 = Lefevre, 1981, Genetics 99: 461-80; 4 = Perrimon, Engstrom, and Mahowald, 1985, Genetics 111: 23-44.

^β Early pupal lethal with apparently normal imaginal disks; mitotic index reduced and metaphase chromosomes abnormally condensed; germ-line clones devoid of maternal lethal effect.

^γ Lethal at larval-pupal boundary and exhibit small disk phenotype; homozygous germ-line clones produce zygotes that arrest in preblastoderm stage.

cytology: Placed in 2D4-6 since included in *Df(1)JC105 = Df(1)2D4-6* and *Df(1)Pgd-kz = Df(1)2D3-4;2F5* but not in *Df(1)64c18 = Df(1)2E1-2;3C2*.

wapm: wings-apart mimic

location: 1-{45}.

discoverer: Schalet.

phenotype: Wings set slightly apart; viability good; females fertile.

cytology: Placed in 12D3 based on the *wapm* phenotype of *Df(1)HA92/Df(1)RK2 = Df(1)12A6-7;12D3/Df(1)12D2-E1;13A2-5*.

warped: see *wp*

warty: see *wa*

Washed eye: see *We*

water wings: see *wtw*

wavoid: see *wd*

wavoidlike: see *wdl*

wavy: see *wy*

waxy: see *wx*

wb: wing blister

location: 2-49.9 (G. Maroni; 0.2 cM to left of *Adh*).

synonym: *l(2)br1* (Woodruff and Ashburner, 1979); A1 group (O'Donnell *et al.*, 1977).

references: O'Donnell, Mandel, Krauss, and Sofer, 1977, Genetics 86: 553-66.

Woodruff and Ashburner, 1979, Genetics 92: 133-49.

Ashburner, Faithfull, Littlewood, Richards, Smith, Velisariou, and Woodruff, 1980, DIS 55: 193-95.

phenotype: Most of the mutants are characterized by homo- or hemizygous lethality, but a visible phenotype has also been reported. O'Donnell *et al.*, 1977, describe a bent-down-wing phenotype occurring when one of their lethals is crossed to other lethals in the same complementation group and the same phenotype when three of their lethals are crossed to deletions for the locus. Woodruff and Ashburner, 1979, describe the viable and fertile wing blister phenotype (a roughly circular area around the crossveins of the wing, forming a blister which often collapses soon after eclosion, distorting the wing blade). The allele showing this phenotype (*wb*¹) is viable and fertile in homozygotes and hemizygotes as well as in heteroallelic combinations with lethal alleles.

alleles: Viable and lethal (or semilethal) alleles are described in the following table.

allele	origin	discoverer	ref ^α	comments
<i>wb</i> ¹	EMS	Ashburner	1, 3	wing blister, viable, fertile
<i>wb</i> ³	EMS	Ashburner	1	wing blister, viable, fertile
<i>wb</i> ⁴	EMS	Ashburner	1	wing blister, viable, fertile
<i>wb</i> ^{BMW22}	EMS	Ashburner	1	lethal
<i>wb</i> ^{BMW32}	EMS	Ashburner	1	lethal
<i>wb</i> ^{CH18}	EMS	O'Donnell	2	lethal
<i>wb</i> ^{CH20}	EMS	O'Donnell	2	lethal
<i>wb</i> ^{CH38}	EMS	O'Donnell	2	lethal
<i>wb</i> ^{CH41}	EMS	O'Donnell	2	lethal
<i>wb</i> ^{CH57}	EMS	O'Donnell	2	lethal
<i>wb</i> ^{CH67}	EMS	O'Donnell	2	lethal
<i>wb</i> ^{CH70}	EMS	O'Donnell	2	lethal
<i>wb</i> ^{CR1}	EMS	Ashburner	1, 3	lethal
<i>wb</i> ^{DM10}	EMS	O'Donnell	2	lethal; semilethal over <i>Df(2L)fn7</i>
<i>wb</i> ^{GM2}	EMS	Maroni	1	lethal
<i>wb</i> ^{HG7}	EMS	Ashburner	1	lethal
<i>wb</i> ^{HG9}	EMS	Ashburner	1	lethal
<i>wb</i> ^{HG10}	EMS	Ashburner	1	lethal
<i>wb</i> ^{HG14}	EMS	Ashburner	1	lethal
<i>wb</i> ^{HG16}	EMS	Ashburner	1	lethal
<i>wb</i> ^{HG17}	EMS	Ashburner	1	lethal
<i>wb</i> ^{HG18}	EMS	Ashburner	1	lethal
<i>wb</i> ^{HG19}	EMS	Ashburner	1	lethal
<i>wb</i> ^{HG24}	EMS	Ashburner	1	lethal
<i>wb</i> ^{HG26}	EMS	Ashburner	1	lethal
<i>wb</i> ^{L412}	EMS	Lindsley	1	lethal
<i>wb</i> ^{OK14}	EMS	O'Donnell	2	lethal

allele	origin	discoverer	ref ^α	comments
wb <i>P115</i>	HD	Shelton	1	lethal
wb <i>PA43</i>	EMS	Ashburner	1	lethal
wb <i>SF11</i>	EMS	Woodruff	3	lethal
wb <i>SF19A</i>	EMS	Woodruff	1, 3	lethal
wb <i>SF20</i>	EMS	Woodruff	3	lethal
wb <i>SF25</i>	EMS	Woodruff	1, 3	lethal
wb <i>SF30</i>	EMS	Woodruff	3	lethal

^α 1 = Ashburner; 2 = O'Donnell, Mandel, Krauss, and Sofer, 1977, Genetics 86: 553-66; 3 = Woodruff and Ashburner, 1979, Genetics 92: 133-49.

cytology: Placed in 34F4 since included in *Df(2L)fn1 = Df(2L)34F4-35A1;35D5-7* but not in *Df(2L)A217 = Df(2L)34F5;35B3*.

wbl: windbeutel

location: 2-86.

synonym: *wind*.

references: Anderson, 1987, TIG 3: 91-97.

Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

phenotype: Maternal expression of *wbl*, one of the "dorsal group" genes, is required for the production of all lateral and ventral pattern elements. In the absence of the wild-type allele, all embryonic cells differentiate dorsal epidermis.

alleles: One allele has been identified (Schüpbach and Wieschaus).

***wd: wavoid**

location: 2-40.

origin: Spontaneous.

discoverer: Kellen, 1941.

references: Kellen, 1945, Genetics 30: 12.

phenotype: Wings waved. Variable penetrance and expressivity, especially in male. Partially suppressed by *y* in both sexes. RK2.

wdh: see *sli*

wdl: wavoidlike

location: 2-39.

origin: Induced by ethyl methanesulfonate.

references: Sandler, 1977, Genetics 86: 567-82.

phenotype: Wavy wings with incomplete L5 vein.

Homozygous viable but female semisterile. Maternal effect results in death of zygotes before egg hatch and is influenced by the X- or Y-chromosome heterochromatin carried by the zygote; effect less severe at 19° than at 25°. Mutation semilethal when heterozygous with a deficiency for the locus.

cytology: Located in 32A-32E; included in *Df(2L)J39 = Df(2L)31A-B;32D-E* but not in *Df(2L)J27 = Df(2L)31B-D; 32A*.

other information: May be an allele of the lost mutation *wd* reported by Kellen (1945, Genetics 30: 12).

***wdn: wings down**

location: 3-100.

discoverer: Morgan.

references: 1929, Carnegie Inst. Washington Publ. No. 399: 187.

phenotype: Wings extended and drooping or even directed ventrally, broad with close crossveins. Overlaps wild type. Low viability. RK3.

***we: wee**

location: 1-3.0.

origin: X ray induced.

discoverer: Muller, 2615.

references: 1935, DIS 3: 30.

Lefevre and Green, 1972, Chromosoma 36: 391-412.

phenotype: Fly dwarfed. Eyes rough; bristles fine; wings spread. Fertility very low. RK2.

***We: Washed eye**

location: 3-43.0.

origin: Spontaneous.

discoverer: Andres, 42e7.

references: 1943, DIS 17: 48.

phenotype: Dominant modifier of *w* that produces partial reversion. Produces spot of dilute red pigment varying in size from dot to nearly whole eye. Homozygous lethal. Classification, fertility, and viability of heterozygote excellent. RK2.

We: see see *Dr*^{We}

weak: see *wk*

wee: see *we*

welt: see *wt*

weltlike: see *wtl*

wg: wingless

location: 2-30.0.

synonym: *Dint-1*.

references: Sharma, 1973, DIS 50: 24, 134.

Sharma and Copra, 1976, Dev. Biol. 48: 461-65.

Babu, 1977, Mol. Gen. Genet. 151: 289-94.

Morata and Lawrence, 1977, Dev. Biol. 56: 227-40.

1977, Nature (London) 265: 211-16.

Deak, 1978, Dev. Biol. 66: 422-41.

Nüsslein-Volhard and Wieschaus, 1980, Nature (London) 287: 785-801.

Vandervorst and Ghysen, 1980, Nature (London) 286: 65-67.

Nüsslein-Volhard, Wieschaus, and Kluding, 1983, DIS 59: 158-60.

1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

Babu and Bhat, 1986, Mol. Gen. Genet. 205: 483-86.

Baker, 1987, EMBO J. 6: 1765-73.

Johnston, Phillips, and Lawrence, 1987, Cell 50: 859-63.

Ryjssewijk, Schuermann, Wagenaar, Parren, Weigel, and Nüsse, 1987, Cell 50: 649-57.

Scott and Carroll, 1987, Cell 51: 689-98.

Baker, 1988a, Dev. Biol. 125: 96-108.

1988b, Development 102: 489-97.

1988c, Development 103: 289-98.

Ingham, Baker, and Martinez-Arias, 1988, Nature (London) 331: 73-75.

Martinez-Arias, Baker, and Ingham, 1988, Development 103: 157-70.

Mohler, 1988, Genetics 120: 1061-72.

Uzsvölgyi, Kiss, Pitt, Arsenian, Ingvarsson, Udvardy, Hamada, Klein, and Sümegi, 1988, Proc. Nat. Acad. Sci. USA 85: 3034-38.

Van den Heuvel, Nüsse, Johnston, and Lawrence, 1989, Cell 59: 739-49.

phenotype: The *wg* gene is involved both in controlling the segmentation pattern of embryos by affecting the pos-

terioormost cells of each parasegment (Baker, 1987) and in controlling the imaginal disk pattern of the meso- and meta-thoracic segments that develop into wing, halter, and notum in pupae and adults (Sharma, 1973; Sharma and Copra, 1976; Morata and Lawrence, 1977). The gene is believed to control segment organization through an intercellular signaling mechanism (Baker, 1987, 1988b; Cabrera, Alonso, Johnston, Phillips, and Lawrence, 1987, Cell 50: 659-63; Ryjsewijk *et al.*, 1987; Martinez-Arias *et al.*, 1988). Mutants may be viable as adults or lethal as embryos or pupae. In embryonic lethal alleles (Babu, 1977; Nüsslein-Volhard and Wieschaus, 1980), each segment shows a mirror-image duplication of the denticle bands at the expense of naked cuticle so that a continuous sheet of denticles (instead of repeated denticle bands) is produced (Cabrera *et al.*, 1987). Dorsal abnormalities are more extreme than ventral ones, the dorsal cuticle being greatly reduced and covered by fine hairs (Baker, 1988a). These embryos lack head structures and filzkörper (Perrimon and Mahowald, 1987, Dev. Biol. 119: 587-600). In the nervous system, a single neuron, RP2, is missing; other neurons in the lineage are normal (Patel, Schafer, Goodman, and Holmgren, 1989, Genes Dev. 3: 890-904). The temperature-sensitive period for *wg*^{l-12}, a heat-sensitive allele that is lethal at 25° (Baker, 1988a; Mohler, 1988), lies between gastrulation and the beginning of dorsal closure (11 hours after egg laying at 25°). In pupal lethal and adult viable alleles, the ready-to-emerge pupae and the adults lack one or both wings and/or halteres, and there is a corresponding duplication of the meso- and metanota (Sharma, 1973; Sharma and Copra, 1976; Morata and Lawrence, 1977; Deak, 1978). This adult phenotype shows incomplete penetrance and variable expressivity and is affected by the ability of the wingless gene to function during the larval period (Baker, 1988a). Low temperature fails to rescue heteroallelic combinations of *wg*¹ or *wg*^{l-18} with the heat-sensitive allele *wg*^{l-12} after the larval stages. Lethal as well as viable *wg* alleles are not cell-autonomous in adult mosaics (Babu and Bhat, 1986; Morata and Lawrence, 1977; Baker, 1988a).

alleles: Mutant alleles of *wg* may be viable, showing a visible phenotype involving loss of wings and/or halteres, or they may be homozygous lethal (usually as embryos but sometimes as pupae). Both types of mutants are included in the following table:

allele	origin	discoverer	synonym	ref ^α	comments
<i>wg</i> ¹	X ray	Sharma		4,6-8	viable
<i>wg</i> ²	EMS	Sharma		8	viable
<i>wg</i> ³	EMS	Sharma		8	viable
<i>wg</i> ³⁻¹	EMS	Sharma		8	viable
<i>wg</i> ⁴	EMS	Sharma		8	viable
<i>wg</i> ⁴⁻¹	EMS	Sharma		8	viable
<i>wg</i> ⁵	EMS	Sharma		8	viable
<i>wg</i> ⁷	EMS	Sharma		8	viable
<i>wg</i> ¹⁰	EMS	Sharma		8	viable
<i>wg</i> ¹¹	EMS	Sharma		8	viable
<i>wg</i> ¹²	X ray	Baker	<i>wg</i> ^{CX1}	3	viable
<i>wg</i> ^{l-1}	EMS	Babu		1	embryonic lethal
<i>wg</i> ^{l-2}	EMS	Babu		1,2	embryonic lethal
<i>wg</i> ^{l-3}	EMS	Babu		1	embryonic lethal
<i>wg</i> ^{l-4}	EMS	Babu		1	embryonic lethal
<i>wg</i> ^{l-5}	EMS	Babu		1	embryonic lethal
<i>wg</i> ^{l-6}	EMS	Babu		1	embryonic lethal
<i>wg</i> ^{l-7}	EMS	Nüsslein-Volhard	<i>wg</i> ^{6K}	9	embryonic lethal
<i>wg</i> ^{l-8}	EMS	Nüsslein-Volhard	<i>wg</i> ^{1G}	9	embryonic lethal
<i>wg</i> ^{l-9}	EMS	Nüsslein-Volhard	<i>wg</i> ^{1S}	9	embryonic lethal

allele	origin	discoverer	synonym	ref ^α	comments
<i>wg</i> ^{l-10}	EMS	Nüsslein-Volhard	<i>wg</i> ^{11D}	9	embryonic lethal
<i>wg</i> ^{l-11}	EMS	Nüsslein-Volhard	<i>wg</i> ^{1I}	9	embryonic lethal
<i>wg</i> ^{l-12} β	EMS	Nüsslein-Volhard	<i>wg</i> ^{1L} , <i>wg</i> ^{1S}	4,9	hs lethal (embryo)
<i>wg</i> ^{l-13}	EMS	Nüsslein-Volhard	<i>wg</i> ^{1N}	9	embryonic lethal
<i>wg</i> ^{l-14}	HD	Baker	<i>wg</i> ^{CPI}	3	embryonic lethal;
<i>wg</i> ^{l-15}	X ray	Baker	<i>wg</i> ^{CX2}	3,4	embryonic lethal
<i>wg</i> ^{l-16}	X ray	Baker	<i>wg</i> ^{CX3}	3,5	pupal lethal
<i>wg</i> ^{l-17}	X ray	Baker	<i>wg</i> ^{CX4}	3,5	embryonic lethal
<i>wg</i> ^P		Baker		4,5	pupal lethal; <i>In(2L)28A1-3;32E-F</i>

^α 1 = Babu, 1977, Mol. Gen. Genet. 151: 289-94; 2 = Babu and Bhat, 1986, Mol. Gen. Genet. 205: 483-86; 3 = Baker, 1987, EMBO J. 6: 1765-73; 4 = Baker, 1988a, Dev. Biol. 125: 96-108; 5 = Baker, 1988b, Development 102: 489-97; 6 = Sharma, 1973, DIS 50: 25, 134; 7 = Sharma and Copra, 1976, Dev. Biol. 48: 461-65; 8 = Sharma and Shekaran, 1983, Indian J. Exp. Biol. 21: 143-49; 9 = Tearle and Nüsslein-Volhard, 1987, DIS 86: 209-69.

β At 18°, homozygous *wg*^{l-12} embryos develop normally and hatch to produce larvae that have a wild-type cuticular pattern, but these mutants do not survive to become adults. *wg*^{l-12}/*wg*^P mutants, however, do survive at 16.5° to become phenotypically wild-type adults (Baker, 1988a).

cytology: *wg* has been located in 28A1-3 by *in situ* hybridization to salivary chromosomes of *In(2L)w*^P = *In(2L)28A1-3;32E-F* heterozygotes, two sites being labelled in the inversion chromosome (Baker, 1987).

molecular biology: DNA from the *wg* gene was cloned by transposon tagging with a *P*-element insertion mutant *wg*^{l-18}. Lesions associated with *wg* mutants were located on a molecular map of the region. Alleles involved are listed below:

allele	molecular biology
<i>wg</i> ¹	deletion from +16.8 to +17.1 kb
<i>wg</i> ^{l-15}	deletion of 0.3 kb
<i>wg</i> ^{l-16}	break between +10.8 and 15.0 kb
<i>wg</i> ^{l-17}	deletion of 2 kb at 5' end of gene
<i>In(2L)wg</i> ^P	28A breakpoint between 16.4 and 17.1 kb

A 3.2 kb transcript was obtained and localized in embryos, where it was first found at the time of cellularization of the blastoderm (Baker, 1987, 1988b). The RNA accumulates as epidermal stripes in the most posterior cells of each parasegment. Transient accumulation of transcript occurs in the mesoderm and the nervous system. *wg* is also expressed in larvae and pupae, but is scarcely detectable in adults. An antisense RNA made from the *wg* transcript produces phenocopies of wingless when injected into wild-type eggs (Cabrera *et al.*, 1987).

The *Drosophila melanogaster* homolog of the mouse mammary oncogene *int-1* has been located in the same cytological region as wingless, cloned and designated *Dint-1* (Ryjsewijk *et al.*, 1987; Urvölgyi *et al.*, 1988); transcripts of 2.9-3.0 kb have been obtained and found to be at approximately the same position on the molecular map and to show the same expression in embryos as *wg*. The identity of *Dint-1* and *wg* was confirmed by positive cross-hybridization of a *Dint-1* cDNA probe to a partial cDNA clone from the 5' half of the *wg* transcript (Ryjsewijk *et al.*, 1987). A partial sequence of *wg* cDNA was obtained (Cabrera *et al.*, 1987) and found to be identical to a region of the complete *Dint-1* sequence (Ryjsewijk *et al.*, 1987). The deduced *Drosophila* protein sequence shows 54% overall identity to the mouse *int-1* sequence (Ryjsewijk *et al.*, 1987; Urvölgyi *et al.*, 1988).

The cysteine-rich protein of *wg* is found in the same region of the embryo as the *wg* mRNA (Van den Heuvel *et al.*, 1989).

wgo: wings-out

location: 1-16.2.
origin: H³TdR-induced.
references: Perez and Kaplan, 1968, DIS 43: 66.
phenotype: Wings divergent, slight thickening of veins, notching effect in 10-15% of flies.
other information: Not an allele of *vs*.

***wgv: wing variance**

location: 1-33.0 (no recombinants with *v* among 905 flies).
discoverer: Fahmy.
references: 1959, DIS 33: 94.
phenotype: Wing position variable; wings drooping, outspread, or upheld. Male sterile. RK2.

wh: whiskers

location: Autosomal.
origin: Neutron induced.
discoverer: Mickey, 54a7.
references: 1963, DIS 38: 29.
phenotype: Many extra vibrissae, which are longer than normal. RK3.

whd: withered

location: 2-61.
references: Strømten, 1974, Hereditas 78: 157-68.
 Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.
phenotype: Wings warped and waved or reduced to shrunken black pupal pads. Synthetic lethal in presence of *su(r)* (Strømten, 1974). RK2.
alleles:

allele	origin	discoverer	ref ^α
<i>whd</i> ¹	spont	Bridges, 38a6	2,3
<i>whd</i> ⁷⁴	EMS	Ashburner	1

^α 1 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95; 2 = CP627; 3 = Stromen, 1974, Hereditas 78: 157-68.

***whg: whiting**

location: Autosomal.
discoverer: Bridges, 13k21.
references: 1916, Genetics 1: 148.
 1919, J. Exp. Zool. 28: 337-84 (fig.).
phenotype: Specific modifier of *w^e*. *w^e*; *whg* has pure white eyes. RK3.

***whh: white head**

location: 3- (not located).
discoverer: Morgan, 13h.
references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 99.
phenotype: Ocelli surrounded by silvery patch. RK3.

whirligig: see *wrl*

whirly: see *wl*

whiskers: see *wh*

white: see *w*

white head: see *whh*

white-marbled: see *w*^{63b}

white ocelli: see *wo*

whiting: see *whg*

who: see *fu*³³

wi: witty eye

location: 2-55.0 (not allelic to *rh*).
origin: Spontaneous.
discoverer: Whitten, 61g.
references: 1963, DIS 38: 31.
 1968, Heredity 23: 263-78.
phenotype: Eyes rough on lower half owing to irregular facets. Extra vibrissae in variable number and distribution. Removal of closely linked modifiers gives rise to dominant form. Penetrance and expression variable and highly sensitive to background genotype and temperature (Whitten, 1968). RK3.

wider wing: see *ww*

wimp: see *RpIII40*

wind: see *wbl*

windbeutel: see *wbl*

wing blister: see *wb*

wing variance: see *wgv*

wingless: see *wg*

wings-apart: see *wap*

wings-apart-like: see *wapl*

wings-apart mimic: see *wapm*

wings down: see *wdn*

wings-out: see *wgo*

***with: with trident**

location: 3- (near *p*).
discoverer: Morgan, 10a.
references: Morgan and Bridges, 1919, J. Gen. Physiol. 1: 639-43.
 Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 31 (fig.).
phenotype: Dark trident pattern on mesonotum. Variable; some overlap of wild type. RK3.

withered: see *whd*

witty eye: see *wi*

wizened: see *wz*

wk: weak

location: 3-42.
origin: Spontaneous.
discoverer: Bridges, 33122.
phenotype: Bristles small, somewhat Minute, and variable. Abdomen disproportionately small. Wings somewhat warped. Viability variable. RK3.

***wl: whirly**

location: 2- (not located).
origin: Spontaneous.
discoverer: Kiil, 43k4.
references: 1946, DIS 20: 66.

phenotype: Acrostichal hairs in irregular rows; incomplete whorls on thorax. RK3.

wo: white ocelli

location: 3-76.2.

discoverer: Bridges, 12f21.

references: 1920, Biol. Bull. 38: 231-36.

Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 66.

Rayle, 1969, DIS 44: 98.

Jones, 1971, DIS 47: 90.

phenotype: Ocelli colorless. Eye color wild type.

Modifies w^e to a lighter and less yellow tone. Interacts with z , producing white-eyed $z/z;wo/wo$ females and $z/Y;wo/wo$ males with a slight deviation from wild-type eye color (Rayle, 1969). RK2.

alleles: wo^1 (Bridges) and wo^{67k} (Rayle).

cytology: Located in 94A-E by deficiency mapping (Jones, 1971).

other information: cd and wo map in same genetic interval and heterozygote cd/wo also shows white eyes; perhaps alleles (Jones, 1971).

wo : see cd^{wo}

wobA: wobbly A (J.C. Hall)

location: Distal X; no crossing over between y and cv (see cytology).

origin: Induced by ethyl methanesulfonate.

references: Grigliatti, Hall, Rosenbluth, and Suzuki, 1973, Mol. Gen. Genet. 120: 107-15.

Schmidt-Nielsen and Hall, 1977, DIS 52: 71-72.

phenotype: Flies cannot coordinate proper sequence of leg movements for normal walking so that legs on one side of the body or the other become entangled with those behind or in front (Grigliatti *et al.*, 1973); legs held out flat when flies are on a horizontal surface. Mutants cannot climb easily or hang upside down; show poor locomotor behavior in counter current experiments involving agitation of flies and their running toward light (Schmidt-Nielsen and Hall, 1977). Males weak and very poorly fertile. Homozygous females inviable. Two genes, $wobA$ and $wobB$, are involved in the extreme phenotype (full uncoordinated walking behavior); both factors were extracted from the original strain. In the absence of $wobB$, walking in $wobA$ flies is only slightly uncoordinated.

cytology: Inseparable from a transposition reported as $Tp(1;2R;3R;1)$ (T. Kaufman cited in Grigliatti *et al.*, 1973).

wobB: wobbly B (J.C. Hall)

location: 1-near 50 (between v and f).

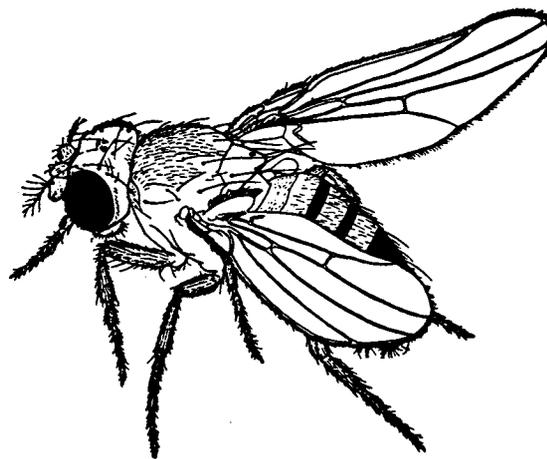
references: Grigliatti, Hall, Rosenbluth, and Suzuki, 1973, Mol. Gen. Genet. 120: 107-15.

Schmidt-Nielsen and Hall, 1977, DIS 52: 71-72.

phenotype: See $wobA$ for digenic phenotype. In absence of $wobA$, walking is not uncoordinated, but the $wobB$ flies show weak locomotion and poor climbing.

wobbly A: see $wobA$

wobbly B: see $wobB$



wp: warped

From Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 216.

***wp: warped**

location: 3-47.5.

discoverer: Bridges, 19k15.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 215 (fig.).

phenotype: Wings small and narrow, dusky, divergent, and warped. RK2.

wr : see fw^{wr}

***Wr: Wrinkle**

location: 2-76.

origin: Spontaneous.

discoverer: Goldschmidt, 1933.

synonym: *Wrinkled* (preoccupied).

phenotype: Wings wrinkled and blistered. Homozygote viable and only slightly more extreme than heterozygote. Development retarded. RK1.

Wrinkled: see W

Wrinkled: see Wr

wrl: whirligig (L.L. Green)

location: 3-54.4 (between $cv-c$ and sbd).

synonym: $ms(3)nc4$.

references: Lewis, Kaufman, Denell, and Talerico, 1980, Genetics 95: 367-81.

Fuller, 1986, Gametogenesis and the Early Embryo, (G.J. Gall, ed.), Symp. Soc. Dev. Biol., 44th, pp. 19-31.

Green, Wolf, McDonald, and Fuller, 1990, Genetics 126: 961-73.

phenotype: Male sterile. Males homozygous for wrl have defects in post-meiotic spermatid differentiation, including disordered spermatid components, disrupted flagellar axonemes, and axonemes lacking one or both central pair microtubules. Axonemes commonly show defects in the

structure of the outer doublet or accessory microtubules. Onion stage early spermatids normal, indicating normal meiosis. Homozygous viable and female fertile. *wrl* acts as a dominant enhancer of tubulin mutants. Alleles obtained by reverting the failure of *wrl* to complement β *Tub85D*ⁿ are dominant male sterile, but are suppressed (restored to fertility) when heterozygous with some mutant alleles of α *Tub84B* or β *Tub85D*. A deficiency for 88C2-E3 generated using *Tp(3;1)kar*⁵¹ and *Tp(3;2)ry*⁺ is semi-dominant male sterile, but is restored to fertility when heterozygous with β *Tub85D*ⁿ. Together these results indicate that *wrl* may be responsible for haploinsufficiency for male fertility located in polytene interval 88C-D. The genetic interactions between mutations in *wrl* and tubulin mutants indicates that the *wrl* gene product could play a role in microtubule function.

alleles:

allele	origin	ref ^α	phenotype
<i>wrl</i>	EMS	1, 2	recessive male sterile; dominant enhancer of tubulin mutants
<i>wrl rv1</i> β	EMS	2	dominant male sterile; phenotype suppressed by tubulin mutants
<i>wrl rv2</i> β	EMS	2	dominant male sterile; phenotype suppressed by tubulin mutants
<i>wrl rv3</i> β	EMS	2	dominant male sterile; phenotype suppressed by tubulin mutants

^α 1 = Lewis, Kaufman, Denell, and Talerico, 1980, Genetics 95: 367-81; 2 = Green, Wolf, McDonald, and Fuller, 1990, Genetics 126: 461-73.
^β Isolated as revertant of failure of *wrl*^{nca} to complement β 21ⁿ.

cytology: Placed in 88C7-D6 because dominant loss of function alleles covered by *Dp(3;3)E11* = *Dp(3;3)88A5-12;88D6-10* but not by *Dp(3;3)E8* = *Df(3;3)88D4-6;88E4-F2*. Recessive male sterile phenotype of *wrl* not uncovered by *Df(3R)red31* = *Df(3R)87F12-14;88C1-3*. Recessive mutant axonemal phenotype of *wrl* uncovered by the synthetic deficiency generated using *Tp(3;1)kar*⁵ = *Tp(3;1)20;87C7-D1;88E2-3* and *Tp(3;2)ry*⁺ = *Tp(3;2)2L;87C2-3;88C2-3*.

ws: waisted

location: 1-1.0.
origin: Induced by L-p -N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).
discoverer: Fahmy, 1955.
references: 1958, DIS 32: 77.
phenotype: Anterior part of abdomen constricted, giving appearance of long, narrow waist. Wings held abnormally and surface wavy. Most flies die shortly after eclosion, but occasional male is viable and fertile. RK3.
alleles: One allele induced by CB. 1506.

wl: welt

location: 2-85.3.
discoverer: Bridges, 32119.
phenotype: Eyes small and narrow with horizontal seam or welt. Many bristles, especially postverticals, doubled or even quadrupled in number. Abdomen chunky. Occasional nicks in wing. Expression overlaps wild type at 19° but is excellent at 25° or higher. RK1.
alleles: *wl*¹ (see phenotype) and *wl*^{ts}, a temperature-sensitive allele producing cell death in wing and eye discs under restrictive conditions (Vikulova and Mglinetz, 1985, Tsitol. Genet. 19: 60-65).

cytology: Placed in 55C based on its inclusion in *Df(2R)Pcl-w5* = *Df(2R)55A-8;55C* but no *Df(2R)PclIIB* = *Df(2R)54F6-55A1;55C1-3* (Deng and Rizki, 1988, Genome 30, suppl. 1: 192).

***wtl: weltlike**

location: 3-59.5.
discoverer: Bridges, 33c7.
phenotype: Eyes seamed and small. Aristae reduced. Wings rather broad. Female sterile. Expression better at 19°. RK3.

***wtw: water wings**

location: 1-38.9.
origin: Induced by DL-p -N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).
discoverer: Fahmy, 1954.
references: 1958, DIS 32: 77-78.
phenotype: Wings short and broad, frequently with incomplete crossveins, and often thickened owing to separation of ventral and dorsal surfaces by fluid. Eyes small and slightly rough. Male genitalia twisted; pigmentation of last abdominal segment in female patchy. Penetrance and viability low. Female infertile. RK3.

***wtw^{clf}: water wings-cleft end**

origin: Induced by DL-p -N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3007).
discoverer: Fahmy, 1953.
synonym: *clf*.
references: 1958, DIS 32: 68.
phenotype: Last male abdominal segment grooved in dorsal midline, has abnormal genitalia. Eyes small; wings short, broad, and slightly divergent. Female fertility low; viability good. Classification difficult. RK3.
alleles: One allele induced by CB. 3007.

wupA : see *hdp*

wupB : see *up*

wvn : see *ddd*

ww: wider wing

location: 1-32.9.
origin: Induced by L-p -N,N-di-(2-chloroethyl)amino-phenylalanine (CB. 3025).
discoverer: Fahmy, 1953.
references: 1958, DIS 32: 78.
phenotype: Wings slightly shorter and broader than normal, frequently upheld, and occasionally truncated. Male viability and fertility good; female viability and fertility reduced. RK3.
alleles: One allele induced by CB. 3026.

***wx: waxy**

location: 2-69.7.
origin: Spontaneous.
discoverer: Ives, 41k15.
references: 1942, DIS 16: 49.
phenotype: Wings heavy textured, more opaque, and smaller than normal. Male completely sterile; female fertile. RK2.

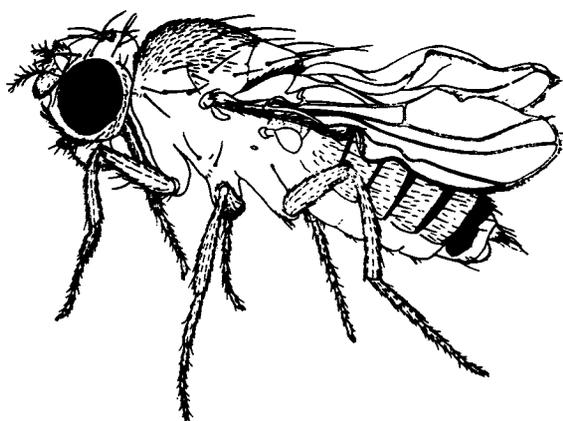
wx^{wxt}: waxy-waxtex

origin: Spontaneous.
discoverer: R. F. Grell, 56k20.
synonym: *wxt*.

references: 1957, DIS 31: 81.

phenotype: Wings slightly spread and curved down distally, texture heavy and waxy, tips pointed. First posterior wing cell narrow, second posterior cell broad and flared. Fertile in both sexes. RK2.

other information: Allelism inferred from similarity in phenotype and genetic location (2-69).



wy: wavy

From Nachtsheim, 1928, Z. Indukt. Abstamm. Vererbungsl. 48: 245-58.

wy: wavy

location: 1-40.7.

phenotype: Wings transversely waved, usually turned up at tip. Abdomen long and narrow. Marginal vein kinked even when other characters overlap wild type. RK2.

alleles:

allele	origin	discoverer	ref ^α	comments
wy¹	spont	Nachtsheim, 26g7	3	wavy wings
wy^{2β}		Ruch	4	more extreme than wy¹ , more upward curl to wings
*wy^{40a}	spont	Haskell, 40a	2	more extreme than wy¹ , more upward curl to wings; wy^{40a} / wy¹ intermediate
wy⁷⁴ⁱ	EMS	Craymer	1	extreme wy allele
*wy²⁷⁴⁻²	X ray	Demerec, 34a		male lethal; associated with <i>T(1;2;3)</i>
wy^{spn}	spont	Waddle	5	male resembles wy¹ ; female has deeply dishd wing distal to wave

^α 1 = Craymer, 1980, DIS 55: 197-200; 2 = Haskell, 1941, DIS 14: 39; 3 = Nachtsheim, 1928, Z. Indukt. Abstamm. Vererbungsl. 48: 245-58; 4 = Parker, 1935, DIS 4: 62; 5 = Waddle, 1977, DIS 52: 3.

^β Synonym: *cx-b*.

cytology: Placed in 11E [Lefevre, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 66-31].

***wz: wizened**

location: 3-47.8.

discoverer: Bridges, 1921.

synonym: *shrunken-3*.

references: Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 241.

phenotype: Small fly; not filled out. Body color dark dull; bristles small. Late hatching. Infertile. RK3.

x1

location: 1-.

origin: Induced by ethyl methanesulfonate.

references: Pak, Grossfield, and White, 1969, *Nature* (London) 222: 351-54.

phenotype: Nonphototactic mutant showing ERG more or less like wild type, although reduced in amplitude and with somewhat different wave form. Other abnormalities include fused wing veins, undeveloped ocelli, and forked bristles.

x2, x3, x4, x5, x6

location: 1-.

origin: Induced by ethyl methanesulfonate.

references: Pak, Grossfield, and White, 1969, *Nature* (London) 222: 351-54.

phenotype: Nonphototactic mutants; external morphology wild type.

x7: see *tan*

x8

location: 1-.

origin: Induced by ethyl methanesulfonate.

references: Pak, Grossfield, and Arnold, *Nature* (London) 227: 518-20.

phenotype: Nonphototactic mutant with normal ERG. Abnormalities include reduced ocelli, fused wing veins, and forked bristles.

x10

location: 1-.

origin: Induced by ethyl methanesulfonate.

references: Pak, Grossfield, and Arnold, *Nature* (London) 227: 518-20.

phenotype: Nonphototactic mutant with normal ERG.

Resembles *sc* in absence of some scutellar, ocellar, and other bristles.

x12: see *norpA*

x13: see *norpA*

x14: see *nonA*

x16: see *norpA*

x24: see *norpA*

x28: see *slrp*

x35: see *rdgA*

x36: see *rdgB*

Xa: see *ap*^{*Xa*}

Xh^{*abo*}: see *ABO*

Xh^{*cr+*}: see *CR*

XI-2: see *dlg1*⁷

xvt: see *pyd*

***xwh*: extra wing hairs**

location: 3-45.

discoverer: Poodrey, 1979.

references: Poodrey, 1980, *The Genetics and Biology of Drosophila* (Ashburner and Wright, eds.). Academic Press, London, New York, San Francisco, Vol. 2d, pp. 443-97.

phenotype: Causes the production of multiple trichomes per cell on the wing and elsewhere on the body; resembles *mwh* although expression of *xwh* on the surface of the wing is less uniform. Arista has many extra branches.

y: yellow

location: 1-0.0.

references: Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. No. 237: 27, 33.

Brehme, 1941, Proc. Nat. Acad. Sci. USA 27: 254-61.

Waddington, 1941, Proc. Zool. Soc. London A III: 173-80.

Sutton, 1943, Genetics 28: 210-17.

Hannah, 1953, J. Exp. Zool. 123: 523-60.

Lefevre, 1970, DIS 45: 32.

Nash and Yarkin, 1974, Genet. Res. 24: 19-26.

Lefevre, 1976, Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 31-66.

Nash, 1976, Dev. Biol. 48: 336-43.

Green, 1980, Ann. Rev. Genet. 14: 109-20.

Lefevre, 1981, Genetics 99: 461-80.

Modolell, Bender, and Meselson, 1983, Proc. Nat. Acad. Sci. USA 80: 1678-82.

Nash, Kamerow, and Merrill, 1983, Biochem. Genet. 21: 1135-41.

Biessmann, 1985, Proc. Nat. Acad. Sci. USA 82: 7369-73.

Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, Cell 40: 327-38.

Mason, 1985, Genetics 110: s33.

Biessmann and Green, 1986, J. Mol. Biol. 191: 573-76.

Chia, Howes, Martin, Meng, Moses, and Tsubota, 1986, EMBO J. 5: 3597-3605.

Geyer, Spana, and Corces, 1986, EMBO J. 5: 2657-62.

Parkhurst and Corces, 1986, Mol. Cell. Biol. 6: 47-53.

Geyer and Corces, 1987, Genes Dev. 1: 996-1004.

Biessmann and Mason, 1988, EMBO J. 7: 1081-86.

Geyer, Green, and Corces, 1988a, Proc. Nat. Acad. Sci. USA 85: 3938-42.

Geyer, Richardson, Corces, and Green, 1988b, Proc. Nat. Acad. Sci. USA 85: 6455-59.

phenotype: The yellow locus controls the melanotic pigment pattern of the cuticle of the adult fly and the pigmented mouth parts and denticle belts of the larval cuticle. *y* mutants can be separated into the following phenotypic classes, each group involving a color change from gray-black to yellow-brown (Nash, 1973, Genetics 74: s191): (1) Mutants that show a total loss of pigmentation from the cuticle (*y*-type) and (2) mutants that show a mosaic pigment pattern, some regions of the cuticle being wild type and others yellow in color (*y*²-type). In the latter type of mutants, at least 40 different adult cuticular structures can express their color independently (Nash and Yarkin, 1974); phenotypes of these mutants indicate that they may play a regulatory role in the expression of yellow (Chia *et al.*, 1986). Some of these type (2) mutants are not temperature-sensitive; others are heat- or cold-sensitive (Nash *et al.*, 1983). For the most part, the yellow gene is autonomous in mosaics, but there is some nonautonomy over limited distances (Hannah, 1953). The function of the gene product in the pigmentation process is still unknown (Biessmann, 1985; Geyer *et*

al., 1986). Hemizygous males are at a mating disadvantage when paired with wild type females (Bastock, 1956, Evolution 10: 421-39), exhibiting a reduced level of locomotion and abnormal courtship (Wilson, Burnet, Eastwood, and Connolly, 1976, Genet. Res. 28: 75-88; Burnet and Wilson, 1980, Genet. Res. 36: 235-47).

alleles: Mutant alleles (but not revertants) are tabulated below. Deficiencies are described in the rearrangement section. Table I includes extant alleles from Russia, Table IIa extant alleles, Table IIb lost alleles.

Table I^α

alleles	origin ^β	comments	cytology
<i>y</i> A66c	γ ray	like <i>y</i> ¹	
<i>y</i> A71k1	γ ray	like <i>y</i> ^{c4}	
<i>y</i> A71l2	γ ray	like <i>y</i> ¹	
<i>y</i> A72a	γ ray	like <i>y</i> ¹	
<i>y</i> A72d1	γ ray	like <i>y</i> ¹	
<i>y</i> A72d3	γ ray	like <i>y</i> ¹	<i>ln(1)1B1-2;20A</i>
<i>y</i> A72d4	γ ray	like <i>y</i> ¹	
<i>y</i> A74b46	C+γ ray	like <i>y</i> ¹	
<i>y</i> A74b51	C+γ ray	like <i>y</i> ^{c4}	<i>ln(1)1B1-2;20D</i>
<i>y</i> A74b121	C+γ ray	like <i>y</i> ¹	
<i>y</i> A74c40	C+γ ray	like <i>y</i> ¹	<i>T(1;4)1B1-2;101F</i>
<i>y</i> A74c166	C+γ ray	like <i>y</i> ^{c4}	<i>ln(1)1B4-9;9D</i>
<i>y</i> A74d1	C+γ ray	like <i>y</i> ¹	
<i>y</i> A74e	γ ray	like <i>y</i> ¹	<i>ln(1)1B1-2;20D</i>
<i>y</i> A74e3	spont	like <i>y</i> ¹	
<i>y</i> A75l2	γ ray	like <i>y</i> ¹	
<i>y</i> A76a110	γ ray	like * <i>y</i> ^{39e}	
<i>y</i> A76a123	γ ray	like <i>y</i> ¹	
<i>y</i> A76b37	γ ray	like <i>y</i> ^{c4}	<i>T(1;2)1B1-2;60F</i>
<i>y</i> A76b94	γ ray	like <i>y</i> ^{td}	<i>ln(1)1B1-2;16C8</i>
<i>y</i> A77d	neutrons	like <i>y</i> ^{c4}	
<i>y</i> A77i	AD+γ ray	like <i>y</i> ¹	
<i>y</i> A78j	γ ray	like <i>y</i> ¹	<i>T(1;3)1B1-2;82A1</i>
<i>y</i> A79b18	γ ray	like <i>y</i> ¹	
<i>y</i> A79b21	γ ray	like <i>y</i> ¹	<i>ln(1)1B1-2;16D</i>
<i>y</i> A79d	neutrons	like <i>y</i> ¹	<i>T(1;2)1A6-B2;23E5</i>
<i>y</i> A79d2	γ ray	like <i>y</i> ¹	<i>ln(1)1B1-2;20A-B</i>
<i>y</i> A79d3	γ ray	like <i>y</i> ¹	
<i>y</i> A79dsd	neutrons+γ ray	females like <i>y</i> ² ; males like <i>y</i> ^{c4} ;	
<i>y</i> A79g	γ ray	like <i>y</i> ¹	
<i>y</i> A79h1	γ ray	like <i>y</i> ¹	
<i>y</i> A81c1	γ ray	like <i>y</i> ¹	
<i>y</i> A81c2	γ ray	like <i>y</i> ¹	
<i>y</i> A81c3	γ ray	like <i>y</i> ¹	
<i>y</i> A81k29	γ ray	like <i>y</i> ^{c4}	
<i>y</i> A82c1	γ ray	like <i>y</i> ¹	
<i>y</i> A82c2	γ ray	like <i>y</i> ¹	
<i>y</i> A82c3	γ ray	like <i>y</i> ¹	
<i>y</i> A83c	γ ray	lethal	
<i>y</i> A83f12	γ ray	like <i>y</i> ¹	
<i>y</i> A83f26	γ ray	like <i>y</i> ¹	
<i>y</i> A83f58	γ ray	like <i>y</i> ^{2s}	
<i>y</i> A83fXL	X ray	like <i>y</i> ¹	<i>ln(1)1B1-2;20E-F</i>
<i>y</i> A84ds	spont	like <i>y</i> ¹	
<i>y</i> A84eS	spont	like <i>y</i> ¹	
<i>y</i> A84e61S	spont	like <i>y</i> ¹	

^α Reference for all alleles: Alexandrov, Ankina, and Alexandrova, 1985, DIS 61: 212-13.

^β Other information: C = caffeine; AD = actinomycin-D.

Table IIa

alleles	origin	discoverer	ref ^α	name and/or comments	cytology
<i>y</i> ¹	spont	E.M. Wallace, 11a	2-5, 11, 17-19, 24, 33, 47-49, 57	adult body yellow, hairs and bristles brown with yellow tips, wing veins, hairs yellow; larval setae, mouth parts yellow to brown	
<i>y</i> ^{1#4}	HD	Green	18	like <i>y</i> ¹	
<i>y</i> ^{1#6}	HD	Green	18	like <i>y</i> ¹	
<i>y</i> ^{1#7}	HD	Green	18	like <i>y</i> ¹	
<i>y</i> ^{1#8}	HD	Green	18	like <i>y</i> ¹	
<i>y</i> ²	spont	Bridges, 25j26	2, 5, 8, 10, 17, 21, 24, 33, 39, 48-49, 51, 57	adult body yellow, hairs and bristles black, wing veins gray; larval mouth parts wild type; <i>y</i> ² / <i>y</i> ^{35a} like <i>y</i> ⁺ / <i>y</i> ⁺ ; reverts to <i>y</i> ¹ with X rays or mutator gene; suppressed by <i>su(Hw)</i> ²	
<i>y</i> ^{2#1}	HD		18	like <i>y</i> ²	
<i>y</i> ^{2#4}	HD		18	like <i>y</i> ²	
<i>y</i> ^{2S}	spont	Bridges	2, 5, 48-49, 51, 57	adult body darker than <i>y</i> ² , bristles lighter; larval mouth parts golden-brown, mouth hooks light	
<i>y</i> ^{3d}	spont	Sturtevant, 1933	2, 5, 48-49	adult body and bristles yellow, wings gray; larval mouth parts yellow, mouth hooks light	
<i>y</i> ^{3M}	spont	Muller	46	like * <i>y</i> ³	
<i>y</i> ^{3Pβ}	X ray	Patterson, 31e25	2, 5, 10, 45, 48a, 49, 52, 59	adult body tan, most bristles black, although patches of yellow bristles and hairs; larval mouth parts light at prongs	<i>In(1)IB1-2;20</i>
<i>y</i> ⁴	X ray	Serebrovsky	2, 10, 15, 48, 51	like <i>y</i> ¹	<i>In(1)IA8-B1;18A3-4</i>
<i>y</i> ¹⁶	EMS		48	adult body yellow; larval mouth parts and microsetae yellow	
<i>y</i> ^{18CH1}		Muller	48-49	like <i>y</i> ¹	
<i>y</i> ²⁵			51	adult body yellow	
<i>y</i> ^{31d}	X ray	Patterson, 31d	5, 49, 53, 59	like <i>y</i> ² ; larval mouth parts light at prongs; <i>y</i> ^{31d} / <i>y</i> ^{35a} like <i>y</i> ^{31d} / <i>y</i> ^{31d}	in <i>In(1)sc</i> ⁸
<i>y</i> ^{31e}				see <i>y</i> ^{3P}	
<i>y</i> ^{34c}	spont	Curry, 34c13	2, 13, 48, 49, 57	adult body and antennae tan; larval mouth parts like <i>y</i> ¹ ; viability excellent	
<i>y</i> ^{50k}	X ray	Green	22	like <i>y</i> ¹	
<i>y</i> ⁵³ⁱ	X ray	Lüning, 53i	32	<i>y</i> / <i>y</i> ⁵³ⁱ Y fertile	
<i>y</i> ^{54j}	spont	Mohler, 55j24	40	adult body and antennae yellow, bristles brownish, wings almost wild type; <i>y</i> ^{54j} / <i>y</i> ¹ intermediate in color, may overlap <i>y</i> ^{54j} homozygotes	
<i>y</i> ^{56k}			48	like <i>y</i> ¹	
<i>y</i> ^{59b}	X ray	Green	8, 21, 22, 48	like <i>y</i> ¹ ; <i>y</i> ^{59b} complements <i>y</i> ² <i>sc</i> ⁺ , but does not complement <i>y</i> ² <i>sc</i>	
<i>y</i> ^{59c}	spont	Clancy, 59c	12, 48, 49	like <i>y</i> ¹	
<i>y</i> ^{60b}	X ray	Green	22	like <i>y</i> ¹	
<i>y</i> ^{61d}	on tritiated medium	Hughes, Hildreth	28	body pale yellow, bristles yellow, wings wild type	
<i>y</i> ^{62a}	spont	Ehrlich, 62a	2, 35, 48, 49	adult body yellow, bristles, hairs brown; tip of male abdomen black; <i>y</i> ^{62a} / <i>y</i> ² like <i>y</i> ² , <i>y</i> ^{62a} / <i>y</i> ¹ like <i>y</i> ¹	
<i>y</i> ^{62d}			2	like <i>y</i> ¹	
<i>y</i> ^{62k19}	spont	Pratt, 62k19	13	like <i>y</i> ¹	
<i>y</i> ^{65f4}	X ray	Lefevre, 65f4	31	body darker than in <i>y</i> ² , bristles dark; fertile	<i>In(1)IB2-3;1B14-C1</i>
<i>y</i> ^{67j}	EMS	Williamson	65	adult body yellow, bristles black in <i>y</i> ^S <i>Yy</i> ^{67j}	
<i>y</i> ^{67k5}	X ray	Lefevre, 67k5	31	yellow; male viable, fertile	<i>T(1;3)IB1-2;98C</i>
<i>y</i> ^{68c}			48	like <i>y</i> ¹	
<i>y</i> ^{68d}				like <i>y</i> ¹	
<i>y</i> ^{68e}	EMS	Hayman, Maddern	23a	like <i>y</i> ¹	
<i>y</i> ^{68e}	EMS	Hayman, Maddern	26a	like <i>y</i> ²	
<i>y</i> ^{68h}	EMS	Hayman, Maddern	26a	like <i>y</i> ¹	
<i>y</i> ^{70a}		Gethmann	48	like <i>y</i> ²	

alleles	origin	discoverer	ref ^α	name and/or comments	cytology
<i>y</i> ^{71f}	spont	Whitney, Lucchesi	64	like <i>y</i> ¹	
<i>y</i> ^{72a}	spont	Thompson	48	like <i>y</i> ²	
<i>y</i> ^{72k}		Green	22a		
<i>y</i> ^{73d}	spont	Periquet, 73d	1a	like <i>y</i> ¹	
<i>y</i> ^{74l}	EMS	Craymer, 74i3	14	like <i>y</i> ¹	
<i>y</i> ⁷⁵		Lefevre	48	like <i>y</i> ²	
<i>y</i> ^{76d28}	HD	Geyer	18	adult cuticle tan, many revertants	
<i>y</i> ^{80e14(6)}	HD	Green	2, 3	like <i>y</i> ¹	
<i>y</i> ^{80h16}	HD	Green	2, 3	like <i>y</i> ¹	
<i>y</i> ⁸²	spont	Blount	3a	like <i>y</i> ¹	
<i>y</i> ^{A3-2}	MR	Green	24	adult body and bristles yellow; <i>y</i> ^{A3-2} / <i>y</i> ² like <i>y</i> ²	
<i>y</i> ^{A3-5}	MR	Green	24	adult body and bristles yellow; <i>y</i> ^{A3-5} / <i>y</i> ² like <i>y</i> ⁺	
<i>y</i> ^{A3-20}	MR	Green	24	adult body and bristles yellow; <i>y</i> ^{A3-20} / <i>y</i> ² like <i>y</i> ⁺	
<i>y</i> ^{bab}	spont	Hanks, Newlin	8, 25, 26, 48-49	yellow-brown abdomen; abdomen brown where black in wild type	
<i>y</i> ^{BC16}	HD	Adler	2	like <i>y</i> ¹	
<i>y</i> ^{bg}		Muller	2, 48-49	like <i>y</i> ²	
<i>y</i> ^{bl}	spont	Sandler	2, 8, 48, 49, 56	yellow bristle; body color wild type, bristles yellow; changes to <i>y</i> ⁺ and <i>y</i> ¹ ; <i>y</i> ^{bl} / <i>y</i> ¹ like <i>y</i> ^{bl} ; <i>y</i> ^{bl} / <i>y</i> ² like <i>y</i> ⁺	<i>Dp(1;1)1B2-3;4F8-9;5D4-5</i>
<i>y</i> ^c	spont		24	yellow-complementing; body and bristles yellow; <i>y</i> ^c / <i>y</i> ² almost wild type	
<i>y</i> ^{c4^γ}	spont	Muller	16, 21, 44, 51	like <i>y</i> ¹ but bristles darker; <i>y</i> ^{c4} / <i>y</i> ^{Sc1} like <i>y</i> ^{c4} ; <i>y</i> ^{c4} / <i>y</i> ² wild type; <i>y</i> ^{c4} / <i>y</i> ² <i>sc</i> like <i>y</i> ²	in <i>In(1)Sc^{SI}+S</i>
<i>y</i> ^{CAA1}	HD	Adler	2	like <i>y</i> ¹	
<i>y</i> ^{CAF2}	HD	Adler	2	like <i>y</i> ¹	
<i>y</i> ^{CAG1}	HD	Adler	2	like <i>y</i> ¹	
<i>y</i> ^{CAM3}	HD	Adler	2	like <i>y</i> ¹	
<i>y</i> ^{CAP1}	HD	Adler	2	like <i>y</i> ¹	
<i>y</i> ^{cl-X}	spont	Muller	7	yellow-cubitus	<i>In(1)1A;20</i>
<i>y</i> ^{cts}	EMS	Craymer, 74f	2, 14	yellow-complementing-ts; <i>y</i> ⁺ at 18°; like <i>y</i> ¹ but lighter at 25°; <i>y</i> ^{cts} / <i>y</i> ² same at all temperatures	
<i>y</i> ^{CU2}	HD	Adler	2	like <i>y</i> ¹	
<i>y</i> ^{d28}	MR	Green	23, 24	like <i>y</i> ² ; a few reversions	
<i>y</i> ^{EMS-112}	EMS	Nash	48a	like <i>y</i> ¹ ; no yellow protein in wing	
<i>y</i> ^{EMS1-35c}	EMS		48	like <i>y</i> ¹	
<i>y</i> ^{f22}	MR	Green	23	like <i>y</i> ¹ ; a few reversions	
<i>y</i> ^{hd1} to <i>y</i> ^{hd14}	HD	Chia	11	yellow-hybrid-dysgenesis; like <i>y</i> ¹	
<i>y</i> ^{j22}	MR	Green	24	adult body and bristles yellow; <i>y</i> ^{j22} / <i>y</i> ² like <i>y</i> ² ; <i>y</i> ⁺ reversions	
<i>y</i> ^M	spont	Nash	48-49	like <i>y</i> ² except for variegation in sex combs and some bristles	
<i>y</i> ^{M1}	spont	Nash	48-49	like <i>y</i> ² except for variegation in some cell types	
<i>y</i> ^{M2}	spont	Nash	48-49	like <i>y</i> ¹ ; no yellow protein in wing	
<i>y</i> ^{M2v}	spont	Nash	2, 48	like <i>y</i> ²	
<i>y</i> ^{Neb}	X ray	Graham	57	like <i>y</i> ¹ except eyes duller	
<i>y</i> ^{ntg}	NNG	Kaufman	29, 48	like <i>y</i> ² ; <i>y</i> / <i>y</i> ^{ntg} are <i>y</i> ²	
<i>y</i> ^{ox}			2, 48	like <i>y</i> ²	
<i>y</i> ^{P59}	spont	Perkovic, 59h	36	yellow of Perkovic; adult body, wings yellow, bristles dark	
<i>y</i> ^{Pπ}	HD	Engels		like <i>y</i> ¹	
<i>y</i> ^{px bl}				see * <i>y</i> ^G	
<i>y</i> ^{r2}	spont		48	some cuticle structures show <i>y</i> variegation at 16°; wild type above 26°	
<i>y</i> ^{R1}	X ray	Roberts	55	yellow of Roberts; male viable, fertile	<i>T(1;2;3)1B4-8;12F;21D2;41;97F</i>
<i>y</i> ^{R2}	X ray	Roberts	55	male viable, fertile	
<i>y</i> ^{R3}	X ray	Roberts	55	male viable, fertile	<i>T(1;2)1B2-3;1E2-3;35F</i>
<i>y</i> ^{R4}	X ray	Roberts	55	female sterile	
<i>y</i> ^{R5}	X ray	Roberts	55	male viable, fertile	
<i>y</i> ^{R6}	X ray	Roberts	55	male viable, fertile	

THE GENOME OF *DROSOPHILA MELANOGASTER*

alleles	origin	discoverer	ref ^α	name and/or comments	cytology
<i>y^{R7}</i>	X ray	Roberts	55	male viable, sterile	<i>T(1;2)1A6-B1;49F</i>
<i>y^{R8}</i>	X ray	Roberts	55	male lethal	<i>T(1;2;3)1A6-B1;47A;67D</i>
<i>y^{R9}</i>	X ray	Roberts	55	female sterile	
<i>y^{R10}</i>	X ray	Roberts	55	male viable, fertile	<i>In(1)1A5-B1;20</i>
<i>y^{R11}</i>	X ray	Roberts	55	male viable, fertile	
<i>y^{R12}</i>	X ray	Roberts	55	male viable, fertile	
<i>y^{R17}</i>	X ray	Roberts	55	male viable, fertile	
<i>y^{R19}</i>	X ray	Roberts	55	male viable, fertile	<i>T(1;3)1A5-B1;87A</i>
<i>y^{R20}</i>	X ray	Roberts	55	male lethal	<i>In(1)1A6-8;1E2-3</i>
<i>y^{R21}</i>	X ray	Roberts	55	male viable, fertile	
<i>y^{R22}</i>	X ray	Roberts	55	male viable, fertile	
<i>y^{R23}</i>	X ray	Roberts	55	female sterile	
<i>y^{R24}</i>	X ray	Roberts	55	male lethal	
<i>y^{R25}</i>	X ray	Roberts	55	female sterile	
<i>y^{R26}</i>	X ray	Roberts	55	male viable, sterile	<i>T(1;3)17C;70C (?)</i>
<i>y^{R28}</i>	X ray	Roberts	55	male lethal	
<i>y^{R29}</i>	X ray	Roberts	55	male viable, sterile	<i>T(1;2;4)1E4-F1;20;26A;35BC;101</i>
<i>y^{R30}</i>	X ray	Roberts	55	male viable, fertile	
<i>y^s</i>				see <i>y^{c4}</i>	
<i>y^{S1}</i>	spont	Singh, 1940	48, 58	<i>yellow of Singh</i> ; like <i>y¹</i>	<i>In(1)1B2-3;20F</i>
<i>y^{Si}</i>				see <i>y^{c4}</i>	
<i>y^{td}</i>	spont	Spencer, 361	5, 6, 48, 49, 57	<i>yellow-tanoid</i> ; adult body rich tan, antennae light yellow, bristles black; larval mouth parts golden brown	
<i>y^{ts1,2,3}</i>	EMS	Nash	48	<i>yellow-ts</i> ; temperature-sensitive (body cuticle)	
<i>y^{ts4,5,6}</i>	EMS	Nash	48, 48a	like <i>y²</i> , but temperature sensitive (for sex combs, wing bristles, and, except for <i>y^{ts5}</i> , bristles of head, thorax, abdomen); no yellow protein in wing	
<i>y^{v2}</i>	spont	Schultz, 35k1	2, 5, 48, 57	<i>yellow-variegated</i> ; adult body color mostly wild type, head bristles mostly black, thoracic bristles often yellow; larval bristles dark with lighter basal prongs	
<i>y^{wm4}</i>			48	<i>yellow-white-mottled</i> ; like <i>y²</i>	

Table IIB

alleles	origin	discoverer	ref ^α	name and/or comments	cytology
<i>*y^{1S}</i>	X ray	Schultz, 34k15	5	like <i>y¹</i>	
<i>*y³</i>	spont	Morgan, 26a	5, 48	adult body tannish; bristles dark brown to black, hairs yellow to black; larval mouth parts golden at basal prongs; mouth hooks, mentum wild type	
<i>*y⁵</i>		Patterson	13	male lethal	<i>In(1)1A-B;14D</i>
<i>*y⁶</i>	X ray		5	body yellow, bristles brown with yellow tips; larval mouth parts like <i>y¹</i>	
<i>*y^{31b}</i>	X ray	Patterson, 31b	5, 48	like <i>y¹</i> ; some <i>sc</i> variegation	in <i>In(1)sc⁸</i>
<i>*y^{31c}</i>	X ray	Patterson, 31c	5	bristles black with some <i>y</i> variegation; larval mouth parts as in <i>y³</i>	in <i>In(1)sc⁸</i>
<i>*y^{35a}</i>	X ray	Stone, 35a	59	like <i>y¹</i> ; larval mouth parts golden; <i>y^{35a/ly¹}</i> like <i>y¹</i> ; <i>y^{35a/ly²}</i> wild type; <i>y^{35a/ly^{31d}}</i> like <i>y^{31d}</i> or <i>y²</i>	
<i>*y^{39e}</i>	spont	Mather, 39e15	34	body yellow, bristles brown	
<i>*y^{40a}</i>	spont	Buzzati-Traverso	9	like <i>y²</i>	
<i>*y^{50e}</i>	spont	Thoday	62		
<i>*y^{51g}</i>	spont	Redfield, 51g	22, 54	body yellow, bristles like <i>y²</i>	
<i>*y^{53e}</i>	X ray	Lüning, 53e12	32	homozygous lethal	
<i>*y^{62b}</i>	radio waves	Mickey, 62b21	38		
<i>*y^{62k}</i>	spont	Mickey, 62k8	38		
<i>*y⁹⁴⁻¹</i>	spont	Moree, 46f6	41, 42	like <i>y¹</i>	
<i>*y²⁶⁰⁻¹¹</i>	X ray	Sutton, 39a	60	like <i>y¹</i> ; male viable but sterile	<i>T(1;3)1B2-3;85F1-5</i>
<i>*y²⁶⁰⁻¹²</i>	X ray	Sutton, 1939	60	like <i>y¹</i>	
<i>*y²⁶⁰⁻¹³</i>	X ray	Sutton, 1939	60	body wild type, bristles yellow; reduced fertility in males	<i>T(1;2)1A4-5;36D</i>
<i>*y²⁶⁰⁻²¹</i>	X ray	Sutton, 1939	60	<i>y^{260-21/ly¹}</i> like <i>y¹</i> ; male lethal	

alleles	origin	discoverer	ref ^α	name and/or comments	cytology
^γ <i>y</i> ²⁶⁰⁻²⁴	X ray	Sutton, 1939	60	like <i>y</i> ¹	
^γ <i>y</i> ²⁶⁰⁻²⁸	X ray	Sutton, 19126	60	like <i>y</i> ¹ ; reduced male viability	
^γ <i>y</i> ²⁶⁰⁻³⁰	X ray	Bishop, 1940	60	like <i>y</i> ¹	
^γ <i>y</i> ²⁶⁰⁻³¹	X ray	Fano, 1941	60	<i>y</i> ²⁶⁰⁻³¹ / <i>y</i> ¹ like <i>y</i> ¹ ; homo- and hemizygous lethal	
^γ <i>y</i> ^{G δ}	spont	Goldschmidt	20	yellow of Goldschmidt; like <i>y</i> ²	<i>In(1)IA;1C3-4</i>
^γ <i>y</i> ^{H51}	spont	Tanaka, 37e30	61	yellow from Hakozaki; body, wings, legs yellow; bristles, hairs black	
^γ <i>y</i> ^N	X ray	Neuhaus	50	yellow of Neuhaus; body color wild type; <i>y</i> ^N / <i>y</i> ¹ like <i>y</i> ^N ; <i>y</i> ^N / <i>y</i> ² like wild type	
^γ <i>y</i> ^O	spont	Kiil, 43k18	30	yellow-orange; body yellow, bristles, hairs black	
^γ <i>y</i> ^S	spont	Cattell, 12d	43	yellow-spot; large yellow spots on dorsal midline near tip of abdomen, on scutellum, and in narrow stripe along thorax	
^γ <i>y</i> ^{S61}	spont	Shuman, 61f	37	yellow of Schuman; like <i>y</i> ¹	
^γ <i>y</i> ^{v1}	X ray	Schultz, 33a11	6	yellow-variegated	
^γ <i>y</i> ^{v56}	X ray	Hinton, Smith	27	variegated for <i>y</i> ; suppressed by extra <i>Y</i> chromosomes	

^α 1 = Anxolabehere and Periquet, 1973, DIS 50: 21; 2 = Biessmann, 1985, Proc. Nat. Acad. Sci. USA 82: 7369-73; 3 = Biessmann and Green, 1986, J. Mol. Biol. 191: 573-76; 3a = Blount, 1982, DIS 58: 154; 4 = Brehme, 1937, Proc. Soc. Exp. Biol. Med. 37: 578-80; 5 = Brehme, 1941, Proc. Nat. Acad. Sci. USA 27: 254-61; 6 = Bridges, 1937, DIS 7: 16; 7 = Brosseau, 1969, DIS 44: 45; 8 = Burnet and Wilson, 1980, Genet. Res. 36: 235-47; 9 = Buzzati-Traverso, 1940, DIS 13: 49; 10 = Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, Cell 40: 327-38; 11 = Chia, Howes, Martin, Meng, Moses, and Tsubota, 1986, EMBO J. 5: 3597-3605; 12 = Clancy, 1960, DIS 34: 48; 13 = CP627; 14 = Craymer, 1980, DIS 55: 197-200; 15 = Dubinin and Friesen, 1932, Biol. Zentralbl. 52: 147-62; 16 = Frye, 1960, DIS 34: 49; 17 = Geyer, Green, and Corces, 1988, Proc. Nat. Acad. Sci. USA 85: 3938-42; 18 = Geyer, Richardson, Corces, and Green, 1988, Proc. Nat. Acad. Sci. USA 85: 6455-59; 19 = Geyer, Spana, and Corces, 1986, EMBO J. 5: 2657-62; 20 = Goldschmidt, 1945, Univ. Calif. Publ. Zool. 49: 307, 398-401; 21 = Green, 1961, Genetics 46: 671-82, 1385-88; 22 = Green, 1962, Genetics 47: 483-88; 22a = Green, 1975, Mutat. Res. 29: 77-84; 23 = Green, 1977, Proc. Nat. Acad. Sci. USA 74: 3490-93; 24 = Green, 1979, Mutat. Res. 59: 291-93; 25 = Hanks and Newlin, 1968, DIS 43: 61; 26 = Hanks and Newlin, 1969, DIS 44: 47; 26a = Hayman and Maddern, 1968, DIS 44: 50; 27 = Hinton and Schmidt, 1956, DIS 30: 121; 28 = Hughes and Hildreth, 1967, DIS 42: 86-87; 29 = Kaufman, 1970, DIS 45: 34; 30 = Kiil, 1946, DIS 20: 66; 31 = Lefevre, 1970, DIS 45: 32; 32 = Lüning, 1953, DIS 27: 58; 33 = Mason, 1985, Genetics 110: s33; 34 = Mather, 1941, DIS 14: 39; 35 = McCloskey, 1963, DIS 37: 50; 36 = Meyer, 1959, DIS 33: 97; 37 = Meyer, 1963, DIS 37: 51; 38 = Mickey, 1963, DIS 38: 29; 39 = Modolell, Bender, and Meselson, 1983, Proc. Nat. Acad. Sci. USA 80: 1678-82; 40 = Mohler, 1956, DIS 30: 79; 41 = Moree, 1946, DIS 20: 66; 42 = Moree, 1947, DIS 21: 69; 43 = Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. No. 237: 27, 33; 44 = Muller, 1946, DIS 20: 68; 45 = Muller and Prokofyeva, 1935, Proc. Nat. Acad. Sci. USA 21: 16-26; 46 = Muller and Valencia, 1947, DIS 21: 70; 47 = Nash, 1975, Genetics 80: s60-61; 48 = Nash, 1976, Dev. Biol. 48: 336-43; 48a = Nash, Kamerow, and Merrill, 1983, Biochem. Genet. 21: 1135-41; 49 = Nash and Yarkin, 1974, Genet. Res. 24: 19-26; 50 = Neuhaus, 1936, DIS 5: 26; 51 = Parkhurst and Corces, 1986, Mol. Cell. Biol. 6: 47-53; 52 = Patterson, 1934, DIS 1: 31; 53 = Patterson, 1935, DIS 4: 12; 54 = Redfield, 1952, DIS 26: 68; 55 = Roberts, 1974, Mutat. Res. 22: 139-44; 56 = Sandler, Hart, and Nicoletti, 1960, DIS 34: 103-4; 57 = Sanger, 1969, DIS 44: 45; 58 = Singh, 1940, DIS 13: 75; 59 = Stone, 1935, DIS 4: 62-63; 60 = Sutton, 1943, Genetics 28: 210-17; 61 = Tanaka, 1937, DIS 8: 11; 62 = Thoday, 1954, DIS 28: 78; 63 = Thompson and Purcell, 1972, DIS 48: 16; 64 = Whitney and Luchesi, 1972, DIS 49: 35; 65 = Williamson, 1968, DIS 43: 65.

^β Synonym: *y*^{31e}.

^γ Synonym: *y*^S (Muller, 1946; preoccupied); *y*^{Si} (Green, 1961; error).

^δ Synonym: *y*^{px-bl}: yellow-plexus blistered.

cytology: Located at the tip of the X chromosome at 1B1 on the cytological map (Lefevre, 1976, 1981).

molecular biology: The yellow locus has been cloned by chromosome walking (Biessmann, 1985) and a *y* transcript of 1.9-2.1 kb identified (Biessmann, 1985; Campuzano *et al.*, 1985; Chia *et al.*, 1986; Parkhurst and Corces, 1986). The transcript consists of two exons processed in both larval and pupal stages into an mRNA of 1990 bp. The nucleotide sequence of yellow genomic DNA has been determined (Geyer *et al.*, 1986; Geyer and Corces, 1987), and the amino acid sequence of the yellow protein predicted from it. Breakpoints of chromosomal aberrations of type 1 and type 2 mutants have been located on the molecular map. Most of the type 1 mutants show structural lesions in the yellow coding region; a majority of the type 2 lesions, however, have been mapped 5' to the transcription start point (Biessmann, 1985; Biessmann and Green, 1986; Chia *et al.*, 1986; Biessmann and Mason, 1988). The allele *y*² is associated with the insertion of the transposable element *gypsy* 700 bp distal to the yellow coding region (Biessmann, 1985; Parkhurst and Corces, 1986); revertants of *y*² are obtained by excision of *gypsy*, leaving

behind a long terminal repeat (LTR) (Geyer *et al.*, 1986) or by replacing the central part of *gypsy* with another transposable element, *wallaby*, and leaving behind all of the 3' LTR and half of the 5' LTR (Geyer *et al.*, 1988a). Another allele, *y*^{76d28}, results from the insertion of a *P* element into the 5' untranslated part of the yellow gene (Geyer *et al.*, 1988b). A high frequency of reversion occurs among the progeny of *y*^{76d28} males carrying the element. Each revertant carries residual *P*-element DNA in the 5' untranslated region. Sequence analysis of several phenotypically wild-type revertants shows imprecise excision of *P*-element sequences within the inverted repeats (20 bp remaining in *y*⁺¹, 4 in *y*⁺¹³, 7 in *y*^{1#7+}, and 340 in *y*⁺¹³⁻¹¹) (Geyer *et al.*, 1988b). Sequencing of two *y* mutants arising from the revertant *y*⁺¹³⁻¹¹ indicates that these mutants are produced by insertion of another *P*-element at the exact site of the original one without duplication of *P* DNA or *y* DNA. *P*-element-mediated transformations of mutants with plasmids carrying *y* DNA with various deletions in the 5' region show which sequences are responsible for regulation of *y* transcription in wings, body, and bristles of adults and mouth parts and denticle belts of larvae (Geyer and Corces, 1987). The

same upstream controlling regions have also been identified using terminal chromosome deficiencies (Biessmann and Mason, 1988).

The yellow polypeptide (60,752 daltons) was localized by immuno-histochemical techniques in the epidermis and adult cuticle of three- and four-day old pupae. Its expression precedes visible melanin deposition by 26 hours. Spatial distribution anticipates the later cuticular melanization pattern. Expression in the embryo was detected in the head region, at the ventral setal belts, and in cells scattered near the ventral nerve cord (Walter, Black, Afshar, Kermabon, Wright, and Biessmann, 1991, Dev. Biol.).

Y^{bb}: see *bb^Y*

Y2: see *fs(1)Ya*

yea: yeast

origin: Induced by ethyl methanesulfonate.

references: Falk and Nash, 1974, Genetics 76: 755-66.

phenotype: A group of genes (all sex-linked) whose mutants require the addition of dead yeast to the standard sucrose *Drosophila* medium in order to show either normal viability or normal rate of development under certain temperature conditions. Some mutants are sensitive to cold or to heat even on yeast-supplemented media. These yeast-auxotroph genes are described in the following table:

locus	genetic location	number of alleles	temperature effects ^α	
			with yeast	without yeast
<i>yea1</i> ^β	1-37	1	lethal at 18°; viable at 25° and 29°	lethal or almost lethal at temperatures tested
<i>yea2</i>	1-16	1	viable at 29°	viable at 20°; lethal at 29°
<i>yea3</i>	1-0.8	1	viable at 18° and 25°; lethal at 29°	lethal at 25°
<i>yea4-1</i> ^γ	1-(3-5)	2	develop normally at 29°	develop slowly at 29°
<i>yea5</i> ^{-2 γ}	1-66	1	viable at 18° and 25°; lethal at 29°	lethal at 25°
<i>yea6</i>	1-45	1	viable at 29°	viable at 20°; lethal at 29°
<i>yea7</i>	1-53	1	develop normally at 29°	develop slowly at 29°; (delay of four to five days)

^α Temperatures used in tests given in table.

^β Females sterile.

^γ Noncomplementing mutant alleles; *yea4-1* shows a developmental delay of about three to five days when grown without yeast at the high temperature.

yellow: see *y*

yem: yema

location: 3- {99}.

origin: Differential screen for genes active during oogenesis whose transcripts disappear at gastrulation.

references: Ait-Ahmed, Thomas, Cavallin, and Rosset, 1987, Dev. Biol. 122: 153-62.

phenotype: Cluster of maternal effect genes active in oogenesis that have been isolated through their messenger RNAs and located by molecular methods at the distal end of 3R.

cytology: Located in 98F3-10 by *in situ* hybridization.

molecular biology: Genomic clone obtained that codes for four transcripts of 4.5, 4, 2.8, and 2.6 kb that are synthesized during oogenesis, are abundant throughout the preblastoderm embryo, and disappear during gastrulation.

All of the transcripts show germ line specific expression in the female. The 4 kb transcript seems to be synthesized in the nurse cells and is transferred to the oocyte; it reappears at postembryonic stages (Ait-Ahmed *et al.*, 1987).

yl: yolkless

location: 1-48.

origin: Induced by ethyl methanesulfonate.

references: Waring, DiOrio and Hennen, 1983, Dev. Biol. 100: 452-63.

Mohler and Carroll, 1984, DIS 60: 236-41.

Perrimon, Mohler, Engstrom, and Mahowald, 1986, Genetics 113: 695-712.

phenotype: Female-sterile locus affecting late oogenesis.

Eggs of mutants lack proteinaceous yolk and collapse; ultrastructurally abnormal chorion at anterior end and especially at base of respiratory filaments. No abnormality detectable in chorion protein synthesis. Uptake of the three yolk proteins by *yl* oocytes is severely reduced, although the proteins accumulate in the hemolymph.

alleles:

allele	synonym	ref ^α
<i>yl 1</i>	<i>fs(1)29</i>	4
<i>yl 2</i>	<i>fs(1)117</i>	4
<i>yl 3</i>	<i>fs(1)445</i>	4
<i>yl 4</i>	<i>fs(1)A148</i>	1
<i>yl 5</i>	<i>fs(1)A305</i>	1
<i>yl 6</i>	<i>fs(1)A332</i>	1
<i>yl 7</i>	<i>fs(1)A1061</i>	1
<i>yl 8</i>	<i>fs(1)A1081</i>	1
<i>yl 9</i>	<i>fs(1)A1130</i>	1
<i>yl 10</i>	<i>fs(1)A1186</i>	1
<i>yl 11</i>	<i>fs(1)K184</i>	2
<i>yl 12</i>	<i>fs(1)K294</i>	2
<i>yl 13</i>	<i>fs(1)K621</i>	2
<i>yl 14</i>	<i>fs(1)11-73</i>	3
<i>yl 15</i>	<i>fs(1)11-380</i>	3
<i>yl 16</i>	<i>fs(1)11-432</i>	3
<i>yl 17</i>	<i>fs(1)12-1259</i>	3
<i>yl 18</i>	<i>fs(1)12-2252</i>	3
<i>yl 19</i>	<i>fs(1)12-5004</i>	3
<i>yl 20</i>	<i>fs(1)12-5262</i>	3
<i>yl 21</i>	<i>fs(1)14-465</i>	3
<i>yl 22</i>	<i>fs(1)5 (2)</i>	3
<i>yl 23</i>	<i>fs(1)205 (2)</i>	3
<i>yl 24</i>	<i>fs(1)L186 (3)</i>	3
<i>yl 25</i>	<i>fs(1)L193 (3)</i>	3
<i>yl 26</i>	<i>fs(1)L196 (3)</i>	3
<i>yl 27</i>	<i>fs(1)L211 (3)</i>	3

^α 1 = Gans, Audit, and Masson, 1975, Genetics 81: 683-704; 2 = Komitopoulou, Gans, Margaritis, Kafatos, and Masson, 1983, Genetics 105: 897-920; 3 = Mohler and Carroll, 1984, DIS 60: 236-41; 4 = Waring, DiOrio, and Hennen, 1983, Dev. Biol. 100: 452-63.

cytology: Located in 12E1-12F1, the region of overlap between *Df(1)g-1 = Df(1)11F10;12F1* and *Df(1)KA9 = Df(1)12E1;13F5* (Waring *et al.*, 1983).

Ylt: see *Pin^{Yt}*

yok: yolky

origin: Induced by ethyl methanesulfonate.

references: Eberl and Hilliker, 1988, Genetics 118: 109-20.

phenotype: Mutants are embryonic lethals with an undispersed yolk plug and no visible Malpighian tubules. Cuticle phenotype normal.

alleles:

allele	synonym
yok 1	<i>l(1)EH160</i>
yok 2	<i>l(1)EH272</i>
yok 3	<i>l(1)EH328</i>
yok 4	<i>l(1)EH352</i>

cytology: Located in 2D1-3A. Included in y^+Y .

Yolk protein: see *Yp*

yolkless: see *yl*

yolky: see *yok*

Yp: Yolk protein

Three yolk proteins, YP1, YP2, and YP3, are synthesized in the fat body and ovarian follicle cells of wild-type adult female flies (Gelti-Douka *et al.*, 1974; Kambysellis, 1977; Bownes and Hames, 1978; Bownes, 1979; Brennan *et al.*, 1980, 1982; Postlethwait and Shirk, 1981). These proteins are the major tyrosine-sulfonated proteins in female flies (Bauerle and Huttner, 1985). After their synthesis, the yolk proteins are secreted into the hemolymph and transported to the egg, where they are converted into the mature form and packaged in the yolk granules (Warren *et al.*, 1979; Brennan *et al.*, 1982). Fat body cells produce approximately equal amounts of YP1, YP2, and YP3 mRNA and protein, but follicle cells synthesize considerably less YP3 transcript and gene product (Barnett *et al.*, 1980; Brennan *et al.*, 1982; Williams and Bownes, 1986). Ovaries transplanted from females into male hosts produce some YP mRNA and protein, but female fat cells transplanted into male hosts do not (Kambysellis, 1977; Postlethwait *et al.*, 1980). The three YP proteins are of similar molecular weight [approximately 44,700-47,000 daltons (Bownes and Hames, 1977; Hames and Bownes, 1978; Warren and Mahowald, 1979; Bownes, 1982)], but have different isoelectric points (Warren and Mahowald, 1979). Each protein is encoded by a single copy, X-linked gene, *Yp1*, *Yp2*, or *Yp3* (Barnett *et al.*, 1980). Regulation occurs by means of 20-hydroxyecdysone (involved in the accumulation of *Yp* transcripts in the fat body of females) and juvenile hormone (involved in the development of ovarian follicles) (Postlethwait and Handler, 1979; Jowett and Postlethwait, 1980; Bownes, 1982a, 1982b, 1986). Male and female larvae and adult males do not normally produce YPs. Some yolk protein, however, is produced in males injected with 20-hydroxyecdysone; juvenile hormone does not induce YP synthesis in male flies (Postlethwait *et al.*, 1980; Bownes and Nothiger, 1981; Bownes, 1982; Bownes *et al.*, 1983a). The products of the sex-determining genes, *ix*, *tra*, *tra2*, and *dsx* may influence the expression of the yolk protein genes (Bownes *et al.*, 1983b; Belote, Handler, Wolfner, Livak, and Baker, 1985, Cell 40: 339-48; Kraus *et al.*, 1988). *X/X* flies carrying *tra/tra*, *tra2/tra2*, or *dsx^D/dsx* do not produce detectable amounts of yolk protein, but intersexes carrying *ix/ix*, *dsx/dsx*, or *dsx^D/+* do synthesize YPs (although in reduced amounts) (Bownes and Nothiger, 1981; Bownes *et al.*, 1983b).

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Yp1**location:** 1-30.**discoverer:** Postlethwait, 80c.**phenotype:** Structural gene for the yolk protein YP1 found in recently-emerged female flies. Protein migrates at different rates in SDS-polyacrylamide gels when encoded by the electrophoretic variants $Yp1^F$ (fast) and $Yp1^S$ (slow), alleles that are female fertile and produce normal amounts of YP1. $Yp1^{ts1}$, which maps near the *Yp1* locus and is believed to be an allele, produces a slow-migrating translation product that is present in reduced amounts in the hemolymph and the ovaries (Bownes and Hodson, 1980); this mutant is female sterile.**alleles:** $Yp1^F$ (= $Yp1^{LC}$), $Yp1^S$ (in Canton-S and Oregon-R stocks), and $Yp1^{ts1}$. The $Yp1^{ts1}$ allele is a female sterile mutant [synonym = *fs(1)1163* (Bownes and Hodson, 1980)]. It is homozygous female sterile at 18° and homo- and heterozygous female sterile at 29°, producing flaccid eggs and reduced amounts of yolk protein at the high temperature (Bownes and Hodson, 1980). $Yp1^{ts1}$ is female sterile over *Df(1)C52*, a deficiency for the *Yp1* locus (Postlethwait and Shirk, 1981).**cytology:** *Yp1* has been located in 8F-9B by *in situ* hybridization of cloned DNA to the salivaries (Barnett *et al.*, 1980; Riddell *et al.*, 1981); cytological location between 9A and 9B since found between the X breakpoints of $T(1;Y)B52 = T(1;Y)9A;YL$ and $T(1;Y)J2a = T(1;Y)9B;YS$.**molecular biology:** The gene for YP1 has been cloned from Canton-S wild type and its nucleotide sequence obtained (Barnett *et al.*, 1980; Hovemann *et al.*, 1981; Hovemann and Galler, 1982; Hung *et al.*, 1982; Hung and Wensink, 1981, 1983; Riddell *et al.*, 1981). *Yp1* is 1635 bp long and present as a single copy, with a short exon followed by a very short intron (76 bp) and a long exon (Hung and Wensink, 1981, 1983). It is transcribed in a proximal to distal direction. The length of the transcript is about 1559 nucleotides plus poly(A) tail; the protein predicted from the nucleotide sequence is made up of 439 amino acids. The mutant $Yp1^{ts1}$ has been cloned and sequenced (Saunders and Bownes, 1986); the mutant protein was found to differ from the wild-type protein by an amino acid substitution (isoleucine to asparagine) at position 92.Transformation experiments indicate that two enhancer sequences upstream from *Yp1* determine its tissue specificity (fat body or follicle cells) (Garabedian *et al.*, 1985, 1986; Shepherd *et al.*, 1985; Tamura, Kunert, and Postlethwait, 1985, Proc. Nat. Acad. Sci. USA 82: 7000-04).A *Yp1-Adh* fusion gene has been used to transform an *Adh*⁻ chromosome; adult females (but not adult males or male and female larvae) expressed enzymatically active ADH (Aprison *et al.*, 1989). *tra2*^{ts2} homozygotes with two X chromosomes show expression of the fusion gene only when they are raised at 16°, the temperature at which they are morphologically female and produce yolk proteins. Endogenous yolk protein genes show the same pattern of temperature expression as the fusion gene in females homozygous for *tra2*^{ts} (Belote, Handler, Wolfner, Livak, and Baker, 1985, Cell 40: 339-48).**Yp2****location:** 1-29.5.**discoverer:** Postlethwait, 79a.**phenotype:** Structural gene for the yolk protein YP2 found in recently-emerged female flies. Protein migrates at different rates in SDS-polyacrylamide gels when encoded by the electrophoretic variants $Yp2^F$ (fast) and $Yp2^S$ (slow), alleles that are female fertile and produce normal amounts of YP2. A mutant $Yp2^M$ (= $Yp2^{12-1245}$) is female fertile but lays fewer eggs than normal (Mohler, Postlethwait, and Shirk) and does not contain yolk protein in the hemolymph or ovaries.**alleles:** $Yp2^F$ (in Canton-S and Oregon-R stocks), $Yp2^S$ [= $Yp2^{Po}$ (Postlethwait and Jowett, 1980)], and $Yp2^M$, [induced by Mohler and believed to be a mutant allele (Mohler, 1977, Genetics 85: 259-72; Mohler and Carroll, 1984, DIS 60: 236-41; Tamura *et al.*, 1985)].**cytology:** *Yp2* has been located in 8F-9B by *in situ* hybridization (Barnett *et al.*, 1980; Riddell *et al.*, 1981); cytological location between 9A and 9B since found between the X breakpoints of $T(1;Y)B52 = T(1;Y)9A;YL$ and $T(1;Y)J2a = T(1;Y)9B;YS$.**molecular biology:** The gene for YP2 has been cloned from Canton-S wild type and its nucleotide sequence obtained (Barnett *et al.*, 1980; Hovemann *et al.*, 1981; Hovemann and Galler, 1982; Hung *et al.*, 1982; Riddell *et al.*, 1981). *Yp2* is 1614 bp long (1225 bp distant from *Yp1*) and is present as a single copy, with a short exon followed by a very short intron (68 bp) and a long exon (Hung and Wensink, 1983). It is transcribed in a distal to proximal direction (opposite from the transcription direction of *Yp1*). There are two transcripts of 1614 and 1698 nucleotides in length. The protein predicted from the nucleotide sequence is made up of 442 amino acids. *Yp1* and *Yp2* show about 53% sequence identity (Yan *et al.*, 1987). The site of tyrosine sulfonation has been identified in this secretory protein (Baeuerle *et al.*, 1988); it is similar in amino acid composition and secondary structure to known tyrosine sulfonation sites in vertebrates.**Yp3****location:** 1-44.**discoverer:** Postlethwait, 79g.**phenotype:** Structural gene for the yolk protein YP3 found in recently-emerged female flies. Protein migrates at different rates in SDS-polyacrylamide gels when encoded by the electrophoretic variants $Yp3^F$ (fast) and $Yp3^S$ (slow), alleles that are female fertile and produce normal amounts of YP3. $Yp3^{RI}$, which maps close to the *Yp3* locus, has no detectable YP3 protein in the hemolymph or ovary, but is not female sterile (Postlethwait and Jowett, 1980; Postlethwait and Shirk, 1981).**alleles:** $Yp3^F$ (= $Yp3^{CB}$), $Yp3^S$ (in Canton-S and Oregon-R stocks), and $Yp3^{RI}$, which behaves like a *cis*-acting regulatory variant and was found by Laurie-Ahlberg in wild stock RI14 (Postlethwait and Shirk, 1981).**cytology:** *Yp3* has been located in 12B-C by *in situ* hybridization (Barnett *et al.*, 1980; Riddell *et al.*, 1981); cytological location between 12A6-7 and 12D3, the region deleted from *Df(1)HA92 = Df(1)12A6-7;12D3*.**molecular biology:** The gene for YP3 has been cloned from Canton-S wild type and its nucleotide sequence obtained (Barnett *et al.*, 1980; Hovemann *et al.*, 1981;

Hovemann and Galler, 1982; Hung *et al.*, 1982; Hung and Wensink, 1981; Riddell *et al.*, 1981; Garabedian *et al.*, 1987; Yan *et al.*, 1987). *Yp3* is 1624 bp long and present as a single copy. It has two small introns and is transcribed in a distal to proximal direction (Yan *et al.*, 1987). The transcript is about 1499 nucleotides plus poly(A) tail in length; the protein predicted from the nucleotide sequence is made up of 420 amino acids. The mutant *Yp3^{R1}* produces no translatable message in females, but carries normal amounts of transcripts that hybridize to the *Yp3* gene probe (Postlethwait and Shirk, 1981). The overall amino acid sequence identity of the three yolk protein genes is 43% (Garabedian *et al.*, 1987). *Yp1* and *Yp3* show about 53% identity in their amino acid sequences; *Yp2* and *Yp3* show about 48% identity. These identical sequences occur mainly in translated regions (Yan *et al.*, 1987).

Two small regions identical to the *Yp1* fat body enhancer region have been found in *Yp3* flanking sequences (Garabedian *et al.*, 1987).

yrt: yurt

location: 3-52.

origin: Induced by ethyl methanesulfonate.

references: Jurgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

phenotype: Embryonic lethal mutation. Embryos show a dorsal posterior hole.

alleles: Three alleles identified, **yrt¹*, *yrt²*, and *yrt³* (weak allele), isolated as *5G*, *9G*, and *10H*.

cytology: Located in 87D14-F12; uncovered by *Df(3R)l26c = Df(3R)87D14-E1;87F11-12* but not by *Df(3R)kar-Sz8 = Df(3R)87C1-2;87D14-E1*.

z: zeste

location: 1-1.0 (to the right of *pn* and *kz*).

references: Gans, 1948, DIS 22: 69-70.

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phenotype: The regulatory gene *zeste* interacts with the white locus as well as with the bithorax and decapentaplegic complexes, changing the phenotypic expression of these loci. z^1 was the first mutant allele identified (Gans, 1948, 1953); the homo- or hemizygotes of this neomorphic mutant show a lemon yellow eye color when carrying two paired copies of w^+ or of the rightmost w^+ alleles [as in $z^1 w^+/z^1 w^+$ females or z^1/Y males with a w^+ duplication (Jack and Judd, 1979)]. $z^1 w^+/Y$ males without the duplication, z^1/z^1 females heterozygous for a w allele belonging to one of the right-hand (*zeste*-suppressing) subloci, or z^+/z^1 females are wild type. An

intralocus duplication for a right sublocus of white produces mottling in z^1 males. z^1 eye color develops autonomously in mosaics and in eye-disk transplants. It is not affected by the number of Y chromosomes in the genotype. A third chromosome mutant *wo* interacts with z^1 or z^{58g} to lighten eye color, producing $z/z;wo/wo$ white-eyed females and $z/Y;wo/wo$ males with a slight deviation from wild-type eye color (Rayle, 1969, DIS 44: 98; Kaufman *et al.*, 1973). z^1 has no effect on the expression of the white gene in ocelli, testes, or larval Malpighian tubules.

The first z^a mutant was also identified by Gans. These mutants are wild type in z^a/Y males and z^a/z^a , $z^a/Df(1)z$, and z^+/z^a females. The heteroallelic combination of z^1/z^a , however, results in yellow-eyed flies. Complementation between w^{sp} and other white alleles does not occur in z^a mutants, although it does occur in z^+ or z^1 flies (Babu and Bhat, 1980). z^a -type alleles, including $z^{ae(bx)}$, as well as the partial revertant of z^1 , z^{11G3} , enhance the mutant phenotype of certain heteroallelic combinations of *BXC* alleles that show transvection (partial complementation) when paired; z^+ and z^1 , however, do not affect these *BXC* alleles (Kaufman *et al.*, 1973; Gelbart and Wu, 1982; Mariani *et al.*, 1985; Pirrotta *et al.*, 1987). All *zeste* mutant alleles tested enhance certain heteroallelic mutant combinations that show transvection in *dpp* (Gelbart and Wu, 1982).

The z^{op} mutants (Lifschytz and Green, 1984), unlike z^1 , require only one copy of w^+ for expression of a *zeste* eye color in homo- and hemizygotes. Heterozygotes over z^+ are *zeste* if they have two copies of w^+ , but are wild type if there is only one copy.

Another mutant, z^{v77h} , requires only one copy of w^+ in males. The eyes are brown variegated in hemi- and homozygous z^{v77h} females and z^{v77h}/Y males, but wild type in homozygous $z^{v77h} Dp(1;1)w^{+2}$ females and $z^{v77h} Dp(1;1)w^{+2}/Y$ males, this allele responding to an increase in w^+ dosage in a manner contrary to that of z^1 (Green, 1984).

Diepoxy-butane-induced mutations (including multilocus deletions) have been generated in an attempt to obtain a null allele of *zeste* (Goldberg *et al.*, 1989). Some of the females that were completely deficient for z [*Df(1)z-deb3/Df(1)z-deb3*, for example] survived and were fertile, indicating that the product of the *zeste* gene is not required for viability or female fertility.

alleles: *Zeste* mutants and rearrangements are described in the following table. Deficiencies for *zeste* are listed as rearrangements. See end of text for more detailed descriptions of certain alleles.

allele	origin	discoverer	ref ^α	comments
z¹	spont	Gans, 46b	5-9, 10-13, 16, 17	eye color wild type in males; lemon yellow at 25°, mottled yellow and brownish red at 19° in homozygous females
z¹⁻³⁵		Jack	11	no detectable DNA rearrangements
z¹⁻⁴²		Jack	11	no detectable DNA rearrangements
z^{11G3}	X ray (from z¹)	Gans	9, 11, 13, 15, 17	eye color wild type in z males and homozygous and heteroallelic z females
z³¹	HD	Simmons	11	
z³²	HD	Simmons	11	
z^{58g}	spont	Gloor	11, 12	eye color wild type in males, lemon yellow in homozygous females; z^{58g}/z^{p69a} zeste mottled; z^{58g}/z^{11G3} wild type; z^{58g}/z^a zeste; no detectable DNA rearrangement
z^{59d}		Green, 59d15	11, 12	z⁺/z^{59d} females have orange mottled eyes; <i>Dp(1;1)2F5-3A1;3A4-5</i>
z^{78c}		Jack	11	no detectable DNA rearrangements
z^{81E}		Green	16	recombinant between z¹ and z^{v77h}
z^{+64b9}	X ray	Green	1, 2, 18	z^{+64b9}/Y males and z^{+64b9}/z^{+64b9} females wild type; <i>In(1)3C1-2;12B</i>
z^{+64b13}	X ray	Green	1, 2	z^{+64b13}/Y males and z^{+64b13}/z^{+64b13} females wild type
z^a	X ray	Gans	3-5, 9, 11, 12	z^a/Y males, z⁺/z^a and z^a/z^a females wild type; enhances certain genotypes in <i>BXC</i> and <i>dpp</i> that show transvection
z^{a68k}	EMS	Gelbart	9, 13	z^{a68k}/Y males and z^{a68k}/z^{a68k} females wild type; enhances certain genotypes in <i>BXC</i> and <i>dpp</i> that show transvection
z^{a691}	EMS	Gelbart	9, 13	z^{a691}/Y males and z^{a691}/z^{a691} females wild type; enhances certain genotypes in <i>BXC</i> and <i>dpp</i> that show transvection
z^{a692}	EMS	Gelbart	9, 11, 13	z^{a692}/Y males and z^{a692}/z^{a692} females wild type; enhances certain genotypes in <i>BXC</i> and <i>dpp</i> that show transvection; no detectable DNA rearrangements
z^{a693}	EMS	Gelbart	9, 11, 13	z^{a693}/Y males and z^{a693}/z^{a693} females wild type; enhances certain genotypes in <i>BXC</i> and <i>dpp</i> that show transvection
z^{a694}	EMS	Gelbart	9, 13	z^{a694}/Y males and z^{a694}/z^{a694} females wild type; enhances certain genotypes in <i>BXC</i> and <i>dpp</i> that show transvection; no detectable DNA rearrangements
z^{ae(bx)β}	γ ray	Lewis	3, 9, 11, 13, 14, 17	males and females wild type in eye color; enhances most <i>bx</i> alleles; <i>In(1)3A3;4F</i>
z^{ae(bx)2}	X ray	Lewis	14	like z^{ae(bx)} ; salivary chromosomes appear normal
z^{op6}	induced in z¹ by EMS	Lifschytz	15, 17	two w⁺ genes not essential for zeste mutant expression in z^{op6}/z^{op6} and z^{op6}/Df(1)z females or z^{op6}/w⁺/Y males
z^{op11}	same as z^{op6}	Lifschytz	15	intermediate between z¹ and z^{op6} (same eye color in z^{op11}/w⁺/Y and z^{op6}/w⁺/Y males, but z⁺/z^{op11} females with two copies of w⁺ have brown eyes); no detectable DNA rearrangements
z^{p69a}		Gelbart	13	hemizygous males wild type; homozygous females brown mottled on yellow ("peppered"); with z¹ , eyes uniform zeste; with z^a , eyes zeste mottled like z^a (with some variegation); <i>P</i> element insertion at coordinate 0 on molecular map in females; revertants obtained
z^{π.1}	HD	Pirrotta	16, 17	like z^a except eyes more orange; <i>P</i> element insertion at coordinate 0 on molecular map in females
z^{π.2}	HD	Pirrotta	16	like z^a except eyes more orange; <i>P</i> element insertion at coordinate 0 on molecular map in females
z^{RN4}	X ray	Green	15	partial revertant of z^{op6}
z^{v77h}	HD	Green	10, 11, 17	brown variegated eye color darkening toward posterior in z^{v77h}/w⁺/Y males and z^{v77h}/z^{v77h}/w⁺ females; lighter at 28°

^α 1 = Arcos-Terán, 1972, *Chromosoma* 37: 233-96; 2 = Arcos-Terán and Beermann, 1968, *Chromosoma* 25: 377-91; 3 = Babu and Bhat, 1980, *Development and Neurobiology of Drosophila* (Siddiqi, Babu, Hall, and Hall eds.). 1980, Plenum Press, New York and London, pp. 35-40; 4 = Babu and Bhat, 1980, *Mol. Gen. Genet.* 183: 400-02; 5 = Bingham, 1980, *Genetics* 95: 341-53; 6 = Gans, 1948, *DIS* 22: 69-70; 7 = Gans, 1953, *Bull. Biol. France Belg., Suppl.* 38: 1-90; 8 = Gans-David, 1949, *Bull. Biol. France Belg.* 83: 136-57; 9 = Gelbart and Wu, 1982, *Genetics* 102: 179-89; 10 = Green, 1984, *Mol. Gen. Genet.* 194: 275-78; 11 = Gunaratne, Mansukhani, Lipari, Liou, Martindale, and Goldberg, 1986, *Proc. Nat. Acad. Sci. USA* 83: 701-05; 12 = Jack and Judd, 1979, *Proc. Nat. Acad. Sci. USA* 76: 1368-72; 13 = Kaufman, Tasaka, and Suzuki, 1973, *Genetics* 75: 299-321; 14 = Lewis, 1959, *DIS* 33: 96; 15 = Lifschytz and Green, 1984, *EMBO J.* 3: 999-1002; 16 = Mariani, Pirrotta, and Manet, 1985, *EMBO J.* 4: 2045-52; 17 = Pirrotta, Manet, Hardon, Bickel, and Benson, 1987, *EMBO J.* 6: 791-99; 18 = Sorsa, Green, and Beermann, 1973, *Nature (London) New Biology* 245: 34-37.

^β Synonym: *e(bx)*.

cytology: Located in salivary chromosome band 3A3 on the basis of its inclusion in $Df(1)w^{258-11} = Df(1)3A3-4;3C2-3$ and $Df(1)64C = Df(1)3A3-4;3C2-3$ (Gans, 1953, Judd, Shen, and Kaufman, 1972, *Genetics* 71: 139-56), but not in $Df(1)K95 = Df(1)3A3-6;3B1$. Also located in 3A3 by *in situ* hybridization to $In(1)z^{ae(bx)}$ (Mariani *et al.*, 1985).

molecular biology: The wild type allele of *zeste* and some of the *zeste* mutants have been cloned and the cDNA sequenced (Mariani *et al.*, 1985; Gunaratne *et al.*, 1986; Pirrotta *et al.*, 1987). Cloning has been accomplished by microdissection of the salivary 3A1-4 region followed by microcloning and chromosome walking; also by "transposon tagging" of the locus with *P* elements, recovering the sequences flanking the 3A3-4 *P* insertion. Probes from the 3' or 5' end of the gene were used to isolate cDNA clones; the length of the longest clone was just over 2300 nucleotides (Pirrotta *et al.*, 1987). No detectable DNA rearrangements were found in many of the mutants (Gunaratne *et al.*, 1986). A transcript of 2.2-2.4 kb was found at all postzygotic stages of development in wild type and most mutant individuals; shorter transcripts were found in $z^{ae(bx)}$, $z^{\pi 1}$, and z^{v77h} . Transcription is most abundant in maternal RNA; it declines during larval growth, but increases again in third instar larvae and pupae (Pirrotta *et al.*, 1988). The gene is transcribed along the *X* from distal to proximal. The z^+ transcript includes three exons and two introns; no differences in exon size or position could be detected in the RNA of the mutants z^1 , z^{op6} , or z^a . *P* factor mediated germ-line transformation to wild-type eye color was carried out in a $z^1 Dp(1;1)w^+$ strain (Gunaratne *et al.*, 1986); in other transformation experiments, yellow-eyed transformants were produced by injecting z^{op6} DNA in a *P* element vector into a $y z^a$ strain (Pirrotta *et al.*, 1987).

The *zeste* protein predicted from the nucleotide sequence of wild type *zeste* cDNA has an amino terminal region with both basic and acidic residues, a second region that is acidic, a third region with few basic or acidic residues but many glutamines and alanines, and a carboxy terminal region with many basic and acidic residues (Pirrotta *et al.*, 1987). The protein binds directly to specific DNA sequences of the white, Ultrabithorax, decapentaplegic, Antennapedia, and engrailed regulatory regions (Benson and Pirrotta, 1987, 1988; Biggin *et al.*, 1988; Mansukhani *et al.*, 1988). Anti-*zeste* antibodies interact with approximately 60 specific bands in polytene chromosomes; this number is drastically increased by heat shock (Pirrotta *et al.*, 1988).

other information: Inversions, translocations, and transpositions with breaks in 3C3, induced as derivatives of z^1 chromosomes carrying tandem duplications of w^+ , result in a range of *zeste* phenotypes in males and females, the eye colors being *zeste*, *zeste* variegated, *zeste* halo, and wild type (Green, 1967, 1984). *E(z)* and *Su(z)* loci have been described (Green, 1967; Kalisch and Rasmuson, 1974, *Hereditas* 78: 97-104; Persson, 1976, *Hereditas* 82: 111-20).

z^1

phenotype: Two synapsed copies of w^+ required for expression of *zeste* eye color (Gans, 1953). Ocelli wild type in color, as are testes and larval Malpighian tubules. Supports transvection at *Ubx* but not at *dpp*.

molecular biology: Comparison between the sequences of z^+ and z^a shows many polymorphisms concentrated in the middle, repetitive part of the gene (Pirrotta *et al.*, 1987). There are no changes in the 5' flanking region or in the untranslated leader. Most of the changes do not affect the predicted amino acid sequence since they are in the introns or in the third position of the codons; the change from A to T (Lys to Met in the middle repetitive part of the protein) is believed to have an important effect on the properties of the mutant product (Pirrotta *et al.*, 1987).

z^{11G3}

phenotype: Partial revertant of z^1 showing wild-type eye color in hemizygous males and homozygous females (Gans, 1953). Almost complete complementation of z^1 eye color. Does not support transvection at *dpp* or *Ubx*.

molecular biology: The sequence of z^{11G3} is identical to that of z^1 except that three nucleotides have been deleted, removing a tyrosine from the amino acid sequence (Pirrotta *et al.*, 1987).

z^a

phenotype: Hemizygous males and homozygous females said to be wild type in eye color (Gans); however, on closer inspection they are seen to have a diluted eye color that becomes brown with age (Pirrotta *et al.*, 1987). z^1/z^a females have *zeste* eyes; $z^a w^{DZL}/z^a w^{ch}$ females have light brown eyes. Does not support transvection at *dpp* or *Ubx*.

molecular biology: No detectable rearrangements except for a DNA insertion (about 15 kb) of unknown significance (Gunaratne *et al.*, 1986).

$z^{ae(bx)}$

phenotype: Mostly wild type, but slight eye color variegation in homozygous females (Lewis, 1959, *DIS* 33: 96). $z^1/z^{ae(bx)}$ females *zeste*. Does not support transvection at *dpp* or *Ubx*.

molecular biology: Lacks about 800 nucleotides of the *zeste* coding region, but otherwise the 5' half of the mutant gene is normal (Pirrotta *et al.*, 1987). The transcript of 1.4-1.8 kb (Mariani *et al.*, 1985; Gunaratne *et al.*, 1986) ends shortly after the 3A3 breakpoint of the inversion [breakpoint placed on molecular map of *zeste* at about +2.0 kb, the origin being the first *EcoRI* site in clone CS1014 (Gunaratne *et al.*, 1986)].

z^{op6}

phenotype: Eye color *zeste* in hemizygous males and homozygous females with only one copy of w^+ ; homozygous females with two copies of w^+ also *zeste*. Heterozygous z^+/z^{op6} females wild type if one copy of w^+ , but *zeste* if two copies; z^{op6} reverts to weaker alleles after EMS or X rays (Lifschytz and Green, 1984).

molecular biology: Carries all the polymorphisms in the z^1 sequence as well as the change from Lys to Met; also a C to T mutation causing a proline to leucine change in the middle region of the protein, increasing its hydrophobic nature (Pirrotta *et al.*, 1987).

$z^{\pi 1}$

phenotype: Hemizygous males and homo- or hemizygous females almost wild type in eye color. Supports transvection at *Ubx* (Pirrotta *et al.*, 1987).

molecular biology: *P* element insertion at coordinate 0 on

molecular map of Mariani *et al.* (within 10 kb of this origin and in the same orientation as the coordinates of Gunaratne *et al.*) plus a deletion of about 300 nucleotides (Pirrotta *et al.*, 1987); otherwise, coding region like that of z^+ .

z^{v77h}

phenotype: Eye color diluted, turning brownish with age in hemizygous males and hemi- and homozygous females with a normal complement of w^+ genes. $z^{a/2} z^{v77h}$ females are wild type; $z^{1/2} z^{v77h}$ females are zeste; z^{v77h} hemizygous males and homozygous females carrying a w^+ duplication in each X are wild type.

molecular biology: Carries deletion of about 320 nucleotides and addition of seven nucleotides in the first exon; P element was probably inserted, followed by excision of the P and the flanking deletion (Pirrotta), the deletion removing the AUG start codon.

z^l : see w^{zl}

z^m : see w^{zm}

*Z: *Zerknittert*

location: 1-5.5.

references: Grüneberg, 1931, Biol. Zentralbl. 51: 219-25. 1934, DIS 2: 8.

phenotype: Wings may be crumpled or incompletely unfolded, but majority overlap wild type. Viability 10% wild type. RK3.

z2: see under *ANTC*

Z600

location: 3-[42].

references: Schultz and Butler, 1989, Genes Dev. 3: 232-42.

Schultz, Schlomchik, Cherbas, and Cherbas, 1989, Dev. Biol. 131: 515-23.

phenotype: Member of cluster of genes that includes *Eip28/29* and *Gdl*. Believed to play role in processes of early development.

cytology: Located in 71C3-D4 (Schultz and Butler, 1989).

molecular biology: Gene cloned, partially sequenced, and a 600-nucleotide transcript (missing sequences from the 5' end) detected in zygotes (Schultz *et al.*, 1989; Schultz and Butler, 1989). *Z600* is located upstream to the neighboring gene *Gdl^M* and overlaps it. The *Z600* gene is expressed abundantly in very early embryos and again at a lower level in adults; the transcript is expressed at a higher level in oviposited eggs than in ovaries. The gene is 1780 bp long, with three exons of 453, 295, and 506 bp and two introns of 56 and 410 bp.

zen: see under *ANTC*

zen-2: see **z2** under *ANTC*

Zerind: see *Fs(3)Sz25*

Zerknittert: see *Z*

zerknüllt: see **zen** under *ANTC*

zeste: see **z**

zip: zipper

location: 2-107.6.

origin: Induced by ethyl methanesulfonate.

references: Nüsslein-Volhard, Wieschaus and Kluding,

1983, DIS 59: 158-60.

Nüsslein-Volhard, Wieschaus and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

Côté, Preiss, Haller, Schuh, and Jäckle, 1987, EMBO J. 6: 2793-2801.

Zhao, Côté, Jähmig, Haller, and Jäckle, 1988, EMBO J. 7: 1115-19.

phenotype: The wild-type allele of *zipper* is expressed in the nervous system during development and was believed to encode an integral membrane protein necessary for normal axon patterning (Zhao *et al.*, 1988); however it has more recently been shown to encode the heavy chain of cytoplasmic myosin (Kierhart). The mutants are embryonic lethals; abnormalities include a small hole in the ventral thorax, distortion of ventral denticle rows, and defects in head involution and dorsal closure (Nüsslein-Volhard *et al.*, 1984; Côté *et al.*, 1987). These defects vary in different alleles and in different embryos from the same egg laying (Côté *et al.*, 1987). The nervous system of mutant embryos also differentiates abnormally, showing local defects in the fasciculation pattern of axons (Zhao *et al.*, 1988), as indicated by antibody stains for neurons and their axons. These CNS abnormalities can be detected after germ band shortening and, together with the molecular data, suggested that neurological rather than epidermal defects are the primary ones in *zip* mutants (Côté *et al.*, 1987).

alleles: *zip¹* (isolation number ID16), a strong allele showing severe cuticular and neurological defects, *zip²* (isolation number IIF107), a weaker allele, and 16 discarded alleles have been reported.

cytology: Located in 60E9-F1 since uncovered by *Df(2R)gsb = Df(2R)60E9-10,60F1-2*, but not by *Df(2R)SB1 = Df(2R)60E10-F1,60F5*.

molecular biology: The 60E9-60F1 region of 2R was cloned by microdissection and the clones were used to start a chromosome walk (Côté *et al.*, 1987). On the molecular map, the *zip* region lies between the proximal breakpoint of *Df(2R)gsb* at -55 to -49 kb ("- values to the left) and the proximal breakpoint of *Df(2R)SB1* at 0 to +3.5 kb ("+" values to the right). A transcript of 2.4 kb was demonstrated by northern blot analysis. The transcript accumulates in the mesodermal-neuroblast region (not in the ectoderm) at the extended germ band stage, but becomes restricted to neural tissue (brain and ventral nerve cord) from the time of germ band shortening up to hatching, not appearing in the precursor cells of the cuticle (Côté *et al.*, 1987).

zip cDNA has been sequenced and the putative amino acid sequence of the protein determined (Zhao *et al.*, 1988). There is a single open reading frame of 1500 bp which encodes a polypeptide of 500 amino acids with several domains, including a putative signal sequence and a transmembrane domain.

other information: Shown to be the same as *Mhc-c*, and is a synonym thereof (Kierhart).

Zolta: see *Fs(3)Sz32*

Zombor: see *Fs(3)Sz36*

Zw: *Zwischenferment*

location: 1-62.9 (Eanes); located between *car* and *sw*.

discoverer: Young.

references: Young, Porter, and Childs, 1964, Science

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- Eanes and Hey, 1986, *Genetics* 113: 679-93.
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- Fouts, Ganguly, Gutierrez, Lucchesi, and Manning, 1988, *Gene* 63: 261-75.
- Lucchesi and Manning, 1988, *Insect Biochem.* 18: 515-19.
- Merriam, 1988, *DIS* 67: 111-36 (clone list).
- phenotype:** Structural gene for glucose 6-phosphate dehydrogenase (Zwischenferment of Warburg) [G6PD (E.C. 1.1.1.49)], the first enzyme in the oxidative part of the pentose phosphate shunt. Electrophoretic variants Zw^A and Zw^B have been described in *Drosophila melanogaster* (Young *et al.*, 1964; Young, 1966). The G6PD produced by Zw^A shows faster migration in starch gel (Young *et al.*, 1964) or acrylamide gel (Peeples, Barnett, and Oliver, 1967, *J. Hered.* 58: 243-45) than that produced by Zw^B . A Zw^A/Zw^B female shows fast- and slow-migrating bands but no hybrid band of intermediate mobility (Young *et al.*, 1964; Steele *et al.*, 1968; Hori and Tanda, 1980, *Jpn. J. Genet.* 55: 211-23). The B variant in both homo- and heterozygotes is characterized by a double band and shows more heat stability than the A variant (Steele *et al.*, 1968). A dominant sex-linked modifier of the electrophoretic mobility of G6PD, $M(G6PD)$, has been described by Komma (1968, *Biochem. Genet.* 1: 229-37). A regulatory element that affects the activity level of G6PD has been reported by Itoh and Hori (*Jpn. J. Genet.* 60: 441-53). The molecular weight of the A variant of G6PD approximates 147,000 and that of the B variant 317,000 according to Steele *et al.* (1968), who used the electrophoretic starch gel method and observed that the B form can dissociate and produce some A-like form. Lee, Langley, and Burkhart (1978, *Anal. Biochem.* 86: 697-706), using gel-filtration chromatography, reported that the B variant has a molecular weight of 240,000. A subunit molecular weight for the purified enzyme was estimated by Lee *et al.* to be 55,000 and by Hori and Tanda to be 69,000, as if the slow B variant represented a tetramer and the fast A variant a dimer of single polypeptides (Hori and Tanda, 1980; Miyashita *et al.*, 1986). Significant amounts of the enzyme are found in the fat body and the intestine of *Drosophila melanogaster* larvae (Cochrane and Lucchesi, 1980, *Genetics* 94: s20). Enzyme levels are raised by dietary sucrose or D-glycerate (Geer, Kamiak, Kidd, Nishimura, and Yemm, 1976, *J. Exp. Zool.* 195: 15-31; Geer, Woodward, and Marshall, 1978, *J. Exp. Zool.* 203: 391-402; Cavener and Clegg, 1981; Cochrane, Lucchesi, and Laurie-Ahlberg, 1983, *Genetics* 105: 601-13). A maternal form of G6PD can be detected up to the early pupal stage (Gerasimova and Smirnova, 1979). Total G6PD activity increases during the larval period, reaches a peak during the third larval instar, drops during pupation, and increases again in the adult (Bijlsma and Van der Meulen-Bruijns, 1979, *Biochem. Genet.* 17: 1131-44; Williamson and Bentley, 1983). The enzyme shows a characteristic staining pattern in imaginal disks (Cunningham, Smith, Makowski, and Kuhn, 1983, *Mol. Gen. Genet.* 191: 238-43).
- Males with one dose of Zw^+ and females with two doses have about the same amount of G6PD activity, *i.e.* show dosage compensation for enzyme activity (Seecof *et al.*, 1969; Gvozdev *et al.*, 1971; Bowman and Simmons, 1973; Faizullin and Gvozdev, 1973; Williamson and Bentley, 1981). Females heterozygous for a Zw deficiency show a corresponding reduction in enzyme activity; males and females with an extra dose of Zw^+ show increased enzyme activity (Seecof *et al.*, 1969; Maroni and Plaut, 1973; Stewart and Merriam, 1975). Contribution of each dose of G6PD to the level of enzyme activity is the same in triploid females as in diploid females (Lucchesi and Rawls, 1973).
- A number of low- and null-activity mutations have been induced at the Zw locus. The mutant alleles are fully viable (Gvozdev *et al.*, 1976; Hughes and Lucchesi, 1977; Bijlsma, 1980; Lucchesi *et al.*, 1979), but the larvae do not grow as well as wild type on a minimal

amino-acid diet lacking fatty acids and whole nucleic acids (Geer *et al.*, 1974). Null alleles at the *ry* locus are also viable, but double mutant combinations of $Zw^-;ry^-$ do not survive (Lucchesi and Manning, 1988). Although Pgd^- flies are lethal, Zw^-Pgd^- flies carrying null alleles for both G6PD, the first enzyme in the pentose phosphate shunt, and 6PGD, the last enzyme, are viable, presumably because the toxic 6-phosphogluconate is not produced (Hughes and Lucchesi, 1977, 1978; Geer *et al.*, 1979; Lucchesi *et al.*, 1979).

Many natural populations throughout the world are polymorphic for the A and B variants (Oakeshott,

Chambers, Gibson, Eanes, and Willcocks, 1983, *Heredity* 50: 67-72). Rare variants from a number of North American populations have been screened by sequential electrophoresis of starch and acrylamid gels to detect molecular heterogeneity (Eanes, 1983, 1984; Eanes and Hey, 1986); the G6PD activity of these lines and also of induced mutants has been measured (see the following table).

alleles: The following table includes electrophoretic (wild-type), low activity, and null alleles at the *Zw* locus. (Synonyms for the Zw^+ alleles, Zw^A and Zw^B , use terminology for allozyme variants from DIS 53: 117).

THE GENOME OF *DROSOPHILA MELANOGASTER*

allele	origin	discov	synonym	ref ^α	mobility; activity ^β	suppression of <i>Pgd</i> -lethality
Zw^A	spont	Young	<i>G6pd</i> ^δ	10-12	fast	
Zw^{AF1}	nature	Eanes		1	>A	strong
Zw^{AF2}	nature	Eanes		1	>A	strong
Zw^{AF3}	nature	Eanes		1	>A	strong
Zw^{AF4}	nature	Eanes		1	>A	strong
Zw^{AS1}	nature	Eanes		1	<A	moderate
Zw^{AS2}	nature	Eanes		1	<A	moderate
Zw^{AS3}	nature	Eanes		1	<A	weak
Zw^{AS4}	nature	Eanes		1	<A	moderate
Zw^{AS5}	nature	Eanes		1	<A	weak
Zw^B	spont	Young	<i>G6pd</i> ⁴	10-12	slow	
Zw^{BF1}	nature	Eanes		1	>B	moderate
Zw^{BS1}	nature	Eanes		1	<B	weak
Zw^{lo1}	spont	Young		2, 7, 8, 10	10-15%	strong
Zw^{lo1a}	EMS (<i>Zw^B</i>)	Gvozdev	<i>Zw^{su(Pgd)1}</i>	4-6	10-20%	strong
Zw^{lo2} γ	nature	Eanes	<i>Zw^{Blo1}</i>	1	20%	strong
Zw^{lo2a}	EMS (<i>Zw^B</i>)	Gvozdev	<i>Zw^{su(Pgd)2}</i>	4-6	4-20%	strong
Zw^{lo5}	EMS (<i>Zw^B</i>)	Gvozdev	<i>Zw^{su(Pgd)5}</i>	4-6	3-20%	weak
Zw^{lo9}	EMS (<i>Zw^A</i>)	Gvozdev	<i>Zw^{su(Pgd)9}</i>	5	90-130%	strong
Zw^{lo12}	EMS (<i>Zw^A</i>)	Gvozdev		5	90-140%	weak
Zw^{lo15}	EMS (<i>Zw^A</i>)	Gvozdev		5	2-5%	weak
Zw^{lo17}	EMS (<i>Zw^A</i>)	Gvozdev		5	0.5-1%	strong
Zw^{lo19}	EMS (<i>Zw^A</i>)	Gvozdev		5	30-60%	strong
Zw^{lo23}	EMS (<i>Zw^A</i>)	Gvozdev		3, 5	7%	weak
Zw^{lo25}	EMS (<i>Zw^A</i>)	Gvozdev		5	30-70%	strong
Zw^{lo26}	EMS (<i>Zw^A</i>)	Gvozdev		5	40-70%	weak
Zw^{lo27}	EMS (<i>Zw^A</i>)	Gvozdev		5	4-10%	strong
Zw^{lo28}	EMS (<i>Zw^A</i>)	Gvozdev		5	20-40%	weak
Zw^{lo29}	EMS (<i>Zw^A</i>)	Gvozdev		5	70-130%	strong
Zw^{lo30}	EMS (<i>Zw^A</i>)	Gvozdev		5	30-70%	strong
Zw^{lo32}	spont (<i>Zw^A</i>)	Gvozdev		5	1-7%	strong
Zw^{lo33}	spont (<i>Zw^A</i>)	Gvozdev		5	2-6%	strong
Zw^{lo35}	spont (<i>Zw^A</i>)	Gvozdev		5	20-180%	strong
Zw^{lo40}	EMS (<i>Zw^A</i>)	Gvozdev		5	20-130%	strong
Zw^{lo43}	EMS (<i>Zw^B</i>)	Gvozdev		5	7-20%	strong
Zw^{lo44}	EMS (<i>Zw^B</i>)	Gvozdev		5	2-10%	strong
Zw^{lo45}	EMS (<i>Zw^B</i>)	Gvozdev		5	4-10%	strong
Zw^{lo47}	spont (<i>Zw^A</i>)	Gvozdev		5	30-110%	weak
Zwⁿ¹	EMS	Hughes, Lucchesi		7	null	strong
Zwⁿ²	nature	Eanes	<i>Zw^{Nash-}</i>	1	null	strong
Zwⁿ³	HD	Nero	<i>Zw^{H7a}</i>	9	null	strong
Zwⁿ⁶	EMS (<i>Zw^A</i>)	Gvozdev	<i>Zw^{su(Pgd)6}</i>	3, 5, 6	null	strong
Zwⁿ⁷	EMS (<i>Zw^A</i>)	Gvozdev	<i>Zw^{su(Pgd)7}</i>	5	null	strong
Zwⁿ⁸	EMS (<i>Zw^A</i>)	Gvozdev	<i>Zw^{su(Pgd)8}</i>	5	null	strong
Zwⁿ¹⁰	EMS (<i>Zw^A</i>)	Gvozdev		5	null	strong
Zwⁿ¹¹	EMS (<i>Zw^A</i>)	Gvozdev		5	null	strong
Zwⁿ¹³	EMS (<i>Zw^A</i>)	Gvozdev		5	null	strong
Zwⁿ¹⁴	EMS (<i>Zw^A</i>)	Gvozdev		5	null	strong
Zwⁿ¹⁶	EMS (<i>Zw^A</i>)	Gvozdev		5	null	strong
Zwⁿ¹⁸	EMS (<i>Zw^A</i>)	Gvozdev		5	null	strong
Zwⁿ²⁰	EMS (<i>Zw^A</i>)	Gvozdev		5	null	strong
Zwⁿ²¹	EMS (<i>Zw^A</i>)	Gvozdev		5	null	strong
Zwⁿ²²	EMS (<i>Zw^A</i>)	Gvozdev		5	null	strong
Zwⁿ²⁴	EMS (<i>Zw^A</i>)	Gvozdev		5	null	strong

^α 1 = Eanes and Hey, 1986, *Genetics* 113: 679-93; 2 = Geer, Bowman, and Simmons, 1974, *J. Exp. Zool.* 187: 77-78; 3 = Gerasimova and Smirnova, 1979, *Dev. Genet.* 1: 97-107; 4 = Gvozdev, Gerasimova, Kogan, and Rosovsky, 1977, *Mol. Gen. Genet.* 153: 191-98; 5 = Gvozdev, Gerasimova, Kogan, Rosovsky, and Smirnova, 1981, *DIS* 56: 53-55; 6 = Gvozdev, Gerasimova, Rosovsky, Kogan, and Braslavskaya, 1978, *DIS* 53: 143-44; 7 = Hughes and Lucchesi, 1977, *Science* 196: 1114-15; 8 = Lucchesi, Hughes, and Geer, 1979, *Curr. Top. Cell. Regul.* 15: 143-54; 9 = Lucchesi and Manning, 1988, *Insect Biochem.* 18: 515-19; 10 = Williamson and Bentley, 1983, *Biochem. Genet.* 21: 1153-67; 11 = Young, 1966, *J. Hered.* 57: 58-60; 12 = Young, Porter, and Childs, 1964, *Science* 143: 140-41.

^β >A = faster than *Zw^A*; <A = slower than *Zw^A*; >B = faster than *Zw^B*; <B = slower than *Zw^B*; % = percent of *Zw^A* G6PD activity; null = no G6PD activity.

^γ P-element insertion at 18D12-13 (Eanes and Hey, 1986).

cytology: Placed in 18D12-13 by *in situ* hybridization (Eanes and Hey, 1986).

molecular biology: A clone containing the G6PD gene sequence (hybridizing to salivary region 18D) was obtained from a *Drosophila melanogaster* genomic library (Ganguly *et al.*, 1985) and its translation product found to be a polypeptide identical to that of the

monomeric subunit of G6PD (molecular weight 55,000). A transcriptional map of the gene shows three introns, dividing the protein-coding region into four exons (Fouts *et al.*, 1988). The transcript is 2.0 kb long and there is one copy of the gene per X chromosome in both males and females (Ganguly *et al.*, 1985). The complete nucleotide sequence of *Zw⁺* DNA and the predicted

amino acid sequence are given by Fouts *et al.* (1988). The sequence of Oregon-R cDNA differs from Canton-S genomic DNA at three sites which do not change the predicted amino acid sequence. The 5' end of the mature mRNA is at nucleotide -289. The first ATG of the transcription unit is at position 1 and the second ATG at position 592; both start sites would result in a protein with a subunit molecular weight of about 55,000, the value predicted by Lee *et al.* (1978). The sequence upstream from each of these sites is GT-rich.

Drosophila melanogaster G6PD is not similar to the human enzyme for the first 42 amino acid residues, but about 65% identity exists throughout the sequence, especially near the N-terminal region and near the central part of the protein (Fouts *et al.*, 1988).

Germline transformation of a Zw^{nl} strain was carried out using a Zw^+ vector. All transformations produced active G6PD enzyme even when the transduced gene was relocated to an autosome, but in an autosomal site no dosage compensation was observed in males (Fouts *et al.*, 1988).

other information: The activity of G6PD is influenced by factors on the second and third chromosomes (Laurie-Alberg *et al.*, 1980; Tanda and Hori, *Jpn. J. Genet.* 58: 591-606; Miyashita *et al.*, 1986). The suggestion of Giesel (1976, *Biochem. Genet.* 14: 823-33) that Zw regulates structural genes on the autosomes has not been supported by studies of the mobility or activity of G6PD in *Drosophila melanogaster* (Lucchesi *et al.*, 1979).

ADDENDUM

Gad: Glutamic acid decarboxylase (J.C. Hall)**location:** 3-[15].**origin:** From molecular cloning, based on cross-hybridization with feline GAD-encoding gene.**references:** Jackson, Newby, and Kulkarni, 1990, *J. Neurochem.* 54: 1068-78.**phenotype:** The structural gene for glutamic acid decarboxylase. *Gad* probes detect putative GAD-encoding 3.1-kb mRNA, from 4-8 h of embryogenesis to adulthood; a 2.6-kb RNA was also found, in embryos and pupae only; *in situ* hybridization detects the RNA(s) in widely, but not uniformly, distributed CNS cortical regions (of adult heads and larvae), with no detectable signals in non-neural tissues.**cytology:** *Gad* cDNA probe hybridized to 64A3-5; adults heterozygous for *Df(3L)HR277 [= Df(3L)63B12;64B12]* and a normal third chromosome display a 30-45% reduction in GAD levels.**molecular biology:** Sequence of cross-hybridizing cDNA clones predicts a 510-amino-acid protein with overall sequence identity to feline coding material of *ca.* 50% (after alignment of *Drosophila* sequence with residues 81-585 of cat GAD); *in vitro* translation of *Drosophila* cDNA leads to *ca.* 57-kd protein (would correspond to a 510-residue polypeptide); this was immunoprecipitable and Western-blotable with anti-rat-GAD, though the latter experiments also found a 76-kd band.**other information:** *Gad* cDNA probe did not cross hybridize with *Ddc* clone, though the sequences of these two genes indicate a region of sequence similarity in their coding regions (corresponding to pyridoxal-phosphate-binding region).**ggy: groggy (J.C. Hall)****location:** 3-between *cu* and *sr* (based on mapping of physiological defects).**origin:** Induced by ethyl methanesulfonate.**references:** Timpe, Moats, Jan, and Jan, 1987, *Neurosci. Abstr.* 13: 578.**phenotype:** Found as heat-sensitive adult paralytic; in physiological experiments on larvae, recordings from neuromuscular junctions show spontaneous depolarizations at room temperature, which probably result from anomalous spontaneous firing of motor neurons (*e.g.*, kainic acid, which blocks evoked junctional potentials, also blocks the spontaneous ones); patch-clamp experiments reveal no abnormalities of potassium-dependent-A or delayed-rectifying currents.**Gtd: Glucose-tasting-defective (J.C. Hall)****location:** 1-(*v-f*, closer to *v*).**origin:** Induced by ethyl methanesulfonate.**references:** Rodrigues, Sathe, Pinto, Balakrishnan, and Siddiqi, 1991, *Mol. Gen. Genet.* 226: 265-766.**phenotype:** Isolated after feeding-preference enrichment, against positive glucose response, of descendants of mutagenized flies. Subsequent detailed testing revealed enhanced attraction to NaCl in feeding-preference test, NaCl tolerance in ingestion test, relative indifference to sucrose, and higher than normal threshold for quinine avoidance; all of these phenotypes semi-dominant. Gustatory responses of larvae normal. No alteration in

electrophysiologically measured response of peripheral (labellar) neurons to NaCl; causes lethality of males hemizygous for the mutation who also carry duplication for normal allele.

cytology: Maps to 10E1 or 11A7, based on inclusion in *Df(1)KA6 = Df(1)10E1-2;11A7-8* but not in *Df(1)m259-4 = Df(1)10C2-3;10E1-2* or *Df(1)m30 = Df(1)10E2;11A6-7*; in fact, females carrying either of the latter two deficiencies in heterozygous combination with *Gtd* are near-normal in sucrose and quinine responses (*i.e.*, closer to wild type than *Gtd/+*); yet, *Gtd* over either *Df(1)v-L2 = Df(1)9F13;10A1* or *Df(1)N105 = Df(1)10F7;11D1* leads to *Gtd/+*-like responses; *Gtd/y⁺Yv⁺#3 (9F4;10E3-4)* males die as second-instar larvae, whereas the *y⁺Yv⁺* duplication (9F4;10A1) does not effect such synthetic lethality; the X factor responsible for the lethality (in conjunction with the larger duplication) was inseparable from *Gtd* locus.**other information:** Fails to complement nearby *gustD* mutations with respect to (recessive-type) quinine tolerance; when heterozygous with *gustC*, which is closely linked, flies show reduced response to sucrose that is more severe than in tests of either homozygous or hemizygous type; complements *gustB* in behavioral assays (*re* homozygous-type responses); three revertants of *Gtd/y⁺Yv⁺#3* lethal interaction also reverted with respect to sucrose-indifference phenotype.**iav: inactive (J.C. Hall)****location:** 1-18.8 (*iav¹*), 20.7 (*hypoB¹*).**synonym:** *hypoB*.**references:** Kaplan, 1977, *DIS* 52: 1.Homyk and Sheppard, 1977, *Genetics* 87: 95-104.Homyk, 1977, *Genetics* 87: 105-28.O'Dell and Burnet, 1986, *DIS* 63: 107-08.O'Dell, Coulon, David, Papin, Fuzeau-Braesch, and Jallon, 1977, *Comptes Rendus Ser. III* 305: 199-202.O'Dell and Burnet, 1988, *Heredity* 61: 199-207.O'Dell, Burnet, and Jallon, 1989, *Heredity* 62: 373-81.**phenotype:** *iav¹* noted (Kaplan, 1977) to be extremely inactive as adult, with normal external morphology; population of mutants remains quiet and spread out evenly in a container; will walk or fly when container disturbed, but settles into inactivity soon afterward. *iav²* noted (Homyk and Sheppard, 1977) to be inactive and difficult to arouse for flight (and, when it does, seems to hover and fixate on objects); to jump and fly abnormally short distances; to exhibit slow running or climbing ability and optomotor response; to be debilitated after mechanical shock (especially in adults more than two weeks old). Gynandromorph analysis of *iav²* adult behavioral impairments suggests primary focus could be neural (Homyk, 1977). Open-field activity tests of *iav¹* and *iav²* (O'Dell and Burnet, 1988) showed both to exhibit reduced speed as well as amount of locomotion (the former meaning number of squares in the activity chambered visited/unit time, the latter the proportion of time spent moving); male activity levels higher than for females (also found for wild-type); lower than wild-type levels of activity at all ages between time of eclosion and approximately one-month; yet, unlike wild type, speed and amount of activity increase (in tests of *iav²*) in parallel between days 1 and 2 of adulthood (reaching a much lower than normal plateau). In tests of homozygous *iav¹*

and *iav*² females (with normal males), mating propensity reduced and courtship durations extended, though such females showed normal compositions of cuticular hydrocarbons and were highly attractive to courting males (O'Dell et al., 1989); mutant males with normal females exhibited slightly reduced mating success; mutant males crossed with mutant females had very low mating success rate. Lower than normal octopamine levels, especially in extracts from adult heads (O'Dell et al., 1987), with dopamine and norepinephrine levels apparently normal.

alleles: *iav*¹, spontaneous (Kaplan, 1977), *iav*², *iav*³, induced by ethyl methanesulfonate [originally *hypob*¹⁻³ (Homyk and Sheppard, 1977)].

other information: Allelism of *iav*¹ to *hypob*¹ shown by O'Dell and Burnet (1986) in open field tests of heterozygotes; they also note that both mutations are recessive.

***irreC*: irregular chiasm-C (J.C. Hall)**

location: 1-1.7 (from cytology, association with *rst*; *irreC*¹ does map between *y* and *cv*).

references: Boschert, Ramos, Tix, Technau, and Fischbach, 1990, *J. Neurogenet.* 6: 153-71.

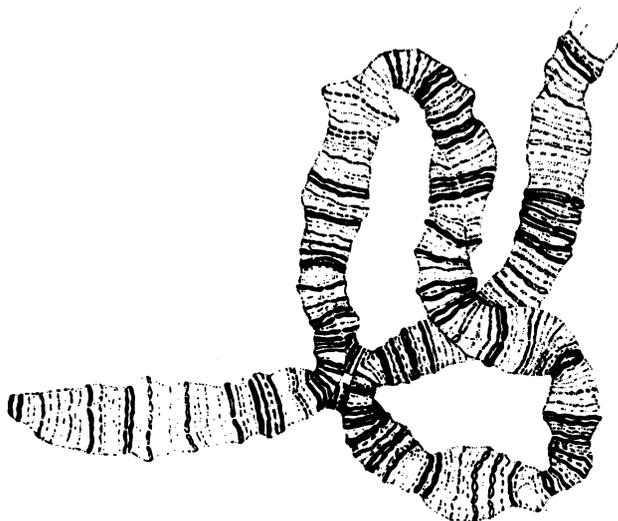
phenotype: In optic ganglia, fiber tracts from the medulla to the lobula plate penetrate lobula neuropile instead of projecting through second optic chiasm; occasionally, ectopic bundles of axonal fibers penetrate lobula-plate neuropile; in most severely deformed individuals, lobula and lobula plate are partly fused. In first optic chiasm, fibers from lamina are misrouted, taking detour around posterior medulla neuropile, penetrating the latter at variable positions on its inner (posterior) face from which positions the fiber tracts turn around and form normal-appearing terminals in retinotopic locations. Gynandromorph analysis showed that eye genotype does not induce optic lobe phenotypes. It also appears as if first- (or second-) chiasm defect does not induce that in the second (or first); among several mutant individuals analyzed (cf. variable expressivity under alleles), there is no correlation between the anatomical abnormalities in these two locations (though there is high correlation between defective first or second chiasm in left and right

sides of head). Optic lobe pioneer axons [larval neurons, which persist into adulthood and can be seen following path of first optic chiasm, and may guide newly growing fibers during formation of imaginal visual system (Tix, Minden, and Technau, 1989, *Development* 105: 739-46)] are displaced in *irreC* pupae, with their axons having followed ectopic pathways.

alleles: *irreC*¹ (*P*-element induced as *irreC*^{UB883}) shows 94% penetrance for optic-lobe defects; good viability and fertility; expressivity highly variable (e.g., anatomical abnormalities can appear in only first but not second chiasm, or vice versa, or in only left or right side of the head). *IrreC*² (X ray induced as *irreC*^{IR34}) shows high penetrance for optic-lobe abnormalities, but variable expressivity; females homozygous for this inversion (see below) do not lay eggs and have rudimentary egg chambers (likely not due to lesion in region 3C, owing to fertility of synthetic deletion described under cytology).

cytology: Located in 3C4-5. *irreC*¹ has a *P* element inserted in 3C (also in 2EF and 4D); *irreC*² is associated with *In(1)2A;3C3-6*, with anatomical defects inseparable from the inversion. The following deletions uncovered *irreC* mutation(s): *Df(1)JC19 = Df(1)2F6;3C5*, *Df(1)N8 = Df(1)3C2-3;3E3-4*, *Df(1)w258-42 = Df(1)3A4-6;3C5-6*, and *Df(1)N71h = Df(1)3C4;3D3-4*; the nearest complementing deletions tested were *Df(1)258-45 = Df(1)3B2-3;3C2-3* and *Df(1)N69h9 = Df(1)3C6;3D1*. Females heterozygous for *Df(1)w258-42/Df(1)N8* are *irreC*-like in their optic lobe anatomy (as well having white and roughest eyes, and seem no worse so than mutant homozygotes or hemizygotes; such females are also fertile).

other information: Associated with the closely linked *rst* locus. Whereas two mutant *rst* alleles (including *rst*⁶) lead to no apparent abnormalities in optic chiasms (cf. Meyerowitz and Kankel, 1978, *Dev. Biol.* 62: 112-42), and *irreC*¹ causes no eye roughening, this mutation over *Df(1)rst vt* leads to rough eyes; some *irreC*² flies show weak eye roughening, and that allele over *rst*⁶ exhibits rough eyes but normal optic lobes.



In(3R)Antp^{LC/+}

Le Calvez, 1948, Bull. Biol. France Belg. 82: 97-113.

CHROMOSOMES

IDENTIFYING SYMBOLS. The standard chromosome sequence or arrangement is the one on which the standard genetic map and the standard polytene chromosome map are based. Chromosome breakage and reunion can give rise to new chromosome sequences; i.e., chromosome aberrations.

As the number of interacting breaks increases, there is a geometrical increase in the number and complexity of possible rearrangements. Rather than a descriptive name and symbol for every type, chromosome aberrations are classified as deficiencies, duplications, inversions, rings, translocations, and transpositions; these are abbreviated *Df*, *Dp*, *In*, *R*, *T*, and *Tp*, respectively. Deficiencies, inversions, rings, and certain duplications and transpositions are intrachromosomal; translocations and other duplications and transpositions are interchromosomal. The abbreviation is followed parenthetically by an indication of the chromosome or chromosomes involved, and then by a specific designation which identifies the particular rearrangement. A brief arbitrarily chosen specific designator is preferred to a lengthy descriptive one. As with mutant symbols, aberration symbols are italicized and do not contain subscripts, or spaces.

Translocations. Translocations are rearrangements in which nonhomologous chromosomes interchange segments and thus involve at least two breaks. No distinction is made in the symbol between simple reciprocal and more complex translocations, nor are the involved arms indicated in the parenthetical chromosomal information. Participating chromosomes are separated by semicolons and listed in the following order: 1 (*X*), *Y*, 2, 3, and 4 [e.g., *T(1;Y;3)127*, *T(1,2,4)^wD2*, and *T(1,4)B^S*]. The chromosomal information (within parentheses) is aligned on the left margin, the different classes being ordered

numerically according to this information (e.g., 1;2, 1;2;3, 1;2;4, 1;3, etc., the *Y* falling between 1 and 2). Within chromosomal classes, translocations are arranged alphabetically according to specific designation. Individual elements of a translocation are denoted by the superscripts *P*, *D*, and *M*. *P* (proximal) refers to the source of the centromere of the element, *D* (distal) to the source of a terminus of different origin from the centromere, and *M* (medial) to the source of any material intercalated between *D* and *P*; segments are designated in the order *P*, *M*, and *D* [e.g., $2^P X^D$ from *T(1;2)Bld*, $2^P 3^M X^D$ of *T(1;2;3)Din*]. This is a departure from previous practice according to which the order was reversed.

Rings. Rejoining of breaks in opposite arms of the same chromosome may give rise to a ring-shaped chromosome. In ring designations the symbol is followed in parentheses by the chromosome involved and then by a specific designation; e.g., *R(1)1*. Ring-shaped *Y* chromosomes are described as *Y* derivatives in the section on special chromosomes.

Inversions. Intrachromosomal aberrations that are not rings and that have at least one section whose map order (either cytological or genetic) is inverted with respect to adjacent regions are designated inversions. In the case of multiple-break intrachromosomal rearrangements, the distinction between inversions and transpositions often becomes ambiguous. An intrachromosomal rearrangement that can be partitioned into a duplicated and a deficient product by exchange with a normal-sequence chromosome is designated a transposition even though it may carry an inverted segment; otherwise, it is designated an inversion. Inversions may involve one arm (paracentric) or both arms (pericentric) of a chromosome; they are symbolized by the abbreviation *In* followed parenthetically by the chromosome arm

or arms involved and then by a specific designation; e.g., *In(2L)Cy*, *In(2LR)bw*^{V1}. *In(1LR)* designates pericentric X-chromosome inversions rather than *Inp(1)*, which was formerly used, and *1L* is implied in *In(1)*. Sometimes one break of a simple autosomal inversion is in the pericentric heterochromatin and is not positioned with respect to the centromere, so that whether the inversion is paracentric or pericentric is indeterminate. In such cases, the parenthetical information consists of chromosome number with no arm designation. Recombination between similar inversions may produce viable recombinant inversions with the left end of one and the right end of the other. Superscripts *L* and *R* are used to identify the sources of the two ends; for example; *In(2R)Cy*^L*bw*^{VDeIR}.

A segment inserted into a new location is in inverted order if its numerical order in salivary-gland-chromosome terminology is inverted with respect to the adjacent segments; it is in dyscentric order if its polarity with respect to the centromere is altered. When a segment from a right arm is inserted into a right arm or left into left, the inverted order is dyscentric; but in left-to-right and right-to-left insertions, the two terms are discordant, the inverted order being eucentric instead of dyscentric.

Transpositions. In this revision, the definition of transposition has been extended beyond that employed by Bridges to include interchromosomal as well as intrachromosomal rearrangements; i.e., we use the transposition category to designate insertional translocations. Transpositions are three-break rearrangements in which a chromosomal segment is removed from one chromosomal region and inserted into another; intrachromosomal transpositions are equivalent to the interchange of two chromosomal segments. The ambiguous distinction between some transpositions and inversions has already been discussed. Among interchromosomal rearrangements, the term transposition is reserved for that class of aberrations in which a segment is deleted from one chromosome and inserted into another, such that a reciprocal duplication and deficiency are generated by random assortment. Reciprocal interchanges of segments between nonhomologous chromosomes are classified as translocations. In the chromosomal designation of transpositions, the donor chromosome precedes the recipient; e.g., *Tp(2;3)P*, *Tp(3;1)ry*³⁵.

Deficiencies. The term deficiency is applied both to deficient chromosomes and deficient genotypes; the distinction between the two is not absolute and the symbol *Df* is used to designate both. A deficient chromosome is one from which a segment, either terminal or interstitial, is missing; whereas, a deficient genotype is one that is hypodiploid or hypotriploid by virtue of having fewer than two or three doses, respectively, of a particular chromosome segment. Deficient chromosomes or deficient genotypes may be produced by induced deletion of segments, by reciprocal translocation between sister or homologous chromatids, as segregants from transpositions, or by recombination between inversions with similar but offset breakpoints. In addition, deficient genotypes may be constructed by combining elements of rearrangements (e.g., reciprocal translocations with similar but offset breakpoints as in the method of segmental aneuploidy). Deficient chromosomes, but not deficient

genomic constructs, are included in this revision. The symbol *Df* is followed by the parenthetical indication of the chromosome arm involved and then by a designator of the specific deficiency. In past editions, deficiencies for genes have been treated as alleles; here we abandon that practice by avoiding superscripts in the specific designations [e.g., *Df(2R)vg-B* not *Df(2R)vg*^B; *Df(3L)st3* not *Df(3L)st*³]; when the deficiency is not named after the deleted gene, however, the superscript is retained [e.g., *Df(1)sc*⁸, not *Df(1)sc*8]. Deficiencies recovered as revertants of dominant mutants are simply denoted as deficiencies for the gene involved. Unless otherwise indicated, deficiencies are assumed to be hemizygous or homozygous lethal. The advent of restriction mapping and nucleotide sequencing has revealed intragenic deletions; these are not treated as deficiencies, but as mutations; at least two adjacent loci must be removed before a chromosome is considered a deficiency.

Duplications. Chromosomes that carry an additional chromosome segment derived either from a sister chromatid, a homologous chromosome or a nonhomologous chromosome are termed duplications and abbreviated *Dp*. As with deficiencies, the distinction can be made between duplicated chromosomes and duplicated genomes, and only the former are described herein. Duplicated chromosomes are formed as the reciprocal product of several of the deficiency-generating events; e.g., reciprocal interchanges between sisters or homologues, segregants from transpositions, and recombinants between inversions with offset breakpoints. In addition, deletion of the majority of a chromosome can generate a small centric element which may be carried as a free duplication. Certain *Y* duplications carrying marker gene(s) are described as *Y* derivatives in the section on special chromosomes. The symbol for duplication follows the same plan as that used for transpositions; the parenthetical chromosomal information contains the chromosome of origin of the duplicated segment listed first followed, after a semicolon, by the recipient chromosome; e.g., *Dp(3;1)O5*, *Dp(1;1)y*^{bl}. When the duplicated segment is carried as a free centric element, the letter *f* (free) follows the semicolon within the parentheses; e.g., *Dp(1;f)101*. A small chromosome segment duplicated *in situ* may be referred to as a repeat, even though it is still symbolized as a duplication; e.g., *Dp(1;1)B*. When the duplicated regions are in the same order, the term tandem repeat is sufficient to specify accurately the new chromosome if the limits of the duplicated segment are known. When these regions are inverted with respect to each other, two reversed repeats are possible, making it necessary to specify which end of the segment is at the axis of symmetry of the repeat; i.e., ABCCBD or ACBBBD. Failure to make such a distinction has given rise to ambiguous descriptions. Recombinations within tandem repeats can lead to formation of triplications and in successive steps to tandem repeats of order higher than three. Such high-order repeats are also symbolized *Dp*, with the number of repeats indicated in the parenthetical chromosomal designation, i.e., *Dp(1;1)* = duplication, *Dp(1;1;1)* = triplication, and so forth.

Combinations of rearrangements. The elementary categories of chromosome aberrations are not mutually exclusive, and some aberrations combine several of them. In such cases the symbol used is the one that

stands highest in the following ranking: $T > \text{interchromosomal } Tp > R > In > \text{intrachromosomal } Tp > Dp > Df$. This is especially so when the components are inseparable. A complicated rearrangement may be separable genetically into its simpler component aberrations, which are usually sufficiently designated with the distinguishing symbol of the original aberration. When, however, the original is named after a phenotype associated with one of the component aberrations, designation of the other component with the symbol of the mutant is inappropriate. A rearrangement superimposed upon another rearrangement may be given a name, which more often than not refers to the entire complex since the newly induced aberration is likely to be inseparable from the original; e.g., $In(2LR)SMI$ is a large pericentric inversion superimposed upon $In(2L)Cy + In(2R)Cy$. Component rearrangements of synthetic combinations of aberrations are occasionally referred to individually, connected with a plus sign; for example, $In(1)sc^{\delta} + In(1)dl-49$ or $In(2L)Cy + In(2R)Cy$. Collecting terms in much the same way as algebraic factoring to further abbreviate the symbol is legitimate; e.g., $In(1)sc^{\delta} + dl-49$ and $In(2L+2R)Cy$. Formerly, chromosomes with more than one inversion were symbolized $Ins()$; we use instead $In()$ for both singly and multiply inverted chromosomes since the presence of more than one inversion is indicated by the specific designation; e.g. $In(1)sc^{S1L}sc^{8R} + S$.

In describing a chromosome, inclusion of several types of information is often desirable; e.g., sequence and gene content. Such categories are separated by a comma followed by a space; e.g., $In(1)dl-49, y w B$, which designates an *X* chromosome carrying the delta-49 inversion, the recessive markers yellow and white, and the dominant marker Bar. Marker genes are listed in the order of the standard genetic map irrespective of their order on the chromosome in question. Three categories of information may be necessary to describe some special chromosomes; e.g., $YSX.YL, In(1)EN+dl-49, y B$, where, besides gene content and sequence, it is informative to designate an abnormal combination of complete chromosome elements.

DESCRIPTIVE SYMBOLS. In addition to identifying symbols just discussed, aberrations are given alternative descriptive symbols indicating points of breakage on the chromosome map; breakpoints are listed in numerical order according to the limits within which they must lie. Each major euchromatic arm as seen in salivary-gland-chromosome preparations is divided into 20 numbered divisions; the heterochromatic complement displays linear differentiation in mitotic prophase and has been subdivided into 61 segments, designated h1 through h61 by Gatti and Pimpinelli (e.g., 1983, Chromosoma 88: 349-73 for the *Y* chromosome). The euchromatic map is numbered sequentially from 1 through 102, with heterochromatic segments h1-h61 interspersed as follows: $YL-YS = h1-h17-h18-h25$; $YL-XR = 1-20 h26-h32-h33-h34$; $2L-2R = 21-40 h35-h37-dh38-h46 41-60$; $3L-3R = 61-80 h47-h52-h53-h58 81-100$; $4 = h59-h61 101-102$; centerpoints indicate centromere positions. Each numbered division on the salivary map is divided into six subdivisions designated by the letters A through F, each of which begins with a heavily staining band; within the lettered subdivisions, the bands are numbered individually. Thus the complete designation of a particular band

consists of its numbered division, its lettered subdivision, and its number; e.g., 3C2. Positions of breakpoints are designated according to the bands between which or the region or regions within which they are known to lie; for example, if a break lies between bands 3C2 and 3C7, its position is designated 3C2-7; for the sake of brevity, the redundant information 3C is omitted from the second half of the notation. Less accurately determined breakpoints may be given less specific designations; e.g., 3C or 3. An example of the total designation, both identifying and descriptive, is as follows: $Tp(2;3)P = Tp(2;3)58E3-F2;60D14-E2;96B5-C1$, items of chromosomal information being separated by semicolons without spaces. Breakpoints are listed in order. Often heterochromatic breakpoints are determined only in polytene preparations; in this case they are designated according to polytene locations 20F, 40, 41, 80, 81, 101 for *X*, *2L*, *2R*, *3L*, *3R*, and *4*, respectively; when prophase determinations have been made h1-h61 designations are employed. *X*-heterochromatin breaks are given arbitrarily as 20F. Apparent terminal deficiencies carry a single breakpoint designation. Descriptions of incompletely analyzed rearrangements incorporate the known information.

Descriptive symbols are used simply as a shorthand method for providing information about the aberration; they supplement rather than substitute for the identifying symbols. We have attempted to give breakpoints according to the revised salivary gland chromosome maps published by C. B. and P. N. Bridges rather than according to C. B. Bridges's original maps, in which individual bands were not numbered. No special notation is used to designate doublet bands; the member of the doublet closer to the breakpoint alone is listed.

Breaks rejoin cyclically to produce chromosome aberrations (e.g., A with B and B with A) and multiple breaks may rejoin in one or more cycles. Thus four breaks may interact to form one four-break rearrangement or two two-break rearrangements. A complex rearrangement consisting of two or more simple cyclic rearrangements is indicated in the descriptive symbol; e.g.

$T(2;3)OR72 = T(2;3)19E;29F + In(2LR)24F;54B$

or

$T(1;2)C314 = T(1;2)5D;40-41$
 $+ T(1;2)9D;51D + T(1;2)20;56F.$

The order in which the component rearrangements are listed in complex descriptive symbols follows the hierarchy according to which the identifying symbol is determined. For a rearrangement superimposed upon a pre-existing rearrangement a similarly compound designation is used except that the plus symbol is replaced by the word on. If one of the new associations of the preexisting rearrangement is broken by the superimposed aberration, then the descriptive symbol is written as though the entire aberration occurred at one time rather than stepwise. An example is: $T(1;4)w^{m52b13}$, which was superimposed upon $In(1)rst^2$, is designated

$T(1;4)2A2-3;3C3-4;20B;101$

since 20B, which was originally adjacent to 3C3, has

become associated with 2A2 and 3C3 with 101. A cyclic rearrangement was produced involving both the preexisting breakpoints and the subsequently occurring ones; i.e., the symbol cannot be written as the old and the new cyclic rearrangements.

NEW ORDERS. In an aberration having only two breakpoints, the new order follows unambiguously from the descriptive symbol. In heterochromatic rearrangements, however, an ambiguity in the position of the breakpoint with respect to the centromere may lead to ambiguities in order. Thus, for example, *T(1;2)8F;40-41* has chromosome 2 broken into two pieces, one extending from 21 to 40 and the other from 41 to 60. Since it is not known which piece is centric, it is not possible to state to which portion of chromosome 2 the acentric portion of the X extending from 1 to 8F is attached. With three or more breakpoints more than one new order is possible; specifying the breakpoints is therefore not sufficient to describe the aberration. We have adopted the following conventions for specifying sequences of aberrations. The sequence of each chromosome involved in an aberration is specified from one end to the other according to salivary gland chromosome terminology. Points of breakage and reunion are indicated by vertical bars, and segments between these points are designated by the most extreme band known to be included at each end, separated by a dash. Thus the order of

$$Tp(2;3)P = T(2;3)58E3-F2;60D12-E2;96B5-C1$$

is represented as follows:

21 - 58E3|60E2 - 60F;
61 - 96B5|60D14 - 58F2|96C1 - 100.

Were the order of the inserted segment 60D14 - 58F2 not known, the segment would have been included within parentheses; i.e.,

61 - 95B5|(58F2 - 60D14)|96C1 - 100;

hierarchies of ambiguities are represented by parentheses within parentheses. Salivary terminology is not italicized except when part of an aberration symbol, either identifying or descriptive. Use of information on order depends only on remembering that chromosome 1 extends from 1 through 20, with the centromere and the proximal heterochromatin in 20F in polytenes or h26-h34 in prophase chromosomes, the Y chromosome from h1 through h25 with the centromere between h17 and h18, chromosome 2 from 21 through 60 with the centromere between 40 and 41, chromosome 3 from 61 through 100 with the centromere between 80 and 81 and chromosome 4 from 101 through 102 with the centromere in 101D. The first breakpoint in *Tp(2;3)P* is listed as 58E3-F2; the first segment indicated in the sequential formula goes through band 58E3, and the inserted segment begins with 58F2. Nothing is implied about the position of the intervening bands 58E4 to 58F1; unless they are specifically described as missing, they are assumed to exist in association with one or the other or both fragments produced by the break. Information on new order is written as follows: each chromosomal element starts at the free end with the lower value and the elements are listed in

ascending order, Y falling between 20 and 21.

When desirable, the centromere position is designated with a centerpoint; in special cases where centromeres and breakpoints coincide, as is frequently true with ring-X chromosomes, the centerpoint replaces the vertical line.

Rings are differentiated from rod-shaped chromosomes by vertical bars at the beginning and end of the element; the circle is broken for linear designation at the breakpoint with the lowest numerical value; e.g., |1A4 - 20.20F - 20A1| for *R(1)2*. In multiple-break rearrangements in which there is a break in autosomal heterochromatin whose position with respect to the centromere is ambiguous, the new order may be written in two ways depending on the position assumed for the heterochromatic break. In such cases, we have usually assumed (for the sake of supplying the remainder of the new order) that the heterochromatic break is in region 40 for breaks in chromosome 2 and 80 for breaks in chromosome 3.

FORMAT. The chromosome aberrations are now listed in alphanumerical order according to symbol, which is in bold face. Names, where necessary, are listed (also in bold face) with cross-references to symbols; synonymic names and symbols appear in italics with cross-references to current symbols. Each aberration description is written in the following format:

symbol: name

cytology: The descriptive symbol as discussed above.

new order: As discussed in the preceding paragraphs.

origin: The inducing agent is listed; aberrations recovered from untreated parents are listed as spontaneous or naturally occurring, depending on whether recovered as a single occurrence or repeatedly.

discoverer: Name, date.

synonym: Alternative symbols or names, or both.

references: Sources of descriptions of the aberrations listed in this section, although bibliographic information may appear under other categories as well.

genetics: Effects of the aberration on the expression of genes near the breakpoints and phenotypic effects not yet attributable to known genes are described. Genes known to be present or absent in duplications and deficiencies indicated. Segregational and recombinational behavior may also be described. Descriptions of aneuploid derivatives are also included in this category.

molecular biology: In long genomic walks in extensively studied regions, the molecular coordinates of rearrangement breakpoints have been determined. These are generally given in terms of kilobases from the origin, which may be a rearrangement breakpoint, the insertion point of a transposable element, or an arbitrarily chosen restriction site. Directionality of molecular coordinates are usually, but not invariably, concordant with that of the cytological map.

other information: In rare instances, information not fitting into other categories is included here.

Groups of rearrangements that share common features or names are tabulated wherever feasible rather than being listed as separate entries. Their distinguishing features are represented in columns whose orders approximate that of the categories of information listed in full entries.

DEFICIENCIES

Del(1): see *Dp(1;f)*

Del(X^{c2}): see *Dp(1;f)R*

Df-3L^K: see *Df(3L)K*

Df(1)0-sc,LVM: see *Df(1)260-1*

Df(1)05-22-1

references: Fleming.

genetics: Deficient for *l(1)IAa* through *l(1)IAd*.

Df(1)1-96

references: Fleming, DeSimone, and White, 1989, Mol. Cell Biol. 9: 719-25.

genetics: Deficient for *l(1)IAa* through *ewg*.

Df(1)1D1

cytology: *Df(1)14D;15C*.

references: Mason, Green, Shaw, and Boyd, 1981, Mutat. Res. 81: 329-43.

genetics: Deficient for *M(1)14F*.

Df(1)2/9A

cytology: *Df(1)20B;20C*.

origin: Induced by MR.

references: Eeken, Sobels, Hyland, and Schalet, 1985, Mutat. Res. 150: 261-75.

genetics: Deficient for *l(1)20Bb¹*.

Df(1)2/19B

cytology: *Df(1)19F*.

origin: Induced by MR.

references: Eeken, Sobels, Hyland, and Schalet, 1985, Mutat. Res. 150: 261-75.

genetics: Deficient for *ftil-l(1)19Fg²*.

Df(1)2F1-3A4

cytology: *Df(1)2F1;3A4*.

discoverer: Green.

references: Perrimon, Engstrom, and Mahowald, 1984, Genetics 108: 559-72.

1985, Genetics 111: 23-41.

genetics: Deficient for *l(1)2Fb-gt*.

molecular biology: Breakpoint mapped to the DNA at +73.5 (Xho site) and +75.0 (Hind III site) by Haenlin, Steller, Pirrotta, and Mohier, 1985, Cell 40: 827-37.

Df(1)5-13

genetics: Deficient for *l(1)IAa* through *l(1)IAc*.

****Df(1)7aA: Deficiency (1) 7a from Austin***

cytology: *Df(1)3C3-5;3C7-9*; inferred from Mackensen's fig. 15F (1935).

origin: X ray induced.

references: Mackensen, 1935, J. Heredity 26: 163-74 (fig.).

genetics: Deficient for *fa* and *spl* but not *w* or *ec*; heterozygous female *N*.

Df(1)10-70d

cytology: *Df(1)8D3;8D8-9*.

origin: Induced by mutator gene *mu*.

references: Green and Lefevre, 1972, Mutat. Res. 16: 59-64.

genetics: Deficient for *lz*.

Df(1)10RA

cytology: 8-13 band deficiency including 7A.

discoverer: Cline.

references: Nicklas and Cline, 1980, Genetics 94: s76.

genetics: Deficient for *Sxl* but not *cm* or *ct*.

Df(1)11-83

cytology: *Df(1)2F2;3A6*.

discoverer: Schalet.

Df(1)12-70b

cytology: *Xh26-Xh33*.

origin: Induced by mutator gene *mu*.

references: Green and Lefevre, 1972, Mutat. Res. 16: 59-64.

Df(1)13-70b

cytology: *Df(1)1A7;1B4*.

origin: Induced by mutator gene *mu*.

references: Green and Lefevre, 1972, Mutat. Res. 16: 59-64.

Df(1)13C3

cytology: *Df(1)20A3;20E-F*.

origin: X ray induced.

references: Schalet and Lefevre, 1973, Chromosoma 44: 183-200.

genetics: Deficient for *wap* to *su(f)*.

****Df(1)14zA***

origin: X ray induced.

discoverer: Mackensen.

references: 1935, J. Heredity 26: 163-74 (fig.).

genetics: Deficient for *f* but not *fw* or *r*.

Df(1)16-, Df(1)17-, Df(1)18-

cytology: A series of deficiencies for the proximal-most X-linked genes; breakpoints tabulated below.

origin: Neutron induced.

discoverer: Munoz.

references: Schalet, 1972, DIS 49: 36-37.

Schalet and Lefevre, 1973, Chromosoma 44: 183-200.

1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 847-902.

Yamamoto and Miklos, 1987, DIS 66: 154-55.

Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31.

deficiency	cytology	genetics
<i>Df(1)16-2-13</i>	{20A1-2;20A4}	<i>wap⁻-uncl⁻</i>
<i>Df(1)16-2-19</i>	19A5;19D3	<i>ot⁻-mal⁻</i>
<i>Df(1)16-3-22</i>	19D1;20A2	<i>mal⁻-eo⁻</i>
<i>Df(1)16-3-35^α</i>	19D2-3;19E3-4	<i>mal⁻-shakB⁻</i>
<i>Df(1)16-3-112</i>	20E;20F	<i>su(f)^{Pb⁻}</i> - <i>su(f)⁻</i>
<i>Df(1)16-3-162</i>	20E;20F	<i>su(f)^{Pb⁻}</i> - <i>su(f)⁻</i>
<i>Df(1)16-129</i>	19F1;19F3	<i>fti⁻-fti⁻</i>
<i>Df(1)16-185</i>	20E;20F	<i>su(f)^{Pb⁻}</i> - <i>su(f)⁻</i>
<i>Df(1)17-8</i>		<i>l(1)IAa⁻-l(1)IAc⁻</i>
<i>Df(1)17-59</i>	19F5;20E-F	<i>l(1)19Fg⁻-su(f)⁻</i>
<i>Df(1)17-87^β</i>	20D-E;20F	<i>sph⁺ su(f)⁻-bb⁻</i>
<i>Df(1)17-137^γ</i>	20A;20E-F	<i>eo⁻-su(f)⁻</i>
<i>Df(1)17-148</i>	20B-C;20E-F	<i>l(1)20Cb⁻-su(f)⁻</i>
<i>Df(1)17-252</i>	20B;20C	<i>fog⁻-l(1)20Ca⁻</i>
<i>Df(1)17-257</i>	19F3;20A1-2	<i>fti⁻-eo⁻</i>
<i>Df(1)17-351</i>	19E;19F	<i>l(1)19Ec⁸⁻-l(1)19Fg²⁻</i>
<i>Df(1)17-408</i>	20A;20F	<i>uncl⁻-su(f)⁻</i>
<i>Df(1)17-439</i>	20A;20B	<i>wap⁻-stn⁻</i>
<i>Df(1)17-466</i>	20A;20B	<i>wap⁻-fog⁻</i>
<i>Df(1)17-489^δ</i>	{19E4-5;20E}	<i>l(1)19Ed⁻-su(f)⁻</i>

deficiency	cytology	genetics
Df(1)18-80	{19F3;19F4}	<i>flil</i> ⁻ - <i>l(1)19Fe</i> ⁻
α	Proximal breakpoint located by Yamamoto and Miklos (1987) between 19E3 and 19E4 instead of 19E6 and 19E7 (Schalet and Lefevre, 1976). The corrected breakpoint is consistent with the viability of <i>Df(1)16-3-35</i> when heterozygous with <i>Df(1)A118 = Df(1)19E4-5;19E8</i> .	
β	Also see Rahman and Lindsley, 1981, Genetics 99: 49-64.	
γ	<i>Df(1)17-137bb</i> ^{L-158} females are phenotypically <i>bb</i> and more than 50% of them show <i>eo</i> .	
δ	See Baird, Schalet, and Wyman, 1990, Genetics 126: 1045-59.	

Df(1)17-8

references: Fleming.
genetics: Deficient for *l(1)IAa* through *l(1)IAc*.

Df(1)17-70b: see *Df(1)70b17*

Df(1)18-70d: see *Df(1)70d18*

Df(1)19

cytology: *Df(1)13F;14E*.
discoverer: Boyd.
references: Steller, Fischbach, and Rubin, 1987, Cell 50: 1139-53.
genetics: Deficient for *disco* and adjacent genes (nearly 5% of X euchromatin deleted).

***Df(1)22**

cytology: *Df(1)20E;20F*.
origin: X ray induced.
synonym: *l(1)22*.
references: Schalet, 1972, DIS 49: 36-37.
Schalet and Lefevre, 1973, Chromosoma 44: 183-200.
genetics: Deficient for *su(f)*; mutant for *sph*.

Df(1)23-70b: see *Df(1)70b23*

Df(1)23-70d: see *Df(1)70d23*

Df(1)24

origin: Induced by hybrid dysgenesis.
discoverer: Sobels.
references: Perrimon, Engstrom, and Mahowald, 1984, Genetics 108: 559-72.
genetics: Deficient for *pn - l(1)2Ea*.

***Df(1)24a**

origin: X ray induced.
discoverer: Mackensen.
references: 1935, J. Heredity 26: 163-74 (fig.).
genetics: Deficient for *w* but not *pn* or *fa*.

Df(1)26B

cytology: *Df(1)19F;20*.
origin: Induced by hybrid dysgenesis.
references: Zusman, Coulter, and Gergen, 1985, DIS 61: 217-18.
Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31.
genetics: Deficient for *l(1)19Ec-eo*. Complements *shakB* but not *l(1)19Ec*, *l(1)19Ed*, or *lf*.

Df(1)30A2

cytology: *Df(1)8F;9B*.
origin: Induced by ethyleneimine.
references: Lim and Snyder, 1968, Mutat. Res. 6: 129-37.

Df(1)46-1

origin: Induced by hybrid dysgenesis.
references: Zusman, Coulter, and Gergen, 1985, DIS 61: 217-18.
genetics: Complements *uncl* but not *fog* or *stm*.

Df(1)48-2

origin: Induced by hybrid dysgenesis.
references: Zusman, Coulter, and Gergen, 1985, DIS 61: 217-18.
genetics: Complements *eo* but not *fog* or *l(1)20Cb*.

Df(1)50D3

cytology: *Df(1)9A3-4;9F12*.
discoverer: Nash.
genetics: *ras*⁻-*l(1)9Fe*.

Df(1)54

cytology: *Df(1)19E8-F1;20B3-C1* superimposed on *In(1)1B3-4;20B-D1^L1B2-3;20B-D1^R*.
new order:
1A - 1B3|20D1 - 20C1|19E8 - 1B3|20D1 - 20F.
19F1 - 20B3 missing; 1B3 duplicated.
origin: X ray induced in *In(1)sc^{SILsc}8R*.
references: Schalet and Lefevre, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 846-902.
Craymer and Roy, 1980, DIS 55: 200-04.
genetics: Deficient for *lfl-l(1)20Bb* and duplicated for *sc*.

Df(1)59: see *Df(1)ras59*

***Df(1)60b**

origin: X ray induced.
discoverer: Mackensen.
references: 1935, J. Heredity 26: 163-74 (fig.).
genetics: Deficient for *f* but not *fw* or *r*.

Df(1)62d18

cytology: *Df(1)3B1-2;3C6-7*.
origin: X ray induced.
discoverer: Judd, 62d18.
references: Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56.
genetics: Deficient for *l(1)3Bb-rst* but not *N*.
molecular biology: Left breakpoint localized to an approximately 4 kilobase restriction fragment fifty kilobases to the left of the origin of a 160 kb walk (Gunaratne, Mansukhani, Lipari, Liou, Martindale, and Goldberg, 1986, Proc. Nat. Acad. Sci. USA 83: 701-05).

Df(1)62g18

cytology: *Df(1)3A1-2;3A4-5*.
origin: X ray induced.
discoverer: Judd, 62g18.
references: Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56.
Kaufman, Shannon, Shen, and Judd, 1975, Genetics 79: 265-82.
Honisch and Campos-Ortega, 1982, DIS 58: 76-77.
genetics: Deficient for *gt - l(1)3Ac* but not *l(1)3Ad*. Embryos hemizygous for the deficiency show defects of the cuticle, central nervous system, and head due to absence of the *gt* locus (Honisch and Campos-Ortega, 1982). Germ-line clones lethal (García-Bellido and Robbins, 1983, Genetics 103: 235-47).

Df(1)64c4

cytology: *Df(1)3A3-4;3C2-3*.
origin: X ray induced.
discoverer: Judd, 64c4.
references: Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56.
 Kaufman, Shannon, Shen, and Judd, 1975, Genetics 79: 265-82.
genetics: Deficient for all known loci between *z* and *w*. Also deficient for *w* but not for *z*. Male lethal in embryo.
molecular biology: Left breakpoint localized to a 4 kilobase BamHI restriction fragment near the origin of a 160-kb walk (Gunaratne, Mansukhani, Lipari, Liou, Martindale, and Goldberg, 1986, Proc. Nat. Acad. Sci. USA 83: 701-05).

Df(1)64c18

cytology: *Df(1)2E1-2;3C2*.
origin: X ray induced.
discoverer: Lefevre.
references: Craymer and Roy, 1980, DIS 55: 200-04.
 Gvozdev, Gerasimova, Kovalev, and Ananiev, 1977, DIS 52: 67-68.
 Perrimon, Engstrom, and Mahowald, 1984, Genetics 108: 559-72.
 1985, Genetics 111: 23-41.
genetics: Deficient for *pn - w*.

Df(1)64f1

cytology: *Df(1)3A9-B1;3B2-3*.
origin: X ray induced.
discoverer: Abrahamson, 64f1.
references: Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56.
 Kaufman, Shannon, Shen, and Judd, 1975, Genetics 79: 365-82.
 Young and Judd, 1978, Genetics 84: 723-42.
genetics: Deficient for *l(1)3Ba*, *per* and *l(1)3Bb* but not *l(1)3Ag* or *l(1)3Bd*. Larval lethal (Judd). Cell lethal in cuticle and in germ-line clones. (Ripoll and García-Bellido, 1979, Genetics 91: 443-53).
molecular biology: Right breakpoint 82-85 kb distal to the *w^a* copia insertion point (Pirrota, Hadfield, and Pretorius, 1983, EMBO J. 2: 927-34; Reddy, Zehring, Wheeler, Pirrota, Hadfield, Hall, and Rosbash, 1984, Cell 38: 701-10).

Df(1)64j4

cytology: *Df(1)3A8-9;3B1-2*.
origin: Found in stocks in separable combination with *Df(1)w²⁵⁸⁻⁴⁵*.
discoverer: Judd, 64j4.
references: Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56.
 Kaufman, Shannon, Shen, and Judd, 1975, Genetics 79: 265-82.
 Young and Judd, 1978, Genetics 88: 723-42.
genetics: Deficient for *l(1)3Ag* and *l(1)3Ba* but not *l(1)3Ae* or *l(1)3Bb*. Also removes *per* (Bargiello and Young, 1984, Proc. Nat. Acad. Sci. USA 81: 2142-46; Bargiello, Jackson, and Young, 1984, Nature 312: 752-54; Reddy, Zehring, Wheeler, Pirrota, Hadfield, Hall, and Rosbach, 1984, Cell 38: 701-10). Cell lethal in cuticle (Ripoll and García-Bellido, 1979, Genetics 91: 443-53). Germ-line clones lethal (García-Bellido and Robbins, 1983, Genet-

ics 103: 235-47).
molecular biology: Right breakpoint between 89 and 98 kb distal to the *w^a* copia insertion point (Pirrota, Hadfield, and Pretorius, 1983, EMBO J. 2: 927-34).

Df(1)65j26

origin: X ray induced.
references: Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56.
 Kaufman, Shannon, Shen, and Judd, 1975, Genetics 79: 365-82.
 García-Bellido and Ripoll, 1973, Nature 273: 399-400.
genetics: Deficient for *gt-l(1)3Ac*. Male lethal in embryo but cell viable in cuticle (Ripoll and García-Bellido, 1979, Genetics 91: 443-53).

Df(1)70

cytology: A series of four distally located deficiencies; breakpoints tabulated below.
origin: Induced by mutator gene *mu*.
references: Green and Lefevre, 1972, Mutat. Res. 16: 59-64.

deficiency	synonym	cytology
<i>Df(1)70b17</i>	<i>17-70b</i>	<i>1A1;1B1-2</i>
<i>Df(1)70b23</i>	<i>23-70b</i>	<i>2F6;3C5</i>
<i>Df(1)70d18</i>	<i>18-70d</i>	<i>1E3;2A3</i>
<i>Df(1)70d23</i>	<i>23-70d</i>	<i>1A1;1B1-2</i>

Df(1)80 - Df(1)82

origin: X ray induced in *Dp(1;4)r⁺*.
synonym: *Df(1)para*.
references: Falk, Roselli, Curtiss, Halladay, and Klufas, 1984, Mutat. Res. 126: 25-34.
genetics: Deficient for *para*.

deficiency	cytology	genetics
<i>Df(1)80e3d^α</i>	<i>14B5-18;15A6-11</i>	<i>l(1)14Ba-l(1)15Ab</i>
<i>Df(1)80e19a</i>	<i>14A1-2;15A6-11</i>	<i>l(1)14Ab-l(1)15Ab</i>
<i>Df(1)80e27a</i>	<i>14A1-2;15A6-11</i>	<i>l(1)14Ba-l(1)15Ab</i>
<i>Df(1)80f3c</i>	<i>14A1-2;14E</i>	<i>l(1)14Aa-l(1)14Ea</i>
<i>Df(1)80f8b</i>	<i>14A1-2;14F</i>	<i>l(1)14Aa-l(1)14Ea</i>
<i>Df(1)80f15f</i>	<i>14B5-18;15A6-11</i>	<i>l(1)14Ba-l(1)15Ab</i>
<i>Df(1)80f18c</i>	<i>14C4-5;15A3-4</i> (Banga and Boyd)	<i>para-l(1)15Aa</i>
<i>Df(1)80f25a</i>	<i>14A1-2;15A6-11</i>	<i>l(1)14Ba-l(1)15Ab</i>
<i>Df(1)80f29d</i>	<i>14B5-18;15A6-11</i>	<i>l(1)14Ba-l(1)15Ab</i>
<i>Df(1)80g12a</i>	<i>14B3-4;15A6-11</i>	<i>l(1)14Ba-l(1)15Ab</i>
<i>Df(1)81f12a</i>	<i>14B5-18;15A6-11</i>	<i>para-l(1)15Aa</i>
<i>Df(1)81f20a</i>	<i>14B5-18;15A3-4</i>	<i>l(1)14Ba-l(1)15Aa</i>
<i>Df(1)81f20e</i>	<i>14B5-18;15A6-11</i>	<i>l(1)14Ba-l(1)15Ab</i>
<i>Df(1)81g11</i>	<i>14B5-18;15A6-11</i>	<i>l(1)14Ba-l(1)15Ab</i>
<i>Df(1)81h24b</i>	<i>14B5-18;14E</i>	<i>l(1)14Ba-para</i>
<i>Df(1)81i21c</i>	<i>14B3-4;14C6-8</i>	<i>l(1)14Ba-para</i>
<i>Df(1)81i25b</i>	<i>14B5-18;15A6-11</i>	<i>l(1)14Ba-l(1)15Ab</i>
<i>Df(1)81j6c</i>	<i>14B5-18;15A6-11</i>	<i>l(1)14Ba-l(1)15Ab</i>
<i>Df(1)81j6e</i>	<i>14B5-18;15A6-11</i>	<i>l(1)14Ba-l(1)15Ab</i>
<i>Df(1)81j16f</i>	<i>14A1-2;15A6-11</i>	<i>l(1)14Aa-l(1)15Ab</i>
<i>Df(1)81j23a</i>	<i>14B5-18;15A6-11</i>	<i>l(1)14Ba-l(1)15Ab</i>
<i>Df(1)81j29i</i>	<i>14B5-18;15A1-2</i>	<i>l(1)14Ba-l(1)14Fa</i>
<i>Df(1)81k19b^α</i>	<i>14B5-18;15B</i>	<i>l(1)14Ba-l(1)15Ab</i>
<i>Df(1)81k21e</i>	<i>14B5-18;15A5</i>	<i>l(1)14Ba-l(1)15Ab</i>
<i>Df(1)81k23b</i>	<i>14B5-18;15A3-4</i>	<i>l(1)14Ba-l(1)15Aa</i>
<i>Df(1)81i11b^β</i>	<i>14B5-18;15A3-4</i>	<i>l(1)14Ba-l(1)15Aa</i>
<i>Df(1)81l8g</i>	<i>14C6-8;15A3-4</i>	<i>para-l(1)14Fa</i>
<i>Df(1)81i12h</i>	<i>14B5-18;15A6-11</i>	<i>l(1)14Bb-l(1)15Ab</i>
<i>Df(1)81i17h</i>	<i>14A1-2;15A1-2</i>	<i>l(1)14Aa-l(1)14Fa</i>
<i>Df(1)81i19i</i>	<i>14B5-18;15A1-2</i>	<i>l(1)14Aa-l(1)14Fa</i>
<i>Df(1)81i28f</i>	<i>14B5-18;15A6-11</i>	<i>l(1)14Ba-l(1)15Ab</i>
<i>Df(1)82a2y</i>	<i>14B5-18;15A3-4</i>	<i>para-l(1)15Aa</i>
<i>Df(1)82a2z</i>	<i>14B5-18;15A3-4</i>	<i>l(1)14Ba-l(1)15Aa</i>
<i>Df(1)82b6w^α</i>	<i>14B5-18;14E</i>	<i>l(1)14Ba-para</i>

deficiency	cytology	genetics
<i>Df(1)82b10w</i> ^β	14C6-8;15A3-4	<i>para-l(1)15Aa</i>
<i>Df(1)82b26c</i>	14C6-8;15A3-4	<i>para-l(1)15Aa</i>
<i>Df(1)82c3k</i> ^α	14B3-4;14E	<i>l(1)14Ba-para</i>
<i>Df(1)82c5b</i>	14B5-18;15A3-4	<i>l(1)14Ba-l(1)15Aa</i>
<i>Df(1)82c19i</i>	14A1-2;14D3-4	<i>l(1)14Ba-para</i>
<i>Df(1)82c25g</i>	14A1-2;15A3-4	<i>l(1)14Aa-l(1)15Aa?</i>
<i>Df(1)82d7e</i>	14B5-18;14D3-4	<i>l(1)14Ba-para</i>

^α Deficient for *diss* (Kulkarni, Steinlauf, and Hall, 1988, Genetics 118: 267-85).
^β Complex.

***Df(1)172-Df(1)247g**

origin: X ray induced.
 references: Patterson, 1932, Am. Naturalist 66: 193-206.

deficiency	genetics
<i>*Df(1)172</i>	<i>pn⁻-ec⁻</i>
<i>*Df(1)231c</i>	<i>v⁻</i>
<i>*Df(1)235</i>	<i>pn⁻-ec⁻</i>
<i>*Df(1)244</i>	<i>m⁻</i>
<i>*Df(1)247a</i>	<i>m⁻</i>
<i>*Df(1)247g</i>	<i>w⁻</i>

Df(1)203: see *Df(1)ras203*

Df(1)208 - Df(1)228

origin: Induced by methyl methanesulfonate.
 references: Parádi, Vogel, and Szilágyi, 1983, Mutat. Res. 111: 145-59.
 genetics: Recessive lethal.

deficiency	cytology
<i>Df(1)208</i>	20C-D;F
<i>Df(1)216</i> ^α	20A;20A
<i>Df(1)217</i> ^α	19D-E;20F
<i>Df(1)218</i>	19F;19F
<i>Df(1)228</i>	19A-B;19A-B

^α Double mutant.

Df(1)217: see *Df(1)ras217*

***Df(1)258-44**: see *T(1;2;3)w^{m258-44}*

Df(1)259

origin: X ray induced in y.
 synonym: *l²⁵⁹*, *l(1)Jl²⁵⁹*.
 references: Lindsley, Edington, and Von Halle, 1960, Genetics 45: 1649-70.
 genetics: Male lethal with normal Y but viable with y⁺Y. Deficient for *l(1)IAa* through *l(1)IAC*.

Df(1)260-1 - *Df(1)260-19

origin: *Df(1)260-1* is spontaneous; the other deficiencies are X ray induced.

deficiency	cytology	ref ^α	genetics
<i>Df(1)260-1</i> ^β	1A1;1B4-6	1, 2, 3, 5, 7	<i>y⁻-l(1)IBb⁻</i> ; male lethal, cell viable
<i>*Df(1)260-2</i>	1B2-3;1B2-3	1, 4, 5	<i>y⁻-ac⁻sc⁺</i> ; male lethal, cell viable
<i>*Df(1)260-5</i>	1A4-5;1A4-5	1, 4, 5	viable
<i>*Df(1)260-10</i>	1A2-3;1A2-3	1, 4, 5	viable
<i>*Df(1)260-19</i>	1A2-3;1A2-3	1, 4, 5	viable

^α 1 = Demerec and Hoover, 1936, J. Hered. 27: 206-12; 2 = Ephrussi, 1934, Proc. Nat. Acad. Sci. USA 10: 420-22; 3 = García-Bellido and Santamaria, 1978, Genetics 88: 469-86; 4 = Sutton, 1940,

Genetics 25: 628-35; 5 = Sutton, 1943, Genetics 28: 214; 6 = Walen, 1961, Genetics 46: 93-103; 7 = White, Decelles, and Enlow, 1983, Genetics 104: 433-48.
^β Germ-line clones lethal (García-Bellido and Robbins, 1983, Genetics 103: 235-47). Eliminates all external sense organs in embryo (Dambly-Chaudière and Ghysen, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 222-28).

***Df(1)262-*Df(1)354**

origin: X ray induced.
 references: Patterson, 1932, Am. Naturalist 66: 193-206.
 genetics: Tabulated.

deficiency	genetics
<i>*Df(1)262</i>	N
<i>*Df(1)267</i>	N
<i>*Df(1)268</i>	<i>car⁻</i>
<i>*Df(1)271</i>	N
<i>*Df(1)274</i>	<i>f⁻</i>
<i>*Df(1)303</i>	N
<i>Df(1)306</i>	<i>y⁻-ac⁻</i>
<i>*Df(1)308</i>	<i>fa⁻-ec⁻</i>
<i>*Df(1)314</i>	<i>pn⁺ w⁻-ec⁻ bi⁺</i>
<i>*Df(1)354</i>	<i>pl⁻</i>

Df(1)278.4B1

cytology: *Df(1)2E3-F3;3A5-B4*.
 discoverer: Wu.
 references: Perrimon, Engstrom, and Mahowald, 1984, Genetics 108: 559-72.
 1985, Genetics 111: 23-41.
 genetics: Deficient for *fs(1)K10-l(1)3Ac*.
 molecular biology: Breakpoint mapped to the DNA between +5.5 (*Sal1* site) and +9.0 (*EcoR1* site) by Haenlin, Steller, Pirrotta, and Mohier, 1985, Cell 40: 827-37.

Df(1)306

origin: Induced by hybrid dysgenesis.
 references: Pelisson, 1981, Mol. Gen. Genet. 183: 123-29.
 genetics: Deficient for y and ac.

Df(1)403

cytology: *Df(1)1A4* (or more complex rearrangement). Acts like a terminal deficiency for all loci distal to *l(1)IAC*.
 synonym: *l(1)403*, *Df(1)l(1)403*.
 references: Maddern, 1977, DIS 52: 82.
 genetics: Exposes *l(1)IAa*, *l(1)IAb* and *l(1)IAC*. Covered by *B^{Sy}y⁺* but not by *B^{Sy}y^{3id}*.

Df(1)724

cytology: *Df(1)20D;20F*.
 synonym: *Df(1)l724*.
 discoverer: Lifschytz.
 references: Ripoll and García-Bellido, 1978, Genetics 91: 443-53.
 genetics: Zygotic lethal but cell viable in cuticle.

Df(1)733

cytology: *Df(1)20D;20F*.
 synonym: *Df(1)l733*.
 discoverer: Lifschytz.
 references: Ripoll and García-Bellido, 1978, Genetics 91: 443-53.
 genetics: Zygotic lethal but cell viable in cuticle.

Df(1)1237

discoverer: Gans.
references: Laurence, Johnson, and Struhl, 1983, Cell 35: 27-34.
genetics: Dominant female sterile (agametic); deficient for *ovo*.

Df(1)7108

cytology: {Df(1)19E1-2;20E-F}.
references: Lifschytz and Jakobovitz, 1978, Mol. Gen. Genet. 161: 275-84.
genetics: Deficient for *run-su(f)*. Covered by *y⁺Ymal⁺*.

Df(1)A

deficiency	cytology	origin	ref ^α	genetics
Df(1)A19^β	20;20F	X ray	8, 9	<i>wap⁻-bb⁻</i>
Df(1)A23	1A1;1A6-7	X ray	7	<i>l(1)1Aa⁻-l(1)1Ac⁻</i>
Df(1)A33	20A;20F	X ray	8, 9	<i>l(1)20Ac⁻-bb⁻</i>
Df(1)A53	19E5;20A1-2	X ray	3, 7	<i>l(1)19Ed⁻-eo⁻</i>
Df(1)A58^γ		X ray	9	<i>mal⁻-run⁻</i>
Df(1)A82	3D4-5;4E3 + <i>In(1)12C6;14A8</i>	X ray	5	<i>l(1)EFa⁻</i>
Df(1)A94	1E4-5;2B11-12	X ray	3, 4, 5, 6, 7	<i>fnf⁻</i>
Df(1)A113	3D6-E1;4F5	X ray	5, 7	<i>slc⁻-rb⁻</i>
Df(1)A118^δ	19E3-4;19E7-8	X ray	3, 6, 8, 9, 10, 12, 13	<i>shakB⁻-vao⁻</i>
Df(1)A122	cytologically normal	X ray	8, 9, 12, 13	<i>wap⁻-l(1)20Ac⁻</i>
Df(1)A129	{20A3-20E}	X ray	8, 9	<i>wap⁻-su(f)⁻</i>
Df(1)A209^β	20A;20F	X ray	8, 9, 11	<i>l(1)20Ac⁻-bb⁻</i> or <i>uncl⁻-bb⁻</i>
Df(1)A211	{20A3;20E}	X ray	8, 9	<i>wap⁻-su(f)⁻</i>
Df(1)At127	1E3-4;2A2-3	EMS	1, 2	<i>l(1)1Ed⁻-l(1)2Ad⁻</i>

^α 1 = Aizenzon and Belyaeva, 1982, DIS 58: 3-7; 2 = Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90; 3 = Baird, Schalet, and Wyman, 1990, Genetics 126: 1045-59; 4 = Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelova, and Zhimulev, 1980, Chromosoma 81: 181-306; 5 = Craymer and Roy, 1980, DIS 55: 200-04; 6 = Kramers, Schalet, Paradi, and Huizer-Hoogteyling, 1983, Mutat. Res. 107: 187-201; 7 = Lefevre; 8 = Lifschytz, 1971, Mutat. Res. 13: 35-47; 9 = Lifschytz and Falk, 1968, Mutat. Res. 6: 235-44; 10 = Lifschytz and Jakobovitz, 1978, Mol. Gen. Genet. 161: 275-84; 11 = Rahman and Lindsley, 1981, Genetics 99: 49-64; 12 = Schalet and Lefevre, 1973, Chromosoma 44: 183-200; 13 = Schalet and Lefevre, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 846-902.

^β Male sterile in combination with *y⁺Ymal⁺* (Rahman and Lindsley, 1981).

^γ *Df(1)A58/Df(1)A118* survives (Schalet and Lefevre, 1976) and shows *vao*.

^δ *Df(1)16-3-35/Df(1)A118* survives (Schalet and Lefevre, 1976) and shows *shakB* (Baird).

***Df(1)A1:** see **Tp(1;4)A1**

***Df(1)A12:** see **T(1;2;4)A12**

Df(1)ac: Deficiency (1) achaete

origin: X ray induced simultaneously with a detachment of an attached X marked with *y*.
references: Muller, 1954, DIS 28: 146-47.
genetics: Deficient for *ac* and probably *y*. Male viable.

Df(1)arth: Deficiency (1) arthritic

origin: X ray induced in *In(1)sc^{S1L}sc^{8R}In(1)dl-49* chromosome.
discoverer: Schalet.
references: Fleming, DeSimone, and White, 1989, Mol. Cell Biol. 9: 719-25.
genetics: Deficient for *arth-ac* but not *sc*. Male and female viable.

Df(1)At127

cytology: *Df(1)1E3-4;2A2-3*.
origin: Induced by ethyl methanesulfonate.
references: Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90.
genetics: Deficient for *l(1)1EFa-l(1)1EFj*.

Df(1)B: Deficiency (1) Bar

cytology: *Df(1)15F9-16A1;16A6-7* superimposed on *Dp(1;1)15F9-16A1;16A7-B1*. All of the left 16A section and all but 16A7 of the right 16A section is included in the deficiency. Sutton (1943), confirmed by Peterson and Laughnan (1963), included *f* as if the left breakpoint were more distal than indicated.

new order:

1 - 15F9|16A7 - 20.

origin: X ray induced in B chromosome.

discoverer: Demerec, 34a.

synonym: *Df(1)B263-20*.

references: Sutton, 1943, Genetics 28: 97-107 (fig.). Peterson and Laughnan, 1963, Proc. Nat. Acad. Sci. USA 50: 126-33.

Laughnan, 1970, Mol. Gen. Genet. 108: 93-96.

genetics: Reversion of B. Deficient for *f-B*. Male lethal.

Df(1)B12-Df(1)B264

origin: X ray induced.

deficiency	cytology	ref ^α	genetics
Df(1)B12^β	19E1;20A1-2	1, 2, 4, 5	<i>run⁻-eo⁻</i>
Df(1)B18	20F;20F	2	includes <i>bb</i>
Df(1)B47^γ	20F;20F	2	includes <i>bb</i>
Df(1)B57	19E1-2;19F1	1, 2, 3, 4, 6	<i>run⁻-unc⁻</i>
Df(1)B82	19F2-3;20F	1, 2	<i>l(1)19Fb⁻-bb⁻</i>
Df(1)B85		2	<i>wap⁻-bb⁻</i>
Df(1)B99		2	includes <i>bb</i>
Df(1)B111	20A;20F	2	<i>wap⁻-bb⁻</i>
Df(1)B155^δ		2	<i>eo⁻-bb⁻</i>
Df(1)B179^ε		2	
Df(1)B182		2	includes <i>bb</i>
Df(1)B199		2	
Df(1)B225		2	
Df(1)B255		2	
Df(1)B264		2	<i>wap⁻-uncl⁻</i>

^α 1 = Falk, 1973, Mutat. Res. 20: 287-90; 2 = Lifschytz and Falk, 1968, Mutat. Res. 6: 235-44; 3 = Lifschytz and Falk, 1969, Genetics 62: 343-52; 4 = Lifschytz and Jakobovitz, 1978, Mol. Gen. Genet. 161: 275-84; 5 = Ripoll and Garcia-Bellido, 1979, Genetics 91: 443-53; 6 = Schalet and Lefevre, 1973, Chromosoma 44: 183-200.

^β Cell viable in cuticle.

^γ Male sterile in combination with *mal⁺Y*.

^δ Also *T(1;2)*.

^ε Also *T(1;3)*.

Df(1)B428

references: Fleming, DeSimone, and White, 1989, Mol. Cell Biol. 9: 719-25.
genetics: Deficient for *l(1)1Aa* through *ewg*.

Df(1)Basc

cytology: *In(1)1B3-4;20F^L1B2-3;20F^R* + *In(1)6A1-3;10F10-11A1* + *Df(1)1A1;1B2*. Deficient for the euchromatin distal to the distal *In(1)sc^{S1}* breakpoint of *Basc*. Origin probably analogous to that of *Df(1)sc⁸*.
discoverer: Abrahamson.
references: Simmons and Lim, 1980, Proc. Nat. Acad. Sci. USA 77: 6042-46.

genetics: Recessive lethal. Covered by y^+Y .

Df(1)bb: Deficiency (1) bobbed

cytology: Deficiencies for most or all of the ribosomal cistrons and neighboring heterochromatin; do not extend into regions not covered by the Y chromosome.

origin: X ray induced.

synonym: *Df(1)bb¹*.

genetics: Deficient for *bb*. X/O male lethal. Segregates irregularly from the Y chromosome. Male sterile or nearly so in combination with *T(Y;A)*'s (Lindsley, Pearson, Rokop, Jones, and Stern, 1979, Genetics 91: s69-70) or with y^+Ymal^+ (Rahman and Lindsley, 1981).

deficiency	% proximal X heterochromatin deleted	ref ^α
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<i>Df(1)bb3a</i>		1
<i>Df(1)bb74</i>	48	1, 2
<i>Df(1)bb158^β</i>	82	1, 2, 3
<i>Df(1)bb452</i>	32	1, 2, 3
<i>Df(1)bb456</i>	72	1
<i>*Df(1)bb481^γ</i>		1

^α 1 = Lindsley, Edington, and Von Halle, 1960, Genetics 45: 1649-70; 2 = Rahman and Lindsley, 1981, Genetics 99: 49-64; 3 = Yamamoto and Miklos, 1978, Chromosoma 66: 71-98.

^β According to Schalet, *Df(1)bb158/Dp(1;f)18* and *Df(1)bb158/Dp(1;f)122* males and *Df(1)bb158/Df(1)mal³* females, all *bb⁺*, show *eo* phenotype in 50% of the flies, while *Df(1)bb158/Df(1)17-137* females, all *bb*, show *eo* phenotype in more than 50% of the flies.

^γ Also carries *In(1)481=In(1)12E-F;14B*.

Df(1)bb-G: see In(1)sc^{4L}sc^{8R}

Df(1)bi: Deficiency (1) bifid

references: Banga, Bloomquist, Brodberg, Pye, Larrivee, Mason, Boyd, and Pak, 1986, Chromosoma 93: 341-46. Oliver, Perrimon, and Mahowald, 1988, Genetics 120: 159-71.

genetics: Deficient for *bi*.

deficiency	cytology	origin	genetics
<i>Df(1)bi-D1^α</i>	4B3-4;4D1-2	X ray	<i>mei9⁻-amb⁻</i>
<i>Df(1)bi-D2^β</i>	4B6-C1;4D7-E1	X ray	<i>lac⁻-svb⁻</i>
<i>Df(1)bi-D3^γ</i>	4C5-6;4C7-8	X ray	<i>lac⁻-amb⁻</i>
<i>Df(1)bi-D4</i>	not visible	X ray	<i>bi⁻-amb⁻</i>
<i>Df(1)bi-DL1</i>	4A3-5;4C15-16	Uc mutator	<i>mei9⁻-amb⁻</i>
<i>Df(1)bi-DL2</i>	4B3-4;4C15-D1	Uc mutator	<i>mei9⁻-amb⁻</i>
<i>Df(1)bi-DL3</i>	3C7-12;4E1-2	Uc mutator	<i>bi⁻-rg⁻</i>
<i>Df(1)bi-DL4</i>	4B1-2;4C15-D1	Uc mutator	<i>mei9⁻-amb⁻</i>
<i>Df(1)bi-DL5</i>	3C7-12;4E1-2	Uc mutator	<i>bi⁻-svb⁻</i>
<i>Df(1)bi-DL6</i>	4B1-2;4C15-16	Uc mutator	<i>mei9⁻-amb⁻</i>
<i>Df(1)bi-DL7</i>	4A3-5;4C15-D1	Uc mutator	<i>mei9⁻-amb⁻</i>
<i>Df(1)bi-RC40</i>	see <i>Df(1)RC40</i>		

^α Includes *mei-9*.

^β Includes *norPA*.

^γ Includes *amb*.

Df(1)br26

cytology: *Df(1)1D-E;2B5*.

origin: Induced by DEB.

references: Kiss, Beaton, Tardiff, Fristrom, and Fristrom, 1988, Genetics 118: 247-59.

genetics: Deficient for *br*.

Df(1)br-rv1

cytology: *Df(1)2B3;2B4*.

origin: X ray induced.

references: Belyaeva, Protopopov, Baricheva, Semeshin, Izquierdo, and Zhimulev, 1987, Chromosoma 95: 295-

310.

genetics: Deficient for *sta* and *br*.

Df(1)C1-Df(1)C10

origin: X ray induced in *In(1)w^{m51b}*.

genetics: Deficient for *su(f)*.

deficiency	cytology	ref ^α
<i>Df(1)C1^β</i>	19E2-3;19E2-3	1, 2, 3
<i>Df(1)C2</i>	20E-F + T(1;2)3C2;34A-B	2
<i>Df(1)C3^γ</i>	20A;20A	2, 3
<i>Df(1)C4</i>	20A;20A	1, 2, 3
<i>Df(1)C5</i>	19E4-8;19E4-8	2
<i>Df(1)C6</i>	20A;20A	2
<i>Df(1)C7</i>	20A;20A	2
<i>Df(1)C10</i>	20A;20A	2

^α 1 = Appels and Hilliker, 1982, Genet. Res. 39: 149-56; 2 = Hilliker and Appels, 1982, Chromosoma 86: 469-90; 3 = Pimpinelli, Sul-livan, Prout, and Sandler, 1985, Genetics 109: 701-24.

^β Deletes *ABO* and *DAL* (Pimpinelli *et al.*, 1985).

^γ Deletes *ABO* but not *DAL* (Pimpinelli *et al.*, 1985).

***Df(1)C15-Df(1)C246**

origin: X ray induced.

discoverer: Lefevre.

deficiency	cytology	ref ^α	genetics
<i>*Df(1)C15</i>	1B14-C1;2A3	4, 6	<i>mu⁻-l(1)2Ad⁻</i>
<i>Df(1)C52^β</i>	8E;9C-D	1, 2, 4, 6, 10, 12	<i>mus(1)109⁻ftw⁺</i>
<i>Df(1)C60</i>	1A4;1A6	6, 11	<i>l(1)1Ac⁻</i> ; cell viable in abdomen
<i>Df(1)C74</i>	19F1;20A4	8, 9	<i>lfl⁻-intro⁻</i>
<i>Df(1)C128^γ</i>	7D1;7D5-6	4-7, 11	<i>sn⁻-mys⁻</i>
<i>Df(1)C149</i>	5A8-9;5C5-6	1, 2, 4, 6, 12	<i>cv⁻-mus(1)105⁻ rux⁺</i>
<i>Df(1)C159</i>	3F1;4F14	6	
<i>Df(1)C237</i>	19F;20	6	<i>lfl⁻-su(f)⁻</i>
<i>Df(1)C246^{δε}</i>	11D-E;12A1-2	3, 4, 6, 12	<i>wy⁻-s⁻</i> slight M

^α 1 = Baker and Smith, 1979, Genetics 92: 833-47; 2 = Baker, Smith, and Gatti, 1982, Proc. Nat. Acad. Sci. USA 79: 1205-09; 3 = Brown and Voelker, 1980, Biochem. Genet. 18: 303-09; 4 = Craymer and Roy, 1980, DIS 55: 200-04; 5 = Deak, Bellamy, Bienz, Dubuis, Fenner, Gollin, Rähmi, Ramp, Reinhardt, and Cotton, 1982, J. Embryol. Exp. Morphol. 69: 61-81; 6 = Lefevre; 7 = Lefevre and Johnson, 1973, Genetics 74: 633-45; 8 = Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-52. 9 = Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31. 10 = Postlethwait and Shirik, 1981, Amer. Zool. 21: 687-700; 11 = Ripoll and García-Bellido, 1979, Genetics 91: 443-53; 12 = Wieschaus, Nüsslein-Volhard and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307.

^β Mutant wing position of *fliiF* and *flw* uncovered in deficiency heterozygotes.

^γ Synonym: *Df(1)sn^{C128}*. High frequency of homeotic transformations found in *Df(3)red/+* progeny of *Df(1)C128/+* females (Gans, Forquignon, and Masson, 1980, Genetics 96: 887-902). Lethal in germ-line clones (García-Bellido and Robbins, 1983, Genetics 103: 235-47).

^δ Seems to include locus involved in early step of sex determination (Scott and Baker, 1986, Genetics 113: s35).

^ε *YXS.YL, Df(1)C246, In(1)EN, y M/YXS.YL, Df(1)g^l In(1)EN, y g f B* females are viable, yellow, and minute (Schalet).

Df(1)c99-w^{m4}

cytology: *Df(1)2E1-2;3C1-2*.

discoverer: Lefevre.

references: Perrimon, Engstrom, and Mahowald, 1985, Genetics 111: 23-41.

***Df(1)C-PL: Deficiency (1) C of Peterson and Laughnan**

cytology: *Df(1)15F;16E*.
origin: Spontaneous; allegedly an asymmetrical exchange.
discoverer: Peterson and Laughnan.
references: 1963, Proc. Nat. Acad. Sci. USA 50: 126-33.
genetics: Deficient for *f* and *B* but not *os*. Male lethal.

Df(1)cho: Deficiency (1) chocolate

reference: Steinmann-Zwicky, 1988, EMBO J. 7: 3889-98.

deficiency	cytology	genetics
<i>Df(1)cho2</i>	3E;4A	<i>mdl⁻ bi⁺</i>
<i>Df(1)cho3</i>	3C;3F	<i>cho⁻ mdl⁺</i>
<i>Df(1)cho5</i>	3D;4A	<i>cho⁻ mdl⁺</i>
<i>Df(1)cho6</i>	1F;4A	<i>mdl⁻ bi⁺</i>
<i>Df(1)cho7</i>	2E-F;4B	<i>mdl⁻ bi⁺</i>
<i>Df(1)cho8</i>	3C;4A	<i>mdl⁻ bi⁺</i>
<i>Df(1)cho10</i>		<i>cho⁻ mdl⁺</i>
<i>Df(1)cho19</i>	3F;4F	<i>ec⁻-fs(1)A1621⁻</i>
<i>Df(1)cho23</i>	3E-F;4F	<i>fl(1)302⁻ ovo⁺</i>
<i>Df(1)cho24</i>		<i>mdl⁻ bi⁺</i>
<i>Df(1)cho25</i>		<i>mdl⁻ bi⁺</i>

Df(1)cin-arth: Deficiency (1) cinnamon-arthritis

references: Kramers, Schalet, Paradi, and Huiser-Hooteyling, 1983, Mutat. Res. 107: 187-201.
genetics: Deficient for *l(1)IA* through *sc*.

Df(1)Cl^Lbb^{DfR}: see *In(1)Cl^Lbb^{DfR}*

Df(1)Cl^Ly^{4R}: see *In(1)Cl^Ly^{4R}*

Df(1)cm: Deficiency (1) carmine

origin: X ray induced.
genetics: Deficient for *cm*.

deficiency	cytology	discoverer	ref ^α
<i>*Df(1)cm-D5</i>	6E5-6;6F2-3	DeFrank	1
<i>*Df(1)cm-H2</i>	6D8-E1;6E6-F1	Hannah	1
<i>*Df(1)cm-H4</i>	6D8-E1;6E6-F1	Hannah	1
<i>Df(1)cm-R8</i>	6E;6E		2

^α 1 = CP627; 2 = Valencia, 1966, DIS 41: 58.

Df(1)cm-In: see *In(1)cm-df*

Df(1)ct2a2-Df(1)ct15b1

origin: X ray induced.
references: Hannah, 1949, Proc. Int. Congr. Genet. 8th, pp. 588-89.
genetics: *cm⁺ ct⁻ sn⁺*.

deficiency	cytology
<i>*Df(1)ct2a2</i>	7B3-6;7B6-7
<i>*Df(1)ct2a3</i>	7B2-3;7C1-2
<i>Df(1)ct4b1^α</i>	7B2-4;7C3-4
<i>*Df(1)ct7a2</i>	7A5-B1;7C4-9
<i>*Df(1)ct7c2</i>	6F11-7A1;7B8-C1
<i>*Df(1)ct10a1</i>	7B3-4;7B6-7
<i>*Df(1)ct10b1^β</i>	6D8-E1;7B7-C1
<i>*Df(1)ct12c2</i>	7B2-3;7B6-7
<i>*Df(1)ct14b1</i>	7B2-3;7C3-4
<i>*Df(1)ct14c1</i>	7B3-4;7B6-9
<i>*Df(1)ct15b1</i>	7B2-4;7B6-7

^α Covered by *Dp(1;3)sn^{13a1}* (Lefevre and Johnson, 1973, Genetics 74: 633-45). Breaks within the *dec-1* locus (Hawley and Waring, 1988, Genes Dev. 2: 341-49). Proximal breakpoint lies between coordinates 3.6 and 4.0 on the molecular map of *dec-1*.

^β *Df(1)ct10b1* should have been deficient for *cm*, but 6E-F may have been transposed and *cm⁺* thus retained.

Df(1)ct78

cytology: *Df(1)6F1-2;7C1-2*.
origin: Derived from unstable X chromosome.
references: Lim, 1979, Genetics 93: 681-701.
 Raymond, Laverty, and Simmons, 1986, DIS 63: 111-14.
genetics: Deficient for *ct*.

***Df(1)ct268-13:** see *In(1)ct²⁶⁸⁻¹³*

***Df(1)ct268-18:** see *In(1)ct²⁶⁸⁻¹⁸*

***Df(1)ct268-20:** see *In(1)ct²⁶⁸⁻²⁰*

***Df(1)ct268-30**

cytology: *Df(1)7B2-3;7C3-4*.
origin: X ray induced.
discoverer: Hoover, 38d.
references: CP627.
genetics: Deficient for *ct* but not *scp* or *sn*.

***Df(1)ct268-37:** see *Tp(1;3)ct²⁶⁸⁻³⁷*

Df(1)ct268-42

cytology: *Df(1)7A5-6;7B8-C1* (Sutton).
origin: X ray induced.
discoverer: Demerec, 40a.
references: CP627.
 Homyk and Pye, 1989, J. Neurogenet. 5: 37-48.
genetics: Deficient for *ct* to *rex* but not *cm*, *scp*, or *sn*.

Df(1)ct-J4

cytology: *Df(1)7A2-3;7C1*.
origin: X ray induced in *In(1)dl-49*.
discoverer: T.K. Johnson.
references: Lefevre and Johnson, 1973, Genetics 74: 633-45.
 Johnson and Judd, 1979, Genetics 92: 485-502.
 Craymer and Roy, 1980, DIS 55: 200-04.
 Homyk and Pye, 1989, J. Neurogenet. 5: 37-48.
genetics: Deficient for *ct* and *rex* but not *shf*. Lethal male embryos show incomplete condensation of the ventral nerve cord [Campos-Ortega and Jimenez, 1979, Development and Neurobiology of *Drosophila* (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York and London, pp. 201-22]. Heterozygotes with wild-type or balancer X chromosomes show thoracic protuberances [100% penetrance and variable expression (Voelker and Wisely, 1982, DIS 58: 150-51)].

Df(1)ct-J6

cytology: *Df(1)6E1;7C1*.
origin: X ray induced.
discoverer: T.K. Johnson.
references: Lefevre and Johnson, 1973, Genetics 74: 633-45.
 Craymer and Roy, 1980, DIS 55: 200-04.
 Homyk and Pye, 1989, J. Neurogenet. 5: 37-48.
genetics: Deficient for *ct* but not *rex*. Heterozygotes show thoracic protuberances as in *Df(1)ct-J4*.

Df(1)(D): see *Df(1)r-D1*

Df(1)D7

cytology: *Df(1)14C7-D1;14E3-F1*.
synonym: *Df(1)l-D7*.
references: Ganetzky, 1984, *Genetics* 108: 897-911.
genetics: Uncovers *para*.

Df(1)D17: see *Df(1)r-D17*

Df(1)D34

cytology: *Df(1)14F1-2;14F6* (Lefevre).
origin: X ray induced.
discoverer: M.M. Green.
synonym: *Df(1)l-D34*.
references: Jarry, 1979, *Mol. Gen. Genet.* 172: 199-202.
 Ganetzky, 1984, *Genetics* 108: 897-911.
genetics: Uncovers several lethal complementation groups including *para* but does not include *r*.

Df(1)D43L1

cytology: *Df(1)19E8;20E-F*.
origin: *Cs*¹³⁷ induced.
discoverer: Himoe.
references: Schalet and Finnerty, 1968, *DIS* 43: 128-29.
 Schalet and Lefevre, 1973, *Chromosoma* 44: 183-200.
 Rahman and Lindsley, 1981, *Genetics* 99: 49-64.
genetics: Deficient for *unc-su(f)*.

Df(1)DA622

cytology: *Df(1)10B8;10D2*.
discoverer: Lefevre.
genetics: Deficient for *dlg*.

Df(1)DCB1-35b

cytology: *Df(1)19F1-2;20E-F*.
origin: Induced by tritiated deoxycytidine.
discoverer: Kaplan.
references: Schalet and Finnerty, 1968, *DIS* 43: 128-29.
 Schalet and Lefevre, 1973, *Chromosoma* 44: 183-200.
 Craymer and Roy, 1980, *DIS* 55: 200-04.
genetics: Deficient for *lfl-su(f)* but not *run* (Gergen and Wieschaus, 1986, *Cell* 45: 289-99).

***Df(1)DCB1-35c**

cytology: Cytologically normal.
origin: Induced by tritiated deoxycytidine.
discoverer: Kaplan.
references: Schalet and Finnerty, 1968, *DIS* 43: 128-29.
 Schalet and Lefevre, 1973, *Chromosoma* 44: 183-200.
genetics: Deficient for *wap* and *intro*.

Df(1)Del lz A: see **In(1)lzA*

Df(1)Del271b: see **T(1;2)271b*

Df(1)dm75e19: Deficiency (1) diminutive

cytology: *Df(1)3C11;3E4* superimposed on *In(1)3E;5E*.
origin: X ray induced.
discoverer: Lefevre.
synonym: *Df(1)HA44*.
references: Craymer and Roy, 1980, *DIS* 55: 200-04.
 McGinnis, Farrell, and Beckendorf, 1980, *Proc. Nat. Acad. Sci. USA* 77: 7367-71.
 Byers, Davis, and Kiger, 1981, *Nature* 289: 79-81.
 Davis and Davidson, 1984, *Mol. Cell. Biol.* 4: 358-67.
genetics: Deficient for *dnc-slc* but not *Sgs-4* (McGinnis *et al.*, 1980). Cell viable in abdominal cuticle (Ripoll and García-Bellido, 1979, *Genetics* 91: 443-53). Slight *dm* phenotype in heterozygotes with *Ubl* (Mortin and

Lefevre, 1981, *Chromosoma* 82: 237-47). Germ-line clones lethal (García-Bellido and Robbins, 1983, *Genetics* 103: 235-47).

genetics: Deficient for *dnc - slc*.

molecular biology: Left breakpoint located on molecular map (McGinnis *et al.*, 1980; Davis and Davidson, 1984).

Df(1)dm77h

cytology: *Df(1)3D5;3F8-9*.
origin: X ray induced.
discoverer: Lefevre.
references: Davis and Davidson, 1984, *Mol. Cell. Biol.* 4: 358-67.
genetics: Deficient for *dm* but not *dnc*.

Df(1)dor1T: Deficiency (1) deep orange

cytology: *Df(1)1E3-4;2B17-18*.
origin: γ ray induced.
discoverer: Gorelova.
references: Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, *DIS* 58: 184-90.
genetics: Deficient for *dor*.

Df(1)dor2T

cytology: *Df(1)2B6;2E1-2*.
origin: γ ray induced.
discoverer: Gorelova.
references: Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, *DIS* 58: 184-90.
genetics: Deficient for *dor*.

Df(1)dor-A94: see *Df(1)A94*

Df(1)DV4: see **T(1;4)w^{mdV4}**

Df(1)E128

cytology: *Df(1)17C;18A*.
origin: Induced by hybrid dysgenesis.
references: Engels and Preston, 1984, *Genetics* 107: 657-78.
genetics: Deficient for *Bx* and *hdp - b*.

Df(1)E150

cytology: *Df(1)14B3-4;14E*.
references: Kulkarni, Steinlauf, and Hall, 1988, *Genetics* 118: 267-85.
genetics: Deficient for *diss*.

Df(1)E160.1

cytology: *Df(1)17A;18A2*.
origin: Induced by hybrid dysgenesis.
references: Engels and Preston, 1984, *Genetics* 107: 657-78.
genetics: Deficient for *Bx* and *hdp - b*.

Df(1)EA113

cytology: *Df(1)20A;20E*.
origin: Induced by ethyl methanesulfonate.
references: Perrimon, Smouse, and Miklos, 1989, *Genetics* 121: 313-31.
genetics: Deficient for *uncl-su(f)*.

Df(1)EH653

cytology: *Df(1)19A2-3;20A-B*.
origin: Induced by ethyl methanesulfonate.
references: Eberl and Hilliker, 1988, *Genetics* 118: 109-

20.

genetics: Deficient for *run* and *fog*, but not for *bb*.

Df(1)f: Deficiency (1) forked

origin: X ray induced.

synonym: *Df(1)f²⁵⁷*.

deficiency	cytology	discoverer	ref ^α	genetics
<i>Df(1)f257-5</i>	15E7-F1;15F2-4	Demerec, 33k	1	<i>M⁺f⁻os⁺</i>
* <i>Df(1)f257-6</i>	15E4-F1;16A1	Bridges, 1917	1, 2	<i>M⁺f⁻B⁻os⁺</i>
* <i>Df(1)f257-9</i>	15E7-F1;16D2-4 ^β	Demerec, 34c	1, 2	<i>M⁺f⁻B⁻lh⁺</i>
* <i>Df(1)f257-27</i>	14F6-15A1;15F5-6 ^β	Demerec, 381	1	<i>M(1)15D⁻f⁻</i>
* <i>Df(1)f257-28</i>	15E7-F1;16E5-F1 ^β	Sutton, 40h	1	<i>f⁻B⁻</i>
* <i>Df(1)f257-31</i>	15E7-F1;15F5-6	Bishop, 41a	1	<i>M⁺f⁻os⁺</i>

^α 1 = CP627; 2 = Sutton, 1943, Genetics 28: 97-107.
^β Superimposed on *Dp(1;1)B = Dp(1;1)15F9-16A1;16A7-B1*.

Df(1)fa-swb: Deficiency (1) facet-strawberry

cytology: Interband deletion between 3C5-6 and 3C7 such that 3C5-6 ("fused" to 3C7) appears thick, while 3C7 appears to be missing.

origin: X ray induced.

discoverer: Lefevre and Kelley.

synonym: *swb^{71b}* (Lefevre and Kelley, 1972, DIS 48: 146-47); *fa^{swb71b}* (Ashburner).

references: Welshons and Keppy, 1975, Genetics 80: 143-55.

Keppy and Welshons, 1977, Genetics 85: 497-506.
 1980, Chromosoma 76: 191-200.

genetics: Eye color strawberrylike (but more mottled) and eye surface rougher than in *fa^{swb62b}* (= *swb^{62b}* of Ives, 1969, DIS 44: 46).

Df(1)FM7

cytology: *Df(1)1A;1B2-3*.

origin: X ray induced.

discoverer: Gyurkovics and Voelker.

references: Voelker, Greenleaf, Gyurkovics, Wisely, Huang and Searles, 1984, Genetics 107: 279-84.

genetics: Deficient for genes from X tip through *ac*.

Df(1)fu: Deficiency (1) fused

genetics: Deficient for *fu*.

deficiency	cytology	origin	ref ^α	lethal stage	Rescue by <i>Dp</i> ^β
<i>Df(1)fu3</i>	17C3-7;17D1-2	DEB	1	larva	+
<i>Df(1)fu5</i>	17B;17E-F	DEB	1		-
<i>Df(1)fu13</i>	17C3-7;17D1-2	X ray	2		+
<i>Df(1)fu15</i>	17C;17D+ <i>In(1)12F;17F</i>	DEB	1	embryo	-
<i>Df(1)fu-A7</i>	17C;18A	DEB	1	embryo	-
<i>Df(1)fu-B10</i>	17C;18A-B	DEB	1		-
<i>Df(1)fu-BX14</i>	17A;17D1-2	X ray	1	embryo	-
<i>Df(1)fu-C3^γ</i>	17A;18B				
<i>Df(1)fu-E5</i>	17C;17E-F	DEB	1		-
<i>Df(1)fu-H4</i>	17C3-7;17D1-2	DEB	1	larva	+
<i>Df(1)fu-I3</i>	17A;18B	DEB	1		+
<i>Df(1)fu-PI</i>	17C3-7;17D1-2	HD	2	larva	+
<i>Df(1)fu-S4</i>	17C;17E-F	DEB	1		-
<i>Df(1)fu-Z4</i>	{17C4-6;17C4-6}	DEB	1	larva	+

^α 1 = Busson, Limbourg-Bouchon, Mariol, Preat, and Lamour-Isnard, 1988, Roux's Arch. Dev. Biol. 197: 221-30. 2 = Holmgren (unpublished).
^β *Dp(1;3)fu⁺* 10.
^γ See Ashburner for further information.

Df(1)fw-HF368: see *Df(1)HF368*

Df(1)g70e

cytology: *Df(1)17A3-4;17C6-7*.

origin: Induced by mutator gene *mu*.

references: Green and Lefevre, 1972, Mutat. Res. 16: 59-64.

Df(1)g-HA92: see *Df(1)HA92*

Df(1)g: Deficiency (1) garnet

cytology: *Df(1)12A;12E* (Nicoletti).

origin: Spontaneous.

discoverer: L. V. Morgan, 24124.

references: Mason, Green, Shaw, and Boyd, 1981, Mutat. Res. 81: 329-43.

Waring, DiOrio, and Hennen, 1983, Dev. Biol. 100: 452-63.

genetics: Deficient for *g*, *l(1)dd4*, *ty* and *ben* but not *wy*, *s*, *na*, *pl*, or *sd*. Uncovers *mus(1)101^{D1}* (Mason *et al.*, 1981). Lethal in male and cell lethal.

Df(1)g2

cytology: *Df(1)12A;12C*.

origin: X ray induced in *In(1)sc²⁹*.

discoverer: Schalet.

genetics: Deficient for *mus101*, *g*, *l(1)dd4*, *ty*, *rdgB*, but not *ben*, or *na*. Covered by *Dp(1;5)LJ9*. Complements *Df(1)C246 = Df(1)11D;12A1-2*.

Df(1)γ2: Deficiency (1) gamma

cytology: *Df(1)7D14;8A3*.

references: Wakimoto and Spradling, 1981-82, Year Book, Carnegie Inst. Washington 81: 190-91.

Homyk and Pye, 1989, J. Neurogenet. 5: 37-48.

genetics: *sesD⁺*.

Df(1)γa3

cytology: *Df(1)7D4;8A3*.

references: Wakimoto and Spradling, 1981-82, Year Book - Carnegie Inst. Washington 81: 190-91.

Df(1)GA

origin: X ray induced.

discoverer: Lefevre.

deficiency	cytology	ref ^α	genetics
<i>Df(1)GA22</i>	20;20	5	<i>wap⁻ - su(f)⁻</i>
<i>Df(1)GA33</i>	19F;20	2,5	<i>unc⁻ - uncl⁻</i>
<i>Df(1)GA34</i>	7A4-5;7B3-4	2,4	<i>l(1)7Ab⁻ - l(1)7Bb⁻</i>
<i>Df(1)GA37</i>	19E;19F	2,4,5	<i>run⁻ - l(1)19Fg⁻</i>
<i>Df(1)GA40</i>	19E;20F	2,5	<i>run⁻ - su(f)⁻</i>
<i>Df(1)GA42^β</i>	20B1;20F	6	<i>fog⁻ - bb⁻</i>
<i>Df(1)GA56</i>	4C5-6;4D1	1	<i>omb⁻ - hnt⁻ svb⁺</i>
<i>Df(1)GA90</i>	20C;20F	5,6	<i>l(1)20Cb⁻ - bb⁻</i>
<i>Df(1)GA102</i>	3D4-5;3F7-8	9	<i>slc⁻, cho⁻ ec⁺</i>
<i>Df(1)GA104</i>	19F;19F	5	<i>fli⁻ - l(1)19Fg⁻</i>
<i>Df(1)GA112^{γδ}</i>	10A11-B1;10C2	8,9	<i>l(1)10Ag⁻ - l(1)10Cd⁻</i>
<i>Df(1)GA113</i>	3D6-E1;?	2	<i>slc⁻</i>
<i>Df(1)GA120</i>		2	
<i>Df(1)GA131</i>	20;20	5	<i>sph⁻ - su(f)⁻</i>

^α 1 = Lawlor, 1980, DIS 55: 81-82; 2 = Lefevre; 3 = Miklos, Kelly, Combe, Leeds, and Lefevre, 1987, J. Neurogenet. 4: 1-19; 4 = Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-52; 5 = Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31; 6 = Rahman and Lindsley, 1980, DIS 45: 123-24; 7 = Rahman and Lindsley, 1981, Genetics 99: 49-64; 8 = Voelker, Wisely, Huang, and Gyurkovics, 1985, Mol. Gen. Genet. 201: 437-45; 9 = Wieschaus, Nüsslein-Volhard and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307.

^β Associated with a *T(1;2)*.

- γ *Df(1)HA85/Df(1)GA112* and *Df(1)m²⁵⁹⁻⁴/Df(1)GA112* females survive to adulthood and show no mutant phenotype (Voelker *et al.*, 1985).
- δ Right DNA breakpoint 16.5 kb to the left of the origin of the *Rp112/15* walk of Biggs, Searles, and Greenleaf, 1985, Cell 2: 611-21 ("+" values to the right and "-" values to the left).

Df(1)GAM201

cytology: *Df(1)3A2-3;3C2-3.*
origin: X ray induced.
discoverer: Lefevre.
references: Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-52.
genetics: Deficient for *z - w*.

Df(1)GE202

cytology: *Df(1)7D12-13;7E3-4.*
origin: X ray induced.
discoverer: Lefevre.
genetics: Deficient for *l(1)7Dj - l(1)7Ea*.

Df(1)GE261

cytology: *Df(1)1A1;1B10-11.*
origin: X ray induced.
discoverer: Lefevre.
genetics: Deficient for *l(1)1Aa - vnd*.

Df(1)GE263

cytology: *Df(1)19F1-2;20A1-2.*
origin: X ray induced.
references: Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31.
genetics: Deficient for *ftil - eo*.

Df(1)h70e

cytology: *Df(1)1A1;1B6.*
origin: Induced by mutator gene *mu*.
references: Green and Lefevre, 1972, Mutat. Res. 16: 59-64.

Df(1)HA11 - Df(1)HF396

origin: X ray induced.
discoverer: Lefevre.

deficiency	cytology	ref ^α	genetics
<i>Df(1)HA11</i>	7D13-14;7D22	3, 4, 6	<i>l(1)adl⁻ - lon⁻ ou⁺</i>
<i>Df(1)HA18</i>	not visible	6	<i>l(1)2Ab⁻ - sta⁻</i>
<i>Df(1)HA32</i>	6E4-5;7A6	1, 6	<i>shf⁺ cm⁻ scp⁺</i>
<i>Df(1)HA44</i>	see <i>Df(1)dm75e19</i>		
<i>Df(1)HA52</i>	12E5;13A4		
<i>Df(1)HA85</i>	10C1-2;11A1-2	1	<i>tyl⁺ dy⁻</i>
<i>Df(1)HA92</i> ^β	12A6-7;12D3	1, 6, 7, 8	<i>s⁺ g⁻ ty⁻ l(1)dd4⁻</i> <i>rdgB⁻ ben⁻ na⁺</i>
<i>Df(1)HA326</i>	includes 19F	11	<i>seg⁻</i>
<i>Df(1)HC133</i> ^γ	9B9-10;9E-F	1, 6, 12	<i>ftw⁻ ftik⁻ sbr⁺</i>
<i>Df(1)HC163</i> ^δ	4A1-2;4D1-2	1, 5	<i>mei-9⁻ - rb⁻</i>
<i>Df(1)HC163B</i> ^ε	4B1-2;4F1-2	5	<i>mei-9⁻ - rb⁻</i>
<i>Df(1)HC194</i>	3A1;3C3-4	6, 8a	<i>gi⁻ - w⁻ rst⁺</i>
<i>Df(1)HC244</i>	3E8;4F11-12	1, 5, 6	<i>ec⁻ - rg⁻ cx⁺</i>
<i>Df(1)HC279</i>		6	<i>shakB⁻ - vao⁻</i> <i>sta⁻ - dor⁻</i>
<i>Df(1)HE333</i>	not visible		
<i>Df(1)HF334</i>	19C3;20E-F	2	<i>mal⁻ - su(f)⁻ bb⁺</i>
<i>*Df(1)HF349</i>	13B5;13E1	1, 6	
<i>Df(1)HF359</i>	{20A-B;20E}	6	<i>l(1)20Bb⁻ - su(f)⁻</i>
<i>Df(1)HF366</i>	3E7-8;5A7	1, 6, 10	<i>ec⁻ - rg⁻ bo⁺</i>
<i>Df(1)HF368</i> ^ζ	11A2;11B9	1, 8, 9	<i>fw⁻ - Lsp1α⁻</i>
<i>Df(1)HF394</i>	13B5;13E1	1, 8	<i>sd⁻</i>
<i>Df(1)HF396</i>	18E1-2;20	1, 6	<i>sw⁻ - su(f)⁻</i>
<i>Df(1)HF406</i>	3C4-5;?		<i>rst⁻</i>

^α 1 = Craymer and Roy, 1980, DIS 55: 200-04; 2 = Durica and Krider, 1978, Genetics 89: 37-64; 3 = Homyk and Emerson, 1985,

Genetics 110: s92; 4 = King and Riley, 1982, Dev. Genet. 3: 69-89; 5 = Lawlor, 1980, DIS 80: 81-82; 6 = Lefevre; 7 = Mason, Green, Shaw, and Boyd, 1981, Mutat. Res. 81: 329-43; 8 = Mortin and Lefevre, 1981, Chromosoma 82: 237-47; 8a = Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-52; 9 = Roberts and Evans-Roberts, 1979, Nature 280: 691-92; 10 = Sodja, Rizki, Rizki and Zafar, 1982, Chromosoma 86: 293-98; 11 = Wieschaus, Audit, and Masson, 1981, Dev. Biol. 88: 92-103. 12 = Zhimulev, Belyaeva, Pokholkova, Kochneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eakin, 1982, DIS 58: 210-14;
^β Synonym: *Df(1)g^{HA92}*. Deficiency uncovers *mus(1)101* (Mason *et al.*, 1981).
^γ Listed as *Df(1)HC163* by Craymer and Roy, 1980.
^δ Deficiency lost according to Craymer and Roy, 1980.
^ε Lethal segregant from *Df(1)HC163* or *Df(1)RC40* (Lawlor, 1980).
^ζ Synonym: *Df(1)fw^{HF368}*.

Df(1)Hk. Deficiency (1) Hyperkinetic

cytology: *Df(1)9A;9C.*
origin: X ray induced.
discoverer: Schlimgen and Kreber.
synonym: *Df(1)Hk^{ΔR2}*.
references: Stern and Ganetsky, 1989, J. Neurogenet. 5: 215-28.
genetics: Deficient for *Hk*.

Df(1)HM: Deficiency (1) Hycanthon Methanesulfonate

origin: Induced by hycanthon methanesulfonate.

deficiency	cytology	ref ^α	genetics
<i>Df(1)HM3</i>	3A;3B-C	2	<i>l(1)3Ac⁻ - l(1)3Bf⁻</i>
<i>Df(1)HM5</i>	20A-B;20?	2	<i>fog⁻ - ?</i>
<i>Df(1)HM8</i>	20A-B;20?	2	<i>fog⁻ - ?</i>
<i>Df(1)HM15</i>		1	<i>ras⁻</i>
<i>Df(1)HM44</i>	19B;19F5-6	2,3	<i>sw⁻ - l(1)19Ff⁻</i>
<i>Df(1)HM45</i>	19D-E;20?	2	<i>run⁻ - ?</i>
<i>Df(1)HM47</i>	20A-B;20?	2	<i>fog⁻ - ?</i>
<i>Df(1)HM49</i>	19E-F;20A	2	<i>lft⁻ - wap⁻</i>
<i>Df(1)HM50</i>	19E-F;20A	2	<i>lft⁻ - wap⁻</i>
<i>Df(1)HM406</i>	3A;3C	2	<i>l(1)3Ad⁻ - rst⁻</i>
<i>Df(1)HM407</i>	20B;20?	2	<i>stn⁻ - ?</i>
<i>Df(1)HM418</i>	20A;20?	2	<i>wap⁻ - ?</i>
<i>Df(1)HM422</i>	20A;20?	2	<i>wap⁻ - ?</i>
<i>Df(1)HM428</i>	1A;1A-B	2	<i>l(1)1Ad⁻ - ewg⁻</i>
<i>Df(1)HM430</i>	20A-B;20C-D	2	<i>wap⁻ - l(1)20Cb⁻</i>
<i>Df(1)HM444</i>	20A-B;20?	2	<i>fog⁻ - ?</i>
<i>Df(1)HM455</i>	20A;20?	2	<i>wap⁻ - ?</i>
<i>Df(1)HM456</i>	10B;10C-D	2,4	<i>l(1)10A⁻ - l(1)10Ce⁻</i>
<i>Df(1)HM458</i>	2F-3A;3A	2	<i>l(1)3Ad⁻ - ?</i>
<i>Df(1)HM459</i>		1	<i>ct⁻ - g</i>
<i>Df(1)HM460</i>	3A	2	<i>mit(1)15⁻ - ?</i>

^α 1 = Ashburner; 2 = Kramers, Schalet, Paradi, and Huiser-Hoogteyling, 1983, Mutat. Res. 107: 187-201; 3 = Miklos, Kramers, and Schalet, 1986, DIS 63: 96-97; 4 = Voelker, Wisely, Huang, and Gyurkovics, 1985, Mol. Gen. Genet. 201: 437-45.

Df(1)j70e

cytology: X proximal.
origin: Induced by mutator gene *mu*.
references: Green and Lefevre, 1972, Mutat. Res. 16: 59-64.

Df(1)J1-259: see *Df(1)259*

Df(1)JA, JC and JF

origin: X ray induced.

discoverer: Lefevre.

deficiency	cytology	ref ^α	genetics
Df(1)JA21	19E5-6;20	4	
*Df(1)JA22	9F13;10A1	4	v ⁻
Df(1)JA26	11A1;11D-E	1,4	fw ⁻ wy ⁺
Df(1)JA27	18A5;18D1-2	1,6	car ⁺ ; fog ⁻ - su(f) ⁻
Df(1)JA52	2C10-D1;2D3-4	4,5	usp ⁺ Actn ⁻ -pha ⁻ Pgd ⁺
Df(1)JA53	3D4-5;3E7		dm ⁻ l(3)3Ee ⁻
Df(1)JA68			
Df(1)JA117	19F;19F	6	l(3)19Ff ⁻ l(3)19Fg ⁻
*Df(1)JA118^β	10F11;11C-D + 19F1;20A1-2	4,7	fw ⁻ tuh-1 ⁻
Df(1)JC4	20A1;20E-F	1,4,7	eo ⁻ - su(f) ⁻
Df(1)JC12		4	eo ⁻ - su(f) ⁻
Df(1)JC19	2F6;3C5	1,3,4,5	pn ⁺ gt ⁻ - rst ⁻
Df(1)JC37	19E2;20A2	4	
Df(1)JC70	4C15-16;5A1-2	1,4	rb ⁺ rg ⁻ cx ⁺
Df(1)JC77	19F;20	6	l(1)19Ff ⁻ - eo ⁻
Df(1)JC102	19E5-7;20A2	4,8	cell lethal in cuticle
Df(1)JC105	2D4-6;2D4-6	4,5	Pgd ⁺ wapl ⁻ - l(1)2Ea ⁻
Df(1)JF4	19E7;20A	4	pcx ⁺
Df(1)JF5^γ	5E5-6;5E6-7	4,9	swa ⁻

^α 1 = Craymer and Roy, 1980, DIS 55: 200-04; 2 = Dura, Randscholt, Deatrick, Erk, Santamaria, Freeman, Freeman, Weddell, and Brock, 1987, Cell 51: 829-39; 3 = Honisch and Campos-Ortega, 1982, DIS 58: 76; 4 = Lefevre; 5 = Perrimon, Engstrom, and Mahowald, 1985, Genetics 111: 23-41; 6 = Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31; 7 = Pyati, 1976, Mol. Gen. Genet. 146: 189-90; 8 = Ripoll and García-Bellido, 1979, Genetics 91: 443-53; 9 = Stephenson and Mahowald, 1987, Dev. Biol. 124: 1-8.

^β Double deficiency.
^γ Induced by diepoxybutane.

Df(1)K5: Deficiency (1) of Kennison

origin: X ray induced.
discoverer: Kennison.
references: Rahman and Lindsley, 1981, Genetics 99: 49-64.
genetics: fog⁺ l(1)20Cb⁻ - bb⁻.

Df(1)K95

cytology: Df(1)3A3-4;3B1-2.
origin: X ray induced.
discoverer: Falk.
references: Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56.
Kaufman, Shannon, Shen, and Judd, 1975, Genetics 79: 265-82.
genetics: Deficient for l(1)3Ac - l(1)3Ba. Cell lethal in cuticle and in germ-line clones (Ripoll and García-Bellido, 1979, Genetics 91: 443-53). Male lethal in embryo.
molecular biology: Associated with 30kb inversion to right of the deletion (Reddy, Zehring, Wheeler, Pirrotta, Hadfield, Hall, and Rosbach, 1984, Cell 38: 701-10). Left breakpoint localized to an approximately 4 kilobase restriction fragment some 70 kb to the right of the origin of a 160 kb walk (Gunaratne, Mansukhani, Lipari, Liou, Martindale, and Goldberg, 1986, Proc. Nat. Acad. Sci. USA 83: 701-05).

Df(1)KA1-Df(1)KC10

origin: X ray induced.
discoverer: Lefevre.

deficiency	cytology	ref ^α	genetics
Df(1)KA1	19E5;20E-F	4,5,8	
Df(1)KA6	10E1;11A7-8	2,5,8	m ⁻ -fw ⁻ rrv ⁺

deficiency	cytology	ref ^α	genetics
Df(1)KA7^β	10A9;10F6-7	2,5,6,8,9	l(1)10Ag ⁺ dsh ⁻ -fid ⁻ tsg ⁺
Df(1)KA9^γ	12E1;13A5	1,2,4,8	ty ⁺ na ⁻ ; incomplete condensation and splitting of ventral nerve cord in hemizygous embryos
Df(1)KA10^δ	11A1;11A7-8	1,2,8	fw ⁻ -tsg ⁻ ; duplicated ventral nerve cord in hemizygous embryos; cell lethal
Df(1)KA14^δ	7F1-2;8C6	2,3,5,8	lon ⁺ om ⁻ -ptg ⁻ ; includes oc
Df(1)KC10	20C;20D	7,8	l(1)20Ca ⁻ - l(1)20Cb ⁻ ; cell viable in cuticle

^α 1 = Campos-Ortega and Jiménez, 1980, Development and Neurobiology of Drosophila (Siddiqi, Babu, Hall, and Hall, eds.), Plenum Press, New York and London, pp. 201-22; 2 = Craymer and Roy, 1980, DIS 55: 200-04; 3 = King and Riley, 1982, Dev. Genet. (Amsterdam) 3: 69-89; 4 = Lefevre; 5 = Kreber and Ganetzky; 6 = Mortin and Lefevre, 1981, Chromosoma 82: 237-47; 7 = Ripoll and García-Bellido, 1979, Genetics 91: 443-53; 8 = Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307. 9 = Zhimulev, Belyaeva, Pokholkova, Kochneva, Fomina, Bgatov, Khyudyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1982, DIS 58: 210-14.
^β Synonym: Df(1)m-KA7.
^γ Also includes rut (Livingston, Sziber, and Quinn, 1984, Cell 37: 205-15).
^δ Germ-line clones lethal (García-Bellido and Robbins, 1983, Genetics 103: 235-47).

Df(1)L10

cytology: Df(1)1A1;1A6-7.
origin: X ray induced.
discoverer: Lefevre.
genetics: Deficient for l(1)1Aa - l(1)1Ac.

Df(1)I24

references: Perrimon, Engstrom, and Mahowald, 1984, Genetics 108: 559-72.
1985, Genetics 111: 23-41.
genetics: Deficient for pn through l(1)2Ea.

Df(1)I32

cytology: Df(1)14B17-C1;15A2-3.
references: Kulkarni, Steinlauf, and Hall, 1988, Genetics 118: 267-85.
genetics: Deficient for diss.

Df(1)L271

origin: Induced by hybrid dysgenesis.
discoverer: Sobels.
references: Perrimon, Engstrom, and Mahowald, 1984, Genetics 108: 559-72.
1985, Genetics 111: 23-41.
genetics: Deficient for fs(1)k10-kz.

Df(1)l-D7: see Df(1)D7

Df(1)l-D34: see Df(1)D34

Df(1)l(1)403: see Df(1)403

Df(1)LB6

cytology: Df(1)19E4;20A2-3.
origin: Induced by mitomycin-C.
discoverer: Baldwin.
references: Schalet and Lefevre, 1973, Chromosoma 73: 83-100.
genetics: Deficient for shakB and -eo but not run.

Df(1)LB7

cytology: *Df(1)19E4*?
origin: Induced by mitomycin-C.
references: Baird, Schalet, and Wyman, 1990, Genetics 126: 1045-59.
 Schalet (unpublished).
genetics: Shows complete *shakB* phenotype over *shakB*¹, but incomplete *shakB* phenotype over *shakB*² or *Df(1)16-3-35* (Baird *et al.*). Deficient for *l(1)19Ec*⁸-*su(f)*.

Df(1)LB23

cytology: *Df(1)19E6-7;20F*.
origin: Induced by ethyl methanesulfonate.
discoverer: Baldwin.
references: Schalet and Lefevre, 1973, Chromosoma 73: 83-100.
genetics: Deficient for *vao*-*su(f)*.

***Df(1)lz: Deficiency (1) lozenge**

origin: X ray induced.
references: Green and Green, 1956, Z. Indukt. Abstamm. Vererbungsl. 87: 708-21.
genetics: Deficient for *lz* and *amx*. Male lethal.

deficiency	cytology
*Df(1)lz1	7E11-F1;8E1-2
*Df(1)lz2	8C14-D2;8E3-4
*Df(1)lz3	8C1-3;8D12-E2
*Df(1)lz5	8D3-5;8F-9A

Df(1)lzA: see *ln(1)lzA

Df(1)m13

cytology: *Df(1)10B8;11A3-5*.
references: Woods, and Bryant, 1989, Dev. Biol. 134: 222-25.
genetics: Deficient for *l(1)10Bg*.

Df(1)m259-4: Deficiency (1) miniature

cytology: *Df(1)10C2-3;10E1-2* (Mortin and Lefevre, 1981, Chromosoma 82: 237-47).
origin: X ray induced.
discoverer: Demerec, 33i.
references: Dorn and Burdick, 1962, Genetics 47: 503-18.
 Craymer and Roy, 1980, DIS 55: 200-04.
 Greenleaf, Weeks, Voelker, Ohnishi, and Dickson, 1980, Cell 21: 785-92.
 Voelker, Wisely, Huang, and Gyurkovics, 1985, Mol. Gen. Genet. 201: 437-45.
genetics: Male lethal. Heterozygote with *m* has *m* phenotype. Heterozygote with *dy* is wild type. Recombines with *m*⁵⁹, *m*^D, *dy*^{61a}, *dy*, and *dy*^{58k}. Includes *tyl* (Craymer and Roy, 1980) and *RpII* (Greenleaf *et al.*, 1980). *Df(1)m 259-4/Df(1)KA6* survives and is extreme *m* (Craymer and Roy, 1980).
molecular biology: Left DNA breakpoint between -19 and -23 kb (Woods and Marsh).

Df(1)m-fw: Df(1) miniature to furrowed

references: Green, 1975, Mutat. Res. 29: 77-84.
genetics: Deficient for *m-fw*.

Df(1)m-KA7: see *Df(1)KA7*

***Df(1)M30: Deficiency (1) Minute-30.**

origin: Spontaneous.
discoverer: Schultz.
references: 1929, Genetics 14: 366-419.
genetics: Deficient for *cv* and *M(1)5D6A*.

Df(1)M31 - Df(1)M176

origin: Induced by ethyl methanesulfonate.

deficiency	cytology	ref ^α	genetics
Df(1)M31	{20A(prox)}	1	<i>uncl</i> ⁻ - <i>ms(1)B</i> ⁻
Df(1)M54	2B10;2B18	2	
Df(1)M82	{20A(prox)}	1	<i>uncl</i> ⁻
Df(1)M158	{20A(prox);20B-D}		<i>uncl</i> ⁻ - <i>ms(1)A</i> ⁻
Df(1)M171	{19E(distal)}		<i>run</i> ⁻ - <i>ms(1)E</i> ⁻
Df(1)M176	{19E(distal)}		<i>run</i> ⁻ - <i>shakB</i> ⁻

^α 1 = Lifschytz and Yakobovitz, 1978, Mol. Gen. Genet. 161: 275-84;
 2 = Lim and Snyder, 1954, Genet. Res. 24: 1-101.

Df(1)mal: Deficiency (1) maroonlike

origin: X ray induced.

deficiency	cytology	ref ^α	genetics
Df(1)mal	{19A1-2;20E-F} + <i>In(1)sc</i> ⁸	2	<i>l(1)carot3</i> ⁺ <i>ot</i> ⁻ - <i>su(f)</i> ⁻ <i>bb</i> ⁺
Df(1)mal3	19A1-2;20E-F	1, 5, 6	<i>l(1)carot3</i> ⁺ <i>ot</i> ⁻ - <i>su(f)</i> ⁻ <i>bb</i> ⁺
Df(1)mal4	{19A1-2;20A2-3}	1, 5, 6	<i>l(1)carot3</i> ⁺ <i>ot</i> ⁻ - <i>eo</i> ⁻ <i>wap</i> ⁺
Df(1)mal5	{19A-B;19E8-F1}	1, 5, 6	<i>l(1)carot3</i> ⁺ <i>sw</i> ⁻ - <i>vao</i> ⁻ <i>unc</i> ⁺
Df(1)mal6	19C3;20A2-3 + <i>In(1)BM1</i>	1, 5, 6	<i>mel</i> ⁺ <i>mal</i> ⁻ - <i>eo</i> ⁻ <i>wap</i> ⁺
Df(1)mal7	{19A1-2;19F-20A1}	1, 5, 6	<i>l(1)carot3</i> ⁺ <i>ot</i> ⁻ - <i>fti</i> ⁻ <i>eo</i> ⁺
Df(1)mal8	18F4-5;19E1	1, 5, 6	<i>l(1)carot3</i> ⁺ <i>ot</i> ⁻ - <i>mel</i> ⁻ <i>run</i> ⁺
Df(1)mal9	{19A1-2;20-E-F} + <i>In(1)sc</i> ⁸	1, 5	<i>l(1)carot3</i> ⁺ <i>ot</i> ⁻ - <i>su(f)</i> ⁻ <i>bb</i> ⁺
Df(1)mal10	19A5-6;19E1 + <i>In(1)sc</i> ⁸	1, 3, 5, 6	<i>ot</i> ⁺ <i>sw</i> ⁻ - <i>mel</i> ⁻ <i>run</i> ⁺ ; <i>Df(1)mal10/sw</i> females lethal; <i>Df(1)mal10/Ubl</i> shows moderate <i>sw</i> effect
Df(1)mal11	19A2-3;19E1 + <i>In(1)sc</i> ⁸	1, 5, 6	<i>l(1)carot3</i> ⁺ <i>ot</i> ⁻ - <i>mel</i> ⁻ <i>run</i> ⁺
Df(1)mal12	19A1;20F + <i>In(1)sc</i> ⁸	1, 4, 5, 6	<i>l(1)carot3</i> ⁺ <i>ot</i> ⁻ - <i>su(f)</i> ⁻ <i>bb</i> ; covered by <i>y</i> ⁺ <i>Ymal</i> ⁺ . <i>Df(1)mal12/bb</i> females <i>bb</i> ; <i>Hw</i> effect.
Df(1)mal13	19A2-3;20E-F	1, 5, 6	<i>mel</i> ⁺ <i>mal</i> ⁻ - <i>su(f)</i> ⁻ <i>bb</i> ⁺
Df(1)mal17^β	19A2-3;19E1 + <i>In(1)sc</i> ⁸ <i>SIL</i> _{sc} <i>8R</i>	1, 5, 6	<i>l(1)lAc</i> ⁻ - <i>ac</i> ⁻ ; <i>bb</i> ; <i>l(1)carot3</i> ⁺ <i>ot</i> ⁻ - <i>mel</i> ⁻ <i>run</i> ⁺ (double deficiency)
Df(1)mal22	19C3;19E1	1, 5, 6	<i>mel</i> ⁺ <i>mal</i> ⁻ - <i>mel</i> ⁻ <i>run</i> ⁺

^α 1 = Chovnick, Finnerty, Schalet, and Duck, 1969, Genetics 62: 145-70; 2 = Grell, 1962, Z. Indukt. Abstamm. Vererbungsl. 93: 371-77; 3 = Mortin and Lefevre, 1981, Chromosoma 82: 237-47; 4 = Schalet, 1969, Genetics 63: 133-53; 5 = Schalet and Finnerty, 1968, DIS 43: 65-66; 6 = Schalet and Lefevre, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 846-902.
^β Effectively a terminal deletion.

Df(1)mm69: Deficiency (1) methyl methanesulfonate

cytology: {*Df(1)3A9;3B1*}.
origin: Induced by methyl methanesulfonate.
synonym: *l(1)m69*.
references: Liu and Lim, 1975, Genetics 79: 601-11.
genetics: Deficient for *l(1)3Ah-l(1)3Ba*.

Df(1)mo

cytology: Breaks in *In(1)w*^{m4h} (distal region).
origin: X ray induced.
references: Reuter and Wolff, 1981, Mol. Gen. Genet. 182: 516-19.

THE GENOME OF *DROSOPHILA MELANOGASTER*

Df(1)N: Deficiency (1) Notch

deficiency	cytology	origin	discoverer	ref ^α	genetics	molec biol ^β
Df(1)N-8	3C2-3;3E3-4 (Lefevre)	spont	Mohr	13, 21, 22, 28	$N^{-}(1)3Ec^{-}$	
*Df(1)N-25		spont	Mohr	3	$w^{+}N^{-}$	
*Df(1)N-26	3C4-5;3C8-9	spont	Mohr	3	$rst^{+}fa^{-}spl^{-}dm^{+}$	
*Df(1)N-29		spont	Eker	3	$w^{-}N^{-}$	
*Df(1)N-33h	3C6-7;3D2-3	spont	Ives	24, 25	$w^{+}fa^{-}ec^{+}$	
*Df(1)N-38g	3C4-5;3C7-8	spont	Curry	3	$rst^{+}fa^{-}spl^{-}dm^{+}$	
Df(1)N-45e	3B4-C1;3C7-8	X ray	Poulson	9, 26, 34	N^{-}	
Df(1)N-5419^γ	3C5-6;3C10-11	spont		13, 16, 17, 20	$vt^{-}N^{-}$; viable and vt over Df(1)w67k30	
Df(1)N-62b1 Df(1)N-62G1	3C7-8 (or 3C7);3D5-6	X ray	Ives	10, 29, 31, 32, 33 6	$N^{55el1+}fa^{8-}dm^{-}$ N^{-} ; covered by Dp(1;2)51b	-19.3 to -18.6 kb
Df(1)N-63b	3C2-3;3E1-2	X ray		18	$rst^{-}dm^{-}$; carries w^{63b} ; Df(1)N-63b/w resembles w^{3P} /w	
Df(1)N-64I16	3C3;3D4		Welshons	11, 12	$rst^{-}dnc^{-}$; males sterile with $w^{+}Y$	see ref 8
Df(1)N64j15	3C3-4;3D3-4	X ray	Welshons	11, 19	$rst^{-}Sgs-4^{-}$; males fertile with $w^{+}Y$	see ref 5
Df(1)N-66h10 Df(1)N-66h12 Df(1)N-66h31	3C1-2;3C6-7	mutable w^{8}	Green	1, 8, 31, 32, 33 8	$w^{-}N^{55el1-}fa^{8+}$	see ref 8 see ref 8 see ref 8
Df(1)N-66i25 Df(1)N-68f19 Df(1)N-69h9	3C1-2;3C6-7 3C1-2;3C6-7 3C6;3D1 (Korge); 3C6;3D4 (Lefevre)	mutable w^{8} mutable w^{8} X ray	Green Green Lefevre	1, 7, 10, 31, 33 7, 10, 31, 33 4, 13, 19	$w^{-}N^{264-40-}N^{Co+}$ $w^{-}N^{55el1-}N^{264-40+}$ $N^{-}Sgs-4^{-}$	+0.8 to +2.7 kb -26.2 to -24.7 kb
Df(1)N-71a Df(1)N-71b9 Df(1)N-71h Df(1)N-75j31	3A6;3C10 (Lefevre) 3C2-3;3E3-4 3C4;3D5 3C7-9;3C10-D1	X ray X ray Green unequal crossover	Lefevre Lefevre Green Welshons	15 15 2, 4, 5, 11, 12 1, 33	N^{-} N^{-} $N^{-}dnc^{-}$ $N^{55el1+}fa^{8-}N^{60g11-}$	see ref 5 see ref 8
Df(1)N-81k1	3C5-6;3C9-10 (Welshons)	X ray	Muskavitch	8	N^{-}	-43.9 to -31.2 kb
Df(1)N-81I4	3C5-6;3D4-5 (Welshons)	X ray	Muskavitch	1, 8	N^{-}	
Df(1)N-81I6^δ	3C7-9;3D2-3 (Welshons)	X ray	Muskavitch	1, 8	$rst^{+}N^{55el1+}fa^{-}N^{j24}$	-14.8 to -9.5 kb
*Df(1)N-264-2 *Df(1)N-264-7	3C6-7;3C7-8 3C6-7;3C8-9 see *In(1)N ²⁶⁴⁻⁷	X ray X ray	Demerec Demerec	28 3	$rst^{+}fa^{-}spl^{-}ec^{+}$	
*Df(1)N-264-13	3C6-7;3C10-11	X ray	Demerec	3	$rst^{+}fa^{-}fa^{n-}$ $spl^{-}dm^{+}$	
*Df(1)N-264-15 *Df(1)N-264-19 *Df(1)N-264-30 *Df(1)N-264-31 *Df(1)N-264-32 *Df(1)N-264-33 *Df(1)N-264-36 *Df(1)N-264-37 Df(1)N-264-38 Df(1)N-264-39^ε *Df(1)N-264-41 *Df(1)N-264-42 *Df(1)N-264-46 *Df(1)N-264-48	3C6-7;3C7-8 3C6-7;3C7-8 3A4-5;3C7-9 3B4-C1;3D1-3 3C3-5;3C7-8 3C6-7;3C7-8 3A3-4;3D2-3 3C6-7;3C7-8 2D3-4;3E2-3 3C6-7;3C7-8 3C6-7;3C8-9 3C4-5;4B4-6 3C6-7;3C7-8 1B6-7;1B10-11 see *In(1)N ²⁶⁴⁻⁴⁸	X ray X ray X ray X ray X ray X ray X ray X ray X ray spont X ray X ray X ray	Demerec Demerec Demerec Demerec Demerec Hoover Demerec Demerec Demerec Slizynska Slizynska Demerec Demerec Demerec Demerec	3 28 27 27 27 27 27 27 27 28, 30 3 3 3 3	$w^{+}fa^{-}spl^{-}$ $w^{+}fa^{-}ec^{+}$ $kz^{+}w^{-}fa^{-}dm^{+}$ $pn^{+}w^{-}dm^{-}ec^{+}$ $w^{+}rst^{-}fa^{-}dm^{+}$ $rst^{+}fa^{-}dm^{+}$ $w^{-}dm^{-}$ $rst^{+}fa^{-}dm^{+}$ $br^{+}pn^{-}dm^{-}$ N^{-} $rst^{+}fa^{-}dm^{+}$ $rst^{+}fa^{-}ec^{-}bi^{+}$ $rst^{+}fa^{-}dm^{+}$	
*Df(1)N-264-49 *Df(1)N-264-51 *Df(1)N-264-54 Df(1)N-264-58	3C4-5;3E8-F1 3C6-7;3C7-8 3C3-5;3C7-8 3B2-3;3D6-7 see Tp(1;3)N ²⁶⁴⁻⁵⁸	X ray radium? X ray X ray	Demerec Demerec Demerec Demerec	3 3 3 3	$rst^{+}fa^{-}ec^{+}$ $rst^{+}fa^{-}dm^{+}$ $rst^{+}fa^{-}dm^{+}$	
*Df(1)N-264-68 *Df(1)N-264-72 *Df(1)N-264-73 *Df(1)N-264-76 *Df(1)N-264-77 *Df(1)N-264-79	3A10-B1;3E8-F1 3C6-7;3C7-9 3C3-4;4C6-7 3B4-C1;3E4-5 3B4-C1;3C7-8 2C10-D1;3C6-7	X ray X ray X ray X ray X ray X ray	Demerec Demerec Demerec Demerec Demerec Demerec	3 3 3 3 3 3	$kz^{+}w^{-}ec^{+}$ $rst^{+}fa^{-}dm^{+}$ $w^{+}rst^{-}bi^{+}$ $pn^{+}w^{-}ec^{+}$ $pn^{+}w^{-}dm^{-}ec^{+}$ $br^{+}kz^{-}rst^{-}dm^{+}$; weak N; fa affected $rst^{+}fa^{-}dm^{+}$	
*Df(1)N-264-81 *Df(1)N-264-86 *Df(1)N-264-89	3C6-7;3C7-8 3C7-8;3E5-6 see *Tp(1;4)N ²⁶⁴⁻⁸⁶ 3B2-3;3F2-3	X ray X ray X ray	Demerec Demerec Demerec	3 3 3	$pn^{+}w^{-}ec^{-}$	

deficiency	cytology	origin	discoverer	ref ^α	genetics	molec biol ^β
*Df(1)N-264-90	3C7-8;3E8-F1	X ray	Demerec	3	w ⁺ spl ⁻ ec ⁻	
*Df(1)N-264-93	3B4-C1;3F3-4	X ray	Demerec	3	pn ⁺ w ⁻ ec ⁻ bi ⁺	
*Df(1)N-264-96	3C6-7;3C7-8	X ray	Demerec	3	rst ⁺ spl ⁻ dm ⁺	
*Df(1)N-264-99	2D2-3;3C11-12	X ray	Demerec	3	pn ⁻ dm ⁻ ec ⁺	
*Df(1)N-264-100	3B4-C1;4B4-5 see *T(1;3)N ²⁶⁴⁻¹⁰⁰	X ray	Demerec	3		
*Df(1)N-264-101	3C4-5;3C7-8	X ray	Demerec	3	rst ⁺ spl ⁻ dm ⁺	
Df(1)N-264-105^ζ	3C6-7;3D2-3	X ray	Demerec	13	rst ⁺ spl ⁻ Sgs-4 ⁻ ec ⁺	
*Df(1)N-264-106	3C6-7;3C7-8	X ray	Demerec	3	rst ⁺ spl ⁻ dm ⁺	
*Df(1)N-264-108	3C3-5;3E7-8 see *In(1)N ²⁶⁴⁻¹⁰⁸			3		
*Df(1)N-264-110	3B4-C1;3D2-3	X ray	Demerec	3	pn ⁺ w ⁻ dm ⁻ ec ⁺	
*Df(1)N-264-111	3C3-5;3C12-D1	X ray	Demerec	3	w ⁺ rst ⁻ spl ⁻ dm ⁺	
*Df(1)N-264-114	3C6-7;3D4-5	spont	Kaufmann	3	w ⁺ rst ⁻ dm ⁻ ec ⁺	
*Df(1)N-264-115	3C3-5;3E2-3	X ray	Sutton	3	w ⁺ rst ⁻ dm ⁻	
*Df(1)N-264-117	3A6-7;3E2-3	X ray	Demerec	3	pn ⁺ w ⁻ dm ⁻ ec ⁺	
*Df(1)N-264-118	3C6-7;3C7-9	spont	Demerec	3	rst ⁺ spl ⁻ dm ⁺	
*Df(1)N-264-120	3C6-7;3D2-3	X ray	Demerec	3	rst ⁺ spl ⁻ dm ⁻ ec ⁺	
*Df(1)N-264-125	3C4-5;3C7-8	X ray	Demerec	3	rst ⁺ spl ⁻ dm ⁺	
*Df(1)N-264-126	3C3-5;3D4-5	spont	Bishop	3	w ⁺ rst ⁻ dm ⁻	
*Df(1)N-264-127	3C6-7;3C7-8	X ray	Demerec	3	rst ⁺ spl ⁻ dm ⁺	
*Df(1)N-264-128	3C6-7;3C7-8	X ray	Demerec	3	rst ⁺ spl ⁻ dm ⁺	
*Df(1)N-264-130	3C6-7;3C7-8	spont	Neel	23	rst ⁺ fa ⁻ dm ⁺	
*Df(1)N-B	3C4-5;3C12-D1	spont	Bernstein	3	rst ⁺ fa ⁻ dm ⁺	
Df(1)N-cm^η	3C7-8;3D3-5	unstable chromosome	Lim	14	N ⁻ cm ⁻	see ref 14
*Df(1)N-EZ	3C6-7;3C7-8	spont	Morgan	3	w ⁺ fa ⁻	
Df(1)N-FM21	3C1-2;3C7-D1		Bingham	19	w ⁻ Sgs-4 ⁻	
Df(1)N-h77.2		X ray on In(1)w ^{m4}		27	w ⁻ dm ⁻ ;bb ⁻	
Df(1)N-M1		X ray on In(1)w ^{m4}		27	w ⁻ dm ⁻ ;bb ⁻	
Df(1)N-M2		X ray on In(1)w ^{m4}		25	w ⁻ dm ⁻ ;bb ⁻	
Df(1)N-M8		X ray on In(1)w ^{m4}		25	w ⁻ dm ⁻ ;bb ⁻	

^α 1 = Artavanis-Tsakonas, Muskavitch, and Yedvobnick, 1983, Proc. Nat. Acad. Sci. USA 80: 1977-81; 2 = Byers, Davis, and Kiger, 1981, Nature 289: 79-81; 3 = CP627; 4 = Craymer and Roy, 1980, DIS 55: 200-04; 5 = Davis and Davidson, 1984, Mol. Cell Biol. 4: 358-67; 6 = Doane, 1969, J. Exp. Zool. 171: 321-42; 7 = Green, 1967, Genetics 56: 467-82; 8 = Grimwade, Muskavitch, Welshons, Yedvobnick, and Artavanis-Tsakonas, 1985, Dev. Biol. 107: 503-19; 9 = Hillman, 1961, Genetics 46: 1395-1409; 10 = Kidd, Lockart, and Young, 1983, Cell 34: 421-33; 11 = Kiger and Golanty, 1977, Genetics 85: 609-22; 12 = Kiger and Golanty, 1979, Genetics 91: 521-35; 13 = Korge, 1977, Chromosoma 62: 155-74; 14 = Johnson-Schlitz and Lim, 1987, Genetics 115: 701-09; 15 = Lefevre, 16 = Lefevre, 1971, DIS 46: 40; 17 = Lefevre and Green, 1972, Chromosoma 36: 391-412; 18 = Lefevre and Wilkins, 1966, Genetics 53: 175-87; 19 = McGinnis, Farrell, and Beckendorf, 1980, Proc. Nat. Acad. Sci. USA 77: 7367-71; 20 = Mohler, 1956, DIS 30: 78; 21 = Mohr, 1919, Genetics 4: 275-82; 22 = Mohr, 1932, Proc. Int. Congr. Genet., 6th, pp. 202; 23 = Neel, 1942, Genetics 27: 530; 24 = Plough and Ives, 1934, DIS 1: 31; 25 = Plough and Ives, 1934, DIS 2: 10,34; 26 = Poulson, 1945, DIS 19: 47; 27 = Reuter, Hoffmann, and Wolff, 1983, Biol. Zentralbl. 102: 281-98; 28 = Slizynska, 1938, Genetics 23: 291-99; 29 = Sorsa and Saura, 1980, Hereditas 92: 341-51; 30 = Welshons, 1958, Proc. Nat. Acad. Sci. USA 44: 254-58; 31 = Welshons, 1974, Genetics 76: 775-94; 32 = Welshons and Keppy, 1975, Genetics 80: 143-55; 33 = Welshons and Keppy, 1981, Mol. Gen. Genet. 181: 319-24; 34 = Wright, 1970, Adv. Genet. 15: 262-85.

^β The zero coordinate used in locating the molecular lesions in the Notch deficiencies is the first Eco R1 site in Canton-S DNA proximal to the 3C7 breakpoint of In(1)N^{76b8} (Kidd et al., 1983); it is 1.1 kb to the right of the zero coordinate used by Artavanis-Tsakonis et al., 1983.

^γ Lacks Sgs-4 sequences (McGinnis et al., 1980).

^δ 3C7 not deleted but appears thinner than normal (Grimwade et al., 1985).

^ε Later reexamination of chromosomes of males from lines carrying w^{ch} and marked N²⁶⁴⁻³⁹ revealed the presence of 3C7 (Welshons).

^ζ Cell lethal and lethal in germ-line clones (Ripoll and Garcia-Bellido, 1979, Genetics 91: 443-53).

^η 50 Notch mutations originating independently in In(1)cm-df = In(1)3D;6D + Df(1)6D;6F (a derivative of the unstable X Uc-1^{UD15}) showed this apparently identical X chromosome deficiency (New order: 1A1 - 3C7 | 6D - 3D | 6F - 20). Results from in situ hybridizations with 20 of these deficiencies indicated a deletion of 10 kb of the sequences from the 3' end of Notch had occurred (Johnson-Schlitz and Lim, 1987). The break position of these Notch deficiencies is within the 2.2 kb Eco R1 fragment with the next 3'-most clone (Kidd et al., 1983), which corresponds to the region between N²⁶⁴⁻⁴⁰ and spl in the genetic map.

Df(1)N12-Df(1)N110

origin: X ray induced.
discoverer: Lefevre.

deficiency	cytology	ref ^α	genetics	deficiency	cytology	ref ^α	genetics
Df(1)N12^β	11D1-2;11F1-2	1, 2, 3, 4, 5, 6, 8	sno ⁻ wy ⁻ s ⁺	Df(1)N110^γ	9B3-4;9D1-2	1, 2, 3, 8	Hk ⁻ fw ⁻ ras ⁺
Df(1)N19	17A1;18A2	1, 2, 3, 8	os ⁺ fu ⁻ hld ⁻				
Df(1)N37^γ	9B5-6;9D1-2	3	Hk ⁺ fw ⁻ ras ⁺				
Df(1)N71	10B5;10D4 10B2-8;10D3-8 (Zhimulev)	1, 3, 8, 9, 10	l(1)10Ag ⁺ dsh ⁻ tyl ⁻ dy ⁺				
Df(1)N73	5C2;5D5-6	1, 3, 7, 8	cv ⁺ rux ⁻ l(1)5CDa ⁻ vs ⁺				
Df(1)N77	19E2-3;20E	3	mell ⁻ su(f) ⁻				
Df(1)N105	10F7;11D1	1, 2, 3, 8	Flu ⁻ fw ⁻				

^α 1 = Craymer and Roy, 1980, DIS 55: 200-04; 2 = Deak, Bellamy, Bienz, Dubuis, Fenner, Gollin, Rähmi, Ramp, Reinhardt, and Cotton, 1982, J. Embryol. Exp. Morphol. 69: 61-81; 3 = Lefevre; 4 = Lefevre and Peterson, 1972, DIS 48: 126-27; 5 = Lefevre and Wright, 1976, Genetics 83: s44-45; 6 = Mortin and Lefevre, 1981, Chromosoma 82: 237-47; 7 = Voelker and Wisely, 1982, DIS 58: 150-51; 8 = Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307. 9 = Zhimulev, Belyaeva, Pokholkova, Kochneva, Fomina, Bgatov, Khudyakov, Patazevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eken, 1982, DIS 58: 210-14; 10 = Zhimulev, Pokholkova, Bgatov,

- β Semeshin, and Belyaeva, 1981, Chromosoma 82: 25-40.
 γ Synonym: *Df(1)sno-N12*.
 γ Listed incorrectly as vermilion deficiency by Kaplan and Trout, 1974, Genetics 77: 721-39.

Df(1)N-r69h9: see *Df(1)N-69h9*

Df(1)NF2

cytology: *Df(1)3C6-7;3E8*.

origin: X ray induced.

discoverer: Lefevre.

genetics: Deficient for *N - slc*.

Df(1)O4: see *Tp(1;3)O4*

Df(1)ovo: Deficiency (1) ovo

deficiency	cytology	ref ^α	genetics
<i>Df(1)ovo4</i>		2	<i>ovo⁻ - rg⁻</i>
<i>Df(1)ovo6</i> ^β	4C5-6;4E2-3	1	<i>peb⁻ - rg⁻</i>
<i>Df(1)ovo7</i> ^γ	4C5-6;4E2-3	1	<i>omb⁻ - rg⁻</i>
<i>Df(1)ovo14</i>		2	<i>ovo⁻ - rg⁻</i>
<i>Df(1)ovo15</i>		2	<i>ec⁻ - fs(1)A1621⁻</i>
<i>Df(1)ovo41</i>		2	<i>rb⁻ - fs(1)A1621⁻</i>
<i>Df(1)ovo44</i>		2	<i>bi⁻ - fs(1)A1621⁻</i>

- α 1 = Oliver, Perrimon, and Mahowald, 1988, Genetics 120: 159-71;
 2 = Steinmann-Zwicky, 1988, EMBO J. 7: 3889-98.
 β Synonym: *Df(1)ovo-D1rG6*.
 γ Synonym: *Df(1)ovo-D1rG7*.

Df(1)para: see *Df(1)80-Df(1)82*

Df(1)PC1

origin: Induced by hybrid dysgenesis.

references: Zusman, Coulter, and Gergen, 1985, DIS 61: 217-18.

genetics: Complements *uncl* and *l(1)20Cb* but not *fog* or *stn*.

Df(1)Pgd35: Deficiency (1) Phosphogluconate dehydrogenase

cytology: *Df(1)2C2-4;2E2-F1* (Nero); *Df(1)2D3;2F5* (Craymer and Roy, 1980). Perrimon *et al.* (1980) believe deficiency listed by Craymer and Roy is *Df(1)Pgd-kz*.

references: Craymer and Roy, 1980, DIS 55: 200-204. Perrimon, Engstrom, and Mahowald, 1984, Genetics 108: 559-72.

genetics: Deficient for *Pgd* and *pn* but not for *l(1)2Ea*. Viable and fertile over *Df(1)X12*.

Df(1)Pgd-kz: Deficiency (1) Phosphogluconate dehydrogenase to kurz

cytology: *Df(1)2D3-4;2F5*. Bands faint. 2F6 preserved.

origin: Co⁶⁰ irradiation of *Pgd^B* Canton-S flies.

references: Gerasimova and Ananiev, 1972, DIS 48: 93. Gvozdev, Gostimsky, Gerasimova, Dubrovskaya, and Braslovskaya, 1975, Mol. Gen. Genet. 141: 269-75. Gvozdev, Gerasimova, Kovalev, Ananiev, 1977, DIS 52: 67-68.

Perrimon, Engstrom, and Mahowald, 1984, Genetics 108: 559-72.

genetics: Deficient for *Pgd-kz-l(1)2Fd* but not for *br*.

Df(1)pn: Deficiency (1) prune

origin: X ray induced.

genetics: Deficient for *pn*.

deficiency	cytology	ref ^α
<i>Df(1)pn1</i> ^β		4
<i>Df(1)pn7a</i> ^γ	2E1;3A4	1, 2, 3, 5, 6
<i>Df(1)pn7b</i> ^γ	1E1-2;2B4-5	
<i>Df(1)pn10</i> ^δ	2C8-9;3A1-2	7
<i>Df(1)pn24</i>	2D5;2F5	3, 6
<i>Df(1)pn38</i> ^ε	2D3;2E3	6

- α 1 = Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90; 2 = Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelova, and Zhimulev, 1980, Chromosoma 81: 281-306; 3 = Ilyina, Sorokin, Belyaeva, and Zhimulev, 1980, DIS 55: 205; 4 = Orevi and Falk, 1975, Mutat. Res. 33: 193-200; 5 = Perrimon, Engstrom, and Mahowald, 1985, Genetics 111: 23-41; 6 = Slobodyanyuk and Serov, 1983, Mol. Gen. Genet. 191: 372-77; 7 = Valencia, 1966, DIS 41: 58.
 β Induced by Lifschytz (1968) on *In(1)sc*^δ.
 γ Double deficiency on same chromosome. *Df(1)1E1-2;2B4-5* deficient for *l(1)Ea-BRC*.
 δ Deficiency superimposed on *In(1)1B3-4;20F^L1B2-3;20F^R + In(1)4D7-E1;11F2-4*; listed as *Df(1)pn^{110Ac4}* in CP627.
 ε Listed as *Df(1)pn36* in Ilyina *et al.*, 1980.

Df(1)pn-ec: see **Tp(1;2)w⁺62g*

Df(1)Q38

origin: Induced by ethyl methanesulfonate.

references: Lifschytz and Falk, Mutat. Res. 8: 147-55.

genetics: Presumed double lethal.

Df(1)Q219

origin: Induced by ethyl methanesulfonate.

references: Lifschytz and Falk, Mutat. Res. 8: 147-55.

Df(1)Q408

origin: Induced by ethyl methanesulfonate.

references: Lifschytz and Falk, 1969, Mutat. Res. 8: 147-55.

Df(1)Q539

cytology: *Df(1)19E6;19F6-20A1*.

origin: Induced by ethyl methanesulfonate.

references: Lifschytz and Falk, 1969, Mutat. Res. 8: 147-55.

Schalet and Lefevre, 1973, Chromosoma 44: 183-200.

Craymer and Roy, 1980, DIS 55: 200-04.

genetics: Deficient for *vao-l(1)19Ff*.

Df(1)r: Deficiency (1) rudimentary

deficiency	cytology	origin	syn ^α	ref ^β	genetics ^γ
<i>Df(1)r-D1</i> ^δ	14D1-2;15D1-2	ICR-170	1	2, 3, 4, 5	<i>para⁻ r⁻</i>
<i>Df(1)r-D17</i> ^{ε, ζ}	14F6;15A6	X ray	2	1, 3, 5	<i>sbl⁻ - r⁻</i>
<i>Df(1)r-19</i> ^δ	14D1;15D1	ICR-170		5	<i>r⁻</i>

- α 1 = *Df(1)(D)*; 2 = *Df(1)D17*;
 β 1 = Falk, McCaughin, and Cogley, 1977, Genetics 86: 765-77; 2 = Ganetzky, 1984, Genetics 108: 897-911; 3 = Jarry, 1979, Mol. Gen. Genet. 172: 199-202; 4 = Mason, Green, Shaw, and Boyd, 1981, Mutat. Res. 81: 329-43; 5 = Naguib and Jarry, 1981, Genet. Res. 37: 199-207;
 γ Two Minutes surround *r*; the one on the left, *M(1)14C*, is new, the one on the right is *M(1)15D* (Lefevre).
 δ Semilethal opposite *r*, *r²⁹*, or *r³⁸*.
 ε Discoverer = M.M. Green.
 ζ Proximal break is determined to be 14C-D by Lilly and Carlson as opposed to Lefevre's determination of 14F6.

Df(1)r75c: see *Tp(1;2)r⁺75c*

Df(1)R: Deficiency (1) of Rahman

origin: X ray induced.

discoverer: Rahman.

references: Rahman and Lindsley, 1981, Genetics 99: 49-64.

genetics: A series of deficiencies for *su(f)* selected on the basis of the distinctive *su(f)/Df(1)su(f)* phenotype (Minute-like).

deficiency	genetics
<i>Df(1)R1</i> ^α	<i>l(1)19Ff⁻ bb⁻</i>
<i>Df(1)R2</i>	<i>uncl⁺ fog⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R3</i>	<i>eo⁺ wap⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R4</i>	<i>l(1)19Ff⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R5</i>	<i>l(1)19Ff⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R6</i>	<i>fog⁺ l(1)20Cb⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R7</i>	<i>uncl⁺ fog⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R8</i>	<i>sph⁺ su(f)^{pb} su(f)⁻ bb⁺</i>
<i>Df(1)R8A</i>	<i>fog⁺ l(1)20Cb⁻ bb⁻</i>
<i>Df(1)R9</i>	<i>l(1)19Ff⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R10</i> ^β	<i>uncl⁺ fog⁻ bb⁻</i>
<i>Df(1)R11</i>	<i>l(1)19Ff⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R12</i>	<i>l(1)20Ac⁺ uncl⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R13</i>	<i>eo⁺ wap⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R14</i>	<i>eo⁺ wap⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R15</i>	<i>eo⁺ wap⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R16</i>	<i>sph⁺ su(f)^{pb} su(f)⁻ bb⁺</i>
<i>Df(1)R17</i>	<i>l(1)20Cb⁺ sph⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R18</i>	<i>sph⁺ su(f)^{pb} su(f)⁻ bb⁺</i>
<i>Df(1)R19</i>	<i>l(1)19Ff⁺ eo⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R20</i>	<i>eo⁺ wap⁻ bb⁻</i>
<i>Df(1)R21</i>	<i>eo⁺ wap⁻ bb⁻</i>
<i>Df(1)R22</i>	<i>eo⁺ wap⁻ su(f)⁻ bb⁻</i>
<i>Df(1)R23</i>	<i>uncl⁺ fog⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R24</i>	<i>eo⁺ wap⁻ bb⁻</i>
<i>Df(1)R25</i>	<i>l(1)20Ac⁺ uncl⁻ bb⁻</i>
<i>Df(1)R26</i>	<i>l(1)20Ac⁺ uncl⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R27</i>	<i>eo⁺ wap⁻ bb⁻</i>
<i>Df(1)R28</i>	<i>eo⁺ wap⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R29</i> ^α	<i>l(1)19Ff⁺ eo⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R30</i>	<i>uncl⁺ fog⁻ bb⁺</i>
<i>Df(1)R31</i>	<i>eo⁺ wap⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R32</i>	<i>eo⁺ wap⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R33</i>	<i>eo⁺ wap⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R34</i> ^β	<i>l(1)19Ff⁻ bb⁻</i>
<i>Df(1)R35</i>	<i>eo⁺ wap⁻ bb⁻</i>
<i>Df(1)R36</i>	<i>uncl⁺ fog⁻ bb⁻</i>
<i>Df(1)R37</i>	<i>l(1)19Ff⁺ eo⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R38</i> ^α	<i>eo⁺ wap⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R39</i>	<i>l(1)19Ff⁻ bb⁻</i>
<i>Df(1)R40</i> ^γ	<i>eo⁺ wap⁻ bb⁻</i>
<i>Df(1)R41</i>	<i>eo⁺ wap⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R42</i>	<i>uncl⁺ fog⁻ bb⁻</i>
<i>Df(1)R43</i>	<i>uncl⁺ fog⁻ bb⁻</i>
<i>Df(1)R44</i>	<i>eo⁺ wap⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R45</i>	<i>l(1)19Ff⁺ eo⁻ su(f)⁻ bb⁺</i>
<i>Df(1)R46</i>	<i>l(1)20Ac⁺ uncl⁻ su(f)⁻ bb⁻</i>
<i>Df(1)R47</i>	<i>eo⁺ wap⁻ bb⁻</i>
<i>Df(1)R48</i>	<i>l(1)19Ff⁺ eo⁻ bb⁻</i>

α Associated with a *T(1;A)*.
 β Associated with a *T(1;2)*.
 γ Associated with a *T(1;3)*.

Df(1)R-9-1

origin: Induced by ethyl methanesulfonate.

references: Lifschytz and Falk, 1969, Mutat. Res. 8: 147-55.

Df(1)R-10-5

origin: Induced by ethyl methanesulfonate.

references: Lifschytz and Falk, 1969, Mutat. Res. 8: 147-55.

Df(1)RA

origin: X ray induced.

discoverer: Lefevre.

deficiency	cytology	ref ^α	genetics
<i>Df(1)RA2</i> ^β	<i>7D10;8A4-5</i>	3, 4, 5, 6, 7	<i>sdr⁻ptg⁻γ</i>
<i>Df(1)RA19</i>	<i>1E3-4;2B9-10</i>	1, 2, 3	<i>l(1)1EFa⁻ fmf⁻</i>
<i>Df(1)RA37</i>	<i>10A6;10B15-17</i>	3, 5, 7, 8, 9, 10	<i>l(1)10Ac⁺ l(1)10Ad⁻ dsh⁻ tyl⁺</i>
<i>Df(1)RA47</i>	<i>10F1;10F9-10</i>	3, 5, 7	<i>dy⁺ jtd⁻ fw⁺</i>

α 1 = Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90; 2 = Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelova, and Zhimulev, 1980, Chromosoma 81: 281-306; 3 = Craymer and Roy, 1980, DIS 55: 200-04; 4 = Gans, Forquignon, and Masson, 1980, Genetics 96: 887-902; 5 = Lefevre; 6 = Mortin and Lefevre, 1981, Chromosoma 82: 237-47; 7 = Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307; 8 = Zhimulev, Belyaeva, Pokholkova, Kochneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1982, DIS 58: 210-14; 9 = Zhimulev, Semeshin, and Belyaeva, 1981, Chromosoma 82: 9-23; 10 = Zhimulev, Semeshin, and Belyaeva, 1981, Chromosoma 82: 25-40.
 β Synonym: *Df(1)oc-RA2*.
 γ *Df(1)RA2/Ubl* females slight *oc*.

Df(1)ras: Deficiency (1) raspberry

deficiency	cytology	discoverer	ref ^α	genetics
<i>Df(1)ras59</i>	<i>9E1;9F10-11</i>	Nash	1	<i>ras⁻</i>
<i>Df(1)ras203</i>	<i>9E1-2;9F13</i>	Nash	1, 2	<i>ras⁻</i>
<i>Df(1)ras217</i>	<i>9A;9E7-8</i>	Nash	1	<i>ras⁻</i>
<i>Df(1)ras-P14</i> ^β	<i>9E1-2;9F3-4</i>	Patzevich	3, 4, 5	<i>ras⁻-(1)9Fa⁻</i>

α 1 = Janca, Woloshyn, and Nash, 1986, Genetics 112: 43-64; 2 = Nash and Janca, 1983, Genetics 105: 957-68; 3 = Zhimulev, Belyaeva, Pokholkova, Kochneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1982, DIS 58: 210-14; 4 = Zhimulev, Semeshin, and Belyaeva, 1981, Chromosoma 82: 9-23; 5 = Zhimulev, Semeshin, and Belyaeva, 1981, Chromosoma 82: 25-40.
 β origin: X ray induced.

Df(1)ras-v17: Deficiency (1) raspberry to vermilion

cytology: *Df(1)9D1-2;10A2-3* superimposed on *In(1)1B3-4;20F^L1B2-3;20F^R + In(1)4D7-E1;11F2-4*.

new order:

1A - 1B3|20F - 11F4|4E1 - 9D1|10A3 - 11F2|4D7 - 1B4|20F.

origin: X ray induced in *In(1)sc^{SIL}sc^{8R} In(1)dl-49*.

synonym: *Df(1)ras-v17Cc.8*.

references: Valencia, 1966, DIS 41: 58.

Johnson, Woloshyn, and Nash, 1979, Mol. Gen. Genet. 174: 287-92.

Zhimulev, Semeshin, and Belyaeva, 1981, Chromosoma 82: 9-23.

genetics: Deficient for *ras-sev*.

molecular biology: 10A2-3 breakpoint mapped to a restriction fragment between + 3.4 and 4.5 kb (Hafen, Basler, Ekström and Rubin, 1987, Science 36: 55-63); see molecular biology under *sev* for coordinates.

Df(1)ras-v-P26

cytology: *Df(1)9C5-6;10A1*.

origin: X ray induced.

discoverer: Zhimulev.

genetics: Deficient for *ras-csk*.

Df(1)rb: Deficiency (1) ruby

deficiency	cytology	origin	ref ^α	genetics
<i>Df(1)rb1</i>	3F6-4A1;4C7-8	γ ray	1	<i>mei-9⁻-amb⁻</i>
<i>Df(1)rb5</i>		γ ray	1	<i>omb⁻-rb⁻</i>
<i>Df(1)rb12</i>	3E3-5;4D3-7	γ ray	1	<i>mei-9⁻-amb⁻</i>
<i>Df(1)rb13</i>	4C5-6;4D3-E1	γ ray	1	<i>lac⁻-hnt⁻</i>
<i>Df(1)rb13a</i>			2	<i>rb⁻-β(1)302⁻</i>
<i>Df(1)rb14</i>			2	<i>rb⁻-rg⁻</i>
<i>Df(1)rb17</i>	3E3-5;4F1-2	γ ray	1	<i>mei-9⁻-amb⁻</i>
<i>Df(1)rb19</i>	3C7-9;4E3-F1	γ ray	1	<i>mei-9⁻-amb⁻</i>
<i>Df(1)rb23</i>			2	<i>ec⁻-peb⁻</i>
<i>Df(1)rb27</i>	3C9-12;4D3-7	γ ray	1	<i>mei-9⁻-amb⁻</i>
<i>Df(1)rb27a</i>			2	<i>bi⁻-rb⁻</i>
<i>Df(1)rb29</i>			2	<i>bi⁻-β(1)302⁻</i>
<i>Df(1)rb30</i>	4B1-2;4F2-4	γ ray	1	<i>mei-9⁻-Sxl⁻</i>
<i>Df(1)rb32</i>	4A6-B2;4E2-F1	γ ray	1	<i>mei-9⁻-svb⁻</i>
<i>Df(1)rb33</i>	3F3-4;4C15-16	γ ray	1	<i>mei-9⁻-hnt⁻</i>
<i>Df(1)rb34</i>			2	<i>bi⁻-peb⁻</i>
<i>Df(1)rb35</i>			2	<i>bi⁻-β(1)302⁻</i>
<i>Df(1)rb41</i>	4B6-C1;4C7-8	γ ray	1	<i>mei-9⁻-amb⁻</i>
<i>Df(1)rb42</i>	4B;4C		2	<i>bi⁻-rb⁻</i>
<i>Df(1)rb44</i>			2	<i>bi⁻-rb⁻</i>
<i>Df(1)rb46</i>	4A3-6;4C6-7	γ ray	1	<i>norpA⁻-amb⁻</i>
<i>Df(1)rb47</i>	4A1-2;4D1-2	γ ray	1	<i>cho⁻-hnt⁻</i>
<i>Df(1)rb48</i>			2	<i>bi⁻-β(1)302⁻</i>
<i>Df(1)rb50</i>			2	<i>bi⁻-rb⁻</i>
<i>Df(1)rb-V</i>	4B4-5;4D5-6	X ray	3	<i>rb⁻</i>

^α 1 = Banga, Bloomquist, Brodberg, Pye, Larrivee, Mason, Boyd, and Pak, 1986, Chromosoma 93: 341-46; 2 = Steinmann-Zwicky, 1988, EMBO J. 7: 3889-98; 3 = Valencia, 1966, DIS 41: 58.

Df(1)RC29

cytology: *Df(1)11A*.
discoverer: Lefevre.
genetics: *cac-fw*.

Df(1)RC40

cytology: *Df(1)4B1;4F1*.
origin: X ray induced.
discoverer: Lefevre.
synonym: *Df(1)bi^{RC40}*.
references: Craymer and Roy, 1980, DIS 55: 400-04.
Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux Arch. Dev. Biol. 193: 296-307.
Bausenwein, Wolf, and Heisenberg, 1986, J. Neurogenet. 3: 87-109.
Bottella, Grond, Saiga, and Edstrom, 1988, EMBO J. 7: 3881-88.
genetics: Deficient for *bi*, *rb*, *rg* and *svb* but not *ec*, *cho*, *bo*, or *cx*. *Df(1)RC40/Ubl* females show slight *bi* phenotype (Mortin and Lefevre, 1981, Chromosoma 82: 237-47). *Df(1)RC40/In(1)omb^{H31}* females show *omb^{H31}* phenotype (Heisenberg).

Df(1)RF19

cytology: *Df(1)7A4-5;7B2-3 + In(1)6A1;19E8*.
origin: X ray induced.
discoverer: Lefevre.
references: Johnson and Judd, 1979, Genetics 92: 485-502.
Nicklas and Cline, 1983, Genetics 103: 617-31.
Lefevre and Leeds, 1983, Genetics 104: s45-46.
genetics: Homozygous lethal. Deficient for *l(1)7Ab⁻-kf⁻*. Completely complements *ctⁿ*, *ct⁶*, and *ct^K*; fails to complement *kf²*. *In(1)HA46/Df(1)RF19* flies show *kf²* phenotype (Lefevre and Leeds, 1983).
molecular biology: Proximal breakpoint 55.2 - 50.5 kb distal to the insertion site of gypsy in *ct⁶* (Jack, 1985,

Cell 42: 869-76).

Df(1)RF19 – Df(1)RF55

origin: X ray induced.
discoverer: Lefevre.

deficiency	cytology	genetics
<i>Df(1)RF19</i>	see separate entry	
<i>Df(1)RF21</i>	19E2;20E-F	<i>run⁻-su(f)⁻</i>
<i>Df(1)RF39</i>	1A1;1B10-11	<i>l(1)1Aa⁻-vnd⁻</i>
<i>Df(1)RF44</i>	1A1;1A6-7	<i>l(1)1Aa⁻-l(1)1Ac⁻</i>
<i>Df(1)RF55</i>	19A;20	

Df(1)RK

references: B. Ganetzky.

deficiency	cytology	origin	genetics
<i>Df(1)RK2</i>	12D2-E1;13A2-5	γ ray	<i>ben⁻eag⁻na⁻</i>
<i>Df(1)RK3</i>	12E2-6;13A6-11	γ ray	<i>eag⁻</i>
<i>Df(1)RK4</i>	12F5-6;13A9-B1	recombination	<i>eag⁻</i>
<i>Df(1)RK5</i>	12E9-11;13A9-B1	recombination	<i>eag⁻</i>

^α Between *In(1)N282.2* and *In(1)P363* (W.R. Engels).
^β Between *In(1)N282.2* and *In(1)N366.2* (W.R. Engels).

Df(1)RpII215-61

cytology: *Df(1)10B8-9;10E1-2*.
origin: Induced by hybrid dysgenesis.
references: Voelker, Wisely, Huang, and Gyurkovics, 1985, Mol. Gen. Genet. 201: 437-45.
genetics: Deficient for *RpII215*.

Df(1)RpII215-172

cytology: *Df(1)10A1-2;11A1-2*.
origin: Induced by hybrid dysgenesis.
references: Voelker, Wisely, Huang, and Gyurkovics, 1985, Mol. Gen. Genet. 201: 437-45.
genetics: Deficient for *RpII215*.

Df(1)rst2: Deficiency (1) roughest

cytology: *Df(1)3C3-4;3C6-7*.
origin: Spontaneous.
discoverer: Bridges, 33d7.
references: Grüneberg, 1937, J. Genet. 34: 169-89.
Emmets, 1937, J. Genet. 34: 191-202.
Gersh, 1965, Genetics 51: 477-80 (fig.).
Gubb, Roote, Harrington, McGill, Durrant, Shelton, and Ashburner, 1985, Chromosoma 92: 116-23.
genetics: Deficient for *rst* and *vt*. Hemizygous males and homozygous females viable; emergence delayed (Lefevre and Green, 1972, Chromosoma 36: 391-412).

Df(1)run112: Deficiency (1) runt

cytology: *Df(1)19D-E*.
origin: Induced by ethyl methanesulfonate.
references: Gergen and Wieschaus, 1986, Cell 45: 289-99.
Gergen, 1987, Genetics 117: 477-85.
genetics: Deficient for *mal*, *run*, *vao*.

Df(1)S39

cytology: *Df(1)1E1;2B5-6* (Lefevre). *Df(1)1E1-2;2B5-6* (Belyaeva et al., 1982) or *Df(1)1E3;2B9 + In(1)1D1-2;1E1* (Lefevre).
origin: X ray induced.
discoverer: Lefevre.
synonym: *Df(1)br^{S39}*.

references: Craymer and Roy, 1980, DIS 55: 200-04.
 Mortin and Lefevre, 1981, Chromosoma 82: 237-47.
 Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova,
 Kramers, and Zhimulev, 1982, DIS 58: 184-90.
 Kramers, Schalet, Paradi, and Huiser-Hoogteyling, 1983,
 Mutat. Res. 107: 187-201.

genetics: Deficient for *l(1)1Ea - l(1)2Bd* but not for *dor*.
 Inversion produces a *su(w^a)* effect (Lefevre).
Df(1)S39/ + females slight *br*, *Df(1)S39/ br* females lethal
 (Lefevre).

molecular biology: 2B5-6 breakpoint between DNA coordinates 217 and 220.8 kb (Chao and Guild, 1986, EMBO J. 5: 143-50).

Df(1)S54

cytology: *Df(1)19E8;20A2*.
origin: X ray induced.
discoverer: Lefevre.
references: Perrimon, Smouse, and Miklos, 1989, Genetics 121: 313-31.
genetics: *unc⁻eo⁻*.

Df(1)s70e

cytology: *Df(1)4D1-2;4D7* (light bands missing).
origin: Induced by mutator gene, *mu*.
references: Green and Lefevre, 1972, Mutat. Res. 16: 59-64.

Df(1)S93: see Tp(1;1)S93

Df(1)SBLA

cytology: *Df(1)17A;17B* (Beer mann).
origin: X ray induced.
references: Lifschytz and Green, 1979, Mol. Gen. Genet. 171: 153-59.
genetics: Male lethal. Uncovers *hdp-a⁵*; *Bx³/Df(1)SBLA* has normal wing phenotype.

Df(1)sbr: Deficiency (1) small bristle

origin: X ray induced.
discoverer: Kochneva.
references: Zhimulev, Semeshin, and Belyaeva, 1981, Chromosoma 82: 9-23.
 Zhimulev, Belyaeva, Pokholkova, Kochneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1982, DIS 58: 210-14.

deficiency	cytology	genetics
<i>Df(1)sbr1</i>	9B9-10;9F13-10A1	<i>ras⁻-l(1)9Fg⁻</i>
<i>Df(1)sbr8</i>	9B1-2;10A1-2	<i>ras⁻-l(1)10Aa⁻</i>
<i>Df(1)sbr9</i>	9A2-4;10A1-2	<i>ras⁻-l(1)10Aa⁻</i>
<i>Df(1)sbr10</i>	9A2-4;9F13-10A1	<i>ras⁻-l(1)9Fg⁻</i>

Df(1)sc^{4L}sc^{8R}: see In(1)sc^{4L}sc^{8R}

Df(1)sc^{4L}sc^{L8R}: see In(1)sc^{4L}sc^{L8R}

Df(1)sc⁸: Deficiency (1) scute-8

cytology: *Df(1)1A1;1B2*.
origin: Spontaneous in *In(1)sc⁸*.
discoverer: Noujdin.
references: 1935, Zool. Zh. 14: 317-52.
 Baker, 1973, Dev. Biol. 33: 429-40.
 Deak, I.I., Bellamy, Bienz, Dubuis, Fenner, Gollin, Rähmi, Ramp, Reinhardt, and Cotton, 1982, J. Embryol. Exp. Morphol. 69: 61-81.
genetics: Deficient for *cin*, *l(1)1Ac*, *y*, *ac*, *Hw*, and *ewg*

(Deak *et al.*, 1982). Male lethal, dies as late embryo; larva nearly complete (Poulson, 1940, J. Expt. Zool. 83: 271-325) but little or no muscular movement (Wright, 1970, Advances in Genetics 15: 262-85).

***Df(1)sc⁸25b - *Df(1)sc⁸WO**

A series of derivatives of either *In(1)sc⁸* or *In(1)sc^{8L}EN^R* that have lost spontaneously the distal euchromatic segment of *In(1)sc⁸*, with or without the loss of adjacent X heterochromatin. The material lost may be replaced with *YS* by means of exchange between *YS* and the heterochromatic segment of the X removed distally by *In(1)sc⁸*; alternatively, the X terminus may not be capped by a recognizable element. The material lost is required for viability; this requirement can be met by either *In(1)EN^R* or *y⁺Y*.

discoverer: Lindsley (except for *Df(1)sc8-M* which was discovered by Mather, 1937).

synonym: *sc⁸c.p.X* [*In(1)sc⁸* derivatives] or *sc⁸ENc.o.X* [*In(1)sc^{8L}EN^R* derivatives].

deficiency	ref ^α	genetics
<i>*Df(1)sc⁸25b^{βδ}</i>	2	<i>y⁻ac⁻bb</i>
<i>*Df(1)sc⁸35a^γ</i>	1	<i>KS y⁻ac⁻bb⁺</i>
<i>*Df(1)sc⁸53c^γ</i>	2	<i>y⁻ac⁻</i>
<i>*Df(1)sc⁸67h^β</i>	1	<i>KS y⁻ac⁻bb⁺</i>
<i>*Df(1)sc⁸89a^β</i>	1	<i>KS y⁻ac⁻bb⁺</i>
<i>*Df(1)sc⁸91b^β</i>	1	<i>KS y⁻ac⁻bb⁺</i>
<i>*Df(1)sc⁸99c^β</i>	2	<i>y⁻ac⁻bb⁺</i>
<i>*Df(1)sc⁸B1^{γδ}</i>	2	<i>y⁻ac⁻</i>
<i>*Df(1)sc⁸C4^γ</i>	2	<i>y⁻ac⁻</i>
<i>*Df(1)sc⁸C6^{γδ}</i>	2	<i>y⁻ac⁻</i>
<i>*Df(1)sc⁸C13^γ</i>	2	<i>y⁻ac⁻</i>
<i>*Df(1)sc⁸D6^γ</i>	2	<i>y⁻ac⁻</i>
<i>*Df(1)sc⁸D8^{γδ}</i>	2	<i>y⁻ac⁻</i>
<i>*Df(1)sc⁸E1^β</i>	1	<i>KS y⁻ac⁻bb⁺</i>
<i>*Df(1)sc⁸F7^γ</i>	2	<i>y⁻ac⁻</i>
<i>*Df(1)sc⁸J3^{γδ}</i>	2	<i>y⁻ac⁻bb</i>
<i>*Df(1)sc⁸K1^{γδ}</i>	2	<i>y⁻ac⁻bb</i>
<i>*Df(1)sc⁸L7^γ</i>	1	<i>KS y⁻ac⁻bb⁺</i>
<i>*Df(1)sc⁸M^β</i>		
<i>*Df(1)sc⁸O7^γ</i>	2	<i>y⁻ac⁻</i>
<i>*Df(1)sc⁸P0^β</i>	1	<i>KS y⁻ac⁻bb⁺</i>
<i>*Df(1)sc⁸P0^γ</i>	1	<i>KS y⁻ac⁻bb⁺</i>
<i>Df(1)sc⁸P7^γ</i>	1	<i>KS y⁻ac⁻bb⁺</i>
<i>*Df(1)sc⁸Q1^{γe}</i>	2	<i>y⁻ac⁻</i>
<i>*Df(1)sc⁸S7^{γδ}</i>	2	<i>y⁻ac⁻bb⁻</i>
<i>*Df(1)sc⁸W0^{γδ}</i>	2	<i>y⁻ac⁻bb⁻</i>

^α 1 = Lindsley, 1955, Genetics 40: 24-44. 2 = Lindsley, 1958, Z. Indukt. Abstamm. Vererbungsleh. 89: 103-22.
^β Spontaneous derivative of *In(1)sc⁸*.
^γ Spontaneous derivative of *In(1)sc^{8L}EN^R*.
^δ Lacks hB and the portion of hA distal to the right breakpoint of *In(1)sc⁸*.
^e Lacks the portion of hA distal to the right breakpoint of *In(1)sc⁸*.

Df(1)sc^{8L}sc^{4R}: see In(1)sc^{8L}sc^{4R}

Df(1)sc^{8L}sc^{L8R}: see In(1)sc^{8L}sc^{L8R}

Df(1)sc^{8L}sc^{S1R}: see In(1)sc^{8L}sc^{S1R}

Df(1)sc²⁶⁰⁻²⁵: see In(1LR)sc²⁶⁰⁻²⁵

Df(1)sc^{L8L}sc^{8R}: see In(1)sc^{L8L}sc^{8R}

Df(1)sc^{L8L}sc^{S1R}: see In(1)sc^{L8L}sc^{S1R}

Df(1)sc^{V1}: see In(1LR)sc^{V1}

Df(1)sc10-1

cytology: *Df(1)1B1-2;1B2-3*.

new order:

1A – 1B1|1B14 – 1B3|1C1 – 20;

1B2 missing.

origin: X-ray-induced derivative of $In(1)ac^3 = In(1)1B2-3;1B14-C1$.

discoverer: Sturtevant, 1930.

references: 1935, DIS 3: 15.

1936, Genetics 21: 444-66.

García-Bellido, 1979, Genetics 91: 491-520.

Villares and Cabrera, 1987, Cell 50: 415-24.

genetics: Mutant for *ac* and *sc*; male viability low. The adults, which occasionally eclose, lack macro- and microchaetae, except those of the eye and wing margin. Homozygous cell clones in wing lack neurons except along anterior margin (Schubiger and Palka, 1985, Dev. Biol. 108: 399-410).

molecular biology: No deletion detected in cloned *ac* region (Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, Cell 40: 327-38). Sequence data indicate that there is a C to T change in nucleotide 669, a C to G change in nucleotide 1143 that converts Ser 101 to Arg, and a C to T change in nucleotide 1147 that generates a stop codon (Villares and Cabrera, 1987).

***Df(1)sc15**

origin: X ray induced.

discoverer: Muller.

references: Patterson and Muller, 1930, Genetics 15: 495-577.

Dubin, 1933, J. Genet. 27: 443-64.

genetics: Mutant for *sc*; deficient for *y* and *ac*. Apparently, *y*⁺ and *ac*⁺ loci were inserted into an autosome and subsequently lost. Originally tested as an allele of *sc* only. Male lethal.

Df(1)sc19: see *Tp(1;2)sc*¹⁹

Df(1)sc-B57

references: González, Romani, Cubas, Modolell, and Campuzano, 1989, EMBO J. 8: 3553-62.

genetics: Deficient for *ASC* and *l(1)1Bb*.

molecular biology: Shown to have left breakpoint between *y* and *ac* and right breakpoint just to the right of that of *Df(1)260-1*.

Df(1)sc-Fah: Deficiency (1) scute of Fahmy

cytology: *Df(1)1A8-B1;1B2-3*.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl) aminophenylalanine (CB. 3007).

discoverer: Fahmy, 1954.

references: 1958, DIS 32: 74.

genetics: Probably mutant for *sc*. Male viable; homozygous female lethal.

Df(1)sc-J4: see *T(1;3)sc*^{J4}

Df(1)sd72b: Df (1) scalloped

cytology: *Df(1)13F1;14B1*.

origin: X ray induced.

discoverer: Lefevre, 72b26.

synonym: *Df(1)sd*.

references: Craymer and Roy, 1980, DIS 55: 200-04.

Ganetzky and Wu, 1982, Genetics 100: 597-614.

Martin and Lefevre, 1981, Chromosoma 82: 237-47.

genetics: Deficient for *sd* but not for *bas*. Extreme *sd* phenotype over *RpII215*^{Ubl}.

Df(1)Si: Deficiency (1) Simmons

origin: Induced by hybrid dysgenesis.

references: Simmons, Raymond, Culbert, and Laverty, 1984, Genetics 107: 49-63.

deficiency	cytology	location of lethal
Df(1)Si1 ^α	1A1;1A8-B1	y
Df(1)Si2	1A1;1A8-B1 + <i>Dp(1;1)19C;19F</i>	y
Df(1)Si3	19A2-3;19D2-E1	mal
Df(1)Si4	19A4-5;19F3-20A	mal
Df(1)Si5	19A2-3;19D2-E1	mal

^α Six deficiency chromosomes with these breakpoints induced.

Df(1)SJ1

origin: Spontaneous.

discoverer: Schalet.

deficiency	genetics
Df(1)SJ1a	<i>l(1)1Aa</i> ⁻ - <i>ewg</i> ⁻ <i>arth</i> ⁺
Df(1)SJ1b	<i>l(1)1Aa</i> ⁻ - <i>l(1)1Ac</i> ⁻ <i>dmd</i> ⁺
Df(1)SJ1c	<i>l(1)1Aa</i> ⁻ - <i>l(1)1Ac</i> ⁻ <i>dmd</i> ⁺
Df(1)SJ1d	<i>l(1)1Aa</i> ⁻ - <i>l(1)1Ac</i> ⁻ <i>dmd</i> ⁺

Df(1)slc-A113: see *Df(1)A113*

DF(1)SMG

cytology: *Df(1)1C;1F*.

origin: γ ray induced.

synonym: *Df(1)1C;1F*.

references: Semenova, Mglinetz, and Glotoff, 1970, Genetika (Moscow) 6: 165-69.

Df(1)sn: Deficiency (1) singed

cytology: $\{Df(1)7C-D1;7D22-E1\}$. Distal breakpoint (7B2-3) of Hinton and Welshons, 1955, moved proximally since not deficient for *ct*.

origin: Spontaneous in *R(1)2*.

discoverer: C. Hinton.

references: Hinton and Welshons, 1955, DIS 29: 125-26.

genetics: Deficient for *sn* but not *ct*, *oc*, or *ptg*.

Df(1)sn-C128: see *Df(1)C128*

Df(1)sno-N12: see *Df(1)N12*

Df(1)sol: Deficiency (1) small optic lobes

cytology: Small deficiency on X.

references: Miklos, Kelly, Coombe, Leeds, and Lefevre, 1987, J. Neurogenet. 4: 1-19.

genetics: Recessive lethal. Deficient for *sol* and four lethals, one to the left and three to the right.

Df(1)Sp: Deficiency (1) Spontaneous

origin: Spontaneous in Amherst laboratory wild-type males, with the exception of *Df(1)Sp(J1.d)* and *Df(1)Sp(ras-v)*.

discoverer: Schalet.

deficiency	cytology	synonym	genetics
Df(1)Sp2F-3A	2F2;3A6 ^α	<i>l(1)11-83</i>	<i>kz</i> ⁺ <i>l(1)2Fb</i> ⁻ - <i>l(1)3Ac</i> ⁻ <i>l(1)3Ad</i> ⁺
Df(1)Sp8A	7F10-8A1;8A2-3	<i>l(1)7-107</i>	<i>gg</i> ⁺ <i>l(1)8Aa</i> ⁻ - <i>l(1)8Ab</i> ⁻ <i>oc</i> ⁺

deficiency	cytology	synonym	genetics
Df(1)Sp20A-F	20A3;20F4	<i>l(1)19-117</i>	<i>eo</i> ⁺ <i>wap</i> ⁻ <i>-su(f)</i> ⁻ <i>bb</i> ⁺
Df(1)Sp(br-dor) ^β _Y		<i>l(1)19-30P</i>	<i>sta</i> ⁺ <i>br</i> ⁻ <i>-dor</i> ⁻
Df(1)Sp(J1a)		<i>l(1)1-96</i>	<i>l(1)1Ab</i> ⁻ <i>-ewg</i> ⁻ <i>arth</i> ⁺
Df(1)Sp(J1b)		<i>l(1)5-13</i>	<i>l(1)1Ab</i> ⁻ <i>-dmd</i> ⁻ <i>l(1)1Aa</i> ⁺
Df(1)Sp(J1c)		<i>l(1)17-18</i>	<i>l(1)1Ab</i> ⁻ <i>-dmd</i> ⁻ <i>l(1)1Aa</i> ⁺
Df(1)Sp(J1d) ^γ		<i>l(1)05-22</i>	<i>l(1)1Aa</i> ⁻ <i>-l(1)1Ab</i> ⁻ <i>cin</i> ⁺
Df(1)Sp(ras-v) ^δ		<i>l(1)04-27</i>	<i>ras</i> ⁻ <i>-v</i> ⁻ <i>Fs(1)10A</i> ⁺
Df(1)Sp(rb) ^ε		<i>l(1)15-81</i>	<i>cho</i> ⁺ <i>mei-9</i> ⁻ <i>-peb</i> ⁻

^α Cytology by Lefevre, who finds *l(1)3Ad* included.
^β Does not complement *Df(1)sta = Df(1)1D3-E1;2A*.
^γ Spontaneous in a cross between wild-type males and *mei-9* females.
^δ Covered by *Dp(1;2)v⁺75d = Dp(1;2)9A2;10C2;40-41*.
^ε Complements *Df(1)JC70 = Df(1)4C15-16;5A1-2*.

Df(1)St472

cytology: *Df(1)2B6-8;2B11-12*.

origin: X ray induced.

discoverer: Pak.

references: Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90.

genetics: Deficient for *dor-fmf*.

Df(1)sta: see **Tp(1;3)sta**

Df(1)su(f): Deficiency (1) suppressor of forked

origin: X ray induced.

deficiency	cytology	ref ^α	genetics ^β
Df(1)su(f)4B ^γ	20A;20F	2, 4	<i>wap</i> ⁻ <i>-su(f)</i> ⁻ <i>bb</i> ⁺
Df(1)su(f)5A	20A;20F	1, 3	<i>wap</i> ⁻ <i>-su(f)</i> ⁻ <i>bb</i> ⁺
Df(1)su(f)724	20B;20F	1	<i>stn</i> ⁻ <i>-su(f)</i> ⁻
Df(1)su(f)733	20D;20F	1	<i>l(1)20Cb</i> ⁻ <i>-su(f)</i> ⁻
Df(1)su(f)754	20A;20F	1	<i>eo</i> ⁻ <i>-su(f)</i> ⁻
Df(1)su(f)795	20A;20F	1	<i>uncl</i> ⁻ <i>-su(f)</i> ⁻
Df(1)su(f)7009	20B;20F	1	<i>stn</i> ⁻ <i>-su(f)</i> ⁻
Df(1)su(f)7085	19E;20F	1	<i>unc</i> ⁻ <i>-su(f)</i> ⁻
Df(1)su(f)9122	19F;20F	1	<i>fii-l</i> ⁻ <i>-su(f)</i> ⁻

^α 1 = Lifschytz and Jakobovitz, 1978, Mol. Gen. Genet. 161: 275-84; 2 = Schalet; 3 = Schalet and Lefevre, 1973, Chromosoma 44: 183-200; 4 = Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307.
^β Constitution with respect to *bb* not provided.
^γ Induced in *In(1)sc^{51L}sc^{8R} In(1)dl-49* (Schalet).

Df(1)su(s): Deficiency (1) suppressor of sable

deficiency	genetics
Df(1)su(s)2	<i>l(1)1Bg</i> ⁻ <i>-brc</i> ⁻
Df(1)su(s)3	<i>l(1)1Bg</i> ⁻ <i>-brc</i> ⁻
Df(1)su(s)4	<i>l(1)1Bb</i> ⁻ <i>-l(1)Ca</i> ⁻

Df(1)su(s)83

cytology: *Df(1)1B10;1D6-E1* (Lefevre; Voelker).

origin: γ ray induced in *y cho* chromosome (Flemming).

references: Voelker, Huang, Wisely, Sterling, Bainbridge, and Hiraizumi, 1989, Genetics 122: 625-42.

genetics: Deficient for *su(s)*.

Df(1)su(s)E2

references: Voelker.

genetics: Deficient for *l(1)1Bg - l(1)1Bk*.

Df(1)svr: Deficiency (1) silver

cytology: *Df(1)1A1;1B9-10* (Lefevre). An apparently terminal deficiency.

origin: Found among progeny of cold-treated female.

discoverer: L. V. Morgan.

references: 1940, DIS 13: 51.

Sutton, 1943, Genetics 28: 213.

Ripoll and García-Bellido, 1979, Genetics 91: 443-53.

White, 1980, Dev. Biol. 80: 332-34.

Kramers, Schalet, Paradi, and Huizer-Hooteyling, 1983, Mutat. Res. 107: 187-201.

genetics: Deficient for *l(1)1Aa - vnd* but not *su(s)* or *sta*. Embryonic lethal; abnormal development of nervous system and cell lethal (Ripoll and García-Bellido).

Df(1)Sxl-bt: Deficiency (1) Sex-lethal

cytology: *Df(1)6E2;7A6*.

references: Nicklas and Cline, 1983, Genetics 103: 617-31.

Cline, 1984, Genetics 107: 231-77.

Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307.

genetics: Deficient for *l(1)6Eb - l(1)7Aa*.

Df(1)Sxl-ra

cytology: *Df(1)6F5;7B1* (Lim); *Df(1)7A1;7B3* (Wieschaus et al., 1984).

origin: Revertant of dominant male lethality of *Sxl^{M#1}*.

discoverer: Cline.

references: Nicklas and Cline, 1983, Genetics 103: 617-31.

Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307.

genetics: Deficient for *Sxl-l(1)7Ad*.

Df(1)T

origin: Induced by triethylenemelamine.

references: Lim and Snyder, 1974, Genet. Res. 24: 1-10.

deficiency	cytology
Df(1)T3 ^α	<i>1A1-2;1A7-8</i>
Df(1)T4	<i>1A1-2;1A5-6</i>
Df(1)T9A	<i>11D3-5;11D10-E1</i>
Df(1)T9B	<i>12A2-4;12A9-B1</i>
Df(1)T19	<i>1A1-2;1A8-B1</i>
Df(1)T24	<i>16A6-B1;16B2-4</i>
Df(1)T30	<i>3C6-7;3C8-9</i>
Df(1)T34	<i>2C2-4;2C10-D1</i>
Df(1)T36	<i>11A3-5;11A10-11</i>

^α Deficient for *l(1)1Ac - cin*.

Df(1)T2-4A

cytology: *Df(1)19B3;19C4*.

origin: Tritiated deoxycytidine.

discoverer: Kaplan.

synonym: *l(1)t2-4a*.

references: Schalet and Finnerty, 1968, DIS 43: 128-29.

Schalet and Lefevre, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 846-902.

genetics: Deficient for *sw-mel*; lethal with *sw* and mutant with *mel*.

Df(1)T2-14A

cytology: *Df(1)19E5-6;19E7-8*.

origin: Induced by tritiated deoxycytidine.

discoverer: Kaplan.

synonym: *l(1)t2-14a*.

references: Schalet and Finnerty, 1968, DIS 43: 128-29.

Schalet and Lefevre, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic

Press, London, New York, San Francisco, Vol. 1b, pp. 846-902.

genetics: Deficient for *lf-vao*. Viable with *Df(1)16-3-35* (Schalet and Lefevre, 1976) in spite of the cytology listed for the deficiencies.

Df(1)T100 - Df(1)T138

origin: Induced by methyl methanesulfonate.
references: Parádi, Vogel, and Szilágyi, 1983, Mutat. Res. 111: 145-59.
genetics: Recessive lethal.

deficiency	cytology
<i>Df(1)T100</i>	19D-E;20A
<i>Df(1)T101</i>	19F;20F
<i>Df(1)T103</i>	19E;20F
<i>Df(1)T105</i>	20A;20F
<i>Df(1)T106</i>	19E-F;19F
<i>Df(1)T107</i> ^α	19C;20A
<i>Df(1)T110</i>	20B;20F
<i>Df(1)T113</i>	19C;20A
<i>Df(1)T114</i> ^α	19F;19F-20A
<i>Df(1)T122</i>	19F;20A
<i>Df(1)T123</i>	19F;20F
<i>Df(1)T124</i> ^α	19D-E;20F
<i>Df(1)T136</i> ^α	20B;20F
<i>Df(1)T137</i> ^α	20A;20F
<i>Df(1)T138</i> ^α	19E;20F

^α Double mutant.

****Df(1)t282-1: Deficiency (1) tan***

cytology: *Df(1)8C2-3;8C14-D1* (Sutton). Green and Green (1956, Z. Indukt. Abstamm. Vererbungsl. 87: 708-21) suggested that the deficiency probably extends farther to the right.
origin: X ray induced.
discoverer: Demerec, 34c.
references: CP627.
genetics: Deficiency for *t*, *lz*, and *amx* but not *dd*, *dvr*, *flp*, *ny*, or *ras*.

Df(1)TEM

origin: Induced by triethylenemelamine.
references: Lim and Snyder, 1974, Genet. Res. 24: 1-10.

deficiency	cytology	ref ^α	genetics
<i>Df(1)TEM1</i>	2E2-F1;3C1-2	1, 3, 5, 6	<i>kz</i> ⁻ - <i>w</i> ⁻
<i>Df(1)TEM6</i>	2F5-3A1;3A3	3	<i>gf</i> ⁻ - <i>z</i> ⁻
<i>Df(1)TEM7</i>	3A3;3B3	3, 4	<i>l(1)3Ac</i> ⁻ - <i>l(1)3Bc</i> ⁻
<i>Df(1)TEM75</i>	2F5-3A1;3C2-4	3	<i>gf</i> ⁻ - <i>w</i> ⁻
<i>Df(1)TEM202</i> ^β	3B2;3C3-5	3, 4	<i>l(1)3Bb</i> ⁻ - <i>w</i> ⁻
<i>Df(1)TEM304</i>	2E2-F1;3A4-6	1, 3, 4	<i>l(1)2Fd</i> ⁻ - <i>l(1)3Ac</i> ⁻
<i>Df(1)TEM501</i>	2E1;3C1-3	1, 2, 3	<i>kz</i> ⁻ - <i>w</i> ⁻

^α 1 = Gvozdev, Gerasimova, Kovalev, and Ananiev, 1977, DIS 52: 67-68; 2 = Lim, 1979, Genetics 93: 681-701; 3 = Lim and Snyder, 1974, Genet. Res. 24: 1-10; 4 = Liu and Lim, 1975, Genetics 79: 601-11; 5 = Raymond, Laverty, and Simmons, 1986, DIS 63: 111-14; 6 = Simmons, Raymond, Culbert, and Laverty, 1984, Genetics 107: 49-63.

^β Deletion includes *per* (Bargiello, Jackson, and Young, 1984, Nature 312: 752-54; Bargiello and Young, 1984, Proc. Nat. Acad. Sci. USA 81: 2142-46). Molecular data show left breakpoint approximately 108 kb distal to *w*^α copia insertion point (Pirrotta, Hadfield, and Pretorius, 1983, EMBO J. 2: 927-34; Reddy, Zehring, Wheeler, Pirrotta, Hadfield, Hall, and Rosbach, 1984, Cell 38: 701-10).

Df(1)v: Deficiency (1) vermilion
origin: X ray induced.

deficiency	cytology	ref ^α	genetics
*<i>Df(1)v63i</i>	9D4-E1;10A11-10B1; see <i>Dp(1;2)v</i> ⁺ 63i		
<i>Df(1)v64f</i>^β	9E7-8;10A1-2	1, 2, 3, 4, 9	<i>ras</i> ⁺ <i>l(1)9Fa</i> ⁻ - <i>l(1)10Aa</i> ⁻
<i>Df(1)v65b</i>	9F13-10A1;11A7-8; see <i>T(1;2)v</i> ^{65b}		
<i>Df(1)v73</i>^γ	10A2;10A5	6	<i>sev</i> ⁻ - <i>l(1)10Ac</i> ⁻
*<i>Df(1)v166</i>	10A1-2;10A6-8	3	<i>v</i> ⁻ - <i>l(1)10Ac</i> ⁻
<i>Df(1)v-B151</i>	9A1-4;10A1-2	8	<i>v</i> ⁻
<i>Df(1)v-JA22</i>	9F13;10A1	5	<i>v</i> ⁻
<i>Df(1)v-L1</i>	10A1;10A5; see <i>T(1;3)v</i> ^{L1}		
<i>Df(1)v-L2</i>	9F13;10A1	3, 4, 9	<i>l(1)9Fg</i> ⁻ - <i>l(1)10Aa</i> ⁻
<i>Df(1)v-L3</i>	9F10;10A7-8	3, 4, 9	<i>flig</i> ⁻ - <i>l(1)10Ae</i> ⁻ <i>rtv</i> ⁺
<i>Df(1)v-L4</i>	9F5-6;10A1-2	3, 4, 9	<i>ras</i> ⁺ <i>sbr</i> ⁻ - <i>l(1)10Aa</i> ⁻
*<i>Df(1)v-L5</i>	9E8-10;10A1-2	3	<i>ras</i> ⁺ <i>v</i> ⁻
*<i>Df(1)v-L6</i>	9E1-2;10A1-2	3	<i>ras</i> ⁻ <i>v</i> ⁻
<i>Df(1)v-L7</i>	9E1-2;10A1-2	3, 9	<i>ras</i> ⁻ - <i>l(1)10Aa</i> ⁻
<i>Df(1)v-L8</i>	9D3-4;10A1-2; see <i>T(1;2;3)v</i> ^{L8}		
*<i>Df(1)v-L9</i>	9D2;10A1-2	3	<i>ras</i> ⁻ <i>v</i> ⁻
*<i>Df(1)v-L10</i>	9D2;10A6-8	3	<i>ras</i> ⁻ <i>v</i> ⁻
<i>Df(1)v-L11</i>	9C4;10A1-2	3, 9	<i>ras</i> ⁻ - <i>l(1)10Aa</i> ⁻
*<i>Df(1)v-L12</i>	9C1;10A2-6	3	<i>ras</i> ⁻ <i>v</i> ⁻
*<i>Df(1)v-L13</i>	9B13-14;10A1-2; see <i>T(1;3)v</i> ^{L13}		
*<i>Df(1)v-L14</i>	9B10-11;10A2-6	3	<i>ras</i> ⁻ <i>v</i> ⁻
<i>Df(1)v-L15</i>	9B1-2;10A1-2	3, 7, 9	<i>HK</i> ⁻ - <i>l(1)10Aa</i> ⁻
*<i>Df(1)v-L16</i>	9F3-5;10A1-2	4	<i>ras</i> ⁺ <i>v</i> ⁻
<i>Df(1)v-M1</i>	9D3;10A1-2	9, 10	<i>ras</i> ⁻ - <i>l(1)10Aa</i> ⁻
<i>Df(1)v-M5</i>	9F13;10A1-2	9, 10	<i>v</i> ⁻ - <i>l(1)10Aa</i> ⁻
<i>Df(1)v-M6</i>	9F11-12;10A1-2	9, 10	<i>l(1)9Fg</i> ⁻ - <i>l(1)10Aa</i> ⁻
<i>Df(1)v-M7</i>	9D3;10A1-2	9, 10	<i>ras</i> ⁻ - <i>l(1)10Aa</i> ⁻
<i>Df(1)v-N48</i>	see <i>Tp(1;1)v</i> ^{N48}		
<i>Df(1)v-P5</i>	9D1-2;10A1-2	9, 10	<i>ras</i> ⁻ - <i>l(1)10Aa</i> ⁻
<i>Df(1)v-P22</i>^δ	10A1;10A2	9, 10	<i>v</i> ⁻ - <i>l(1)10Aa</i> ⁻
<i>Df(1)v-VAM19</i>	9E1;10A	5	<i>v</i> ⁻

^α 1 = Craymer and Roy, 1980, DIS 55: 200-04; 2 = Green; 3 = Lefevre, 1969, Genetics 63: 589-600; 4 = Lefevre, 1971, Genetics 67: 497-513; 5 = Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-52; 6 = Ripoll and García-Bellido, 1979, Genetics 91: 443-53; 7 = Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307; 8 = Zhimulev, Pokholkova, Bgatov, Umbetova, Solovjeva, Khudyakov, and Belyaeva, 1987, Biol. Zentrabl. 106: 699-720; 9 = Zhimulev, Belyaeva, Pokholkova, Kochneva, Fomina, Bgatov, Kudyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1982, DIS 58: 210-14; 10 = Zhimulev, Semeshin, and Belyaeva, 1981, Chromosoma 82: 9-23.

^β 10A1-2 breakpoint mapped to a restriction fragment at -2.7 to -1.3 kb (Hafen, Kasler, Edström, and Rubin, 1987, Science 236: 55-63); see molecular biology under *sev* for coordinates).

^γ Lethal in germ-line clones (García-Bellido and Robbins, 1983, Genetics 103: 235-47).

^δ γ ray induced in *T(1;Y)B149*.

Df(1)V51: Deficiency (1) Valencia

cytology: *Df(1)19E1-2;19E3-4*.
origin: X ray induced.
references: Valencia, 1970, DIS 45: 37-38.

****Df(1)vB: Deficiency (1) vermilion of Bridges***

origin: Spontaneous.
discoverer: Bridges, 16e9.
references: 1919, J. Gen. Physiol. 1: 645-56.
genetics: Deficient for *v*. Genetic map shortened 1-3 units.

Df(1)VE624

cytology: *Df(1)7E4;8B*.
discoverer: Lefevre.
references: Perrimon, Engstrom, and Mahowald, 1989,

Genetics 121: 333-52.

Df(1)VE696

cytology: *Df(1)19F1;20F.*

discoverer: Lefevre.

references: Perrimon, Engstrom, and Mahowald, 1989, Genetics 121: 333-52.

Df(1)vt: Deficiency (1) verticals

cytology: *Df(1)3C2-3;3C6-7* inferred from Young and Judd's figure 1 (1978).

references: Young and Judd, 1978, Genetics 88: 723-42.

genetics: Deficient for *rst-vt*.

Df(1)w^{m4L}rst^{3R}: see *In(1)w^{m4L}rst^{3R}*

Df(1)w^{m4L}w^{mJR}: see *In(1)w^{m4L}w^{mJR}*

Df(1)w^{ma}N: Deficiency (1) white-marbled Notch

cytology: *Df(1)3C2-3;3E2-3.*

origin: X ray induced.

references: Lefevre and Moore, 1968, Genetics 58: 557-71.

genetics: Deficient for *rst, spl, dm,* and *N*. Heterozygous females viable and fertile. Eyes "white-marbled" (like *w^{sp}*).

Df(1)w^{mJL}rst^{3R}: see *In(1)w^{mJL}rst^{3R}*

Df(1)w26d: Deficiency (1) white

cytology: Deletion of 3C2-6 + interband 3C6-7; 3C1 "fused" to 3C7, forming a thick band.

origin: *Df(1)w67k30* + *Df(1)fa-swb* recombinant.

references: Keppy and Welshons, 1977, Genetics 85: 497-506.

1980, Chromosoma 76: 191-200.

genetics: Deficient for *w, rst,* and *vt*. *fa^{swb}* phenotype absent although *fa^{swb}* can be recovered as a recombinant product from *Df(1)w26D*.

Df(1)w55j

cytology: *3C2;3C2.*

origin: Unequal recombination in *w^{a/w} a⁴* female.

references: Green, 1959, Heredity 13: 303-15.

1963, Z. Indukt. Abstamm. Vererbungsl. 94: 200-14.

Goldberg, Paro, and Gehring, EMBO J. 1: 93-98.

genetics: Homozygous viable. Deficient for *w^{sp}* and *w^e* sites.

molecular biology: Copia insertion near breakpoint.

Df(1)W61

cytology: *Df(1)10B8;10E1-2.*

genetics: Deficient for *RplI215*.

Df(1)w62g26: see Tp(1;2)w^{+62g}

Df(1)w63b: see Df(1)N63b

Df(1)w64b: see T(1;2)w^{+64b}

Df(1)w64c

cytology: *Df(1)2D6-E1;3C2-3.*

origin: X ray induced.

discoverer: Lefevre, 64c18.

references: Stewart and Merriam, 1974, Genetics 76: 301-09.

Craymer and Roy, 1980, DIS 55: 200-04.

genetics: Deficient for *pn, gt,* and *w*.

Df(1)w64d: see In(1)w^{-64d}

Df(1)w67

origin: Derived from mutable *w⁸*, except for X-ray-induced *Df(1)w67k30*.

deficiency	cytology	syn.	ref ^α	genetics	male viab.?
<i>Df(1)w67c2^β</i>	<i>3C1-2;3C2-3</i>	<i>w-1</i>	1,2,6	<i>w⁻rst⁺vt⁺</i>	+
<i>Df(1)w67c3</i>	<i>3C1-2;3C3-4</i>	<i>w-4</i>	5, 6	<i>w⁻rst⁺vt⁺</i>	+ Y
<i>Df(1)w67c9</i>	<i>3C1-2;3C2-3</i>	<i>w-2</i>	6	<i>w⁻rst⁺vt⁺</i>	+
<i>Df(1)w67c11</i>	<i>3C1-2;3C5-6</i>	<i>wrv-4</i>	6, 8	<i>w⁻rst⁺vt⁺</i>	+ Y
<i>Df(1)w67c14</i>	<i>3C1-2;3C5</i>	<i>wr-2</i>	6	<i>w⁻rst⁺vt⁺</i>	+ Y
<i>Df(1)w67c23</i>	<i>3C1-2;3C5</i>	<i>wv-1</i>	6	<i>w⁻rst⁺vt⁺</i>	+ Y
<i>Df(1)w67c23.1</i>	<i>3C1-2;3C5</i>	<i>wrv-1</i>	6	<i>w⁻rst⁺vt⁺</i>	+ Y
<i>Df(1)w67c23.2</i>	<i>3C1-2;3C6-7</i>	<i>wrv-5</i>	6	<i>w⁻rst⁻vt⁻</i>	-
<i>Df(1)w67d4</i>	<i>3C1-2;3C6-7</i>	<i>wrv-6</i>	6	<i>w⁻rst⁻vt⁻</i>	-
<i>Df(1)w67d7</i>	<i>3C1-2;3C3-4</i>	<i>w-3</i>	6	<i>w⁻rst⁺vt⁺</i>	+ Y
<i>Df(1)w67d17</i>	<i>3C1-2;3C5</i>	<i>wr-3</i>	6	<i>w⁻rst⁺vt⁺</i>	+ Y
<i>Df(1)w67d22</i>	<i>3C1-2;3C5-6</i>	<i>wrv-2</i>	6	<i>w⁻rst⁻vt⁻</i>	+ Y
<i>Df(1)w67d24</i>	<i>3C1-2;3C5-6</i>	<i>wrv-3</i>	6, 8	<i>w⁻rst⁻vt⁻</i>	+ Y
<i>Df(1)w67d38</i>	<i>3C1-2;3C5</i>	<i>wr-1</i>	6	<i>w⁻rst⁺vt⁺</i>	+ Y
<i>Df(1)w67e8^δ</i>	<i>3C1-2;3C6-7</i>	<i>wrv-7</i>	3,6,8	<i>w⁻rst⁻vt⁻</i>	-
<i>Df(1)w67k30^{δe}</i>	<i>3C1-2;3C6-7</i>	<i>wrv-X</i>	4,6,7	<i>w⁻rst⁻vt⁻</i>	-

^α 1 = Goldberg, Paro, and Gehring, 1982, EMBO J. 1: 93; 2 = Green, 1969, Genetics 61: 429-41; 3 = Kaufman, Shannon, Shen, and Judd, 1975, Genetics 79: 265-82; 4 = Lefevre, 1970, DIS 45: 32; 5 = Lefevre and Green, 1971, DIS 46: 141; 6 = Lefevre and Green, 1972, Chromosoma 36: 391-412; 7 = Ripoll and Garcia-Bellido, 1978, Genetics 91: 443-53; 8 = Welshons and Welshons, 1985, Genetics 110: 465-77.

^β Molecular biology: 3C2-3 (right or proximal) breakpoint between 2.4 and 3.5 kb to the right of the *w^a* copia insertion point (Pirrota and Bröckl, 1984, EMBO J. 3: 563-68; Pirrota, Hadfield, and Pretorius, 1983, EMBO J. 2: 927-34).

^γ Slow to emerge.

^δ Cell viable.

^e Covered by *Dp(1;3)w^{veo}*. Suppresses *fa^{swb}*. Deletes 5' half of the transcription unit immediately upstream of *N* (Kidd, Kelley, and Young, 1986, Mol. Cell Biol. 6: 3094-3108).

Df(1)w68a - Df(1)w71c

origin: X ray induced in *In(1)z^{+64b9} = In(1)3C1-2;12B8-9*.

references: Sorsa, Green, and Beermann, 1973, Nature (London) New Biol. 245: 34-37.

genetics: Male lethal.

deficiency	cytology ^α	new order ^β	genetics
<i>Df(1)w68a^γ</i>	see <i>Tp(1;3)w^{68a}</i>		
<i>Df(1)w70e^γ</i>	<i>3B5-C1;3C1-2 + 12A10-B1;12B8-9</i>	1	<i>crm⁺w⁻;g⁻</i>
<i>Df(1)w70j</i>	<i>3A10-B1;3C1-2 + 12A7-8;12B8-9</i>	2	<i>crm⁻-w⁻;g⁻</i>
<i>Df(1)w70k</i>	<i>3A;3C1-2</i> (tentative)	3	<i>crm⁻-w⁻;g⁺</i>
<i>Df(1)w70l</i>	<i>3B;3C1-2 + In(1)4C-12A</i> (tentative)	4	<i>crm⁻-w⁻;g⁺</i>
<i>Df(1)w71b</i>	<i>2D;3C1-2 + 12A10-B1;12B8-9</i>	5	<i>z⁻-w⁻;g⁻</i>
<i>Df(1)w71c</i>	faint band to right of 3C1 missing		<i>w⁻</i> ; partial suppressor of <i>z</i>

^α All deficiencies superimposed on *In(1)z^{+64b9} = In(1)3C1-2;12B8-9*.

^β 1 = 1A1-3B5|12A10-3C2|12B9-20; 3C1 + 12B1-8 missing.
2 = 1A1-3A10|12A7-3C2|12B9-20; 3B1-C1 + 12B1-8 missing.
3 = 3A-3C1 missing.
4 = 1A1-3B|12B8-12B|4C-12A|4C-3C2|12B9-20; 3B-C1 missing.
5 = 1-2D|12A10-3C2|12B9-20; 2D-3C1 + 12B1-8 missing.

^γ Deficiency heterozygous with *Df(1)w258-45* is viable (Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56).

Df(1)w78

cytology: *Df(1)3C1-2;3C2-3*; inferred from genetic data.
origin: Spontaneous in *Dp(1;1)w^aw^{sp}*, z male; simultaneous with recovery of transposon *w⁺II(78h24)*.
references: Rasmuson and Montell, 1980, DIS 55: 127. Rasmuson, Montell, Rasmuson, Svahlin, and Westerberg, 1980, Mol. Gen. Genet. 177: 567-70.
genetics: Deficient for *w* but not for *rst* or *vt*.

Df(1)w79

cytology: *Df(1)3C2-8;3C6-7* superimposed on *In(1)3C1-2;3C7-8*.
origin: Spontaneous derivative of *In(1)N^{66h26}*.
new order:
 1A – 3C1|3C7|3C8 – 20.
 3C2-6 missing.
references: Welshons and Keppy, 1981, Mol. Gen. Genet. 181: 319-24.
genetics: Deficient for *w*, *rst*, *vt*, and *N*.

Df(1)w80k1

cytology: *Df(1)3C2;3C2*.
origin: Spontaneous (presumably induced by recombination between FB elements).
references: Collins and Rubin, 1984, Nature 308: 323-27.
genetics: Mutant for *w*. Male viable and fertile.
molecular biology: Sequences within *w^c* insertion partially deleted and sequences in left copy of the *w^t* duplication absent.

Df(1)w86.8

references: Kaufman, Tasaka, and Suzuki, 1973, Genetics 75: 299-321.
genetics: Dominant suppressor of *z* and *z^a*. Deficient for *w*.

Df(1)W172

cytology: *Df(1)10A1-2;11A1-2*.
genetics: Deficient for *RpII215*.

Df(1)w258

origin: X ray induced.
discoverer: Demerec.

deficiency	cytology	ref ^α	genetics
<i>Df(1)w258-3</i>	3B2-3;3C1-2		<i>br⁺w⁻fa⁺</i>
<i>Df(1)w258-11</i>	3A2-3;3C3-5	1, 2, 4	<i>kz⁺z⁻w⁻rst⁺</i> ; embryonic lethal
<i>*Df(1)w258-14</i>	3A3-4;3C2-3	4, 5	<i>kz⁺w⁻rst⁺</i>
<i>Df(1)w258-42</i>	3A4-6;3C5-6	1, 2	<i>kz⁺l(1)3Ad⁻-rst⁻</i> ; embryonic lethal
<i>Df(1)w258-45^β</i>	3B2-3;3C2-3	1, 2, 3	<i>kz⁺l(1)3Bc⁻-w⁻rst⁺</i> ; embryonic lethal
<i>Df(1)w258-48</i>	3A9-B1;3C2-3	5	<i>kz⁺w⁻rst⁺</i>

^α 1 = Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56; 2 = Kaufman, Shannon, Shen, and Judd, 1975, Genetics 79: 265-82; 3 = Lefevre, 1968, DIS 43: 141, 165; 4 = Slizyńska, 1938, Genetics 23: 291-99; 5 = Wright, 1970, Adv. Genet. 15: 261-395.
^β Proximal member of double deficiency *Df(1)64j4 Df(1)w258-45* (Kaufman *et al.*, 1975). Germ-line clones lethal (García-Bellido and Robbins, 1983, Genetics 103: 235-47). Deficiency extends from 85-90 kb to the left to approximately 65 kb to the right of the *w^a* copia insertion point (Pirrotta, Hadfield, and Pretorius, 1983, EMBO J. 2: 927-34; Reddy, Zehring, Wheeler, Pirrotta, Hadfield, Hall, and Rosbach, 1984, Cell 38: 701-10).

Df(1)w-c: Deficiency (1) white-crimson

cytology: *Df(1)3C2;3C2*.
origin: Spontaneous (presumably induced by recombination between FB elements).
references: Collins and Rubin, 1984, Nature 308: 323-27.
genetics: Mutant for *w*. Male viable and fertile.
molecular biology: Sequences adjacent to and extending to the left of the *w^c* insertion are deleted, while sequences to the right are conserved. Restriction maps of the following six deletions have been made: *Df(1)w-c80j2*, *Df(1)w-c81b1*, *Df(1)w-c81b4*, *Df(1)w-c81b8*, *Df(1)w-c82a2*, and *Df(1)w-c82d1*.

Df(1)w-DZL

origin: In *w^{DZL/w^{DZL}}* females. Presumed to be a rearrangement generated by *w^{DZL}*.

deficiency	cytology	ref ^α	genetics
<i>Df(1)w-DZL-1</i>	3A2-4;3C1-2	1	<i>l(1)3Bf⁻-w⁻</i>
<i>Df(1)w-DZL-2^{β γ}</i>	3C1-2;3D2-3	1, 2, 3, 4	<i>w^{DZL}-N⁻</i>
<i>Df(1)w-DZL-3</i>	3B4-5;3C1-2	1	<i>l(1)3Bf⁻-w⁻</i>
<i>Df(1)w-DZL-14^{β δ}</i>	3C1-2;3C7-9	1, 2, 3	<i>w^{DZL}-N⁻</i>

^α 1 = Bingham, 1980, Cold Spring Harbor Symp. Quant. Biol. 45(2): 519-25; 2 = Bingham, Levis, and Rubin, 1981, Cell 25: 693-704; 3 = Zachar and Bingham, 1982, Cell 30: 529-41; 4 = Bingham and Zachar, Cell 40: 819-25.
^β *Df(1)w-DZL-2* begins at or very near the right-hand FB segment of the *w^{DZL}* transposon, removing sequences at or near the transposon insertion and extending to the right (away from *w*) for several hundred kilobases, leaving the *w* locus, the unique sequence, and the left-hand FB segment of the *w^{DZL}* transposon (Bingham and Zachar, 1985, Cell 40: 819-25). The left breakpoint of *Df(1)w-DZL-14* lies between coordinates +8.0 and +10.6, with 0 the site of the copia insertion in *w^a* (Levis, Bingham, and Rubin, 1982, Proc. Nat. Acad. Sci. USA 79: 564-68).
^γ Associated with *w^{DZL}*; removes sequence at/or near the *w^{DZL}*.
^δ TE insertion and extending proximally (Bingham and Zachar, 1982). Associated with *w⁺*; lacks most of *w^{DZL}* TE (Zachar and Bingham, 1982).

Df(1)w-ec: see *Tp(1;2)w-ec*

Df(1)w-m49a: see *Tp(1;3)w^{m49a}*

***Df(1)w-m53a:** see *Tp(1;2)w^{m53a}*

Df(1)w-N14a: Deficiency (1) white to Notch

cytology: *Df(1)2C8-9;3E3-4*.
origin: X ray induced.
discoverer: Lefevre.
genetics: Deficient for *usp-slc*.

Df(1)w-N71a

cytology: *Df(1)3A4-6;3C10-11*.
origin: Spontaneous in crossover experiment involving *Df(1)N-5419/w* females.
discoverer: Lefevre.
references: Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56. Craymer and Roy, 1980, DIS 55: 200-04.
genetics: Deficient for *l(1)3Ad*.

Df(1)w-NFM20

cytology: *Df(1)3C1-2;3C7-D1* (Bingham).
references: McGinnis, Farrell, and Beckendorf, 1980, Proc. Nat. Acad. Sci. USA 77: 7367-71.
genetics: Deficient for *w*, *N*, and *Sgs-4*.
molecular biology: Lacks *w* and *Sgs-4* sequences.

Df(1)w-r: Deficiency (1) white-recombinant

origin: Regular product of unequal exchange between 3C1-2 (or 3) of a w^a , w^{a2} , or w^{rdp} chromosome and the 3A or 3C-D region of a specific homologue.

deficiency	cytology	ref ^α	genetics
Df(1)w-rG	3A3-4;3C1-2	1	$z^+ w^-$ (or w) rst^+
Df(1)w-rJ1 ^β	3A1-2;3C2-3	2, 3, 4, 5	$gt^+ tko^- w^-$ (or w) rst^+ ; embryonic lethal and lethal in germ-line clones ^γ
Df(1)w-rJ2	3A8-9;3C2-3	2, 3, 4, 5	$l(1)3Ag^+ l(1)3Ah^- w^-$ (or w) rst^+ ; embryonic lethal
Df(1)w-rJ3 ^δ	3C1-2;3C12-D1	2, 3	w^- (or w) $-dm^-$ (or dm)

^α 1 = Green, 1959, Genetics 44: 1243-56; 2 = Judd, 1961, Genetics 46: 1687-97; 3 = Judd, 1964, DIS 39: 59-60; 4 = Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56; 5 = Kaufman, Shannon, Shen, and Judd, 1975, Genetics 79: 265-82.

^β Survives as w female when heterozygous with $Df(1)w^{m4L} rst^{3R}$; such females display reduced crossing over and increased nondisjunction for all pairs of homologues. Deletes *per* locus (Bargiello and Young, 1984, Proc. Nat. Acad. Sci. USA 81: 2142-46).

Molecular biology: 3A3-4 (left or distal) breakpoint localized to an approximately 4-kb restriction fragment 2-6 kb to the left of the origin of a 160-kb walk in the vicinity of z (Gunaratne, Mansukhani, Lipari, Liou, Martindale, and Goldberg, 1986, Proc. Nat. Acad. Sci. USA 83: 701-05).

^γ According to García-Bellido and Robbins, 1983, Genetics 103: 235-47.

^δ Survives as w female when heterozygous with $Df(1)w258-45$, $Df(1)w-rJ1$, or $Df(1)w-rJ2$.

Df(1)w-vco: see ***Tp(1;3)w^{vco}***

Df(1)w-Vogt

cytology: $Df(1)3A9-B1;3C3-4$.

references: Wright, 1970, Adv. Genet. 15: 261-395.

genetics: Deficient for w . Lethal phase in embryo.

Df(1)w-zh: see ***Tp(1;3)w^{zh}***

Df(1)wl-2

cytology: $Df(1)19E6;20F$.

origin: X ray induced.

references: Lifschytz and Falk, 1969, Mutat. Res. 8: 147-55.

genetics: Deficient for *lf-bb*.

Df(1)wrv: see ***Df(1)w67***

Df(1)wy2: Deficiency (1) wavy

cytology: $Df(1)11A6-12;11F2-4$.

origin: X ray induced.

discoverer: Goralski.

genetics: Deficient for *M(1)11F* and *wy*.

Df(1)wy26

cytology: $Df(1)11B17-C1;11E9-10$.

origin: X ray induced.

discoverer: Goralski.

genetics: Deficient for *wy*.

Df(1)X1

cytology: $Df(1)20D-E;20F$.

origin: X ray induced.

references: Lifschytz and Falk, 1969, Mutat. Res. 8: 147-55.

Schalet and Lefevre, 1973, Chromosoma 44: 183-200.

Rahman and Lindsley, 1981, Genetics 99: 49-64.

genetics: Deficient for *sph-bb*. Male viable and fertile with $B^S Y^+$, viable and sterile with $y^+ Ymal^+$.

Df(1)X10

cytology: $Df(1)2F5-3A1;3C3$; status of 2F6 in doubt.

origin: X ray induced.

discoverer: R. Falk.

references: Lifschytz, 1967, DIS 42: 89.

Kaufman, Shannon, Shen, and Judd, 1975, Genetics 79: 265-82.

Lifschytz and Green, 1984, EMBO J. 3: 999-1002.

genetics: Deficient for *gt-w*; covered by $w^+ Y$ (Lifschytz, 1967). Lethal in embryonic period. Used as marker for $z^- w^-$ (Lifschytz and Green, 1984).

Df(1)X12

cytology: $Df(1)2F6-3A1;3B5-3C1$; status of 2F6 in doubt.

origin: X ray induced.

discoverer: R. Falk.

references: Lifschytz, 1967, DIS 42: 89.

Kaufman, Shannon, Shen, and Judd, 1975, Genetics 79: 265-82.

Lifschytz and Green, 1984, EMBO J. 3: 999-1002.

Perrimon, Engstrom, and Mahowald, 1984, Genetics 108: 559-72.

genetics: Deficient for *phl-l(1)3Bf*. Lethal in embryonic period. Used as marker for $z^- w^-$ (Lifschytz and Green, 1984).

Df(1)X15: see ***Df(1)y-X15***

Df(1)y: Deficiency (1) yellow

deficiency	cytology	origin	ref ^α	genetics
Df(1)y1		EMS	1	$cin^+ arth^- ac^-$
Df(1)y13	1A1;1B1-3	X ray	3, 4, 5, 6	$l(1)IAc^- ac^-$; males with $y^+ Y$ fertile, with $l(1)IAc^+ Y$ sterile and <i>arth</i>
Df(1)y14	1A4-6;1A8-B1	X ray	3, 4, 5, 6	$l(1)IAc^- ac^-$; males with $y^+ Y$ sterile, with $l(1)IAc^+ Y$ sterile and <i>arth</i>
Df(1)y15	1A4-5;1A8-B1	X ray	3, 4, 5, 6	$cin^- y^-$, <i>ac</i> hypomorph; males with $y^+ Y$ fertile, with $l(1)IAc^+ Y$ fertile and <i>arth</i>
Df(1)y16		X ray	2	$y^- ac^-$; homozygous female sterile
Df(1)y18		X ray	2	$y^- ac^-$; homozygous female sterile
Df(1)y27		X ray	2	$y^- ac^-$; homozygous female sterile
Df(1)y64		EMS	1	$l(1)IAc^- y^- ac^+$

^α 1 = Padilla and Nash, 1977, Mol. Gen. Genet. 155: 171-77; 2 = Roberts, 1973, Genetics 74: s231; 3 = Roberts, 1973, DIS 50: 23; 4 = Roberts, 1974, Mutat. Res. 22: 139-44; 5 = Roberts, 1975, Genetics 80: 135-42; 6 = Schalet and Roberts, 1973, DIS 50: 23.

Df(1)y^{3PL}sc^{8R}: see ***In(1)y^{3PL}sc^{8R}***

Df(1)y^{4L}sc^{4R}: see ***In(1)y^{4L}sc^{4R}***

Df(1)y^{4L}sc^{8R}: see ***In(1)y^{4L}sc^{8R}***

Df(1)y^{4L}sc^{9R}: see ***In(1)y^{4L}sc^{9R}***

Df(1)y74 – Df(1)y75

deficiency	cytology	origin α	ref β	genetics
Df(1)y74I4.2γ	1A1;1B4	1	2, 3	<i>l(1)IAa⁻-sc⁻</i>
Df(1)y74k10.1	1A1;1B5-6	1	2, 3, 5	<i>l(1)IAa⁻-l(1)Bh⁻ l(1)Bi⁺</i>
Df(1)y74k10.3	1A1;1B5-6	1	2, 3	<i>l(1)IAa⁻-sc⁻</i>
Df(1)y74k24.1	1A1;1B9-10	1	1, 2, 3, 5	<i>l(1)IAa⁻-l(1)IBi⁻ su(s)⁺</i>
Df(1)y74k24.2	1A1;1B5-6	1	2, 3	<i>l(1)IAa⁻-sc⁻</i>
Df(1)y75a22.1	1A1;1B5-6	1	3	<i>l(1)IAa⁻-sc⁻</i>
Df(1)y75e	1A1;1B6-9	2	4	<i>l(1)IAa⁻-sc⁻</i>

α 1 = y² mutator stock of M.M. Green; 2 = TEM.
 β 1 = Craymer and Roy, 1980, DIS 55: 200-04; 2 = Garcia-Bellido, 1979, Genetics 91: 491-520; 3 = Lefevre; 4 = Lim, 1979, Genetics 93: 681-701; 5 = Voelker.
 γ Viable and strong *sc* over *Dp(1;4)sc-H* (García-Bellido, 1979).

Df(1)y-A: Deficiency (1) yellow of Alexandrov

references: Alexandrov, Ankina, and Alexandrova, 1985, DIS 61: 212-13.

deficiency	cytology	origin α	genetics
Df(1)y-A71k2	1B1;1B2	3	y ^{34C-}
Df(1)y-A71l1	1B1;1B2	3	y ⁻
Df(1)y-A72d2	1B1;1B2	3	y ⁻
Df(1)y-A74b151	1B1;1B2	2	y ⁻
Df(1)y-A74d2	1B1;1B2	3	y ⁻
Df(1)y-A74d30	1B1;1B2	2	y ⁻
Df(1)y-A74d40	1B1;1B2	2	y ⁻
Df(1)y-A74e2	1B1;1B2	2	y ⁻
Df(1)y-A74k	1B1;1B2	3	y ^{c4-}
Df(1)y-A75l1	1B1;1B2	3	y ⁻
Df(1)y-A75l3	1B1;1B2	3	y ⁻
Df(1)y-A76i	1B1;1B2	1	y ⁻
Df(1)y-A76j	1B1;1B2	1	y ^{c4-}
Df(1)y-A76k	1B1;1B2	1	y ^{c4-}
Df(1)y-A77a	1B1;1B2	1	y ⁻
Df(1)y-A78a	1B1;1B2	1	y ⁻
Df(1)y-A78dβ	1A3;1B1-2	3	lethal
Df(1)y-A79bγ	1A5-6;1B6	4	lethal
Df(1)y-A79d1β	1A3;1B1-2	3	lethal
Df(1)y-A79h2β	1A3;1B1-2	3	lethal
Df(1)y-A82c38γ	1A6;1B4	4	lethal

α 1 = actinomycin-D and γ rays; 2 = caffeine and γ rays; 3 = α rays; 4 = neutrons.
 β *Df(1)y/y⁺Y* males not viable.
 γ *Df(1)y/y⁺Y* males viable.

Df(1)y-ac: Deficiency (1) yellow-achaete

references: Biessmann and Mason, 1988, EMBO J. 7: 1081-86.

Walter, Black, Afshar, Kermabon, Wright, and Biessmann, 1990.

phenotype: Homozygous viable *y-ac* deficiency.

molecular biology: Removes entire *y* gene coding region (Biessmann and Mason, 1988).

Df(1)y-X: Deficiency (1) yellow-X ray

origin: All deficiencies X ray induced in *In(1)sc⁸* with the exception of *Df(1)y-X2rl*.

deficiency	cytology	ref α	genetics
Df(1)y-X2	deletion of X tip + part of 20F	3, 4	<i>l(1)IAc⁻-ac⁻, bb⁻</i> ; (170 rRNA sequences); covered by y ⁺ Y; <i>Df/bb</i> females <i>bb^l</i>
Df(1)y-X2rlβ	deletion of X tip + more of 20F	1, 4	<i>l(1)IAc⁻-ac⁻ bb^l</i>
Df(1)y-X5	deletion of X tip + 20A4-5 through 20F; capped by 2L(?)	5, 6	<i>l(1)IAc⁻-ac⁻, fog⁻-bb⁻</i>

deficiency	cytology	ref α	genetics
Df(1)y-X7	deletion of X tip + 20C-D through 20F	3	<i>l(1)IAc⁻-ac⁻, l(1)20Cb⁻-bb⁻</i> ; males viable with <i>B^SYy⁺</i> and <i>y⁺Ymal⁺</i>
Df(1)y-X9	deletion of X tip + 20B-C through 20F	3	<i>l(1)IAc⁻-ac⁻, fog⁻-bb⁻</i> ; males lethal with <i>B^SYy⁺</i> , viable with <i>y⁺Ymal⁺</i>
Df(1)y-X15	deletion of X tip + 20D-E through 20F + inversion with 8C break capped by 4 γ	2, 3, 5	<i>l(1)IAc⁻-ac⁻, sph⁻-bb⁻</i> ; males with <i>y⁺Y</i> lethal, with <i>B^SYy⁺</i> viable and fertile, with <i>y⁺Ymal⁺</i> viable and sterile.
Df(1)y-X16	deletion of X tip + 20B-C through 20F	3	<i>l(1)IAc⁻-ac⁻, fog⁻-bb⁻</i> ; males with <i>B^SYy⁺</i> lethal, with <i>y⁺Ymal⁺</i> viable

α 1 = Procunier and Tartof, 1978, Genetics 88: 67-79; 2 = Rahman and Lindsley, 1981, Genetics 99: 49-64; 3 = Schalet, 1968, DIS 43: 64; 4 = Schalet, 1969, Genetics 63: 133-53; 5 = Schalet and Lefevre, 1973, Chromosoma 44: 184-200; 6 = Zusman and Wieschaus, 1987, Genetics 115: 725-36.
 β Derived from *Df(1)y-X2* by reduction in number of rDNA genes under the influence of *Ybb⁻*.
 γ new order: 102-101|8C-20D|8C-1B|20F.

Df(1)yI-1

cytology: *Df(1)I9E6;20A1-2*.

origin: X ray induced.

references: Lifschytz and Falk, 1969, Mutat. Res. 8: 147-55.

genetics: Deficient for *lf-eo*.

Df(1)yT: Deficiency (1) yellow Terminal

cytology: Putative terminal deficiencies with breaks between 1B1 and 1F.

origin: Irradiation of *mu-2* females (Mason, Strobel, and Green, 1984, Proc. Nat. Acad. Sci. USA 81: 6090-94; Biessman and Mason, 1988, EMBO J. 7: 1081-86).

synonym: *Df(1)y^{RT}* (Sherald and Voelker, 1985, DIS 61: 155).

deficiency α	ref β	genetics	mol biol γ
Df(1)yT1a-276	2, 3	y ²⁻ y ¹⁺	71.5-69.5
Df(1)yT1a-394	2, 3	y ²⁻ y ¹⁺	72.3-71.6
Df(1)yT1a-473	2, 3	y ²⁻ y ¹⁺	72.3-71.6
Df(1)yT1a-653	2, 3	y ²⁻ y ¹⁺	72.3-71.6
Df(1)yT1a-741	2, 3	y ²⁻ y ¹⁺	71.5-69.5
Df(1)yT1b-81	2, 3	y ¹⁻ ac ⁺	67.8-67.7
Df(1)yT1b-94	2, 3	y ¹⁻ ac ⁺	67.7-67.2
Df(1)yT1b-96	2	y ¹⁻ ac ⁺	
Df(1)yT1b-303	2, 3	y ¹⁻ ac ⁺	67.2-66.3
Df(1)yT1b-488	2, 3	y ¹⁻ ac ⁺	64.2-63.2
Df(1)yT1b-497	2, 3	y ¹⁻ ac ⁺	65.6-65.3
Df(1)yT1b-625	2, 3	y ¹⁻ ac ⁺	65.3-64.8
Df(1)yT1b-627	2	y ¹⁻ ac ⁺	
Df(1)yT1b-628	2, 3	y ¹⁻ ac ⁺	67.2-66.3
Df(1)yT1b-685	2, 3	y ¹⁻ ac ⁺	45.4-43.9
Df(1)yT1b-688	2	y ¹⁻ ac ⁺	
Df(1)yT1b-689	2, 3	y ¹⁻ ac ⁺	67.2-66.3
Df(1)yT1b-733	2	y ¹⁻ ac ⁺	
Df(1)yT2-26	2	ac ⁻ sc ⁺	
Df(1)yT2-29	2, 3	ac ⁻ sc ⁺	42.3-39.5
Df(1)yT2-83	2, 3	ac ⁻ sc ⁺	50.2-49.4

CHROMOSOMES - DEFICIENCIES

deficiency α	ref β	genetics	mol biol γ	deficiency α	ref β	genetics	mol biol γ
<i>Df(1)yT2-84</i>	2,3	ac ⁻ sc ⁺	53.2-52.4	<i>Df(1)yT4-25</i>	2	<i>l(1)lBb⁻ l(1)lBc⁺</i>	
<i>Df(1)yT2-85</i>	2,3	ac ⁻ sc ⁺	46.0-45.4	<i>Df(1)yT6-151</i>	2	svr ⁻ elav ⁺	
<i>Df(1)yT2-86</i>	2,3	ac ⁻ sc ⁺	53.2-52.4	<i>Df(1)yT6-188</i>	2	svr ⁻ elav ⁺	
<i>Df(1)yT2-89</i>	2,3	ac ⁻ sc ⁺	46.0-45.4	<i>Df(1)yT6-244</i>	2	svr ⁻ elav ⁺	
<i>Df(1)yT2-90</i>	2,3	ac ⁻ sc ⁺	52.4-50.2	<i>Df(1)yT6-291</i>	2	svr ⁻ elav ⁺	
<i>Df(1)yT2-92</i>	2,3	ac ⁻ sc ⁺	50.6-49.4	<i>Df(1)yT6-437</i>	2	svr ⁻ elav ⁺	
<i>Df(1)yT2-93</i>	2,3	ac ⁻ sc ⁺	49.4-47.9	<i>Df(1)yT6-450</i>	2	svr ⁻ elav ⁺	
<i>Df(1)yT2-95</i>	2,3	ac ⁻ sc ⁺	54.3-53.2	<i>Df(1)yT6-506</i>	2	svr ⁻ elav ⁺	
<i>Df(1)yT2-97</i>	2,3	ac ⁻ sc ⁺	55.8-54.3	<i>Df(1)yT6-522</i>	2	svr ⁻ elav ⁺	
<i>Df(1)yT2-99</i>	2,3	ac ⁻ sc ⁺		<i>Df(1)yT6-544</i>	2	svr ⁻ elav ⁺	
<i>Df(1)yT2-103</i>	2,3	ac ⁻ sc ⁺	42.1-41.5	<i>Df(1)yT7-104</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-150</i>	2,3	ac ⁻ sc ⁺	33.8-33.4	<i>Df(1)yT7-107</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-165</i>	2,3	ac ⁻ sc ⁺	45.4-43.9	<i>Df(1)yT7-108</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-195</i>	2,3	ac ⁻ sc ⁺	62.4-61.8	<i>Df(1)yT7-109</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-212</i>	2,3	ac ⁻ sc ⁺	56.8-56.3	<i>Df(1)yT7-114</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-214</i>	2,3	ac ⁻ sc ⁺	33.4-32.7	<i>Df(1)yT7-115</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-216</i>	2,3	ac ⁻ sc ⁺	52.4-50.6	<i>Df(1)yT7-129</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-233</i>	2,3	ac ⁻ sc ⁺	38.4-36.3	<i>Df(1)yT7-130</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-242</i>	2,3	ac ⁻ sc ⁺	61.2-60.3	<i>Df(1)yT7-155</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-252</i>	2,3	ac ⁻ sc ⁺	42.1-41.5	<i>Df(1)yT7-157</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-288</i>	2,3	ac ⁻ sc ⁺	46.0-45.4	<i>Df(1)yT7-178</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-293</i>	2,3	ac ⁻ sc ⁺	57.6-56.6	<i>Df(1)yT7-191</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-304</i>	2,3	ac ⁻ sc ⁺	44.4-43.9	<i>Df(1)yT7-194</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-331</i>	2,3	ac ⁻ sc ⁺	61.2-60.3	<i>Df(1)yT7-208</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-336</i>	2,3	ac ⁻ sc ⁺	47.9-46.8	<i>Df(1)yT7-218</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-343</i>	2,3	ac ⁻ sc ⁺	36.6-33.8	<i>Df(1)yT7-229</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-351</i>	2,3	ac ⁻ sc ⁺	47.9-46.8	<i>Df(1)yT7-234</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-356</i>	2,3	ac ⁻ sc ⁺	52.4-50.6	<i>Df(1)yT7-250</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-398</i>	2,3	ac ⁻ sc ⁺	58.9-58.2	<i>Df(1)yT7-263</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-438</i>	2,3	ac ⁻ sc ⁺	50.6-49.4	<i>Df(1)yT7-284</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-503</i>	2,3	ac ⁻ sc ⁺	54.3-53.2	<i>Df(1)yT7-292</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-520</i>	2,3	ac ⁻ sc ⁺	52.4-50.6	<i>Df(1)yT7-296</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-524</i>	2,3	ac ⁻ sc ⁺	42.3-42.1	<i>Df(1)yT7-301</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-551</i>	2,3	ac ⁻ sc ⁺	42.3-39.5	<i>Df(1)yT7-311</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-558</i>	2,3	ac ⁻ sc ⁺	58.9-58.2	<i>Df(1)yT7-329</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-589</i>	2,3	ac ⁻ sc ⁺	62.9-62.4	<i>Df(1)yT7-341</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-590</i>	2,3	ac ⁻ sc ⁺	52.4-50.6	<i>Df(1)yT7-348</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-618</i>	2,3	ac ⁻ sc ⁺	39.5-37.1	<i>Df(1)yT7-358</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-623</i>	2,3	ac ⁻ sc ⁺	45.4-44.4; 36.7-34.4	<i>Df(1)yT7-366</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-624</i>	2,3	ac ⁻ sc ⁺	54.3-53.2	<i>Df(1)yT7-371</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-626</i>	2,3	ac ⁻ sc ⁺	59.7-58.9	<i>Df(1)yT7-376</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-629</i>	2,3	ac ⁻ sc ⁺	42.3-39.5	<i>Df(1)yT7-377</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-630</i>	2,3	ac ⁻ sc ⁺	45.4-43.9	<i>Df(1)yT7-400</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-631</i>	2,3	ac ⁻ sc ⁺	37.1-35.5	<i>Df(1)yT7-401</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-633</i>	2,3	ac ⁻ sc ⁺	62.4-61.8	<i>Df(1)yT7-406</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-634</i>	2	ac ⁻ sc ⁺		<i>Df(1)yT7-406</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-637</i>	2,3	ac ⁻ sc ⁺	49.4-47.9	<i>Df(1)yT7-435</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-643</i>	2,3	ac ⁻ sc ⁺	46.0-45.4	<i>Df(1)yT7-446</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-650</i>	2,3	ac ⁻ sc ⁺	37.1-35.5	<i>Df(1)yT7-448</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-655</i>	2,3	ac ⁻ sc ⁺	55.8-54.3	<i>Df(1)yT7-458</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-659</i>	2,3	ac ⁻ sc ⁺	62.4-61.8	<i>Df(1)yT7-518</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-691</i>	2,3	ac ⁻ sc ⁺	59.9-59.7	<i>Df(1)yT7-525</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-695</i>	2	ac ⁻ sc ⁺		<i>Df(1)yT7-532</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-696</i>	2,3	ac ⁻ sc ⁺	60.2-59.9; 53.2-52.4; 47.9-45.8	<i>Df(1)yT7-547</i>	2	elav ⁻ vnd ⁺	
<i>Df(1)yT2-701</i>	2,3	ac ⁻ sc ⁺	53.2-52.4	<i>Df(1)yT8-9</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
<i>Df(1)yT2-730</i>	2,3	ac ⁻ sc ⁺	49.4-47.9	<i>Df(1)yT8-12</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
<i>Df(1)yT2-734</i>	2	ac ⁻ sc ⁺		<i>Df(1)yT8-18</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
<i>Df(1)yT2-735</i>	2	ac ⁻ sc ⁺		<i>Df(1)yT8-101</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
<i>Df(1)yT2-738</i>	2	ac ⁻ sc ⁺		<i>Df(1)yT8-124</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
<i>Df(1)yT2-739</i>	2,3	ac ⁻ sc ⁺	60.3-59.9	<i>Df(1)yT8-128</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
<i>Df(1)yT2-742</i>	2,3	ac ⁻ sc ⁺	47.9-46.8	<i>Df(1)yT8-143</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
<i>Df(1)yT2-744</i>	2	ac ⁻ sc ⁺		<i>Df(1)yT8-148</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
<i>Df(1)yT2-748</i>	2,3	ac ⁻ sc ⁺	38.4-36.3	<i>Df(1)yT8-158</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
<i>Df(1)yT2-749</i>	2	ac ⁻ sc ⁺		<i>Df(1)yT8-184</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
<i>Df(1)yT3-15</i>	2	sc ⁻ <i>l(1)lBb⁺</i>		<i>Df(1)yT8-185</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
<i>Df(1)yT3-22</i>	2	sc ⁻ <i>l(1)lBb⁺</i>		<i>Df(1)yT8-197</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
<i>Df(1)yT3-24</i>	2	sc ⁻ <i>l(1)lBb⁺</i>		<i>Df(1)yT8-206</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
<i>Df(1)yT3-55</i>	2	sc ⁻ <i>l(1)lBb⁺</i>		<i>Df(1)yT8-241</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
<i>Df(1)yT4-8</i>	2	<i>l(1)lBb⁻ l(1)lBc⁺</i>		<i>Df(1)yT8-271</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
<i>Df(1)yT4-19</i>	2	<i>l(1)lBb⁻ l(1)lBc⁺</i>		<i>Df(1)yT8-361</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
<i>Df(1)yT4-20</i>	2	<i>l(1)lBb⁻ l(1)lBc⁺</i>		<i>Df(1)yT8-362</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
				<i>Df(1)yT8-368</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	
				<i>Df(1)yT8-369</i>	2	vnd ⁻ <i>l(1)Bg⁺</i>	

THE GENOME OF *DROSOPHILA MELANOGASTER*

deficiency α	ref β	genetics	mol biol γ
<i>Df(1)yT8-385</i>	2	<i>vnd</i> ⁻ <i>l(1)Bg</i> ⁺	
<i>Df(1)yT8-409</i>	2	<i>vnd</i> ⁻ <i>l(1)Bg</i> ⁺	
<i>Df(1)yT8-420</i>	2	<i>vnd</i> ⁻ <i>l(1)Bg</i> ⁺	
<i>Df(1)yT8-500</i>	2	<i>vnd</i> ⁻ <i>l(1)Bg</i> ⁺	
<i>Df(1)yT8-530</i>	2	<i>vnd</i> ⁻ <i>l(1)Bg</i> ⁺	
<i>Df(1)yT9-7</i>	2, 4	<i>l(1)lBg</i> ⁻ <i>l(1)Bh</i> ⁺	
<i>Df(1)yT9-21</i>	2, 4	<i>l(1)lBg</i> ⁻ <i>l(1)Bh</i> ⁺	
<i>Df(1)yT9-220</i>	2, 4	<i>l(1)lBg</i> ⁻ <i>l(1)Bh</i> ⁺	
<i>Df(1)yT9-275</i>	2, 4	<i>l(1)lBg</i> ⁻ <i>l(1)Bh</i> ⁺	
<i>Df(1)yT10-102</i>	2, 4	<i>l(1)lBh</i> ⁻ <i>l(1)Bi</i> ⁺	
<i>Df(1)yT10-106</i>	2, 4	<i>l(1)lBh</i> ⁻ <i>l(1)Bi</i> ⁺	
<i>Df(1)yT10-581</i>	2, 4	<i>l(1)lBh</i> ⁻ <i>l(1)Bi</i> ⁺	
<i>Df(1)yT10-665</i>	2, 4	<i>l(1)lBh</i> ⁻ <i>l(1)Bi</i> ⁺	
<i>Df(1)yT10-673</i>	2, 4	<i>l(1)lBh</i> ⁻ <i>l(1)Bi</i> ⁺	
<i>Df(1)yT10-677</i>	2, 4	<i>l(1)lBh</i> ⁻ <i>l(1)Bi</i> ⁺	
<i>Df(1)yT12-173</i>	2, 4	<i>M(1)Bl</i> ⁻ <i>su(s)</i> ⁺	
<i>Df(1)yT12-187</i>	2, 4	<i>M(1)Bl</i> ⁻ <i>su(s)</i> ⁺	
<i>Df(1)yT12-200</i>	2, 4	<i>M(1)Bl</i> ⁻ <i>su(s)</i> ⁺	
<i>Df(1)yT12-206</i>	2, 4	<i>M(1)Bl</i> ⁻ <i>su(s)</i> ⁺	
<i>Df(1)yT12-246</i>	2, 4	<i>M(1)Bl</i> ⁻ <i>su(s)</i> ⁺	
<i>Df(1)yT12-667</i>	2, 4	<i>M(1)Bl</i> ⁻ <i>su(s)</i> ⁺	
<i>Df(1)yT13-423</i> ^{δ}	2, 4	<i>su(s)</i> ⁻ <i>l(1)lBk</i> ⁺	
<i>Df(1)yT13-464</i> ^{δ}	2, 4	<i>su(s)</i> ⁻ <i>l(1)lBk</i> ⁺	
<i>Df(1)yT14-546</i>	2, 4	<i>l(1)lBk</i> ⁻ <i>l(1)lCa</i> ⁺	
<i>Df(1)yT14-576</i>	2, 4	<i>l(1)lBk</i> ⁻ <i>l(1)lCa</i> ⁺	
<i>Df(1)yT15-5</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-6</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-28</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-56</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-105</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-158b</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-161</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-171</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-177</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-183</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-189</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-219</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-222</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-270</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-274</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-286</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-300</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-318</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-325</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-327</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-334</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-364</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-365</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-383</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-390</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-392</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-405</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-408</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-410</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-412</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-433</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-449</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-460</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-468</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-502</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-509</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-511</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-517</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-533</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-543</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-548</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-552</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-564</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-568</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-575</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	

deficiency α	ref β	genetics	mol biol γ
<i>Df(1)yT15-586</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-597</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT15-599</i>	2, 4	<i>l(1)Ca</i> ⁻ <i>mul</i> ⁺	
<i>Df(1)yT16-14</i>	2, 4	<i>mul</i> ⁻ <i>tw</i> ⁺	
<i>Df(1)yT16-411</i>	2, 4	<i>mul</i> ⁻ <i>tw</i> ⁺	
<i>Df(1)yT16-499</i>	2, 4	<i>mul</i> ⁻ <i>tw</i> ⁺	
<i>Df(1)yT16-534</i>	2, 4	<i>mul</i> ⁻ <i>tw</i> ⁺	
<i>Df(1)yT16-554</i>	2, 4	<i>mul</i> ⁻ <i>tw</i> ⁺	
<i>Df(1)yT17-570</i>	2, 4	<i>tw</i> ⁻ <i>l(1)lDa</i> ⁺	
<i>Df(1)yT17-600</i>	2, 4	<i>tw</i> ⁻ <i>l(1)lDa</i> ⁺	
<i>Df(1)yT18-319</i>	2, 4	<i>l(1)Da</i> ⁻ <i>brc</i> ⁺	
<i>Df(1)yT19-16</i>	2, 4	<i>brc</i> ⁻ <i>l(1)lDc</i> ⁺	
<i>Df(1)yT19-253</i>	2, 4	<i>brc</i> ⁻ <i>l(1)lDc</i> ⁺	

α The number preceding the hyphen designates the intergenic interval in which the break has occurred, and the numbers following the hyphen represent independent occurrences.

β 1 = Campos, Grossman, and White, 1985, *J. Neurogenet.* 2: 197-218; 2 = Mason, Voelker, Rosen, Campos, White, and Lim, 1986, *DIS* 63: 164-65; 3 = Ruiz-Gómez and Modolell, 1988, *Genes Dev.* 1: 1238-46; 4 = Voelker.

γ DNA coordinates from Caramolino, Ruiz-Gómez, Guerrero, Campuzano, and Modolell, 1982, *EMBO J.* 1: 1185-91 and Campuzano, Carramolino, Cabrera; Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, *Cell* 40: 3227-38. Origin (coordinate 0) is the distal EcoRI site within the *sc*^{S2} molecular deficiency ("+" values to the left, "-" values to the right). *Df(1)yT2-623* and *Df(1)yT2-696* have multiple breakpoints. The unstable chromosome ends lose approximately 75 base pairs per generation (Biessman and Mason, 1988, *EMBO J.* 7: 1081-86). The presented data represent the extents of the deficiencies as of 1985.

δ Broken within *su(s)*.

Df(1)z: Deficiency (1) zeste

deficiency	cytology	origin	ref α	genetics β
<i>Df(1)z</i>	3A2-3;3A3-4		2	<i>z</i> ⁻
<i>Df(1)z⁺6</i>	3A6;3A7		1	<i>z</i> ⁺
<i>Df(1)z⁺33</i>	3C(partial)		2	<i>z</i> ⁺
<i>Df(1)z⁺45</i>	3C1;3C3		1	<i>z</i> ⁺
<i>Df(1)z⁺46</i>	3C3-4;3C5-6		2	<i>z</i> ⁺
<i>*Df(1)z1</i>	2C2-3;3E2-3	X ray	1	<i>pn</i> ⁻ <i>w</i> ⁻
<i>*Df(1)z2</i>	2D4-5;3C3-4	X ray	1	<i>pn</i> ⁻ <i>w</i> ⁻
<i>*Df(1)z3</i>	2C5-6;3B2-3	X ray	1	<i>pn</i> ⁻ <i>z</i> ⁻
<i>*Df(1)z4</i>	2C5-6;3A9-B1	X ray	1	<i>pn</i> ⁻ <i>z</i> ⁻
<i>*Df(1)z5</i>	3A1-3;3A4-6	X ray	1	<i>z</i> ⁻
<i>*Df(1)z6</i>	3A6-8;3C10-11	X ray	1	<i>z</i> ⁺ <i>w</i> ⁻
<i>Df(1)z-deb3</i>	2E-F;3A3	DEB	3	<i>gt</i> ⁻ <i>z</i> ⁻
<i>Df(1)z-deb42</i>		DEB	3	<i>gt</i> ⁻ <i>l(1)3Ac</i>
<i>Df(1)z-deb54</i>		DEB	3	<i>gt</i> ⁻ <i>l(1)3Ag</i>
<i>Df(1)z-deb59</i>		DEB	3	<i>gt</i> ⁻ <i>l(1)3Ac</i>
<i>Df(1)z-deb62</i>		DEB	3	<i>gt</i> ⁻ <i>l(1)3Bc</i>
<i>Df(1)z-deb65</i>		DEB	3	<i>gt</i> ⁻ <i>crm</i> ⁻
<i>Df(1)z-deb72</i>		DEB	3	<i>gt</i> ⁻ <i>crm</i> ⁻
<i>Df(1)z-deb81</i>		DEB	3	<i>gt</i> ⁻ <i>l(1)3Ba</i>
<i>Df(1)z-deb85</i>		DEB	3	<i>gt</i> ⁻ <i>l(1)3Ba</i>
<i>Df(1)z-deb87</i>		DEB	3	<i>gt</i> ⁻ <i>z</i> ⁻
<i>Df(1)z-deb92</i>	2E-F;3A3	DEB	3	<i>gt</i> ⁻ <i>z</i> ⁻
<i>Df(1)z-deb311</i>		DEB	3	<i>gt</i> ⁻ <i>l(1)3Ag</i>
<i>Df(1)z-deb511</i>		DEB	3	<i>gt</i> ⁻ <i>l(1)3Bf</i>
<i>Df(1)z-deb910</i>		DEB	3	<i>gt</i> ⁻ <i>z</i> ⁻

α 1 = Gans, 1953, *Bull. Biol. Fr. Belg. Supp.* 38: 1-90; 2 = Gelbart, 1971, Ph.D. Thesis, Univ. of Wisconsin; 3 = Goldberg, Colvin, and Mellin, 1989, *Genetics* 123: 145-55.

β Experiments of Goldberg *et al.*, 1989, show that females without a *zeste* locus are fertile and of normal or near normal fecundity.

Df(1)ZWD5

cytology: Band at 3C in *Df(1)ZWD5/+* salivaries wedge-shaped indicating small deficiency.

origin: Irradiation of z^1 males.

references: Smolik-Utlaut and Gelbart, 1987, Genetics 116: 285-98.

genetics: Homozygous lethal. Notch wing in *Df(1)ZWD5/+*.

Df(Y)bb: see *Ybb*⁻

Df(YL) and Df(YS)

origin: X ray or γ ray induced in $B^S Y y^+$ (Lindsley *et al.*, 1972; Hazelrigg *et al.*, 1982) or $y^+ Y$ (Brosseau, 1960); γ ray induced in $y^+ Y$ (Schwartz).

genetics: Recovered as male-sterile Y chromosomes; presence of deficiencies established by cytological examination of prophase and metaphase figures. Missing fertility factors in deficiencies of Brosseau, Hazelrigg *et al.*, and Schwartz indicated.

deficiency	cytology α	ref β	genetics
<i>Df(YL)A5</i>		3	$kl-3^- kl-2^+ kl-1^-$
<i>Df(YL)A10</i>		3	$kl-3^- kl-1^-$
<i>Df(YL)A11</i>		3	$B^S kl-5^- kl-3^-$
<i>Df(YL)A17</i>		3	$kl-3^- kl-1^-$
<i>Df(YL)A29^Y</i>		3	$kl-3^-$
<i>Df(YL)A32</i>		3	$kl-5^- kl-3^-$
<i>Df(YL)A33^Y</i>			
<i>Df(YL)A39</i>		3	$kl-5^- kl-3^-$
<i>Df(YL)A49^{\delta}</i>		3	$kl-3^- kl-2^-$
<i>Df(YL)A78</i>	$B^S Xh;h10;87B$	2, 5	
<i>Df(YL)A80</i>	$B^S Xh;h7 + h21;35B$	2, 5	
<i>Df(YL)A109</i>	$B^S Xh;h13;83E$	2, 5	
<i>Df(YL)A111</i>	$h5;h11 + h14;28D$	2, 5	
<i>Df(YL)A158</i>	$h3;h14;63A$	2, 5	
<i>Df(YL)B2^{\epsilon}</i>	$h3;h8$	1, 2	$kl-5^- kl-3^-$
<i>Df(YL)B4</i>		3	$kl-5^- kl-3^-$
<i>Df(YL)B12</i>		3	$B^S kl-5^- kl-3^-$
<i>Df(YL)B14</i>		3	$kl-5^- kl-1^-$
<i>Df(YL)B172</i>	$B^S Xh;h3;95A$	2, 5	
<i>Df(YL)B225</i>	$h3;h10 + B^S Xh;73D$	2, 5	
<i>Df(YL)B236</i>	$B^S Xh;h16;25D-E$	2, 5	
<i>Df(YL)D211</i>	$B^S Xhj;h3;26A$	2, 5	
<i>Df(YL)G8</i>	$h3;h8;85F$	2, 5	
<i>Df(YL)G18</i>		4	$kl-5^- kl-1^-$
<i>Df(YL)G19</i>		4	$kl-3^- kl-2^-$
<i>Df(YL)G20</i>		4	$kl-3^- kl-2^-$
<i>Df(YL)G21</i>		4	$kl-5^- kl-2^-$
<i>Df(YL)G22</i>		4	$kl-5^- kl-2^-$
<i>Df(YL)G23</i>		4	$kl-5^- kl-2^-$
<i>Df(YL)G24</i>		4	$kl-5^- kl-2^-$
<i>Df(YL)G26</i>		4	$kl-5^- kl-3^-$
<i>Df(YL)G27</i>		4	$kl-5^- kl-3^-$
<i>Df(YL)G28</i>		4	$kl-5^- kl-3^-$
<i>Df(YL)G29</i>		4	$kl-5^- kl-3^-$
<i>Df(YL)G30</i>		4	$kl-5^- kl-3^-$
<i>Df(YL)G48</i>	$h3;h7 + Xhy^+;88C$	2, 5	
<i>Df(YL)H52</i>	$h3;h16 + Xhy^+;27E$	2, 5	
<i>Df(YL)H121</i>	$h3;h13 + h16;26B$	2, 5	
<i>Df(YL)H143</i>	$B^S Xh;h14;59F$	2, 5	
<i>Df(YL)J59</i>	$h3;h16 + Xhy^+;43A$	2, 5	
<i>Df(YL)J96</i>	$B^S Xh;h8 + h16;25A$	2, 5	
<i>Df(YL)J112</i>	$B^S Xhj;h3 + h25D;71B-C$	2, 5	
<i>Df(YL)J118</i>	$h3;h10 + h21;22D$	2, 5	
<i>Df(YL)J136</i>	$h3;h9 + B^S Xh;26F$	2, 5	
<i>Df(YL)J154</i>	$h1-2;h3 + h25D;63A$	2, 5	
<i>Df(YL)L132</i>	$B^S Xh;h16 + Xhy^+;83C-D$	2, 5	
<i>Df(YL)R136</i>	$h3;h7;22D$	2, 5	
<i>Df(YL)R158</i>	$B^S Xhj;h10 + Xhy^+;32F$	2, 5	
<i>Df(YL)S5</i>	$h13;h14-15$	2	$kl-1^-$
<i>Df(YL)S6</i>	$h13;h17$	2	$kl-1^-$
<i>Df(YL)S7</i>	$h3;h8-9$	2	$kl-5^- kl-3^-$
<i>Df(YL)S8</i>	$y^+ Xh;h8$	2	$kl-5^- kl-3^-$

deficiency	cytology α	ref β	genetics
<i>Df(YL)S9</i>	$h3;h11$	2	$kl-5^- kl-2^-$
<i>Df(YL)S10</i>	$y^+ Xh;h11$	2	$kl-5^- kl-2^-$
<i>Df(YL)S11</i>	$h3;h11$	2	$kl-5^- kl-2^-$
<i>Df(YL)S13^Y</i>	$h3;h9$	2	$kl-5^- kl-3^-$
<i>Df(YL)S14^Y</i>	$y^+ Xh;h14$	2	$kl-5^- kl-1^-$
<i>Df(YL)S21^{\zeta}</i>	$y^+ Xh;h2$	2	$kl-5^-$
<i>Df(YS)A6</i>		3	$ks-1^- ks-2^-$
<i>Df(YS)A29^Y</i>		3	$ks-1^-$
<i>Df(YS)A33^Y</i>		3	$ks-1^-$
<i>Df(YS)B4</i>	$h21;h25$	1, 2	$ks-1^- ks-2^-$
<i>Df(YS)B13^{\eta}</i>		3	$ks-1^- ks-2^-$
<i>Df(YS)G4</i>		4	$ks-1^- ks-2^-$
<i>Df(YS)G5</i>		4	$ks-1^- ks-2^-$
<i>Df(YS)S12</i>	$h18;h25$	2	$ks-1^- ks-2^-$
<i>Df(YS)S13^Y</i>	$h22;h25$	2	$ks-1^- ks-2^-$
<i>Df(YS)S14^Y</i>	$h19;h25$	2	$ks-1^- ks-2^-$

α Many Y deficiencies are also Y-autosome translocations. Three-break translocations that have lost a segment between two Y chromosome breakpoints are listed with three breakpoints, the last one being the autosomal breakpoint of the translocation (for example, *Df(YL)A78=B^SXh;h10;87B*), whereas deficiencies that are independent of the Y-autosome-translocation breakpoints are listed with two deficiency breakpoints plus two translocation breakpoints when known (for example, *Df(YL)A80=B^SXh;h7 + h21;35B*). New orders of these rearrangements are provided with description of the like-designated Y-autosome translocations. Deficiencies unassociated with translocations are listed with two breakpoints (for example, *Df(YL)B2=h3;h8*).

β 1 = Brosseau, 1960, Genetics 45: 257-74; 2 = Gatti and Pimpinelli, 1983, Chromosoma 83: 349-73; 3 = Hazelrigg, Fornili, and Kaufman, 1982, Chromosoma 87: 535-59; 4 = Kennison; 5 = Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethman, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84.

γ Occurs in chromosome carrying two noncontiguous deletions, one in YL and the other in YS (for example, *Df(YL)A29* and *Df(YS)A29*).

δ Also see *In(YL)A49*.

ϵ Also see *In(YL)B2*.

ζ Also see *T(Y;3)S21*.

η Also see *T(Y;2)B13*.

Df(YS)bb: see *Ybb*⁻

Df(2)M-33a: see *Df(2R)M60E*

Df(2L)061b

cytology: *Df(2L)38F7;39A6*.

origin: X ray induced.

references: Siegel, 1981, Genetics 98: 505-27.

Df(2L)1: see *Df(2L)TW1*

Df(2L)2: see *Df(2L)TW2*

Df(2L)3: see *Df(2L)TW3*

Df(2L)9: see *Df(2L)TW9*

Df(2L)12: see *Df(2L)TW12*

Df(2L)30A;C

cytology: *Df(2L)30A;30C* (Laverly).

references: Schüpbach and Wieschaus, 1989, Genetics 121: 101-17.

genetics: Deficient for *mat(2)cellQC13*, *mat(2)cellRH36*, *rem*.

Df(2L)50: see *Df(2L)TW50*

Df(2L)64j

cytology: *Df(2L)34D1-2;35B9 - C1*.

origin: X ray induced.

discoverer: E.H. Grell, 1964.

references: Grell, Jacobson, and Murphy, 1968, *Annals N.Y. Acad. Sci.* 151: 441-55.
 Nash, 1970, *Genetics* 64: 471-79.
 Woodruff and Ashburner, 1979, *Genetics* 92: 117-32.
 Gubb, Shelton, Roote, McGill, and Ashburner, 1984, *Chromosoma* 91: 54-64.
 Gubb, Roote, Harrington, McGill, Durrant, Shelton, and Ashburner, 1985, *Chromosoma* 92: 116-23.
 Lasko and Ashburner, 1988, *Nature (London)* 335: 611-17.
genetics: Deficient for *l(2)34Db-ck* but not *M(2)30A*, *vasa* or *M(2)36F*. Suppresses *H*.
molecular biology: Left breakpoint 13.5 - 17.6 kb distal to the 0 point of the 35B-C walk of Lasko and Ashburner ("+" values to right, "-" values to left).

Df(2L)65: see *Df(2L)TW65*
Df(2L)68: see *Df(2L)TW68*
Df(2L)75c: see ***In(2L)75c***
Df(2L)81: see *Df(2L)L138D*
Df(2L)84: see *Df(2L)TW84*
Df(2L)119: see *Df(2L)TW119*
Df(2L)130: see *Df(2L)TW130*
Df(2L)137: see *Df(2L)TW137*
Df(2L)150: see *Df(2L)TW150*
Df(2L)158: see *Df(2L)TW158*
Df(2L)161: see *Df(2L)TW161*
Df(2L)201: see *Df(2L)TW201*
Df(2L)202: see *Df(2L)TW202*
Df(2L)203: see *Df(2L)TW203*

Df(2L)282
 cytology: *Df(2L)25F2-3;25F4-26A1*.

Df(2L)330: see *Df(2L)TW330*

Df(2L)429.9: see ***T(2;3)429.9***

Df(2L)2802
 cytology: *Df(2L)25F2-3;25F4-26A1*.
 origin: Induced by ethyl methanesulfonate.
 references: Kotarski, Pickert, and MacIntyre, *Genetics* 105: 371-86.
 genetics: Includes break of *T(Y;2)G105*. Deficient for *l(2)25Fb* and *l(2)25Fd*.

Df(2L)50075a: see *Df(2L)Gpdh75*

Df(2L)50078a: see *Df(2L)Gpdh78*

Df(2L)A: Deficiency (2L) Aaron
 origin: X ray induced; selected as ADH null on 1-penten-3-ol.
 discoverer: Aaron.

deficiency	cytology	ref ^α	genetics
<i>Df(2L)A47</i>	34E1;35B2	1, 2, 4	<i>wb⁻osp⁻</i>
<i>Df(2L)A48</i>	35B2-3;35D5-7	1, 2, 3, 4, 6	<i>osp⁻-lace⁻</i>
<i>Df(2L)A63</i> ^γ	not visible	2, 3, 4	<i>osp⁻-Adh⁻</i>
<i>Df(2L)A72</i>	35B2-3;35B7-8	1, 2, 3; 5, 6	<i>osp⁻-l(2)35Cb⁻</i>
<i>Df(2L)A76</i> ^δ		1	<i>Adh⁻</i>

deficiency	cytology	ref ^α	genetics
<i>Df(2L)A167</i> ^δ		1	<i>Adh⁻</i>
<i>Df(2L)A178</i> ^γ ^ε	35B1-2;35B1-2	2, 3, 4, 5	<i>noc⁻-Adh⁻</i>
<i>Df(2L)A215</i>	35A3;35B7-8	1, 2	<i>wb⁻-rd</i>
<i>Df(2L)A217</i>	34F5;35B3	1, 2	<i>l(2)34Fd⁻-Adh⁻</i>
<i>Df(2L)A220</i>	35B1-2;35B9	2	<i>osp⁻-l(2)35Cb⁻</i>
<i>Df(2L)A245</i>	35A4;35B2	2, 3	<i>l(2)35Ba⁻-Adh⁻</i>
<i>Df(2L)A246</i>	34F4;35D3-4	1, 2	<i>wb⁻-l(2)35Dg⁻</i>
<i>Df(2L)A260</i>	35B1-2;35B1-2	2, 3, 4	<i>l(2)35Ba⁻-Adh⁻</i>
<i>Df(2L)A263</i>	34E5-F1;35C3-9	2	<i>l(2)34Fa⁻-l(2)35Cd⁻</i>
<i>Df(2L)A264</i>	35B1-3;35B8-9	2, 5	<i>osp⁻-l(2)35Cb⁻</i>
<i>Df(2L)A266</i>	35B2-3;35B2-3	2	<i>l(2)35Ba⁻-Adh⁻</i>
<i>Df(2L)A267</i> ^β	35B2-3;35B10-C1	2, 3	<i>noc⁻-vasa⁻</i>
<i>Df(2L)A376</i> ^ζ	34E3;35C4-5	1, 2, 3	<i>rk⁻-dgl⁻</i>
<i>Df(2L)A377</i>	34F1-4;35D5-7	1, 2	<i>wb⁻-l(2)35Dd⁻</i>
<i>Df(2L)A379</i> ^η ^θ	35B1-3;35B1-3 + <i>In(2LR)35B1-3</i> ; 40-41;57A8-10	2	<i>noc⁻-Adh⁻</i>
<i>Df(2L)A385</i> ^δ		1	
<i>Df(2L)A400</i> ^l	35A1-4;35B10	1, 2, 3	<i>pu⁻-l(2)35Cb⁻</i>
<i>Df(2L)A445</i> ^θ	not visible	2	<i>noc⁻-Adh⁻</i>
<i>Df(2L)A446</i>	see <i>Ip(2;2)A446</i>		

^α 1 = Aaron, 1979, *Mutat. Res.* 63: 127-37; 2 = Ashburner, Aaron, and Tsubota, 1982, *Genetics* 102: 421-35; 3 = Ashburner, Detwiler, Tsubota, and Woodruff, 1983, *Genetics* 104: 405-31; 4 = Gubb, Roote, Harrington, McGill, Durrant, Shelton, and Ashburner, 1985, *Chromosoma* 92: 116-23; 5 = Gubb, Roote, McGill, Shelton, and Ashburner, 1986, *Genetics* 112: 551-75; 6 = Gubb, Shelton, Roote, McGill, and Ashburner, 1984, *Chromosoma* 91: 54-64.
^β Right breakpoint mapped to the DNA at about 2.5 kb in the 35B-C walk of Lasko and Ashburner, 1988, *Nature (London)* 335: 611-17 ("+" values to right, "-" values to left).
^γ *Df(2L)A63* induced simultaneously with, but separable from, *T(2;3)CA1* and *T(2;3)CA2*; *Df(2L)A178* induced simultaneously with, but separable from, *T(2;3)CA3*.
^δ Lethal with *Df(2L)A47* and *Df(2L)A48*.
^ε Semilethal with *noc*. Males and females homozygous viable and fertile with strong *noc* and *osp* phenotypes.
^ζ Lethal with *Df(2L)b75* but viable and *rk* with *Df(2L)b-L*. Another reference: Simpson, 1983, *Genetics* 105: 615-32.
^η new order: 21 - 35B1-3|57A8-10 - 40-41|35B1-3 - 40-41|57A8-10 - 60.
^θ Males and females homozygous viable and fertile.
^l Induced simultaneously with, but separable from, *T(2;3;4)CA4*.

Df(2L)A1: see *Df(2L)pr-A1*

Df(2L)A16: see *Df(2L)pr-A16*

Df(2L)a17
 cytology: *Df(2L)35C;36A*.
 discoverer: Roth.

references: Wustmann, Szidonya, Taubert, and Reuter, 1989, *Mol. Gen. Genet.* 217: 520-27.

Df(2L)A20: see *Df(2L)pr-A20*

Df(2L)a25
 cytology: *Df(2L)35C;36A*.
 discoverer: Roth.

references: Wustmann, Sidonya, Taubert, and Reuter, 1989, *Mol. Gen. Genet.* 217: 520-27.

Df(2L)A244
 cytology: Not visible cytologically but believed, on the basis of complementation data, to be a deficiency in 2L.
 origin: X ray induced.
 references: Siegel, 1981, *Genetics* 98: 505-27.

Df(2L)Adh1: see *Df(2L)nNxFl*

Df(2L)Adh2: see *Df(2L)nNxFl*

Df(2L)ade2

origin: Induced by hybrid dysgenesis.
references: Tiong, Keizer, Nash, Bleskan, and Patterson, 1989, *Biochem. Genet.* 27: 333-48.
genetics: Deficient for *ade2*.

deficiency	cytology
Df(2L)ade2-1	<i>Df(2L)25F;26B5-6</i>
Df(2L)ade2-2	<i>Df(2L)25F2-3;26DE</i>
Df(2L)ade2-3	<i>Df(2L)26A;26B5-6</i>

Df(2L)Adh78

cytology: *Df(2L)35B1;35D5-7*.
origin: Spontaneous in dysgenic strain T-007; selected as ADH null on 1-penten-3-ol.
discoverer: Bencze.
synonym: *Df(2L)17813*.
references: Ashburner, Tsubota, and Woodruff, 1982, *Genetics* 102: 401-20.
genetics: Deficient for *l(2)35Ba-l(2)35Dg*.

Df(2L)AdhnBR41

origin: Induced by ENU.
discoverer: Lee.
genetics: Deficient for *l(2)34Fa-Adh*.

Df(2L)al: Deficiency (2L) aristaless

cytology: *Df(2L)21B8-C1;21C8-D1*.
origin: X ray induced.
discoverer: E. B. Lewis, 1940.
references: 1945, *Genetics* 30: 147-51.
 Korochkina and Golubovsky, 1978, *DIS* 53: 197-200.
 Nüsslein-Volhard, Wieschaus and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 267-82.

genetics: Deficient for *al*, *ex*, *ush* and *ds* but not for *lgl*, *net*, or *S*. Homozygotes lethal, heterozygotes Minute. Heterozygotes with *In(2LR)al*⁸ survive poorly; escapers have reduced aristaes, broad thoraces, arched wings, incomplete veins, and large eyes (Koroshkina and Golubovsky, 1978).

Df(2L)ARR1

cytology: *Df(2L)35A3-4;35B9-C1*.
new order:
 21 – 35A3-4|YLY|y⁺ ac⁺|YS|35B9-C1 – 60.
origin: X-ray-induced reconstitution of chromosome 2 from *Y^P2^D* of *T(Y;2)A80* and *2^PY^D* of *T(Y;2)R15*.
references: Woodruff and Ashburner, 1979, *Genetics* 92: 117-32.
 Gubb, Shelton, Roote, McGill, and Ashburner, 1984, *Chromosoma* 91: 54-64.
genetics: Deficient for *el-Su(H)*; includes *Adh*. Carries *y⁺ac⁺* from *T(Y;2)R15*.

Df(2L)ast: Deficiency (2L) asteroid

origin: γ ray induced.
references: Roberts, Brock, Rudden, and Evans-Roberts, 1985, *Genetics* 109: 145-56.
genetics: Deficient for *ast*.

deficiency	cytology
Df(2L)ast1	<i>21C7-8;23A1-2</i>
Df(2L)ast2	<i>21D1-2;22B2-3</i>
Df(2L)ast3	<i>21D1-2;21E1-2</i>
Df(2L)ast4	<i>21D1-2;21E1-2</i>
Df(2L)ast5	<i>21E1-2;21F3-22A1</i>
Df(2L)ast6	<i>21E1-2;21E2-3</i>
Df(2L)ast10 ^α	<i>21D2-3;22A1-2</i>

^α From complementation data.

Df(2L)b: Deficiency (2L) black

deficiency	cytology	origin	ref ^α	genetics
Df(2L)b23	34E;35C	MRF ^β	16	b ⁻
Df(2L)b24	34D;35C	MRF ^β	16	b ⁻
Df(2L)b36f	see T(2;3)dp			
Df(2L)b74c6	34D1-2;34E1-2	caffeine + γ ray	1, 2	l(2)34Db ⁻ -l(2)35Bb ⁻
Df(2L)b75^γ	34D4-6;34E5-6	X ray	4, 6, 15	l(2)34Db ⁻ -l(2)34Fa ⁻
Df(2L)b77c	34D2-4;34F4-35A1-2	actinomycin-D + γ ray	1, 2	b ⁻ -rk ⁻ -pu ⁺
Df(2L)b78j	34D3;35A4	X ray	1, 2	l(2)34Db ⁻ -l(2)34De ⁻
Df(2L)b79a3	34D2-4;34D8-E1	neutrons	1, 2	b ⁻ j ⁺
Df(2L)b79b3	34D1-2;34E5-6	neutrons	1, 2	l(2)34Db ⁻ -l(2)34Fa
Df(2L)b79b4	34D2-4;34E6-F1	neutrons	1, 2	l(2)34Db ⁻ -wb ⁻
Df(2L)b79b8	34D2-4;34F4-35A1	neutrons		b ⁻ -rk ⁻ j ⁺
Df(2L)b80c1	34D3;34D8-E1	γ ray	3	l(2)34Db ⁻ -l(2)34Eb ⁻
Df(2L)b80e3	34C4;35A4	γ ray	5, 7	l(2)34Da ⁻ -l(2)35Aa ⁻
Df(2L)b80e4	34C4;35D1-2	γ ray	12, 14	l(2)34Da ⁻ -sna ⁻
Df(2L)b80f1	34C3;34E3-6	γ ray	2	l(2)34Da ⁻ -l(2)34Eb ⁻
Df(2L)b80k	34D2-4;35B10-C1	γ ray	1, 2	l(2)34Da ⁻ -j ⁻
Df(2L)b80l	34D2-4;34E2-4	neutrons	1, 2	l(2)34Db ⁻ -l(2)34Eb ⁻
Df(2L)b81a1^δ	34D3;35B1-2	γ ray	7	l(2)34Db ⁻ -noc ⁻
Df(2L)b81f1	34D2-4;35A3-4	neutrons	1, 2	l(2)34Db ⁻ -l(2)34Dd ⁻
Df(2L)b81f1A	34D1-2;35F1-2			l(2)34Db ⁻ -l(2)34Fa
Df(2L)b81f2	34D2-4;35B4-5	neutrons	1, 2	l(2)34Db ⁻ -l(2)35Bb ⁻
Df(2L)b81h1		γ ray (on CyO)	8	l(2)34Db ⁻ -b ⁻
Df(2L)b81i1^ε	34C1;35B4	γ ray	7	l(2)34Db ⁻ -Adh ⁻
Df(2L)b81i4	34D3;34D8			l(2)34Db ⁻ -l(2)34Ea ⁻
Df(2L)b82a1		γ ray	11	l(2)34Ca ⁻ -l(2)34Eb ⁻
Df(2L)b82a2	34D1-2;34E1-2	γ ray	11	l(2)34Ca ⁻ -j ⁻
Df(2L)b82a3^ζ		HD	13	l(2)34Dd ⁻ -b ⁻
Df(2L)b83d29a	35D2-4;35E2-6	neutrons	1, 2	l(2)34Db ⁻ -pu ⁻
Df(2L)b83i1	34D2-4;34E2-5	neutrons	1, 2	l(2)34Db ⁻ -l(2)34Dg ⁻
Df(2L)b83i2	see T(2;3)b ⁸³ⁱ²			
Df(2L)b84a1	34D3;35B1-2	γ ray	9	l(2)34Db ⁻ -Adh ⁻
Df(2L)b84a2	34C3;35A4	γ ray	9	l(2)34Da ⁻ -el ⁻
Df(2L)b84a3	34D3;35B3-4	γ ray	9	l(2)34Db ⁻ -l(2)35Bf ⁻
Df(2L)b84a4	34D3;35B1-2	γ ray	9	l(2)34Db ⁻ -noc ⁻
Df(2L)b84a5	34D3;35B2-3	γ ray	9	l(2)34Db ⁻ -noc ⁻
Df(2L)b84a6	34D3;35B6-7	γ ray	9	l(2)34Db ⁻ -l(2)35Bf ⁻
Df(2L)b84a7	34C1-2;35B1-2	γ ray	9	l(2)34Da ⁻ -Adh ⁻
Df(2L)b84a8	34D3;35B1-2	γ ray	9	l(2)34Db ⁻ -noc ⁻
Df(2L)b84a9	not visible	γ ray	9	l(2)34Dc ⁻ -l(2)34Ea ⁻
Df(2L)b84h1	34D3;35A4	γ ray	7	l(2)34Dc ⁻ -l(2)35Aa ⁻
Df(2L)b84h14	34D2-4;34F4-35A1-2	X ray	1, 2	l(2)34Db ⁻ -l(2)34Fa ⁻
Df(2L)b84h50	34D2-4;35C1-3	X ray	1, 2	l(2)34Da ⁻ -pu ⁻
Df(2L)b85b1	34D2-4;34E2-6	γ ray	1, 2	b ⁻ j ⁺
Df(2L)b85b2	34D2-4;34D8-E1	γ ray	1, 2	b ⁻ j ⁺
Df(2L)b85c1	34D2-4;34F4-35A1	neutrons	1, 2	b ⁻ -j ⁻ -rk ⁺
Df(2L)b85f1	34C4-5;34F5	γ ray	2, 9	l(2)34Db ⁻ -wb ⁻
Df(2L)b85f1A	34D2-4;34F3-4	γ ray	1, 2	b ⁻ -rk ⁻ -pu ⁺
Df(2L)b85f2	34D2-4;34E6-F1			
Df(2L)b87E25	34B12-C1;35B10-C1			
Df(2L)b-L^η	34D3;34E3-5	spont	3-5, 11	l(2)34Db ⁻ -rk ⁻
Df(2L)b85f2	34D2-4;34E6-F1			l(2)34Da ⁻ -rk ⁻
Df(2L)b87E25	34B12-C1;35B10-35C1			b ⁻

^α 1 = Alexandrov, 1984, DIS 60: 45-47; 2 = Alexandrov and Alexandrova, 1986, DIS 63: 159-61; 3 = Angel; 4 = Ashburner, Aaron, and Tsubota, 1982, Genetics 102: 421-35; 5 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91; 6 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95; 7 = Ashburner, Tsubota, and Woodruff, 1982, Genetics 102: 401-20; 8 = Durrant; 9 = Harrington; 10 = G. Johnson; 11 = Lutkin and Baker, 1979, Mutat. Res. 61: 221-27; 12 = McGill; 13 = Simpson, 1983, Genetics 105: 615-32; 14 = Struhl; 15 = Woodruff and Ashburner, 1979, Genetics 92: 117-32; 16 = Yannopoulos, Stamatis, Zacharopoulou, and Pelecanos, 1981, Mutat. Res. 83: 383-93.

^β Induced by Male Recombination Factor 23.5 from natural population in Greece.

^γ Viable over Df(2L)fn1, Df(2L)75c, and Df(2L)C75RL but lethal over Df(2L)W and Df(2L)A376.

^δ Viable and noc over Df(2L)A446. Right breakpoint mapped to the DNA (Chia, Karp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706).

^ε Induced on In(2LR)Gla.

^ζ Synonym: b^{CP}.

^η In mei-W68 male. Df(2L)b-L/Df(2L)A376 viable but rk.

Df(2L)B5: see Df(2L)pr-B5

Df(2L)B10-1: see T(Y;2;3)B10-1

Df(2L)B80: see T(Y;2)B80

Df(2L)B119

cytology: {Df(2L)39D-E} (not cytologically detectable).

origin: γ ray induced.

references: Siegel, 1981, Genetics 98: 505-27.

Df(2L)B209: see **T(Y;2)B209**

Df(2L)C

cytology: Deficient for most of the 2L heterochromatin. (40;40).
origin: γ -ray-induced reconstitution of chromosome 2 by detachment of compound second chromosomes.
synonym: *Df(2L)PR31*.
references: Hilliker and Holm, 1975, Genetics 81: 705-21.
genetics: Deficient for *lt*.

Df(2L)C'

cytology: Deficient for much of the 2L heterochromatin and duplicated for much of the 2R heterochromatin.
origin: γ -ray-induced reconstitution of chromosome 2 by detachment of compound second chromosomes.
synonym: *Df(2L)PR31*.
references: Hilliker and Holm, 1975, Genetics 81: 705-21. Hilliker, 1976, Genetics 83: 765-82.
genetics: Deficient for 28 EMS-induced lethal alleles in seven complementation groups (Hilliker, 1976); duplicated for *rl*⁺ with an *rl*⁺ locus on each side of the centromere (Hilliker, 1981, DIS 56: 72-74).

Df(2L)C2: see **Df(2L)L138D-C2**

Df(2L)C15: see **Df(2L)L138D-C15**

Df(2L)C75RL: see **In(2L)75c^LC158.1^R**

***Df(2L)C263: Deficiency (2L) Crossover suppressor**

cytology: *Df(2L)25F;26F*.
origin: Associated with *In(2L)C263*.

Df(2L)cl: Deficiency (2L) clot

origin: X ray induced.
genetics: Deficient for *cl* and surrounding lethal complementation groups from *l(2)25Eb* to *l(2)25Eg* (Kotarski *et al.*, 1983).

deficiency	cytology	ref ^α	genetics
Df(2L)cl1	25D2-4;25F2-4	1, 3, 5, 6	<i>tkv</i> ⁻ - <i>E(var)</i> ⁻
Df(2L)cl2	25D2-4;25F2-4	1, 3, 5, 6	<i>tkv</i> ⁻ - <i>E(var)</i> ⁻
Df(2L)cl7^β	25D5-6;26A7	1, 2, 4, 6, 7	<i>l(2)25De</i> ⁻
Df(2L)cl-h1^γ	25D4-5;25F1-2	5, 6	<i>tkv</i> ⁻ - <i>mid</i> ⁻
Df(2L)cl-h2^γ	25D6;25E4-5	5, 6	<i>l(2)25De</i> ⁻ - <i>l(2)26Aa</i> ⁻
Df(2L)cl-h3^γ	25D2-3;26B2-5	6	<i>tkv</i> ⁻
Df(2L)cl-h4^γ	25D6;25F3-4	5, 6	<i>l(2)25Ea</i> ⁻ - <i>l(2)25Ef</i> ⁻

^α 1 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95; 2 = Knipple and MacIntyre, 1984, Mol. Gen. Genet. 198: 75-83; 3 = Kotarski, Pickert, and MacIntyre, 1983, Genetics 105: 371-86; 4 = Racine, Langley, and Voelker, 1980, Environ. Mutagen. 2: 167-77; 5 = Semeshin and Szidonya, 1985, DIS 61: 148-54; 6 = Szidonya and Reuter, 1988, DIS 67: 77-79 and 1988, Genet. Res. 51: 197-208; 7 = Velissariou and Ashburner, 1980, Chromosoma 77: 13-27.
^β Enhances variegation of *In(1)y^{m4}* and *In(1)y^{3P}* (Locke, Kotarski, and Tartof, 1988, Genetics 120: 181-98).
^γ Isolated over *Dp(2;2)B3 = Dp(2;2)23E2-3;26E2-F1*.

Df(2L)Cy^Lt^R: see **In(2L)Cy^Lt^R**

***Df(2L)d: Deficiency (2L) dachs**

origin: Spontaneous in *d* stock.
discoverer: Bridges, 15j6.
synonym: *d*¹: *dachs-lethal*.
references: Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 277.
genetics: Homozygous lethal. Gives decreased crossing

over in *d-b* region.

Df(2L)D

cytology: Deficient for more than one-half of the proximal 2L heterochromatin.
origin: γ -ray-induced reconstitution of chromosome 2 by detachment of compound second chromosomes.
references: Hilliker and Holm, 1975, Genetics 81: 705-21. Hilliker, 1976, Genetics 83: 765-82.
genetics: Deficient for four EMS-induced semilethal loci proximal to *lt*.
other information: Six such deficiencies recovered.

Df(2L)D'

cytology: Deficient for more than one-quarter of the proximal 2L heterochromatin.
origin: γ -ray-induced reconstitution of chromosome 2 by detachment of compound second chromosomes.
references: Hilliker and Holm, 1975, Genetics 81: 705-21. Hilliker, 1976, Genetics 83: 765-82
genetics: Deficient for two EMS-induced lethal loci proximal to *lt*. Adults very small and late in hatching.
other information: Eleven such deficiencies recovered.

Df(2L)D6: see **T(Y;2)D6**

Df(2L)dl2034: see **Df(2L)H20**

Df(2L)dl-H20: see **Df(2L)H20**

Df(2L)do1

cytology: *Df(2L)35B1-2;35D2*.
origin: Diepoxycetane induced.
discoverer: Detwiler.
references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91. Ashburner, Tsubota, and Woodruff, 1982, Genetics 102: 401-20.
genetics: Deficient for *l(2)34Fd-sna*. Includes *el*, *noc*, *Adh*, *osp*, and *rd*.

Df(2L)dp: Deficiency (2L) dumpy

deficiency	cytology	origin	ref ^α	genetics ^β
Df(2L)dp	25A3-4;25B	spont ^γ	1	<i>dp</i> ⁻
Df(2L)dp79b	22A2-3;22D5-E1			
Df(2L)dp-h19^δ	24F1-2;24F6-7	X ray	2, 3	<i>l(2)24Eb</i> ⁻ - <i>dp</i> ⁻
Df(2L)dp-h24^δ	not visible	X ray	3	<i>M(2)25A</i> ⁻ - <i>dp</i> ⁻
Df(2L)dp-h25^δ	24E4;25B2	X ray	2, 3	<i>dw24F</i> ⁻ - <i>l(2)25Ac</i> ⁻
Df(2L)dp-h28^δ	24D8;24F6-7	X ray	2, 3	<i>l(2)24Ea</i> ⁻ - <i>dp</i> ⁻

^α 1 = Lutkin and Baker, 1979, Mutat. Res. 61: 221-27; 2 = Semeshin and Szidonya, 1985, DIS 61: 148-54. 3 = Szidonya and Reuter, 1988, DIS 67: 77-79 and 1988, Genet. Res. 51: 197-208;
^β Includes complementation groups localized in the deleted regions by Szidonya (unpubl.).
^γ In *mei-W68* male.
^δ Isolated over *Dp(2;2)B3 = Dp(2;2)23E2-3;26E2-F1*.

Df(2L)dp-cl: Deficiency (2L) dumpy-clot

origin: X ray induced.
references: Szidonya and Reuter, 1988a, DIS 67: 77-79. 1988b, Genet. Res. 51: 197-208.
genetics: Deficient for *dp-cl*; includes haplo-insufficient genes.

deficiency ^α	cytology
Df(2L)dp-cl-h1	24F7-25A1;26A2-3
Df(2L)dp-cl-h2	24E4-F1;26B9-C1

deficiency ^α	cytology
Df(2L)dpp-cl-h3	24F7-25A1;25E2-4

^α Isolated over *Dp(2;2)B3 = Dp(2;2)23E2-3;26E2-F1*.

Df(2L)dpp: Deficiency (2L) decapentaplegic genetics: Deficient for *dpp*.

deficiency	cytology	origin	discov ^α	ref ^β	dpp class
Df(2L)dpp14	22E4-F2;22F3-23A1	X ray	7	2, 3	<i>d-V</i>
Df(2L)dpp19 ^γ	22F2-3;22F3-4	X ray	7	2, 3	<i>d-III</i>
Df(2L)dpp32 ^δ	22E3-F1;23A1-2	X ray	2	1	<i>Hin-Df</i>
Df(2L)dpp33	22F1-2;23A1-2	HD	3	3	<i>d-V</i>
Df(2L)dpp34 ^δ	22E2-3;23A2-4	X ray	2	1	<i>Hin-Df</i>
Df(2L)dpp38 ^δ	22A1-2;22F3	γ ray	4	1	<i>Hin-Df</i>
Df(2L)dpp39 ^δ	22A1-3;23A2-4	γ ray	4	1	<i>Hin-Df</i>
Df(2L)dpp40 ^δ	22E1-2;23A1-2	γ ray	4	1	<i>Hin-Df</i>
Df(2L)dpp43 ^δ	22A;23A3-B1	γ ray	4	1	<i>Hin-Df</i>
Df(2L)dpp46 ^δ	22F1-2;22F2-3	γ ray	4	1	<i>Hin-Df</i>
Df(2L)dpp51 ^δ	21F;23B1-2	γ ray	4	1	<i>Hin-Df</i>
Df(2L)dpp53 ^δ	22A1-2;23A3-7	γ ray	4	1	<i>Hin-Df</i>
Df(2L)dpp55	22F;23A	HD	8	1	<i>d-III</i>
Df(2L)dpp59	22A;23A	γ ray	5	1	<i>Hin-Df</i>
Df(2L)dpp62	22B;22F	γ ray	5	1	<i>Hin-Df</i>
Df(2L)dpp79	22F1-2;22F4-23A1	γ ray	6	1	<i>d-V</i>
Df(2L)dpp84	22B;23A	γ ray	1	1	<i>Hin-Df</i>
Df(2L)dpp85	22E1-2;23A	γ ray	1	1	<i>Hin-Df</i>

^α 1 = Blackman; 2 = Gelbart; 3 = Hoffmann; 4 = Irish; 5 = L. Posakony; 6 = Segal; 7 = Spencer; 8 = St. Johnston.
^β 1 = Gelbart; 2 = Segal and Gelbart, 1985, Genetics 109: 119-43; 3 = Spencer, Hoffmann, and Gelbart, 1982, Cell 28: 451-61.
^γ Partially complements *dpp4*; resulting phenotype enhanced by *z* alleles (Gelbart and Wu, 1982, Genetics 102: 179-89).
^δ Synonym: *Df(2L)dpp^{Hin32}*, etc. (Irish and Gelbart, 1987, Genes and Development 1: 868-79).
^ε Induced along with *T(2;3)Irish = T(2;3)55D-E;98F*.

Df(2L)dpp21: see **T(2;3)dpp²¹**

Df(2L)DS

origin: Induced by triethylenemelamine.

references: Sinclair, Moore, and Grigliatti, 1980, Genetics 94: s96.

Moore, Sinclair, and Grigliatti, 1981, Genetics 105: 327-44.

deficiency	cytology	genetics
Df(2L)DS5	39A1-2;39E7-F1	<i>pr⁻-His⁻</i>
Df(2L)DS6	38F5;39E7-F1	<i>crc⁻-His⁻</i>
Df(2L)DS8	39A6-7;39D2-3	<i>crc⁻</i>
Df(2L)DS9	39A1-2;39B2-3	<i>crc⁺ His⁺</i>

Df(2L)DTD2: see **Tp(2;1)DTD2**

Df(2L)DTD33: see **Tp(2;3)DTD33**

Df(2L)DTD48

cytology: *Df(2L)22E2-4;22F4-23A1*.

origin: X ray induced.

references: Spencer, Hoffmann, and Gelbart, 1982, Cell 28: 451-61.

genetics: Deficient for the entire *dpp* locus.

Df(2L)E: see **T(2;3)E**

Df(2L)E53

cytology: Not visibly deficient.

origin: Induced by ethyl methanesulfonate.

references: T.R.F. Wright, 1983.

genetics: Deficient for *l(2)36Fc* and *msl-1*, but not for

l(2)36Fb, *l(2)36Fd*, or *l(2)36Fe*. Lethal over *Df(2L)TW3*, *Df(2L)E71*, *Df(2L)M36F-S6*, and *Df(2L)M36F-S5*; viable over *Df(2L)TW119*.

Df(2L)E55

cytology: *Df(2L)37D2-E1;37F5-38A1*.

origin: Induced by ethyl methanesulfonate.

references: Wright, Hodgetts, and Sherald, 1976, Genetics 84: 267-85.

Brittnacher and Ganetzky, 1983, Genetics 103: 659-73.

Nüsslein-Volhard, Wieschaus and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

genetics: Deficient for *ref(2)P* and *spi*. Lethal over *Df(2L)TW330*, *Df(2L)VA15*, *Df(2L)VA20*, *Df(2L)VA22*, *Df(2L)VA23*, *Df(2L)VA25*, *Df(2L)TW9*, *Df(2L)TW12*, *l(2)37Da*, *l(2)37Db*, *l(2)37Fa*, *l(2)37Fb*, *l(2)37Fc*, and *l(2)37Fd*. Viable, but shows non-transparent wing phenotype over *Df(2L)TW150*, *Df(2L)TW84*, and *Df(2L)OD16*.

Df(2L)E66: Deficiency (2L)

Enhancer of variegation

cytology: *Df(2L)25F2-3;26A1-B1*.

origin: *P*-element induced mutagenesis.

references: Locke, Kotarski, and Tartof, 1988, Genetics 120: 181-98.

genetics: Enhances position-effect variegation of *In(1)w^{m4}*. Recessive lethal.

Df(2L)E71

cytology: *Df(2L)36F2-6;37C6-D1*.

origin: Induced by ethyl methanesulfonate.

references: Wright, Hodgetts, and Sherald, 1976, Genetics 84: 267-85.

Belote and Lucchesi, 1980, Genetics 96: 165-86.

Wright, Beermann, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, Chromosoma 83: 45-58.

genetics: Deficient for *msl-1 - fs(2)TW1* (Wright, 1983) but not for *dl* or *l(2)37Da*. Lethal over *Df(2L)M36F*, *Df(2L)E53*, *l(2)36Fc*, *l(2)36Fd*, *l(2)36Fe*, and *Df(2L)SD^{rv77}*.

Df(2L)E110

cytology: *Df(2L)25F3-26A1;26D3-11*.

origin: *P*-element induced mutagenesis.

references: Locke, Kotarski, and Tartof, 1988, Genetics 120: 181-98.

genetics: Enhances position-effect variegation of *In(1)w^{m4}*. Recessive lethal.

Df(2L)E(SD): Deficiency (2L) Enhancer of Segregation Distorter

origin: γ ray induced in *SD-5* chromosome.

references: Ganetzky, 1977, Genetics 86: 321-55.

genetics: Deficient for *l(2)40Fa - l(2)40Ff*. Partial revertant of *Sd*.

deficiency	cytology	synonym	male fecundity
Df(2L)E(SD)1 ^α	40A-B;40E	<i>Df(2L)SD^{Rev}-1</i>	intermediate
Df(2L)E(SD)3 ^β	40A-B;40F	<i>Df(2L)SD^{Rev}-3</i>	low
Df(2L)E(SD)36	40A-B;40F	<i>Df(2L)SD^{Rev}-36</i>	low

^α Involved in translocation of 3R tip to base of 2R [*T(2;3)E(SD)1*].
^β Carries *In(2L)E(SD) = In(2L)29E;30A*.

Df(2L)ed1: Deficiency (2L) echinoid

cytology: *Df(2L)24A3-4;24D3-4*.
discoverer: Szidonya.
synonym: *Df(2L)ed-Sz1*.
references: Reuter and Szidonya, 1983, *Chromosoma* 88: 277-85.
 Szidonya and Reuter, 1988a, *DIS* 67: 77-79.
 1988b, *Genet. Res.* 51: 197-208.
genetics: Includes *ed* but not *M(2)24D* or *M(2)24F*.

Df(2L)ed2

cytology: *Df(2L)24A3-4;24D3-4*.
discoverer: Szidonya.
synonym: *Df(2L)ed-Sz2*.
references: Reuter and Szidonya, 1983, *Chromosoma* 88: 277-85.
genetics: Includes *ed* but not *M(2)24D* or *M(2)24F*.

Df(2L)ed-dp: Deficiency (2L) echinoid to dumpy

cytology: *Df(2L)24C3-5;25A2-3*.
origin: X ray induced.
references: Szidonya and Reuter, 1988a, *DIS* 67: 77-79.
 1988b, *Genet. Res.* 51: 197-208.
genetics: Deficient for *ed-dp*; includes *haplo*-insufficient genes. Isolated over *Dp(2;2)B3 = Dp(2;2)23E2-3;26E2-F1*.

Df(2L)el: Deficiency (2L) elbow

deficiency	cytology	origin	ref ^α	genetics
<i>Df(2L)el15</i>	<i>35B1-2;35C5</i>	γ ray	5	<i>l(2)34Fc⁻-l(2)35Cb⁻</i>
<i>Df(2L)el16</i>	not visible	γ ray	5	<i>el⁻-noc⁻</i>
<i>Df(2L)el17</i>	<i>34F1-2;35A4</i>	γ ray	5	<i>wb⁻-Adh⁻</i>
<i>Df(2L)el18</i>	see <i>ln(2L)el18</i>			
<i>Df(2L)el20</i>	<i>34F4;35C5</i>	EMS	5	<i>wb⁻-l(2)35Da⁻</i>
<i>Df(2L)el28</i>		γ ray		<i>el⁻-Su(H)⁻</i>
<i>Df(2L)el77</i>	<i>35A1-3;35B3</i>	γ ray	3, 6	<i>wb⁻-Adh⁻</i>
<i>Df(2L)el80f1</i>	<i>34E3;35D7</i>	γ ray	1, 2, 4	<i>rk⁻-lace⁻</i>
<i>Df(2L)el80i1</i>	<i>34E1-2;35C2-3</i>	γ ray	1	<i>l(2)34Eb⁻-l(2)35Cb⁻</i>
<i>Df(2L)el81i1</i>	<i>34F5;35B1-2</i>	γ ray	1	<i>wb⁻-Adh⁻</i>
<i>Df(2L)el82f1</i>	<i>34E1-2;35C3-5</i>	EMS	1	<i>l(2)34Eb⁻-l(2)35Ce⁻</i>

^α 1 = Angel; 2 = Ashburner, Detwiler, Tsubota, and Woodruff, 1983, *Genetics* 104: 405-31; 3 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, *DIS* 55: 193-95; 4 = Ashburner and Harrington, 1984, *Chromosoma* 89: 329-37; 5 = G. Johnson; 6 = Woodruff and Ashburner, 1979, *Genetics* 92: 117-32.

Df(2L)esc10

cytology: *Df(2L)33A8-B1;33B2-3* (G. Richards).
origin: γ ray induced.
references: Struhl, 1981, *Nature* 293: 36-41.
 Frei, Baumgartner, Edström, and Noll, 1985, *EMBO J.* 4: 979-87.
 Frei, Schuh, Baumgartner, Burri, Noll, Jürgens, Seifert, Nauber, and Jäckle, 1988, *EMBO J.* 7: 197-204.
genetics: Deficient for *esc* but not for *sal*. Homozygotes viable and extreme *esc* when mother *esc⁺/esc⁻*, but lethal in first larval instar with homeotic transformation of segments when mother *esc⁻/esc⁻*. Heterozygotes with *esc²*, *esc⁴*, *esc⁵*, *esc⁶*, *esc⁸*, or *esc⁹* show same extreme phenotypes as homozygotes.
molecular biology: Embryos deficient for *esc* show indiscriminate expression of segment-specific homeotic genes (Struhl and Akam, 1985, *EMBO J.* 4: 3259-64). Distal breakpoint mapped to the DNA at about -310 to -290 kb and proximal breakpoint mapped at about +70 kb

("-" values to right, "+" values to left) in the F walk of Frei *et al.*, 1985.

Df(2L)esc-P2-0

cytology: *Df(2L)33A1-2;33B1-2*.
origin: Hybrid dysgenesis.
discoverer: Struhl.
references: Jürgens, 1988, *EMBO J.* 7: 189-96.
 Frei, Schuh, Baumgartner, Burri, Noll, Jürgens, Seifert, Nauber, and Jäckle, 1988, *EMBO J.* 7: 197-204.
genetics: Deficient for *sal* and *esc*.
molecular biology: Distal breakpoint mapped to the DNA at about -476 to -442 kb (Frei *et al.*, 1985).

Df(2L)esc-P3-0

cytology: *Df(2L)33A1-2;33E*.
origin: Hybrid dysgenesis.
discoverer: Struhl.
references: Jürgens, 1988, *EMBO J.* 7: 189-96.
 Frei, Schuh, Baumgartner, Burri, Noll, Jürgens, Seifert, Nauber, and Jäckle, 1988, *EMBO J.* 7: 197-204.
genetics: Deficient for *sal* and *esc*.

Df(2L)F

cytology: Deficient for heterochromatin between *lt* and the secondary constriction.
origin: γ-ray-induced reconstitution of chromosome 2 by detachment of compound second chromosomes.
references: Hilliker and Holm, 1975, *Genetics* 81: 705-21.
 Hilliker, 1976, *Genetics* 83: 765-82.
genetics: *M* and *lt⁺* phenotypes. Deficient for lethal loci *l(2)40Fa* and *l(2)40Fd* (Hilliker, 1976).
other information: Two such deficiencies recovered.

Df(2L)FB15 : see *Df(2L)L138D*

Df(2L)fn: Deficiency (2L) formaldehyde induced

origin: Formaldehyde induced. Selected as ADH null on 1-penten-3-ol.
discoverer: Sofer.

deficiency	cytology	ref ^α	genetics
<i>Df(2L)fn1</i>	<i>34F4-35A1;35D5-7</i>	3, 4, 7, 8	<i>wb⁻-lace⁻</i>
<i>Df(2L)fn2</i>	<i>35A3;35B2</i>	3, 4, 7, 8	<i>el⁻-Adh⁻</i>
<i>Df(2L)fn3^β</i>	<i>35B1;35B3-4</i>	3, 4, 5, 6, 7, 8	<i>l(2)35Ba⁻-l(2)35Bb⁻</i>
<i>Df(2L)fn5</i>	<i>34F5;35C3</i>	7	<i>wb⁻-l(2)35Cb⁻</i>
<i>Df(2L)fn7</i>	<i>34E1-2;35B3-5</i>	2, 3, 5, 6, 7, 8	<i>j⁻-Adh⁻</i>
<i>Df(2L)fn12</i>	<i>34D3;35B10</i>	7	<i>{rk⁻-ck⁻}</i>
<i>Df(2L)fn15</i>	<i>35B1-2;35B7-C5</i>	7	<i>el⁻-ck⁻</i>
<i>Df(2L)fn26</i>	<i>34E3;35D8-E1</i>	3, 7	<i>j⁻-l(2)35Ea⁻</i>
<i>Df(2L)fn27^β</i>	<i>35B1;35D1-2</i>	2, 3, 4, 5, 7	<i>noc⁻-lace⁻</i>
<i>Df(2L)fn30^γ</i>	<i>34C6-7;35B9-C1</i>	7	<i>l(2)34Db⁻-vasa⁻</i>
<i>Df(2L)fn31</i>	<i>34D3;35B3-5</i>	2, 6, 7	<i>l(2)34Db⁻-l(2)35Bf⁻</i>
<i>Df(2L)fn36</i>	<i>35A3;35B4</i>	7	<i>wb⁻-Adh⁻</i>
<i>Df(2L)fn52^β</i>	not visible	1	<i>osp⁻-Adh⁻</i>

^α 1 = Ashburner; 2 = Ashburner, 1982, *Genetics* 101: 447-59; 3 = Ashburner, Detwiler, Tsubota, and Woodruff, 1983, *Genetics* 104: 405-31; 4 = Ashburner, Tsubota, and Woodruff, 1982, *Genetics* 102: 401-20; 5 = Gubb, Roote, Harrington, McGill, Durrant, Shelton, and Ashburner, 1985, *Chromosoma* 92: 116-23; 6 = Gubb, Shelton, Roote, McGill, and Ashburner, 1984, *Chromosoma* 91: 54-64; 7 = O'Donnell, Mandell, Krauss, and Sofer, 1977, *Genetics* 86: 553-66; 8 = Woodruff and Ashburner, 1979, *Genetics* 92: 117-33.

^β See deficiency map under *l(2)35* for molecular coordinates of left breakpoints.

^γ Right breakpoint around 12.5 kb in 35C walk of Lasko and Ashburner (1988, *Nature* 335: 611-17).

Df(2L)G

cytology: *Df(2L)36B5-6;40F*.
origin: Aneuploid segregant from *T(2;Y)G/+*.

Df(2L)GdhA: see *Df(2L)GpdhA*

Df(2L)GM: see *Df(2L)Sco-1*

Df(2L)Got2: Deficiency (2L) Glutamate oxaloacetic transaminase

cytology: *Df(2L)22A;22B5-8*.
origin: Segmental aneuploid from *T(Y;2)J69* and *T(Y;2)H56*.
references: Racine, Langley and Voelker, 1980, Environ. Mutagen. 2: 167-77.
 Chase and Kankel, 1987, J. Neurobiol. 18: 15-41.
genetics: Deficient for *Got2*.

Df(2L)Gpdh75: Deficiency (2L) Glycerol phosphate dehydrogenase

cytology: *Df(2L)25F2-3;25F4-26A1*.
origin: γ ray induced.
discoverer: Voelker.
synonym: *Df(2L)50075a*.
references: Racine, Langley, and Voelker, 1980, Environ. Mutagen. 2: 167-77.
 Kotarski, Pickert, and MacIntyre, 1983, Genetics 105: 371-86.
genetics: Deficient for *Gpdh*.

Df(2L)Gpdh78

cytology: Not visibly deficient.
origin: X ray induced.
discoverer: Voelker.
synonym: *Df(2L)50078a*.
references: Racine, Langley, and Voelker, 1980, Environ. Mutagen. 2: 167-77.
 Kotarski, Pickert, and MacIntyre, 1983, Genetics 105: 371-86.
genetics: Deficient for *l(2)25Fc - Gpdh*. Lethal with *Df(2L)GpdhA* and *Df(2L)cl7*.

Df(2L)Gpdh101

cytology: *Df(2L)25E1-2;26A2-5*.
references: Cook, Shaffer, Bewley, MacIntyre, and Wright.
genetics: Deficient for *Gpdh*.

Df(2L)GpdhA

cytology: *Df(2L)25D7-E1;26A8-9* (Kotarski *et al.*, 1983).
origin: X ray induced.
synonym: *Df(2L)GdhA*.
references: Grell, 1967, Science 158: 1319-20.
 1968, Genetics 60: 184-85.
 O'Brien and MacIntyre, 1972, Genetics 71: 127-38.
 Racine, Langley, and Voelker, 1980, Environ. Mutagen. 2: 167-77.
 Kotarski, Pickert, and MacIntyre, 1983, Genetics 105: 371-86.
 Knipple and MacIntyre, 1984, Mol. Gen. Genet. 198: 75-83.
genetics: Deficient for *cl-l(2)26Ae* (Kotarski *et al.*, 1983). Enhances variegation of *In(1)w^{m4}* and *In(1)y^{3P}* (Locke, Kotarski, and Tartof, 1988, Genetics 120: 181-98).

Df(2L)GT4

cytology: *Df(2L)34F3;35B2*.
origin: γ rays.
discoverer: Durrant.
genetics: Deficient for *l(2)34Fa⁻-noc⁻*.

Df(2L)GT5

origin: γ rays.
discoverer: Durrant.
genetics: Deficient for *b-noc*.

***Df(2L)H:** see **TP(2;Y)H**

Df(2L)H20

cytology: *Df(2L)36A8-9;36E3-4*.
origin: X ray induced.
discoverer: Nüsslein-Volhard.
synonym: *Df(2L)dl2034; Df(2L)dl-H20*.
references: Simpson, 1983, Genetics 105: 615-32.
 Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.
 Steward and Nüsslein-Volhard, 1986, Genetics 113: 665-78.
genetics: Deficient for *l(2)36Aa* through *ninaD*, but not for *M(2)36F*. Lethal over *Df(2L)TW119*, *Df(2L)M36F-S5*, *Df(2L)TW137*, *Df(2L)T317*, *Df(2L)TW201*, *Df(2L)203*, *Df(2L)330*, and *Df(2L)VA18*.

Df(2L)H68

cytology: *Df(2L)36B-C1;37A1-B1* (Wieschaus).
origin: X ray induced.
references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.
genetics: Deficient for *dl, l(2)36Ba - l(2)37Ac* but not *Mhc*. Lethal over *Df(2L)H20*, *Df(2L)T317*, *Df(2L)TW119*, *Df(2L)TW137*, *Df(2L)M36F-S5*, *Df(2L)VA18*, *Df(2L)TW201*, and *Df(2L)50*.

Df(2L)H151

cytology: *Df(2L)25F;26A*.
references: Kotarski, Pickert, and MacIntyre, 1983, Genetics 105: 371-86.

Df(2L)hk18: Deficiency (2L) hook

cytology: *Df(2L)36E4-6;37B9-C1*.
origin: X ray induced in *SD-72* chromosome.
references: Brittnacher and Ganetzky, 1983, Genetics 103: 659-73.
genetics: Deficient for *l(2)36Fb, Tft*, and *hk*, but not *M(2)36F*. Lethal over *Df(2L)M36F-S5*, *Df(2L)M36F-S6*, *Df(2L)TW158*, *Df(2L)TW130*, and *l(2)36Fb-Dox*.
molecular biology: Proximal breakpoint between -52.15 and -50.55 kb (see deficiency map under *l(2)37* for coordinates).

Df(2L)hk39

cytology: *Df(2L)36F6-37A1;37D1-2*.
origin: X ray induced in *SD-Roma* chromosome.
references: Brittnacher and Ganetzky, 1983, Genetics 103: 659-73.
genetics: Deficient for *Tft* and *hk* but not *l(2)37Db*. Lethal over *Df(2L)Sd57* and *Df(2L)Sd77*.

Df(2L)hk-UC1

origin: X ray induced in Canton-S.
discoverer: Gibbs and Marsh.
genetics: Deficient for *l(2)31Ba - Dox-A2* but not for

l(2)37Bg.

molecular biology: Right breakpoint at -56.75 to -54.95 to the left of *Ddc* (-0.06 to +3.76).

Df(2L)hk-UC2

origin: From 15 generations of repeated backcrossing of Canton-S males to M strain females homozygous for *nub b rdo hk pr cn*.

discoverer: Gibbs and Marsh.

references: Eveleth and Marsh, 1986, *Nucleic Acid Res.* 4: 6169-83.

genetics: Deficient for *hk-l(2)37Cb - l(2)37Cc* but not for *l(2)37Cd*.

molecular biology: Right breakpoint at +8.15 to +10.95 to the right of *Ddc* (-0.06 to +3.76).

Df(2L)J: Deficiency (2L) Jammed

origin: X ray induced as *J* revertants.

synonym: *Df(2L)J-der; Df(2L)J-rv.*

deficiency	cytology	ref ^α	genetics
<i>Df(2L)J2</i> ^β	31B;32A	1, 2, 5	<i>J</i> ⁻ - <i>da</i> ⁻
<i>Df(2L)J4</i> ^β	31A-B;31F-32A	1, 2, 4	<i>M(2)30A</i> ⁻ - <i>da</i> ⁻
<i>Df(2L)J27</i> ^β	31B-E;32A	4, 5	<i>J</i> ⁻ - <i>da</i> ⁻
<i>Df(2L)J39</i>	31A-B;32D-E	2, 4, 5	<i>M(2)30A</i> ⁻ - <i>abo</i> ⁻
<i>Df(2L)J77</i>		3	<i>J</i> ⁻ - <i>l(2)54</i> ⁻
<i>Df(2L)J106</i>		3	<i>J</i> ⁻ - <i>l(2)54</i> ⁻
<i>Df(2L)J233</i>		3	<i>J</i> ⁻ - <i>l(2)54</i> ⁻

^α 1 = Mange and Sandler, 1973, *Genetics* 73: 73-86; 2 = Sandler, 1975, *Israel J. Med. Sci.* 11: 1124-34; 3 = Salas and Lengyel, 1984, *DIS* 60: 243-44; 4 = Sandler, 1977, *Genetics* 86: 567-82; 5 = Voelker, Ohnishi, and Langley, 1978, *Biochem. Genet.* 17: 769-83.

^β Lethal over *da*.

Df(2L)JK12

cytology: *Df(2L)37D2-E1;38A8-C1* (Kennison).

origin: γ ray induced.

discoverer: Kennison.

genetics: *pr⁻l(2)37Dc⁻Bl⁺*; lethal in combination with *T(Y;2)H174 = T(Y;2)YS;37D* and *Df(2L)TW158 = Df(2L)37B2-8;37E2-F4*.

Df(2L)L

cytology: *Df(2L)38B;40*.

origin: Segmental deficiency comprising the *Y^{P2D}* element of *T(Y;2)P7 = T(Y;2)h9-10;38b* and the *2^{PYD}* element of *T(Y;2)B190 = T(Y;2)40*.

synonym: *T(Y;2)P57^LB190^R*.

references: Khesin and Leibovitch, 1978, *Mol. Gen. Genet.* 162: 323-28.

Chernyshev, Leibovitch, and Khesin, 1980, *Mol. Gen. Genet.* 178: 663-68.

genetics: Deficient for *His*. Variegated *w* position effect in *Dp(1;3)w^{vco}* weakened in *Df(2L)L/* + flies.

Df(2L)L138D

cytology: *Df(2L)39A;39D-E*, with indeterminate quantities of *Y* heterochromatin intercalated between the chromosome-2 breakpoints.

origin: γ-ray-induced reconstitutions of chromosome 2 from the *Y^{P2D}* element of *T(Y;2)L138* and the *2^{PYD}* element of *T(Y;2)B209*, *T(Y;2)B251*, or *T(Y;2)H54*. Deficient for most but not all of the tandemly repeated Histone genes.

discoverer: J. Siegel.

references: 1981, *Genetics* 98: 505-29.

genetics: Deficient for *His*.

deficiency	2 ^P Y ^D component
<i>Df(2L)L138D-81</i> ^α	<i>T(Y;2)H54</i>
<i>Df(2L)L138D-C2</i>	<i>T(Y;2)B209</i>
<i>Df(2L)L138D-C15</i>	<i>T(Y;2)B209</i>
<i>Df(2L)L138D-FB15</i> ^β	<i>T(Y;2)B251</i>
<i>Df(2L)L138D-X1</i>	<i>T(Y;2)B209</i>
<i>Df(2L)L138D-X14</i>	<i>T(Y;2)B209</i>
<i>Df(2L)L138D-XR</i>	<i>T(Y;2)B209</i>

^α Large segment of Hoechst-33258-bright, *Y*-derived material intercalated between second-chromosome breakpoints; neither *bb⁺* nor any *Y* fertility factor included.

^β Produces *M* phenotype in heterozygotes. Presumably deficient for *M(2)39F* and therefore extends further to right than other *Df(2L)L138D*'s.

Df(2L)It: Deficiency (2L) light

deficiency	approximate cytology	origin	ref ^α	genetics
<i>Df(2L)It1</i>	prox. to 40A	X rays on <i>SD-Roma</i>	1	<i>It⁻-E(SD)⁻</i>
<i>Df(2L)It2</i>	40A-F	γ rays on <i>SD-5</i>	2, 3	<i>l(2)40Fa⁻-l(2)40Fg⁻</i>
<i>Df(2L)It5</i>	prox. to 40A	X rays on <i>SD-Roma</i>	1	<i>It⁻-E(SD)⁻</i>
<i>Df(2L)It8</i>	prox. to 40A	X rays on <i>SD-Roma</i>	1	<i>It⁻-E(SD)⁻</i>
<i>Df(2L)It8a</i>	40B-D	γ rays on <i>SD-5</i>	2, 3	<i>It⁻-E(SD)⁻</i>
<i>Df(2L)It13</i>	prox. to 40A	X rays on <i>SD-Roma</i>	1	<i>It⁻-E(SD)⁺</i>
<i>Df(2L)It23</i>	prox. to 40A	X rays on <i>SD-72</i>	1	<i>It⁻-E(SD)⁻</i>
<i>Df(2L)It25</i>	prox. to 40A	X rays on <i>SD-72</i>	1	<i>It⁻-E(SD)⁻</i>
<i>*Df(2L)It27</i>	40A	γ rays on <i>SD-5</i>	2, 3	<i>It⁻</i>
<i>Df(2L)It45</i>	40A-B	γ rays on <i>SD-5</i>	2, 3	<i>l(2)40Fa⁻-It⁻</i>
<i>Df(2L)It47</i>	40A-B	γ rays on <i>SD-5</i>	2, 3	<i>l(2)40Fa⁻-It⁻</i>
<i>Df(2L)It59</i>	prox. to 40A	X rays on <i>SD-Roma</i>	1	<i>It⁻-E(SD)⁻</i>
<i>Df(2L)It61</i>	40B-F	γ rays on <i>SD-5</i>	2, 3	<i>It⁻-l(2)40Fg⁻</i>
<i>Df(2L)It73</i>	prox. to 40A	X rays on <i>SD-Mad</i>	1	<i>It⁻-E(SD)⁻</i>

^α 1 = Brittnacher and Ganetzky, 1984, *Genetics* 107: 423-34; 2 = Hilliker, 1976, *Genetics* 83: 765-82; 3 = Sharp, Hilliker, and Holm, 1985, *Genetics* 110: 671-88.

Df(2L)M9: see *In(2LR)M9*

Df(2L)M11: see *Df(2L)M24F11*

Df(2L)M15: see *Tp(2;Y)G-M15*

Df(2L)M18: see *Df(2L)M36F18*

Df(2L)M24F: Deficiency (2L) Minute

genetics: *M* phenotype.

deficiency	cytology	origin	syn	ref ^α	genetics (cont)
<i>Df(2L)M24F11</i> ^β	24D3-4; 25A2-3	X ray	<i>M11</i>	6, 8, 9	<i>ang⁺ed⁻-dp⁻</i> <i>l(2)25Ad⁺</i>
<i>Df(2L)M24F-B</i> ^{βγ}	24D8-E1; 24F7-25A1	spont	<i>M-z^B</i>	1, 2, 3, 5, 6, 7, 9	<i>ang⁺l(2)24Efa⁻</i> <i>-dp⁻tkv⁺</i>
<i>Df(2L)M24F-C</i>	24D2-5; 25A2-3	spont	<i>M-z^C</i>	4, 5	<i>ed⁻-dp⁻</i> <i>l(2)cg⁺</i>

^α 1 = Curry, 1939, *DIS* 12: 46; 2 = Duttagupta and Roy, 1984, *DIS* 60: 92; 3 = Duttagupta, Kar, and Roy, 1984, *DIS* 60: 93; 4 = Morgan, Bridges, and Schultz, 1938, *Year-book-Carnegie Inst. Washington* 37: 307; 5 = Morgan, Schultz, Bridges, and Curry, 1939, *Year-book-Carnegie Inst. Washington* 38: 276-77; 6 = Reuter and Szidonya, 1983, *Chromosoma* 88: 277-85; 7 = Roy, Manna, and Duttagupta, 1984, *J. Biosci.* 6: 87-95; 8 = Semeshin and Szidonya, 1985, *DIS* 61: 148-61; 9 = Szidonya and Reuter, 1988, *DIS* 67: 77.

^β Suppresses position-effect variegation in *In(1)w^{m4h}*.

^γ Enhances *bw^{V1}* (Duttagupta *et al.*, 1984).

Df(2L)M36F

genetics: M phenotype.

deficiency	cytology	origin α	syn	discov ref β	genetics (cont)
Df(2L)M36F18	36B3-8; 36D1-E1		M18	1	<i>l(2)36Ba</i> ⁻ <i>l(2)36Da</i> ⁻
Df(2L)M36F-S5 ^{γ}	36D1-E1; 36F1-37A1	1	M(2)H ^{S5}	2, 3	<i>l(2)36Da</i> ⁻ <i>-rdo</i> ⁻ <i>dl</i> ⁺
Df(2L)M36F-S6 ^{δ}	36E6-F1; 36F7-9	2	M(2)m ^{S6}	2, 3	<i>l(2)36Fb</i> ⁻ <i>-l(2)36Fe</i> ⁻

α 1 = In stock labelled M(2)H^{S5}; 2 = X ray induced.
 β 1 = Mohler and Wieschaus; 2 = Schultz, 1933; 3 = Wright, Hodgetts, and Sherald, 1976, Genetics 84: 267-85.
 γ Lethal over Df(2L)M36F-S6 (claimed by Schultz to complement this deficiency, but found by Wright not to do so). Also lethal over Df(2L)TW119, Df(2L)H20; Df(2L)TW50, Df(2L)TW137, Df(2L)E71, Df(2L)TW3, Df(2L)VA16, and Df(2L)hk18 (Wright).
 δ Lethal over Df(2L)M36F-S5, Df(2L)TW50, Df(2L)hk18, Df(2L)E71, Df(2L)TW3, and Df(2L)E53 (Wright).

Df(2L)M-B: see Df(2L)M24F-B

Df(2L)M-C: see Df(2L)M24F-C

Df(2L)M-HS5: see Df(2L)M36F-S5

Df(2L)M-mS6: see Df(2L)M36F-S6

Df(2L)M-zB: see Df(2L)M24F-B

Df(2L)M-zC: see Df(2L)M24F-C

Df(2L)Mdh: Deficiency (2L) Malate dehydrogenase

cytology: Df(2L)30D-F;31F.
 origin: X ray induced.
 synonym: Df(2L)Mdh-2J.
 references: E. H. Grell, 1969, Genetics 61: s23.
 Sandler, 1977, Genetics 86: 567-82.
 genetics: J revertant. Deficient for M(2)30A-da.

Df(2L)N22-3

cytology: Df(2L)30A1-2;30D1-2.
 discoverer: Schüpbach.
 references: Wustmann, Szidonya, Taubert, and Reuter, 1989, Mol. Gen. Genet. 217: 520-27.

Df(2L)N22-5

cytology: Df(2L)29D1-2;30C4-D1.
 discoverer: Schupbach.
 references: Wustmann, Szidonya, Taubert, and Reuter, 1989, Mol. Gen. Genet. 217: 520-27.

Df(2L)n7813

cytology: Df(2L)35B1;35D5-7.
 origin: Hybrid dysgenesis.
 discoverer: Benzce.
 genetics: Deficient for *l(2)35Ba* - *l(2)35Dg*⁻.

Df(2L)net18: see *In(2LR)net*¹⁸

Df(2L)net

deficiency	cytology	ref α	genetics
Df(2L)net62 ^{β}	21A;21B4-5	1, 3	<i>net</i> ⁻ <i>l(2)gl</i> ⁻
Df(2L)net-PM47C	21A1;21B6-7	2	<i>net</i> ⁻
Df(2L)net-PMC	21A1;21B6-7	2	<i>net</i> ⁻ <i>kis</i> ⁻
Df(2L)net-PMF	21A1;21B7-8	2	<i>net</i> ⁻ <i>kis</i> ⁻

α 1 = Golubovsky, Kulakov, and Korochkina, 1978, Genetika 14: 294-305; 2 = Kennison; 3 = Korochkina and Golubovsky, 1978, DIS 53: 197-200.

β X ray induced.

Df(2L)nNx F1

cytology: Df(2L)35B3;35B10 (Ashburner).
 origin: X ray induced.
 discoverer: Maroni.
 synonym: Df(2L)Adh1.
 genetics: Deficient for *osp-l(2)35Cb*.
 molecular biology: Left breakpoint mapped to the DNA (Chia, Karp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706).

Df(2L)nNx F2

cytology: Df(2L)35B3;35B10 (Ashburner).
 origin: X ray induced.
 discoverer: Maroni.
 synonym: Df(2L)Adh2.
 genetics: Deficient for *osp-l(2)35Cb*.
 molecular biology: Left breakpoint mapped to the DNA (Chia, Karp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706).

Df(2L)noc: Deficiency (2L) no-ocelli

origin: γ ray induced.
 references: Ashburner.

deficiency	cytology	discoverer	genetics
Df(2L)noc10	34F1-2;35B1-2	Harrington	<i>l(2)34Fa</i> ⁻ <i>-Adh</i> ⁻
Df(2L)noc11	34E3;35A2	Harrington	<i>l(2)34Fa</i> ⁻ <i>-l(2)35Cd</i> ⁻
Df(2L)noc13	35A1-2;35B2	Harrington	<i>wb</i> ⁻ <i>-Adh</i> ⁻
Df(2L)noc20 ^{α}	34F1-2;35B2	Roote	<i>l(2)34Fa</i> ⁻ <i>-Adh</i> ⁻

α Induced in CyO, *Adh*^{nB}. *Adh* deficient by molecular criteria (Karp). Df(2L)noc20/*osp*⁻ flies have extreme Curly wings. Proximal DNA breakpoint between +11.8 and +13.7 kb (Chia, Karp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706).

Df(2L)NST

origin: X ray induced in Canton-S males.
 discoverer: Gibbs and Marsh.
 references: Pentz and Wright, 1986, Genetics 112: 843-59.
 Marsh, Erfle, and Leeds, 1986, Genetics 114: 453-67.
 genetics: Deficient for *l(2)37Bd* - *l(2)37Cg* but not for *l(2)37Da*.
 molecular biology: Left breakpoint at -37.8 to -33.8 kb, approximately 30 kb distal to *Ddc*.

Df(2L)OD

origin: Induced by diepoxybutane.

deficiency	discoverer α	genetics
Df(2L)OD9	2	<i>rdo</i> ⁺ <i>hk</i> ⁻ <i>-Ddc</i> ⁻ <i>ref(2)P</i> ⁺
Df(2L)OD12	2	<i>ref(2)P</i> ⁺ <i>l(2)37Fa</i> ⁻
Df(2L)OD15 ^{β}	2, 3	<i>hk</i> ⁻ <i>-l(2)37Be</i> ⁻ <i>amd</i> ⁺
Df(2L)OD16 ^{γ}	2	<i>ref(2)P</i> ⁺ <i>l(2)37Fd</i> ⁻ <i>-l(2)37Fg</i> ⁻
Df(2L)OD21	2	<i>ref(2)P</i> ⁺ <i>l(2)37Fc</i> ⁻
Df(2L)OD-P5	1	<i>ref(2)P</i> ⁻ <i>l(2)37Ea</i> ⁻ <i>l(2)37Fa</i> ⁻ <i>l(2)37Fb</i> ⁻

α 1 = Contamine, Peritjean, and Ashburner, 1989, Genetics 123: 525-33; 2 = Gay and Contamine; 3 = Wright.
 β Proximal DNA breakpoint between -64.65 and -65.25 kb (see deficiency map under *l(2)37* for coordinates).
 γ Only part of 38A deleted. Shows non-transparent wings when heterozygous with Df(2L)E55.

Df(2L)odd: Deficiency (2L) odd skipped

cytology: Df(2L)21A;24B.
 references: Nüsslein-Volhard, Wieschaus, and Kluding,

1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.
genetics: Deficient for *odd*.

Df(2L)osp: Deficiency (2L) outspread

origin: γ ray induced.

discoverer: Detwiler.

references: Ashburner, Tsubota, and Woodruff, 1982, Genetics 102: 401-20.

Ashburner, Detwiler, Tsubota, and Woodruff, 1983, Genetics 104: 405-31.

deficiency	cytology	genetics
Df(2L)osp18 ^{$\alpha\beta$}	35B1-2;35C4-5	<i>osp</i> ⁻ - <i>dgl</i> ⁻
Df(2L)osp29 ^{$\beta\gamma$}	35B1-3;35E6	<i>osp</i> ⁻ - <i>l(2)35Bb</i> ⁻ - <i>l(2)35Eb</i> ⁻
Df(2L)osp38	35A1-2;35C4-5	<i>wb</i> ⁻ - <i>l(2)35Cd</i> ⁻
Df(2L)osp141 ^{δ}	34F5;35B5	<i>l(2)34Fa</i> ⁻ - <i>Adh</i> ⁻
Df(2L)osp144 ^{$\beta\epsilon$}	not evident	<i>noc</i> ⁻ - <i>osp</i> ⁻
Df(2L)osp204 ^{β}	see <i>Tp(3;2)osp204</i>	<i>noc</i> ⁻ - <i>osp</i> ⁻

- α Also see Simpson, 1983, Genetics 105: 615-32.
- β Left and/or right breakpoints mapped to the DNA (Chia, Karp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706).
- γ Heterozygotes with *Sco* viable and without enhanced *Sco* phenotype.
- δ Induced simultaneously with *T(2;3)CA7* and *T(2;3)CA8*.
- ϵ Deleted for about 50 kb of DNA within both the *osp* and *noc* loci (S. McGill).
- ζ Gubb, Roote, Harrington, McGill, Durrant, Shelton, and Ashburner, 1985, Chromosoma 92: 116-23.

Df(2L) π 1a1

cytology: *Df(2L)34D3;35B4* (Ashburner).
origin: Hybrid dysgenesis; selected as ADH null.
discoverer: Craymer.
genetics: Deficient for *l(2)34Db-l(2)35Bf*; includes *Adh*.

Df(2L)PA4

cytology: *Df(2L)35B1-3 + Df(2L)35D1;36A1-2*.
origin: γ ray induced in *Sco* chromosome; selected as ADH null.
discoverer: Angel.
references: Ashburner, Detwiler, Tsubota, and Woodruff, 1983, Genetics 104: 405-31.
 Simpson, 1983, Genetics 105: 615-32.
 Ashburner and Harrington, 1984, Chromosoma 89: 329-37.
genetics: Deficient for *noc-Adh* and *sna-l(2)35Ec*. Revertant for *Sco*.

Df(2L)PM

references: Caggese, Caizzi, Bozzetti, Barsanti, and Ritossa, 1988, Biochem. Genet. 26: 571-84.

deficiency	cytology	genetics
Df(2L)PM1	21A1;21B3-5	<i>net</i> ⁻ <i>Gst</i> ⁺
Df(2L)PM4	21A1;21B1-2	<i>net</i> ⁻ <i>Gst</i> ⁺
Df(2L)PM6	21A1;21B2-3	<i>net</i> ⁻ <i>Gst</i> ⁺
Df(2L)PM44	21A1;21B2-4	<i>net</i> ⁻ <i>Gst</i> ⁺
Df(2L)PM45	21A1;21B4-6	<i>net</i> ⁻ <i>Gst</i> ⁻
Df(2L)PM47C	21A1;21B6-7	<i>net</i> ⁻ <i>Gst</i> ⁻
Df(2L)PM51	21A1;21B3-5	<i>net</i> ⁻ <i>Gst</i> ⁺
Df(2L)PM59	21A1;21B3-5	<i>net</i> ⁻ <i>Gst</i> ⁺
Df(2L)PM82	21A1;21B2-4	<i>net</i> ⁻ <i>Gst</i> ⁺
Df(2L)PM91	21A1;21B4-6	<i>net</i> ⁻ <i>Gst</i> ⁻
Df(2L)PMC	21A1;21B4-6	<i>net</i> ⁻ <i>Gst</i> ⁻
Df(2L)PMF	21A1;21B7-8	<i>net</i> ⁻ <i>Gst</i> ⁻
Df(2L)PMG	21A1;21B4-6	<i>net</i> ⁻ <i>Gst</i> ⁻

Df(2L)pr: Deficiency (2L) purple

origin: X ray induced.

synonym: *pr* may be omitted as in *Df(2L)A1*, *Df(2L)B5*, etc.; *Df(2L)pr21* also called *Df(2L)SD-7 pr21*.

deficiency	cytology	ref ^{α}	genetics
Df(2L)pr2b	38B5-C1;38D2-E1		<i>pr</i> ⁻
Df(2L)pr8b	38A2-5;39A2-B1		<i>pr</i> ⁻
Df(2L)pr21	37E3-F1;38C6-10	2, 3	<i>pr</i> ⁻
Df(2L)pr26	37D5-6;38C8-10	2, 3	<i>ref(2)P</i> ⁻ - <i>pr</i> ⁻
Df(2L)pr28	37D3-E1;38C6-9		<i>pr</i> ⁻
Df(2L)pr40	38A2-5;38A6-D1		<i>pr</i> ⁻
Df(2L)pr47	38B1-2;38C1-2	2	<i>pr</i> ⁻
Df(2L)pr49	38B3-6;38C6-10	2	<i>pr</i> ⁻
Df(2L)pr65	38A3-5;38D3-5	2	<i>pr</i> ⁻
Df(2L)pr67	38A5-8;39C-E	2	<i>pr</i> ⁻ - <i>l(2)crc</i> ⁻
Df(2L)pr69	38B1-2;38C5-6	2	<i>pr</i> ⁻
Df(2L)pr76	37D;38E	1	<i>pr</i> ⁻
Df(2L)pr-A1		4	<i>pr</i> ⁻
Df(2L)pr-A2		4	<i>pr</i> ⁻
Df(2L)pr-A3		4	<i>pr</i> ⁻
Df(2L)pr-A4		4	<i>pr</i> ⁻
Df(2L)pr-A5		4	<i>pr</i> ⁻
Df(2L)pr-A6		4	<i>pr</i> ⁻
Df(2L)pr-A7		4	<i>pr</i> ⁻
Df(2L)pr-A8	36F8-11;38B6-C1	4	<i>Tfi</i> ⁻ - <i>pr</i> ⁻
Df(2L)pr-A9		4	<i>pr</i> ⁻
Df(2L)pr-A14	37D2-7;39A4-7	3, 4, 7	<i>Sd</i> ⁻ - <i>pr</i> ⁻
Df(2L)pr-A16	37B2-12;38D2-5	3, 4, 7	<i>hk</i> ⁻ - <i>pr</i> ⁻
Df(2L)pr-A20	38A3-4;38B6-C1	4	<i>pr</i> ⁻
Df(2L)pr-B5	38A7-8;39A4-C4	4	<i>pr</i> ⁻
Df(2L)pr-B10	38A7-8;39A4-C4	4	<i>pr</i> ⁻
Df(2L)pr-B11	38A7-8;39A4-C4	4	<i>pr</i> ⁻
Df(2L)pr-B12	38A7-8;39A4-C4	4	<i>pr</i> ⁻
Df(2L)pr-B14	38A7-8;39A4-C4	4	<i>pr</i> ⁻
Df(2L)pr-F59 ^{β}	37D;38F	5	<i>ref(2)P</i> ⁻ - <i>pr</i> ⁻
Df(2L)pr-F65 ^{β}		5	<i>hk</i> ⁻ - <i>l(2)crc</i> ⁻
Df(2L)pr-F99 ^{β}		5	<i>pr</i> ⁻
Df(2L)pr-F144 ^{β}		5	<i>ref(2)P</i> ⁻ - <i>l(2)crc</i> ⁻
Df(2L)pr-F257 ^{β}		5	<i>ref(2)P</i> ⁻ - <i>l(2)crc</i> ⁻
Df(2L)pr-F280 ^{β}		5	<i>ref(2)P</i> ⁻ - <i>l(2)crc</i> ⁻
Df(2L)pr-F286 ^{β}		5	<i>hk</i> ⁻ - <i>l(2)crc</i> ⁻
Df(2L)pr-R	37D1;38C1	6	<i>pr</i> ⁻

- α 1 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-195; 2 = Brittnacher and Ganetzky, 1983, Genetics 103: 659-73; 3 = Gay and Contamine; 4 = Ganetzky, 1977, Genetics 86: 321-55; 5 = Nakamura, 1973, Thesis, Paris-Sud; 6 = Roberts, 1971, DIS 46: 122; 7 = T.R.F. Wright.
- β Derived from *Dp(2;Y)G*.

Df(2L)PR7F

cytology: In 2L heterochromatin just distal to the centromere.
discoverer: Hilliker.
references: Brittnacher and Ganetzky, 1984, Genetics 107: 423-34.

Df(2L)prd1.25: Deficiency (2L) paired

cytology: *Df(2L)33B6-7;33E1-2 + T(Y;2)21;40*.
discoverer: Nüsslein-Volhard.
references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.
 Nüsslein-Volhard, Kluding, and Jürgens, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 145-54.
genetics: Deficient for *prd*.

Df(2L)prd1.7

location: *Df(2L)33B2-3;34A1-2*.
discoverer: Nüsslein-Volhard.
references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

Nüsslein-Volhard, Kluding, and Jürgens, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 145-54.
genetics: Deficient for *prd*.
molecular biology: Distal breakpoint mapped to the DNA (Frei, Baumgartner, Edström, and Noll, 1985, EMBO J. 4: 979-87).

Df(2L)prd2.27: see *Tp(2;3)prd*^{2,27}

Df(2L)prd5.12: see *Tp(2;Y)prd*^{5,12}

Df(2L)Prl: Deficiency (2L) Proxless

cytology: *Df(2L)32F1-3;33F1-2* (Ashburner).
discoverer: B.S. Baker.
references: Jürgens, 1988, EMBO J. 7: 189-96.
 Frei, Schuh, Baumgartner, Burri, Noll, Jürgens, Seifert, Nauber, and Jäckle, 1988, EMBO J. 7: 197-204.
genetics: Associated with *Prl*. Deficient for *prd*, *sal* (Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82), and *nub* but not *b* (Ashburner).
molecular biology: Distal breakpoint mapped to the DNA at about -430 to -400 kb (Frei *et al.*, 1988).

Df(2L)prx

origin: X ray induced.
discoverer: Simpson.

deficiency	cytology	genetics
<i>Df(2L)prx3</i>	35D3;35D4 ^α	<i>sna</i> ⁻ - <i>l(2)35Dd</i> ⁻
<i>Df(2L)prx13</i>	35D1;35D4	<i>l(2)35Ce</i> ⁻ - <i>l(2)35Dg</i> ⁻
<i>Df(2L)prx18</i>	not visible	
<i>Df(2L)prx19</i>	35C1;35D1-2	<i>l(2)35Cb</i> ⁻ - <i>l(2)35Dd</i> ⁻
<i>Df(2L)prx24</i>	35B3-35E1-2	<i>l(2)35Bb</i> ⁻ - <i>l(2)35Dg</i> ⁻
<i>Df(2L)prx31</i>	35D1;35D4	<i>rd</i> ⁻ - <i>l(2)35Dg</i> ⁻

^α Probably not detectable.

Df(2L)rd9: Deficiency (2L) reduced

cytology: *Df(2L)35A4;35B3*.
origin: γ ray induced.
discoverer: Thompson.
genetics: Deficient for *noc-Adh*.

Df(2L)RMD: Deficiency (2L) Roy Manna Duttgupta

cytology: Not visible.
origin: X ray induced.
references: Roy, Manna, and Duttgupta, 1984, J. Biosci. 6: 87-95.

deficiency	synonym	genetics ^α
<i>Df(2L)RMD42</i>	<i>l(2)42</i>	<i>l(2)24EFa</i> ⁻ - <i>l(2)24EFd</i> ⁻
<i>Df(2L)RMD113</i>	<i>l(2)113</i>	<i>l(2)24EFc</i> ⁻ - <i>l(2)24EFd</i> ⁻
<i>Df(2L)RMD149</i>	<i>l(2)149</i>	<i>l(2)24EFb</i> ⁻ - <i>l(2)24EFd</i> ⁻
<i>Df(2L)RMD202</i>	<i>l(2)202</i>	<i>l(2)24EFa</i> ⁻ - <i>l(2)24EFc</i> ⁻
<i>Df(2L)RMD239</i>	<i>l(2)239</i>	<i>l(2)24EFb</i> ⁻ - <i>l(2)24EFc</i> ⁻
<i>Df(2L)RMD261</i>	<i>l(2)261</i>	<i>l(2)24EFa</i> ⁻ - <i>l(2)24EFc</i> ⁻
<i>Df(2L)RMD269</i>	<i>l(2)269</i>	<i>l(2)24EFa</i> ⁻ - <i>l(2)24EFg</i> ⁻ <i>M24F</i> ⁻ - <i>dw24F</i> ⁻ - <i>dp</i> ⁻

^α Order of loci unknown.

Df(2L)S: Deficiency (2L) Star

origin: X ray induced.
discoverer: E.B. Lewis, 1940.
references: 1945, Genetics 30: 147-51.

deficiency	cytology	genetics
*Df(2L)S1	21C3-4;22A2-3	<i>ds</i> ⁻ - <i>ast</i> ⁻

deficiency	cytology	genetics
Df(2L)S2	21C6-D1;22A6-B1	<i>ds</i> ⁻ - <i>shr</i> ⁻
Df(2L)S3^α	21D2-3;21F2-22A1	<i>S</i> ⁻ - <i>ast</i> ⁻
*Df(2L)S4	21C3-4;22B2-3	<i>ds</i> ⁻ - <i>shr</i> ⁻
*Df(2L)S5	21C2-3;22A3-4	<i>ex</i> ⁻ - <i>ast</i> ⁻
*Df(2L)S7	21C3-4;21F2-22A1	<i>ds</i> ⁻ - <i>ast</i> ⁻

^α Uncovers *ninaA*.

Df(2L)S^{56f}: see *In(2LR)S^{56f}*

Df(2L)S1 (of Simpson): see *Df(2L)SS1*

***Df(2L)S-der: Deficiency (2L) Star derived**

cytology: *Df(2L)21D4-E1;21E2-3*.
new order:
 Y|21D4 – 21A;
 60 – 21E3|101.
origin: Synthetic; a combination of *Y^{P2D}* from *T(Y;2)21E* = *T(Y;2)21D4-E1* and *2^{F4D}* from *T(2;4)ast^V* = *T(2;4)21E2-3;101*.
discoverer: E. B. Lewis.
references: 1945, Genetics 30: 137-66.
genetics: Deficient for *S* and *ast*. Homozygous lethal.

Df(2L)sc19: Deficiency (2L) scute19

origin: X ray induced loss of *y⁺* from *Tp(1;2)sc¹⁹*.
references: Semeshin and Szidonya, 1985, DIS 61: 148-54.
 Szidonya and Reuter, 1988a, DIS 67: 77-79.
 1988b, Genet. Res. 51: 197-208.

deficiency	cytology	genetics ^α
Df(2L)sc19-1	24D5-6;25C8	<i>ed</i> ⁻ - <i>l(2)25</i> ⁻
Df(2L)sc19-3	24E3;25A6-7	<i>dw24F</i> ⁻ - <i>l(2)25Aa</i> ⁻
Df(2L)sc19-4	25A5;25E4-5	<i>sif</i> ⁻ - <i>mid</i> ⁻
Df(2L)sc19-5	25A5;25D6	<i>sif</i> ⁻ - <i>l(2)25Dd</i> ⁻
Df(2L)sc19-6	24F1-2;25C5	<i>l(2)24Ec</i> ⁻ - <i>l(2)25Ce</i> ⁻
Df(2L)sc19-7	24D2-4;25C2-3	<i>ed</i> ⁻ - <i>l(2)25Cc</i> ⁻
Df(2L)sc19-8	24C2-8;25C2-8	<i>ed</i> ⁻ - <i>l(2)25</i> ⁻
Df(2L)sc19-9	24D4-5;25F4-26A1	
Df(2L)sc19-10	25A4-5;25B9-C1	<i>sif</i> ⁻ - <i>l(2)25Be</i> ⁻
Df(2L)sc19-11	24D2-4;25B2-4	<i>ed</i> ⁻ - <i>l(2)25Ac</i> ⁻
Df(2L)sc19-12	24A4-5;26A6-B1	<i>sif</i> ⁻ - <i>l(2)26Aa</i> ⁻
Df(2L)sc19-13	24E2-4;25B2-5	<i>dw24F</i> ⁻ - <i>l(2)25Ba</i> ⁻

^α Includes complementation groups localized in the deleted regions by Szidonya (unpublished).

Df(2L)Sco: Deficiency (2L) Scutoid

origin: X ray induced revertants of *Sco*.
synonym: *Df(2L)Sco^{R+}*.

deficiency	cytology	discoverer	ref ^α	genetics
Df(2L)Sco4	see <i>In(2L)Sco^{rv4}</i>		4	
Df(2L)Sco7^β	34D5;35D5-7	E. H. Grell	1, 5	<i>b</i> ⁻ – <i>rd</i> ⁻
Df(2L)Sco10	35B+35D	Ashburner	2, 6	<i>noc</i> ⁻ to <i>Adh</i> ⁻ <i>sna</i> ⁻ to <i>l(2)35De</i> ⁻
Df(2L)Sco14	35B+35D	Ashburner	2, 3, 6	<i>noc</i> ⁻ to <i>Adh</i> ⁻ <i>sna</i> ⁻ to <i>l(2)3Dg</i> ⁻
Df(2L)Sco16	35D;35D	Ashburner	2	<i>sna</i> ⁻ to <i>l(2)35Ec</i> ⁻
Df(2L)Sco18	35B+35D	Ashburner	2, 3	<i>noc</i> ⁻ to <i>Adh</i> ⁻ <i>sna</i> ⁻ to <i>l(2)35Eb</i> ⁻
Df(2L)Sco19	35D;35D	Ashburner	2	<i>sna</i> ⁻
Df(2L)Sco23	see <i>Tp(2;1)Sco^{rv23}</i>			
Df(2L)Sco25	35D;35D	Ashburner	2	<i>sna</i> ⁻ to <i>lace</i> ⁻

^α 1 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91; 2 = Ashburner, Detwiler, Tsubota, and Woodruff, 1983, Genetics 104: 405-31; 3 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95; 4 = Ashburner and Harrington, 1984, Chromosoma 89: 329-37; 5 = O'Donnell,

Mandel, Krauss, and Sofer, 1977, *Genetics* 86: 553-66; 6 = Woodruff and Ashburner, 1979, *Genetics* 92: 117-32.

β Left breakpoint of deletions mapped to the DNA (Chia, Karp, McGill, and Ashburner, 1985, *J. Mol. Biol.* 186: 689-706; McGill, Chia, Karp, and Ashburner, 1988, *Genetics* 119: 647-61).

Df(2L)Sco1-DV7

cytology: Resembles *In(2LR)Sco^{rv1}* (Ashburner).

origin: γ ray induced in *In(2LR)Sco^{rv1}*.

discoverer: G. Johnson.

genetics: Deficient for *sna-l(2)35De*.

Df(2L)Sd: Deficiency (2L) Segregation distorter

origin: X ray induced.

deficiency	cytology	chromosomal ref ^α origin	genetics
Df(2L)Sd2	37D1-2;38D2-E1	SD-Roma 1	<i>Sd⁻-ms(2)⁻</i> ;
Df(2L)Sd14	37D1-2;38C1-2	SD-Roma 1	<i>Sd⁻-pr⁻</i>
Df(2L)Sd37	37D2-7;38A6-B2	SD-5 1, 2, 3, 4	<i>Sd⁻-ref(2)P⁻</i> <i>pr⁺</i>
Df(2L)Sd57^β	37D1-2;38C1-2	SD-Roma 1, 4	<i>l(2)37Cf⁻-pr⁻</i>
Df(2L)Sd68^γ	37B3-7;38E3-5	SD-Mad 1	<i>hk⁻-ms(2)38C⁻</i>
Df(2L)Sd77^δ	37D2-7;38C1-2	SD-Mad 1, 4	<i>fs(2)TWI⁻-pr⁻</i>

α 1 = Brittnacher and Ganetzky, 1983, *Genetics* 103: 659-73; 2 = Ganetzky, 1977, *Genetics* 86: 321-55; 3 = Gay and Contamine; 4 = T.R.F. Wright.

β Distal breakpoint +38.55 - +40.55 kb [for coordinates see deficiency map under l(2)37].

γ Deficiency able to generate *cad* gene transcripts (Macdonald and Struhl, 1986, *Nature* 324: 537-45).

δ Distal breakpoint +47.05 - +52.55 kb [for coordinates see deficiency map under l(2)37].

Df(2L)SD-7^{pr21}: see *Df(2L)pr21*

Df(2L)SD-Mad-d77: see *Df(2L)Sd77*

Df(2L)SD-R-m57: see *Df(2L)Sd57*

Df(2L)sna-S1: see *Df(2L)SS1*

Df(2L)spd: Deficiency (2L) spade

cytology: *Df(2L)27D-E;28C*.

discoverer: E.H. Grell.

genetics: Deficient for *spd*.

Df(2L)SS1

cytology: Not visibly deficient.

discoverer: Simpson.

origin: X ray induced.

synonym: *Df(2L)sna-S1*; *Df(2L)S1* (of Simpson).

references: Grau, Carteret, and Simpson, 1984, *Genetics* 108: 347-60.

genetics: Deficient for *rd-l(2)35Dg* (Ashburner).

Df(2L)Su(Pc): Deficiency (2L) Suppressor of Polycomb

cytology: *Df(2L)37D2-E1;38A8-C1*.

origin: γ ray induced.

discoverer: Kennison.

references: Kennison and Russell, 1987, *Genetics* 116: 75-86.

genetics: Dominant suppressor of *Pc*.

Df(2L)Sw-L: Deficiency (2L) Swedish-L

cytology: Tip of 2L contains deficiency.

origin: Naturally occurring condition in some Swedish strains.

discoverer: Gustafson, 1937.

genetics: No phenotypic effect.

Df(2L)T317

cytology: Breakpoints unknown; associated with a *T(2;4)* (Steward and Nüsslein-Volhard).

references: Steward and Nüsslein-Volhard, 1986, *Genetics* 113: 665-78.

genetics: Deficient for *dl, l(2)36Bd-ninaD* but not *M(2)36F*. Lethal over *Df(2L)H20, Df(2L)H68, Df(2L)TW119, Df(2L)TW137, Df(2L)M36F-S5, Df(2L)VA18, and Df(2L)201*.

Df(2L)TE21A

cytology: *Df(2L)21A;21B4-6*; extra bands that fail to pair with normal homologue inserted (Kennison).

origin: γ ray induced in *TE21A*.

discoverer: Ising.

synonym: *Df(2L)TE75*.

references: Ising and Block, 1980, Cold Spring Harbor Symp. Quant. Biol. 45: 527-44.

genetics: *w⁺rst⁺* inserted in 21B with loss of terminal bands of 2L [*l(2)gl⁺net⁻l(2)al⁺*]; proximal to breakpoint of *In(2L)shv³⁴ = In(2L)21B6-C1;22F1-2*.

Df(2L)TE35A: Deficiency (2L) Transposing Element in 35AB

origin: γ ray induced in *TE35A* except for spontaneous deficiencies *TE35A-48* and *TE35A-54*.

synonym: *Df(2L)TE146*.

genetics: Deficient for *w* and *rst* except for *Df(2L)TE35A-48* and *Df(2L)TE35A-54* which carry *w⁺* and *rst⁺*.

deficiency	cytology	discoverer	ref ^α	genetics
Df(2L)TE35A-1	35B1;35C4-5	Gubb	2	<i>el⁻-dgl⁻</i>
Df(2L)TE35A-2	34F4;35D7	Gubb	2	<i>wb⁻-l(2)35Dg</i>
Df(2L)TE35A-2a	see <i>T(2;3)TE35A-2a</i>			
Df(2L)TE35A-3	34F5;35B3	Gubb	2	<i>l(2)34Fa⁻-Adh⁻</i>
Df(2L)TE35A-4^β	34F1-2;35A2	Ashburner	1, 2	<i>l(2)34Fa⁻-noc⁻</i>
Df(2L)TE35A-5^γ	34D2;35C1	Ashburner	2	<i>l(2)34Db⁻--vasa⁻</i> <i>l(2)35Cb⁻</i>
Df(2L)TE35A-5a	see <i>T(2;3)TE35A-5a</i>			
Df(2L)TE35A-6	35B1;35C1	Ashburner	2	<i>el⁻-l(2)35Cb⁻</i>
Df(2L)TE35A-7	35A3-4;35B2	Ashburner	2	<i>l(2)35Aa⁻-Adh⁻</i>
Df(2L)TE35A-8^β	34E4-5;35A2-3	Ashburner	1, 2	<i>rk⁻-osp⁻</i>
Df(2L)TE35A-9	34F1;35B2	Ashburner	2	<i>l(2)34Fa⁻-Adh⁻</i>
Df(2L)TE35A-10^β	34F5;35B2	Durrant	1, 2	<i>wb⁻-noc⁻</i>
Df(2L)TE35A-11^β	not visible	Durrant	1, 2	<i>noc⁻-Adh⁻</i>
Df(2L)TE35A-12^β	cytologically not visible	Durrant	1, 2	<i>pu⁻-noc⁻</i>
Df(2L)TE35A-13	cytologically not visible			
Df(2L)TE35A-13	see <i>In(2L)TE35A-13</i>			
Df(2L)TE35A-14	35A4;35D4	Durrant	2	<i>el⁻-rd⁻</i>
Df(2L)TE35A-15	34F3;35B2	Durrant	2	<i>wb⁻-Adh⁻</i>
Df(2L)TE35A-46^β	35B;35B	Roote	1	<i>noc⁻-Adh⁻</i>
Df(2L)TE35A-50^β	35B1-2;35B1-2	Roote	1	<i>noc⁻-Adh⁻</i>
Df(2L)TE35A-54^β	35B;35B	Roote	1	<i>noc⁻-Adh⁻</i>
Df(2L)TE35A-54a	35B;35B	Ashburner		<i>noc⁻-Adh⁻</i>

α 1 = Chia, Karp, McGill, and Ashburner, 1985, *J. Mol. Biol.* 186: 689-706; 2 = Gubb, Roote, Harrington, Durrant, Shelton, and Ashburner, 1985, *Chromosoma* 92: 116-23.

β Molecular biology: Breakpoint(s) mapped to the DNA [Chia *et al.*, 1985; for coordinates see deficiency map under l(2)35A-E].

γ Right breakpoint mapped to the DNA between +14.5 and +18 kb on the restriction map of the *vasa* region [Lasko and Ashburner, 1988, *Nature* (London) 335: 611-17].

Df(2L)TE35BC

origin: γ ray induced in TE35BC.
synonym: Df(2L)TE36.
genetics: Deficient for *w* and *rst* except for Df(2L)TE35BC-23, Df(2L)TE35BC-28, and Df(2L)TE35BC-31, which have remnants of the bands in TE35BC and carry *rst*⁺.

deficiency	cytology	discov	ref ^α	genetics
Df(2L)TE35BC-1	35C1;35D1	Gubb	1, 2, 3	Su(H) ⁻ -ck ⁻ ;vasa ⁺
Df(2L)TE35BC-3	35C1;35D2	Gubb	1, 3	ck ⁻ -lace ⁻
Df(2L)TE35BC-4	35B4;35C3	Gubb	1, 3	l(2)35Bb ⁻ -dgl ⁻
Df(2L)TE35BC-6	34D3-6;35C1	Gubb	3	l(2)34Db ⁻ -l(2)35Cb ⁻
Df(2L)TE35BC-7 ^β	35B3; 35B9-10	Gubb	3	noc ⁻ -ck ⁻ ;vasa ⁺
Df(2L)TE35BC-8	35B1-2; 35E1-2	Gubb	3	eI ⁻ -l(2)35Dg ⁻
Df(2L)TE35BC-23	34D1-2; 35B10-C1	Shelton	3	l(2)34Db ⁻ -ck ⁻
Df(2L)TE35BC-24	34B4-6; 35E1-2	Shelton	3	l(2)35Bg ⁻ -l(2)35Ea ⁻
Df(2L)TE35BC-28	35B2;35B7	Shelton	3	l(2)35Bb ⁻ -ck ⁻
Df(2L)TE35BC-29 ^γ	35A3;35C1	Shelton	3	wb ⁻ -vasa ⁻
Df(2L)TE35BC-31	34F5;35B10	Shelton	3	wb ⁻ -ck ⁻
Df(2L)TE35BC-34	35B4;35D4	Shelton	3	l(2)35Bb ⁻ -l(2)35Dd ⁻
Df(2L)TE35BC-35	35B2;35D4	Shelton	3	l(2)35Bb ⁻ -lace ⁻

^α 1 = Ashburner, 1982, Genetics 101: 447-59; 2 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velisariou, and Walker, 1981, DIS 56: 186-91; 3 = Gubb, Shelton, Roote, McGill, and Ashburner, 1984, Chromosoma 91: 54-64.
^β Molecular biology: Left breakpoint mapped to the DNA at -70 kb (Chia, Karp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706).
^γ Left breakpoint mapped to the DNA between -3 and -2 kb (Lasko and Ashburner, 1988, Nature 335: 611-17).

Df(2L)TE36A

origin: γ ray induced in TE36A.
synonym: Df(2L)TE116.
references: Lasko and Ashburner, 1988, Nature (London) 335: 611-17.

deficiency	cytology	genetics	molecular biology ^α
Df(2L)TE35D-4	35C1-2;?	l(2)35Cb ⁻	+15.5 to +18.5 kb
Df(2L)TE35D-16	35B9-C1;?	vas ⁻ to l(2)35Cb ⁻	-5.1 to -3.7 kb (circa)
Df(2L)TE35D-18	35B9-C1;?	vas ⁻ to l(2)35Cb ⁻	-5.1 to -3.8 kb (circa)

^α DNA coordinates for the distal breakpoints of the deficiencies (0 point is the EcoRI site immediately proximal to the starting point of the walk; "+" values to right, "-" values to left).

***Df(2L)TE37C-1**

cytology: Df(2L)37C2-5;38F5-39A1 (broken within the transposition).
origin: γ ray induced in TE37C.
synonym: Df(2L)TE42-1.
references: Marsh and Wright, 1979, Genetics 92: s74-75. Wright, Beerman, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, Chromosoma 83: 45-58.
genetics: *w*⁺ but not *rst*⁺ deleted. Deficient for l(2)37Cf-B1.

Df(2L)TE38A-1

cytology: Df(2L)38A6-7;38B6-C1.
origin: γ ray induced in TE38A.
synonym: Df(2L)TE48-1.
discoverer: Robertson.
references: T.R.F. Wright.
genetics: Deficient for *pr* and TE38A. Lethal over Df(2L)TW150, but viable over Df(2L)TW130.

Df(2L)TE38A-2

cytology: Df(2L)37F6-38A1;40.
origin: γ ray induced in TE38A of Ising.
synonym: Df(2L)TE48-2.
discoverer: T.R.F. Wright.
genetics: Deficient for *pr*, M(2)39F, and TE38A of Ising. Lethal over Df(2L)TW150, but viable over Df(2L)TW130.

Df(2L)TE42: see Df(2L)TE37C

*Df(2L)TE48: see Df(2L)TE38A

Df(2L)TE146: see Df(2L)TE35A

Df(2L)tkv2: Deficiency (2L) thick veins

cytology: Df(2L)25D2-5;25D6-E1.
origin: X ray induced.
discoverer: Szidonya.
synonym: Df(2L)tkv-Sz2.
references: Reuter and Szidonya, 1983, Chromosoma 88: 277-85. Szidonya and Reuter, 1988a, DIS 67: 77-79. 1988b, Genet. Res. 51: 197-208.
genetics: Deficient for *tkv*-l(2)25De but not for l(2)25Ea.

Df(2L)tkv3

cytology: Df(2L)25A2-3;25D5-E1.
origin: X ray induced.
discoverer: Szidonya.
synonym: Df(2L)tkv-Sz3.
references: Reuter and Szidonya, 1983, Chromosoma 88: 277-85. Szidonya and Reuter, 1988a, DIS 67: 77-79. 1988b, Genet. Res. 51: 197-208.
genetics: Deficient for *slf*-*tkv*. Females sterile; males semisterile.

Df(2L)TW: Deficiency (2L) Ted Wright

origin: Df(2L)TW1-Df(2L)TW161 are X ray induced, Df(2L)TW201-Df(2L)TW203 are γ ray induced, Df(2L)TW330 formaldehyde or ethyl methanesulfonate induced.
synonym: Df(2L)1-Df(2L)330.

deficiency	cytology	ref ^α	genetics
Df(2L)TW1 ^β	38A7-B1;39C2-3	5, 8, 10	pr ⁻ -Bl ⁻ crc ⁺
Df(2L)TW2	37D2-E1;38E6-9	3, 8, 10	ref(2)P ⁻ -Bl ⁻ crc ⁺
Df(2L)TW3 ^γ	36F7-37A1;37B2-8	1, 7, 8, 10	Tjfi ⁻ -Roi ⁻ hk ⁺
Df(2L)TW9	37E2-F4;38A6-B1	3, 8, 10	Bl ⁺ ref(2)P ⁻ -pr ⁻
Df(2L)TW12	37E2-F4;39D1-2	5, 8, 10	ref(2)P ⁺ pr ⁻ -crc ⁻
Df(2L)TW50 ^δ	36E4-F1;38A6-7	1, 8, 10	rdo ⁺ M(2)36F ⁻ -ref(2)P ⁻ pr ⁺
Df(2L)TW65	37F5-38A1;39E2-F1	3, 4, 5, 8, 10	pr ⁻ -His ⁻
Df(2L)TW68	not confirmed	10	b ^D and strong M
Df(2L)TW84	37F5-38A1;39D3-E1	3, 4, 5, 8, 10	ref(2)P ⁺ pr ⁻ -His ⁻ (partial)
Df(2L)TW119 ^δ		8, 10	dI ⁻ -rdo ⁻ M(2)36F ⁺
Df(2L)TW130 ^ε	37B9-C1;37D1-2	2, 9, 10	hk ⁻ -fs(2)TW1 ⁻
Df(2L)TW137 ^δ	36C2-4;37B9-C1	1, 8, 10	dI ⁻ -msl ⁻ 1 ⁺ hk ⁺
Df(2L)TW150	37F5-38A1;38B2-C1	3, 8, 10	ref(2)P ⁺ l(2)37Fd ⁻ -l(2)A113 ⁻
Df(2L)TW158 ^ζ	37B2-8;37E2-F4	1, 3, 6, 8, 10	msl ⁻ 1 ⁺ hk ⁻ -l(2)37Ea ⁻ ref(2)P ⁺
Df(2L)TW161	38A6-B1;40A4-B1	3, 4, 5, 10	pr ⁻ -His ⁻ ; moderate M
Df(2L)TW201	36E-F;37A	8	l(2)36Da ⁺ rdo ⁻ -Tjfi ⁻ hk ⁺
Df(2L)TW202 ^δ	36F2-5;37A	8	rdo ⁺ M(2)36F ⁻ -Tjfi ⁻ hk ⁺
Df(2L)TW203 ^η	36E4-F1;37B9-C1	8, 9	l(2)36Da ⁺ rdo ⁻

deficiency	cytology	ref ^α	genetics
Df(2L)TW330^δ	36E4-F1;37D1-2	8	-hk ⁻ l(2)36Da ⁺ rdo ⁻ -fs(2)TW1 ⁻
^α	1 = Belote and Lucchesi, 1980, Genetics 96: 165-86; 2 = Brittnacher and Ganetzky, 1983, Genetics 103: 659-73; 3 = Gay and Contamine; 4 = Moore, Procnier, Cross, and Grigliatti, 1979, Nature 282: 312-14; 5 = Moore, Sinclair, and Grigliatti, 1983, Genetics 105: 327-44; 6 = Struhl, 1981, Nature 293: 36-41; 7 = Voelker and Langley, 1978, DIS 53: 185; 8 = T.R.F. Wright; 9 = Wright, Beerman, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, Chromosoma 83: 45-58; 10 = Wright, Hodgetts, and Sberald, 1976, Genetics 84: 267-85.		
^β	Associated with T(2;3)40;65 (García-Bellido, Moscoso del Prado, and Botas, 1983, Mol. Gen. Genet. 192: 253-63).		
^γ	Associated with, but separable from, T(2;3)53A;99 and In(3L)64C-F;71C (García-Bellido et al., 1983).		
^δ	Molecular biology: Distal DNA breakpoints given (Steward, McNally, and Schedl, 1984, Nature 311: 262-65).		
^ε	Phenotype hk over Df(2L)TW203. Molecular biology: Deletes the entire 100 kb of DNA which carries Ddc and includes the coding region for the genes l(2)37Ba-l(2)37Cf (Gilbert, Hirsh, and Wright, 1984, Genetics 106: 679-94).		
^ζ	esc not included in this deficiency (Struhl, 1981).		
^η	Induced Tft revertant. Phenotype rdo over Df(2L)TW119, hk over Df(2L)TW130 or Df(2L)VA23. Molecular biology: Proximal breakpoint distal to -59 kb (Ddc coordinates from Gilbert, Hirsh, and Wright, 1984).		

Df(2L)VA4: Deficiency (2L) Valencia

cytology: Df(2L)29E5;29E6.

origin: X ray induced.

references: Valencia, 1970, DIS 45: 37-78.

Df(2L)V106

cytology: Df(2L)37B;37B.

origin: X ray induced.

references: Valencia, 1970, DIS 45: 37-38.

Df(2L)VA

origin: ^γ ray induced [Df(2L)VA1-Df(2L)VA14, Df(2L)VA18]; ^γ ray or ethyl methanesulfonate induced [Df(2L)VA15-Df(2L)VA17, Df(2L)VA19, Df(2L)VA22]; diepoxybutane induced [Df(2L)VA20, Df(2L)VA21, Df(2L)VA23, Df(2L)VA24] or resulting from hybrid dysgenesis with Harwich P [Df(2L)VA25].

deficiency	cytology	ref ^α	genetics	mol. biol ^β
Df(2L)VA1		8	pr ⁻ ; lethal over Df(2L)TE37C-1	
Df(2L)VA2		8	pr ⁻ ; lethal over Df(2L)TE37C-1	
Df(2L)VA3		8	pr ⁻ ; lethal over Df(2L)TE37C-1	
Df(2L)VA4		8	pr ⁻ ; lethal over Df(2L)TE37C-1	
Df(2L)VA5		8	pr ⁻ ; lethal over Df(2L)TE37C-1	
Df(2L)VA6^γ	37D2-E1; 38F2-39A1	3, 9	pr ⁻	
Df(2L)VA7		9	pr ⁻ ; lethal over Df(2L)TE37C-1	
Df(2L)VA8	37D2-3; 38F5-39A1	3, 9	pr ⁻	
Df(2L)VA10		6, 8	pr ⁻ ; lethal over Df(2L)TE37C-1	
Df(2L)VA11		6, 8	pr ⁻ ; lethal over Df(2L)TE37C-1	
Df(2L)VA12	37C2-5; 38B2-C1	7, 8, 10	Ddc ⁺ l(2)37Cf ⁻ - pr ⁻	1
Df(2L)VA13	37C2-5; 38C2-D1	1, 8, 10	Ddc ⁺ l(2)37Cf ⁻ - pr ⁻	2

deficiency	cytology	ref ^α	genetics	mol. biol ^β
Df(2L)VA14		3, 7, 8	hk ⁻ - l(2)37Cf ⁻ l(2L)37Db ⁺	
Df(2L)VA15		2, 8	hk ⁻ - l(2)37Db ⁻	
Df(2L)VA16		3, 4, 8	M(2)36F ⁺ hk ⁻ - fs(2)TW1 ⁻ l(2)37Db ⁺	
Df(2L)VA17	37B9-C1; 37F5-38A1	5, 8, 10	amd ⁺ Ddc ⁻ - fs(2)TW1 ⁻	3
Df(2L)VA18	36C4-D1; 37C2-5	5, 8	dl ⁺ l(2)36Da ⁻ - Ddc ⁻ l(2)37Ca ⁺	4
Df(2L)VA19	37C2-D1; 38A6-B1	8	l(2)37Cf ⁻	5
Df(2L)VA20	37B2-8; 37D2-E1	3, 4, 8	hk ⁻ - fs(2)TW1 ⁻	
Df(2L)VA21	36D1-F1; 37C2-D1	4, 8	dl ⁺ l(2)36Da ⁻ - l(2)37Cg ⁻ fs(2)TW1 ⁺	6
Df(2L)VA22	{36E4-F1; 37D2-E1}	8	l(2)36Da ⁺ rdo ⁻ - l(2)37Cf ⁻ l(2)37Db ⁺	
Df(2L)VA23^δ	37B9-C1; 37D2-E1	4, 8	hk ⁻ - l(2)37Db ⁻	
Df(2L)VA24		4, 8	hk ⁻ - l(2)37Ea ⁻	
Df(2L)VA25^ε		4, 8	M(2)36F ⁺ hk ⁻ - fs(2)TW1 ⁻	
^α	1 = Bishop; 2 = Black; 3 = Brittnacher and Ganetzky, 1983, Genetics 103: 659-73; 4 = Cecil; 5 = Cecil and Wade; 6 = Steward; 7 = Tomsett; 8 = T.R.F. Wright; 9 = T.R.F. Wright and E.Y. Wright; 10 = Wright, Beerman, Marsh, Bishop, Steward, Black, Tomsett, and Wright, 1981, Chromosoma 83: 45-58.			
^β	Ddc DNA coordinates from Gilbert, Hirsh, and Wright, 1984, Genetics 106: 679-84: 1 = Distal breakpoint between +25.5 and +28.1 kb; 2 = Distal breakpoint between +30.8 and +35 kb; 3 = Distal breakpoint within the Ddc gene between +1.4 and 2.4 kb removing the first 5' exon; 4 = Proximal breakpoint between +5.1 and +5.6 kb; 5 = Distal breakpoint between +30 and +32 kb (E.S. Pentz); 6 = Proximal breakpoint between +27 and +30 kb (E.S. Pentz).			
^γ	Viable over Df(2L)VA25 but with wings held down and out.			
^δ	Viable over Df(2L)TW203 but shows hk phenotype.			
^ε	Wings held outstretched when over l(2)37Db and held down and outward when over Df(2L)VA6.			

Df(2L)VV5

cytology: Df(2L)22A3;22E4.

origin: X ray induced.

discoverer: Velissariou.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.

Df(2L)VV9

cytology: Df(2L)30B3;30C9.

origin: ^γ ray induced.

discoverer: Velissariou.

references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

Df(2L)W

cytology: Df(2L)35A2-3;35B3-5.

origin: Spontaneous in natural population (Greece).

synonym: l(2)AR8.

references: Woodruff and Ashburner, 1979, Genetics 92: 117-32.

genetics: Deficient for l(2)34Fa-Adh.

Df(2L)X1 : see Df(2L)L138D

Df(2L)X14 : see Df(2L)L138D

Df(2L)XR : see Df(2L)L138D

Df(2R)017

cytology: *Df(2R)56F5;56F15*.

origin: Induced by ethyl methanesulfonate.

references: Duttagupta and Shellenbarger, 1980, Development and Neurobiology of *Drosophila* (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York and London, pp. 25-33.

genetics: Includes *l(2)56Fd*, *l(2)56Fe*, *l(2)56Ff*, and *l(2)56Fg*.

Df(2R)2J: see *Df(2R)A''*

Df(2R)6C: see *Df(2R)A''*

Df(2R)7B: see *Df(2R)Pcl17B*

Df(2R)10G: see *Df(2R)A''*

Df(2R)11B: see *Df(2R)Pcl11B*

Df(2R)14C: see *Df(2R)A''*

Df(2R)42

cytology: *Df(2R)42C3-8;42D2-3*.

origin: Probably X ray induced. Found on chromosome with *vg^S*.

discoverer: Bridges, 36b.

references: Morgan, Bridges, and Schultz, 1938, Year Book - Carnegie Inst. Washington 37: 304-9.

genetics: Deficient for no tested loci. Homozygous lethal.

Df(2R)43A

cytology: *Df(2R)43A2;43B1*.

Df(2R)44CE

cytology: *Df(2R)44C;44E1-4*.

discoverer: Yannopoulos.

references: Hooper and Scott, 1989, Cell 59: 751-65.

genetics: Deficient for *tuf*.

Df(2R)173

cytology: Not visible.

origin: Induced by ethyl methanesulfonate.

references: Duttagupta and Shellenbarger, 1980, Development and Neurobiology of *Drosophila* (Siddiqi, Babu, Hall, and Hall, eds.). Plenum Press, New York and London, pp. 25-33.

genetics: Includes *l(2)56Fa*, *l(2)56Fb*, and *M(2)56F*.

Df(2R)193A

cytology: *Df(2R)44C;44C*.

Df(2R)A

cytology: Deficient for about half of the 2R heterochromatin including *rl* (Hilliker, 1976).

origin: γ -ray-induced reconstitution of chromosome 2 by detachment of compound second chromosomes (Hilliker and Holm, 1975).

references: Hilliker and Holm, 1975, Genetics 81: 705-21. Hilliker, 1976, Genetics 83: 765-82.

genetics: Deficient for *rl*. Complements *Df(2R)M41A4* and *Df(2R)M41A8* but not *Df(2R)M41A10* (Hilliker, 1976).

other information: Thirty-three such deficiencies recovered.

Df(2R)A'

cytology: Deficient for most of the 2R heterochromatin including *rl*. Extends distal to *Df(2R)A* and *Df(2R)A''* (Hilliker, 1976).

origin: γ -ray-induced reconstitution of chromosome 2 by detachment of compound second chromosomes (Hilliker and Holm, 1975).

references: Hilliker and Holm, 1975, Genetics 81: 705-21. Hilliker, 1976, Genetics 83: 765-82.

genetics: Deficient for *rl* and *M(2)41A*. Lethal over *Df(2R)M41A4*, *Df(2R)M41A8*, and *Df(2R)M41A10* (Hilliker, 1976).

Df(2R)A''

cytology: *Df(2R)41* (heterochromatin). Deficient for about two-thirds of the 2R heterochromatin including *rl*. Extends distal to *Df(2R)A* (Hilliker, 1976).

origin: γ -ray-induced reconstitution of chromosome 2 by detachment of compound second chromosomes (Hilliker and Holm, 1975).

references: Hilliker, 1976, Genetics 83: 765-82.

Ganetzky, 1977, Genetics 86: 321-55.

genetics: Deficient for *rl*. Complements *Df(2R)M41A4* and *Df(2R)M41A8* but not *Df(2R)M41A10* (Hilliker, 1976).

***Df(2R)a-ba2: Deficiency (2R) arc-broad angular**

cytology: *Df(2R)58D5-6;58D7-8*.

origin: Spontaneous.

discoverer: Goldschmidt.

references: 1945, Univ. Calif. (Berkeley) Publ. Zool. 49: 363-73, 388-89.

genetics: Associated with *a^{ba2}*.

Df(2R)AA21: see In(2R)AA21

Df(2R)B

cytology: Deficient for less than half of the 2R heterochromatin not including *rl* (Hilliker, 1976).

origin: γ -ray-induced reconstitution of chromosome 2 by detachment of compound second chromosomes (Hilliker and Holm, 1975).

references: Hilliker and Holm, 1975, Genetics 81: 705-21. Hilliker, 1976, Genetics 83: 765-82.

genetics: Not deficient for *rl*. Complements *Df(2R)M41A4* and *Df(2R)M41A8* but not *Df(2R)M41A10*.

Df(2R)Ba

cytology: *Df(2R)60E3;60E6*

origin: γ ray induced.

discoverer: Moscoso del Prado.

synonym: *Art²*, *Ba^{MP}*.

references: Sunkel and Whittle, 1987, Wilhelm Roux's Arch. Dev. Biol. 196: 124-32.

Cohen, Brönnner, Küttner, Jürgens, and Jäckle, 1989, Nature 338: 432-34.

Cohen and Jürgens, 1989, EMBO J. 8: 2045-55.

genetics: Deficient for *Ba*; entire putative transcription unit deleted. Heterozygote shows partial transformation of arista to tarsal segments. Dominant phenotype suppressed in either *Pcl¹⁰* or *Df(2R)en28* heterozygotes. *Df(2R)Ba/+* shows almost complete suppression of the extra-sex combs phenotypes of *Pc^{4/+}*, *Pc^{T7/+}*, *Pcl^{10/+}*, *Pcl^{11/+}*, *Antp^{Scx/+}*, *Msc/+*, and *Msc^{T3/+}*.

molecular biology: Deleted for the region from approximately coordinate 90 kb through 180 kb and beyond on the molecular map of Cohen *et al.*

Df(2R)Ba4: Deficiency (2R) Brista

cytology: *Df(2R)60D1-2;60F5*.
origin: EMS-induced.
references: Sunkel, 1983, Ph.D. Thesis, University of Sussex.
genetics: Deficient from *sp* to *2R* tip. Heterozygotes have missing legs, and abnormal eye facets. Lethal over *Ba*.

Df(2R)Ba-MP (J. Kennison)

cytology: *Df(2R)60E3-4;60E5-6*.
origin: X ray induced.
discoverer: Moscoso del Prado.
synonym: *Scnp*^{AL}; *Art*²; *Dll*^{MP}.
genetics: Recessive lethal. Heterozygotes show partial transformation of arista to tarsal segments.
molecular biology: Deletion removes the entire *Ba* coding region [Cohen, Brönnner, Küttner, Jürgens, and Jackle, 1989, Nature (London), 338: 432-34].

Df(2R)BP7

cytology: *Df(2R)57E;58A1*.
origin: Constructed from *T(2;3)P49 = T(2;3)57D;81F* and *T(2;3)BTD73 = T(2;3)58A1;81F*.
references: Schejter, and Shilo, 1989, Cell 56: 1093-1104.
genetics: Does not complement lethal mutations of *Egfr*.

Df(2R)bw^{AL}Cy^R: see *In(2R)bw^{AL}Cy^R*

Df(2R)bw^{VDe1L}Cy^R: see *In(2R)bw^{VDe1L}Cy^R*

Df(2R)bw^{VDe2L}Cy^R: see *In(2R)bw^{VDe2L}Cy^R*

Df(2R)bw5: Deficiency (2R) brown

cytology: *Df(2R)59D10-E1;59E4-F1*.
origin: Spontaneous.
discoverer: Mohr, 31k28.
references: Wright, 1970, Advances in Genetics 15: 262-85.
genetics: Deficient for *bw*. Homozygous lethal in embryo (Tsai, 1955, Genetics 40: 601).

Df(2R)bw-D23

cytology: *Df(2R)59D4-5;60A1-2* (Richards).
origin: X ray induced.
synonym: *Df(2R)bw^{Drv23}*; *Df(2R)bw^{D+R23}*.
references: Simpson, 1983, Genetics 105: 615-32.
genetics: Deficient for *bw* but not for *twi*.

Df(2R)bw-D31

cytology: *Df(3R)59B6-8;60A8-16* (Richards).
origin: X ray induced.
synonym: *Df(2R)bw^{Drv31}*; *Df(2R)bw^{D+R31}*.
references: Simpson, 1983, Genetics 105: 615-32.
genetics: Deficient for *bw* and *twi*.

Df(2R)bw-GY

cytology: *Df(2R)59B;59E*.
origin: Recovered as apparent recombinant from a test cross of a dysgenic-type male that was heterozygous for a maternally derived *dp b cn bw* second chromosome and paternally derived autosome extracted from a natural population and capable of promoting male recombination. An MRF (Male Recombination Factor) chromosome, *MRF23.5*, was utilized. The duplication was recovered from a male heterozygous for *MRF23.5*.
references: Yannopoulos, Stamatis, Zacharopoulou, and Pelecanos, 1981, Mutat. Res. 83: 383-93.

genetics: Deficient for *bw*.

Df(2R)bw-K

cytology: *Df(2R)59D2-5;59E1-3*.
origin: Spontaneous.
references: Koliantz, 1971, DIS 46: 52.
genetics: Deficient for *bw*.

***Df(2R)bw-R40: Deficiency (2R) brown-Rearranged**

cytology: *Df(2R)59C5-6;59E2-3*.
origin: X ray induced.
discoverer: Slatis.
references: 1955, Genetics 40: 5-23.

Df(2R)bw-S46

cytology: *Df(3R)59D8-11;60A7* (Richards).
origin: X ray induced.
references: Simpson, 1983, Genetics 105: 615-32.
genetics: Deficient for *bw* but not *twi*.

Df(2R)C113

cytology: *Df(2R)41A;41A*.
references: Tano, 1966, Jpn. J. Genet. 41: 299-308.
genetics: Deficient for *l(2)41Ae*. Complemented by *M(2)41A* and *M(2)39F* but not by *Df(2R)r110A*.

Df(2R)CA53

cytology: *Df(2R)43E6;44B5-9*.
origin: γ ray induced.
discoverer: Durrant.
genetics: Deficient for *cn*. Ocelli small in heterozygotes.

Df(2R)CA58

cytology: *Df(3R)43A3;43F6*.
origin: γ ray induced.
discoverer: Ashburner.

Df(2R)CC2

cytology: *Df(2R)57C3;57C6*.
origin: γ ray induced.
references: O'Donnell, Boswell, Reynolds, and Mackay, 1989, Genetics 121: 273-80.
genetics: Deficient for *l(2)57Cb-Pu*.

Df(2R)cn: Deficiency (2R) cinnabar

origin: Most deficiencies recovered as apparent *cn* double recombinants from test crosses of dysgenic-type males that were heterozygous for a maternally derived *dp b cn bw* second chromosome and paternally derived autosomes extracted from natural populations and capable of promoting male recombination. Rearranged derivatives occurred both singly and in clusters. Two MRF (Male Recombination Factor) chromosomes, *MRF23.5* and *MRF31.1*, were utilized. Most deficiencies of MRF origin were recovered from males heterozygous for *MRF23.5*; *1b* and *2b* recovered from males heterozygous for *MRF31.1*.

deficiency	cytology	origin	ref ^α
<i>Df(2R)cn1</i>	42E;44A	MRF	3
<i>Df(2R)cn1b</i>	43D;44B		1
<i>Df(2R)cn2</i>	42E;43F	MRF	3
<i>Df(2R)cn2b</i>	42B;43E		1
<i>Df(2R)cn3</i>	42E;44B	MRF	3
<i>Df(2R)cn4</i>	43D;44C	MRF	3
<i>Df(2R)cn5</i>	43E;43E	MRF	3
<i>Df(2R)cn6</i>	43D;43E	MRF	3
<i>Df(2R)cn7</i>	43D;44C +	MRF	3

deficiency	cytology	origin	ref ^α
Df(2R)cn8	<i>In(2R)47C;54B</i> 43C;44B	MRF	3
Df(2R)cn9	42E;44C	MRF	3
Df(2R)cn10	43C;44C + <i>In(2R)44A;54B</i>	MRF	3
Df(2R)cn11	43E;43E	MRF	3
Df(2R)cn12	43E;43F	MRF	3
Df(2R)cn13	43D;43E + <i>In(2R)50F;54B</i>	MRF	3
Df(2R)cn14	43E + <i>In(3R)37C;59C</i>	MRF	3
Df(2R)cn15	42E;43F	MRF	3
Df(2R)cn16	42E;43E	MRF	3
Df(2R)cn17	43C;44B + <i>T(2;3)57C;69E</i>	MRF	3
Df(2R)cn18	43E;44B + <i>In(2R)43B;54A</i>	MRF	3
Df(2R)cn19	43E;43F + <i>In(2R)57C;59C</i>	MRF	3
Df(2R)cn20	43D;44C	MRF	3
Df(2R)cn21	43E;43F	MRF	3
Df(2R)cn81	43D1-7;44A3-7	γ ray	2
Df(2R)cn83c	43C5-D1;44B6-C1		1
Df(2R)cn87a	42B4-C1;43F-44A1		1
Df(2R)cn87e	42B4-C1;43F-44A1		1
Df(2R)cn88b	42A;42E		1
Df(2R)cn-h1	43B1-C1;43E6-15		1
Df(2R)cn-h2	43D2-43E3;43E15-F1		1
Df(2R)cn-h3	43C1-D1;43E15-F1		1

^α 1 = Ashburner; 2 = Gubb 81il; 3 = Yannopoulos, Stamatis, Zacharopoulou, and Pelecanos, 1981, *Mutat. Res.* 83: 383-93.

Df(2R)Cy-sf: Deficiency (2R) Curly-safranin

origin: X ray induced.

references: Davis and MacIntyre, 1988, *Genetics* 120: 755-66.

genetics: Deficient for *sf*.

Df(2R)Cy^Lbw^{VDe1R}: see *In(2R)Cy^Lbw^{VDe1R}*

Df(2R)Cy^Lbw^{VDe2R}: see *In(2R)Cy^Lbw^{VDe2R}*

Df(2R)D17

cytology: *Df(2R)57B5;58B1-2*.

discoverer: O'Donnell.

references: Schüpbach and Wieschaus, 1986, *Wilhelm Roux's Arch. Dev. Biol.* 195: 302-17.

O'Donnell, Boswell, Reynolds, and Mackay, 1989, *Genetics* 121: 273-80.

genetics: Deficient for *tud* (Schüpbach and Wieschaus, 1980), and *Pu* (O'Donnell *et al.*, 1989).

Df(2R)Egfr: Deficiency (2R)

Epidermal growth factor

references: Price, Clifford, and Schüpbach, 1989, *Cell* 56: 1085-92.

deficiency	cytology	origin	synonym	genetics
Df(2R)Egfr3	57E1;57F11	EMS	<i>flb</i> ^{3F18}	<i>mat(2)N⁻-Egfr⁻</i>
Df(2R)Egfr5	57D2-8;58D1	γ ray	<i>Df(2R)top</i> ^{5D}	<i>mat(2)N⁻-Egfr⁻</i>
Df(2R)Egfr18	57E4-11;57F1	γ ray	<i>Df(2R)top</i> ^{18A}	<i>Egfr⁻</i>

Df(2R)en: Deficiency (2R) engrailed

genetics: Deficient for *en*. Enhance *Pc* (Sato, Russell, and Denell, 1983, *Genetics* 105: 357-70).

deficiency	cytology	origin ^α	discoverer ^β	ref ^γ
Df(2R)en28	complex; see <i>In(2R)en</i> ²⁸	1	2	1, 2, 3, 7
Df(2R)en30^δ	48A3-4;48C6-8	1	2	1, 2, 3, 5, 7

deficiency	cytology	origin ^α	discoverer ^β	ref ^γ
Df(2R)en-A^ε	47D3;48A5-6	2	1	2, 3, 4
Df(2R)en-B^ε	47E3;48B2	2	1	2, 4
Df(2R)en-SFX31^ζ	48A1;48B5-7	2	3	5, 6
^α	1 = γ rays; 2 = X rays;			
^β	1 = Ashburner; 2 = Eberlein and Russell; 3 = Kornberg.			
^γ	1 = Eberlein, 1982, <i>Genetics</i> 100: s21-22; 2 = Eberlein and Russell, 1983, <i>Dev. Biol.</i> 100: 227-37; 3 = Epper and Sánchez, 1983, <i>Dev. Biol.</i> 100: 387-98; 4 = Gubb, 1985, <i>Wilhelm Roux's Arch. Dev. Biol.</i> 194: 236-46; 5 = Kuner, Nakanishi, Ali, Drees, Gustavson, Theis, Kauvar, Kornberg, and O'Farrell, 1985, <i>Cell</i> 42: 309-16; 6 = Kornberg, 1981, <i>Proc. Nat. Acad. Sci. USA</i> 78: 1095-99; 7 = Russell and Eberlein, 1979, <i>Genetics</i> 92: s109.			
^δ	Homozygous viable. Left breakpoint between DNA coordinates 13 and 20 kb (Kuner <i>et al.</i> , 1985). Claimed to be a viable allele by Kuner <i>et al.</i> despite claimed deficiency and failure to test homozygote by Eberlein and Russell. (Kuner <i>et al.</i> , 1985).			
^ε	Also deficient for <i>shn</i> and <i>sha</i> (Nüsslein-Volhard, Wieschaus, and Kluding, 1984, <i>Wilhelm Roux's Arch. Dev. Biol.</i> 193: 267-82).			
^ζ	Homozygous lethal extends from coordinate -95 to +115 (Kuner <i>et al.</i> , 1985).			

Df(2R)ES1

cytology: Distal break at 60F1-2.

references: Côté, Preiss, Haller, Schuh, Kienlin, Seifert and Jäckle, 1987, *EMBO J.* 6: 2793-801.

genetics: Deficiency for *zip-gsb*.

molecular biology: 60F1-2 break at +54 kb.

Df(2R)eve: Deficiency (2R) even-skipped

cytology: *Df(2R)46C3-4;46C9-11*.

discoverer: Nüsslein-Volhard.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 267-82.

Nüsslein-Volhard, Kluding, and Jürgens, 1985, *Cold Spring Harbor Symp. Quant. Biol.* 50: 145-54.

genetics: Deficient for *eve*.

Df(2R)exu

cytology: *Df(2R)57A2;57B1*.

genetics: Deficient for *exu*.

Df(2R)F4-1

cytology: *Df(2R)57A3;57B1-2*.

genetics: Deficient for *exu*.

Df(2R)F36

cytology: *Df(2R)57B16-17;57C6-7* (Schüpbach and Wieschaus, 1986).

origin: γ ray induced.

synonym: *Df(2R)Pu-rF36*.

references: Mackay, Reynolds, and O'Donnell, 1985, *Genetics* 111: 885-904.

Schüpbach and Wieschaus, 1986, *Wilhelm Roux's Arch. Dev. Biol.* 195: 302-17.

genetics: Deficient for *l(2)57Ba-Pu*. GTP cyclohydrolase activity lost in eye but present elsewhere.

Df(2R)gsb: Deficiency (2R) gooseberry

cytology: *Df(2R)60E9-10;60F1-2*.

genetics: Deficient for *gsb*.

Df(2R)IIX62

cytology: *Df(2R)60E9-10;60F1-2*.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 267-82.

Côté, Preiss, Haller, Schuh, Kienlin, Seifert, and Jäckle, 1987, *EMBO J.* 6: 2793-2801.

genetics: Deficient for *gsb* and *zip*. *Df(2R)gsb/Df(2R)SB1*

embryos express *gsb* but not *zip* (Côté *et al.*, 1987).

molecular biology: Proximal breakpoint at -55 to -49 kb and distal breakpoint at +40 to +42.5 kb (Côté *et al.*, 1987), with the proximal breakpoint of *Df(2R)SB1* approximately at 0.

Df(2R)Jp7

cytology: *Df(2R)52F;53A*.

discoverer: Saxton.

references: McDonald and Goldstein, 1990, Cell 61: 991-1000.

genetics: Deficient for *Kin*.

Df(2R)K11

cytology: *Df(2R)57C3-4;57D7-8*.

origin: γ ray induced.

references: O'Donnell, Boswell, Reynolds, and Mackay, 1989, Genetics 121: 273-80.

genetics: Deficient for *Pu*.

Df(2R)KL

origin: X ray.

references: Davis and MacIntyre, 1988, Genetics 120: 755-66.

genetics: Deficient for *c*.

deficiency	cytology
<i>Df(2R)KL9</i>	52D3;52D7-9
<i>Df(2R)KL32</i>	52C5-D1;52E2-5
<i>Df(2R)KL69</i>	52D2-3;52D9-E1
<i>Df(2R)KL99</i>	52B5-C1;52E2-5

Df(2R)Kr: Deficiency (2R) Krüppel

genetics: Deficient for *Kr*.

deficiency	synonym	cytology	origin	ref ^{α}
<i>Df(2R)Kr1</i>		60F2;60F5	spont	2, 3, 4
<i>Df(2R)Kr4</i>	<i>Kr^{B80}</i>	60F2;60F5	X ray	3, 4
<i>Df(2R)Kr6</i>	<i>Kr^{6A69}</i>	60F3;60F5	EMS	3, 4
<i>Df(2R)Kr10</i> ^{β}	<i>Kr^{SB1}</i>	60E10;60F5	X ray	3
<i>Df(2R)Kr14</i>	<i>Kr^{L14}</i>	60F2;60F5	EMS	1, 3

^{α} I = Jürgens, Kluding, Nüsslein-Volhard, and Wieschaus, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95; 2 = Nüsslein-Volhard and Wieschaus, 1980, Nature 287: 795-801; 3 = Preiss, Rosenberg, Kienlin, Seifert, and Jäckle, 1985, Nature 313: 27-32; 4 = Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Dev. Biol. 104: 172-86.

^{β} Recovered as reversion of *If*. The deficient region is replaced by transposed bands probably derived from chromosome 4.

Df(2R)I(2)30

cytology: *Df(2R)52D6-7;52A12-B1*.

origin: Induced by ethyl methanesulfonate.

discoverer: Doane.

references: Davis and MacIntyre, 1988, Genetics 120: 755-66.

genetics: Homozygous lethal.

Df(2R)L4: Deficiency (2R) Lobe

cytology: *Df(2R)51A2;52A12-B1*.

origin: X ray induced.

synonym: *Df(2R)L^{rv4}*; *Df(2R)L^{+R4}*.

references: Baker and Ridge, 1980, Genetics 94: 383-423.

genetics: Deficient for *L*. No complementation of *tra-2* for male sterility (Belote and Baker, 1983, Dev. Biol. 95: 512-17).

Df(2R)L7

cytology: *Df(2R)50D;51B5-C2*.

origin: X ray induced.

synonym: *Df(2R)L^{rv7}*; *Df(2R)L^{+R7}*.

references: Baker and Ridge, 1980, Genetics 94: 383-423.

genetics: Deficient for *L*. No complementation of *tra-2*.

Df(2R)L48

cytology: *Df(2R)51A1;51B4* (MacIntyre).

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

genetics: Deficient for *Asx* and *L*.

Df(2R)M41A: Deficiency (2R) Minute 41A

origin: X ray induced.

genetics: Homozygous lethal.

deficiency	cytology	synonym	ref ^{α}	genetics
<i>Df(2R)M41A</i>	salivaries appear normal	<i>M(2)S2</i>	2, 3, 4	<i>M(2)41A⁻-tk⁻</i>
<i>Df(2R)M41A4</i>	salivaries appear normal	<i>M(2)S2⁴</i>	1, 2, 3, 4, 5	<i>l(2)41Ae⁻-ap⁻</i> ; includes <i>Dip-A</i>
<i>Df(2R)M41A8</i>	salivaries appear normal	<i>M(2)S2⁸</i>	1, 2, 3, 5	<i>l(2)41Ae⁻-stw⁻ap⁺</i>
<i>Df(2R)M41A10</i>	41A;mitotic 2R 3/4 normal size	<i>M(2)S2¹⁰</i>	1, 3, 4, 5	<i>l(2)41Aa⁻-l(2)41Ah⁻stw⁺</i>
<i>Df(2R)M41A50j</i>		<i>M(2)S2^{50j}</i>	5	<i>rl⁺l(2)41Ae⁻stw⁺</i>
<i>*Df(2R)M41A-D</i>	41A;41C; see	<i>*M(2)S2^D</i>		
<i>*Df(2R)M41A-vg11^{β}</i>		<i>M(2)S2^{vg11}</i>	2, 3	<i>rl⁻-msf⁻</i>

^{α} 1 = Hilliker and Holm, 1975, Genetics 81: 705-21; 2 = Morgan, Bridges, and Schultz, 1938, Year Book - Carnegie Inst. Washington 37: 306; 3 = Morgan, Schultz, Bridges, and Curry, 1939, Year Book - Carnegie Inst. Washington 38: 273-77; 4 = Morgan, Schultz, and Curry, 1940, Year Book - Carnegie Inst. Washington 39: 251-55; 5 = Tano, 1966, Jpn. J. Genet. 41: 299-308; 6 = Voelker and Langley, 1978, Genetica 49: 233-36.

^{β} Arose simultaneously with *vg¹¹*.

***Df(2R)M58F**

cytology: *Df(2R)57F11-58A1;58F8-59A1*.

origin: Spontaneous.

discoverer: Bridges, 23g15.

synonym: **Df(2R)M-l*.

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 231.

Bridges, 1937, Cytologia (Tokyo), Fujii Jub., Vol. 2: 745-55.

genetics: Deficient for *px*, *l(2)Su(H)*, *M(2)58F*, and probably *a*. Homozygote dies in egg stage; eggs recognizable by thin chorion (Li, 1927, Genetics 12: 1-58).

Df(2R)M60E

cytology: *Df(2R)60E2-3;60E11-12*.

origin: X ray induced.

discoverer: Schultz, 33a7.

synonym: *Df(2)M-33a*; *Df(2R)M-c33a*; *M(2)115*.

references: Bridges, 1937, Cytologia (Tokyo), Fujii Jub., Vol. 2: 745-55.

genetics: Deficient for *M(2)60E*. Homozygous lethal. Fairly strong *M* with good viability in combination with *Binsn* and *In(2LR)bw^{v32g}* (Campos-Ortega and Waitz, 1978, Wilhelm Roux' Arch. Entwicklungsmech. Org.

184: 155-70).

Df(2R)MP1

cytology: *Df(2R)57C1-2;57D1-2*.

origin: γ ray induced.

references: O'Donnell, Boswell, Reynolds, and Mackay, 1989, *Genetics* 121: 273-80.

***Df(2R)Np: Deficiency (2R) Notopleural**

cytology: *Df(2R)44F1-2;45E1-2* (Bridges).

origin: Spontaneous.

discoverer: Nichols-Skoog, 33b20.

references: Bridges, Skoog, and Li, 1936, *Genetics* 21: 788-95 (fig.).

Li, 1936, *Peking Nat. Hist. Bull.* 11: 39-48.

genetics: Not deficient for *cn*, *blo*, or *en*. *Df(2R)Np/T(2;3)dp* lethal.

Df(2R)Ore-R: Deficiency (2R) Oregon-R

cytology: *Df(2R)60F2-3;60F5*.

origin: Naturally occurring in Oregon-R stock but not in Oregon-R-C stock (Roberts, 1974, *Genetics* 77: s54-55).

discoverer: Bridges, 3615.

genetics: No detectable phenotypic effect in homozygote.

Df(2R)P: Deficiency (2R) Pale

cytology: *Df(2R)58E3-F1;60D14-E2*.

origin: Aneuploid segregant from *Tp(2;3)P/ +*.

Df(2R)P32

cytology: *Df(2R)43A3;43A6* (Sinclair).

references: Schüpbach, and Wieschaus, 1989, *Genetics* 121: 101-17.

genetics: Uncovers *tor*, *scra*, *mat(2)cellIRE43*.

Df(2R)PC4

cytology: *Df(2R)55A;55F* (Jürgens).

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 267-82.

Schüpbach and Wieschaus, 1986, *Wilhelm Roux's Arch. Dev. Biol.* 195: 302-17.

genetics: Deficient for *thr*, *stau*, *Pcl* (Jürgens, 1985, *Nature* 316: 153-55); also for *Bc*, *Dox1*, *ff*, and *wt* (Deng and Rizki, 1988, *Proc. Int. Congr. Genet.*, 16th, p. 192).

Df(2R)PC18

cytology: *Df(2R)57B16-17;57C6-7*.

origin: γ ray induced.

references: O'Donnell, Boswell, Reynolds, and Mackay, 1989, *Genetics* 121: 273-80.

genetics: Deficient for *l(2)57Ba-Pu*.

Df(2R)PC29

cytology: *Df(2R)55C1-2;56B1-2*.

Df(2R)PC66

cytology: *Df(2R)55D2-E1;55E3-4*.

Df(2R)Pcl: Deficiency (2R) Polycumblike

genetics: Deficient for *Pcl*. When heterozygous with wild type, flies carrying the *Pcl* deficiency show transformation of second and third legs to first legs.

deficiency	cytology	discoverer	synonym	ref α
Df(2R)Pcl7B ^{β}	<i>54E8-F1;55B9-C1</i>	E. B. Lewis	<i>Df(2R)7B</i>	1
Df(2R)Pcl11B ^{β}	<i>54F6-55A1;55C1-3</i>	E.B. Lewis	<i>Df(2R)11B</i>	1,2
Df(2R)Pcl-W5	<i>55A-B;55C</i>	J. Williams		2

- α 1 = Duncan, 1982, *Genetics* 102: 49-70; 2 = Sato, Russell, and Denell, 1983, *Genetics* 105: 357-70.
- β Enhances *Pcl*³.
- γ Also deficient for *Bc*, *Dox1*, and *ff* (Deng and Rizki, 1988, *Proc. Int. Congr. Genet.*, 16th, p. 192).
- δ Also deficient for *ff* and *wt* (Deng and Rizki, 1988).

Df(2R)PF1

cytology: *Df(2R)57C5;57D1*.

origin: DEB induced.

references: O'Donnell, Boswell, Reynolds, and Mackay, 1989, *Genetics* 121: 273-80.

genetics: Deficient for *Pu*.

Df(2R)pk: Deficiency (2R) prickle

origin: X ray induced.

discoverer: Gubb.

deficiency	cytology	ref α	genetics
Df(2R)pk78b			<i>pwn⁻-sple⁻</i>
Df(2R)pk78d			<i>pwn⁻-sple⁻</i>
Df(2R)pk78k	<i>42E3;43C3</i>	1, 2	<i>pk⁻-so⁻cn⁺</i>
Df(2R)pk78n		2	<i>pwn⁻-sple⁻</i>
Df(2R)pk78r	<i>43A1;43C7</i>	2	<i>pwn⁻-sple⁻</i>
Df(2R)pk78s	<i>42C1-7;</i> <i>43F5-8 +</i> <i>ln(2R)42C1-7;</i> <i>59F5-8</i>	1, 2	<i>pk⁻-cn⁻</i>
Df(2R)pk78w			<i>pwn⁻-sple⁻</i>

- α 1 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, *DIS* 56: 186-91; 2 = Gubb and Garcia-Bellido, 1982, *J. Embryol. Exp. Morphol.* 68: 37-57.

Df(2R)PK1

cytology: *Df(2R)57C5;57F6*.

origin: DEB induced.

references: O'Donnell, Boswell, Reynolds, and Mackay, 1989, *Genetics* 121: 273-80.

genetics: Deficient for *Pu-Egfr*.

Df(2R)PI12

cytology: *Df(2R)57C4;57D8-9*.

origin: DEB induced.

references: O'Donnell, Boswell, Reynolds, and Mackay, 1989, *Genetics* 121: 273-80.

McLean, Boswell, and O'Donnell, 1990, *Genetics* 126: 1007-19.

genetics: Deficient for *Pu-l(2)57Db*.

Df(2R)PI13

cytology: *Df(2R)57B13-14;57D8-9* (O'Donnell).

references: Schüpbach, and Wieschaus, 1989, *Genetics* 121: 101-17.

genetics: Uncovers *tud*.

Df(2R)PL3

cytology: *Df(2R)57B18-20;57D8-9*.

origin: DEB induced.

discoverer: O'Donnell.

references: Schüpbach and Wieschaus, 1986, *Wilhelm Roux's Arch. Dev. Biol.* 195: 302-17.

O'Donnell, Boswell, Reynolds, and Mackay, 1989, *Genetics* 121: 273-80.

genetics: Deficient for *tud* and *Pu*.

Df(2R)Pu-D17

cytology: *Df(2R)57B4;58B*.

Df(2R)Pu^{rF36}: see *Df(2R)F36*

Df(2R)Px: Deficiency (2R) Plexate

deficiency	cytology	origin	discov ^α	ref ^β	comments
Df(2R)Px1	60B8-10; 60D1-2	spont	1	1	<i>Fo⁺ sp⁻ -ba⁻</i>
Df(2R)Px2	60C5-6; 60D9-10	X ray	3	1	<i>sp⁺ ba⁻ -Pin⁻ Kr⁺</i>
Df(2R)Px4	[see <i>In(2LR)Px⁴</i>]		4	2	<i>Px⁻ -ba⁻; vesicles</i>
*Df(2R)Px5		spont in <i>In(2LR)bw^{V1}</i>	4	4	<i>sp⁺ bs⁻ -ba⁻; vesicles; veins more regular than in <i>Px¹</i></i>
*Df(2R)Px-Bsl^γ		X ray	2	3	thick veins; blisters

^α 1 = Bridges, 22f6; 2 = Oliver, 29b1; 3 = Schultz, 3211; 4 = Thompson, 56f, 57.

^β 1 = Bridges, 1937, Cytologia, Fujii Jub. Vol. 2: 745-55; 2 = Burdick, 1956, DIS 30: 69; 3 = Oliver, 1939, DIS 11: 47; 4 = Thompson, 1963, DIS 38: 28.

^γ Probably a *Px* deficiency (Bridges). Synonym: *B51*.

Df(2R)R7

cytology: Complex.

origin: γ ray induced.

references: Lasko and Pardue, 1988, Genetics 120: 495-502.

genetics: Deficient for *l(2)49Da - l(2)49Ea*.

Df(2R)rl: Deficiency (2R) rolled

deficiency	cytology	origin ^α	ref ^β	genetics
Df(2R)rl1	41A	2	2	<i>rl⁻ stw⁺</i>
Df(2R)rl1G	41A	1	1, 2	<i>l(2)41Ab⁻ -rl⁻</i>
Df(2R)rl2J	41A+	1	1, 2	<i>rl⁻ -uex⁻</i>
Df(2R)rl4J	41A	1	1, 2	<i>l(2)41Ab⁻ -rl⁻</i>
Df(2R)rl5B	41A	1	1, 2	<i>l(2)41Ab⁻ -rl⁻</i>
Df(2R)rl6C	41A+	1	1, 2	<i>l(2)41Ab⁻ - l(2)41Ae⁻</i>
Df(2R)rl9B	41A+	1	1, 2	<i>l(2)41Ab⁻ - l(2)41Ae⁻</i>
Df(2R)rl10a	41A	?	3	<i>rl⁻ stw⁺</i>
Df(2R)rl10b^γ	41A	?		<i>rl⁻ -stw⁻</i>
Df(2R)rl10G	41A+	1	1, 2	<i>l(2)41Ab⁻ - l(2)41Ae⁻</i>
Df(2R)rl12E	41A	1	1, 2	<i>l(2)41Ab⁻ -rl⁻</i>
Df(2R)rl13D	41A	1	1, 2	<i>l(2)41Ab⁻ -rl⁻</i>
Df(2R)rl14C	41A+	1	1, 2	<i>l(2)41Ab⁻ - l(2)41Ae⁻</i>
Df(2R)rl18C	41A+	1	1, 2	<i>l(2)41Ab⁻ - l(2)41Ae⁻</i>
Df(2R)rl-B	41A+	1	1, 2	<i>l(2)41Ab⁻ - l(2)41Ae⁻</i>
Df(2R)rl-D	41A+	1	1, 2	<i>l(2)41Ab⁻ - l(2)41Ae⁻</i>
Df(2R)rl-F	41A	1	1, 2	<i>l(2)41Ab⁻ -rl⁻</i>
Df(2R)rl-I	41A+	1	1, 2	<i>l(2)41Ab⁻ - l(2)41Ae⁻</i>

^α 1 = Recovered as a detachment product of *C(2L)RM, SH3/C(2R)RM, VK1*, *bw* (Hilliker and Holm, 1975, Genetics 81: 705-21); 2 = Induced in a *cn bw* chromosome (Sharp *et al.*, 1985).

^β 1 = Hilliker, 1976, Genetics 83: 765-82; 2 = Sharp, Hilliker, and Holm, 1985, Genetics 110: 671-88; 3 = Tano, 1966, Jpn. J. Genet. 41: 299-308.

^γ Stock listed (1982, DIS 57) but no references.

Df(2R)Rsp: Deficiency (3R) Responder

cytology: Deficient for 2R heterochromatin.

origin: X-ray-induced losses of sensitivity to distortion by *SD*.

synonym: *Df(2R)Rsp^{ins}*.

references: Ganetzky, 1977, Genetics 86: 321-55.

genetics: Insensitive to distortion by *SD*; lethal in combination with *Df(2R)M-S2-10*; deficient for *rl* and lethals as tabulated below:

deficiency	deficient for
Df(2R)Rsp1	<i>Rsp⁻ -rl⁻</i>
Df(2R)Rsp11	<i>Rsp⁻ -rl⁻</i>
Df(2R)Rsp31	<i>Rsp⁻ -uex⁻</i>

Df(2R)SB1

cytology: *Df(2R)60F1;60F5*.

origin: X ray induced.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

Preiss, Rosenberg, Kienlin, Seifert, and Jäckle, 1985, Nature 313: 27-32.

Côté, Preiss, Haller, Schuh, Kienlin, Seifert, and Jäckle, 1987, EMBO J. 6: 2793-2801.

Baumgartner, Bopp, Burri, and Noll, 1987, Genes and Development 1: 1247-67.

genetics: Deficient for *gsb* and *Kr* but not *zip*. *Df(2R)gsb/Df(2R)SB1* embryos express *gsb* but not *zip* (Côté *et al.*, 1987).

molecular biology: Deletion for entire cloned *Kr* region; proximal breakpoint at 0 to +3.5 kb on the molecular map (Côté *et al.*, 1987).

Df(2L)sf-49

cytology: *Df(2L)51B3-4;51C7-E2*.

Df(2R)sple: Deficiency (2R) spiny legs

cytology: *Df(2R)42E3;43C3*.

origin: X ray induced.

references: Gubb and García-Bellido, 1982, J. Embryol. Exp. Morphol. 68: 37-57.

genetics: Deficient for *pk*, *pwn*, and *sple*.

Df(2R)ST1

cytology: *Df(2R)43B3-4;43E18*.

origin: γ ray induced.

discoverer: Tsubota.

references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

genetics: Deficient for *pk*, *so*, *cos2* and *cn*, the heterozygotes showing small ocelli (Ashburner).

Df(2R)Sw: Deficiency (2R) Swedish

cytology: *Df(2R)60F3-4;60F5*.

origin: Natural condition of Swedish-b.

discoverer: Catcheside, 36120.

genetics: No phenotypic effect.

Df(2R)TE19: see *Df(2R)TE51D*

Df(2R)TE51D

origin: X ray induced in *TE51D* of Ising.

discoverer: R. MacIntyre.

synonym: *Df(2R)TE19*.

genetics: $w^- rst^-$.

deficiency	cytology
Df(2R)TE51D-11	51E3-4;52A6-10
Df(2R)TE51D-17	51D9-E1;51E5
Df(2R)TE51D-18	51E3;52C9-D1
Df(2R)TE51D-58	51D9-E1;52A13-B1

Df(2R)trix: Deficiency (3R) transformer intersex

cytology: *Df(2R)51A2-4;51B6*.

discoverer: B.S. Baker.

references: Breen and Duncan, 1986, Dev. Biol. 118: 442-56.

Belote and Baker, 1987, Proc. Nat. Acad. Sci. USA 84: 8026-30.

genetics: Deficient for *Asx* and *tra2*. Homozygous *trix* larvae show abdominal setal-belt phenotype in thorax.

Df(2R)twi: Deficiency (2R) twist

cytology: *Df(2R)59C3-4;59D1-2* (Richards).

origin: X ray induced.

synonym: *Df(1)twi-S60*.

references: Simpson, 1983, Genetics 105: 615-32.

genetics: Deficient for *twi*.

Df(2R)V13: Deficiency (2R) Valencia

cytology: *Df(2R)49D-E;50B + T(2;3)60D;96F*.

origin: X ray induced with *T(2;3)V13*.

references: Valencia, 1970, DIS 45: 37-38.

Df(2R)V30

cytology: *Df(2R)41B;41C*.

origin: X ray induced.

references: Valencia, 1970, DIS 45: 37-38.

Df(2R)vg: Deficiency (2R) vestigial

deficiency	cytology	origin	ref ^α	genetics ^β
Df(2R)vg33	49D;50A	γ ray	2	<i>l(2)49Da⁻</i> <i>-l(2)49Fm⁻</i> ; <i>vg/Df = vg</i>
Df(2R)vg52	49C-D;50B-C	γ ray	Lasko	<i>vg/Df = vg</i>
Df(2R)vg56	49D;49F	γ ray	2, 5	<i>vg⁻-l(2)49Fa⁻</i> ; <i>vg/Df = vg</i>
Df(2R)vg57		γ ray	Lasko	<i>vg/Df = vg</i>
Df(2R)vg62	49B8-D1;49E1-F2	γ ray	2	<i>l(2)49Da⁻</i> <i>-l(2)49Ea⁻</i>
Df(2R)vg79a	49D1;49E2 + <i>In(2LR)34B2-C1</i> ; 49B12-C3	neutrons	1, 2	
Df(2R)vg79b3	49C4;49E2 + <i>T(2;3)49C2-3;94A2-3</i>	γ ray	1, 2	
Df(2R)vg79d2	49C2-D2;49D7-E1	neutrons	1	<i>vg⁻</i>
Df(2R)vg79d8	49C4-D1;49D7-E1	neutrons	1, 2	<i>l(2)49Da⁻</i> <i>-l(2)49Ea⁻</i>
Df(2R)vg80I1	49D2-3;49E7-F1	neutrons	1	<i>vg⁻</i>
Df(2R)vg81		γ ray	2	<i>l(2)49Da⁻</i> <i>-l(2)49Ea⁻</i>
Df(2R)vg83b	49B2-3;49E7-F1	γ ray	1	<i>vg⁻</i>
Df(2R)vg83c31	49C3-D2; 49F15-50A1	γ ray	1	<i>vg⁻</i>
Df(2R)vg83f15	49C3-D1;49E7-F1	γ ray	1	<i>l(2)49Da⁻</i> <i>-l(2)49Ea⁻</i>
Df(2R)vg83f36	49C3-D1;49E7-F1	γ ray	1	<i>vg⁻</i>
Df(2R)vg83f58	49B12-C1;49D7-E1	γ ray	1	<i>vg⁻</i>
Df(2R)vg83I2a	49D2-3;49D7-E1	neutrons	1	<i>vg⁻</i>
Df(2R)vg83I-N	49C3-D1;50B9-C1	neutrons	1	<i>vg⁻</i>
Df(2R)vg84f65	49B11-C1;49D7-F1	X ray	1	<i>vg⁻</i>
Df(2R)vg84h49	49B2-5;49F10-11	X ray	1	<i>vg⁻</i>
Df(2R)vg85f1	49B11-C1;49E7-F1	γ ray	1	
Df(2R)vg85f2	49B11-C1;49E7-F1	γ ray	1	

deficiency	cytology	origin	ref ^α	genetics ^β
Df(2R)vg104	49C4;49F13	γ ray	2	<i>l(2)49Da⁻</i> <i>-l(2)Fk⁻</i>
Df(2R)vg106		γ ray	2	<i>l(2)49Da⁻</i> <i>-l(2)49Fj⁻</i>
Df(2R)vg107		γ ray	2	<i>l(2)49Da⁻</i> <i>-l(2)49Ea⁻</i>
Df(2R)vg120^Y		γ ray	2	<i>l(2)49Da⁻</i> <i>-l(2)49Ea⁻</i>
Df(2R)vg124^Y		γ ray	2	<i>l(2)49Da⁻</i> <i>-l(2)49Ea⁻</i>
Df(2R)vg133		γ ray	2	<i>l(2)49Da⁻</i> <i>-l(2)49Dc⁻</i>
Df(2R)vg135	48C-D;49D	γ ray	2	<i>l(2)49Da⁻</i> <i>-l(2)49Dc⁻</i>
Df(2R)vg136	breakpoints not cytologically detectable	γ ray	2, 5	<i>vg⁻</i> <i>-l(2)49Ea⁻</i>
Df(2R)vg-B^δ	49D3-4; 49F15-50A3	spont	2, 3, 5	<i>l(2)49Da⁻</i> <i>-l(2)49Fo⁻</i>
Df(2R)vg-C	49B2-3;49E7-F1	X ray	2, 3	<i>sca⁻-l(2)49Dc⁻</i>
Df(2R)vg-D^{δ ε}	49C1-2;49E4-5	spont	2, 3	<i>sca⁻-l(2)49Ea⁻</i>
*Df(2R)vg-I	49C2-D1;50A2-3		3	<i>sca⁻-l(2)49Dc⁻</i>
Df(2R)vg-P2		HD	2	<i>l(2)49Da⁻</i> <i>-l(2)49Fm⁻</i> <i>l(2)49Da⁻</i> <i>-l(2)49Ea⁻</i>
Df(2R)vgR7				<i>l(2)49Da⁻</i> <i>-l(2)49Ea⁻</i>
Df(2R)vg-S	49B12-C1; 49F15-50A1	X ray	3, 4	<i>sca⁻-l(2)49Dc⁻</i>

^α 1 = Alexandrov and Alexandrova, 1987, DIS 66: 185-87; 2 = Lasko and Pardue, 1988, Genetics 120: 495-502; 3 = Morgan, Bridges, and Schultz, 1938, Year Book-Carnegie Inst. Washington 37: 304-06; 4 = Muller, 1930, J. Genet. 22: 299-334. 5 = Williams and Bell, 1988, EMBO J. 7: 1355-63.

^β Homozygous lethal.

^γ Heterozygous females have reduced fertility.

^δ Enhances *BicD* in heterozygotes (Mohler and Wieschaus, 1986, Genetics 112: 803-22).

^ε *Df(2R)vg^D/+* flies show dominant flightlessness (Cripps, and Sparrow, 1989, Dev. Genet. 10: 98-105) and loss of bristles on legs and anterior dorsal thorax (Morgan, *et al.*, 1938).

Df(2R)VV4L

cytology: *Df(2R)43B1;43B2*.

origin: X ray induced with *Df(2R)VV4R*.

discoverer: Velissariou.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.

Df(2R)VV4R

cytology: *Df(2R)44F1;45A1-2*.

origin: X ray induced with *Df(2R)VV4L*.

discoverer: Velissariou.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.

Df(2R)WVG

cytology: *Df(2)52A4-B1;52D7-E1*.

discoverer: Gelbart.

references: Chase and Kankel, 1987, J. Neurobiol. 18: 15-41.

Davis and MacIntyre, 1988, Genetics 120: 755-66.

Df(2R)XTE-11

cytology: *Df(2R)51E3-4;52A6-10*.

references: Chase and Kankel, 1987, J. Neurobiol. 18: 15-41.

Davis and MacIntyre, 1988, Genetics 120: 755-66.

genetics: Deficient for *TE51D*.

Df(2R)XTE-18

cytology: *Df(2R)51E3-4;52C9-D1*.
 references: Chase and Kankel, 1987, J. Neurobiol. 18: 15-41.
 Davis and MacIntyre, 1988, Genetics 120: 755-66.
 genetics: Deficient for *TE51D*.

Df(2R)Y: Deficiency (2R) Ytterborn

cytology: *Df(2R)59B;59F*.
 origin: X ray induced.
 references: Ytterborn, 1968, Hereditas 59: 49-62.

Df(3L)1-Df(3L)10

cytology: *Df(3L)80F;80F*.
 origin: Detachment of *C(3L)RMIC(3R)RM*.
 references: Marchant and Holm, 1988, Genetics 120: 519-32.

deficiency	genetics
Df(3L)1-16	<i>l(3)80Fa⁻ - l(3)80Fg⁻</i>
Df(3L)1-166	<i>l(3)80Fg⁻ - l(3)80Fj⁻</i>
Df(3L)2-66	<i>l(3)80Fh⁻ - l(3)80Fj⁻</i>
Df(3L)3-9	<i>l(3)80Fj⁻</i>
Df(3L)5-84	<i>l(3)80Fb⁻ - l(3)80Fj⁻</i>
Df(3L)8A-80	<i>l(3)80Ff⁻ - l(3)80Fg⁻</i>
Df(3L)9-52	<i>l(3)80Fa⁻ - l(3)80Fj⁻</i>
Df(3L)9-56	<i>l(3)80Fi⁻ - l(3)80Fj⁻</i>
Df(3L)10-39	<i>l(3)80Fd⁻ - l(3)80Fj⁻</i>
Df(3L)10-58	<i>l(3)80Fc⁻ - l(3)80Fg⁻</i>

Df(3L)29A6

cytology: *Df(3L)66F3;67B1*.
 origin: Induced by DEB (isolated as lethal over *Df(3L)AC1*).
 synonym: *l(3)67Ee*.
 references: Leicht and Bonner, 1988, Genetics 119: 579-93.
 genetics: No 67B heat shock puff.

Df(3L)1227

cytology: *Df(3L)63C1-2;63F1-2*.
 references: Fristrom.

Df(3L)AC1: Deficiency (3L) Adelaide Carpenter

cytology: *Df(3L)67A2;67D11-13*.
 origin: Uncovered among progeny of males treated with ethyl methanesulfonate.
 discoverer: Carpenter.
 references: Leicht and Bonner, 1988, Genetics 119: 579-93.
 genetics: Deficient for *M(3)67C [=M(3)i]* (Leicht and Bonner, 1988) and *αTub67C* (Matthews).

Df(3L)ASC: see *Df(3L)Pc*

Df(3L)BK

origin: X ray induced in *Dp(3;3)S2*.
 references: Leicht and Bonner, 1988, DIS 67: 54-56.

deficiency	cytology	new order
Df(3L)BK9	<i>68E2-3;69A1-2</i>	<i>61A - 68E2-3 69A1-2 - 100F</i>
Df(3L)BK10	<i>71C1-2;71F4-5</i>	<i>61A - 71C1-2 71F4-5 - 100F</i>
Df(3L)BK15	<i>68B1-2;68C6-8</i>	<i>61A - 68B1-2 68C6-8 - 100F</i>
Df(3L)BK140^α	<i>67F1;68C7-8</i>	<i>61A - 67F1 68C7-8 - 100F</i>

^α Also see Regan and Fuller, 1988, Genes Dev. 2: 82-92.

Df(3L)Brd: Deficiency (3L) Bearded

origin: γ ray induced.
 discoverer: Posakony.
 genetics: Deficient for *Brd*; phenotypically wild-type over *Brd^T*.

deficiency	cytology	synonym
Df(3L)Brd6	<i>70E;71F</i>	<i>Df(3L)Brd^{R6}</i>
Df(3L)Brd12	<i>70E;71B</i>	<i>Df(3L)Brd^{R12A}</i>
Df(3L)Brd15	<i>70E;71E1</i>	<i>Df(3L)Brd^{R15}</i>
Df(3L)Brd18	<i>70D;71F</i>	<i>Df(3L)Brd^{VR18}</i>

Df(3L)brm11: Deficiency (3L) brahma

cytology: *Df(3L)71F;72D* (Kennison).
 origin: Induced by P-M hybrid dysgenesis.
 discoverer: Kennison and Tamkun, 1987.
 genetics: *brm^{-th}*. Lethal in combination with *Df(3L)st-g24 = Df(3L)72D1-2;73A9-10* but survives in combination with *Df(3L)st-e4 = Df(3L)72D5-10;73A5* and *Df(3L)st-b11 = Df(3L)72D7-9;73D1-2*.

Df(3L)Cat: Deficiency (3L) Catalase

cytology: *Df(3L)75C1-2;75F1*.
 origin: γ ray induced.
 synonym: *Df(3L)Cat^{DH104}*.
 references: Mackay, Hollar, and Bewley, 1986, Genetics 113: s31.
 Bewley, Mackay, and Cook, 1986, Genetics 113: 919-38.
 genetics: Deficiency heterozygotes show dosage effect for catalase activity.

***Df(3L)D**: see **T(Y;2;3)D**

Df(3L)DTS5-3

cytology: *Df(3L)72F3-4;73E2-F1* (Ashburner).
 origin: X-ray-induced reversion of dominant effect of *l(3)73Ai* selected at 29°.
 discoverer: Faithfull.
 synonym: *Df(3L)DTS-5^{R+3}*.
 genetics: Deficient for *l(3)73Ai*.

Df(3L)DTS5-4

cytology: *Df(3L)72D3-6;73B6-C4 + T(Y;3)CA16* (broken at 90B-C).
 origin: X-ray-induced revertant of dominant effect of *l(3)DTS-5*.
 discoverer: Faithfull.
 synonym: *Df(3L)DTS-5^{R+4}; Df(3L)st^A*.
 references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91 (listed here incorrectly as *Df(3L)DTS-5^{R+3}*).
 genetics: Deficient for *l(3)73Ai*, *st* and *tra* but not for *as* or *th*.

Df(3L)E

origin: X ray induced.
 genetics: Homozygous lethal.

deficiency	cytology	ref ^α	comments	mol biol ^β
Df(3L)E5	<i>?;73A9-10</i>	1	<i>tra⁺</i>	+80 kb (prox.)
Df(3L)E34^γ	<i>73A1-2;?</i>	1, 2	<i>tra⁻</i>	-36 kb (dist.)
Df(3L)E36^δ	<i>?;73B1-2</i>	1, 2	<i>tra⁻</i>	+120 kb (prox.)
Df(3L)E48	<i>?;73A11</i>	1	<i>tra⁻</i>	+90 kb (prox.)
Df(3L)E52	<i>73A1-2;73A9-10</i>	1	<i>tra⁻</i>	-38 kb (dist.); +85 kb (prox.)

deficiency	cytology	ref ^α	comments	mol biol ^β
^α	1 = Butler, Pirrotta, Irminger-Finger, and Nöthiger, 1986, EMBO J. 5: 3607-13; 2 = Henkemeyer, Gertler, Goodman, and Hoffmann, 1987, Cell 51: 821-28.			
^β	The zero coordinate used in locating the molecular lesions is the 73A3-4 breakpoint of <i>In(3LR)st^{a27}</i> , "+" values to the right, "-" values to the left (McKeown, Belote, and Baker, 1987, Cell 48: 489-99). The distal and proximal molecular breaks of the deficiencies made by Butler <i>et al.</i> , 1986, were estimated using this zero coordinate. (<i>Bam</i> HI site, midway between <i>Df(3L)E5</i> and <i>Df(3L)E52</i> , was the original zero coordinate of Butler <i>et al.</i>).			
^γ	Synonym: <i>Df(3L)st-E34</i> ; includes <i>st-l(3)73Bb</i> but not <i>l(3)73Bc</i> (Henkemeyer <i>et al.</i> , 1987).			
^δ	Synonym: <i>Df(3L)st-E36</i> ; includes <i>st-Abl</i> but not <i>l(3)73Bb</i> (Henkemeyer <i>et al.</i> , 1987).			

Df(3L)e146c4

cytology: *Df(3L)67A;67A*.
genetics: *l(3)67Ac-l(3)67Ad*.

Df(3L)emc: Deficiency (3L) extra macrochaetae

cytology: *Df(3L)61C5-6;61D3-4*.
origin: X ray induced.
discoverer: Ellis.
genetics: Deficient for *emc*; rare homozygous escapers.

Df(3L)emc-E12

cytology: *Df(3L)61A;61D3*.
references: Alonso, and García-Bellido 1988, Roux's Arch. Dev. Biol. 197: 328-38.
genetics: Deficient for *emc*.

Df(3L)Est6: Deficiency (3L) Esterase 6

discoverer: Richmond.

Df(3L)Ez: Deficiency 3L Enhancer of zeste

references: Jones and Gelbart, 1990, in press.
genetics: Deficient for *E(z)*.

deficiency	cytology	origin	synonym
<i>Df(3L)Ez1</i>	67E3-4;67F1-3	γ ray of <i>E(z)</i> ¹	<i>E(z)</i> ^{1R2}
<i>Df(3L)Ez2</i>	67E1-2;67E5-7 + <i>In(3L)64B;67E</i>	γ ray of <i>E(z)</i> ¹	<i>E(z)</i> ^{1R3}
<i>Df(3L)Ez3</i>	67E3-4;67E6-7	γ ray of <i>E(z)</i> ¹	<i>E(z)</i> ^{1R9}
<i>Df(3L)Ez4</i>	67E1-4;68A1-2	γ ray of <i>E(z)</i> ¹	<i>E(z)</i> ^{1R12}
<i>Df(3L)Ez5</i>	67D9-13;68F	γ ray of <i>E(z)</i> ¹	<i>E(z)</i> ^{1R13}
<i>Df(3L)Ez6</i>	67E1-2;67E3-5	γ ray of <i>E(z)</i> ⁶⁰	<i>E(z)</i> ^{51R1}
<i>Df(3L)Ez7</i>	67E1-4;67F1-3	EMS	<i>E(z)</i> ⁵⁶

Df(3L)Fz-CAL5: Deficiency (3L) frizzled

cytology: *Df(3L)70C2-7;70E1-3*.
origin: Hybrid dysgenesis induced.
references: Adler, Charlton, and Vinson, 1987, Dev. Genet. 8: 99-119.
genetics: Strong *fz* phenotype over *fz*; lethal over *fz*^{D21}, *fz*^{M21}.

Df(3L)Fz-D21

cytology: *Df(3L)70D3;70E3-8*.
origin: γ ray induced.
discoverer: Adler.
references: Freeman, Nüsslein-Volhard, and Glover, 1986, Cell 46: 457-58.
Adler, Charlton, and Vinson, 1987, Dev. Genet. 8: 99-119.
genetics: Deficient for *fz* and *gnu* (Freeman *et al.*, 1986).

Df(3L)Fz-GF3b

cytology: *Df(3L)70B;70C6*.

Df(3L)Fz-GS1a

cytology: *Df(3L)70C6-15;70E4-6*.

Df(3L)Fz-M21

cytology: *Df(3L)70D2-3;71E4-5*.
origin: γ ray induced.
discoverer: Adler.
references: Freeman, Nüsslein-Volhard, and Glover, 1986, Cell 46: 457-58.
Adler, Charlton, and Vinson, 1987, Dev. Genet. 8: 99-119.
genetics: Deficient for *fz*.

****Df(3L)Gl1: Deficiency (3L) Glued***

cytology: *Df(3L)70C;71B*.
origin: X ray induced.
synonym: *Df(2L)Gl^{rv1}*; *Df(3L)Gl^{+R1}*.
references: Harte and Kankel, 1982, Genetics 101: 477-501.
genetics: Deficient for *Gl* and *fz*. Lethal over *In(3L)D*, *D*³.

Df(3L)Gl2

cytology: *Df(3L)70C;70F*.
origin: X ray induced.
synonym: *Df(3L)Gl^{rv21}*; *Df(3L)Gl^{+R2}*.
references: Harte and Kankel, 1982, Genetics 101: 477-501.
Garen, Miller, and Paco-Larson, 1984, Genetics 107: 645-55.
genetics: Deficient for the entire *Gl* region and *fz*. Lethal over *In(3L)D*, *D*³.

Df(3L)h

origin: Induced by hybrid dysgenesis.
references: Campbell, Hilliker, and Phillips, 1986, Genetics 112: 205-15.

deficiency	cytology	genetics
<i>Df(3L)h1</i> ^α	68A3-4;68B4-C1	<i>l(3)68Ac-l(3)68Be</i> ⁻
<i>Df(3L)h2</i> ^α	[68A;68B]	<i>l(3)68Ae-l(3)68Bd</i> ⁻
<i>Df(3L)h5</i> ^α	68A3-4;68B4-C1	<i>l(3)68Ad-l(3)68Be</i> ⁻
<i>Df(3L)h8</i> ^α	68A3-4;68B4-C1	<i>l(3)68Ad-l(3)68Be</i> ⁻
<i>Df(3L)h9</i>	invisible ^β	<i>l(3)68Ad</i> ⁻
<i>Df(3L)h76</i> ^α	68A deficiency	<i>l(3)68Ae-l(3)68Ag</i> ⁻

^α Deficient for *Sod*.
^β No major rearrangements of 68A-C, but a small aberration, possibly a deficiency, in 68A proximal to 68A2.

****Df(3L)h100.390: Deficiency (3L) hairy***

cytology: *Df(3L)66D2-5;66D14-E1*.
origin: X ray induced.
discoverer: Alexander.
references: Ward and Alexander, 1957, Genetics 42: 42-54.
genetics: Deficient for *h*.

Df(3L)h-i22

cytology: *Df(3L)66D10-11;66E1-2*.
origin: X ray induced.
discoverer: Belote.
references: Ingham, Pinchin, Howard, and Ish-Horowicz, 1985, Genetics 111: 463-86.
genetics: Deficient for *h* and the chorion protein genes on 3L (*Cp-15*, *Cp-16*, *Cp-17*, and *Cp-18*), and *abd* but not *se*. Homozygous lethal.

Df(3L)Hn: Deficiency (3L) Henna

cytology: *Df(3L)66A;66B*.
origin: X ray induced simultaneously with *T(2;3)Hn = T(2;3)53E-54A;77A;94F;96A*.
discoverer: Van Atta, 30k.
references: 1932, Am. Nat. 66: 93-95.
 1932, Genetics 17: 637-59.
 Lewis, 1956, DIS 30: 130.
genetics: Mutant or deficient for *Hn*. Homozygous lethal.

Df(3L)in61: Deficiency (3L) inturned

cytology: *Df(3L)76F;77D*.
references: Arajärvi and Hannah-Alava, 1969, DIS 44: 73-74.
genetics: Deficient for *in*.

Df(3L)JK18: see *Df(3L)kto2*

***Df(3L)K: Deficiency (3L) of Krivshenko**

cytology: *Df(3L)61A;61C1-2*; an apparent terminal deficiency.
origin: Probably X ray induced.
discoverer: Krivshenko, 5614.
synonym: *Df-3L^K*.
references: 1959, DIS 33: 95.

Df(3L)kto2: Deficiency (3L) kohtalo

cytology: *Df(3L)76B1-2;76D5* (Ashburner).
origin: Induced by P-M hybrid dysgenesis.
discoverer: Kennison, 1987.
synonym: *Df(3L)JK18*.
genetics: Deficient for *kto* and *ash1*.

Df(3L)l3

cytology: *Df(3L)68A8-B1;68B4-C1*. Status of 68A7 not clear.
origin: Induced by ethyl methanesulfonate.
references: Campbell, Hilliker, and Phillips, 1986, Genetics 112: 205-15.
genetics: Includes *l(3)68Ba-l(3)68Be*. Not deficient for *Sod*.

Df(3L)lxd: Deficiency (3L) low xanthine dehydrogenase

references: Schott and Finnerty, 1983, Genetics 104: s62-63.
 Schott, Baldwin, and Finnerty, 1986, Biochem. Genet. 24: 509-27.

deficiency	cytology	genetics
<i>Df(3L)lxd2</i>	68A2-3;68C5-7	<i>lxd⁻</i>
<i>Df(3L)lxd6^α</i>	67E1-2;68C1-2	<i>hay⁻ - lxd⁻</i>
<i>Df(3L)lxd8</i>	68A2-3;68A5-6	<i>lxd⁻</i>
<i>Df(3L)lxd9^β</i>	68A3-4;68B4-C1	<i>lxd⁻ - sod⁻</i>
<i>Df(3L)lxd15</i>	67E;68C10-15	<i>hay⁻ - lxd⁻</i>

^α See Regan and Fuller, 1988, Genes Dev. 2: 82-92.
^β See Campbell, Hilliker, and Phillips, 1986, Genetics 112: 205-15.

Df(3L)Ly: Deficiency (3L) Lyra

cytology: *Df(3L)70A2-3;70A5-6*.
origin: X ray induced.
discoverer: Dubinin, 1929.
references: Morgan, Bridges, and Schultz, 1937, Year Book - Carnegie Inst. Washington 36: 301.
genetics: Associated with the mutant, *Ly*. Lethal in combination with *Df(3L)M69E*.

Df(3L)Ly2

cytology: *Df(3L)70A2-3;70A5-6*. Same as *Df(3L)Ly* (Zhimulev and Feldman, 1982).
origin: Spontaneous (possibly caused by male recombination inducer).
references: Waddle, 1977, DIS 52: 3.
 Zhimulev and Feldman, 1982, DIS 58: 152.
genetics: Deficient for *Ly*. Phenotype of *Ly*² similar to *Ly* except that eyes tend to be rougher.

***Df(3L)M69E**

cytology: Probably includes bands in 70A.
origin: X ray induced.
discoverer: Demerec, 33j25.
synonym: **Df(3L)M-h33j*.
references: 1935, DIS 3: 27.
 Coyne, 1935, DIS 4: 59.
 Mossige, 1938, Hereditas 24: 110-16.
genetics: Deficient for *M(3)69E*; lethal homozygous and in combination with *Df(3L)Ly*.

**Df(3L)M-h33j*: see *Df(3L)M69E*

Df(3L)Mg26: Deficiency (3L) Mglinetz

cytology: *Df(3L)66C;66D*.
origin: γ ray induced.
references: Mglinetz, 1972, Genetika (Moscow) 8: 82-92.

Df(3L)Mg27

cytology: *Df(3L)61C;66D*.
origin: γ ray induced.
references: Mglinetz, 1972, Genetika (Moscow) 8: 82-92.

***Df(3L)Mz: Deficiency (3L) from Minute (2) z stock**

cytology: Loss of several bands from tip of *3L(61A)*.
origin: Spontaneous.
discoverer: Bridges, 36h28.
references: CP627.

Df(3L)N²⁶⁴⁻⁶: see *T(1;3)N²⁶⁴⁻⁶*

Df(3L)P20: Deficiency (3L) Pasadena 20

cytology: *Df(3L)67F;68E3-4*.
origin: Recombinant from *Tp(3;3)P20/ +*.
references: Lewis, 1980, DIS 55: 207-08.
genetics: *Df(3L)P20/ +* flies slightly Minute and seem to have reduced fertility in females.

Df(3L)Pc: Deficiency (3L) Polycomb

deficiency	cytology	discoverer	ref ^α	genetics
<i>Df(3L)Pc^β</i>	78D1-2;79A4-C1	Jürgens	1	<i>Pc⁻</i>
<i>Df(3L)Pc-MK</i>	78A3;79E1-2	Denell	3-5	<i>Pc⁻</i>
<i>*Df(3L)Pc-T6^γ</i>	77E1;79A1	Tiong		<i>ri⁻Pc⁻M</i>
<i>Df(3L)Pc-T7^γ</i>	78E2-3;79E5	Tiong	2	<i>ri⁺Pc⁻</i>
<i>*Df(3L)Pc-W2</i>		Williams		<i>Pc⁻</i>

^α 1 = Haynie, 1983, Dev. Biol. 100: 399-411; 2 = Kennison and Russell, 1987, Genetics 116: 75-86; 3 = Sato and Denell, 1985, Dev. Biol. 110: 53-64; 4 = Sato, Hayes, and Denell, 1984, Dev. Genet. 4: 185-98; 5 = Sato, Russell, and Denell, 1983, Genetics 105: 357-70.
^β Synonym: *Df(3L)ASC*.
^γ Origin: γ ray induced.

Df(3L)Pst8: see *Df(3L)st8P*

Df(3L)Pst-B7: see *Df(3L)st7P*

Df(3L)R: Deficiency (3L) Rough

discoverer: J. Bonner.

references: Johnson and Friedman, 1983, Proc. Nat. Acad. Sci. USA 80: 2990-94.

Sliter, Henrich, Tucker, and Gilbert, 1989, Genetics 123: 327-36.

deficiency	origin	cytology	genetics
Df(3L)R		62B7;62B12	<i>Aprt⁻ R⁻</i>
Df(3L)R-E	X ray	62B8-9;62B12-C1	<i>R⁻ - l(3)62Bi⁻</i>
Df(3L)R-G2	γ ray	62B2-4;62B11-12	<i>l(3)62Ba⁻ - l(2)62Bg⁻</i>
Df(3L)R-G5	γ ray	62A10-B1;62C4-D1	<i>l(3)62Ba⁻ - l(2)62Ca⁻</i>
Df(3L)R-G7	γ ray	62B8-9;62F2-5	<i>R⁻ - ecd⁻</i>
Df(3L)R-R2	X ray	62B2-4;62D3-5	<i>l(3)62Ba⁻ - ecd⁻</i>

Df(3L)ri79c: Deficiency (3L) radius incompletus

cytology: *Df(3L)77B-C;77F-78A*.

discoverer: Jürgens.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

genetics: Deficient for *kni* and *ri*.

Df(3L)ri-K2

cytology: *Df(3L)76D;77E-F*.

discoverer: Gubb.

references: García-Bellido, Moscoso del Prado, and Botas, 1983, Mol. Gen. Genet. 192: 253-63.

genetics: Deficient for *ri*.

***Df(3L)ru100.392: Deficiency (3L) roughoid**

cytology: *Df(3L)61E;62A10-B1*.

origin: X ray induced.

discoverer: Alexander.

references: Ward and Alexander, 1957, Genetics 42: 42-

54.

genetics: Deficient for *ru*.

***Df(3L)ru100.393**

cytology: *Df(3L)61E;62A4-6*.

origin: X ray induced.

discoverer: Alexander.

references: Ward and Alexander, 1957, Genetics 42: 42-54.

genetics: Deficient for *ru*.

***Df(3L)ru300.234**

cytology: *Df(3L)61E;62A2-4*.

origin: X ray induced.

discoverer: Alexander.

references: Ward and Alexander, 1957, Genetics 42: 42-54.

genetics: Deficient for *ru*.

***Df(3L)ru-K1: Deficiency (3L) roughoid of Krivshenko**

cytology: *Df(3L)62A12-B1;62B2-3*.

origin: X ray induced.

discoverer: Krivshenko, 1957.

references: 1958, DIS 32: 81.

genetics: Has rough and slightly reduced eyes in combination with *ru* but, judging from cytology, probably not deficient for *ru*.

***Df(3L)ru-K2**

cytology: *Df(3L)61F4-5;62A10-B1*.

origin: X ray induced.

discoverer: Krivshenko, 1957.

references: 1958, DIS 32: 81.

genetics: Deficient for *ru*.

Df(3L)st: Deficiency (3L) scarlet

deficiency	cytology	origin	discoverer	synonym	ref ^α	genetics	molecular biology ^β
Df(3L)st	72D7;74A3				3, 10	<i>st⁻ tra⁻</i>	
Df(3L)st2^γ	73A2-3;73A5	γ ray	Faithfull		1, 9	<i>st⁻</i>	
Df(3L)st3	72E5-F1;73A3-4	γ ray	Faithfull		1, 9	<i>th st⁻</i>	
Df(3L)st4	72D7-11;73B7-C11	γ ray	Faithfull		1, 6, 9	<i>st⁻ tra⁻</i>	
Df(3L)st6	72E1-2;73A3-4	γ ray	Faithfull		9	<i>st⁻ tra⁺</i>	
Df(3L)st7	73A3-4;74A3	γ ray	Faithfull		9	<i>st⁻ tra⁻</i>	
Df(3L)st7P	73A1-2;73A7-9 + <i>In(3L)P</i>	X ray	Bonner	<i>PstB7</i>	8	<i>l(3)73Aa⁻-l(3)73Ag⁻ tra⁺</i>	+80 to +85
Df(3L)st8P	72E4;73B4 + <i>In(3L)P</i>	X ray	Bonner	<i>Pst8</i>		<i>l(3)73Aa⁻-Abi⁻ db⁺</i>	
Df(3L)st62	73A2-3;73B3-5	X ray		100.62	6,8,12	<i>l(3)73Aa⁻-Abi⁻ db⁺</i>	
Df(3L)st100.62	72F3-7;73B3				13	<i>st⁻</i>	
Df(3L)st103	73E3;74A6	X ray	S. Smith	<i>SS103</i>	2, 3	<i>bul⁻-tra⁻</i>	
Df(3L)st106	72E3;73A3-4	X ray	S. Smith	<i>SS106</i>	2, 11, 12	<i>bul⁻-tra⁻</i>	
*Df(3L)st171	72E4-5;74C2-3	X ray		100.171	13	<i>st⁻</i>	
*Df(3L)st200	72E4-5;73A10-B1	X ray		100.200	13	<i>st⁻</i>	
Df(3L)st-a20	73A3;73A4	X ray	Belote		4, 8	<i>l(3)73Aa⁺ l(3)73Ab⁻-st⁻</i> <i>l(3)73Ac⁺</i>	-45 to -40; +45 to +55
Df(3L)st-b8	72D5-11;74B2					<i>st⁻</i>	
Df(3L)st-b11	72D10-11;73D1-2	X ray	Belote		4	<i>l(3)73Aa⁻-db⁻</i>	
Df(3L)st-e4	72D5-10;73A5	X ray	Belote		4, 8	<i>l(3)73Aa⁻-st⁻ l(3)73Ac⁺</i>	+60
*Df(3L)st-e5	72E2;73D4 + <i>In(3L)72E5-F1;73A1-2</i>	X ray	Belote		4	<i>l(3)73Aa⁻-Abi⁻</i>	
Df(3L)st-E5	?;73A9-10					<i>b st⁻ tra⁺</i>	+2 to 0
Df(3L)st-e10	{73A;73A}	dysgenesis	Green, 81e10	<i>81e</i>		<i>st⁻ lethal</i>	
Df(3L)st-E34	73A1-2; ?					<i>b st⁻ tra⁺</i>	+2 to 0
Df(3L)st-E36	?; 73B1-2					<i>b st⁻ tra⁻</i>	~ +115
Df(3L)st-E48	?; 73A11					<i>b st⁻ tra⁻</i>	~ -33
Df(3L)st-E52	73A1-2; 73A 9-10					<i>b st⁻ tra⁻</i>	-6 to -13
						<i>b st⁻ tra⁻</i>	~ +117; -2 to -6
Df(3L)st-f13	72C1-D1;73A3-4	X ray	Belote		4, 8	<i>l(3)73Aa⁻-st⁻ l(3)73Ac⁺</i>	+20 to +25
Df(3L)st-g18	72E2-7;74F4-75A2 + <i>In(3LR)65A1-2;99A1-2</i>	X ray	Belote		4, 5	<i>l(3)73Aa⁻-db⁻⁰</i>	
Df(3L)st-g24	72D1-2;73A9-10	X ray	Belote		4, 6, 8	<i>l(3)73Aa⁻-tra⁻ l(3)73Aj⁺</i>	+105
*Df(3L)st-g82	72D10-11;73B1-2 (see <i>Tp(3;1)st-g82</i>)	X ray	Belote		4	<i>l(3)73Aa⁻-tra⁻ l(3)73Aj⁺</i>	
Df(3L)st-j7	73A1-2;73B1-2	X ray	Belote		4, 6, 8	<i>l(3)73Aa⁻-l(3)73Aj⁻</i>	+140
Df(3L)st-j7;RB87	73A1-2;73B1-2;87D	dysgenesis			8		
Df(3L)st-j11	72E2;73E1-2	X ray	Belote		4	<i>st⁻</i>	
Df(3L)st-k2	73A2-3;73D2-3	dysgenesis	Green, 81k7	<i>81K7(2)</i>			
Df(3L)st-k3	{73A;73A}	dysgenesis	Green, 81k19	<i>81K19(3)</i>		<i>st⁻ lethal</i>	
Df(3L)st-k7	72A2-73A4;74E1-2	dysgenesis	Green, 81k19	<i>81K19(7)</i>		<i>st⁻</i>	
Df(3L)st-k10	73A3-4;73D1-2	dysgenesis	Green, 81k17	<i>81K17(12)</i>	5	<i>st⁻ lethal</i>	
Df(3L)st-LM3	{73A;73A}	X ray	Marsh and Mock		7	<i>bul⁻ st⁻-l(3)73Ai⁻</i>	
Df(3L)st-LM19	{73A;73A}	X ray	Marsh and Mock		7	<i>bul⁻ st⁻-l(3)73Ai⁻</i>	

^α 1 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91; 2 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95; 3 = Baker and Ridge, 1980, Genetics 94: 383-423; 4 = Belote and McKeown; 5 = Belote and McKeown, 1985, DIS 61: 33-34; 6 = Butler, Pirotta, Irminger-Finger, and Nöhiger, 1986, EMBO J. 5: 3607-13; 7 = Henkemeyer, Gertler, Goodman, and Hoffmann, 1987, Cell 51: 821-28; 8 = Marsh and Mock, 1985, DIS 61: 214; 9 = McKeown, Belote, and Baker, 1987, Cell 48: 489-99; 10 = Velissariou and Ashburner, 1981, Chromosoma 84: 173-85; 11 = Voelker, Ohnishi, and Langley, 1978, Biochem. Genet. 17: 947-56; 12 = Walker and Ashburner, 1981, Cell 26: 269-77; 13 = Ward and Alexander, 1957, Genetics 42: 42-54.

^β DNA coordinates (kb) of proximal breakpoints in the walk in region 73A-B of McKeown and Belote, which originates at the left breakpoint of *In(3LR)st^{a27}* ("+" values to right, "-" values to left). *Df(3L)st-E* coordinates from Butler *et al.*, 1986; coordinate 0 halfway between right breakpoints of *Df(3L)st-E5* and *Df(3L)st-E52*; positive values to the left.

^γ Semilethal in combination with *TM3*; lethal in combination with *TM1* and *TM6* (Ashburner).

^δ Heterozygote viable but weak.

Df(3L)std11: see In(3L)std11

Df(3L)th: Deficiency (3L) thread

deficiency	cytology	origin ^α	ref ^β	genetics	deficiency	cytology	origin ^α	ref ^β	genetics
Df(3L)th1^γ	71F3-5;72D6-8	1	1, 4	<i>th⁻</i>	Df(3L)th113	72A2;72D1-2	2	2	<i>th⁻</i>
Df(3L)th2^γ	70F1-3;72E3-5	1	1, 4	<i>th⁻</i>	Df(3L)th117	72A1;72D5	2	2, 5	<i>th⁻-Pgm⁻</i>
Df(3L)th45	72A5;72D6	2	2	<i>th⁻</i>					
Df(3L)th70i	72A2;72D6	2	3	<i>th⁻</i>					
Df(3L)th70ki	71C3-4;72C1-2	2	3	<i>th⁻</i>					
Df(3L)th70kil	71F3-4;73A3-4	2	3	<i>th⁻</i>					
Df(3L)th70l	72A2;72D	2	3	<i>th⁻</i>					
Df(3L)th102	72B1;72D12	2	2, 5	<i>th⁻-Pgm⁻</i>					
*Df(3L)th105	72A2-B1;73A4-5	2	6	<i>th⁻-st⁻</i>					
Df(3L)th112	71F3-5;72D12	2	2	<i>th⁻</i>					

^α 1 = γ ray induced; 2 = X ray induced.

^β 1 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91; 2 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95. 3 = Korge, 1972, DIS 48: 20; 4 = Velissariou and Ashburner, 1981, Chromosoma 84: 173-85; 5 = Voelker, Ohnishi, and Langley, 1978, Biochem. Genet. 17: 769-83; 6 = Ward and Alexander, 1957, Genetics 42: 42-54.

γ Dominant female-sterile.

Df(3L)tra: Deficiency (3L) transformer

cytology: *Df(3L)73A4;74A1-2*.
origin: X ray induced.
discoverer: B.S. Baker.
references: McKeown, Belote, and Baker, 1987, Cell 48: 489-99.
genetics: Deficient for *l(3)73Ac* through *db*; also includes *C-abl*; does not include *st*; broken between *st*⁺ and the next lethal complementation group to the right.
molecular biology: Distal breakpoint in the vicinity of +25 to +30 kb, "+" values to the right, "-" value to the left (McKeown *et al.*, 1987).

Df(3L)V13: see Tp(3;3)V13

Df(3L)V127

cytology: *Df(3L)71C1;71C2*.
origin: X ray induced.
references: Valencia, 1970, DIS 45: 37-38.

Df(3L)ve: Deficiency (3L) veinlet

cytology: *Df(3L)61E1;62A2-3*. 61E and 61F not puffed.
origin: X ray induced.
references: Korge, 1970, Chromosoma 30: 430-64.
genetics: Deficient for *ve*.

Df(3L)vin: Deficiency (3L) vin

deficiency	cytology	origin α	ref β	genetics
<i>Df(3L)vin1</i>	67E5-7;68E3-4	1	1, 2	<i>rs</i> ⁻ - <i>vin</i> ⁻
<i>Df(3L)vin2</i>	67F2-3;68D6	2	1	<i>rs</i> ⁻ - <i>vin</i> ⁻
<i>Df(3L)vin3</i>	68C5-6;68E3-4	1	1, 2, 3, 4	<i>rt</i> ⁻ - <i>Lsp2</i> ⁻
<i>Df(3L)vin4</i>	68B1-3;68F3-6	1	1, 2, 3, 6	<i>rt</i> ⁻ - <i>Lsp2</i> ⁻
<i>Df(3L)vin5</i>	68A2-3;69A1-3	1	1, 2, 4	<i>Sgs</i> ³⁻ - <i>Lsp2</i> ⁻
<i>Df(3L)vin6</i>	68C8-11;69A4-5	1	1, 2	<i>vin</i> ⁻ - <i>app</i> ⁻
<i>Df(3L)vin7</i>	68C8-11;69B4-5	1	1, 2	<i>rt</i> ⁻ - <i>app</i> ⁻
<i>Df(3L)vin8</i> ^{γ}	68C2-3;68F3-6		5	<i>vin</i> ⁻
<i>Df(3L)vin9</i> ^{γ}	68C5-6;69A5-B1		5	<i>vin</i> ⁻
<i>Df(3L)vin10</i> ^{δ}				<i>vin</i> ⁻
<i>Df(3L)vin11</i> ^{δ}				<i>vin</i> ⁻
<i>Df(3L)vin66</i>	68A2-3;68D3	1	1, 2	<i>Sgs</i> ³⁻ - <i>vin</i> ⁻

α 1 = γ ray induced; 2 = X ray induced.
 β 1 = Akam, Roberts, Richards, and Ashburner, 1978, Cell 13: 215-25; 2 = Crosby and Meyerowitz, 1986, Genetics 112: 785-802; 3 = Garcia-Bellido, Moscoso del Prado, and Botas, 1983, Mol. Gen. Genet. 192: 253-63; 4 = Gautam, 1983, Mol. Gen. Genet. 189: 495-500; 5 = D.B. Roberts; 6 = Schott, Baldwin, and Finnerty, 1986, Biochem. Genet. 24: 509-27.
 γ Discoverer: Hoogwerf.
 δ No cytology or genetics given (Ashburner).

***Df(3L)Vn: Deficiency (3L) Vein**

cytology: *Df(3L)64C12-D1;65D2-E1*.
origin: Spontaneous.
discoverer: Mohr, 28j21.
references: 1932, Proc. Intern. Congr. Genet., 6th., Vol 1: 199-212.
 1938, Norske Videnskaps-Akad. 4: 1-7.
 Mohr and Mossige, 1943, Norske Videnskaps-Akad. 7: 1-51 (fig.).
genetics: Deficient for *ju*, *dv*, and *Me* but not for *abd*, *Hn*, or *se*. Mutant or deficient for *Vn*.

***Df(3L)VV8**

cytology: *Df(3L)74B1-2;74F1-2*.
origin: γ ray induced.
discoverer: Velissariou.
references: Ashburner, Angel, Detwiler, Faithfull, Gubb,

Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

Df(3L)VW1

cytology: *Df(3L)69E2-F1;70C1*.
origin: γ ray induced.
discoverer: Walker.
references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.
genetics: Minute phenotype; presumably deficient for *M(3)69E*.

Df(3L)VW3

cytology: *Df(3L)76A3;76B2*.
origin: γ ray induced.
discoverer: Walker.
references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.
genetics: Minute phenotype; presumably deficient for *M(3)76A*.

Df(3L)W: Deficiency (3L) Wrinkled

origin: X ray induced.
synonym: *Df(3L)W^{rv}*; *Df(3L)W^{+R}*.
genetics: Deficient for *W*.

deficiency	cytology	discoverer	ref α
<i>Df(3L)W4</i> ^{β}	75B8-11;75C5-7	Segraves	2, 3
<i>*Df(3L)W7</i>	74D3-5;75C3-7	Faithfull	1
<i>Df(3L)W10</i> ^{γ}	75A6-7;75C1-2	Segraves	2, 3

α 1 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou and Woodruff, 1980, DIS 55: 193-95; 2 = Segraves and Hogness, 1984, Genetics 107: s96-97. 3 = Seagraves and Hogness, 1990, Genes Dev. 4: 204-19.
 β Proximal to 75B puff.
 γ Includes 75B puff and *E75* gene (Segraves and Hogness, 1984).

Df(3L)X37

genetics: Deficient for *Src1*.

Df(3R)+1

cytology: *Df(3R)92A11-B2;92C3-D1*.
genetics: Deficient for *l(3)SG65*.

Df(3R)1c77: see Df(3R)Tp12

Df(3R)3g74: see Df(3R)Tp11

Df(3R)4-7

cytology: *Df(3R)81F;81F*.
genetics: Deficient for *l(3)81Fa-l(3)81Fb*.

Df(3R)4SCB

cytology: *Df(3R)84A6-B1;84B2-3*.
discoverer: G. Jürgens.
references: Struhl, 1981, Nature 292: 635-38.
 Garber, Kuroiwa, and Gehring, 1983, EMBO J. 2: 2027-36.
 Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.
genetics: Deficient for *Scr*, *ftz*, and *Antp*. Homozygous lethal (Howard and Ingham, 1986, Cell 44: 949-57).
molecular biology: Deletes sequences to left of a point 100 kb to the left of the proximal breakpoint of *In(3R)Hu* (Garber *et al.*, 1984).

Df(3R)5B-RXP

cytology: *Df(3R)97A;98A1-2*.

discoverer: Anderson.

Df(3R)5f77: see *Df(3R)Tpl3*

Df(3R)9A99

cytology: *Df(3R)83F2-84A1;84B1-2*. Defines maximum leftward extent of *Antp*.

discoverer: G. Jürgens.

references: Garber, Kuroiwa, and Gehring, 1983, EMBO J. 2: 2027-36.

Kuroiwa, Hafen, and Gehring, 1984, Cell 37: 825-31.

Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

genetics: Deficient for *Scr* and *ftz*. Complements all *Antp* alleles tested.

molecular biology: Proximal breakpoint between coordinates +170 and +180 on DNA map of region 84B1-2 (Garber *et al.*, 1983).

Df(3R)10-65

cytology: *Df(3R)81F;81F*.

genetics: Deficient for *l(3)81Fa-l(3)81Fb*.

Df(3R)10d77-7: see *Df(3R)Tpl4*

Df(3R)25g77: see *Df(3R)Tpl5*

Df(3R)26b77: see *Df(3R)Tpl6*

Df(3R)28b77: see *Df(3R)Tpl7*

Df(3R)28f77: see *Df(3R)Tpl8*

Df(3R)29c76: see *Df(3R)Tpl9*

Df(3R)30c76: see *Df(3R)Tpl10*

***Df(3R)89EF**

cytology: *Df(3R)89D7-E1;90A2-3*.

origin: Synthetic. Made by combining the 4^P3R^D element of one *T(3;4)* with the 3R^P4^D element of another.

discoverer: Dubovsky and Kelstein.

references: 1936, Eksperim. Med. No. 11: 65-84.

Kelstein, 1938, Biol. Zh. (Moscow) 7: 1145-69.

Pipkin, 1959, Texas Univ. Publ. 5914: 69-88.

genetics: One of a series of deficiencies for the middle of 3R synthesized and carefully studied by Dubovsky and Kelstein. Deficient for abdominal components of *BXC*. Heterozygous male has rotated genitalia which may be feminized both in structure and color, has sex combs. Heterozygous female sterile.

Df(3R)109

references: Casanova, Sánchez-Herrero, and Morata, 1986, Cell 47: 627-36.

genetics: Deficient for *Ubx* and *iab2* but not *iab5-7*.

Df(3R)148.5: see *Df(3R)Cha-M1*

Df(3R)159.22

cytology: *Df(3R)87E3-4;87E6-8*.

origin: Induced by diepoxybutane.

references: Nagoshi and Gelbart, 1987, Genetics 117: 487-502.

genetics: Deficient for *l(3)87Ee-l(3)87Ef*.

molecular biology: Distal breakpoint 3-8 kb proximal to the distal breakpoint of *Df(3R)Ace-HD1* at 72.5 - 77.8 kb (DNA walk of Bender, Spierer and Hogness, 1983, J.

Mol. Biol. 168: 17-33).

Df(3R)229: see *Df(3R)E-229*

Df(3R)293

cytology: *Df(3R)87E;88A*.

references: Parkhurst, Harrison, Remington, Spana, Kelley, Coyne, and Corcas, 1988, Genes Dev. 2: 1205-15.

Df(3R)A41

cytology: *Df(3R)84B1-2;84D1-2*.

origin: X ray induced.

discoverer: Abbott, 1985.

references: Cavener, Otteson, and Kaufman, 1986, Genetics 114: 111-23.

Cavener, Corbett, Cox, and Whetten, 1986, EMBO J. 5: 2939-48.

Whetten, Organ, Krasney, Cox-Foster, and Cavener, 1988, Genetics 120: 475-84.

genetics: Deficient for *Antp* but not for *ftz*.

molecular biology: Left breakpoint at about +140 kb on the molecular map of Cavener *et al.*, (1986).

Df(3R)A113

cytology: *Df(3R)100A-B;100F*.

references: Van Vactor, Krantz, Reinke, and Zipursky, 1988, Cell 52: 281-90.

**Df(3R)Ace-HD1: Deficiency (3R)
Acetylcholinesterase**

cytology: *Df(3R)87E3-4;87E5-6*.

origin: Hybrid dysgenesis.

references: Nagoshi and Gelbart, 1987, Genetics 117: 487-502.

genetics: Deficient for *Ace-l(3)87Ee*; does not fully remove *Ace* activity, but behaves as recessive *Ace* lethal.

molecular biology: Proximal breakpoint between +49 and +50.5 kb; distal breakpoint between +72.5 and +77.8 kb (DNA walk of Bender, Spierer, and Hogness, 1983, J. Mol. Biol. 168: 17-33).

**Df(3R)Aldoxⁿ¹: Deficiency (1)
Aldehyde oxidase**

cytology: *Df(3R)89A1-2*.

origin: Spontaneous.

references: Langhout and van Breugel, 1985, DIS 61: 181.

genetics: Deficient for *Aldox*.

Df(3R)Antp^{73b+RX1}: see *Df(3R)Antp-X1*

Df(3R)Antp^{73b+RX2}: see *Df(3R)Antp-X2*

Df(3R)Antp^{73b+RX3}: see *Df(3R)Antp-X1*

Df(3R)Antp^{73b+RX4}: see *Df(3R)Antp-X2*

Df(3R)Antp^{+R1P}: see *Df(3R)Antp1P*

Df(3R)Antp^{Ns+R17}: see *Df(3R)Antp17*

Df(3R)Antp^{Ns+RC7}: see *Df(3R)Antp7*

Df(3R)Antp^{BL dsx^{Drv3R}}: see *In(3R)Antp^{BL dsx^{Drv3R}}*

Df(3R)Antp^{rA60}: see *Df(3R)Antp2*

Df(3R)Antp^{rA75}: see *Df(3R)Antp6*

Df(3R)Antp1P: Deficiency (3R) Antennapedia

cytology: *Df(3R)84B3;84D1-2 + In(3R)84B3;85C2-3*.
origin: Deficient recombinant between *In(3R)Antp^{rv1P}* (also a transposition) and a normal sequence.
discoverer: Green.
synonym: *Df(3R)Antp^{+R1P}*.
references: Lewis, Kaufman, and Denell, 1980, DIS 55: 85-87.
 Lewis, Kaufman, Denell, and Talerico, 1980, Genetics 95: 367-81.
genetics: Deficient for the distal portion of the 84B1-2 doublet and reverted for *Antp*; deficient for *roe* (Lewis *et al.*, 1980).

Df(3R)Antp2

cytology: *Df(3R)84B2;84D3*.
synonym: *Df(3R)Antp^{RA60}*.
origin: X ray induced.
references: Abbott and Kaufman, 1986, Genetics 114: 919-42.
genetics: Not *Antp* over wild-type. Complements *Antp²³* and *Antp¹*.

Df(3R)Ant6

cytology: *Df(3R)84B2;84C6*.
synonym: *Df(3R)Antp^{RA75}*.
origin: X ray induced.
references: Abbott and Kaufman, 1986, Genetics 114: 919-42.
genetics: Not *Antp* over wild type. Complements *Antp²³* and *Antp¹*.

Df(3R)Antp7

cytology: *Df(3R)84B1-2;84D*.
origin: Induced by ethyl methanesulfonate in *Antp^{NS}*.
discoverer: Struhl.
synonym: *Df(3R)Antp^{NS+RC7}* (Struhl, 1981; Garber *et al.*, 1983).
references: Struhl, 1981, Nature 292: 635-38.
 Garber, Kuroiwa, and Gehring, 1983, EMBO J. 2: 2027-36.
genetics: Deficient for *Antp* but not for *ftz* or *Scr* (Struhl 1981; Riley, Carroll, and Scott, 1987, Genes and Development 1: 716-30).
molecular biology: Proximal breakpoint 154 kb to the left of the proximal breakpoint of *In(3R)Hu* (Garber *et al.*, 1983).

Df(3R)Antp17

cytology: *Df(3R)84A6;84D13-14* (Baker); *84B1;84D11-12* (Lewis *et al.*, 1980).
origin: X ray induced in *Antp^{NS}*.
synonym: *Df(3R)Antp^{NS+R17}* (Duncan and Kaufman, 1975).
references: Duncan and Kaufman, 1975, Genetics 80: 733-52.
 Lewis, Kaufman, and Denell, 1980, DIS 55: 85-87.
 Lewis, Kaufman, Denell, and Talerico, 1980, Genetics 95: 367-81.
 Wakimoto, Lewis, and Kaufman, 1980, DIS 55: 140-41.
 Seeger, Haffley, and Kaufman, 1988, Cell 55: 589-600.
genetics: Deficient for *Scr-roe*; includes *Est-C* (Ohnishi and Voelker, 1982, DIS 58: 120). Associated with dominant reduced-sex-comb phenotype. Fails to complement the recessive lethality of *Scr^{Msc}*, *Antp*, and *Scx*. Embryonic lethal with *Df(3R)Scx*. Male heterozygotes

semisterile (Kaufman).

molecular biology: Distal (84D13-14) breakpoint located at -31 kb (Baker and Wolfner; 0 point = Hind III site in *αTub84D*, "+" values to the right, "-" values to the left).

Df(3R)Antp72: see In(3R)Antp^{rv72}

Df(3R)Antp-Scx: see Df(3R)Scx2

Df(3R)Antp-X1

cytology: *Df(3R)83F4-84A1;84B1-2 + Df(3R)84B6-C1;84C5-6*; deficiencies for two noncontiguous segments juxtaposed by *In(3R)Antp^{73b}* = *In(3R)84B1-2;84C5-6*.
new order: 61-83F4|84B6-84B2|84C6-100.
origin: X-ray-induced in *In(3R)Antp^{73b}*.
synonym: *Df(3R)Antp^{73b+RX1}* (Cavener *et al.*, 1986); *Df(3R)Antp^{73b+RX3}* (Hazelrigg and Kaufman, 1983).
references: Hazelrigg and Kaufman, 1983, Genetics 105: 581-600.
 Cavener, Otteson, and Kaufman, 1986, Genetics 114: 111-23.
 Cavener, Corbett, Cox, and Whetten, 1986, EMBO J. 5: 2939-48.
genetics: Deficient for *Antp*; shows reduced-sex-comb phenotype. Marked with *Ki*.
molecular biology: Distal breakpoint at about +156 kb in the molecular map of Cavener *et al.*, 1986 (ref. #2).
other information: Isolated independently of *Df(3R)Antp-X2* (Hazelrigg and Kaufman, 1983).

Df(3R)Antp-X2

cytology: *Df(3R)83F4-84A1;84B1-2 + Df(3R)84B6-C1;84C5-6*; deficiencies for two noncontiguous segments juxtaposed by *In(3R)Antp^{73b}* = *In(3R)84B1-2;84C5-6*.
new order: 61-83F4|84B6-84B2|84C6-100.
origin: X-ray-induced in *In(3R)Antp^{73b}*.
synonym: *Df(3R)Antp^{73b+RX2}* (Cavener *et al.*, 1986); *Df(3R)Antp^{73b+RX4}* (Hazelrigg and Kaufman, 1983).
references: Hazelrigg and Kaufman, 1983, Genetics 105: 581-600.
 Cavener, Otteson, and Kaufman, 1986, Genetics 114: 111-23.
genetics: Deficient for *Antp*; shows reduced-sex-comb phenotype. Marked with *red e*.

Df(3R)awd7: Deficiency (3R) abnormal wing disks.

references: Biggs, Tripoulas, Dearolf, and Shearn, 1988, Genes Dev. 2: 1333-43.
genetics: Deficient for *awd* and both flanking transcription units.

Df(3R)awd21

cytology: Deficient for 100C-D.
references: Biggs, Tripoulas, Hersperger, Dearolf, and Shearn, 1988, Genes Dev. 2: 1333-43.
genetics: Deficient for *awd*.

Df(3R)β2t: see In(3R)Scr^{MscL} Antp^{BR}

Df(3R)Bd: Deficiency (3R) Beaded

cytology: *Df(3R)97D;97F-98A*.
origin: X ray induced.
discoverer: P. Lewis.
synonym: *Df(3R)Ser^{+82f24}*.

genetics: Fails to complement lethality of *Bd*³; also slightly enhances *nd* on the X chromosome. Deficient for *ro* and *Bd*.

Df(3R)bxd100: Deficiency (3R) bithoraxoid

cytology: *Df(3R)89B5-6;89E2-3*.
origin: Aneuploid recombinant from *Tp(3;3)bxd*¹⁰⁰/+.
discoverer: E. B. Lewis.
references: Struhl, 1982, Genetics 102: 737-49.
genetics: Deficient for *ss*^a, *bx*, and *Ubx*; mutant for *bxd*.

Df(3R)bxd110: see Tp(3;3)bxd¹¹⁰

Df(3R)by: Deficiency (3R) blistery

origin: X ray induced.
references: Kempfues, Raff, Raff, and Kaufman, 1980, Cell 21: 445-51.
 Kempfues, Raff, and Kaufman, 1983, Genetics 105: 345-56.

deficiency	cytology	genetics
<i>Df(3R)by10</i>	85D8-12;85E7-F1	by ⁻ -M(3)85E ⁻
<i>Df(3R)by61</i>	85D11-14;85F16	by ⁻ -M(3)85E ⁻
<i>Df(3R)by62</i>	85D11-14;85F16	by ⁻ -M(3)85E ⁻
<i>Df(3R)by77</i>	85D8-12;86B4	by ⁻ -M(3)85E ⁻
<i>Df(3R)by416</i>	85D10-12;85E1-3	by ⁻

Df(3R)c3G2

cytology: *Df(3R)89A2-3;89A4-5*.
references: Hughes, Nelson, Yanuk, and Szauter (unpublished).
genetics: Deficient for *c(3)G*.

Df(3R)C4

cytology: *Df(3R)89E;90A*.
origin: X ray induced in *Mcp*.
discoverer: Crosby.
references: Kuhn, Woods, and Andrew, 1981, Genetics 99: 99-107.
 Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.
 Struhl and White, 1985, Cell 45: 507-19.
 Casanova, Sánchez-Herrero, and Morata, 1986, Cell 47: 627-36.
genetics: Deficient for *iab6-iab9* (Karch *et al.*, 1985). Includes *Abd-B* and is associated with slight reductions in *Ubx* and *abd-A* activity.
molecular biology: Proximal breakpoint 133.5-137 kb distal to the right breakpoint of *In(3R)Cbx*^{rv1}.

Df(3R)ca: Deficiency (3R) claret

origin: X ray induced in *Dp(3;1)B152*.
genetics: Deficient for *ca* and *M(3)99B*.

deficiency	cytology	synonym	ref ^α
<i>Df(3R)ca1A</i> ^β	98F14;100B5-7 (Strecker)	<i>Dp(3;1)1A</i>	1, 2
<i>Df(3R)ca3</i>	98F14;99E5-F1	<i>Dp(3;1)3</i>	1, 2
<i>Df(3R)ca19</i>	98F14;99F7-8	<i>Dp(3;1)19</i>	1
<i>Df(3R)ca27</i>	99B4-5;99B10-C1	<i>Dp(3;1)27</i>	1, 2
<i>Df(3R)ca34</i>	98F14;99E5-F1	<i>Dp(3;1)34</i>	1, 2
<i>Df(3R)ca35</i>	98F14;99D3-5	<i>Dp(3;1)35</i>	1
<i>Df(3R)ca45B</i>	98F14;99C1-2	<i>Dp(3;1)45B</i>	1
<i>Df(3R)ca46A</i>	98F14;99B5-9	<i>Dp(3;1)46A</i>	1, 2
<i>Df(3R)ca48</i>	98F14;100B7-8; complex	<i>Dp(3;1)48</i>	1, 2
<i>Df(3R)ca52</i>	99A9-10;99C5-6	<i>Dp(3;1)52</i>	1
<i>Df(3R)ca67A</i>	98F14;99D9-E1	<i>Dp(3;1)67A</i>	1, 2
<i>Df(3R)ca67N</i> ^β	98F14;99D6-9	<i>Dp(3;1)67N</i>	1, 2

deficiency	cytology	synonym	ref ^α
<i>Df(3R)ca74</i>	98F14;99C2-6	<i>Dp(3;1)74</i>	1, 2
<i>Df(3R)ca78</i>	98F14;99C5-7	<i>Dp(3;1)78</i>	1, 2
<i>Df(3R)ca79</i> ^β	98F14;100B4-5 (Strecker)	<i>Dp(3;1)79</i>	1, 2
<i>Df(3R)ca88</i>	98F14;99F6-8	<i>Dp(3;1)88</i>	1, 2
<i>Df(3R)ca93</i>	98F14;99F9-10	<i>Dp(3;1)93</i>	1, 2
<i>Df(3R)ca97</i>	98F14;99E1-2	<i>Dp(3;1)97</i>	1
<i>Df(3R)ca124P</i>	99A2-3;99E4-5 ^γ	<i>Dp(3;1)124P</i>	1, 2
<i>Df(3R)ca138P</i>	99B5-9;99F9-100A1	<i>Dp(3;1)138P</i>	1
<i>Df(3R)ca150P</i>	98F14;100B1-2	<i>Dp(3;1)150P</i>	1, 2
<i>Df(3R)ca152P</i> ^β	98F14;100A1-2 (Strecker)	<i>Dp(3;1)152P</i>	1, 2
<i>Df(3R)ca165P</i>	99B2-4;99C5-6	<i>Dp(3;1)165P</i>	1
<i>Df(3R)ca-L127</i>	99B5-6;99E4-F1	<i>Dp(3;1)L127</i>	1
<i>Df(3R)ca-R10</i> ^β	98F14;99D6-9	<i>Dp(3;1)R10</i>	1, 2
<i>Df(3R)ca-R14</i> ^β	99A8-9;99D1-2	<i>Dp(3;1)R14</i>	1, 2
<i>Df(3R)ca-R15</i>	98F14;99F7-8	<i>Dp(3;1)R15</i>	

^α 1 = Frisardi and MacIntyre, 1984, Mol. Gen. Genet. 197: 403-13; 2 = Kongsuwan, Dellavalle, and Merriam, 1986, Genetics 112: 539-50.
^β Cytology of Frisardi and MacIntyre modified by Kongsuwan *et al.*
^γ Distal break in the first intron of *Mlc2* (Warmke, Kreuz, and Falkenthal, 1989, Genetics 122: 139-51).

Df(3R)CA

origin: Hybrid dysgenesis.

deficiency	cytology	ref ^α	genetics	molec. biol. ^β
<i>Df(3R)CA1</i>	84E12-13;85A6-11	2	<i>l(3)84Ef⁻-l(3)85Ac⁻</i> includes <i>Dhod</i> and <i>p</i>	+33
<i>Df(3R)CA3</i>	84F2-6;85A6-11	1, 2	<i>l(3)84Ef⁻-l(3)85Ac⁻</i> includes <i>Dhod</i> and <i>p</i>	+33
<i>Df(3R)CA4B</i>	85A6-11;85C3-6	2	<i>Dhod⁺</i> <i>p⁻-hb⁺</i>	+22

^α 1 = Bender, Turner, and Kaufman, 1987, Dev. Biol. 119: 418-32; 2 = Jones and Rawls, 1988, Genetics 120: 733-42.
^β DNA coordinates from 85A map (Jones and Rawls, 1988).

Df(3R)Cha: Deficiency (3R) Choline Acetyltransferase

deficiency	cytology	discoverer	synonym	ref ^α	genetics
<i>Df(3R)Cha1</i>	91A1-2;91E	Greenspan	<i>Def-Cat</i> ; <i>Def-Cha</i>	5, 6	<i>Cha⁻</i>
<i>Df(3R)Cha1a</i>	91C2-3;91F5-92A1	Myers, Gelbart	<i>Df(3R)Cha^{M1}</i>	1-3	<i>Cha⁻-l(3)91Ff⁻</i>
<i>Df(3R)Cha2</i>	91C7-D1;92A2	Myers, Gelbart	<i>Df(3R)Cha^{M2}</i>	2, 3	<i>Cha⁻-Dl⁻</i>
<i>Df(3R)Cha5</i>	91B3;91D1	Gelbart	<i>Df(3R)148.5-1</i>	3-5	<i>fru⁻-Cha⁻</i>
<i>Df(3R)Cha7</i>	91A;91F3	Myers, Gelbart	<i>Df(3R)Cha^{M7}</i>	2-4	<i>gl⁻-Cha⁻</i>
<i>Df(3R)Cha9</i>	91C7-D1;92A2	Myers, Gelbart	<i>Df(3R)Cha^{M9}</i>	2,4	<i>Cha⁻-Dl⁻</i>
<i>Df(3R)Cha12</i>	91A;91D1-2 + <i>In(3R)90F;100A1</i>			3	<i>Cha⁻</i>

^α 1 = Alton, Fechtel, Kopczynski, Shepard, Kooh, and Muskavitch, 1989, Dev. Genet. 10: 261-72; 2 = Alton, Fechtel, Terry, Meikle, and Muskavitch, 1988, Genetics 118: 235-45; 3 = Ashburner, 4 = Gailey and Hall, Genetics 121: 773-85; 5 = Gorczyca and Hall, 1984, J. Neurogenet. 1: 289-313; 6 = Greenspan, 1980, J. Comp. Physiol. 137: 83-92.

Df(3R)CP1

cytology: *Df(3R)84A5;84B1*.
references: Source of deficiency unknown, cytology from Kaufman's laboratory.

Df(3R)crb87-4

cytology: Df(3R)95F15;96A1.
references: Wustmann, Szidonya, Taubert, and Reuter, 1989, Mol. Gen. Genet. 217: 520-27.
genetics: Deficient for *crb*.

Df(3R)crb87-5

cytology: Df(3R)95F7-9;96A17-18.
references: Wustmann, Szidonya, Taubert, and Reuter, 1989, Mol. Gen. Genet. 217: 520-27.
genetics: Deficient for *crb*.

Df(3R)cu

cytology: Df(3R)86C1-2;86D8.
discoverer: Holden.
synonym: Df(3R)cu40.
references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.
 Ohnishi and Voelker, 1982, DIS 58: 120.
genetics: Deficient for *Odh* and *cu*.

Df(3R)D: Deficiency (3R) Duncan

discoverer: Duncan.

deficiency	cytology	ref ^α	genetics
Df(3R)D6	84D2-3;84F11-16	1	roe ⁻ - dsx ⁻
Df(3R)D7	84D3-5;84F1-2	1	p ⁻ - dsx ⁻
Df(3R)D8 ^β	84D3-5;85A2-4	2	l(3)84Ef ⁻ - l(3)85Aa ⁻

^α 1 = Cavener, Otteson, and Kaufman, 1986, Genetics 114: 111-23;
 2 = Jones and Rawls, 1988, Genetics 120: 733-42.
^β At molecular map coordinate -57 on the 85A DNA map (Jones and Rawls, 1988).

Df(3R)Dfd13: Deficiency (3R) Deformed

cytology: Df(3R)83E3;84A4-5.
origin: X-ray-induced reversion of *Dfd*.
synonym: Df(3R)Dfd^{+RX13}.
references: Hazelrigg and Kaufman, 1983, Genetics 105: 581-600.
genetics: Reduced-sex-comb phenotype in heterozygotes. Deficient for *Dfd*, *Scr* and *ftz* but not *Antp*.

Df(3R)DG

deficiency	cytology	genetics
Df(3R)DG1	90E1-2;91C3-7	sr ⁻ - gl ⁻
Df(3R)DG2	89E-F;91B1-2	sr ⁻ - gl ⁻
Df(3R)DG3	89F;90F	sr ⁻
Df(3R)DG4	90E1-2;90F3-11	sr ⁻
Df(3R)DG5	90E-F;91E-F	gl ⁻

Df(3R)Dhod15: Deficiency (3R) Dihydroorotase

cytology: {Df(3R)85A;85A}.
discoverer: Rawls and Jones.
references: Bender, Turner, and Kaufman, 1987, Dev. Biol. 119: 418-32.
genetics: Deficient for *Dhod*.

Df(3R)Dipr: Deficiency (3R) Distal into proximal

origin: X ray induced revertants of *rn*^D (= *Dipr*).
references: Kerridge, 1981, Mol. Gen. Genet. 184: 519-23.

genetics: Deficient for *rn*. Lethal with *Df(3R)Antp17*.

deficiency	cytology	synonym
Df(3R)Dipr2	84F;84F	
Df(3R)Dipr4	84F;84F	
Df(3R)Dipr8	84F;84F	
Df(3R)Dipr11	84D;84F	Df(3R)Dipr ^{+R1}
Df(3R)Dipr12	84D;84F	Df(3R)Dipr ^{+R2}
Df(3R)Dipr15	84D;84F + In(3R)84D3-4;84F6-11	Df(3R)Dipr ^{+R5}
Df(3R)Dipr17	84D;84F	Df(3R)Dipr ^{+R7}
Df(3R)Dipr19	84D;84F	Df(3R)Dipr ^{+R9}

Df(3R)DI: Deficiency (3R) Delta

deficiency	cytology	origin	discoverer	ref ^α	genetics
Df(3R)DI1	90D2-4;92A1-2	spont	Schultz	4	DI ⁻
Df(3R)DI2	91D3;92A1-2	X ray	Myers		DI ⁻
Df(3R)DI5 ^β	91F5-13;92E1-11	X ray		5,7	DI ⁻ - tRNA - val3b ⁻
Df(3R)DI12	91F6-13;92A1	spont	Bridges	2	DI ⁻
Df(3R)DI-A143	91F13;92A2-3	HD	Tepass	8	DI ⁻
Df(3R)DI-BX6	92A1-2	X ray		1	DI ⁻
Df(3R)DI-BX12	91F1-2;92D3-6	X ray	Muskavitch	1, 2	l(3)91Fb ⁻ - DI ⁻
Df(3R)DI-FX2	91D3;92A5-8	X ray		6,8	DI ⁻
Df(3R)DI-FX3	91F11;92A3	X ray		6,8	DI ⁻
*Df(3R)DI-H	91C6-D1;92A2-3	chemical	Auerbach	3, 4	DI ⁻
Df(3R)DI-HD28	91F6-13;92A2-3	HD		1, 2	l(3)91Fd ⁻ - DI ⁻
Df(3R)DI-KX4	92A2;92B3-10	X ray			DI ⁻
Df(3R)DI-KX5	91C;92E	X ray	Vässin	8	DI ⁻
Df(3R)DI-KX9	91F6-10;92B4-8	X ray	Vässin	8	DI ⁻
Df(3R)DI-KX10	91F6-10;92B	X ray	De la Concha	8	DI ⁻
Df(3R)DI-KX12	92A2;92A + In(3)67C-D;92A2	X ray	Vässin	8	DI ⁻
Df(3R)DI-KX16	91C;92B11-C1	X ray	Vässin	8	DI ⁻
Df(3R)DI-KX17	91F10-13;92E	X ray	Vässin	8	DI ⁻
Df(3R)DI-KX18	91F13-92A1; 92D7-E3	X ray	Vässin	8	DI ⁻
Df(3R)DI-KX20	91F1-5;92A2	X ray	Vässin	8	DI ⁻
Df(3R)DI-KX21	91F3;92A2	X ray	Vässin	8	DI ⁻
Df(3R)DI-KX23	91C7-D3;92A5-8	X ray	Vässin	8	DI ⁻
Df(3R)DI-KX24	91A1-2;92B9-11	X ray	Vässin	8	DI ⁻
Df(3R)DI-M2	91C7-D1;93A1		Myers, Gelbart	2	DI ⁻
Df(3R)DI-P	91F10;92B3	HD	Vässin	8	DI ⁻
Df(3R)DI-PX	91D1;92A7	X ray	Vässin	6, 8	DI ⁻
Df(3R)DI-X43	91F11;92A8-10	X ray	Campos-Ortega	2, 6, 8	DI ⁻

^α 1 = Alton, Fechtel, Kocpczynski, Shepard, Kooh, and Muskavitch, 1989, Dev. Genet. 10: 261-72; 2 = Alton, Fechtel, Terry, Meikle, and Muskavitch, 1988, Genetics 118: 235-45; 3 = CP627; 4 = Auerbach, 1943, DIS 17: 49; 5 = Larson, Miller, Spiegelman, Hayashi, Tener, Sinclair, and Grigliatti, 1982, Mol. Gen. Genet. 185: 390-96; 6 = Lehmann, Jiménez, Dietrich, and Campos-Ortega, 1983, Wilhelm Roux's Arch. Dev. Biol. 192: 62-74; 7 = Scavarda, O'Tousa, and Pak, 1983, Proc. Nat. Acad. Sci. USA 80: 4441-45; 8 = Vässin and Campos-Ortega, 1987, Genetics 116: 433-45.
^β Also deficient for *ninaE* (Larson *et al.*, 1982).

Df(3R)dsx: Deficiency (3R) doublesex

origin: X ray induced.
genetics: Revertants of dominant alleles at the *dsx* locus affecting chromosomally female flies.

deficiency	cytology	synonym ^α	ref ^β
Df(3R)dsx1	84D6-7;85A1-3	Df(3R)dsx ^{D+R1}	4
Df(3R)dsx2D	84D11;84F16	Df(3R)dsx ^{D+R2}	4
Df(3R)dsx2M ^γ	84C1-3;84E1 (Baker)	Df(3R)dsx ^{Mas+R2}	1, 3
Df(3R)dsx3	84D11-14;84E12-13	Df(3R)dsx ^{Mas+R3}	1
Df(3R)dsx5 ^δ	84E1-2;84F11-12	Df(3R)dsx ^{D+R5}	2, 4
Df(3R)dsx8 ^ε	84D13-14;85A1-3	Df(3R)dsx ^{D+R8}	2
Df(3R)dsx10D ^ε	84D11-12;85A1-3	Df(3R)dsx ^{D+R10}	6
Df(3R)dsx10M	84D3;84F1-2	Df(3R)dsx ^{Mas+R10}	1
Df(3R)dsx11 ^ζ	84D8-9;85A1-3	Df(3R)dsx ^{Mas+R11}	1, 5
Df(3R)dsx15	84D11;84E8	Df(3R)dsx ^{Mas+R15}	1

deficiency	cytology	synonym α	ref β
Df(3R)dsx21	84D11-12,84E8	<i>Df(3R)dsx^{Mas+R21}</i>	1
Df(3R)dsx27	84D9 (within); 85A1-3 (near distal edge)	<i>Df(3R)dsx^{Mas+R27}</i>	1
Df(3R)dsx28	84D13-E1,85A4-5	<i>Df(3R)dsx^{Mas+R28}</i>	1
Df(3R)dsx29	84C8-D1 (interband); 84F6-7 (within)	<i>Df(3R)dsx^{Mas+R29}</i>	1, 3
Df(3R)dsx33 η	84D10-11 (interband); 85A1-3 (distal edge)	<i>Df(3R)dsx^{Mas+R33}</i>	1
Df(3R)dsx34	84D3-4;85B4-5	<i>Df(3R)dsx^{Mas+R34}</i>	1
Df(3R)dsx37	84D8;85B3-5	<i>Df(3R)dsx^{Mas+R37}</i>	1
Df(3R)dsx43	84D13-14,84E6-8	<i>Df(3R)dsx^{Mas+R43}</i>	1
Df(3R)dsx48	see <i>In(3R)dsx⁴⁸</i>		

α Original strain (*dsx^D* or *dsx^{Mas}*) indicated in superscript.

β 1 = Baker, Hoff, Kaufman, Wolfner, and Hazelrigg, 1991, Genetics 127: 125-38; 2 = Baker and Ridge, 1980, Genetics 94: 383-423; 3 = Cavener, Corbett, Cox, and Whetten, 1986, EMBO J. 5: 2939-48; 4 = Duncan and Kaufman, 1975, Genetics 80: 733-52; 5 = Jones and Rawls, 1988, Genetics 120: 733-42; 6 = Kaufman.

γ Proximal (84C1-3) breakpoint estimated to be at about +26 to +27 kb, based on location of 84C1-2 breaks of *T(2;3)Ta¹* at +34 kb and *Df(3R)Scx²* at +41 kb (Baker and Wolfner, 1988, Genes Dev. 2: 477-89). Also deficient for *hat* and *nac*.

δ Part of 84E1-2 fused with part of 84F11-12. Proximal breakpoint at about -4kb on the molecular map of Cavener *et al.*, 1986.

ϵ Deficient for *Su(var)304* as well as *dsx^D* (Reuter, Dorn, Wustmann, Friede, and Rauh, 1986, Mol. Gen. Genet. 202: 481-87).

ζ Broken within 85A1-3, deleting centromere proximal but not centromere distal material (B.S. Baker; Jones and Rawls, 1988).

η Proximal breakpoint at about +134 kb on the molecular map of Cavener *et al.*, 1986.

Df(3R)e: Deficiency (3R) ebony

origin: *Df(3R)e-G* chromosomes were γ ray induced, other deficiencies X ray induced.

deficiency	cytology	ref α	genetics
*Df(3R)e4.39	93B,93F	1	<i>e⁻</i>
Df(3R)e671	93B5-7,93D3	6	<i>e⁻</i>
*Df(3R)e100.172	93B7-19,93F10-94A1	12	<i>e⁻</i>
*Df(3R)e100.256	93A5-B1,93F5-9	12	<i>e⁻</i>
Df(3R)e-B52	93C3-6,93F5-8		
Df(3R)e-D7 β	93C3-6,93F6-8	2, 3, 7, 8, 10, 11	<i>l(3)93Cb⁻-l(3)93Fa⁻</i>
Df(3R)e-F1	93B6-7,93E1-2	4, 7, 8, 10, 11	<i>l(3)93Cb⁻-l(3)93Di⁻</i>
Df(3R)e-F2 β	93A6-B1,93D7-10	4, 7, 8, 10	<i>l(2)93Ba⁻-l(3)93Dh⁻</i>
Df(3R)e-F3 β	93B8-13,93E6-11	4, 7, 8, 10	<i>l(3)93Ba⁻-l(3)93Dj⁻</i>
Df(3R)e-F4 β	93C3-6,93F11-14	4, 7, 8, 10	<i>l(3)93Ca⁻-l(3)93Fc⁻</i>
Df(3R)e-GC3	93C3-6,94A	7, 8	<i>l(3)93Cb⁻-l(3)93Fc⁻</i>
Df(3R)e-GC9	93B11-13,93D9-10	7, 8	<i>l(3)93Ba⁻-l(3)93Dh⁻</i>
Df(3R)e-Gp4	93B11-13,93D7-9	7, 8	<i>l(3)93Ba⁻-Hsr93D⁻</i>
Df(3R)e-H4 γ	93C3-6,93F6-8	7, 8	<i>l(3)93Cc⁻-l(3)93Fc⁻</i>
Df(3R)e-H5 γ	93B11-13,93D4-6	5, 7, 8	<i>l(3)93Ba⁻-l(3)93Da⁻</i>
Df(3R)e-H6 γ	93C3-6,94A	7, 8	<i>l(3)93Cb⁻-l(3)93Fc⁻</i>
Df(3R)e-N19	93B,94	11	<i>e⁻</i>
Df(3R)e-N26	93B,93F	11	<i>l(3)93Ba⁻-e⁻</i>
Df(3R)e-R1 δ	93B3-5,93D2-4	7, 8, 9	<i>l(3)93Ba⁻-e⁻</i>
Df(3R)e-R6 δ	93B4-5,94A5-16	9	<i>e⁻</i>

α 1 = Alexander, 1960, Genetics 45: 1019-22; 2 = Caggese, Caizzi, Morea, Scalenghe, and Ritossa, 1979, Proc. Nat. Acad. Sci. USA 76: 2385-89; 3 = D'Alessandro, Ritossa, and Scalenghe, 1977, DIS 52: 46; 4 = Fortebraccio, Scalenghe, and Ritossa, 1977, DIS 52: 102; 5 = Henikoff, 1980, DIS 55: 61-62; 6 = Korge, 1972, DIS 48: 20; 7 = Mohler and Pardue, 1982, Chromosoma 86: 457-67; 8 = Mohler and Pardue, 1984, Genetics 106: 249-65; 9 = Rawls, 1980, Mol. Gen. Genet. 178: 43-49; 10 = Scalenghe and Ritossa, 1976, Atti Accad. Naz. Lincei 13: 439-528; 11 = Scalenghe and Ritossa, 1977, Chromosoma 63: 317-26; 12 = Ward and Alexander, 1957, Genetics 42: 42-54.

β Cytology according to Mohler and Pardue, 1984 (different breakpoints given by Scalenghe and Ritossa, 1977).
 γ Discovered by S. Henikoff.
 δ Discovered by B.S. Baker.

Df(3R)E45

cytology: *Df(3R)100C5;100F*.

origin: P-element induced mutagenesis.

references: Locke, Kotarski, and Tartof, 1988, Genetics 120: 181-98.

genetics: Enhances position-effect variegation in *In(1)w^{m4}*.

Df(3R)E79

cytology: *Df(3R)86F1-2;87B8-10* (Gausz).

origin: Induced by ethyl methanesulfonate.

references: Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz, 1981, Genetics 98: 775-89.

Ohnishi and Voelker, 1981, Biochem. Genet. 19: 75-85.

genetics: Deficient for *Dip-C* and *l(3)86Fa - l(3)87Bg*. Lacks heat shock puff site at 87A7.

Df(3R)E229

cytology: *Df(3R)86F6-7;87B1-2* (Gausz).

origin: Induced by ethyl methanesulfonate.

synonym: *Df(3R)229* (Ish-Horowicz and Pinchin, 1980).

references: Ish-Horowicz, Pinchin, Gausz, Gyurkovics, Bencze, Goldschmidt-Clermont, and Holden, 1979, Cell 17: 565-71.

Ish-Horowicz and Pinchin, 1980, J. Mol. Biol. 142: 231-45.

Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz, 1981, Genetics 98: 775-89.

Jürgens, Wieschhaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

genetics: Deficient for *l(3)86Fe - l(3)87Ae*. Lacks heat shock puff site at 87A7. Uncovers *sad* (Jürgens *et al.*, 1984).

Df(3R)E307

cytology: *Df(3R)87B2-4;87D1-2* (Gausz).

origin: Induced by ethyl methanesulfonate.

references: Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz, 1981, Genetics 98: 775-89.

genetics: Deficient for *Dip-C - l(3)87Bg*. Lacks heat shock puff site at 87C1.

Df(3R)ea: Deficiency (3R) easter

cytology: *Df(3R)88E7-13;89A1*.

origin: Induced by hybrid dysgenesis.

references: Chasan and Anderson, 1989, Cell 56: 591-600.

genetics: Fertile revertant of dominant *easter* allele.

Df(3R)Espl: Deficiency (3R) Enhancer of spl

genetics: Deficient for *E(spl)*.

deficiency	cytology	origin	synonym	ref α
Df(3R)Espl1	96F7,97A6	X ray	<i>E(spl)</i> ^{8D06}	1
Df(3R)Espl2	<i>In(3R)96C3-9;96E5-12;97A3-4</i> (deficient for 96E5,97A4)	X ray	<i>E(spl)</i> ^{A7.13.2}	2
Df(3R)Espl3 β	96F1,97B1		<i>E(spl)</i> ^{BX36}	4
Df(3R)Espl4	96F8-9,96F12-13	X ray	<i>E(spl)</i> ^{RB25.1}	2
Df(3R)Espl5	<i>In(3R)96F2;96F12-14;99C</i> (deficient for 96F12-14)		<i>E(spl)</i> ^{R1}	3
Df(3R)Espl6	96F5,97A9-10	X ray	<i>E(spl)</i> ^{R2}	3
Df(3R)Espl7	96F9,97A4-6	X ray	<i>E(spl)</i> ^{R3}	3
Df(3R)Espl8	96F9-10,97A6-10	X ray	<i>E(spl)</i> ^{R23.1}	2
Df(3R)Espl9 γ	96F5-7,96F12-14	X ray	<i>l(gro)</i> ^{X1}	4

deficiency	cytology	origin	synonym	ref ^α
Df(3R)Esp10^γ	96F5-7;97B1	X ray	<i>l(gro)</i> ^{X72}	4
^α	1 = Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Roux's Arch. Dev. Biol. 93: 283-95; 2 = Knust, Bremer, Vässin, Ziemer, Tepass, and Campos-Ortega, 1987, Dev. Biol. 122: 262-73; 3 = Lehmann, Dietrich, and Campos-Ortega, 1985, J. Neurogenet. 2: 291-308; 4 = Preiss, Hartley, and Campos-Ortega, 1988, EMBO J. 7: 3917-27.			
^β	Discoverer: Muskavitch.			
^γ	Discoverer: Preiss.			

Df(3R)F76

cytology: {*Df(3R)84F;84F*}.
genetics: *roe*⁻ - *rn*⁻.

Df(3R)fs293

cytology: *Df(3R)?;88A1-2*.
genetics: Deficient for *ems*.

Df(3R)G1

cytology: *Df(3R)85A4-5;85A6-11*.
discoverer: Porter.
references: Bender, Turner, and Kaufman, 1987, Dev. Biol. 119: 418-32.
Jones and Rawls, 1988, Genetics 120: 733-42.
genetics: Deficient for *Dhod*, *p*, *l(3)85Ac-l(3)85Ag*.
molecular biology: DNA coordinates of the 85A proximal breakpoint and the 85A distal breakpoint are -49 and +45 respectively ("+" values to the right and "-" values to the left or centromere end of map).

Df(3R)GB14

cytology: *Df(3R)85D12;85E10*.

Df(3R)GB104

cytology: *Df(3R)85D11-13;85E10*.
genetics: Deficient for α *Tub85E*.

Df(3R)GC14

cytology: {*Df(3R)93D6-7;93D9-10*}.
origin: γ ray induced.
references: Mohler and Pardue, 1982, Chromosoma 86: 457-67.
1984, Genetics 106: 249-65.
genetics: Deficient for *Hsr-93D* and *l(3)93Dh*.

Df(3R)GE

origin: X ray induced loss of *TE88A*.
references: Gausz, Hall, Spierer, and Spierer, 1986, Genetics 112: 65-78.

deficiency	cytology	genetics	DNA breakpoints (proximal) ^α
Df(3R)GE26	87E1-2		-30, -23
Df(3R)GE41^β	87E4	<i>Ace</i> ⁺ <i>l(3)87Ee</i> ⁻ - <i>l(3)87Ef</i> ⁻	+56.5, +58.8
Df(3R)GE99	87E1-2		

^α *ry-Ace* walk, "+" values to right, "-" values to left (Gausz *et al.*, 1986).
^β Other molecular references: Hall and Spierer, 1986, EMBO J. 5: 2949-54; Nagoshi and Gelbart, 1987, Genetics 117: 487-502; Fournier, Karch, Bride, Hall, Bergé, and Spierer, 1989, J. Mol. Biol. 210: 15-22.

Df(3R)gl: Deficiency (3R) glass

deficiency	cytology	genetics
Df(3R)gl-BX1	90F8-11;91B1-2	<i>sr</i> ⁺ <i>gl</i> ⁻ - <i>fru</i> ⁻
Df(3R)gl-BX5	91B1-2;91D1-2	
Df(3R)gl-BX6	90C9-10;90F2-11	<i>sr</i> ⁻ <i>gl</i> ⁺
Df(3R)gl-BX10	90C7-8;91B1-2	<i>sr</i> ⁻ - <i>fru</i> ⁻

Df(3R)gro: Deficiency (3R) groucho genetics: Deficient for *E(spl)*.

deficiency	cytology
Df(3R)gro-X1	96F5-7;96F12-14
Df(3R)gro-X2	96F5-7;97B1

Df(3R)H5: see *Df(3R)kar-H5*

Df(3R)H10: see *Df(3R)kar-H10*

Df(3R)H13: see *Df(3R)kar-H13*

Df(3R)H-B79

cytology: *Df(3R)92B3-11;92F8-13*.
references: Wustmann, Szidonya, Taubert, and Reuter, 1989, Mol. Gen. Genet. 217: 520-27.
Bang, Hartenstein, and Posakony, 1991, Development 111: 89-104.
genetics: Deficient for *H*.

Df(3R)hh: Deficiency (3R) hedgehog

cytology: *Df(3R)92F11-14;94E2-5*.
origin: γ ray induced.
synonym: *Df(3R)hh-GR2*.
references: Mohler, 1988, Genetics 120: 1061-72.
genetics: Deficient for *hh*.

Df(3R)Hu: Deficiency (3R) Humeral

cytology: *Df(3R)84A6-B1;84B3-6 + Df(3R)84D4-5;84F1-2*; deficiencies for two non-contiguous deletions juxtaposed by *In(3R)Hu*.
new order: 61 - 84A6|84D4 - 84B6|86C5 - 84F2|86C6 - 100.
origin: Induced by ethyl methanesulfonate in *In(3R)Hu*.
synonym: *Df(3R)Hu^{rvX1}*; *Df(3R)Hu^{+RX1}*.
references: Hazelrigg and Kaufman, 1983, Genetics 105: 581-600.
Cavener, Otteson, and Kaufman, 1986, Genetics 114: 111-23.
genetics: Deficient for *Antp* and *Hu* but not *ftz*. Male heterozygote shows reduced sex comb phenotype.
molecular biology: Proximal end of the deficiency at +88; "+" values to the right, "-" values to the left. (Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, Cell 35: 763-76).

Df(3R)I9

cytology: *Df(3R)92B1-3;92C1-3*.
genetics: Deficient for *l(3)SB65*.

Df(3R)JK6: see *Df(3R)urd*

Df(3R)K9

cytology: *Df(3R)91B1-5;92C3-D1*.
genetics: Deficient for *l(3)SB65*.

Df(3R)kar: Deficiency (3R) karmoisin
origin: X ray induced.

deficiency	cytology	ref ^α	genetics
Df(3R)kar1W^{β,δ}	87A6-7;87D12-13	2, 4, 5, 6	<i>Hsp70</i> ⁻ - <i>ry</i> ⁻
Df(3R)kar3J^{β,γ}	87B15-C1;87C9-D1	1, 2, 4, 5, 6	<i>l(3)87Ca</i> ⁻ - <i>l(3)87Cd</i> ⁻
Df(3R)kar3I^γ	87C2-3;87D3-4	2, 3, 4, 5, 6, 8	<i>l(3)87Ca</i> ⁻ - <i>Men</i> ⁻
Df(3R)kar3Q^{β,γ}	87B2-4;87C9-D3	2, 4, 5, 6, 7, 8	<i>l(3)87Bb</i> ⁻ - <i>Men</i> ⁻
Df(3R)kar5F^δ	86E5;87E1-2	1	<i>Hsp70</i> ⁻ - <i>kar</i> ⁻

^α 1 = Ashburner; 2 = Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowitz, 1981, Genetics 98: 775-89; 3 = Hall and Kankel,

1976, Genetics 83: 517-35; 4 = Ish-Horowitz, Holden, and Gehring, 1977, Cell 12: 643-52; 5 = Ish-Horowitz, Pinchin, Gausz, Gyurkovics, Bencze, Goldschmidt-Clermont, and Holden, 1979, Cell 17: 565-71; 6 = Ish-Horowitz, Pinchin, Shedd, Artavanis-Tsakonas, and Mirault, 1979, Cell 18: 1351-58; 7 = Ohnishi and Voelker, 1981, Biochem. Genet. 19: 75-85; 8 = Voelker, Ohnishi, Langley, Gausz, and Gyurkovics, 1981, Biochem. Genet. 19: 525-34.

- β All or almost all homozygous embryos fail to hatch; embryos (or nonfeeding larvae) have transparent Malpighian tubules.
- γ Lacks 87C1 heat shock puff site; complements *Df(3R)ry*.
- δ Lacks 87A7 and 87C1 heat shock puff sites.

Df(3R)kar-D

origin: X ray induced in *In(3R)AFA = In(3R)86C;93D6-7* chromosome.

deficiency	cytology	ref ^α	genetics
<i>Df(3R)kar-D1</i> ^{β,γ}	87A7-8;87D1-2	1, 2, 3, 4, 5, 6, 7	<i>Dip - C⁻ - kar⁻</i>
<i>Df(3R)kar-D2</i> ^γ	87A6-7;87D4-5	1, 2, 5, 6, 7	<i>Dip - C⁻ - Men⁻</i>
<i>Df(3R)kar-D3</i> ^δ	86E16-18;87D3-4	2, 3, 4, 6, 7	<i>l(3)87Ac⁻ - Men⁻</i>
<i>Df(3R)kar-D4</i>	87B11-C2;87E12-F1	2,6	<i>kar⁻</i>

- α 1 = Caggese, Caizzi, Morea, Scalenghe, and Ritossa, 1979, Proc. Nat. Acad. Sci. USA 76: 2385-89; 2 = Costa, Ritossa, and Scalenghe, 1977, DIS 52: 140; 3 = Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowitz, 1981, Genetics 98: 775-89; 4 = Ish-Horowitz, Pinchin, Gausz, Gyurkovics, Bencze, Goldschmidt-Clermont, and Holden, 1979, Cell 17: 565-71; 5 = Ohnishi and Voelker, 1981, Biochem. Genet. 19: 75-85; 6 = Scalenghe and Ritossa, 1976, Atti Acad. Naz. Lincei 13: 439-528; 7 = Voelker, Ohnishi, Langley, Gausz, and Gyurkovics, 1981, Biochem. Genet. 19: 525-34.
- β Two deficiencies [*Df(3R)kar-D1* and *Df(3R)kar-D2*] thought to be identical and listed separately through stock error according to Caggese *et al.*, 1979.
- γ Partial deletion of *Hsp70* site at 87A producing 40 kb *Hsp70* (Ish-Horowitz and Pinchin, 1980, J. Mol. Biol. 142: 231-45). Lacks *Hsp70* site at 87C1.
- δ Lacks 87A7 and 87C1 heat shock puff sites. Uncovers *sad* (Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95).

Df(3R)kar-H

origin: X ray induced.

deficiency	cytology	ref ^α	genetics
<i>Df(3R)kar-H1</i> ^β	87A10;87D11	1, 3	<i>Hsp70⁻ - kar⁻</i>
<i>Df(3R)kar-H2</i> ^β	87B3;87D1-4	1	<i>Hsp70⁻ - kar⁻</i>
<i>Df(3R)kar-H3</i>	87C2-3;87D3-4	1	<i>kar⁻</i>
<i>Df(3R)kar-H5</i> ^β	87A1-2;87D5-7	1, 3, 4	<i>l(3)87Aa⁻ - kar⁻</i>
<i>Df(3R)kar-H6</i>	87B6;87C8	2	<i>Dip - C⁻ - kar⁻</i>
<i>Df(3R)kar-H9</i> ^γ	86F4;87F11	1	<i>Hsp70⁻ - kar⁻</i>
<i>Df(3R)kar-H10</i> ^β	87A1-2;87D6-7	1, 2, 3, 4	<i>svp⁻ - kar⁻</i>
<i>Df(3R)kar-H11</i> ^β	87B10;87F4	1, 3	<i>Hsp70⁻ - kar⁻</i>
<i>Df(3R)kar-H12</i> ^β	87C7;87F1-2	1	<i>kar⁻</i>
<i>Df(3R)kar-H13</i> ^β	87B1-2;87D14-E1	1, 2, 4	<i>Dip - C⁻ - kar⁻</i>
<i>Df(3R)kar-H14</i> ^γ	86F10-11;87D1-2	1, 2	<i>Dip - C⁻ - kar⁻</i>

- α 1 = Henikoff, 1979, Genetics 93: 105-15; 2 = Ohnishi and Voelker, 1981, Biochem. Genet. 19: 75-85; 3 = Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowitz, 1981, Genetics 98: 775-89; 4 = Gausz, Hall, Spierer, and Spierer, 1986, Genetics 112: 65-78.
- β Lacks 87C1 heat shock puff site.
- γ Lacks 87A7 and 87C1 heat shock puff sites.

Df(3R)kar-IG27

cytology: *Df(3R)87B5;87D6* (Hilliker *et al.*, 1980).

discoverer: Gelbart.

- references: Hall and Kankel, 1976, Genetics 83: 517-35. Hilliker, Clark, Chovnick, and Gelbart, 1980, Genetics 95: 95-110. Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72. Spierer, Spierer, Bender, and Hogness, 1983, J. Mol.

Biol. 168: 35-50.

genetics: Deficient for *kar-87De*; includes *Men*. Lacks heat shock puff site at 87C1.

molecular biology: 87D breakpoint between 184 and 191 kb to the left of the left breakpoint of *In(3R)Cbx^{rv1}* (Spierer *et al.*, 1983; Hogness, 1983, J. Mol. Biol. 168: 17-33).

Df(3R)kar-Sz

origin: X ray induced.

deficiency	cytology	ref ^α	genetics
<i>Df(3R)kar-Sz5</i> ^β	86E20-F1;87F3-4	1	<i>Hsp70⁻ - kar⁻</i>
<i>Df(3R)kar-Sz8</i>	87C1-2;87D14-E1	1, 5, 6	<i>kar⁻</i>
<i>Df(3R)kar-Sz11</i> ^γ	87C7-8;87E5-6	1, 2, 4, 7, 8	<i>kar⁻ - l(3)87Ee⁻</i> ; includes <i>Men</i>
<i>Df(3R)kar-Sz12</i> ^δ	87B1-3;87C8-9	1, 2, 3	<i>l(3)87Ca⁻ - kar⁻</i>
<i>Df(3R)kar-Sz13</i> ^β	86E6-7;87C8-D1	1	<i>Hsp70⁻ - kar⁻</i>
<i>Df(3R)kar-Sz15</i> ^δ	87B1-2;87E1-2	1, 3	<i>Hsp70⁻ - kar⁻</i>
<i>Df(3R)kar-Sz16</i> ^ε	87C2;87C9-D1	1, 5, 6, 8	<i>kar⁻ - Men⁻</i>
<i>Df(3R)kar-Sz21</i>	87C7;87C8-9	1, 2	<i>l(3)87Cc⁻ - kar⁻</i>
<i>Df(3R)kar-Sz23</i> ^δ	86E6-7;87C9-D1	1	<i>Hsp70⁻ - kar⁻</i>
<i>Df(3R)kar-Sz27</i>	87C7;87F1	1, 2	<i>kar⁻ - ry⁻</i>
<i>Df(3R)kar-Sz28</i>	87C7-8;87E9-10	1	<i>kar⁻</i>
<i>Df(3R)kar-Sz29</i>	87C3-4;87C9-D1	1, 2	<i>l(3)87Ca⁻ - l(3)87Cd⁻</i>
<i>Df(3R)kar-Sz30</i> ^δ	87B2-4;87D2-3	1, 8	<i>Hsp70⁻ - Men⁻</i>
<i>Df(3R)kar-Sz31</i> ^β	86C6-7;87C9-D1	1, 2	<i>l(3)87Cc⁻ - kar⁻</i>
<i>Df(3R)kar-Sz33</i>	87C1-2;87E4-5	1	<i>kar⁻</i>
<i>Df(3R)kar-Sz37</i>	87C5-6;87D14-E1	1, 2	<i>l(3)87Cb⁻ - pic⁻</i>
<i>Df(3R)kar-Sz40</i> ^δ	87B2-3;87D1-3	1	<i>Hsp70⁻ - kar⁻</i>
<i>Df(3R)kar-Sz72</i> ^ζ	87E1-3;87F13-14	1	<i>kar⁻</i>

- α 1 = Gausz, Awad, and Gyurkovics, 1980, DIS 55: 45-46; 2 = Gausz, Bencze, Gyurkovics, Ashburner, Ish-Horowitz, and Holden, 1979, Genetics 93: 917-34; 3 = Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowitz, 1981, Genetics 98: 775-89; 4 = Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72; 5 = Ish-Horowitz and Pinchin, 1980, J. Mol. Biol. 142: 231-45; 6 = Ish-Horowitz, Pinchin, Gausz, Gyurkovics, Bencze, Goldschmidt-Clermont, and Holden, 1979, Cell 17: 565-71; 7 = Spierer, Spierer, Bender, and Hogness, 1983, J. Mol. Biol. 168: 35-50; 8 = Voelker, Ohnishi, Langley, Gausz, and Gyurkovics, 1981, Biochem. Genet. 19: 525-34.
- β Lacks 87A7 and 87C1 heat shock puff sites.
- γ Molecular coordinates of distal breakpoint between +82 and +93 (Spierer *et al.*, 1983).
- δ Lacks 87C1 heat shock puff site.
- ε Broken within *Hsp70* at 87C1; heat shock protein small (Ish-Horowitz and Pinchin, 1980). Survives 20 minutes of heat shock at 40.5° if given pretreatment for 30 minutes at 33-35° [Mitchell, Moller, Peterson, and Lipps-Sarmiento, 1979, Dev. Genet. 1: 181-92].
- ζ Distal to *kar* (at 87C8 according to Gausz *et al.*, 1981).

Df(3R)Kx1

cytology: *Df(3R)86C1;87B5*.

discoverer: Vässin.

- references: Reuter, Gausz, Gyurkovics, Friede, Bang, Spierer, Hall, and Spierer, 1987, Mol. Gen. Genet. 210: 429-36.

genetics: Dominant suppressor of *w^{m4h}* variegation.

Df(3R)L16

cytology: *Df(3R)96A1-10;96E*.

origin: X ray induced.

- references: Gonzales, Molina, Casal, and Ripoll, 1989, Genetics 123: 371-77.

genetics: Practically lethal over *asp* in late larvae, with mitotic abnormalities of the brain identical to those of *Df(3R)Su9/asp* larvae; *Df(3R)Su9/TM6B* larval brains normal. Escapers [*Df(3R)L16/asp*] are Minute, showing cuticular defects and male and female sterility.

Df(3R)I26c

cytology: *Df(3R)87D14-E1;87F11-12* (Gelbart, Lefevre).
discoverer: Chovnick.
discoverer: Gelbart.
references: Hall and Kankel, 1976, *Genetics* 83: 517-35.
 Hilliker, Clark, Chovnick, and Gelbart, 1980, *Genetics* 95: 95-110.
 Hilliker, Chovnick, and Clark, 1981, *DIS* 56: 64-72.
 Spierer, Spierer, Bender, and Hogness, 1983, *J. Mol. Biol.* 68: 35-50.
 Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95.
genetics: Deficient for *l(3)87Eb* to region distal to *l(3)87Ek*. Uncovers *yrt* (Jürgens *et al.*, 1984).
molecular biology: Molecular coordinates of proximal breakpoint between -129 and -115 (Spierer *et al.*, 1983).

Df(3R)I26d

cytology: *Df(3R)87D11-13;87E3-5* (Gelbart, Hilliker, Lefevre).
discoverer: Chovnick.
references: Hall and Kankel, 1976, *Genetics* 83: 517-35.
 Hilliker, Clark, Chovnick, and Gelbart, 1980, *Genetics* 95: 95-110.
 Hilliker, Chovnick, and Clark, 1981, *DIS* 56: 64-72.
 Spierer, Spierer, Bender and Hogness, 1983, *J. Mol. Biol.* 68: 35-50.
 Nagoshi and Gelbart, 1987, *Genetics* 117: 487-502.
genetics: Deficient for *pic-l(3)87Ef* (includes *Ace*).
molecular biology: Molecular coordinates of distal breakpoint between +79.5 and +90.5, of proximal breakpoint between -160.5 and -157.5 (Spierer *et al.*, 1983).

Df(3R)IC4a

cytology: *Df(3R)87E5-7;87E11-F1* (Gelbart).
discoverer: Chovnick.
references: Hall and Kankel, 1976, *Genetics* 83: 517-35.
 Hilliker, Clark, Chovnick, and Gelbart, 1980, *Genetics* 95: 95-110.
 Hilliker, Chovnick, and Clark, 1981, *DIS* 56: 64-72.
 Spierer, Spierer, Bender, and Hogness, 1983, *J. Mol. Biol.* 68: 35-50.
genetics: Deficient for *l(3)87Eg-l(3)87Ej*.
molecular biology: Molecular coordinates of proximal breakpoint between +73 and +85 (Spierer *et al.*, 1983).

Df(3R)LIN

cytology: *Df(3R)84A4-5;84B1-2*.
origin: Induced by ethyl methanesulfonate.
references: Frohnhofer and Nüsslein-Volhard, 1986, *Nature (London)* 324: 120-25.
 Pultz, Diederich, Cribbs, and Kaufman, 1988, *Genes Dev.* 2: 901-20.
genetics: Deficient for *bcd*.

Df(3R)M1: Deficiency (3R) of Morata

discoverer: Casanova and Morata.
references: Sánchez-Herrero, Vernós, Marco, and Morata, 1985, *Nature* 313: 108-13.
 Casanova, Sánchez-Herrero, and Morata, 1986, *Cell* 47: 627-36.
genetics: Deficient for the entire *BXC* and *l(3)89Ea*, but not *l(3)89Ee*.

Df(3R)M86D: Deficiency (3R) Minute

cytology: *Df(3R)86D1-2;86D4* (Ashburner *et al.*, 1980).
origin: X ray induced.
discoverer: Schultz, 33a10.
synonym: *Df(3R)M-S31*.
references: 1940, *DIS* 13: 51.
 Ashburner, Richards, and Velissariou, 1980, *DIS* 55: 196.
genetics: Deficient for *Odh*, *M(3)86D*, *cu*, *neu* (Clark, 1983, *Biochem. Genet.* 21: 375-90).

Df(3R)M-S31: see *Df(3R)M86D*

Df(3R)M95A

cytology: *Df(3R)94D;95A3*.
origin: X ray induced.
synonym: *Df(3R)MSu244*.
references: Reuter, Dorn, Wustmann, Friede, and Rauh, 1986, *Mol. Gen. Genet.* 202: 481-87.
genetics: Deficient for *M(3)95A* and *Su(var)3-11*.

Df(3R)M96A

cytology: *Df(3R)95E6-8;96A1-5*.
origin: Recombination between elements of *T(Y;3)HI73 = T(Y;3)95E6-8* and *T(Y;3)G73 = T(Y;3)96A1-5*.
references: Gonzáles, Molina, Casal, and Ripoll, 1989, *Genetics* 123: 371-77.
genetics: Deficient for *M(3)96A*. Haplo-insufficient. Heterozygotes are Minute showing full penetrance and expressivity for the thin bristle phenotype.

Df(3R)MAP

origin: X ray induced.

deficiency	cytology	ref ^α	genetics
<i>Df(3R)MAP2</i> ^β	84A1-2;84A4-5	2	<i>lab⁻pb⁻zen⁺</i>
<i>Df(3R)MAP8</i> ^{γδ}	84A4-5;84A4-5	1-3	<i>lab⁺pb⁻zen⁺</i>
<i>Df(3R)MAP11</i>		1-3	<i>zen⁻bcd⁻ama⁻Dfd⁻</i>
<i>Df(3R)MAP13</i> ^δ	84A4-5;84A4-5		<i>lab⁺pb⁻zen⁺</i>
<i>Df(3R)MAP117</i>		3	<i>zen⁻-Antp⁻</i> (and beyond)

^α 1 = Diederich, Merrill, Pultz, and Kaufman, 1989, *Genes Dev.* 3: 399-414; 2 = Pultz, Diederich, Cribbs, and Kaufman, 1988, *Genes Dev.* 2: 901-20; 3 = Seeger, Haffley, and Kaufman, 1988, *Cell* 55: 598-600.

^β Distal breakpoint at -54 kb on molecular map of the *ANTC* complex (Pultz *et al.*, 1988).

^γ Distal breakpoint at -50kb and proximal breakpoint at -110 kb on molecular map of the *ANTC* complex (Pultz *et al.*, 1988).

^δ Synonyms: *Df(3R)pb-map8* and *Df(3R)pb-map13*.

Df(3R)Mg28: Deficiency (3R) Mglinetz

cytology: *Df(3R)87D;87F*.
origin: γ ray induced.
references: Mglinetz, 1972, *Genetika (Moscow)* 8(2): 82-92.

Df(3R)Mg32

cytology: *Df(3R)98F;100C*.
origin: γ ray induced.
references: Mglinetz, 1972, *Genetika (Moscow)* 8(2): 82-92.

Df(3R)ML457

cytology: *Df(3R)87A;87C*.
origin: X ray induced.
references: Mukhina and Zhimulev, 1980, *DIS* 55: 209.

Df(3R)MSu2: Deficiency (3R) Minute Suppressor

cytology: *Df(3R)100F3-5*.

origin: X ray induced.

references: Reuter, Dorn, Wustmann, Friede, and Rauh, 1986, Mol. Gen. Genet. 202: 481-87.

genetics: Exhibits a Minute phenotype and dominant suppression of variegated-type position effect.

other information: The genetic map position and the inferred moderate phenotype are at odds with the location of the deficiency at 100F3-5.

Df(3R)MSu244: see *Df(3R)M95A*

Df(3R)N

origin: X ray induced.

references: Gausz, Hall, Spierer, and Spierer, 1986, Genetics 112: 65-78.

Nagoshi and Gelbart, 1987, Genetics 117: 487-502.

deficiency	cytology	genetics
<i>Df(3R)N40</i>	87E1-2	<i>l(3)87Ec</i> ⁻
<i>Df(3R)N42</i>	87E4	<i>Ace</i> ⁻
<i>Df(3R)N63</i>	87E5-6	<i>l(3)87Ee</i> ⁻
<i>Df(3R)N69</i>	87D14-E1	<i>sim</i> ⁻
<i>Df(3R)N74</i>	87D13-14	<i>pic</i> ⁻
<i>Df(3R)N78</i>	87E4	<i>Ace</i> ⁻

Df(3R)Na: see *In(3R)Na*

Df(3R)o: Deficiency (3R) opsin

origin: γ ray induced.

discoverer: O'Tousa.

deficiency	cytology	ref ^α	genetics
<i>Df(3R)o11</i>	<i>Df(3R)92B5-6;92B7-8</i>	1	<i>ninaE</i> ⁻
<i>Df(3R)oB16</i>	<i>Df(3R)92A12-B1;92E7-15</i>	2	<i>ninaE</i> ⁻ <i>ort</i> ⁺
<i>Df(3R)oF4</i>	<i>Df(3R)92A1-3;92D5-9</i>	2	<i>ninaE</i> ⁻ <i>ort</i> ⁻

^α 1 = O'Tousa, Baehr, Martin, Hirsh, Pak, and Applebury, 1985, Cell 40: 839-50; 2 = O'Tousa, Leonard, and Pak.

Df(3R)O5: see *Tp(3;1)O5*

Df(3R)p: Deficiency (3R) pink

genetics: Deficient for *p*.

deficiency	cytology	origin	discov. or ref ^α	molec. biol. ^β
<i>Df(3R)p4</i>	84D6-10;85A1-2 + <i>T(2;3)34D;85A1-2</i>	X ray	4, 5	
<i>Df(3R)p5</i>		MR	1, 2	+24 - +29
<i>Df(3R)p7</i>	{85A;85A}		1, 2	
<i>Df(3R)p13</i> ^{γ δ}	84F2;85B1	X ray	1, 4, 5, 8	
<i>Df(3R)p16</i>	{85A;85A}	MR	1, 2	+24 - +45
<i>Df(3R)p17</i>	{85A;85A}		1, 2	
<i>Df(3R)p19</i>	{85A;85A}	MR	1, 2	+24 - +33
<i>Df(3R)p21</i>	84F4-6;85C4-6	X ray	4, 5	
<i>Df(3R)p25</i> ^δ	85A5-7;85A11	X ray	1, 4, 5	+60
<i>Df(3R)p30</i> ^δ	84F8-10;85D3-5 (B.S. Baker)	X ray	4, 5	
<i>Df(3R)p40</i> ^δ	84E8-9;85B6 + <i>In(3LR)64;90 + T(2;3)55;75</i>	X ray	4, 5	
<i>Df(3R)p46</i>	84D4-6;85D6	X ray	4, 5	
<i>Df(3R)p66</i> ^ε	84D4-6;85B6-C1 + <i>T(2;3)25D;84D4-6 + T(2;3)25D;85B6-C1</i>			
<i>Df(3R)p118</i> ^ε	84F;85A			
<i>Df(3R)p712</i>	84D4-6;85B6 + <i>T(2;3)25D;85B</i>	X ray	4	
<i>Df(3R)p819</i>	85A3;85B6 +	X ray	4	

deficiency	cytology	origin	discov. or ref ^α	molec. biol. ^β
	<i>T(2;3)41;87C + Tp(3;3)64F;67F;97</i>			
<i>Df(3R)p-bs12</i>	85A;?			
<i>Df(3R)p-bs13</i>	85A;?			
<i>Df(3R)p-XM66</i> ^ε	85A3;85B6		3	
<i>Df(3R)p-XT6</i>	85A3;85C1-2			
<i>Df(3R)p-XT9</i> ^ε	84F14;85C-D	X ray	7	
<i>Df(3R)p-XT15</i>	{85A;85A}			
<i>Df(3R)p-XT26</i>	85A3;85C1-2	X ray	6, 7	
<i>Df(3R)p-XT27</i>	{85A;85A}	X ray	7	
<i>Df(3R)p-XT103</i>	85A2;85C1-2	X ray	6, 7	
<i>Df(3R)p-XT104</i>	{85A;85A}			
<i>Df(3R)p-XT118</i> ^δ	84F1;85B	X ray	3, 7	

^α 1 = Bender, Turner, and Kaufman, 1987, Dev. Biol. 119: 418-32; 2 = M.M. Green; 3 = Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95; 4 = Kempthues, Raff, Raff, and Kaufman, 1980, Cell 21: 445-51; 5 = Kempthues, Raff, and Kaufman, 1983, Genetics 105: 345-56; 6 = Lehmann and Nüsslein-Volhard, 1986, Cell 47: 141-52; 7 = Lehmann and Nüsslein-Volhard, 1987, Dev. Biol. 119: 402-07; 8 = Tautz, Lehmann, Schnürch, Schuh, Seifert, Kienlin, Jones and Jäckle, 1987, Nature (London) 327: 383-89.

^β DNA coordinates from 85A map (Jones and Rawls, 1988, Genetics 120: 733-42) ("+" values to right, "-" values to left or centromere end of map). Proximal breakpoints of deficiencies 5, 16, and 19; distal breakpoint of deficiency 25.

^γ Distal breakpoint of *Df(3R)p13* molecularly mapped to the DNA (Tautz *et al.*, 1987).

^δ Includes *Dhod* (Porter and Rawls, 1984, Mol. Gen. Genet. 193: 27-32).

^ε Uncovers *hb*.

^ζ Deficient for *pum* (Lehman and Nüsslein-Volhard, 1987, Nature 329: 167-70).

Df(3R)P2: Deficiency (3R) Pasadena

cytology: *Df(3R)89D9-E1;89E2-3*.

origin: X ray induced.

discoverer: E.B. Lewis.

references: 1980, DIS 55: 207-08.

genetics: Deficient for *abx-iab*³. Genetically similar to *Df(3R)Ubx109*.

molecular biology: Right breakpoint 75-81 kb to the right of the right breakpoint of *In(3R)Cbx^{rv1}* (Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96).

Df(3R)P9

cytology: *Df(3R)89D9-E1;89E4-5*.

origin: X ray induced.

discoverer: E.B. Lewis.

references: 1978, Nature 276: 565-70.

1980, DIS 55: 207-08. Jiménez and Campos-Ortega, 1981, Wilhelm Roux's Arch. Dev. Biol. 190: 370-73.

Morata, 1982, Am. Zool. 22: 57-64.

Levine, Hafen, Garber, and Gehring, 1983, EMBO J. 2: 2037-46.

Hafen, Levine, and Gehring, 1984, Nature 307: 287-89.

genetics: Deficient for entire *BXC*; uncovers *tuh-3* (Kuhn, Woods, and Andrew, 1984, DIS 60: 134-35). Homozygotes die in late embryonic or early larval stages, showing transformation of metathorax and anterior first abdominal segment to mesothorax (Lewis, 1978; Hayes, Sato, and Denell, 1984, Proc. Nat. Acad. Sci. USA 81: 545-49) and transformation of posterior meso- and metathorax to prothorax (Hayes *et al.*, 1984; Ganger, Fehon, and Schubiger, 1985, Nature 313: 395-97).

When incubated at 18°, homozygous *Df(3R)P9* embryos do not complete germ band shortening but *Df(3R)P9/Dp(3;3)P5* heterozygotes go through normal development (Ganger and Schubiger, 1984, DIS 60: 108-09). *Df(3R)P9/+* flies show reduced male pigmentation on AB5 and AB6, are sterile, and have deformed genitalia; *Df(3R)P9/Mcp* flies are partially fertile. *Df(3R)P9/Df(3R)Ubx109* larvae have a short tracheal trunk between AB7 and AB8 and a posterior spiracle in AB8 (Lewis, 1978).

molecular biology: Right breakpoint 225-230 kb to the right of the right breakpoint of *In(3R)Cbx^{rvl}* (Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96).

Df(3R)P10: see *Tp(3;2)P10*

Df(3R)P-10 - Df(3R)P-79

origin: X ray induced loss of *TE87A*.

references: Reuter, Gausz, Gyurkovics, Friede, Bang, Spierer, Hall, and Spierer, 1987, Mol. Gen. Genet. 210: 429-36.

genetics: Suppress *w^{m4h}* variegation.

deficiency	cytology
<i>Df(3R)P-10</i>	86E6-9;87A9-B1
<i>Df(3R)P-21</i>	86E19-F1;87B11-15
<i>Df(3R)P-29</i>	87A4-5;87B4-5
<i>Df(3R)P-35</i>	86F3-4;87B5-8
<i>Df(3R)P-79</i>	86E1-2;87B1-2

Df(3R)P13

cytology: *Df(3R)89C-D;89E*.

origin: X ray induced.

discoverer: Ramey.

references: Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.

molecular biology: Right breakpoint 78-81 kb to the right of the right breakpoint of *In(3R)Cbx^{rvl}*.

Df(3R)P14

cytology: *Df(3R)90C2-D1;91A2-3*.

origin: X ray induced.

discoverer: E. B. Lewis.

references: Grell, 1976, Genetics 83: s28-29.

Detwiler and MacIntyre, 1978, Biochem. Genet. 16: 1113-32.

genetics: Deficient for *DNase-1*, *sr*, and *gl* but not *k* or *DI*.

Df(3R)P47: see *Tp(3;3)P47*

Df(3R)P88: see *Df(3R)ss*

Df(3R)P115: see *Tp(3;1)P115*

Df(3R)Pc-T7: Deficiency (3R) Polycomb

origin: γ ray induced.

discoverer: Tiong.

references: Kennison and Russell, 1987, Genetics 116: 75-86.

genetics: Deficient for *Pc*.

Df(3R)Po: Deficiency (3R) Pyridoxal oxidase

references: Hughes, Nelson, Yanuk, and Szauter.

genetics: Deficient for *Po* (Hughes *et al.*) and *mor* (Kennison).

deficiency cytology

Df(3R)Po3 89A1-2;89A11-13
Df(3R)Po4 88F7-89A1;89A11-13

Df(3R)Pr: Deficiency (3R) Prickly

origin: X ray induced.

discoverer: Preiss.

references: Preiss, Hartley, and Artavanis-Tsakonas, 1988, EMBO J. 7: 3917-27.

deficiency	cytology	synonym
<i>Df(3R)Pr1</i>	96F11-14;97E2-3	<i>Pr^{rev1}</i>
<i>Df(3R)Pr4</i>	96F11-14?;97B	<i>Pr^{rev4}</i>
<i>Df(3R)Pr6</i>	96F11-14;98A	<i>Pr^{rev6}</i>
<i>Df(3R)Pr10</i>	96F11-14;97C1	<i>Pr^{rev10}</i>
<i>Df(3R)Pr-P9</i>	96F11-14;97D8-10	<i>Pr^{P9}</i>

Df(3R)r1-G6

cytology: *Df(3R)93A2-B1;93E-F*.

references: Vincent.

genetics: Deficient for the gene encoding (Na⁺K⁺) α ATPase, α *Atp*.

Df(3R)R29

references: Whetten, Organ, Krasney, Cox-Foster, and Cavener, 1988, Genetics 120: 475-84.

genetics: *Df(3R)A41/Df(3R)R29* flies lack GLD enzyme activity.

Df(3R)red: Deficiency (3R) red Malpighian tubules

genetics: *Df(3R)red/+* progeny of *Df(1)C128/+* females show a high frequency of homeotic transformations (Gans *et al.*, 1980).

deficiency	cytology	ref ^{α}	genetics
<i>Df(3R)red1</i>	88B1;88D3-4	2, 4	<i>trx⁻red⁻</i>
<i>Df(3R)red2l</i>	88A12-B1;88B2-3	1, 9	<i>trx⁻red⁻</i>
<i>Df(3R)red3l</i>	87F12-14;88C1-3	1, 2, 3, 8	<i>Dip-B⁻red⁻</i>
<i>Df(3R)red-P1</i>	88B1;88D3-4	2	<i>trx⁻red⁻</i>
<i>Df(3R)red-P6</i>	88B1;88D3-4	2	<i>trx⁻red⁻</i>
<i>Df(3R)red-P52</i>	88A12-B1;88B4-5	1, 5, 6, 7	<i>trx⁻su(Hw)⁻</i>
<i>Df(3R)red-P93</i>	88B-C (Lewis)	2, 5, 7	<i>trx⁻red⁻</i>

^{α} 1 = Capdevila and García-Bellido, 1981, Wilhelm Roux's Arch. Dev. Biol. 190: 339-50 2 = Gans, Forquignon, and Masson, 1980, Genetics 96: 887-902; 3 = Hall and Kankel, 1976, Genetics 83: 517-35; 4 = Ingham, 1980, DIS 55: 63-64; 5 = Ingham, 1981, Wilhelm Roux's Arch. Dev. Biol. 190: 339-50; 6 = Ingham and Whittle, 1980, Mol. Gen. Genet. 179: 607-14; 7 = Lewis, 1981, Developmental Biology Using Purified Genes (Brown and Fox, eds.), Academic Press, New York, pp. 189-208; 8 = Ohnishi and Voelker, 1981, Biochem. Genet. 19: 75-85; 9 = Spillman and Nöhiger, 1978, DIS 53: 163.

Df(3R)rn: Deficiency (3R) rotund

references: Agnel, Kerridge, Vola, and Griffin-Shea, 1989, Genes Dev. 3: 85-95.

genetics: Deficient for *rn*.

deficiency	cytology	origin	discoverer
<i>Df(3R)rn3</i>	84D3;84D4	X ray	Hannah-Alava
<i>Df(3R)rn5</i>	84D3;84D4	X ray	Williams
<i>Df(3R)rn17^{α}</i>	84B-C;84D9-12	DEB	Kerridge
<i>Df(3R)rn19</i>	84D3;84D4	DEB	Kerridge
<i>Df(3R)rn20^{α}</i>	84D3;84D4	DEB	Kerridge
<i>Df(3R)rn22^{α}</i>	84B-C;84D9-10	DEB	Kerridge

^{α} Deficiency includes *roe*.

Df(3R)ry: Deficiency (3R) rosy
origin: X ray induced.

deficiency	cytology	ref ^α	genetics	molecular coordinates of breakpoint ^β
Df(3R)ry1	87D;87E-F	1	kar ⁻ - ry ⁻	
Df(3R)ry27	87D1-2;87F1-2	2, 6, 7, 8	l(3)87Da ⁻ - l(3)87Ek ⁻	
*Df(3R)ry28		7, 8	ry ⁻	
*Df(3R)ry29		7, 8	ry ⁻	
*Df(3R)ry30		7, 8	ry ⁻	
*Df(3R)ry31		7, 8	ry ⁻	
*Df(3R)ry32		7, 8	ry ⁻	
*Df(3R)ry33		7, 8	ry ⁻	
*Df(3R)ry34		7, 8	ry ⁻	
Df(3R)ry36	87E;87E	4, 7, 8, 9	mesA ⁻ - ry ⁻ ; also snk ⁻	proximal -201 to -213;
*Df(3R)ry51		7, 8	ry ⁻	
Df(3R)ry52		7, 8	kar ⁻ - l(3)87Eb ⁻	
*Df(3R)ry66		7, 8	ry ⁻	
*Df(3R)ry70		7, 8	ry ⁻	
Df(3R)ry74	87D8;87D12	6	ry ⁻	
Df(3R)ry75	87D1-2;87D14-E1	4, 6, 9	l(3)87Da ⁻ - pic ⁻	distal -125 to -139
Df(3R)ry76	87D13-E1;88B12-14	6	ry ⁻	
*Df(3R)ry77^γ			l(3)87Da ⁻ - pic ⁻	
*Df(3R)ry78^γ			mes ⁻ - Ace ⁻	
Df(3R)ry81	87C1-3;87D14-E2	2,3,4,9,10	l(3)87Ca ⁻ - pic ⁻ ; includes Men	distal -100 to -112
Df(3R)ry85	87B15-C1;87F15-88A1	2, 10	l(3)87Ca ⁻ - l(3)87EK ⁻ ; includes Men	
Df(3R)ry614	87D2-4;87D11-14	2,3,4,9	l(3)87Ca ⁻ - pic ⁻	distal -125 to -139
Df(3R)ry615^δ	87B12-15;87E8-11	2, 10	l(3)87Ca ⁻ - l(3)87Ej ⁻ ; includes Men	
Df(3R)ry619	87D7-9;87E12-F1	2, 3, 4, 9	mesA ⁻ - l(3)87Ek ⁻	proximal -201 to -213
Df(3R)ry1168	87B15-C1;87E9-12	3, 4	l(3)87Ca ⁻ - l(3)87Ej ⁻	
Df(3R)ry1301	87D2-4;87E1-2	3, 4, 9	l(3)87Da ⁻ - l(3)87Ec ⁻	distal +15 to +21.5 ^e
Df(3R)ry1402	87D2-4;87D14-E2	2, 3, 4, 9	l(3)87Da ⁻ - l(3)87Eb ⁻	distal -39 to -60
Df(3R)ry1607	87D3-4;87E2-3	3, 4, 9	l(3)87Da ⁻ - Ace ⁻	distal +26 to +32 ^e
Df(3R)ry1608	87D4-6;87E1-2	3, 4, 9	l(3)87Dd ⁻ - l(3)87Eb ⁻	distal -17 to -41
Df(3R)ry-K	87B;87E (Richmond)	5, 8	l(3)S1 Ace ⁻	

^α 1 = Grell, E.H., 1962, Z. Indukt. Abstamm. Vererbungsl. 93: 371-77; 2 = Hall and Kankel, 1976, Genetics 83: 517-35; 3 = Hilliker, Chovnick, and Clark, 1981, DIS 56: 64-72; 4 = Hilliker, Clark, Chovnick, and Gelbart, 1980, Genetics 95: 95-110; 5 = Kernaghan, 1964, DIS 39: 62-64; 6 = Lefevre, 1971, DIS 46: 40; 7 = Schalet, 1964, DIS 39: 62-64; 8 = Schalet, Kernaghan, and Chovnick, 1964, Genetics 50: 1261-68; 9 = Spierer, Spierer, Bender, and Hogness, 1983, J. Mol. Biol. 168: 35-50; 10 = Voelker, Ohnishi, Langley, Gausz, and Gyurkovics, 1981, Biochem. Genet. 19: 525-34.

^β Coordinates of either proximal or distal breakpoints given with respect to arbitrarily-chosen 0 (Spierer *et al.*, 1983), "+" values to right, "-" values to left.

^γ Described in CP627.

^δ Also see García-Bellido, Moscoso del Prado, and Botas, 1983, Mol. Gen. Genet. 192: 253-63.

^e Gausz, Hall, Spierer, and Spierer, 1986, Genetics 112: 65-78; Hall and Spierer, 1986, EMBO J. 5: 2949-54.

Df(3R)ro: Deficiency (3R) rough

deficiency	cytology	discoverer	ref ^α	genetics
Df(3R)ro80b	97D1;97D15	Peter Lewis	1,2	Tl ⁻ , ro ⁻
Df(3R)ro82b	96F11-14;97F3-11		3	ro ⁻
Df(3R)ro-XB3	97D1-2;97D9	Peter Lewis	1,4	Tl ⁻ , ro ⁻ ; 40% viable over Tl ^{rv}
Df(3R)ro-z1	97D1-2;97D15	Peter Lewis	4	ro ⁻

^α 1 = Anderson, Jürgens, and Nüsslein-Volhard, 1985, Cell 42: 779-89; 2 = Anderson, Bokla, and Nüsslein-Volhard, 1985, Cell 42: 791-98; 3 = Preiss, Hartley, and Artavanis-Tsakonas, 1988, EMBO J. 7: 3917-27; 4 = Tomlinson, Kimmel, and Rubin, 1988, Cell 55: 771-84.

Df(3R)roe: Deficiency (3R) roughened eye

cytology: 84A6-B1;84D4-9.

discoverer: Jürgens.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.

genetics: Deficient for *Scr*, *ftz*, and *Antp*.

Df(3R)ry: Deficiency (3R) rosy

origin: X ray induced. (Other information in table at top of page).

Df(3R)ry⁺: see **Tp(3;2)ry⁺**

Df(3R)S462: Deficiency (3R) of Shaw

cytology: Df(3R)89D1-2;90D1.

origin: Aneuploid recombinant from *Tp(3;3)S462/+*.

discoverer: Shaw, 1973.

references: Lewis, 1980, DIS 55: 207-08.

genetics: *Df(3R)S462/+* viable but ecloses one or two days later than wild type.

Df(3R)sbd: Deficiency (3R) stubbloid

origin: X ray induced except for *Df(3R)sbd104*, which is an aneuploid recombinant from *Tp(3;3)sbd¹⁰⁴/+*.

deficiency	cytology	ref ^α	genetics
Df(3R)sbd14	89B5;89C		
Df(3R)sbd15	88F9-A1;89B9-10		
Df(3R)sbd26^β	89B9-10;89C7-D1	1, 4	Aldox-1 ⁺ sbd ⁻
Df(3R)sbd45^γ	89B4;89B10	1, 4	Aldox-1 ⁺ sbd ⁻
Df(3R)sbd104	89B5;89C; see <i>Tp(3;3)sbd¹⁰⁴</i>	3, 5	sbd ⁻ - ss ⁻
Df(3R)sbd105^δ	88F9-89A1;89B9-10	2, 4	cv-c ⁺ Aldox-1 ⁻ - sbd ⁻ ss ⁺

^α 1 = Grell, 1984, Genetics 108: 425-43; 2 = Lewis, 1948, DIS 22: 72-73; 3 = Lewis, 1980, DIS 55: 207-208; 4 = Spillman and Nöthiger, 1978, DIS 53: 124; 164; 5 = Struhl, 1984, Nature 308: 454-57.

^β Heterozygotes with *In(3R)sbd¹⁷* viable and *sbd*; heterozygotes with *In(3R)sbd¹²*, *sbd¹³*, *sbd¹⁸*, and *In(3R)sbd²¹* lethal with adult escapers that are *sbd*.

^γ Heterozygotes with *In(3R)sbd¹⁷* viable and *sbd*; heterozygotes with *sbd³²* lethal with adult escapers that are *sbd*. Uncovers *pnr*

(Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95).

♂ Crossing over in *X* reduced in *Df(3R)sbd105/+* females (Hinton, 1966, Genetics 55: 157-64); no effect on spontaneous male recombination (Lutkin and Baker, 1979, Mutat. Res. 61: 221-27). Uncovers *spt* and *pnr* (Jürgens *et al.*, 1984).

Df(3R)SCB-XL2

cytology: *Df(3R)84A4-5;84B1-2*.

origin: X ray induced.

discoverer: Jürgens.

references: Pultz, Diederich, Cribbs, and Kaufman, 1988, Genes Dev. 2: 901-20.

genetics: Variegating for *pb*. *Df(3R)SCB-XL2/pb⁻* flies show the maxillary and labial palp phenotypes of *pb* null flies.

Df(3R)Scr: Deficiency (3R) Sex combs reduced

cytology: *Df(3R)84A1-2;84B1-2*.

origin: γ ray induced.

discoverer: Sinclair, 1977.

references: Kaufman, Lewis, and Wakimoto, 1980, Genetics 94: 115-33.

Lewis, Kaufman, and Denell, 1980, DIS 55: 85-87.

Lewis, Kaufman, Denell, and Tallero, 1980, Genetics 95: 367-81.

Lewis, Wakimoto, Denell, and Kaufman, 1980, Genetics 95: 383-97.

Wakimoto, Lewis, and Kaufman, 1980, DIS 55: 140-41.

Wakimoto and Kaufman, 1981, Dev. Biol. 81: 51-64.

Garber, Kuroiwa, and Gehring, 1983, EMBO J. 2: 2027-36.

genetics: Deficient for *pb-Hu* plus four lethal complementation groups proximal to *pb*. Associated with dominant reduced-sex-comb phenotype. Fails to complement the recessive lethality of *Msc*, *Antp*, and *Antp^{Scx}*. Embryonic lethal with *Df(3R)Antp17*.

molecular biology: Deletes sequences to left of molecular coordinate +100, *In(3R)Hu* breakpoint being coordinate 0 in the breakpoint DNA walk of Garber *et al.*, 1983 ("+" values to the left, "-" values to the right).

Df(3R)Scx2: Deficiency (3R) Sexcombs extra

cytology: *Df(3R)84A4-5;84C1-2*. Break near C1, probably C2 or C3 (B. Baker).

origin: X ray induced.

synonym: *Df(3R)Scx^{W+RX2}*; *Df(3R)Antp-Scx*.

references: Hazelrigg and Kaufman, 1983, Genetics 105: 581-600.

Cavener, Corbett, Cox, and Whetten, 1986, EMBO J. 5: 2939-48.

Pultz, Diederich, Cribbs, and Kaufman, 1988, Genes Dev. 2: 901-20.

genetics: Deficient for *pb*, *ftz*, *Scx^W* and *Antp*. Homozygous lethal. *Df(3R)Scx2/Df(3R)MAP2* viable and *pb*.

molecular biology: Distal (84C1-2) breakpoint located at about +41 kb (Baker and Wolfner, 1988, Genes Dev. 2: 477-89); 0 point = *HindIII* site in *α Tub84B*, "+" values to the right, "-" values to the left).

Df(3R)Scx4

cytology: *Df(3R)84B3;84D1-2*.

origin: X ray induced.

synonym: *Df(3R)Scx^{W+RX4}*.

references: Hazelrigg and Kaufman, 1983, Genetics

105: 581-600.

Cavener, Otteson and Kaufman, 1986, Genetics 114: 111-23.

Agnel, Kerridge, Vola, and Griffin-Shea, 1989, Genes Dev. 3: 85-95.

genetics: Deficient for *Scx^W*, *Antp*, *rn*, and *roe* but not *ftz*. Homozygous lethal.

Df(3R)SMG39: Deficiency (3R) Semenova Mglinetz Glotoff

cytology: *Df(3R)90C;91D*.

origin: γ ray induced.

references: Semenova, Mglinetz, and Glotoff, 1970, Genetika (Moscow) 6: 165-69.

****Df(3R)sr: Deficiency (3R) stripe***

origin: X ray induced.

discoverer: Alexander.

references: Ward and Alexander, 1957, Genetics 42: 42-54.

genetics: Deficient for *sr*.

deficiency	cytology
* <i>Df(3R)sr100.394</i>	90C2-7;90F3-7
* <i>Df(3R)sr300.24</i>	90C2-4;91A2-5
* <i>Df(3R)sr300.101</i>	90D2-4;91A6-8

Df(3R)ss: Deficiency (3R) spineless

cytology: Deficient for bands in 89C-D; associated with *In(3LR)P88=In(3LR)61A;89C-D*.

origin: X ray induced.

discoverer: E.B. Lewis.

synonym: *Df(3R)P88*.

references: García-Bellido, Moscoso del Prado, and Botas, 1983, Mol. Gen. Genet. 192: 253-63.

genetics: Deficient for *ss*.

Df(3R)ss-a

cytology: *Df(3R)89B;89D* (Holmgren).

origin: Induced by ethyl methanesulfonate.

synonym: *ss^{aC15}*.

references: Struhl, 1982, Genetics 102: 737-49.

genetics: Deficient for *ss*. Lethal over *Df(3R)bxd100*.

Df(3R)su(Hw)7: Deficiency (3R) Suppressor of Hairy wing

cytology: *Df(3R)88A9;88B2*.

genetics: Deficient for *su(Hw)*⁷.

Df(3R)Su9-b: Deficiency (3R) Suppressor

cytology: *Df(3R)95A;97A*.

origin: Spontaneous.

references: Gonzales, Molina, Casal, and Ripoll, 1989, Genetics 123: 371-77.

genetics: Deficient for *M(3)96A-Pr*. Dominant lethal.

Df(3R)Su(var)3-6: Deficiency (3R) Suppressor of variegation

cytology: *Df(3R)87B1;87D11*.

origin: X ray induced.

references: Reuter, Dorn, Wustmann, Friede, and Rauh, 1986, Mol. Gen. Genet. 202: 481-87.

Reuter, Gausz, Gyurkovics, Friede, Bang, Spierer, Hall, and Spierer, 1987, Mol. Gen. Genet. 210: 429-36.

genetics: Deficient for *Su(var)3-6*. Recessive lethal.

Df(3R)SX1

cytology: *Df(3R)89E;89E*.

origin: X ray induced.

references: Tiong, Bone, and Whittle, 1985, *Mol. Gen. Genet.* 200: 335-42.

Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, *Cell* 43: 81-96.

genetics: Deficient for *iab2 - iab9*.

molecular biology: Proximal breakpoint 27.5-35 kb to the right of the right breakpoint of *In(3R)Cbx^{rv1}* (Karch *et al.*, 1985).

Df(3R)T

origin: Derived from transposing element *TE86F*.

deficiency	cytology	ref ^α	genetics
<i>Df(3R)T-7</i>	86F1-2;86F4-7	2	<i>l(3)86Fa⁻ - l(3)86Fb⁻</i>
<i>Df(3R)T-10^β</i>	86F1-2;87C6-7	1, 2	<i>l(3)87Ca⁻ - l(3)87Cb⁻</i>
<i>Df(3R)T-32^β</i>	86E2-4;87C6-7	1, 2	<i>l(3)87Ca⁻ - l(3)87Cb⁻</i>
<i>Df(3R)T-41</i>	86F1-2;87C1-2	2, 4	lacks 87A7 and 87C1 heat shock puff sites
<i>Df(3R)T-45^β</i>	86E;87B5-6	2, 5	<i>l(3)86Fa⁻ - l(2)86Fb⁻</i> ; lacks 87A7 heat shock puff site
<i>Df(3R)T-47</i>	86F1-2;87A9	2, 5	<i>l(3)86Fa⁻ - l(3)87Ac⁻</i> ; lacks 87A7 heat shock puff site; <i>mgr⁻</i>
<i>Df(3R)T-55^γ</i>	86F1-2;87A6-7	2, 3	<i>l(3)86Fa⁻ - l(3)87Ab⁻</i>
<i>Df(3R)T-61</i>	86F1-2;87A9	2	lacks 87A7 heat shock puff site; <i>mgr⁻</i>
<i>Df(3R)T-63</i>	86F1-2;87A5-7	2, 6	<i>sad⁻</i>

^α 1 = Gausz, Bencze, Gyurkovics, Ashburner, Ish-Horowicz, and Holden, 1979, *Genetics* 98: 917-34; 2 = Gausz, Gyurkovics, Bencze, Awad, Holden, and Ish-Horowicz, 1981, *Genetics* 98: 775-89; 3 = Goldberg, Paro, and Gehring, 1982, *EMBO J.* 1: 93-98; 4 = Ish-Horowicz and Pinchin, 1980, *J. Mol. Biol.* 142: 231-45; 5 = Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95; 6 = Ohnishi and Voelker, 1981, *Biochem. Genet.* 19: 75-85.

^β Suppresses *w^{mth}* variegation (Reuter, Gausz, Gyurkovics, Friede, Bang, Spierer, Hall, and Spierer, 1987, *Mol. Gen. Genet.* 210: 429-36).

^γ Isolated as pale apricot variant from stock carrying *TE86F* (Ish-Horowicz). Deletion breakpoint 9 kb to left of *Hsp70*.

Df(3R)TI: Deficiency (3R) Toll

references: Anderson, Jürgens, and Nüsslein-Volhard, 1985, *Cell* 42: 779-89.

genetics: Revertants of *Tl*. Heterozygous females viable, fertile.

deficiency	cytology	origin	synonym
<i>Df(3R)TI-D</i>	97B;97E-F	X ray	<i>Df(3R)TI84cRXD</i>
<i>Df(3R)TI-I</i>	97B;97E	X ray	<i>Df(3R)TI5BRXI</i>
<i>Df(3R)TI-K</i>	97A;97D-E	EMS	<i>Df(3R)TI5BREK</i>
<i>Df(3R)TI-P</i>	97A;98A1-2	X ray	<i>Df(3R)TI5BRXP</i>
<i>Df(3R)TI-Q^α</i>	97A;97D1-2	X ray	<i>Df(3R)TI5BRXQ</i>
<i>Df(3R)TI-X^α</i>	97B;97D1-2	X ray	<i>Df(3R)TI9QRX</i>

^α Cytologically visible break within *Tl*. The 97D1-2 breakpoint is located in the 6.0 kb EcoRI fragment of the Toll clone (Hashimoto, Hudson, and Anderson, 1988, *Cell* 52: 269-79).

***Df(3R)tll: Deficiency (3R) tailless**

origin: X ray induced.

deficiency	cytology	ref ^α	genetics
<i>Df(3R)tll-e^γ</i>	100A1-2;100B5-9	1, 3-5	<i>tll⁻ - Chp⁻</i>
<i>Df(3R)tll-f</i>	100A2-3;100C1-2	4	<i>tll⁻ - Chp⁻</i>
<i>Df(3R)tll-g</i>	?;100B3-4	1, 4, 5	<i>tll⁻</i>
<i>Df(3R)tll-pgx</i>	100A1-2;100B1-2	2	<i>tll⁻</i>

^α 1 = Mahoney and Lengyel, 1987, *Dev. Biol.* 122: 464-70; 2 = Pignoni, Baldarelli, Steingrimsson, Diaz, Patapouliau, Merriam, and Lengyel, 1990, *Cell* 62: 151-63; 3 = Strecker, Kongsuwan, Lengyel, and Merriam, 1986, *Dev. Biol.* 113: 64-76; 4 = Strecker, Merriam, and Lengyel, 1988, *Development* 102: 721-34; 5 = Van Vactor, Krantz, Reinke, and Zipursky, 1988, *Cell* 52: 281-90.

^γ Molecular biology: Homozygous deficiency embryos show six instead of seven *ftz* protein stripes (Mahoney and Lengyel, 1987).

Df(3R)Tpl: Deficiency (3R) Triplo-lethal

genetics: Deficient for *Tpl* region at 83D-E. Viable over *Tpl* duplications, but lethal over wild-type 3's or *Tpl* deficiencies.

other information: Two more putative deficiencies (synonyms: *20c76* and *25e76*) were viable over wild-type 3's, but the stocks were lost because of male sterility and not tested further.

deficiency	cytology	origin ^α	ref ^β	synonym
<i>Df(3R)Tpl1</i>	83D4-5;83E1-2	synthetic, γ ray	1, 2	<i>Df(3R)3g74</i>
<i>Df(3R)Tpl2</i>	83D4-5;84A1-2	X ray	1, 2	<i>Df(3R)1c77</i>
<i>Df(3R)Tpl3</i>	83D4-5;84A4-5	EMS	1, 2	<i>Df(3R)5f77</i>
<i>Df(3R)Tpl4</i>	83D4-5;83E1-2	EMS	1, 2	<i>Df(3R)10d77-7</i>
<i>Df(3R)Tpl5</i>	83D1-2;83E1-2	EMS	1, 2	<i>Df(3R)25g77</i>
<i>Df(3R)Tpl6</i>	83D1-2;84A4-5	γ ray	1, 2	<i>Df(3R)26b77</i>
<i>Df(3R)Tpl7</i>	83D4-5;83E1-2	γ ray	1, 2	<i>Df(3R)28b77</i>
<i>Df(3R)Tpl8</i>	83D4-5;83E1-2	EMS	1, 2	<i>Df(3R)28f77</i>
<i>Df(3R)Tpl9</i>	83D4-5;84A4-5	γ ray	1, 2	<i>Df(3R)29c76</i>
<i>Df(3R)Tpl10</i>	83C1-2;84B1-2	γ ray	1, 2	<i>Df(3R)30c76</i>
<i>Df(3R)Tpl11^γ</i>	lacks 83E	EMS	3	<i>tpl¹⁶</i>
<i>Df(3R)Tpl12^γ</i>	lacks 83E	EMS	3	<i>tpl²⁵</i>
<i>Df(3R)Tpl13^γ</i>	lacks 83E	EMS	3	<i>tpl³⁶</i>
<i>Df(3R)Tpl14^γ</i>	lacks 83E	EMS	3	<i>tpl³⁷</i>

^α *Df(3R)Tpl1* was induced in *Df(3R)Tpl/Dp(3;3)Tpl* females, while the other deficiencies were induced in wild-type males (Keppy and Denell, 1979).

^β 1 = Denell and Keppy, 1979, *Genetics* 93: 117-30; 2 = Keppy and Denell, 1979, *Genetics* 91: 421-41; 3 = Roehrdanz and Lucchesi, 1980, *Genetics* 95: 355-66.

^γ Also deficient for *Ki*. Salivaries lack quinacrine-bright region at 83D-E.

Df(3R)trxE8: Deficiency (3R) trithorax

origin: EMS induced.

discoverer: Kennison.

genetics: *red⁺trx⁻su(Hw)⁻cv-c⁺*.

Df(3R)trxE12

origin: EMS induced.

discoverer: Kennison.

genetics: *red⁻trx⁻su(Hw)⁻cv-c⁺*.

Df(3R)Ubx: Deficiency (3R) Ultrabithorax

deficiency	cytology	origin	discov	ref ^α	genetics
<i>Df(3R)Ubx19</i>	89D1-2;89E1-2				
<i>Df(3R)Ubx43</i>	89D;89E1-2(?)	X ray	Lewis, 73f		<i>Ubx⁻</i>
<i>Df(3R)Ubx109^β</i>	89D1-2;89E1-2		Lewis	1, 2, 3	<i>abx⁻ - iab8⁻</i>
<i>Df(3R)Ubx-C1</i>	{89E1-2;89E1-2}			4	<i>Ubx⁻ (partial)</i>
					<i>abdA⁻</i>
<i>Df(3R)Ubx-R13^γ</i>	89D1-2;89E1-2	X ray	Ramey		<i>Cbx⁺ Ubx⁻</i>
<i>Df(3R)Ubx-R29^γ</i>	89D;89E	X ray	Ramey		<i>Cbx⁺ Ubx⁻</i>
<i>Df(3R)Ubx-R32^γ</i>	89C;89E	X ray	Ramey		<i>Cbx⁺ Ubx⁻</i>

^α 1 = Jiménez and Campos-Ortega, 1981, *Wilhelm Roux's Arch. Dev. Biol.* 190: 370-73; 2 = Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, *Cell* 43: 81-96; 3 = Lewis, 1978, *Nature* 276: 565-70; 4 = Sánchez-Herrero, Vernós, Marco, and Morata, 1985, *Nature* 313: 108-13.

^β Molecular biology: Right breakpoint 86 to 93 kb distal to the right breakpoint of *In(3R)Cbx^{rv1}*

^γ Synonym: *Cbx^{rev-R17.13N}, Cbx^{rev-R17.29E}, Cbx^{rev-R17.32H}*

Df(3R)urd: Deficiency (3R) urdur

cytology: Lacks several bands in 87F.
origin: γ ray induced.
discoverer: Kennison, 1983.
synonym: *Df(3R)JK6;urd*¹.
references: Kennison and Tamkun, 1988, Proc. Nat. Acad. Sci. USA 85: 8136-8140.
genetics: Viable in combination with *Df(3R)ry27* = *Df(3R)87D1-2;87F1-2* and *Df(3R)red-P52* = *Df(3R)88A12-B1;88B4-5*. Lethal in combination with *Df(3R)l26c* = *Df(3R)87D14-E1;87F11-12* and with *Df(3R)red31* = *Df(3R)87F12-14;88C1-3*.

Df(3R)V: Deficiency (3R) Vincent

discoverer: Vincent.
references: Jones and Rawls, 1988, Genetics 120: 733-42.

deficiency	cytology	genetics	mol. biol.	α
<i>Df(3R)V2</i>	84E6-9;85A3-5	<i>l(3)84Eg⁻-Dhod⁻</i>	+19	
<i>Df(3R)V5</i>	85A5-6;85A6-11	<i>l(3)85Ab⁻-hb⁻</i>		
<i>Df(3R)V8</i>	85A4-5;85A6-11	<i>l(3)84Eg⁻-l(3)85Ad⁻</i>	+37	

α DNA coordinates of the 85A (distal) breakpoint.

Df(3R)VW4

cytology: *Df(3R)85E11;85F11*.
origin: γ ray induced.
discoverer: Walker.
references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.
genetics: Minute, presumably deficient for *M(3)85E*.

Df(3R)WIN

origin: X ray induced.
genetics: Deficient for *pb* but not *lab*. *Df(3R)WIN/Df(3R)MAP2* viable and *pb⁻*.

deficiency	cytology	ref	α
<i>Df(3R)WIN1</i>	84A4-5;84B1-2	3	
<i>Df(3R)WIN3</i>	84A4-5;84C1-2	1, 2, 3	
<i>Df(3R)WIN11</i>	83E1-2;84A4-5		

α 1 = Cavener, Otteson, and Kaufman, 1986, Genetics 114: 111-23; 2 = Hays, Deuring, Robertson, Prout and Fuller, 1989, Mol. Cell Biol. 9: 875-84; 3 = Pultz, Diederich, Cribbs, and Kaufman, 1988, Genes Dev. 2: 901-20.

Df(3R)X2

cytology: *Df(3R)84A4-5;84B1-2*.
origin: X ray induced.
references: Pultz, Diederich, Cribbs, and Kaufman, 1988, Genes Dev. 2: 901-20.
genetics: Deficient for *pb* but not *lab*. *Df(3R)X2/Df(3R)MAP2* viable and *pb⁻*.

Df(3R)X3F

cytology: *Df(3R)99D1-2;99E1*.

Df(3R)XS

cytology: *Df(3R)96A1-7;96A21-25*.

Df(3R)XTA1

cytology: *Df(3R)95B;95D*.
discoverer: Jürgens.
references: Wustmann, Szidonya, Taubert, and Reuter, 1989, Mol. Gen. Genet. 217: 520-27.

Df(4)3-Df(4)40

origin: X ray induced.

deficiency	cytology	ref	α	deficient for
<i>Df(4)3</i>	102E2-10;102F2-10	1-4		<i>l(4)102EFa-sv</i>
<i>Df(4)11</i>	102E2-10;102F2-10	1-5		<i>l(4)102EFa-spa</i>
<i>Df(4)34</i>	{102E-F}	1		<i>spa</i>
<i>Df(4)38</i>	102E2;102E10	2-5		<i>l(4)102EFa-l(4)102EFc</i>
<i>Df(4)40</i>	102E2-10;102F1-10	2-5		<i>sv-spa</i>

α 1 = CP627; 2 = Hochman, 1971, Genetics 67: 235-52; 3 = Hochman, 1974, Cold Spring Harbor Symp. Quant. Biol. 38: 581-89; 4 = Hochman, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 903-28; 5 = Hochman, Gloor, and Green, 1964, Genetica 35: 109-26.

Df(4)2c: see *Df(4)bt-D*

Df(4)62e: see *Df(4)M101-62e*

Df(4)62f: see *Df(4)M101-62f*

Df(4)63a: see *Df(4)M101-63a*

Df(4)bt-D: Deficiency (4) bent-Dominant

cytology: *Df(4)101B6-10*.
origin: X ray induced.
discoverer: Schultz.
references: Bridges, 1935, Biol. Zh. (Moscow) 4: 401-20. Hochman, Gloor, and Green, 1964, Genetica (The Hague) 35: 109-26. Hochman, 1971, Genetics 67: 235-52. 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 903-28.
genetics: Deficient for *l(4)102Cda-l(4)102Cdb*. *bt* phenotype no longer observed (Hochman, 1976).

Df(4)Cat: Deficiency (4) Cataract

origin: X ray induced.
discoverer: Belgovsky, 1936.
references: 1937, DIS 8: 7. Morgan, 1941, DIS 14: 52. Hochman, Gloor, and Green, 1964, Genetica (The Hague) 35: 109-26. Hochman, 1971, Genetics 67: 235-52.
genetics: Deficient for *l(4)102EFa-sv*.

Df(4)ci-D: Deficiency (4) cubitus interruptus-Dominant

origin: X ray induced.
discoverer: Ruch, 32a18.
references: Bridges, 1935, Biol. Zh. (Moscow) 4: 401-20. Hochman, Gloor, and Green, 1964, Genetica (The Hague) 35: 109-26. Hochman, 1971, Genetics 67: 235-52.
genetics: Deficient for *l(4)102ABb* and *l(4)102ABc*.

Df(4)G: Deficiency (4) of Gloor and Green

cytology: *Df(4)102E2-10*; tip of 4R lost and remainder of chromosome 4 capped with X-chromosomal material, including 1A (Hochman).
origin: X ray induced.
discoverer: Gloor and Green, 1957.
references: Hochman, Gloor, and Green, 1964, Genetica 35: 109-26. Hochman, 1971, Genetics 67: 235-52. 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London,

CHROMOSOMES - DEFICIENCIES

New York, San Francisco, Vol. 1b, pp. 903-28.

genetics: Deficient for *l(4)102EFa-l(4)102EFf*; fails to complement *sv* and *spa*. Lethal homozygous and in heterozygous combination with *Df(4)3*, *Df(4)11*, *Df(4)12*, *Df(4)24*, and *Df(4)34*. *y*⁺ and *ac*⁺ linked terminally.

Df(4)M: see *Df(4)M101*

Df(4)M2: see *Df(4)M101-2*

Df(4)M3: see *Df(4)M101-3*

Df(4)M4: see *Df(4)M101-4*

Df(4)M62e: see *Df(4)M101-62E*

Df(4)M62f: see *Df(4)M101-62f*

Df(4)M63-a: see *Df(4)M101-63a*

Df(4)M101: Deficiency (4) Minute

deficiency	cytology	origin	synonym	ref ^α	deficient for
<i>Df(4)M101</i> ^β	101E-F; 102B6-17	spont	<i>Df(4)M</i>	1, 2, 5, 7	<i>M(4)101</i> <i>-l(4)102ABi</i>
* <i>Df(4)M101-2</i>	{102A1-2; 102B2-5}	X ray	<i>Df(4)M2</i>	1	<i>ci-ar</i>

deficiency	cytology	origin	synonym	ref ^α	deficient for
* <i>Df(4)M101-3</i>	{101E-F; 102B2-5}	X ray	<i>Df(4)M3</i>	2, 3	<i>M(4)101-ar</i>
<i>Df(4)M101-4</i>	{101E-F; 102B2-5}	X ray	<i>Df(4)M4</i>	4	<i>M(4)101-Ce</i>
<i>Df(4)M101-62e</i> ^γ	101E; 102D13-E1		<i>Df(4)M62e</i>	3	<i>ar-ey</i>
<i>Df(4)M101-62f</i>	101E; 102B10-17	γ ray	<i>Df(4)M62f</i>	3	<i>M(4)101-ar</i>
<i>Df(4)M101-63a</i> ^δ	101F2-102A1; 102A2-5		<i>Df(4)M63a</i>	3, 5, 6 not <i>ci</i> ^D .	<i>M(4)101-ci</i> ,

^α 1 = Bridges, 1935, Biol. Zh. 4: 401-20; 2 = Bridges, 1935, Tr. Dinam. Razvit. 10: 469-70; 3 = CP627; 4 = Glass, 1944, DIS 18: 40; 5 = Hochman, 1971, Genetics 67: 235-52; 6 = Hochman, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 903-28; 7 = Slyzinski, 1944, J. Hered. 35: 322-24.

^β Right break to left of 102B9-10 according to Bridges, to right according to Slyzinski.

^γ Recovered by Fahmy from progeny of male injected with *Drosophila* DNA.

^δ Recovered by Fahmy from progeny of male injected with thymus extract from leukemic mice ("Gross Factor"). Eclosion delayed.

Df(w-ec)^{64d}: see *Tp(1;2)w-ec*

DUPLICATIONS

Doubler: see $Dp(1;1)B^S RMG$

$Dp(1)3C1$: see $Dp(1;1)w^{rG}$

Dp(1;1)3C3

cytology: $Dp(1;1)3C2-3;3C3-5$; tandem duplication of right-most sites of w (w^e and w^{sp}).

synonym: $Dp3C3$; $Dp(w^+R)$; $Dp(w^e;w^{sp})$.

references: Green, 1963, *Z. Vererbungsl.* 94: 200-14.

1967, *Biol. Zentralbl.* 86: 211-20.

Arcos-Teran, 1972, *Chromosoma* 37: 233-96.

genetics: Homozygotes wild type in eye color. Males and females with z and the duplication are z .

Dp(1;1;1)3C3

cytology: Triplication of right-most sites of w inferred (Green, 1967). No thickening of 3C3 visible in polytene chromosomes (Lefevre).

origin: From homozygous $Dp(1;1)3C3$ females.

synonym: $Tr(w^+R)$; $Dp(1;1)3C3$ (triplication).

references: Green, 1967, *Biol. Zentralbl.* 86: 211-20.

Arcos-Teran and Beerman, 1968, *Chromosoma* 25: 377-91.

Arcos-Teran, 1972, *Chromosoma* 37: 233-96.

genetics: Homozygotes wild type in eye color. Males and females with z and the duplication are z .

Dp(1;1)4E;6F

cytology: $Dp(1;1)4E2-F1;6F1-2$. Reversed repeat.

new order:

1A – 6F|6F – 4E|6F – 20.

origin: Derived from *Uc* unstable X.

references: Lim, 1979, *Genetics* 93: 681-701.

genetics: Unstable.

****Dp(1;1)100: Duplication (1;1) 100***

origin: Spontaneous product of exchange between $Dp(1;f)100$ and proximal heterochromatin of $C(1)RM$.

****Dp(1;1)105***

cytology: Metaphase X chromosome has one arm of normal length and one about 40% normal length.

new order:

1 – 20F·|6 – 1.

origin: X-ray-induced deletion of most of X euchromatin was recovered as a $C(1)RM/Dp(1;f)105$ female, which by detachment produced $Dp(1;1)105$ in the succeeding generation.

discoverer: Dobzhansky, 1930.

references: 1932, *Biol. Zentralbl.* 52: 493-509.

genetics: Contains wild-type alleles of y through dx and also probably bb .

Dp(1;1)112

origin: Spontaneous product of exchange between $Dp(1;f)112$ and proximal heterochromatin of an attached-X.

****Dp(1;1)138***

origin: X-ray-induced deletion of most of X euchromatin that was recovered as a $C(1)RM/Dp(1;f)138$ female, which by detachment produced $Dp(1;1)138$ in the subsequent generations.

discoverer: Dobzhansky, 1930.

references: 1935, *Z. Indukt. Abstamm. Vererbungsl.*

68: 134-62.

genetics: Extends from locus of r to base of X; carries *B*. Female nearly wild type, but male has low viability and is sterile.

****Dp(1;1)258-46***

cytology: $Dp(1;1)2B4-7;3A4-6$; reversed repeat (Sutton).

new order:

1 – 2B4|3A4 – 2B7|2B7 – 20 or

1 – 3A4|3A4 – 2B7|3A6 – 20.

origin: X ray induced.

discoverer: Demerec, 38l.

genetics: Originally appeared as w but reverted to w^+ . No effect on *pn-gt* reported.

Dp(1;1)Ax^{28a}: Duplication (1;1) Abruptex

cytology: Single-band duplication believed to involve 3C7 by Morgan, Schultz, and Curry (1941); identity of extra band not confirmed by Lefevre (1953, *Genetics* 38: 345-59).

new order:

1A – 3C7|3C7 – 20.

origin: X ray induced.

references: Morgan, Schultz, and Curry, 1941, *Year Book - Carnegie Inst. Washington*

Welshons, 1971, *Genetics* 68: 259-68.

genetics: *Ax* phenotype.

Dp(1;1)B: Duplication (1;1) Bar

cytology: $Dp(1;1)15F9-16A1;16A7-B$; a tandem duplication (Bridges, 1936, *Science* 83: 210-11; Muller, Prokofyeva-Belgovskaya, and Kossikov, 1936, *Dokl. Acad. Nauk SSSR* 1: 87-88).

new order:

1 – 16A7|16A1 – 20.

origin: Spontaneous.

discoverer: Tice, 13b.

references: 1914, *Biol. Bull.* 26: 221-30.

genetics: Position effect for *B*, apparently resulting from juxtaposition of 16A1 with 16A7, which may undergo mutation to less extreme forms (e.g., B^i). Produces normal and triplicated [$Dp(1;1)BB$] products by unequal crossing over.

****Dp(1;1)B²⁶³⁻²⁸***

cytology: $Dp(1;1)15F9-16A1;16A3-4;16A6-7;16A7-B1$.

new order:

1 – 16A3|16A7|16A1 – 20.

origin: X-ray-induced deletional derivative of $Dp(1;1)B^i B^i = Dp(1;1)15F9-16A1;16A7-B1$.

discoverer: Demerec, 34b.

references: Sutton, 1943, *Genetics* 28: 97-107.

****Dp(1;1)B²⁶³⁻⁴⁸: see Tp(1;1)B²⁶³⁻⁴⁸******Dp(1;1)B^S-H: Duplication (1;1) Bar Stone of Hinton***

new order:

1A1-1B3|20F-3C2|20F|1A4-3C1|20F-16A1|102F;

position of centromere indeterminate; adjacent to either 3C1 or 3C2.

origin: Generated by double exchange in a $R(1)2, In(1)w^{vC}/In(1)sc^{4L}sc^{8R}/X^{P4D}$ female, one exchange occurring between the B^S element and $R(1)2$ and the other between $In(1)sc^{4L}sc^{8R}$ and $R(1)2$.

references: Hinton, 1957, *Genetics* 42: 55-65.

phenotype: These linear derivatives of the w^{vC} ring do not undergo the somatic loss characteristic of the unstable ring; they do, however, display indications of variable levels of instability, presumably related to the degree of instability of the generating ring. This is recognized by reduced recovery and elevated primary nondisjunction in $Dp(1;1)B^S-H/In(1)dl49$ females. Exchange between the B^S duplication and the long arm of the same chromatid regenerates single rings whose stabilities reflect those of the parental linear chromosome. Exchange between the B^S duplication and a normal homologue generates tandem-metacentric compound chromosomes [$C(1)TM-H$].

$Dp(1;1)B^S RAG$: Duplication (1;1) Bar of Stone Reversed Acrocentric

cytology: $Dp(1;1)15F9-16A1;20$.

new order:

·20|1A – 20|20 – 16A1|102F.

origin: Spontaneous recombinant between the distal X of a $C(1)RA$ and X^{P4D} from $T(1;4)B^S$.

discoverer: Lindsley and Sandler.

references: 1963. Methodology in Basic Genetics (W. J. Burdette, ed.). Holden-Day, Inc., San Francisco, pp. 390-403.

genetics: Generates reversed acrocentric compound X chromosomes in $Dp(1;1)B^S RAG/+$ female, usually by a double exchange in which one exchange occurs between the duplicated segment of one strand and the homologous region of its sister and the other between the duplication-bearing X and its normal homolog. Rate of $C(1)RA$ generation about 6×10^{-4} .

$Dp(1;1)B^S RMG$: Duplication (1;1) Bar of Stone Reversed Metacentric

cytology: $Dp(1;1)15F9-16A1;20$.

new order:

1 – 20·20 – 16A1|102F.

origin: Spontaneous recombinant between $C(1)RM$ and the X^{P4D} element of $T(1;4)B^S$.

discoverer: Muller.

synonym: *Doubler*.

references: 1936, DIS 6: 8.

Lindsley and Sandler, 1963, Methodology in Basic Genetics (W. J. Burdette, ed.). Holden-Day, Inc., San Francisco, pp. 390-403.

genetics: Generates reversed metacentric compound X chromosomes in $Dp(1;1)B^S RMG/+$ female by crossing over between the duplicated segment and either the X to which it is attached or the homologous X at a rate of about 2.5×10^{-4} .

$Dp(1;1)B^S TAG$: Duplication (1;1) Bar of Stone Tandem Acrocentric

cytology: $Dp(1;1)15F9-16A1;20$.

new order:

·20 – 1A|20 – 16A1|102F.

origin: X-ray-induced recombinant between the distal heterochromatin of an X chromosome with a terminal heterochromatin segment derived from y^{+Y} and the proximal heterochromatin of the X^{P4D} element of $T(1;4)B^S$.

discoverer: Lindsley and Sandler.

references: 1963, Methodology in Basic Genetics (W. J. Burdette, ed.). Holden-Day, Inc., San Francisco, pp. 390-403.

genetics: Ineffective in generating tandem acrocentric compound X chromosomes.

$Dp(1;1)B^S TMG$: Duplication (1;1) Bar of Stone Tandem Metacentric

cytology: $Dp(1;1)15F9-16A1;20$ added as a second arm to $In(1)sc^{8L}EN^R$.

new order:

1A – B2|20B – 1A|20·20 – 16A1|102F.

origin: Spontaneous recombinant between the X in normal sequence of a $C(1)TM$ and the X^{P4D} element of $T(1;4)B^S$.

discoverer: Lindsley and Sandler.

references: 1963, Methodology in Basic Genetics (W. J. Burdette, ed.). Holden-Day, Inc., San Francisco, pp. 390-403.

genetics: Generates tandem metacentric compound X chromosomes in $Dp(1;1)B^S TMG/+$ female by recombination between the duplication and the base of a homolog in normal sequence, at a rate of about 20×10^{-4} .

$Dp(1;1)B^S TRG$: Duplication (1;1) Bar of Stone Tandem Ring-genic

See $C(1)TMB^S$ subsection in compound chromosomes.

$Dp(1;1)bb^{DfL}Cl^R$

cytology: $Dp(1;1)4A5-B1;4D2-3 + Dp(1;1)17A6-B1;20B-C$.

origin: Associated with $In(1)bb^{DfL}Cl^R$.

references: CP627.

$Dp(1;1)bb^{DfL}y^{4R}$

cytology: $Dp(1;1)1A8-B1;4D2-3 + Dp(1;1)18A3-4;20B-C$.

origin: Associated with $In(1)bb^{DfL}y^{4R}$.

references: CP627.

$Dp(1;1;1)BB$: Duplication (1;1;1) Bar + Bar

cytology: $Dp(1;1)15F9-16A1;16A7-B1$; a tandem triplication [Bridges, 1936, Science 83: 210-11 (fig.)].

new order:

1 – 16A7|16A1 – 16A7|16A1 – 20.

origin: Spontaneous through unequal crossing over in B/B female.

discoverer: Zeleny.

references: 1920, J. Exp. Zool. 30: 292-324.

Sturtevant, 1925, Genetics 10: 117-47.

genetics: Either or both B regions may carry a less extreme derivative of B (i.e., B^iB , BB^i , or B^iB^i). Number of duplicated segments may be either increased or decreased by unequal crossing over.

*** $Dp(1;1)Bt$: Duplication (1;1) Branchlet**

cytology: $Dp(1;1)3B2-C1;6F6-7$; tandem repeat (Darby).

new order:

1 – 6F4|3C1 – 20.

origin: Induced by ^{32}P .

discoverer: Bateman, 1950.

references: 1950, DIS 24: 54.

1951, DIS 25: 77.

$Dp(1;1)Bx^f$: Duplication (1;1) Beadex-recessive

cytology: $Dp(1;1)17A;17E-F$ (E. B. Lewis).

origin: Spontaneous.

discoverer: Ives, 35k.

references: 1937, DIS 7: 6.

Green, 1952, Proc. Nat. Acad. Sci. USA 38: 949-53.

1953, Genetics 38: 91-105.

1953, Z. Indukt. Abstamm. Vererbungsl. 85: 435-49.

genetics: Duplicated for *os*⁺, *Bx*⁺, and *fu*⁺. Does not yield unequal crossovers as does *Bx*^{r49k}.

Dp(1;1)Bx^{r49k}

cytology: *Dp(1;1)17A;17C* (E. B. Lewis).

origin: Spontaneous.

discoverer: Mossige, 49k22.

references: 1950, DIS 24: 61.

Green, 1953, Z. Indukt. Abstamm. Vererbungsl. 85: 435-49.

Green, 1968, Mol. Gen. Genet. 103: 209-17.

genetics: Duplicated for *Bx*⁺ but not for *os*⁺ or *fu*⁺. Unequal crossing over yields wild types and triplications. Quadruplications have also been produced.

Dp(1;1)Cl^Ly^{4R}

cytology: *Dp(1;1)1A8-B1;4A5-B1+Df(1)17A6-B1;18A3-4..*

new order:

1 - 4A|17A6-1B1|18A4 - 20.

origin: Associated with *In(1)Cl^Ly^{4R}*.

references: CP627.

Dp(1;1)Co: Duplication (1;1) Confluens

cytology: *Dp(1;1)3C4-5;3D6-E1*; tandem duplication (Schultz, 1941, DIS 14: 54-55).

new order:

1 - 3D6|3C5 - 20.

origin: Recovered among progeny of cold-treated fly.

discoverer: Gottschewski, 34c.

references: 1935, DIS 4: 7, 14, 16.

1937, DIS 8: 12.

1937, Z. Indukt. Abstamm. Vererbungsl. 73: 131-42.

genetics: The *Co* phenotype arises from a duplication of the Notch locus (salivary band 3C7).

Dp(1;1)ct^{7B}

cytology: *Dp(1;1)6F1-2;7C1-2*. Reversed repeat.

new order:

1 - 6F|7C - 6F|6F - 20.

origin: Derived from *Uc* unstable X.

references: Lim, 1979, Genetics 93: 681-701.

genetics: Unstable.

Dp(1;1)G: Duplication (1;1) Gelbart

references: Gelbart, 1971, Ph.D. Thesis.

duplication	cytology
Dp(1;1)GJ4	<i>1B6-7;3C1-2;3C2-3</i> ^α
Dp(1;1)GJ5	<i>3C2;3C3 + In(1)1B;10A</i>
Dp(1;1)GM9	<i>3B;3F</i>
Dp(1;1)GM10	<i>3A2-3;3C3</i>

^α new order: 1 - 1B6|3C2 - 1B7|3C2 - 20 (tentative).

Dp(1;1)Gr: Duplication (1;1) Green

cytology: *Dp(1;1)3A2-3;8B4-C1*; tandem repeat. More than ¼ the length of euchromatic X at metaphase.

origin: Spontaneous.

references: Kalisch, 1973, Chromosoma 41: 237-42.

1975, Theoret. Appl. Genet. 46: 169-80.

1976, Genet. Res. 26: 275-82.

1980, DIS 55: 206-07.

Charton-Struck and Kalisch, 1981, DIS 56: 28-29.

genetics: Homo- and hemizygous lethal. Heterozygous females viable; show reduction in crossing over within and adjacent to the duplication and increase in crossing over in distal parts of 2 and 3. Duplication chromosome marked by *y*² *sc* distally and (*w*⁻ *spl sn*³)(*w*^c *sn*³) within the duplication.

Dp(1;1)hdp-b: Duplication (1;1) heldup-b

origin: Induced in progeny of dysgenic flies.

references: Engles and Preston, 1984, Genetics 107: 657-78.

genetics: Mutant for *hdp-b* as result of break at 17C, site of *P* factor in strain π2.

duplication	cytology	chrom. design.	new order
Dp(1;1)hdp12	<i>17C;17C</i>		
Dp(1;1)hdp-b1 ^α		B395.2	1A - 17C dp 17C - 20.
Dp(1;1)hdp-b2	<i>12F;13E;17C</i>	F14f	1A - 17C 12F - 13E 17C - 20.
Dp(1;1)hdp-b3	<i>5A;7A;17C;19F</i>	L40	1A - 7A 19F - 17C 5A - 17C 19F - 20
Dp(1;1)hdp-b4 ^β		N71	1A - 17C dp 17C - 20.
Dp(1;1)hdp-b5 ^α		N86	1A - 17C dp 17C - 20.
Dp(1;1)hdp-b6	<i>4F;5E;17C</i>	N224	1A - 5E 17C - 4F 17C - 20.
Dp(1;1)hdp-b7	<i>4F;5E;17C</i>	N254	1A - 17C 5E - 4F 17C - 20.

^α Dp for one of more bands.

^β Dp for several bands.

Dp(1;1)hdp-b^{rv}: Duplication (1;1) heldup-b reverted

cytology: *Dp(1;1)5E;7A*; reversed repeat.

new order:

1A - 5E|7A - 5E|5E - 20.

synonym: *Dp(1;1)019*.

origin: Induced in progeny of *hdp-b* fly (*P* factors in rearranged chromosome at *5E* and *17C*).

references: Engles and Preston, 1984, Genetics 107: 657-78.

genetics: Reverted for *hdp-b*.

Dp(1;1)IMNB-8

cytology: Inversion of segment overlapping the tandem duplication *Dp(1;1)MNB-8*.

new order:

1A - 16F|16F - 16A1|16A7 - 16A1|17E1 - 17A2|16F - 20.

origin: Spontaneous derivative of *Dp(1;1)MNB-8*.

references: Holmquist, 1972, Chromosoma 36: 413-52.

genetics: Has a *B os*⁺ phenotype and a (*B*⁺ *B*)(*os*⁺ *os*) genotype.

Dp(1;1)jnR1-A

cytology: Small duplication in 6F-7A region.

references: Mainz, Salz, Cline, and Schedl, 1985, Cell 43: 521-29.

genetics: Duplicated for *Sxl*⁺ and *l(1)7Aa*.

Dp(1;1)jnR1-B

cytology: Small duplication in 6F-7A region.

references: Mainz, Salz, Cline, and Schedl, 1985, Cell 43: 521-29.

genetics: Duplicated for *l(1)7Aa* but not *Sxl*⁺.

Dp(1;1)L-B^S: Duplication (1;1) Lindsley

origin: Replacement of *y*⁺ in *Dp(1;1)L-y*⁺ with *B*^S from *B*^S*Yy*⁺ resulting in the production of a *B*^S(*het*) *y cv v f bb*⁻ chromosome.

genetics: Carries *B*^S distal to and *y cv v f bb*⁻ proximal to the heterochromatin.

Dp(1;1)L-y^{31d}

origin: Replacement of y⁺ in Dp(1;1)L-y⁺ with y^{31d} from FM6 resulting in production of a y^{31d} (het) y cv v f bb⁻ chromosome.

genetics: Carries y^{31d} distal to and y cv v f bb⁻ proximal to the distal heterochromatin.

Dp(1;1)L-y⁺

origin: X-ray-induced recombination in a B^SYy⁺/FM6/YSX, Df(1)bb3a, y cv v f female resulting in a YSX, Df(1)bb3a, y⁺ KS y cv v f bb⁻ chromosome.

references: Lindsley and Sandler, 1963, Methodology in Basic Genetics (Burdette, ed.). Holden-Day, Inc., San Francisco, pp. 390-403.

genetics: Carries y⁺ distal to and y cv v f bb⁻ proximal to the distal heterochromatin.

Dp(1;1)lz-1: Duplication (1;1) lozenge

cytology: Dp(1;1)8D;8F; tandem duplication.

origin: X ray induced in lz^{50e} +/+ lz^{y4} female.

references: Bender, 1967, Genetics 55: 249-54.

Peeples, Geisler, Whitcraft, and Oliver, 1969, Genetics 62: 161-70.

genetics: Partial complementation for lz. Hemizygous males have slightly rough eyes, thin or missing posterior postalar bristles, and good fertility; homozygous females have rougher eyes, thin or missing posterior postalar bristles, and markedly reduced fertility although secondary reproductive structures are normal. Tarsal claws are normal in both sexes.

Dp(1;1)lz-2

cytology: Dp(1;1)8D1+; small tandem duplication.

origin: X ray induced in lz^{50e} +/+ lz^{y4} female.

references: Bender, 1967, Genetics 55: 249-54.

Peeples, Geisler, Whitcraft, and Oliver, 1969, Genetics 62: 161-70.

De la Concha, Dietrich, Weigel, and Campos-Ortega, 1988, Genetics 118: 499-508.

genetics: Partial complementation for lz. Duplication may include t⁺. Dp(1;1)lz-2 males have less rough eyes than Dp(1;1)lz-1 males, normal posterior postalar bristles, and good fertility; homozygous Dp(1;1)lz-2 females have moderately rough eyes, thin or missing posterior postalar bristles, and good fertility; tarsal claws normal in both sexes.

Dp(1;1)MNB-8

cytology: Dp(1;1)15F9-16A1;16A7-B1;17E2-F1 + Df(1)16 E-F1;17A2-3; tandem repeat with three copies of 16A1-7.

new order:

1A - 16E|17A3 - 17E1|16A1 - 16A7|16A1 - 20.

origin: Spontaneous derivative of Dp(1;1)NB-8.

synonym: MLD, modified long duplication.

references: Gabay and Laughnan, 1970, Genetics 65: 249-65.

Laughnan, Gabay, and Montgomery, 1971, DIS 47: 64.

Holmquist, 1972, Chromosoma 36: 413-52.

genetics: Has a B os⁺ phenotype and a (B⁺ os⁺) (B os) genotype. Intrachromosomal recombination in some lines results in exceptional male offspring (Peterson and Laughnan, 1964, Genetics 50: 275-76; Gabay and Laughnan, 1970; Laughnan et al., 1971).

Dp(1;1)NB-8

cytology: Dp(1;1)15F9-16A1;16A7-B1;17E2-F1; tandem repeat with three copies of 16A1-7.

new order:

1A - 17E1|16A1 - 16A7|16A1 - 20.

origin: Derived from Dp(1;1)NBB-8 by exchange with wild type X (crossover in distal 16A1-17E2 segment of the duplication).

synonym: SLD, standard long duplication.

references: Gabay and Laughnan, 1970, Genetics 65: 249-65.

genetics: Has a B os⁺ phenotype and a (B⁺ os⁺) (B os) genotype.

***Dp(1;1)NBB-8**

cytology: Dp(1;1)15F9-16A1;17E2-F1 superimposed on Dp(1;1)15F9-16A1;16A7-B1; four copies of 16A1-7.

new order:

1 - 16A7|16A1 - 17E2|16A1-16A7|16A1 - 20.

origin: Spontaneous in a B/f B os car female.

discoverer: Peterson and Laughnan.

synonym: OLD, original long duplication.

references: 1963, Proc. Nat. Acad. Sci. USA 50: 126-33.

Gabay and Laughnan, 1970, Genetics 65: 249-65.

genetics: Male viability reduced. Has a BB phenotype. Genetically it is (B os⁺) (B os).

Dp(1;1)O19

cytology: Dp(1;1)5E;7A.

new order:

1A - 5E|7A - 5E|5E - 20.

origin: Induced by hybrid dysgenesis.

references: Engles and Preston, 1984, Genetics 107: 657-78.

Dp(1;1)pn2: Duplication (1;1) prune

synonym: TE100.

genetics: Duplicated for Pgd.

Dp(1;1)S93: see Tp(1;1)S93

Dp(1;1)sc^{4L}sc^{8R}

cytology: Dp(1;1)1B2-3;1B3-4.

origin: Associated with In(1)sc^{4L}sc^{8R}.

Dp(1;1)sc^{4L}y^{4R}

cytology: Dp(1;1)1A8-B1;1B3-4 + Dp(1;1)18A3-4;h26.

origin: Associated with In(1)sc^{4L}y^{4R}.

Dp(1;1)sc^{8L}sc^{4R}

cytology: Dp(1;1)h26;h32.

origin: Associated with In(1)sc^{8L}sc^{4R}.

Dp(1;1)sc^{8L}sc^{L8R}

cytology: Dp(1;1)h30-31;h32.

origin: Associated with In(1)sc^{8L}sc^{L8R}.

Dp(1;1)sc^{8L}y^{4R}

cytology: Dp(1;1)1A8-B1;1B2-3 + Dp(1;1)18A3-4;h32.

origin: Associated with In(1)sc^{8L}y^{4R}.

Dp(1;1)sc²⁶⁰⁻²⁵

origin: Aneuploid recombinant from In(1LR)sc²⁶⁰⁻²⁵.

Dp(1;1)sc^{L8L}sc^{4R}

cytology: Dp(1;1)h26;h30-31.

origin: Associated with In(1)sc^{L8L}sc^{4R}.

Dp(1;1)sc^{L8L}sc^{8R}

cytology: *Dp(1;1)1B2-3;1B3-4*.

origin: Associated with *In(1)sc^{L8L}sc^{8R}*.

Dp(1;1)sc^{S1L}sc^{4R} (L. Robbins)

cytology: *Dp(1;1)20F;1B3-4*. *NO* and portions of surrounding heterochromatin duplicated at 1B3-4 inferred from genetics.

new order:

1 – 1B3|20F|1B4 – 20F.

origin: *Rex*-induced reinversion of *In(1)sc^{S1L}sc^{4R}*.

references: Swanson, 1984, Ph.D. thesis, Michigan State Univ.

Robbins and Swanson, 1988, *Genetics* 120: 1053-59.

genetics: One of two mitotic exchange products induced by *Rex* in *In(1)sc^{S1L}sc^{4R}* [see also *Dp(1;1)sc^{S1L}sc^{4R}*]. Euchromatin and heterochromatin are restored to normal sequence except for duplication of an inverted segment of 20F containing the *NO* and adjacent material at 1B3-4. Presence of *NO* material at both ends of the chromosomes has been confirmed by examination of the *bb* phenotypes of the separated ends, with varied levels of *bb* expression observed for the ends of independently derived chromosomes. Presence of adjacent heterochromatin in the duplicated segment is inferred from the structure of *In(1)sc^{S1L}sc^{4R}*.

Dp(1;1)sc^{S1L}sc^{8R}

cytology: *Dp(1;1)1B2-3;1B3-4*.

origin: Associated with *In(1)sc^{S1L}sc^{8R}*.

Dp(1;1)sc^{S1L}sc^{L8R}

cytology: *Dp(1;1)h30-31;h32*.

origin: Associated with *In(1)sc^{S1L}sc^{8R}*.

Dp(1;1)sc^{V1}: see *In(1LR)sc^{V1}*

Dp(1;1)SM1: Duplication (1;1) Semenova Mglinetz

cytology: *Dp(1;1)1A1;3E*.

origin: γ ray induced.

references: Semenova, Mglinetz, and Glotoff, 1970, *Genetika (Moscow)* 6(4): 165-69.

Mglinetz and Semenova, 1970, *Genetika (Moscow)* 6(8): 86-94.

Dp(1;1)SM2

cytology: *Dp(1;1)3A;11E*.

origin: γ ray induced.

references: Semenova, Mglinetz, and Glotoff, 1970, *Genetika (Moscow)* 6(4): 165-69.

Mglinetz and Semenova, 1970, *Genetika (Moscow)* 6(8): 86-94.

Dp(1;1)sn^{S93}: see *Tp(1;1)sn^{S93}*

***Dp(1;1)Th: Duplication (1;1) Theta**

origin: X-ray-induced detachment of *C(1)RM* with X-ray-induced deletion of most of the *X* euchromatin.

discoverer: Muller.

references: Muller and Painter, 1929, *Am. Nat.* 63: 197.

Patterson, 1930, *Genetics* 15: 141-49.

Muller, 1932, *Proc. Intern. Congr. Genet.*, 6th., Vol. 1: 213-55.

Stern, 1956, *Wilhelm Roux's Arch. Dev. Biol.* 149: 1-25.

genetics: Fragment of *X* chromosome, including *y*⁺, *sc*⁺,

and *bb*⁺ attached to right of *X* centromere. Causes development of interalar bristle not ordinarily present in *D. melanogaster* (Stern, 1956).

Dp(1;1)w: Duplication (1;1) white

cytology: *Dp(1;1)3A;3C*.

new order:

1 – 3C|3A – 20.

origin: Spontaneous as a recombinant from *w^{ch/w^{sp}}*.

discoverer: E. B. Lewis, 55j.

references: 1957, *DIS* 31: 84.

genetics: Loci of *w* and *rst* within duplicated section. Unequal crossing over gives normal and triplicated products. Quintuplication also produced.

Dp(1;1)w^{60h21}: see *Dp(1;1)w^{rG}*

Dp(1;1)w^{60h30}: see *Dp(1;1)w^{rG2}*

Dp(1;1;1)w^{81e1}

origin: Spontaneous (presumably induced by recombination between FB elements).

references: Collins and Rubin, 1984, *Nature* 308: 323-27.

genetics: Mutant for *w*. Male viable and fertile.

molecular biology: *w^c* insertion conserved and flanked on the left by two copies of the sequence duplicated in *wⁱ* and on the right by a single copy of the sequence.

Dp(1;1;1)w^{82a3}

origin: Spontaneous (presumably induced by recombination between FB elements).

references: Collins and Rubin, 1984, *Nature* 308: 323-27.

genetics: Mutant for *w*. Male viable and fertile.

molecular biology: 1 kb of DNA deleted from *w^c* inversion; otherwise, rearrangement same as *Dp(1;1;1)w^{81e1}*.

Dp(1;1)w⁺¹

cytology: *Dp(1;1)3C1-2;4A*; tandem duplication.

origin: Induced by ethyl methanesulfonate.

references: Green and Lefevre, 1979, *Chromosoma* 74: 329-35.

Green, 1984, *Mol. Gen. Genet.* 194: 275-78.

genetics: Male phenotype *w⁺* except with *z*. Duplicated males fertile, but do not survive as well as wild-type males. Reversion to non-duplicated chromosome in males and females. *Dp(1;1)w⁺¹*, *z* males are *z*.

Dp(1;1)w⁺²

cytology: *Dp(1;1)3C1-2;3C6-7* (probably); tandem duplication.

origin: Induced by ethyl methanesulfonate.

references: Green and Lefevre, 1979, *Chromosoma* 74: 329-35.

Green, 1984, *Mol. Gen. Genet.* 194: 275-78.

genetics: Phenotype *w⁺*. Males rarely survive and survivors are sterile; females are viable. Reversion to non-duplicated chromosome in females. *Dp(1;1)w⁺²*, *z* males are *z* (Rasmuson, Svahlin, Montell, and Olofsson, 1978, *Mutat. Res.* 54: 33-38).

Dp(1;1)w^{+61e19}

cytology: Tandem duplication of proximal part of *w* plus transposon inserted between duplications.

origin: Unequal crossing over.

synonym: *Dp(1;1)w^{+R61e19}*; *Dp(1;1)w^{+R}*; "DP" (Green, 1963).

references: Green, 1963, *Z. Vererbungsl.* 94: 200-14.

Rasmuson and Green, 1974, Mol. Gen. Genet. 133: 249-60.
 Rasmuson, Montell, Rasmuson, Svahlin, and Westerberg, 1980, Mol. Gen. Genet. 177: 567-70.
 Goldberg, Sheen, Gehring, and Green, 1983, Proc. Nat. Acad. Sci. USA 80: 5017-21.
 Fujikawa and Kondo, 1986, Genetics 112: 505-22.

genetics: Duplication males with *z* are *z* (Rasmuson, Svahlin, Montell, and Olofsson, 1978, Mutat. Res. 54: 33-38). *Dp(1;1)w⁺61e19*, *z/Dp(1;Y)y-z* males show *z* variegation. Duplication semistable, reverting to *z⁺*, the reversion occurring with a higher frequency in *mei-9* than in wild-type strains (Fujikawa and Kondo, 1986).

molecular biology: About 80 kb of DNA in proximal part of *w* duplicated (Gunaratne, Mansukhani, Lipari, Liou, Martindale, and Goldberg, 1986, Proc. Nat. Acad. Sci. USA 83: 701-05).

Dp(1;1)w⁺61e19

cytology: Tandem triplication thought to involve sub-locus 4 (or 4 and 5) of the *w* locus.

origin: Unequal crossing over.

synonym: *Tr(1)w⁺61e19*; "TR" (Green, 1963).

references: Green, 1963, Z. Vererbungsl. 94: 200-14.

Rasmuson and Green, 1974, Mol. Gen. Genet. 133: 249-60.

Rasmuson, Montell, Rasmuson, Svahlin, and Westerberg, 1980, Mol. Gen. Genet. 177: 567-70.

genetics: Triplication males with *z* are *z*.

Dp(1;1)w^a: Duplication (1;1) white-apricot

cytology: *Dp(1;1)3A10-B1;3C3-5* (Gersh, 1962, Genetics 47: 1393-98).

new order:

1 - 3C3|3B1 - 20.

origin: Spontaneous from *w^a/w^a* female; recovered once as a recombinant and once as a presumed recombinant.

discoverer: Green.

references: 1959, Genetics 44: 1243-56.

Dp(1;1)w^aw⁺

cytology: Tandem duplication of entire *w* locus marked by *w^a* in left segment and *w⁺* in right segment.

origin: Unequal crossing over in *w^a/w⁺* females.

synonym: *Dp(w^a;w⁺)*.

references: Green, 1966; Genetics 54: 881-85.

1967, Biol. Zentralbl. 86: 211-20.

1969, Mol. Gen. Genet. 103: 209-17.

genetics: Male phenotype *w⁺* except with *z*.

Dp(1;1)wⁱ: Duplication (1;1) white-ivory

references: Green, Todo, Ryo, and Fujikawa, 1986, Proc. Nat. Acad. Sci. USA 83: 6667-71 [except for *Dp(1;1;1;1)wⁱ*].

genetics: Heterozygote with *Dp(1;1)z* shows *z* eye color. Eye color of *wⁱ* duplication males and homozygous *wⁱ* duplication females darkens with increased dosage of *wⁱ*. Duplications unstable, reverting to *w⁺*.

molecular biology: Quantitative increase in white locus DNA (indicated by Southern blot hybridization) as *wⁱ* dosage increased.

duplication	cytology	origin
<i>Dp(1;1)wⁱ</i>	3A;3C; serial tandem duplication	X ray
<i>Dp(1;1;1)wⁱ</i>	serial tandem triplication	unequal Xover
<i>Dp(1;1;1;1)wⁱ α</i>	serial tandem quadruplication	unequal Xover

duplication	cytology	origin
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<i>Dp(1;1;1;1)wⁱ β</i>	serial tandem quintuplication	unequal Xover
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α
β
 Homozygous females are sterile.
 Discoverer: E.B. Lewis.
 Synonym: *Qn(1)wⁱ*; *Dp(1;1)wⁱQn*.
 References: Bowman, 1969, Mutat. Res. 7: 409-15.

Dp(1;1)w^{m51bL}w^{m4R} (L. Robbins)

cytology: *Dp(1;1)20F;3C1-2*. Variable portions of *NO* duplicated at 3C1-2; inferred from genetics.

new order:

1 - 3C1|20F|3C2 - 20F.

origin: *Rex*-induced reinversion of *In(1)w^{m51bL}w^{m4R}*.

discoverer: Robbins 87g.

references: Robbins and Swanson, 1988, Genetics 120: 1053-59.

genetics: One of two mitotic exchange products induced by *Rex* in *In(1)w^{m51bL}w^{m4R}* (see also *Dp(1;f)w^{m51bL}w^{m4R}*). Euchromatin and heterochromatin are restored to normal sequence except for duplication of *NO* material at 3C1-2. In effect, each such chromosome is a half-tetrad containing both of the products of a single *Rex*-induced exchange. Examination of the *bb* phenotypes of the separated *NO* regions of a series of independently generated *Dp(1;1)w^{m51bL}w^{m4R}* chromosomes indicates that both can be mutant, both can be *bb⁺*, or either one can be mutant and the other wild type (Robbins).

Dp(1;1)w^{rdp}

cytology: Tandem duplication of the proximal part of *w*.

references: Jack and Judd, 1978, Proc. Nat. Acad. Sci. USA 76: 1368-72.

genetics: Carries *w^{bf}* in distal part of *w*. Formed by recombination between offset *roo* insertion elements in *w^{ric}* and *w^{bf}*, reciprocal product is *w^{r,def}* (Davis, Shen, and Judd, 1987, Proc. Nat. Acad. Sci. 84: 174-78).

Dp(1;1)w^{rdp+}

cytology: Tandem duplication of the proximal part of *w*.

references: Jack and Judd, 1978, Proc. Nat. Acad. Sci. USA 76: 1368-72.

genetics: No *w^{bf}* in distal part of *w*.

Dp(1;1)w^{rg}: Duplication (1;1) white-recombinant of Green

cytology: *Dp(1;1)3A3-4;3C1-2* or *Dp(1;1)3A4-5;3C2-3* (Gersh, 1967).

new order:

1 - 3C1|3A4 - 20.

origin: A regular product of asymmetric exchange between 3C1-2 of a *w^a* or *w^{a2}* chromosome and 3A3-4 of specific homologs. Reciprocal of *Df(1)w^{rg}*.

discoverer: Green, 60h21.

synonym: *Dp(1;1)3C1; Dp(1;1)w^{60h21}*.

references: 1961, Genetics 46: 1555-60.

Gersh, 1962, Genetics 47: 1393-98 (fig.).

1967, Genetics 56: 309-19.

Pearson, 1976, Hereditas 82: 57-62.

genetics: Covers *z* up to and including at least part of *w*.

Dp(1;1)w^{rg2}

cytology: *Dp(1;1)3B2-C1;3C3-5* [Gersh, 1962, Genetics 47: 1393-98 (fig.)].

new order:

1 - 3C3|3C1 - 20.

origin: Spontaneous by recombination.

discoverer: Green, 60h30.

synonym: *Dp(1;1)w^{60h30}*.

references: CP627.

***Dp(1;1)w^{rJ2}*: Duplication (1;1)
white-recombinant of Judd**

cytology: *Dp(1;1)3A6-8;3C1-3* [could be same as *Dp(1;1)w^{rG}*].

new order:

1 - 3C1|3A8 - 20.

origin: A regular product of unequal exchange between the 3C1-3 region of a chromosome carrying *w^{rdp}* with the 3A4-8 region of specific homologs. Probably reciprocal recombinant of *Df(1)w^{rJ2}*.

discoverer: Judd, 1961.

synonym: Mutant locus designated *w^{r,dup}*.

references: 1961, Proc. Nat. Acad. Sci. USA 47: 545-50.

Dp(1;1)w^{sp}w^{17G}

cytology: *Dp(1;1)3A1-2;3C3-4*; tandem repeat of entire *w* locus.

origin: Unequal crossing over between *X* marked with *w^{sp}* and *X* marked with *w^{17G}*.

synonym: *Dp(w^{sp};w^{17G})*.

references: Green, 1973, Genetics Supplement 73: 187-94.

Rasmuson, Green, and Karlsson, 1974, Mol. Gen. Genet. 133: 237-47.

Rasmuson and Green, 1974, Mol. Gen. Genet. 133: 249-60.

Rasmuson, Montell, Rasmuson, Svahlin, and Westerberg, 1980, Mol. Gen. Genet. 177: 567-70.

genetics: Complementary (maroon) eye color in males resulting from interaction between markers *w^{sp}* and *w^{17G}*. Unstable; duplication stocks carrying *z* produce *w⁺* males which, in turn, produce males with *z* eyes, white deficiencies, and transpositions of the *w* locus to other chromosomes.

***Dp(1;1)y^{bl}*: Duplication (1;1) yellow-bristle**

cytology: *Dp(1;1)1B2-3;4F8-9;5D4-5* (Nicoletti, Lindsley).

new order:

1A - 1B2|5D4 - 4F9|1B3 - 20.

origin: Spontaneous.

discoverer: Sandler.

references: Sandler, Hart, and Nicoletti, 1960, DIS 34: 103-4.

genetics: Mutant for *y*; duplicated for *cv*. Regularly generates further rearrangements; has produced losses of the duplicated segment, which are accompanied by changes in phenotype from *y^{bl}* to *y*-like and a translocation between the tips of *X* and *2L* accompanied by a change from *y^{bl}* to *y⁺*.

molecular biology: Three discontinuities in DNA sequence at coordinates 40.8-41.5, 53.2-55.0, and 63.3-65.5 (Biessmann, 1985, Proc. Nat. Acad. Sci. USA 82: 7369-73).

****Dp(1;1)z*: Duplication (1;1) zeste**

origin: X ray induced.

references: Gans, 1953, Bull. Biol. Fr. Belg., (Suppl.)

38: 1-90.

duplication	cytology	new order	genetics
* <i>Dp(1;1)z1</i>	1E2-3;4B4-5 tandem repeat	1A-4B4 1E3-20	male lethal
* <i>Dp(1;1)z2</i>	2C10-D1;4D2-4 + 1n(1)z2		
* <i>Dp(1;1)z4^α</i>	2B16-C1;3B-C1	1-3B 3C1-2C1 3C1-20	male viable and fertile; female homozygote viable and semi-fertile male lethal
* <i>Dp(1;1)z8</i>	2B18-C1;4B4-5	1-4B4 2C1-20	male lethal

^α 3C2-3 may also be duplicated (Gersh, 1967, Genetics 56: 309-19).

Dp(1;1)z59d

cytology: *Dp(1;1)2F5-3A1;3A4-5* (Gersh). Tandem duplication of *z*.

new order:

1 - 3A4|3A1 - 20.

origin: X ray induced in *y²su(w^a)z*.

discoverer: Green, 59d15.

references: 1961, Genetics 46: 1555-60.

Gersh, 1962, Genetics 47: 1393-98 (fig.).

Jack and Judd, 1979, Proc. Nat. Acad. Sci. USA 76: 1368-72.

genetics: Contains *z* allele but no *w* gene. When two doses of *w⁺* present, *z⁺/Dp(1;1)z* females have orange mottled eyes while *z⁺/z* females have wild-type eyes (Jack and Judd, 1979).

Dp(1;Y)IAc⁺: see *l(1)IAc⁺Y*

Described in section on *Y* derivatives.

Dp(1;Y)1E

cytology: *YSX.YL* from which all euchromatic genes have been removed except those located in 1A through 1E.

origin: X ray induced.

discoverer: Masterson, 1961.

synonym: *T(1;Y)1E*; *YSsc⁺y.YL* (Ehrlich, 1965).

references: Clancy, 1964, Genetics 50: 241 (abstract).

Ehrlich, 1965, Can. J. Genet. Cytol. 7: 430-32.

Masterson, 1968, DIS 43: 61, 161.

Lucchesi and Bischoff, 1969, Genetics 61: s37-38.

genetics: Carries *y* and wild-type alleles of *ac - su(w^a)*.

Male viable and fertile (Masterson, 1968). Variegates strongly for *dor* and *dor^l* (Lucchesi and Bischoff, 1969).

Dp(1;Y)2E

cytology: *YSX.YL* from which all euchromatic genes have been removed except those between 1A and 2B15 (Belyaeva *et al.*, 1982). [An additional deficiency involving *su(s)⁺ - sta⁺* is reported by Rayle and Hoar (1969) in a *Dp(1;Y)2E* obtained from Clancy.]

discoverer: Masterson, 1961.

synonym: *T(1;Y)2E*.

references: Clancy, 1964, Genetics 50: 241 (abstract).

Masterson, 1968, DIS 43: 61, 161.

Lucchesi and Bischoff, 1969, Genetics 61: s37-38.

Rayle and Hoar, 1969, DIS 44: 94.

Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers and Zhimulev, 1982, DIS 58: 184-90.

genetics: Carries *y* and wild-type alleles of *ac* and *dor* or *l(1)2Bh*; possibly deficient for *su(s) - sta*. Male viable and fertile. Slight variegation for *dor* (Lucchesi and Bischoff, 1969).

Dp(1;Y)59k9-1

cytology: *Dp(1;Y)2A2-B1*.
 references: Rayle and Hoar, 1969, DIS 44: 94.
 genetics: Carries *sta*⁺.

Dp(1;Y)60e17.4-3

cytology: *Dp(1;Y)1E2-4*.
 references: Rayle and Hoar, 1969, DIS 44: 94.
 genetics: Carries *y*⁺ - *su(w^a)*⁺.

Dp(1;Y)68h20

cytology: *Dp(1;Y)2B3-5*.
 references: Rayle and Hoar, 1969, DIS 44: 94.
 genetics: Carries *y*⁺ - *sta*⁺.

Dp(1;Y)B^S_v⁺y⁺: see *B^S_v⁺Yy⁺*
 Described in section on *Y* derivatives.

Dp(1;Y)B^S_w⁺: see *B^S_w⁺Y*
 Described in section on *Y* derivatives.

Dp(1;Y)B^S_w⁺y⁺: see *B^S_w⁺y⁺Y*
 Described in section on *Y* derivatives.

Dp(1;Y)B^S_y^{61d}: see *B^S_y^{61d}Y*
 Described in section on *Y* derivatives.

Dp(1;Y)B^S_y^{67j}: see *B^S_y^{67j}Y*
 Described in section on *Y* derivatives.

Dp(1;Y)B^S_y^{bl2}: see *B^S_y^{bl2}Y*
 Described in section on *Y* derivatives.

Dp(1;Y)B^S_Y: see *v⁺Yy⁺3*
 Described in section on *Y* derivatives.

Dp(1;Y)B^{SV}: see *B^{SV}Y*
 Described in section on *Y* derivatives.

Dp(1;Y)bw⁺: see *bw⁺Y*
 Described in section on *Y* derivatives.

Dp(1;Y)bw⁺y⁺: see *bw⁺Yy⁺*
 Described in section on *Y* derivatives.

Dp(1;Y)cin⁺: see *cin⁺Y*
 Described in section on *Y* derivatives.

Dp(1;Y)ct⁺y⁺: see *ct⁺y⁺Y*
 Described in section on *Y* derivatives.

Dp(1;Y)dor: Duplication (1;Y) deep orange

origin: γ ray induced in attached-XY, *dor* males.
 discoverer: Gorelova.
 synonym: *dorY*.
 references: Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90.

duplication	cytology	genetics
<i>Dp(1;Y)dor1T</i>	1A;2C1-2	
<i>Dp(1;Y)dor3T</i>	1A;2E1-2	
<i>Dp(1;Y)dor6T</i>		<i>y</i> ⁺ - <i>arm</i> ⁺
<i>Dp(1;Y)dor9T</i>		<i>y</i> ⁺ - <i>hfw</i> ⁺
<i>Dp(1;Y)dor10T</i>	1A;2C1-2	
<i>Dp(1;Y)dor13T</i>	1A;2B9-10	<i>y</i> ⁺ - <i>hfw</i> ⁺
<i>Dp(1;Y)dor17T</i>	1A;2C1-2	<i>y</i> ⁺ - <i>arm</i> ⁺
<i>Dp(1;Y)dor18T</i>	1A;2D1-2	
<i>Dp(1;Y)dor21T^α</i>	1A;2B1-2 + 3 thick bands	

^α The three thick condensed bands pair with the 2B3-4 - 2B9-10 bands of the wild type X. No 2B5 puff in this duplication.

Dp(1;Y)dor⁺: see *dor⁺Yy⁺*
 Described in section on *Y* derivatives.

Dp(1;Y)l(1)J1⁺: see *l(1)1Ac⁺Y*
 Described in section on *Y* derivatives.

Dp(1;Y)mal²y⁺: see *y⁺Ymal⁺2*
 Described in section on *Y* derivatives.

Dp(1;Y)mal⁺

cytology: *Dp(1;Y)19A1;20F* (Eberl).
 new order: YL|19A1 - 20F|YS.
 origin: X-ray-induced deletion of majority of euchromatin [l(1)J1⁺ through *car*⁺] from *YSX*·YL, *In(1)EN*.
 discoverer: E.H. Grell.
 synonym: *Ymal⁺*.
 references: Brosseau, Nicoletti, Grell, and Lindsley, 1961, Genetics 46: 339-46.
 Rahman and Lindsley, 1981, DIS 46: 339-46.
 Eberl and Hilliker, 1988, Genetics 118: 109-20.
 genetics: Carries *sw⁺-bb⁺*.

Dp(1;Y)pn⁻w⁺: see *pn⁻w⁺Y*
 Described in section on *Y* derivatives.

Dp(1;Y)sc^{S1}: see *YL·usc^{S1}*
 Described in section on *Y* derivatives.

Dp(1;Y)sc^{V1}: see *sc^{V1}·Y⁺*
 Described in section on *Y* derivatives.

Dp(1;Y)shi⁺: see *shi⁺Y*
 Described in section on *Y* derivatives.

Dp(1;Y)su(f)⁻: see *su(f)⁻Y*
 Described in section on *Y* derivatives.

Dp(1;Y)Sz: Duplication (1;Y) Szeged

origin: X ray induced in Oregon-R (premeiotic?).
 discoverer: Koczka and Kiss.
 synonym: *YSz*.
 references: Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90.

duplication	X cytology	genetics
<i>Dp(1;Y)Sz71/4</i>	1A;2C1-2	<i>y</i> ⁺ - <i>arm</i> ⁺
<i>Dp(1;Y)Sz78/4</i>	1A;2B17-18	<i>y</i> ⁺ - <i>arm</i> ⁺
<i>Dp(1;Y)Sz170</i>	1A;2B17-18	<i>y</i> ⁺ - <i>arm</i> ⁺
<i>Dp(1;Y)Sz280</i>	1A;2C1-2 + <i>Df(1)2B3-4;2B7-8</i>	<i>y</i> ⁺ - <i>sta</i> ⁺ , <i>BRC⁻-hfw⁻</i> , <i>l(1)Bg⁺-arm⁺</i>
<i>Dp(1;Y)Sz303</i>	1A;2B17-18	<i>y</i> ⁺ - <i>arm</i> ⁺

Dp(1;Y)v⁺y⁺: see *v⁺Yy⁺*
 Described in section on *Y* derivatives.

Dp(1;Y)w⁺: see *w⁺Y*
 Described in section on *Y* derivatives.

Dp(1;Y)w⁺303

cytology: *Dp(1;Y)2D1-2;3D3-4*.
 origin: X ray induced.
 discoverer: Lefevre.
 references: Perrimon, Engstrom, and Mahowald, 1984, Genetics 108: 559-72.
 1985, Genetics 111: 23-41.
 genetics: Carries *w⁺* and wild-type alleles of loci distal and proximal to *w⁺*.

Dp(1;Y)w⁺y⁺: see *w⁺y⁺Y*

Described in section on *Y* derivatives.

***Dp(1;Y)y²*: Duplication (1;Y) yellow**

origin: γ ray induced in attached -XY, *y² su(w^a) w^a* males.

discoverer: Gorelova.

synonym: *y²Y*.

duplication	cytology	ref ^α	genetics
<i>Dp(1;Y)y² 21T</i>	1A;2B6(2B7-8?)	1,2	<i>y⁺-hfw⁺</i>
<i>Dp(1;Y)y² 22T</i>	1A;2B9-10	1	<i>y⁺-fmf⁺</i>
<i>Dp(1;Y)y² 40T</i>	1A;2B9-11	1,2	<i>y⁺-fmf⁺</i>
<i>Dp(1;Y)y² 43T</i>	1A;2E1-2	1	
<i>Dp(1;Y)y² 53T</i>	1A;2B6(2B7-8?)	1,2	<i>y⁺-hfw⁺</i>

^α 1 = Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90; 2 = Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelova, and Zhimulev, 1980, Chromosoma 81: 281-306.

Dp(1;Y)y² 61l: see *y²Y61l*

Described in section on *Y* derivatives.

Dp(1;Y)y² 67g: see *y²Y67g*

Described in section on *Y* derivatives.

Dp(1;Y)y²sc

cytology: *Dp(1;Y)1E-F;2B-C;3A*.

new order:

1A1 - 1E-F|2B-C - 3A.

references: Eberl and Hilliker, 1988, Genetics 118: 109-20.

Dp(1;Y)y^{59b}2: see *y^{59b}Y2*

Described in section on *Y* derivatives.

Dp(1;Y)y⁺mal¹: see *y⁺Ymal¹*

Described in section on *Y* derivatives.

Dp(1;Y)y⁺mal²: see *y⁺Ymal²*

Described in section on *Y* derivatives.

Dp(1;Y)y⁺mal⁺102 - Dp(1;Y)y⁺mal⁺126:
see *y⁺Ymal⁺102 - y⁺Ymal⁺126*

Described in section on *Y* derivatives.

Dp(1;Y)y⁺sc: see *y⁺scY*

Described in section on *Y* derivatives.

***Dp(1;Y)y-z*: Duplication (1;Y) yellow-zeste**

references: Gunaratne, Mansukhani, Lipari, Liou, Martindale, and Goldberg, 1986, Proc. Nat. Acad. Sci. USA 83: 701-05.

genetics: Contains wild-type alleles of *gt* and *tko* but not *l(1)3Ac*. *Dp(1;1)w⁺61e19/Dp(1;Y)y-z* males show *z* variegation.

molecular biology: *X* breakpoint localized to an approximately 4-kb restriction fragment 7-8 kb to the right of the origin of a 160-kb walk in the vicinity of *z*.

Dp(1;Y;2)B^S5

cytology: Piece of *Y* carrying *B^S* attached to 2*L*.01 unit to left of locus of *al*.

origin: *X* ray induced in *B^SYy⁺*.

references: Novitski, Ehrlich, and Becker, 1971, DIS 47: 91-92.

genetics: Carries *B^S* and fertility factor *kl-5*; claimed to carry *ks-1* and possibly *kl-4* (= *kl-3?*) as well.

Dp(1;Y;2)bw⁺: see *X bw⁺YL*

Described in section on *XY* combinations.

Dp(1;Y;3)H: see *sc^{VI}·YSmwh⁺*

Described in section on *Y* derivatives.

Dp(1;Y;3)M1-3

cytology: Duplication of 61 to 62A-B, including *mwh⁺*, on tip of *Y**SX* chromosome distal to the *KS* factors.

origin: *X* ray induced in *T(Y;3)P6*, *mwh⁺ ve^{+/} y⁺YSX*, *y⁺KS y cv v f*; *mwh ve* males.

synonym: *mwh⁺YSX*.

references: García-Bellido and Ripoll, 1973, DIS 50: 92.

genetics: Male viable and fertile over *YL* chromosome. Homozygous females lethal. No variegation for the *mwh⁺* of the duplication in nullo-*Y* males.

Dp(1;Y;4)B^S

origin: *X* ray induced in *B^SYy⁺/C(1)RM*, *y² su(wa^a) w^a* *bb* females.

references: Parker, 1965, Mutat. Res. 2: 523-29.

genetics: *Y* carries *B^S* and shows *Y-4* linkage.

Dp(1;Y;4)y⁺

origin: *X* ray induced in *B^SYy⁺/C(1)RM*, *y² su(wa^a) w^a* *bb* females.

references: Parker, 1965, Mutat. Res. 2: 523-29.

genetics: *Y* carries *y⁺* and shows *Y-4* linkage.

Dp(1;2)51b*: see *Tp(1;2)51b

Dp(1;2)51bV76e

cytology: *Dp(1;2)3C1-2;3D6-E1;40-41;52E*.

new order:

21 - 41|3C2 - 3D6|52E - 41|52E - 60 or
21 - 40|52E - 40|3C2 - 3D6|52E - 60.

origin: Spontaneous.

references: Poulson and Lefevre, 1982, Genetics 100: s54-55.

genetics: When duplication covers *l(1)N^B*, *N^{55e}*, *N^{60g11}*, *N²⁶⁴⁻⁴⁰*, *N²⁶⁴⁻¹⁰³*, or *N^{Nic}*, some males show variegated or roughened eyes, abnormal legs, missing or multiple bristles, and missing or fused ocelli.

Dp(1;2)63i*: see *Dp(1;2)v⁺63i

Dp(1;2)65b*: see *Tp(1;2)v^{65b}

Dp(1;2)76f

cytology: Deletion of *N⁺* to *dm⁺* region in *Dp(1;2)51b*. Retains functions of *w⁺ - rst⁺*.

origin: Induced by ethyl methanesulfonate.

references: Welshons and Welshons, 1985, Genetics 110: 465-77.

1986, Genetics 113: 337-54.

genetics: Covers *w*, *rst*, and *vt* but not *N*.

Dp(1;2)(w-ec)^{64d}*: see *Tp(1;2)w-ec

Dp(1;2;3)pn3*: see *Tp(1;2;3)pn3

****Dp(1;2)A12*: see **Tp(1;2)A12***

****Dp(1;2)A124*: see **Tp(1;2)A124***

Dp(1;2)B^S

cytology: Second chromosome carrying fragment of *B^SYy⁺* with the marker *B^S* attached to 2*L* tip.

origin: *X* ray induced in female with *B^SYy⁺*.

references: Novitski, Ehrlich, and Becker, 1971, DIS 47: 91-92.

Novitski, 1976, The Genetics and Biology of Drosophila (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b., pp. 562-68.

Dp(1;2)B^{S3}

cytology: B^S attached to 2L 0.1 unit to left of locus of *al*.
origin: X ray induced in B^SYy⁺.
references: Novitski, Ehrlich, and Becker, 1971, DIS 47: 91-92.
genetics: Homozygous viable and fertile. Carries B^S but no male fertility factors; probably only X-derived material translocated to 2L.

*Dp(1;2)ct^{7c1}: see *Tp(1;2)ct^{7c1}

Dp(1;2)E1

cytology: Dp(1;2)1A-F;60A.
references: Jacobson, Yim, Grell, and Wobbe, 1982, Cell 30: 817-23.
genetics: Carries normal alleles of y⁺ and su(s)⁺.

Dp(1;2)f⁺

cytology: f⁺ inserted near centromere of 2.
origin: X ray induced.
references: Shukla and Auerbach, 1980, Genet. Res. 36: 41-56.
genetics: Variegates for *f*. Transposes to other chromosome sites on 2 or 4.

Dp(1;2)FN107: see Tp(1;2)sn⁺72d

Dp(1;2)Hw^K

cytology: Dp(1;2)1B2-3;1E-F;21C2-3.
new order:
 21A - 21C2|1B3 - 1E|21C3 - 60.
discoverer: Kreber.
references: Craymer.
genetics: Mutant for *Hw*.

Dp(1;2)K1: Duplication (1;2) of Krivshenko

cytology: Dp(1;2)1B1-3;20;29A-B; deficient for 1B3-20. Larval ganglion cells in metaphase show a rod-shaped element, believed to be distal 2L capped with the X centromere, and a J-shaped element, believed to be the X tip attached to the rest of 2 with the 2 centromere.

new order:

1A1 - 1B1|29B - 60;
 20|29A - 21.

origin: X ray induced in Canton-S wild type.
synonym: T(1;2)K1.
references: Krivshenko, 1956, DIS 30: 74-76.
genetics: Bipartite duplication, the 2^PX^D segment carrying y⁺ and the X^P2^D segment carrying bb⁺. Flies hyperploid for the X^P2^D segment survive; flies with a normal X but homozygous for Dp(1;2)K1 die.

Dp(1;2)Nst: see Tp(1;2)w-ec

Dp(1;2)pn1: see Tp(1;2)pn1

Dp(1;2)pn-ec: see Tp(1;2)w^{62g26}

Dp(1;2)r⁺75c: see Tp(1;2)r⁺75c

Dp(1;2)rb⁺71g: see Tp(1;2)rb⁺71g

Dp(1;2)sc¹⁹: see Tp(1;2)sc¹⁹

Dp(1;2)sc²⁶⁰⁻²⁷: see Tp(1;2)sc²⁶⁰⁻²⁷

Dp(1;2)sn⁺72d: see Tp(1;2)sn⁺72d

Dp(1;2)v^{65b}: see Tp(1;2)v^{65b}

Dp(1;2)v⁺63i

cytology: Dp(1;2)9E1;10A11;56A.
origin: X ray induced.
discoverer: Lefevre.
synonym: Dp(1;2)63i; Tp(1;2)v⁺63i.
references: 1969, Genetics 63: 589-600.
 Hall and Kankel, 1976, Genetics 83: 517-35.
 Craymer and Roy, 1980, DIS 55: 200-04.
 Lefevre, 1981, Genetics 99: 461-80.
 Zhimulev, Belyaeva, Pokholkova, Kotchneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1981, DIS 56: 192-96.
genetics: Covers *ras - l(1)10Ag*, including v. *Rp11215* not included.

Dp(1;2)v⁺75d: see Tp(1;2)v⁺75d

Dp(1;2)w^{51b}: see Tp(1;2)51b

Dp(1;2)w⁺51b7: see Tp(1;2)51b

Dp(1;2)w⁺62g26: see *Tp(1;2)w⁺62g26

Dp(1;2)w⁺70h

cytology: Dp(1;2)3A7-8;3C2-3;3IA3.
discoverer: Green.
references: Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56.
 Jack and Judd, 1979, Proc. Nat. Acad. Sci. USA 76: 1368-72.
genetics: Covers *mit(1)15 - w*.
molecular biology: Right breakpoint some 28 kb to the right of the w^a copia insertion point (Pirrota, Hadfield, and Pretorius, 1983, EMBO J. 2: 927-34).

*Dp(1;2)w^{m52b}: see *Tp(1;2)w^{m52b}

*Dp(1;2)w^{m53a}: see *Tp(1;2)w^{m53a}

*Dp(1;2)w^{m258-44}: see *Tp(1;2;3)w^{m258-44}

Dp(1;2)w-ec: see Tp(1;2)w-ec

Dp(1;2;3)pn3: see Tp(1;2;3)pn3

Dp(1;3)51

cytology: X broken distally and tip attached to centric part of 3; also broken proximally and centromere of X attached to acentric part of 3. Interstitial part of X without centromere lost. Third-chromosome breakpoint not determined.

origin: X ray induced.

discoverer: Weltman.

references: Lindsley and Sandler, 1958, Genetics 43: 547-63.

genetics: Bipartite duplication, the 3^PX^D segment carrying sc⁺-su(w^a)⁺ and the X^P3^D segment carrying bb⁺.

Dp(1;3)126

origin: X ray induced.

discoverer: Dobzhansky, 1930.

references: 1935, Z. Indukt. Abstamm. Vererbungsl. 68: 143.

genetics: Duplicated for *r*, *M(1)15D*, *f*, and *B* but not *sl* or *os*; variegates for *f* and *M(1)15D* (Schultz). Duplicated section inserted into chromosome 3 between *st* and *cu*. Also an inversion in 3L. Viability, fertility, and phenotype of Dp(1;3)126/+ male and female normal.

Dp(1;3)142

cytology: X broken distally and tip attached to centric part of 3; also broken proximally and centromere of X attached to acentric part of 3. Interstitial part of X without centromere lost. Third-chromosome breakpoint not determined.

origin: X ray induced.

discoverer: Weltman.

references: Lindsley and Sandler, 1958, *Genetics* 43: 547-63.

genetics: Bipartite duplication, the $3^P X^D$ segment carrying $sc^+ - su(w^a)^+$ and the $X^P 3^D$ segment carrying $su(f)^+ - bb^+$.

Dp(1;3)B^S

cytology: Duplication with B^S and some heterochromatin on 3L tip; B^S located to left of *ru*.

origin: X ray induced in $B^S Y^+$.

discoverer: Leigh.

references: Novitski, Grace, and Strommen, 1981, *Genetics* 98: 257-73.

Puro and Novitski, 1982, *DIS* 58: 126.

genetics: Carries B^S . Female homozygotes viable and fertile, male homozygotes viable but sterile. Used in construction of *C(3)EN* (Novitski *et al.*, 1981).

Dp(1;3)B^{S3i}: see **Tp(1;3)B^{S3i}**

***Dp(1;3)ct^{11a}:** see ***Tp(1;3)ct^{11a}**

***Dp(1;3)ct^{12c1}:** see ***Tp(1;3)ct^{12c1}**

***Dp(1;3)ct²⁶⁸⁻³⁷:** see ***Tp(1;3)ct²⁶⁸⁻³⁷**

Dp(1;3)ct^{J8}: see **Tp(1;3)ct^{J8}**

Dp(1;3)D^{spot}: see **Tp(1;3)N²⁶⁴⁻⁵⁸**

Dp(1;3)E1

cytology: *Dp(1;2)1A-F;61A*.

references: Jacobson, Yim, Grell, and Wobbe, 1982, *Cell* 30: 817-23.

genetics: Carries normal alleles of genes from the tip of X through *brc* (Voelker).

Dp(1;3)f⁺71b: see **Tp(1;3)f⁺71b**

Dp(1;3)G^{spot}: see **Dp(1;3)w^{67k27}**

Dp(1;3)in^{61j2}: Duplication (1;3) inturned

cytology: *Dp(1;3)20;77B-C*. Nucleolus moved to *in* region.

origin: X ray induced.

references: Arajjarvi and Hannah-Alava, 1969, *DIS* 44: 73-74.

Hannah-Alava, 1971, *Mol. Gen. Genet.* 113: 191-203.

genetics: Homozygous lethal, and lethal with some *in* alleles induced by X ray. Expression of *in* variable with other *in* alleles, becoming more nearly wild type with addition of a Y (Hannah-Alava, 1971).

Dp(1;3)JC153: see **Tp(1;3)JC153**

Dp(1;3)K2: Duplication (1;3) of Krivshenko

cytology: *Dp(1;3)20A-B;20D-F;80-81* superimposed on *In(1)1B2-3;20B-D1*. Inferred from genetic data since salivary chromosomes do not reveal an aberration. In ganglial metaphase, chromosome 3 is a rod-shaped and a J-shaped element.

new order:

1A – 1B2|20B|80 – 100;

20F|80 – 61.

Tentative.

origin: X ray induced in *In(1)sc⁸*.

synonym: *T(1;3)K2*.

references: Krivshenko, 1956, *DIS* 30: 76.

genetics: Bipartite duplication. Irradiated *In(1)sc⁸* broken in distal region between y^+ and bb^+ and also near the centromere. Chromosome 3 broken near the centromere, whether to left or right of the centromere is not known. Tip of X chromosome with y^+ and ac^+ is attached to the chromosome 3 centromere, and one arm of this chromosome is attached to the X centromere. Bulk of the X chromosome is thus acentric and lost. Homozygote viable and moderately fertile.

Dp(1;3)L^{spot}: see **Dp(1;3)w^{m49a}**

Dp(1;3)m⁺84f

cytology: *Dp(1;3)10B;10F-11A;98*.

new order:

61 – 98|10B – 10F|98 – 100.

origin: Spontaneous.

discoverer: Voelker.

genetics: Covers *Df(1)m259-4* and therefore includes wild-type alleles of *tyl* and *RpII215* (Voelker). Exists only as the duplication (deficiency segregant lost).

Dp(1;3)MNB-8

cytology: Breaks near the two 16F regions of *Dp(1;1)MNB-8* with resulting acentric fragment inserted between 67E1 and 67E7 of 3; deficient X lost.

new order:

61A – 67E|16F – 16A1|16A7-

16A1|17E1 – 17A1|67E – 100.

origin: Spontaneous derivative of *Dp(1;1)MNB-8*.

references: Holmquist, 1972, *Chromosoma* 36: 413-52.

genetics: Carries *B os⁺*.

Dp(1;3)N^{50k}: see **Tp(1;3)N^{50k}**

Dp(1;3)N²⁶⁴⁻⁵⁸: see **Tp(1;3)N²⁶⁴⁻⁵⁸**

***Dp(1;3)N²⁶⁴⁻¹⁰⁰:** see ***Tp(1;3)N²⁶⁴⁻¹⁰⁰**

Dp(1;3)O4: see **Tp(1;3)O4**

Dp(1;3)pn25: see **Tp(1;3)pn25**

Dp(1;3)pn26: see **Tp(1;3)pn26**

Dp(1;3)ras^v: see **Tp(1;3)ras^v**

***Dp(1;3)sc²⁶⁰⁻²⁰:** see ***T(1;3)sc²⁶⁰⁻²⁰**

Dp(1;3)sc^{J4}: see **T(1;3)sc^{J4}**

Dp(1;3)sn^{13a1}: see **Tp(1;3)sn^{13a1}**

Dp(1;3)sta: see **Tp(1;3)sta**

Dp(1;3)v⁺74c: see **Tp(1;3)v⁺74c**

***Dp(1;3)w⁺54a4:** see ***Tp(1;3)w⁺54a4**

***Dp(1;3)w⁺54c10:** see ***Tp(1;3)w⁺54c10**

Dp(1;3)w⁺67k: see **Tp(1;3)w⁺67k**

Dp(1;3)w^{halo}: see **Tp(1;3)w^{halo}**

Dp(1;3)w^{m49a}: see **Tp(1;3)w^{m49a}**

Dp(1;3)w^{m264-58}: see *Dp(1;3)N²⁶⁴⁻⁵⁸*

Dp(1;3)w^{vco}: see *Tp(1;3)w^{vco}*

Dp(1;3)w^{zh}: see *Tp(1;3)w^{zh}*

Dp(1;3)z7: see *T(1;3)z⁷*

Dp(1;3;4)7

cytology: *Dp(3;4)62E;78D;79F;102F* + *Dp(1;3)1B;61A* from *T(1;3)sc¹⁴*

new order:

1A - 1B|61A - 62F|(78D - 79F)|102F - 101.

references: Duncan and Lewis, 1982, Symp. Soc. Dev. Biol., 40th, pp. 533-54.

phenotype: Covers homozygous lethal *Pc* mutants.

Dp(1;4)20G1L

cytology: Duplication for bands 1A1 to 3C2 translocated to 4. 3C1 only may be included (Gans, 1953) or 3C1 and 3C2 (Gersh, 1967).

references: Gans, 1953, Bull. Biol. Fr. Belg. (Suppl.) 38: 1-90.

Gersh, 1967, Genetics 56: 309-19.

Dp(1;4)174

cytology: Duplication linked with 4 but cytology not worked out.

origin: X ray induced.

discoverer: Weltman.

references: Lindsley and Sandler, 1958, Genetics 43: 547-63.

genetics: Probably bipartite duplication like *Dp(1;3)51*. Carries *sc⁺* but not *bb⁺*.

Dp(1;4)193

cytology: X tip attached to 4 at left end.

origin: Derived from *Dp(1;4)1021*.

synonym: *y⁺.spa^{pol}*.

references: Williamson, Parker, and Manchester, 1970, Mutat. Res. 9: 287-97, 299-306.

Parker, 1970, Mutat. Res. 9: 307-32.

genetics: Carries *y⁺* and *ac⁺* as well as *ci⁺*, *ey⁺*, and *spa^{pol}*. (Williamson *et al.*, 1970). Shows 66% segregation from the compound X in *C(1)RM* females without a Y.

Dp(1;4)1021

cytology: Heterochromatic left arm of 4 capped by euchromatic tip of X.

references: Parker, 1969, Mutat. Res. 7: 393-407.

Parker and Busby, 1973, Mutat. Res. 18: 33-46.

O'Tousa, 1982, Genetics 102: 503-04.

genetics: Carries *y⁺* and *ac⁺*.

****Dp(1;4)A1***: see ****Tp(1;4)A1***

****Dp(1;4)A12***: see ****Tp(1;4)A12***

***Dp(1;4)exd⁺*: Duplication extradenticle wild type**

discoverer: D. Falk.

references: Wieschaus, Nüsslein-Volhard, and Jürgens, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 296-307.

genetics: Covers *exd*.

duplication	cytology	synonym
<i>Dp(1;4)exd^{81h24b}</i>	13F;14B5-18;14E1-4;16A2	<i>Dp(1;4)81h24b</i>
<i>Dp(1;4)exd^{82b26c}</i>	13F;14D1-2;15A3-5;16A2	<i>Dp(1;4)82b26c</i>

other information: Also described as *Df(1)81h24b* and *Df(1)82b26c*.

Dp(1;4)mg

cytology: *Dp(1;4)2B7-C1;3C1-2;102F*.

discoverer: Green.

references: Robbins, 1977, Genetics 87: 655-84.

1980, Genetics 96: 187-200.

1984, Genetics 108: 361-75.

genetics: Covers lethals between *z* and *w*; includes *pn⁺* but is *w* or *w⁻*. *Dp(1;4)mg* hyperploids have reduced viability and homozygotes having an otherwise normal genotype are lethal. Homozygotes survive if the fly is heterozygous, homozygous or hemizygous for deficiencies in the *z-w* region. Viability of *Dp(1;4)mg* hyperploids improves at lower temperatures and in XO males, suggesting position-effect-variegation of the transposed genes (Robbins).

****Dp(1;4)N²⁶⁴⁻⁸⁵***: see ****Tp(1;2;4)N²⁶⁴⁻⁸⁵***

****Dp(1;4)N²⁶⁴⁻⁸⁶***: see ****Tp(1;4)N²⁶⁴⁻⁸⁶***

***Dp(1;4)r⁺*: Duplication (1;4) rudimentary - wild type**

cytology: *Dp(1;4)14A1-2;16A7-B1;102F2-3*.

origin: X-ray-induced derivative of *T(1;4)B^S = T(1;4)15F9-16A1;16A7-B1;102F2-3* of most of the X euchromatin from the 4^PX^D element deleted; likely to carry genes from X terminus.

new order:

1A|14A2 - 16A7|102F2 - 101.

discoverer: Green.

synonym: *Dp(1;4)r⁺f⁺*.

references: 1963, Genetica 34: 242-53.

Jarry, 1979, Mol. Gen. Genet. 172: 199-202.

Craymer and Roy, 1980, DIS 55: 200-04.

Naguib and Jarry, 1981, Genet. Res. 37: 199-207.

Ganetzky and Wu, 1982, Genetics 100: 597-614.

Falk, Roselli, Curtis, Holliday, and Klufas, 1984, Mutat. Res. 126: 25-34.

Ganetzky, 1984, Genetics 108: 897-911.

Kulkarni, Steinlauf, and Hall, 1988, Genetics 118: 267-85.

genetics: Carries normal alleles of *exd*, *para*, *r* and *f* appended to the right end of chromosome 4. Usually lethal in two doses.

Dp(1;4)w^{m5}: see ***T(1;4)w^{m5}***

****Dp(1;4)w^{m51c}***: see ****Tp(1;4)w^{m51c}***

Dp(1;4)w^{m65g}

cytology: *Dp(1;4)3B1-2;3C4-5;101*.

origin: X ray induced.

references: Lefevre, 1968, DIS 43: 62-63, 165.

Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56.

Robbins, 1980, Genetics 96: 187-200.

1984, Genetics 108: 361-75.

genetics: Contains wild-type alleles of *l(1)3Bb-rst*. Covers *Df(1)w^{m4Lrst^{3R}}* and *Df(1)w^{64d8}* lethality. *Df(1)w²⁵⁸⁻⁴⁵*; *Dp(1;4)w^{m65g}* male zygotes are lethal in

the absence of a *Y*. Duplication shows variegation, *XO* males carrying *w* and *Dp(1;4)^{m65g}* having almost white eyes (Robbins, 1980).

***Dp(1;4)w^{VD1}*: Duplication (1;4)
white-variegated of Demerec**

cytology: *Dp(1;4)3C1-4;101A-D*.

origin: X ray induced in *y*.

discoverer: Demerec, 33j19.

references: CP627.

genetics: Variegated for *w* but not *ci*. *X* broken between *w* and *rst*; 4 probably broken in left arm.

****Dp(1;f)1*: Duplication (1;free)**

origin: X-ray-induced deletion of most of *X* euchromatin.

discoverer: Muller.

synonym: *Del(1)1*.

references: Painter and Muller, 1929, *J. Heredity* 20: 287-98.

Muller and Painter, 1932, *Z. Indukt. Abstamm. Vererbungsl.* 62: 316-65.

genetics: Contains wild-type alleles of *y*, *sc*, *br*, *pn*, and *bb*.

****Dp(1;f)2***

origin: X-ray-induced deletion of most of *X* euchromatin.

discoverer: Muller.

synonym: *Del(1)2*.

references: Painter and Muller, 1929, *J. Heredity* 20: 287-98.

Muller and Painter, 1932, *Z. Indukt. Abstamm. Vererbungsl.* 62: 316-65.

genetics: Contains wild-type alleles of *y*, *sc*, *br*, and *bb*.

Dp(1;f)3

cytology: *Dp(1;f)1D4-E1;20D*. 4 times the size of chromosome 4 at metaphase.

new order:

1A – 1D3|20A – 20F.

origin: X-ray-induced deletion of most of *X* euchromatin.

discoverer: Weltman, 1954.

references: Lindsley and Sandler, 1958, *Genetics* 43: 547-63.

Grell, 1964, *Genetics* 50: 151-66.

Rayle and Hoar, 1969, *DIS* 44: 94.

Schalet and Lefevre, 1973, *Chromosoma* 44: 183-200.

Sandler and O'Tousa, 1979, *Genetics* 91: 537-51.

genetics: Carries wild-type alleles of *y-tw* and *wap-bb*. Includes *ABO-X* (Parry and Sandler, 1974, *Genetics* 77: 535-39). Disjoins regularly from *XY*, shows 3% nondisjunction from *C(1)RM*, and causes 18% nondisjunction of *In(1)dl-49* from + in *In(1)dl-49/+/Dp(1;f)3* female.

Dp(1;f)3bb⁻

cytology: *Dp(1;f)1D3-E1;20A-F*. 2 times size of chromosome 4 at metaphases; lacks *NO*.

origin: X ray induced in *Dp(1;f)3*.

discoverer: Parry.

references: Sandler and O'Tousa, 1979, *Genetics* 91: 537-51.

genetics: Deficient for *bb*.

****Dp(1;f)10***

origin: X-ray-induced deletion of most of *X* euchromatin.

discoverer: Weltman, 1954.

references: Lindsley and Sandler, 1958, *Genetics*

43: 547-63.

genetics: Carries wild-type alleles of *y*, *ac*, and *sc* but not *su(f)* or *bb*. Sixty-one percent nondisjunction from *XY*, 45% from *C(1)RM*, and regular disjunction of + from *In(1)dl-49* in *In(1)dl-49/+/Dp(1;f)10* female.

****Dp(1;f)12***

cytology: 3.4-4 times the size of chromosome 4 at metaphase; lacks only the distal-most part of heterochromatic segment *hD* (Cooper).

origin: X-ray-induced deletion of most of *X* euchromatin.

discoverer: Weltman, 1954.

references: Lindsley and Sandler, 1958, *Genetics* 43: 547-63.

genetics: Contains wild-type alleles of *y*, *ac*, *sc*, *su(w^a)*, *su(f)*, and *bb* but not *pn* or *car*. Disjoins regularly from *XY*, shows 4% nondisjunction from *C(1)RM*, and causes 19% nondisjunction of + from *In(1)dl-49* in *In(1)dl-49/+/Dp(1;f)12* female.

****Dp(1;f)14***

origin: X-ray-induced deletion of most of *X* euchromatin.

discoverer: Muller.

synonym: *Del(1)14*.

references: Painter and Muller, 1929, *J. Heredity* 20: 287-98.

Muller and Painter, 1932, *Z. Indukt. Abstamm. Vererbungsl.* 62: 316-65.

genetics: Contains wild-type alleles of *y*, *sc*, and *bb* but not *br*.

Dp(1;f)18

cytology: *Dp(1;f)1E4-F1;19-20*.

origin: X-ray-induced deletion of most of *X* euchromatin.

discoverer: Weltman, 1954.

references: Lindsley and Sandler, 1958, *Genetics* 43: 547-63.

Rayle and Hoar, 1969, *DIS* 44: 94.

Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, *DIS* 58: 184-90.

genetics: Contains wild-type alleles of *y-l(1)1Ec* and *bb* but not *l(1)Ed* (Voelker) or *car*.

Dp(1;f)24

cytology: *Dp(1;f)1B5;19-20* (Lefevre).

origin: X-ray-induced deletion of most of *X* euchromatin.

discoverer: Muller.

synonym: *Del(1)24*.

references: 1932, *Proc. Intern. Congr. Genet.*, 6th., Vol. 1: 213-55.

García-Bellido, 1979, *Genetics* 91: 491-520.

White, Decelles, and Endow, 1983, *Genetics* 104: 433-48.

genetics: Mutant for *sc* and carries wild-type alleles of *l(1)1Ac*, *y*, *ac*, and *l(1)Bb* but not *l(1)Ec*.

Dp(1;f)52

cytology: *Dp(1;f)1B10-C4;19-20* (Gersh). 3.7-4 times the size of chromosome 4 at metaphase; lacks only the distal-most part of heterochromatic segment *hD* (Cooper).

origin: X-ray-induced deletion of *X* euchromatin.

discoverer: Weltman, 1954.

references: Lindsley and Sandler, 1958, *Genetics* 43: 547-63.

Schalet and Finnerty, 1968, *DIS* 43: 128-29.

genetics: Contains wild-type alleles of *y*, *ac*, *sc*, *su(f)*, and

bb but not *su(w^a)*, *pn*, or *car*. Segregates normally from *XY*, shows 3% nondisjunction from *C(1)RM*, and causes 13% nondisjunction of + from *In(1)dl-49* in *In(1)dl-49/+/Dp(1:f)52*.

Dp(1:f)60g

origin: A spontaneous exchange between the distally located heterochromatin of *In(1)sc⁸, y^{31d}*, and the proximal heterochromatin of a normal *X*. Occurred in a triploid female.

discoverer: Mohler, 60g.

references: 1960, DIS 34: 52.

genetics: Carries *y^{31d}*, *ac⁺*, and probably two copies of *su(f)⁺* but not *car⁺*.

other information: The reciprocal product, a reversed acrocentric compound *X [C(1)RA60g]* was recovered from the same fly.

Dp(1:f)65X^{C2}

origin: X ray induced in *R(1)2* males.

discoverer: Thompson, 1965.

references: Gethman, 1967, DIS 42: 39, 70.

genetics: Carries wild-type alleles of *y*, *ac*, *sc*, and *su(f)* but not *pn* or *car*. Covers deficiency of *C(1)RA60g*. Somatic unstable; mosaics frequent. Lethal in 30% of the zygotes.

***Dp(1:f)100**

cytology: Two-thirds the length of normal *X* at metaphase.

origin: Spontaneous deletion of most of *X* euchromatin.

discoverer: L. V. Morgan, 221.

synonym: *sc-Dp*.

references: 1938, Genetics 23: 423-62.

genetics: Contains wild-type alleles of *y-pn* and *fu-bb*. Phenotype of duplication-bearing female nearly wild type, but occipital bristles and hairs are present, eyes are a trifle smaller and rougher, and wings have straighter outer margins and sometimes scalloped inner margins. In male, duplication more than 99% lethal.

Dp(1:f)101

cytology: *Dp(1:f)2A2;20E-F* (Lefevre); *Df(1:f)2B1-2;19F5-20A* (Belyaeva *et al.*, 1982); one-fourth the length of normal *X* at metaphase.

new order:

1A – 2A2|20F.

origin: X-ray-induced deletion of most of *X* euchromatin.

discoverer: Dobzhansky, 1930.

references: 1932, Tr. Lab. Genet. (Leningrad) 9: 193-216.

1935, Z. Indukt. Abstamm. Vererbungsl. 68: 134-62.

Rayle and Hoar, 1969, DIS 44: 95.

Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90.

genetics: Contains wild-type alleles of *y-l(1)IFc* and *bb* but not *l(1)IFd* (Voelker) or *su(f)*. With duplication, both sexes viable and wild type except for presence of occipital bristles.

molecular biology: DNA breakpoint (2B1-2) between coordinates 31.3 and 35.3 (Chao and Guild, 1986, EMBO J. 5: 143-50).

***Dp(1:f)102**

cytology: One-fifth the length of normal *X* at metaphase.

origin: X-ray-induced deletion of most of *X* euchromatin.

discoverer: Dobzhansky, 1930.

references: 1932, Biol. Zentralbl. 52: 493-509.

1935, Z. Indukt. Abstamm. Vererbungsl. 68: 134-62.

genetics: Contains *y⁺* to *rb⁺* inclusive and not *bb⁺*. Usually male lethal, but female survives and has occipital bristles, narrow parallel-sided wings, branched posterior crossveins, and heavier bristles on thorax.

***Dp(1:f)106**

cytology: Metaphase length about 4 times that of chromosome 4.

origin: X-ray-induced deletion of most of *X* euchromatin.

discoverer: Dobzhansky, 1930.

references: 1932, Biol. Zentralbl. 52: 493-509.

genetics: Contains wild-type alleles of *y*, *sc*, and *svr* but not *bb*.

Dp(1:f)107

cytology: Metaphase length about one-fifth that of a normal *X*.

origin: X-ray-induced deletion of most of *X* euchromatin.

discoverer: Dobzhansky, 1930.

references: 1932, Biol. Zentralbl. 52: 493-509.

genetics: Contains wild-type alleles of *y*, *sc*, *svr*, *su(s)* (Voelker) and *bb*.

Dp(1:f)112

cytology: *Dp(1:f)1E4-F1;19-20* (Gersh); slightly longer than chromosome 4 at metaphase.

origin: X-ray-induced deletion of most of *X* euchromatin.

discoverer: Dobzhansky, 1930.

references: 1932, Biol. Zentralbl. 52: 493-509.

Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90.

genetics: Contains wild-type alleles of *y-l(1)IEc* but not *l(1)IEd* or *bb*. Both sexes viable and have occipital bristles.

Dp(1:f)118

cytology: About one-fourth the length of normal *X* at metaphase.

origin: X-ray-induced deletion of most of *X* euchromatin.

discoverer: Dobzhansky, 1930.

references: 1932, Biol. Zentralbl. 52: 493-509.

genetics: Contains wild-type alleles of *y*, *sc*, *svr*, *su(s)* (Voelker) and *bb* but not *kz*.

Dp(1:f)118YM

cytology: About size of chromosomes 4 at metaphase.

origin: Presumably same as *Dp(1:f)118*.

references: Yamamoto and Miklos, 1977, Chromosoma 60: 283-96.

genetics: Contains wild-type alleles of *y*, *sc*, and *svr* but not *bb* (Yamamoto and Miklos, 1977).

Dp(1:f)122

cytology: *Dp(1:f)1E4-F1;19-20* (Gersh).

origin: X-ray-induced deletion of most of *X* euchromatin.

discoverer: Weltman, 1954.

references: Lindsley and Sandler, 1958, Genetics 43: 547-63.

La Volpe, La Mantis, Gargiulo, and Malva, 1984, Mol. Gen. Genet. 194: 485-88.

genetics: Carries wild-type alleles of *y*, *ac*, *sc*, and *bb* but not *su(w^a)*, *pn*, *car*, or *su(f)*. Disjoins regularly from *XY*, shows 6% nondisjunction from *C(1)RM*, and causes 9% nondisjunction of + from *In(1)dl-49* in *In(1)dl-49/+/Dp(1:f)122*.

Dp(1;f)134-Dp(1;f)137

origin: X-ray-induced deletion of most of euchromatin from X chromosome marked with y^2 or y^+ .
discoverer: Dobzhansky.

duplication	cytology	ref ^α	genetics
*Dp(1;f)134 ^β	deletion of most of X euchromatin	2	$y^+ - br^+$; not bb^+
Dp(1;f)135 ^β	deletion of most of X euchromatin	3	$y^2 - su(s)^+$ (Voelker); bb^+
*Dp(1;f)136 ^γ	metaphase length 1/4 normal X	1	$y^2 - pn^+$; bb^+
*Dp(1;f)137	metaphase length 1/5 normal X	1	$y^2 - w^+$; not bb^+

^α 1 = Dobzhansky, 1932, Biol. Zentralbl. 52: 493-509; 2 = Dobzhansky, 1935, Z. Indukt. Abstamm. Vererbungsl. 68: 134-62; 3 = Sivertzev-Dobzhansky and Dobzhansky, 1933, Genetics 18: 173-92.
^β Both sexes viable and wild type except for presence of occipital bristles.
^γ Viability low; shows spread wings and occipital bristles.

Dp(1;f)164

cytology: Dp(1;f)1B;20F (Gersh). Approximate size of chromosome 4 at metaphase (Yamamoto and Miklos, 1977).

origin: X-ray-induced deletion of most of X euchromatin.

discoverer: Weltman, 1954.

references: Lindsley and Sandler, 1958, Genetics 43: 547-63.

Yamamoto and Miklos, 1977, Chromosoma 60: 283-96.

genetics: Carries wild-type alleles of y and ac but not sc , $su(w^a)$, pn , car , $su(f)$, or bb . Disjoins essentially randomly from XY, shows 36% nondisjunction from C(1)RM, and does not interfere with disjunction of + from *In(1)dl-49* in *In(1)dl-49/+|Dp(1;f)164* female.

***Dp(1;f)167**

cytology: 3.7-4 times the size of chromosome 4 at metaphase; lacks only the distalmost heterochromatic segment *hD* (Cooper).

origin: X-ray-induced deletion of most of X euchromatin.

discoverer: Weltman, 1954.

references: Lindsley and Sandler, 1958, Genetics 43: 547-63.

genetics: Carries wild-type alleles of y , ac , sc , $su(w^a)$, $su(f)$, and bb but not pn or car . Disjoins regularly from XY, shows 3% nondisjunction from C(1)RM, and causes 16% nondisjunction of + from *In(1)dl-49* in *In(1)dl-49/+|Dp(1;f)167* female.

Dp(1;f)179

origin: X-ray-induced deletion of most of X euchromatin.

discoverer: Weltman, 1954.

references: Lindsley and Sandler, 1958, Genetics 43: 547-63.

genetics: Carries wild-type alleles of y , ac , sc , and $su(w^a)$ but not pn , car , $su(f)$, or bb . Disjoins regularly from XY, shows 20% nondisjunction from C(1)RM, and causes 2% nondisjunction of + from *In(1)dl-49* in *In(1)dl-49/+|Dp(1;f)179* female.

Dp(1;f)749 - Dp(1;f)1518

origin: X ray induced deletion of most of X euchromatin from *In(1)sc*^δ.

discoverer: Krivshenko and Cooper, 1953.

duplication	cytology	length as multiple of 4	ref ^α	genetics
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duplication	cytology	length as multiple of 4	ref ^α	genetics
*Dp(1;f)749	1B12-13;20	3-4		$y^+ - svr^+$, bb^+
Dp(1;f)797	2B4-5;20	2-3		$y^+ - svr^+$, bb^+
Dp(1;f)816	not visible	0.7	2, 3	$y^+ - sc^+$, bb^+
Dp(1;f)819	1D3-4;20	2.9		$y^+ - svr^+$, bb^+
Dp(1;f)856	1D3-4;20	3	2, 3, 4, 5	$y^+ - svr^+$, bb^+
Dp(1;f)1144	not visible	1.1	2, 3, 5	$y^+ - ac^+$, bb^-
Dp(1;f)1148		2		$y^+ - ac^+$, bb^+
Dp(1;f)1156		2.6		$y^+ - ac^+$, bb^+
Dp(1;f)1158		2.3		$y^+ - ac^+$, bb^+
Dp(1;f)1159		2.7		$y^+ - ac^+$, bb^+
Dp(1;f)1160		3.1		$y^+ - ac^+$, bb^+
Dp(1;f)1162		0.5	2, 3	$y^+ - ac^+$, bb^-
Dp(1;f)1170		1.9		$y^+ - ac^+$, bb^-
Dp(1;f)1173		3.2-3.6	5, 6	$y^+ - ac^+$, bb^+
Dp(1;f)1185		1.8		$y^+ - ac^+$, bb^+
Dp(1;f)1186		1.6	2, 3	$y^+ - ac^+$, bb^-
Dp(1;f)1187		>0.3 ^β	2, 3, 5	$y^+ - ac^+$, bb^-
Dp(1;f)1191		0.7		$y^+ - ac^+$, bb^-
Dp(1;f)1193		1	2, 3	$y^+ - ac^+$, bb^-
Dp(1;f)1194		3.1		$y^+ - ac^+$, bb^+
Dp(1;f)1201		2.2		$y^+ - ac^+$, bb^+
Dp(1;f)1204		0.9	2, 3	$y^+ - ac^+$, bb^-
Dp(1;f)1205		0.7		y^+ , bb^-
Dp(1;f)1206		0.5		y^+ , bb^-
Dp(1;f)1208		2		$y^+ - ac^+$, bb^+
Dp(1;f)1209		1.9		$y^+ - ac^+$, bb^+
Dp(1;f)1328 ^γ	2A2-3;20	2.1	2, 3	$y^+ - su(w^a)^+$, bb^+
*Dp(1;f)1330	2B10-11;20	2.6		$y^+ - svr^+$, bb^+
*Dp(1;f)1331	1E-F;20	1.9		$y^+ - svr^+$, bb^+
Dp(1;f)1337 ^δ	2B8-9;19-20	1.4	1, 2, 3, 7	$y^+ - hfw^+$, bb^-
Dp(1;f)1339	1D-E;20	1.1	2, 3	$y^+ - su(w^a)^+$, bb^-
*Dp(1;f)1341	2C-D;20	>3		$y^+ - svr^+$, bb^+
Dp(1;f)1342		>3		$y^+ - svr^+$, bb^+
Dp(1;f)1343	1F;20	2.6	2	$y^+ - su(w^a)^+$, bb^+
*Dp(1;f)1345	1C;20	1.7		$y^+ - svr^+$, bb^+
Dp(1;f)1346 ^γ	1B12-13;20	2	2, 3, 5	$y^+ - svr^+$, bb^+
Dp(1;f)1479 ^ε	1C;20	2.1		$y^+ - su(s)^+$, bb^+
Dp(1;f)1488	2A;20	2.5	2, 3	$y^+ - su(w^a)^+$, bb^+
*Dp(1;f)1489	1D;20	1.8		$y^+ - svr^+$, bb^+
Dp(1;f)1492 ^γ	1B10-12;20	1.9		$y^+ - svr^+$, bb^+ (?)
Dp(1;f)1494	1B10-14;20	2.7		$y^+ - svr^+$, bb^+
Dp(1;f)1498	1F;20	3.3	2, 3	$y^+ - su(w^a)^+$, bb^+
Dp(1;f)1501	2A;19E4-F1	4.4		$y^+ - svr^+$, bb^+
Dp(1;f)1512	1F;19E4-F1	3.6		$y^+ - svr^+$, bb^+
Dp(1;f)1513	1B10-14;20	>2		$y^+ - svr^+$, bb^+
Dp(1;f)1514	1B12-13;20	1.9		$y^+ - svr^+$, bb^+
Dp(1;f)1518	2A4-B1;20	3.9		$y^+ - svr^+$, bb^+

^α 1 = Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90; 2 = Grell, 1964, Genetics 50: 151-66; 3 = Grell, 1964, Proc. Nat. Acad. Sci. USA 52: 226-32; 4 = Krider, Yedvobnick, and Levine, 1979, Genetics 92: 879-89; 5 = Parry and Sandler, 1974, Genetics 77: 535-39; 6 = Procunier and Tartof, 1978, Genetics 88: 67-79; 7 = Rayle and Hoar, 1969, DIS 44: 94.
^β 1300 kb (Karpen and Spradling, 1990, Cell 63: 97-107; Karpen and Spradling, unpublished). Assorts randomly with respect to sex chromosomes.
^γ Lethal with *In(1)sc*^{4L}_{sc}^{8R}.
^δ Variegates for *dor*, *hfw*, and *BRC* in male with no Y chromosome.
^ε Includes *su(s)*⁺ according to Voelker.

*Dp(1;f)A1: see *Tp(1;3)A1

*Dp(1;f)A12: see *Tp(1;2;4)A12

Dp(1;f)AM

cytology: Dp(1;f)1C3-4;8C17-D1;9C1-2;16E1-2; 0.2 length of normal X.

new order:

1A - 1C3|9C1 - 8D1|16E3 - 20.

origin: X ray induced in *In(1)AM* = *In(1)8C17-D1;16E2-3*.

references: Bender and Barr, 1971, Nature (London) New

Biol. 231: 217-19.

Kalisch and Hägele, 1973, Chromosoma 44: 265-83.

genetics: Semilethal in males. Does not replicate its DNA in synchrony with homologous region of normal X (Kalisch and Hägele, 1973).

***Dp(1;f)eq: Duplication (1;free) from equational producer**

origin: X-ray-induced deletion of most of euchromatin from X chromosome carrying eq.

discoverer: Schultz, 34k4.

genetics: Contains y^+ to pn^+ , inclusive, and bb^+ . Male fertile but rather inviable; has occipital bristles; eyes rough, wings spread, wing veins thickened. Female has occipital bristles; wings straight edged and coarse textured. Female with two duplications occasionally survives and shows extreme spread wings and rough eyes.

Dp(1;f)GE-1

cytology: Dp(1;f)4C7;20D-F.

references: Gelbart, 1971, PhD. Thesis, Univ. of Wisconsin.

Dp(1;f)LJ: Duplication (1;free) La Jolla

origin: γ -ray-induced deletion of most of the euchromatin from $In(1)sc^{29} = In(1)1B;13A2-5$.

references: Hardy, Lindsley, Livak, Lewis, Siversten, Joslyn, Edwards, and Bonaccorsi, 1984, Genetics 107: 591-610.

duplication	cytology	new order	genetics
Dp(1;f)LJ4			na^+
Dp(1;f)LJ8			$g^y; na^+$
Dp(1;f)LJ9^α	1B;12A6-10; 13A2-5;20	1A-1B 13A2-12A10 20	$y^+ sc^{29} l(1)g^+ Ste^+ na^+$

^α Variegates for g, as in Dp(1;f)LJ9/FM7 and Dp(1;f)LJ9/In(1)d149, $y m^2 g^4$ males (Schalet). Dp(1;f)LJ9/YXS.YL, In(1)EN, Df(1)g⁺, y, g, f, B males viable and fertile (Schalet).

Dp(1;f)R: Duplication (1;free) from Ring X

cytology: Dp(1;f)1A3-4;3A1-2;20A1-F.

new order:

|1A4 - 3A|20-20F - 20A1|

origin: Spontaneous deletion of most of euchromatin from R(1)2.

cytology: Schultz, 35d10.

synonym: Dp(1;f)X^{c2}.

references: Ananiev and Gvozdev, 1974, Chromosoma 45: 173-91.

Gvozdev, Gerasimova, and Birstein, 1974, Mol. Gen. Genet. 130: 251-60.

genetics: Covers y to kz but not bb. Variegation for dor, ac, svr, pn, and kz decreased as Y's are added. Variegation for y insensitive to Y's.

***Dp(1;f)R1-*Dp(1;f)R43**

origin: X-ray-induced-deletion of most of euchromatin from R(1)2.

discoverer: Pontecorvo.

synonym: Del(X^{c2})1-Del(X^{c2})43.

duplication	cytology	new order	ref ^α
*Dp(1;f)R1^β			1
*Dp(1;f)R35	1A3-4;17A4-5; 19F-20A1	17A5 - 20.20F	2
*Dp(1;f)R36	1A3-4;17A4-5; 19F-20A1	17A5 - 20.20F	2

duplication	cytology	new order	ref ^α
*Dp(1;f)R37	1A3-4;16F2-3; 19F-20A1	16F3 - 20.20F	2
*Dp(1;f)R38	1A3-4;1F;20	1A4 - 1F 20.20F - 20A1	1
*Dp(1;f)R40	1A3-4;1F4-5;20	1A4 - 1F4 20.20F - 20A1	2
*Dp(1;f)R41	1A3-4;1F4-5;20	1A4 - 1F4 20.20F - 20A1	2
*Dp(1;f)R42	1A3-4;2A2-3;20	1A4 - 2A2 20.20F - 20A1	2
*Dp(1;f)R43	1A3-4;1F4-5;20	1A4 - 1F4 20.20F - 20A1	2

^α 1 = Pontecorvo, 1942, DIS 16: 65; 2 = Slizynska, 1942, DIS 16: 67.

^β Deletion of most of X euchromatin.

***Dp(1;f)R53d**

cytology: Dp(1;f)1A3-4;1F-2A;20.

new order:

|1A4 - 1F|20-20F - 20A1|

origin: X-ray-induced deletion of most of euchromatin from R(1)2.

discoverer: S. Brown, 1953.

synonym: Del(X^{c2})53d.

references: 1955, DIS 29: 70.

Brosseau, 1955, DIS 29: 106.

genetics: Contains wild-type alleles of y, ac, sc, and su(s); covers Df(1)260-1. Female tolerates two duplications; male tolerates only one. Fly hemi- or homozygous for y and the duplication shows mosaicism for y. There is probably both variegation for y and loss of the duplication.

***Dp(1;f)RA**

cytology: Dp(1;f)1A3-4;1F-2A;20 (Slizynska).

new order:

|1A4 - 1F|20-20F - 21A1|

origin: X-ray-induced deletion of most of euchromatin from R(1)2.

discoverer: Pontecorvo.

references: 1942, DIS 16: 65.

Dp(1;f)sc^{7.2}

origin: Deletion of majority of euchromatin from $In(1)sc^7 = In(1)B4-6;5D3-6$.

references: Garcia-Bellido, 1979, Genetics 91: 491-520.

genetics: Carries wild-type alleles of neither cv nor car.

***Dp(1;f)sc²⁶⁰⁻²⁷: Duplication (1;free) scute**

cytology: Dp(1;f)1A8-B1;19F.

origin: Aneuploid segregant from $Tp(1;2)sc^{260-27}/+$.

Dp(1;f)sc^{SIL}sc^{4R} (L. Robbins)

cytology: Dp(1;f)1-1B3;20F, inferred from genetics.

origin: Rex-induced mitotic exchange in $In(1)sc^{SIL}sc^{4R}$.

references: Swanson, 1984, Ph.D. thesis, Michigan State Univ.

Swanson, 1987, Genetics 115: 271-76.

genetics: One of two mitotic exchange products induced by Rex in $In(1)sc^{SIL}sc^{4R}$ [see also Dp(1;1)sc^{SIL}sc^{4R}]. Material between the two NO regions of $In(1)sc^{SIL}sc^{4R}$ is deleted. Recovered at 1-5% as sterile X/Dp male offspring of Rex/+ or Rex/Rex female by $In(1)sc^{SIL}sc^{4R}/Y$ male crosses. Recovered as X/Dp/Y males at a rate of 10⁻³ from crosses of Rex/In(1)sc^{SIL}sc^{4R} females to X/Y males. Independently generated Dp(1;f)sc^{SIL}sc^{4R} chromosomes recovered from X/Dp/Y males have different bb^{Rex} phenotypes. By analogy with Ybb^{Rex}, the proximal and distal heterochromatic segments are probably intact and in normal order.

Dp(1;f)sc^{V2} (L. Robbins)

cytology: *Dp(1;f)1-1B3;20F*, inferred from genetics.
origin: *Rex*-induced mitotic exchange in *In(1)sc^{V2}*.
references: Swanson, 1987, *Genetics* 115: 271-76.
genetics: A *Rex*-induced mitotic exchange product of *In(1)sc^{V2}*. Material between the two segments of the divided *NO* region of the inversion is deleted. Recovered as sterile *X/Dp* male offspring of *Rex*-bearing females and *In(1)sc^{V2}/Y* males. By analogy with other *Rex*-generated chromosomes, the heterochromatic constitution should be normal except for *bb^{Rex}* mutations induced during the exchange event.

***Dp(1;f)w^{m3}: Duplication (1;free) white-mottled**

cytology: *Dp(1;f)3C-D;19-20*; breakpoints inferred from genetic data.
origin: X ray induced.
discoverer: Muller, 1925.
references: 1930, *J. Genet.* 22: 299-334.
genetics: *w/Dp(1;f)w^{m3}* male has variegated eyes and is sterile; *C(1)RM*, *w/Dp(1;f)w^{m3}* female has variegated eyes and is fertile.

Dp(1;f)X^{c2}: see *Dp(1;f)R*

***Dp(1;f)y-sc: Duplication (1;free) for yellow and scute**

origin: X-ray-induced deletion of most of X euchromatin.
cytology: Oliver, 32k21.
references: 1937, *DIS* 7: 19.
phenotype: Carries wild-type alleles of *y* and *sc* but not *pn*.

Dp(1;f)y⁺

cytology: Small fragment from tip of X.
references: Roberts, 1969, *Genetics* 63: 387-404.
genetics: Carries *y⁺*.

Dp(1;f)w^{m51bL}w^{m4R} (L. Robbins)

cytology: *Dp(1;f)1-3C1;20F*, inferred from genetics.
origin: *Rex*-induced mitotic exchange in *In(1)w^{m51bL}w^{m4R}*.
references: Swanson, 1987, *Genetics* 115: 271-76.
genetics: One of two mitotic exchange products induced by *Rex* in *In(1)w^{m51bL}w^{m4R}* (see also *Dp(1;1)w^{m51bL}w^{m4R}*). Material between the two *NO* regions of *In(1)w^{m51bL}w^{m4R}* is deleted. Recovered at 1-5% as sterile *X/Dp* male offspring of *Rex/+* or *Rex/Rex* female by *In(1)w^{m51bL}w^{m4R}/Y* male crosses. By analogy with *Dp(1;f)sc^{SIL}sc^{4R}/Y* and *Ybb^{Rex}*, independent occurrences probably are *bb^{Rex}* with the other heterochromatic segments intact and in normal order.

Dp(1;f)z9: Duplication (1;free) zeste

cytology: *Dp(1;f)3E7-F1;19-20*.
origin: X-ray-induced deletion of most of euchromatin from *z*-bearing X chromosome.
discoverer: Gans.
references: 1953, *Bull. Biol. France Belg.*, Suppl. 38: 1-90.
 Jack and Judd, 1979, *Proc. Nat. Acad. Sci. USA* 76: 1368-72.
genetics: Contains *z* and wild-type alleles of *y* through *dm*. Eye color *zeste* in *z w⁺* males carrying the duplication.

Dp(2;1)AT

cytology: *Dp(2;1)5A-7A;36D1-2;37D1-2*. Duplicated for both 5A-7A and 36D-37D.
new order:
 1A – 7A|36D1-2 – 37D1-2|5A – 20.
references: Marsh and Wright, 1986, *Genetics* 112: 249-65.

Dp(2;1)B19

cytology: *Dp(2;1)9B-C;24D2-5;25F1-2*.
new order:
 1A – 9B|25F1 – 24D5|9C – 20.
origin: X ray induced.
references: Reuter and Szidonya, 1983, *Chromosoma* 88: 277-85.
genetics: Viable and female fertile. Wild type over *Df(2L)M24F11*, *Df(2L)M24F-B*, and *Df(2L)tkv3*.

Dp(2;1)C239: see **Tp(2;1)C239**

Dp(2;1)DTD2: see **Tp(2;1)DTD2**

Dp(2;1)OR19: see **Tp(2;1)OR19**

Dp(2;1)Sco^{rv23}: see **Tp(2;1)Sco^{rv23}**

Dp(2;1;f)Sco^{rv23}-X: Duplication (2;1;f) Scutoid-reverted-X

cytology: *Df(1)1B1-2;20* on *Dp(2;1)20;34F1-2;35C1-2*.
new order:
 1A – 1B1|20F|(34F2 – 35B1|35D1 – 35C1|35B4 – 35B8|35B3 – 35B2)|20F;
 21 – 34F1|35D2 – 60.
origin: γ -ray-induced deletion of *Dp(2;1)Sco^{rv23}*.
discoverer: S. Tsubota.
synonym: *Dp(2;1;f)Sco^{R+23}*.
references: Ashburner.
genetics: Covers *l(2)34Fa* to *l(2)35Da*; carries active *Adh* allele. Mutant for *noc*. Three independent isolates (-X2, -X3, and -X4) recovered with compound -X females.

Dp(2;1;3;f)Sco^{rv23}-X6

cytology: *Dp(1;3)1B9-10;20F;h34;96B;97F;98F1-2* superimposed on *Dp(2;1)Sco^{rv23}*.
new order:
 1A1 – 1B9|96B – 61;
 h34 – h29|(34F2 – 35A4|(35C2 – 35C5)|35B4 – 35C1|35B3 – 35B1|h29 – 20F|97F – 96B|98F2 – 100 (tentative). 1B10-20F and 97F-98F1 missing.
origin: γ ray induced.
discoverer: S. Tsubota.
synonym: *Dp(2;1)Sco^{R+23}*.
references: Ashburner.
genetics: Duplication for 2; acts like *Dp(2;1)Sco^{rv23}*, covering *l(2)34Fa* - *l(2)35Da* and including *Adh*.

Dp(2;Y)B231: see **Tp(2;Y)B231**

Dp(2;Y)bw⁺: see **Tp(2;Y)bw⁺**

Dp(2;Y)C: see **cn⁺Y**

Dp(2;Y)C: see **Tp(2;Y)C**

Dp(2;Y)Ddc⁺: see **Dp(2;Y)HI**

Dp(2;Y)G: see **Tp(2;Y)G**

***Dp(2;Y)H:** see ***Tp(2;Y)H**

Dp(2;Y)H1

cytology: *Dp(2;Y)36B4-5;40F* + *Df(2L)37F4-38A1;39C2-D1*.

new order:

Y|36B5 – 37F4|39D1 – 40F|Y.

origin: γ ray induced in *Dp(2;Y)G*.

synonym: *Dp(2;Y)Ddc*⁺.

references: Clark, Pass, Venkataraman, and Hodgetts, 1978, *Mol. Gen. Genet.* 162: 287-97.

Hodgetts, 1980, *DIS* 55: 63.

genetics: Covers *rdo-hk*, *Ddc*, and *lt* but not *pr*. *Y* fertility not affected.

Dp(2;Y)H2

cytology: *Dp(2;Y)36B4-5;40F* + *Df(2L)38B2-C1;39E2-3*.

new order:

Y|36B5 – 38B2|39E3 – 40F|Y.

origin: γ ray induced in *Dp(2;Y)G*.

references: Hodgetts, 1980, *DIS* 55: 63.

genetics: Covers *rdo-hk*, *Ddc*, and *lt* but not *pr*. *Y* fertility not affected.

Dp(2;Y)H3

cytology: *Dp(2;Y)36B4-5;40F* + *Df(2L)37E2-F1;40B-F*.

new order:

Y|36B5 – 37E2|40F|Y.

origin: γ ray induced in *Dp(2;Y)H1*.

references: Hodgetts, 1980, *DIS* 55: 63.

genetics: Covers *rdo-hk* and *Ddc* but not *pr* or *lt*. *Y* fertility not affected.

Dp(2;Y)prd: see ***Tp(2;Y)prd***

Dp(2;Y)R24:** see *Tp(2;Y)R24***

Dp(2;Y)Rsp: Duplication (2;Y) Responder

cytology: Insertion of 2R heterochromatin into *B^SYy*⁺.

origin: Designation of several *Y* chromosomes formed by reconstitution from *T(Y;2)* males by irradiation of mature translocation-bearing sperm (Lyttle, 1984, *Genetics* 106: 423-34).

synonym: *RspY*.

references: Lyttle and Ault, 1985, *Genetics* 110: s23. Lyttle, 1989, *Genetics* 121: 751-63.

genetics: Sensitivity to SD varies depending on the number of *Rsp* repeat sequences included in the reconstituted *Y*.

Dp(2;Y)Sd: Duplication (2;Y) Segregation distorter

new order:

YL|36D3 – 40F|YL·YS.

origin: X-ray-induced reconstitution of *Y* from *T(Y;2)b10*.

synonym: *Dp(2;Y)B10-4*.

references: Lyttle, 1984, *Genetics* 106: 423-34.

Lyttle, Brittnacker, and Ganetzky, 1986, *Genetics* 114: 183-202.

Lyttle, 1986, *Genetics* 114: 203-16.

genetics: Carried *Sd* and *E(Sd)* as well as *B^S* from *B^SY* at the end of *YL*.

Dp(2;2)41A

cytology: Tandem duplication for material in 41A.

origin: Spontaneous in the *In(2L)Cy* + *In(2R)Cy* chromosome of a balanced *In(2L)Cy* + *In(2R)Cy/Df(2R)M41A10* stock.

discoverer: Schultz, 1945.

references: CP627.

genetics: Acts as a suppressor of *Df(2R)M41A* and perhaps as a partial suppressor of *L*. Fly heterozygous for the duplication appears more stocky than normal.

Dp(2;2)92

cytology: *Dp(2;2)30B;34A*; tandem duplication.

discoverer: Reuter.

references: De la Concha, Dietrich, Weigel, and Campos-Ortega, 1988, *Genetics* 118: 499-508.

genetics: Duplication for *bib*⁺.

Dp(2;2)A446: see ***Tp(2;2)A446***

***Dp(2;2)Adh2: Duplication (2;2) Alcohol dehydrogenase**

cytology: *Dp(2;2)32D3;35C1*; direct tandem duplication.

new order:

21A – 35C1|32D3 – 40.

origin: X ray induced as interchange between homologues in female.

references: Grell, 1969, *Genetics* 69: s23.

Nash, 1970, *Genetics* 64: 471-79.

Ashburner, 1982, *Genetics* 101: 447-59.

genetics: Carries *Adh^F* and *Adh^S*. Dominant enhancer of *H*.

Dp(2;2)Adh3

cytology: *Dp(2;2)34B1-2;35B3*; tandem repeat.

new order:

21 – 35B3|34B1-2 – 60.

origin: X ray induced as interchange between homologues in female.

references: Grell, 1969, *Genetics* 61: s23.

Nash, 1970, *Genetics* 64: 471-79.

Ashburner, 1982, *Genetics* 101: 447-59.

Ashburner, Detwiler, Tsubota, and Woodruff, 1983, *Genetics* 104: 405-31.

genetics: Duplicated for *b-Adh* in the order (*b⁺ rk^{EMS} el⁺ Adh^D*) (*b rk⁺ el Adh^F*); carries *rd^S*. Slight suppressor of *H* (Nash, 1970; Ashburner, 1982). Suppresses *Cu* and *Cu⁷⁵* (Woodruff and Ashburner, 1979, *Genetics* 92: 117-32) and the bristle phenotype of *Sco^{rv1}* and *Sco^{rv11}* (Ashburner *et al.*, 1983).

Dp(2;2)AM

origin: *P*-element induced mutagenesis.

references: Locke, Kotarski, and Tartof, 1988, *Genetics* 120: 181-98.

genetics: Suppresses Minute phenotype of *M(2)24F*.

duplication	cytology	suppression of	
		<i>In(1)w^{m4}</i>	variegation
<i>Dp(2;2)AM3^α</i>	24A;26F		+
<i>Dp(2;2)AM6</i>	23B;25F4-26A1		-
<i>Dp(2;2)AM7</i>	23D;25F2-3		-
<i>Dp(2;2)AM17^α</i>	23D-E;26C		+
<i>Dp(2;2)AM18^α</i>	24F-25A;27A		+
<i>Dp(2;2)AM20</i>	23E;25F2-4		-
<i>Dp(2;2)AM22</i>	22E-F;24F-25A		-
<i>Dp(2;2)AM25</i>	23A;24F		-
<i>Dp(2;2)AM26</i>	23D-E;25D-E		-
<i>Dp(2;2)AM33^α</i>	21E-F;28C-D		+
<i>Dp(2;2)AM34^α</i>	24;27		+
<i>Dp(2;2)AM35</i>	22B;26A5-6		-
<i>Dp(2;2)AM37^α</i>	23D-E;27B-C		+
<i>Dp(2;2)AM42</i>	23C;25F-26A		-

^α Counteracts the *In(1)w^{m4}* enhancing effect of *Df(2L)E66*, which is deficient for *E(van)26A*.

Dp(2;2)AZ

cytology: *Dp(2;2)33B1-3;34A1-2;50A-B* +
Df(2L)34B;35C.

new order:

21 – 50A|34A1 – 33B1|50B – 60.

origin: Spontaneously deleted derivative of *Dp(2;2)GYL*.

discoverer: Zacharopoulou.

references: Yannopoulos, Zacharopoulou, and Stamatis, 1982, *Mutat. Res.* 96: 41-51.

Dp(2;2)B

origin: X ray induced. Recovered as *trans* suppressors of *Df(2L)M24F11*; most are tandem repeats.

references: Reuter and Szidonya, 1983, *Chromosoma* 88: 277-85.

duplication	cytology	homozygous viability	female fertility	Suppression of <i>w^m</i>
<i>Dp(2;2)B1</i>	24D2-5;25A1-2	-	-	-
<i>Dp(2;2)B2</i>	23E4-F1;26D9-E1	+	-	+
<i>Dp(2;2)B3</i>	24A2-B1;26C1-2	+	-	+
<i>Dp(2;2)B4</i>	22C-D;25A3-4	-	-	-
<i>Dp(2;2)B5</i>	23F6-24A1;25E2-F1	+	+	-
<i>Dp(2;2)B6</i>	24D2-5;24F5-25A1	+	+	-
<i>Dp(2;2)B7</i>	24D1-2;24E5-F1	+	+	-
<i>Dp(2;2)B8</i>	22D4-E1;25F2-3	-	-	-
<i>Dp(2;2)B9</i>	23A1-2;26C1-2	-	-	+
<i>Dp(2;2)B10</i>	24C8-D1;27D-E	+	-	+
<i>Dp(2;2)B11</i>	22C-D;25C9-D1	-	-	-
<i>Dp(2;2)B12</i>	24D2-5;24F2-25A1	+	+	-
<i>Dp(2;2)B13</i>	24D2-5;25A4-6	+	+	-
<i>Dp(2;2)B14</i>	24D2-5;25A2-3	+	+	-
<i>Dp(2;2)B15</i>	23D5-E1;26B9-C1	-	-	+
<i>Dp(2;2)B16</i>	24C8-D1;26C2-D1	+	-	+
<i>Dp(2;2)B17</i>	23A3-B1;25C3-8	+	+	-
<i>Dp(2;2)B23</i>	24D2-5;24F2-25A1	+	+	-
<i>Dp(2;2)B24</i>	24C2-8;27E-28A	-	-	+
<i>Dp(2;2)B25</i>	22B2-4;25A2-3	-	-	-
<i>Dp(2;2)B26</i>	24B2-C1;25A7-B1 + <i> Tp(2;2)</i>	+	+	-
<i>Dp(2;2)B27</i>	22D1-6;26B2-C1	-	-	+
<i>Dp(2;2)B28</i>	23F2-24A1;25B2-C1 + <i> Tp(2;2)37B9-C1;39C2-D1</i>	-	-	-

Dp(2;2)b81f1

cytology: *Dp(2;2)34D3;35B2.*

new order:

21 – 35B2|34D3 – 60.

origin: γ ray induced.

discoverer: G. Johnson.

***Dp(2;2)bw^{AL}Cy^R**

cytology: *Dp(2;2)58A4-B1;59D.*

origin: Associated with *In(2R)bw^{AL}Cy^R*.

references: CP627.

Dp(2;2)bw^{V34kL}: see *In(2R)bw^{V34k}*

Dp(2;2)bw^{VDe1L}Cy^R

cytology: *Dp(2;2)58A4-B1;59E2-4.*

origin: Associated with *In(2R)bw^{VDe1L}Cy^R*.

references: CP627.

Dp(2;2)bw^{VDe2L}Cy^R

cytology: *Dp(2;2)58A4-B1;59D6-E1.*

origin: Associated with *In(2R)bw^{VDe2L}Cy^R*.

references: CP627.

Dp(2;2)C75RL: see *In(2L)75c^LC158.1^R*

Dp(2;2)C158.1^LSco^{rv11R}

see *In(2L)C158.1^RSco^{rv11R}*

Dp(2;2)C158.1^LSco^{rv17R}

see *In(2L)C158.1^LSco^{rv17R}*

Dp(2;2)C163.41^LC158.1^R

see *In(2L)C163.41^LC158.1^R*

Dp(2;2)C619

cytology: *Dp(2;2)26A;28E.*

new order:

21A – 28E|26A – 60.

origin: X ray induced in oocyte.

discoverer: Roberts and Thomas, 1965.

references: Roberts, 1966, *Genetics* 54: 969-79.

Thomas and Roberts, 1966, *Genetics* 53: 855-62.

genetics: Homozygous viable. Reduces recombination in *2L*. Map distance between *al* and *pr* reduced from 44.2 to 7.3 in *Dp(2;2)C619/+* and to 17.0 in *Dp(2;2)C619* homozygotes.

Dp(2;2)cn⁺: Duplication (2;2) cinnabar - wild type

cytology: *Dp(2;2)41F;44B.*

new order:

21 – 44B|41F – 60.

origin: Recovered as apparent *dp b bw* double recombinant from a test cross of a dysgenic-type male that was heterozygous for a maternally derived *dp b cn bw* second chromosome and a paternally derived autosome extracted from a natural population and capable of promoting male recombination. A MRF (Male Recombination Factor) chromosome, *MRF23.5*, was utilized. The duplication was recovered from a male heterozygous for *MRF23.5*.

references: Yannopoulos, Zacharopoulou, and Stamatis, 1982, *Mutat. Res.* 96: 41-51.

genetics: Covers *cn*.

Dp(2;2)Cy^Lbw^{VDe1R}

cytology: *Dp(2;2)41B2-C1;42A2-3.*

origin: Associated with *In(2R)Cy^Lbw^{VDe1R}*.

references: CP627.

Dp(2;2)Cy^Lbw^{VDe2R}

cytology: *Dp(2;2)41A-B;42A2-3.*

origin: Associated with *In(2R)Cy^Lbw^{VDe2R}*.

references: CP627.

Dp(2;2)Cy^LC129^R: see *In(2R)Cy^LC129^R*

Dp(2;2)Cy^Rbw^{V34kR}: see *In(2R)bw^{V34k}*

Dp(2;2)D85-G42

cytology: *Dp(2;2)84C-D;85E.*

origin: Segmental aneuploidy.

references: Kempfues, Raff, Raff, and Kaufman, 1980, *Cell* 21: 445-51.

Kempfues, Raff, and Kaufman, 1983, *Genetics* 105: 345-56.

genetics: Carries *p⁺*.

Dp(2;2)da: Duplication (2;2) daughterless

origin: γ ray induced.

references: Cronmiller and Cline, 1986, *Dev. Genet.* (Amsterdam) 7: 205-21.

genetics: Carries two doses of *da*.

duplication	cytology ^α	genetics of homozygote
<i>Dp(2;2)da119</i>	31A;34A	viable, sterile
<i>Dp(2;2)da125</i>	30A-B;34B-C	lethal, sterile

^α Tandem duplication.

***Dp(2;2)da⁺*: Duplication (2;2) daughterless - wild type**

origin: γ ray induced.

references: Cronmiller and Cline, 1986, Dev. Genet. (Amsterdam) 7: 205-21.

genetics: Carries wild-type alleles of *da* and *M(2)30A*.

duplication	cytology	genetics of homozygote
<i>Dp(2;2)da⁺18</i>	30-31;31-32	semi-viable; semi-fertile
<i>Dp(2;2)da⁺20</i>	30C-D;34A-B	few survivors; sterile
<i>*Dp(2;2)da⁺44</i>	not visible	viable, fertile

Dp(2;2)dpp¹¹: see ***Tp(2;2)dpp¹¹***

Dp(2;2)dpp¹³: see ***Tp(2;2)dpp¹³***

Dp(2;2)dpp²¹: see ***Tp(2;2)dpp²¹***

Dp(2;2)dpp^{23L}DTD^{21R}:
see ***In(2L)dpp^{23L}DTD^{21R}***

***Dp(2;2)DTD48*: Duplication (2;2) Disrupter of Transvection at Decapentaplegic**

cytology: *Dp(2;2)22E2-4;22F4-23A1;53D1-2*.

origin: X ray induced.

references: Spenser, Hoffmann, and Gelbart, 1982, Cell 28: 451-61.

genetics: Duplication for all regions of *dpp*. Disrupts transvection at *dpp*.

****Dp(2;2)E(H)*: Duplication (2;2) Enhancer of Hairless**

cytology: *Dp(2;2)35B6-8;35B8-10*. Difficult to observe in heterozygotes because of ectopic pairing (Nash, 1970).

references: Nash, 1970, Genetics 64: 471-79.

Ashburner, 1982, Genetics 101: 447-59.

genetics: Dominant enhancer of *H* owing to the presence of two doses of *Su(H)⁺*. Homozygous viable.

***Dp(2;2)E(var)29A#19*: Duplication (2;2) Enhancer of variegation**

cytology: *Dp(2;2)28D1-12;29F3-30A2 + In(2L)28D1-12;37B1-13*.

origin: P-element induced mutagenesis.

synonym: *Dp(2;2)E19*.

references: Locke, Kotarski, and Tartof, 1988, Genetics 120: 181-98.

genetics: Carries two doses of *Su(var)2-5⁺* = *Su(var)205⁺*. Enhances position-effect variegation of *In(1)w^{m4}*. Recessive lethal.

Dp(2;2)E(var)29A#39A

cytology: *Dp(2;2)28B2-C9;29B2-C5*.

origin: P-element induced mutagenesis.

synonym: *Dp(2;2)E39A*.

references: Locke, Kotarski, and Tartof, 1988, Genetics 120: 181-98.

genetics: Carries two doses of *Su(var)2-5⁺* = *Su(var)205⁺*. Enhances position-effect variegation of

In(1)w^{m4}. Recessive lethal.

***Dp(2;2)Eni*: Duplication (2;2) Enigma**

cytology: Complete copy of 2L appended to the end of 2R to produce 2L.2R2L chromosome. Deficient for 60D-F.

new order:

21 – 60D|40B – 21.

origin: Thought to be reciprocal crossover product of exchange generating a *F(2R)*.

references: Fitz-Earle, 1979, Genetics 92: s34.

Holm, 1980, Theor. Appl. Genet. 57: 247.

genetics: Carried in combination with *F(2R)*. Recombination between *F(2R)* and 2R of *Dp(2;2)Eni* generates a normal second chromosome and an acrocentric second chromosome that is 41 – 60D|40 – 21 and deficient for 60D-F, as reciprocal products.

Dp(2;2)GM: see ***Df(2L)Sco-1***

***Dp(2;2)GYL*: Duplication (2;2) of George Yannopoulos**

cytology: *Dp(2;2)33B1-2;35C1-3;50A1-4*.

new order:

21 – 50A4|35C1 – 33B2|50B5 – 60.

origin: Recovered as apparent *dp cn bw* double recombinant from a test cross of a dysgenic male that was heterozygous for a maternally derived *dp b cn bw* second chromosome and a paternally derived autosome extracted from a natural population and capable of promoting male recombination. A MRF (Male Recombination Factor) chromosome, *MRF23.5*, was utilized. The duplication was recovered from a male heterozygous for *MRF23.5*.

references: Yannopoulos, Stamatis, Zacharopoulou, and Pelecanos, 1981, Mutat. Res. 83: 383-93.

Ashburner, 1982, Genetics 101: 447-59.

Yannopoulos, Zacharopoulou, and Stamatis, 1982, Mutat. Res. 96: 41-51.

Hatzopoulos, Monastirioti, Yannopoulos, and Louis, 1987, EMBO. J. 6: 3091-96.

genetics: Covers *esc* (Struhl) and all loci in region 34-35 up to *l(2)35Cb*. Enhances *H* (Ashburner, 1982). Unstable, transposing and producing shorter derivatives. Homozygous viable.

molecular biology: Mobile element *hobo* present at or near breakpoints of GYL derivatives (Hatzopoulou, 1987).

Dp(2;2)GYL1

cytology: *Dp(2;2)33B1-2;35C1-3;50A4-B5*.

origin: Derivative of *Dp(2;2)GYL*.

references: Ashburner.

genetics: Covers *esc – l(2)35Cb*.

Dp(2;2)GYL4

cytology: *Dp(2;2)34A1-2;35C1-3;50A1-4*.

new order:

21 – 50A1|35C1 – 34A2|50A4 – 60.

origin: Spontaneous derivative of *Dp(2;2)GYL*.

references: Hatzopoulos, Monastirioti, Yannopoulos, and Louis, 1987, EMBO J. 6: 3091-96.

Dp(2;2)GYS

cytology: *Dp(2;2)34D1-2;35C1-3;50A1-4*.

new order:

21 – 50A1|35C1 – 34D2|50A4 – 60.

origin: Spontaneous derivative of *Dp(2;2)GYL*.

references: Ashburner, 1982, Genetics 101: 447-59.
Yannopoulos, Zacharopoulou, and Stamatis, 1982, Mutat. Res. 96: 41-51.
genetics: Distally covers lethals in region in common between *Df(2L)64j* and *Df(2L)b80e3*; proximal limit of duplication presumably same as that of *Dp(2;2)GYL*. Enhances *H*. Homozygous viable.

Dp(2;2)GYSS

cytology: *Dp(2;2)34D1-2; 35B1-2;59F1-3*.
new order:
21 – 59F2|35B1 – 34D2|59F3 – 60.
origin: Spontaneous derivative of *Dp(2;2)GYS*.
references: Ashburner.
genetics: Covers all lethals in region in common between *Df(2L)64j* and *Df(2L)b80e3*.

Dp(2;2;2)I

cytology: Tandem triplication of 33A;40B with all members inverted.
new order:
21A – 33A|40B – 33A|40B – 33A|40B – 33A|40B – 60.
origin: Induced by triethylenemelamine.
synonym: *Dp(2;2)Tr-I*.
references: Slizynska, 1968, Genet. Res. 11: 201-08.

Dp(2;2;2)II

cytology: Triplication of 41A;44D with only middle member inverted.
new order:
21A – 44D|44D – 41A|41A – 60.
origin: Induced by nitrogen-mustard.
synonym: *Dp(2;2)Tr-II*.
references: Slizynska, 1968, Genet. Res. 11: 201-08.

Dp(2;2)It⁺: Duplication (2;2) light - wild type

cytology: Chromosome duplicated for heterochromatin between *It* and the secondary constriction.
origin: γ-ray-induced detachment product of compound second chromosomes.
references: Hilliker and Holm, 1975, Genetics 81: 705-21.
genetics: Carries duplication for 2L heterochromatin with *It⁺*.

Dp(2;2)M: Duplication (2;2) Mglinetz

origin: Recovered as apparent recombinants from γ-irradiated males.
references: Mglinetz, 1971, Genetika (Moscow) 7(8): 108-14.

duplication	cytology
<i>Dp(2;2)M2</i>	58F;59D-60E
<i>Dp(2;2)M4</i>	44B;46B
<i>Dp(2;2)M6</i>	56F;57D
<i>Dp(2;2)M7</i>	50F;54B
<i>Dp(2;2)M8</i>	57B;60D
<i>Dp(2;2)M11</i>	23A;25A
<i>Dp(2;2)M12</i>	39D;41
<i>Dp(2;2)M13</i>	37F;40
<i>Dp(2;2)M14</i>	48E;50A

Dp(2;2)M24F⁺: Duplication (2;2) Minute wild-type

origin: X ray induced in wild-type females. Recovered as *trans* suppressors of *M(2)24F*.
references: Roberts and Broderick, 1982, Genetics 102: 75-89.

synonym: *Dp(2;2)SuM2z; Dp(2;2)Mz*.
genetics: Covers and suppresses *M(2)24F*. Most of the duplications suppress crossing over when heterozygous.

duplication	cytology ^α
<i>Dp(2;2)M24F⁺1</i>	24F8-25A1;25A1-3
<i>Dp(2;2)M24F⁺2</i>	24F6;25A3
<i>Dp(2;2)M24F⁺3</i>	24F1;25A1-3
<i>Dp(2;2)M24F⁺4</i>	24F1;25A3
<i>Dp(2;2)M24F⁺5^β</i>	24F1;25A3;26C
<i>Dp(2;2)M24F⁺6</i>	24D1;25A3
<i>Dp(2;2)M24F⁺7</i>	24A1-2;25A1-3
<i>Dp(2;2)M24F⁺8</i>	24A1-2;25A3-4
<i>Dp(2;2)M24F⁺9</i>	24E1-2;25E1-2
<i>Dp(2;2)M24F⁺10</i>	24F8-25A1;26C1-2
<i>Dp(2;2)M24F⁺11</i>	22D6;25A1-3
<i>Dp(2;2)M24F⁺12</i>	24E1-2;26F
<i>Dp(2;2)M24F⁺13</i>	22F;25E2
<i>Dp(2;2)M24F⁺14</i>	21B7;25A3-4
<i>Dp(2;2)M24F⁺15</i>	23D1;26F
<i>Dp(2;2)M24F⁺16</i>	23E-F;27D-F1
<i>Dp(2;2)M24F⁺17</i>	22A3;25F2
<i>Dp(2;2)M24F⁺18</i>	23D1-2;27B4
<i>Dp(2;2)M24F⁺19</i>	22A1;27C2
<i>Dp(2;2)M24F⁺20</i>	23F;29B

^α Tandem duplications (except for #5).
^β New order: 21 – 26C|25A3 – 24F1|26C – 60.

Dp(2;2)M56F⁺2

cytology: *Dp(2;2)56C-D;59C-D*; tandem repeat.
origin: X ray induced.
synonym: *Dp(2;2)Minute 2*.
references: Nix, 1973, Mol. Gen. Genet. 120: 309-18.
genetics: Suppresses *M(2)56F*. Includes site of rRNA-5S genes (Wimber and Steffensen, 1970, Science 170: 639-41). Homozygous lethal and lethal with *Dp(2;2)M56F⁺2*. Fertility of heterozygotes good.

Dp(2;2)M56F⁺4

cytology: *Dp(2;2)56E-F;58A-B*.
origin: X ray induced.
synonym: *Dp(2;2)Minute 4*.
references: Nix, 1973, Mol. Gen. Genet. 120: 309-18.
genetics: Suppresses *M(2)56F*. Left break of duplication within site of rRNA-5S genes. Homozygous lethal and lethal with *Dp(2;2)M56F⁺2*. Fertility of heterozygotes good.

Dp(2;2)Mdh: Duplication (2;2) Malate dehydrogenase

cytology: Tandem duplication in region of "gooseneck" in 2L.
origin: X ray induced.
synonym: *Dp(2;2)Mdh-2*.
references: Grell, 1969, Genetics 61: s23.
genetics: Carries *Mdh1*. Compensates for Minute effect of *Df(2L)Mdh*.

Dp(2;2)MS3: Duplication (2;2) Mglinetz Semenova

cytology: *Dp(2;2)47B;48E*.
origin: γ ray induced.
references: Mglinetz and Semenova, 1970; Genetika (Moscow) 6(8); 86-94.

Dp(2;2)MS4

cytology: *Dp(2;2)42A;55E*.
origin: γ ray induced.
references: Mglinetz and Semenova, 1970, Genetika

(Moscow) 6(8): 86-94.

Dp(2;2)MS5: see **Tp(2;2)MS5**

Dp(2;2)MVD1-2

cytology: *Dp(2;2)21E;25A1;33B*.

references: Irish and Gelbart, 1987, *Genes Dev.* 1: 868-79.

Dp(2;2)NS

cytology: *Dp(2;2)34A1-2;35C1-3;50B + In(2R)50B1-2;56D9-10* (Ashburner).

new order:

21 – 50B1|56D9 – 50B2|34A2 – 35C1|56D10 – 60.

origin: Spontaneous derivative of *Dp(2;2)GYL*.

references: Yannopoulos, Zacharopoulou, and Stamatis, 1982, *Mutat. Res.* 96: 41-51.

Dp(2;2)Px⁴: see *In(2LR)S^{56f}*

Dp(2;2)S: Duplication (2;2) Star

cytology: *Dp(2;2)21D2-3;21E2-3*; tandem repeat.

new order:

21A – 21E2|21D3 – 60.

origin: Spontaneous as interchange between homologous chromosomes.

discoverer: E. B. Lewis, 39i.

references: 1941, *Proc. Nat. Acad. Sci. USA* 27: 31-35. 1945, *Genetics* 30: 137-66.

genetics: Duplicated segment contains the loci of *S* and *ast*. *ast* mutant in both members of the duplication (+ *ast* + *ast*). Duplication appears wild type when homozygous or when heterozygous with *ast*. Heterozygous with *S*, it has normal or only slightly roughened eyes. Various combinations of *S* and *ast* alleles have been introduced into the duplication. Through unequal crossing over, a triplication and a quintuplication of the region [*i.e.* *Dp(2;2;2)S* and *Dp(2;2;2;2;2)S*] have been synthesized.

Dp(2;2)S^{56f}

cytology: *Dp(2;2)21C6-D1;22A3-B1*.

origin: Associated with *In(2LR)Px⁴*.

references: CP627.

Dp(2;2)Sco^{rv17L}C158.1^R:
see *In(2L)Sco^{rv17L}C158.1^R*

Dp(2;2)Sco2: see *Df(2L)Scol*

Dp(2;2)SM5: Duplication (2;2) Second Multiple

cytology: *Dp(2;2)42A2-3;42D;53C;58A4-B1;58F*.

origin: Associated with *In(2LR)SM5*.

references: CP627.

**Dp(2;2)SMG44: Duplication (2;2)
Seminova Mglinetz Glotoff**

cytology: *Dp(2;2)47B;48E*.

origin: γ ray induced.

references: Semenova, Mglinetz, and Glotoff, 1970, *Genetika (Moscow)* 6(4): 165-69.

Mglinetz and Semenova, 1970, *Genetika (Moscow)* 6(8): 86-94.

Dp(2;2)SMG45

cytology: *Dp(2;2)42A;55E*.

origin: γ ray induced.

references: Semenova, Mglinetz, and Glotoff, 1970, *Genetika (Moscow)* 6(4): 165-69.

Mglinetz and Semenova, 1970, *Genetika (Moscow)*

6(8): 86-94.

Dp(2;2)TE35A: see **Tp(2;2)TE35A**

Dp(2;3)17: see **Tp(2;3)17**

Dp(2;3)C328: see **T(2;3)C328**

Dp(2;3)dp: see **Tp(2;3)dp**

Dp(2;3)DTD33: see **Tp(2;3)DTD33**

Dp(2;2)GT3: see **T(2;3)GT3**

Dp(2;3)GYL2

cytology: *Dp(2;3)42C;44A;64A*.

origin: Recovered as apparent double recombinant involving *cn* from a test cross of a dysgenic-type male that was heterozygous for a maternally derived *dp b cn bw* second chromosome and a paternally derived autosome extracted from a natural population and capable of promoting male recombination. A MRF (Male Recombination Factor) chromosome, *MRF23.5*, was utilized. The duplication was recovered from a male heterozygous for *MRF23.5*.

references: Yannopoulos, Stamatis, Zacharopoulou, and Pelecanos, 1981, *Mutat. Res.* 83: 383-93.

Dp(2;3)Me: see **Tp(2;3)Me**

Dp(2;3)osp³: see **Tp(2;3)osp³**

Dp(2;3)P: see **Tp(2;3)P**

Dp(2;3)P32: see **Tp(2;3)P32**

Dp(2;3)Su-en

cytology: Insertion of two bands, presumably including *en⁺*, into 62C.

discoverer: Russell.

references: Morata, Kornberg, and Lawrence, 1983, *Dev. Biol.* 99: 27-33.

Dp(2;3)TE35A-203: see **Tp(2;3)TE35A-203**

Dp(2;3)tkv3: see **Tp(2;3)tkv3**

Dp(2;3;3)S: Duplication (2;3;3) Synthetic
new order:

61A – 89D|41 – 48C|84D – 100F.

origin: Recombination between *T(2;3)205* and *T(2;3)P8*.

discoverer: Craymer, 1979.

synonym: *Dp(2;3;3)205^PP8^D*.

genetics: Lethal when heterozygous with normal 3. Useful in selecting duplications and deficiencies in 84D-89D.

Dp(2;f)1: Duplication (2;free) 1

cytology: *Dp(2;f)21;41* (left breakpoint tentative) superimposed on *In(2LR)40F;59E*. Small metacentric, 1/3 length of the X (Grell, 1970).

new order:

21|41 – 40F|59E – 60. Tentative.

origin: X-ray-induced derivative of *In(2LR)bw^{V32g}*; possibly a deletion of most of the long arm.

discoverer: E. H. Grell, 1959.

references: CP627.

Grell, 1970, *Genetics* 64: 337-65.

genetics: Carries centromere of 2, most of the proximal heterochromatin and probably all of the loci between *bw* and the tip of 2R (Grell, 1970). Suppresses *w^{m4}* variegation.

Dp(2;f)Bl: Duplication (2;f) Bristle

cytology: *Dp(2;f)38C;41*; considerably larger than 4 in metaphase, appearing as small compact ring. Basal region triplicated in salivaries.

origin: X ray induced in *Bl* male; recovered with *C(2)EN*.

discoverer: Novitski.

synonym: *R(2)Bl*.

references: Puro and Nokkala, 1977, *Chromosoma* 63: 273-86.

Novitski and Puro, 1978, *DIS* 53: 205-06.

Falk, 1981, *DIS* 56: 37-38.

genetics: Contains *Bl* but not *pr*⁺. May show some somatic loss (Novitski and Puro, 1978; Falk, 1981).

Dp(2;f)rl⁺: Duplication (2;f) rolled

cytology: Ring with heterochromatic breaks on either side of the centromere.

origin: X ray induced in females; recovered in crosses of irradiated females with *C(2L)RM/C(2R)RM* males.

synonym: *R(2)rl*⁺.

references: Hilliker, 1980, *Genetics* 90: 85-91.

genetics: Carries *rl*⁺. Mitotically unstable in neuroblast tissue.

Dp(3;1)1A: see *Df(3R)ca1A*

Dp(3;1)3: see *Df(3R)ca3*

Dp(3;1)19: see *Df(3R)ca19*

Dp(3;1)27: see *Df(3R)ca27*

Dp(3;1)34: see *Df(3R)ca34*

Dp(3;1)35: see *Df(3R)ca35*

Dp(3;1)45B: see *Df(3R)ca45B*

Dp(3;1)46A: see *Df(3R)ca46A*

Dp(3;1)48: see *Df(3R)ca48*

Dp(3;1)52: see *Df(3R)ca52*

Dp(3;1)67A: see *Df(3R)ca67A*

Dp(3;1)67N: see *Df(3R)ca67N*

Dp(3;1)68

cytology: Insertion of 89D-E into proximal part of the X.

references: Lewis, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 155-64.

synonym: *Dp(3;1)P68*.

genetics: Rescues *Cbx Ubx* homozygotes.

Dp(3;1)74: see *Df(3R)ca74*

Dp(3;1)78: see *Df(3R)ca78*

Dp(3;1)79: see *Df(3R)ca79*

Dp(3;1)88: see *Df(3R)ca88*

Dp(3;1)93: see *Df(3R)ca93*

Dp(3;1)97: see *Df(3R)ca97*

Dp(3;1)124P: see *Df(3R)ca124P*

Dp(3;1)138P: see *Df(3R)ca138P*

Dp(3;1)150P: see *Df(3R)ca150P*

Dp(3;1)152P: see *Df(3R)ca152P*

Dp(3;1)165P: see *Df(3R)ca165P*

Dp(3;1)B152

cytology: *Dp(3;1)XR;YL;98F14*. (Kongswan *et al.*, 1986). (Kongswan *et al.*, 1986).

origin: Detachment of *C(1)RM* with *Y^{P3}D* element of *T(Y;3)B152 = T(Y;3)h3;98F*.

synonym: *Dp(3;1)Acp^h-1⁺*.

references: Kankel and Hall, 1976, *Dev. Biol.* 48: 1-24.

Morrison and MacIntyre, 1978, *Genetics* 88: 487-97.

Frisardi and MacIntyre, 1984, *Mol. Gen. Genet.*

197: 403-13.

Kongswan, Dellavalle, and Merriam, 1986, *Genetics* 112: 539-50.

genetics: Carries *ca*⁺*bv*⁺.

Dp(3;1)bxd¹¹¹: Duplication (3;1) bithoraxoid

cytology: *Dp(3;1)4D;89E3-4;90B*.

references: Lewis, 1981, ICN-UCLA Symposia on Molecular and Cellular Biology (Brown and Fox, eds.). Academic Press, New York, pp. 189-208.

genetics: Carries *bxd*⁺ inserted in X.

Dp(3;1)H135

cytology: *Dp(3;1)20F;96C*.

origin: Detachment of *C(1)RM* with *Y^{PX}D* element of *T(Y;2)H135 = T(Y;2)h21;96C*.

references: Meyerowitz and Kankel, 1978, *Dev. Biol.* 62: 112-42.

genetics: Carries wild-type alleles of *ro* and *ca*.

Dp(3;1)L127: see *Df(3R)ca-L127*

Dp(3;1)H163

cytology: *Dp(3;1)1A;98B*.

origin: X-ray-induced replacement of *y*⁺ of *Dp(1;1)L-y*⁺ with *3^D* of *T(Y;3)H63 = T(Y;3)YL;98B*.

references: Harris, 1977, *DIS* 52: 68.

genetics: Carries *ca*⁺, *Acp^h-1*⁺, and *bv*⁺; marked with *y*.

Dp(3;1)mwh⁺: Duplication (3;1) multiple wing hairs - wild type

cytology: Terminus of *3L* appended to left end of X.

origin: X-ray-induced replacement of *y*⁺ of *Dp(1;1)L-y*⁺ with *3^D* of a *T(Y;3)* broken near the end of *3L*.

discoverer: Merriam.

synonym: *Dp(3;1)FG1*.

references: Postlethwait, 1978, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 2c, pp. 359-441.

genetics: Carries *mwh*⁺.

Dp(3;1)N²⁶⁴⁻⁶: see *Tp(3;1)N²⁶⁴⁻⁶*

Dp(3;1)O5: see *Tp(3;1)O5*

Dp(3;1)P68: see *Dp(3;1)68*

Dp(3;1)P115: see *Tp(3;1)P115*

Dp(3;1)R10: see *Df(3R)ca-R10*

Dp(3;1)R14: see *Df(3R)ca-R14*

Dp(3;1)R15: see *Df(3R)ca-R15*

Dp(3;1)R87

cytology: *Dp(3;1)20F;97A*.
origin: Detachment of *C(1)RM* with the $Y^{P}X^D$ element of $T(Y;2)R87 = T(Y;3)YS;97A$.
references: Meyerowitz and Kankel, 1978, *Dev. Biol.* 62: 112-42.
genetics: Carries ca^+ but not ro^+ .

Dp(3;1)ry³⁵: see **Tp(3;1)ry³⁵**

Dp(3;1)Tpl: Duplication (3;1) Triplolethal

cytology: *Dp(3;1)* derivative of *Dp(3;Y)Tpl11*.
origin: Derived from *Dp(3;Y)Tpl11* by detachment of *C(1)RM*, $y v bb$.
synonym: *Dp(3;1)11a76*.
references: Keppy and Denell, 1979, *Genetics* 91: 421-41.
genetics: Covers *Tpl* region (83D-E); carried in combination with complementary deficiency.

Dp(3;1)w⁺67k27: see **Tp(3;1)w⁺67k27**

Dp(3;1)w^{zh}: see **Tp(3;1)w^{zh}**

Dp(3;Y)7a77: see **Dp(3;Y)Tpl7**

Dp(3;Y)11a7b: see **Dp(3;Y)Tpl11**

Dp(3;Y)awd⁺: see **YKpn**
 Described in section on *Y* derivatives.

Dp(3;Y)P92: see **Tp(3;Y)P92**

Dp(3;Y)Rg-pbx: see **Dp(3;Y)trx^D**

Dp(3;Y)Tpl7: Duplication (3;Y) Triplolethal

cytology: *Dp(3;Y)YL;82D1;83E1-2;83E4;100C6-D1-2*.
new order: $YS.YL^P|82D1 - 83E2|100D1-2 - 100F$.
origin: γ ray induced *Df(3R)83E1-4;100C6-D2* in $Y^{P}3^D$ element of $T(Y;3)B155 = T(Y;3)YL;82D1$.
synonym: *Dp(3;Y)7a77; TplY7*.
references: Keppy and Denell, 1979, *Genetics* 91: 421-41.
genetics: Covers *Tpl* region (83D-E); marked with y^+ on *YS*.

Dp(3;Y)Tpl11

cytology: *Dp(3;Y)YS;83D4-5;84F4;99E1*.
new order: $YL.YS^P|83D5 - 84F4|99E1 - 100F$.
origin: γ ray induced *Df(3R)84F4;99E1* in $Y^{P}3^D$ element of $T(Y;3)L132 = T(Y;3)h21-Xhy^+;83D4-5$.
synonym: *Dp(3;Y)11a76; TplY11*.
references: Keppy and Denell, 1979, *Genetics* 91: 421-41.
genetics: Covers *Tpl* region; marked with B^S on *YL*.

Dp(3;Y)trx^D: Duplication (3;Y) trithorax-Dominant

cytology: Insertion of the more proximal rearrangement point of *In(3R)trx^D = In(3R)85AB;88C-E* into the *Y* chromosome.
origin: X ray induced.
discoverer: E.B. Lewis.
synonym: *Dp(3;Y)Rg-pbx; TplYtrx^D*.
references: Capdevila and García-Bellido, 1981, *Wilhelm Roux's Arch. Dev. Biol.* 190: 339-50.
genetics: Shows *trx^D* phenotype.

Dp(3;Y;1)M

cytology: *1A1;h23-Xhy⁺;62AB*.
new order: $20 - 1A|h21 - h23|62A - 61A$.
origin: X-ray-induced replacement of y^+ on *Dp(1;1)L-y⁺* by 3^D of $T(Y;3)P6 = T(Y;3)62A-B$. (Three independent occurrences, *M1, M2* and *M3*).
references: García-Bellido and Ripoll, 1973, *DIS* 50: 92. Minaña and García-Bellido, 1982, *Wilhelm Roux's Arch. Dev. Biol.* 191: 331-34.
genetics: Males viable and fertile over *YL* chromosome; females homozygous lethal. Carries mwh^+ and ve^+ ; no variegation for mwh^+ in nullo-*Y* males.

Dp(3;2)dpp⁶⁰: see **Tp(3;2)dpp⁶⁰**

Dp(3;2)dpp⁶⁶: see **In(2L)dpp⁶⁶**

Dp(3;2)dpp⁸⁰: Duplication (3;2) decapentaplegic

cytology: *Dp(3;2)22F1-2;85D;86E*.
new order: $21A - 22F|85D - 86E|22F - 60F$.
origin: γ ray induced.
discoverer: Segal.
references: Gelbart.
genetics: *d-III* mutant.

Dp(3;2)FM27: see **Tp(3;2)N2-27**

Dp(3;2)MS6: see **Tp(3;2)MS6**

Dp(3;2)MS7: see **Tp(3;2)MS7**

Dp(3;2)P10: see **Tp(3;2)P10**

Dp(3;2)ry⁺: see **Tp(3;2)ry⁺**

Dp(3;3)9k75-1: see **Dp(3;3)Tpl9k1**

Dp(3;3)9k75-2: see **Dp(3;3)Tpl9k2**

***Dp(3;3)19k75:** see ***Dp(3;3)Tpl19k**

Dp(3;3)21173: see **Dp(3;3)Tpl21**

Dp(3;3)121.3 - Dp(3;3)149.14

origin: Spontaneous exchange between homologues in *Dfd kar² l(3)87Df Sb/ry⁴⁰⁰* females; recovered as apparent *l(3)87Df⁺ ry⁺* recombinants.
references: Gelbart and Chovnick, 1979, *Genetics* 92: 849-59.

duplication	cytology	exchange orient. ^α	genetics
Dp(3;3)121.3	87B;87E	?	wild type
Dp(3;3)122.3	87B;88B	A	<i>Dfd</i>
Dp(3;3)124.3	85F;88A	A	<i>Dfd</i>
Dp(3;3)124.5	87B;88F	A	<i>Dfd</i>
Dp(3;3)124.6	87D;87F	?	wild type
Dp(3;3)148.4	86E;87E	B	<i>Sb</i>
Dp(3;3)148.8	87B;87D	B	<i>Sb</i>
Dp(3;3)149.4	87E;87F	A	<i>Dfd</i>
Dp(3;3)149.9	86C;86F	B	<i>Sb</i>
Dp(3;3)149.12	86C;87F	B	<i>Sb</i>
Dp(3;3)149.14	87C;87F	B	<i>Sb</i>

^α A = *Dfd [kar² l(3)87Df ry⁺] [kar⁺ l⁺ ry⁴⁰⁰] Sb⁺*; B = *Dfd⁺ [kar⁺ l⁺ ry⁴⁰⁰] [kar² l(3)87Df ry⁺] Sb⁺*. ? = Presumably unequal exchange with second exchange either between *Dfd* and the duplication or between the duplication and *Sb*.

***Dp(3;3)Antp⁵⁰*: Duplication (3;3) Antennapedia**

cytology: *Dp(3;3)84B1-2* + extra band immediately following 84B1-2 (Hannah-Alava).

origin: X ray induced.

discoverer: Piternick, 1950.

references: Bulyzhenkov and Ivanov, 1978, *Genetika* (Moscow) 14: 456-62.

Kaufman, Lewis, and Wakimoto, 1980, *Genetics* 94: 115-33.

Lewis, Kaufman, Denell and Talerico, 1980, *Genetics* 95: 367-81.

genetics: Mutant for *Antp⁵⁰*. Homozygous lethal in first instar larvae (Bulyzhenkov and Ivanov, 1978).

Dp(3;3)Antp⁶⁰

cytology: *Dp(3;3)84B1-2* + extra band that splits 84B1-2 doublet into three thin bands.

origin: X ray induced.

discoverer: Hannah-Alava.

references: Bulyzhenkov and Ivanov, 1978, *Genetika* (Moscow) 14: 456-62.

Kaufman, Lewis, and Wakimoto, 1980, *Genetics* 94: 115-33.

Lewis, Kaufman, Denell, and Talerico, 1980, *Genetics* 95: 367-81.

genetics: Mutant for *Antp*. Homozygous lethal in first instar larvae (Bulyzhenkov and Ivanov, 1978).

Dp(3;3)Antp^{rv8}

cytology: Tandem *Dp(3;3)84D5-8;85F5-8* superimposed on *In(3R)84B1-2;84C5-6*.

new order:

61 – 84B1|84C5 – 84B2|84C6 – 85F5|84D8 – 100.

origin: X-ray-induced revertant of *Antp^{73b}*.

discoverer: Hazelrigg.

synonym: *Dp(3;3)Antp^{73b+RX6}*; *Dp(3;3)Antp^{73b+RX8}*.

references: Kempfues, Raff, Raff, and Kaufman, 1980, *Cell* 21: 445-51.

Hazelrigg and Kaufman, 1983, *Genetics* 105: 581-600.

De la Concha, Dietrich, Weigel, and Campos-Ortega, 1988, *Genetics* 118: 499-508.

genetics: Fails to complement *Antp* lethality. Covers *βTub85D* and *neu*. Seems entirely possible that duplication separable from revertant.

Dp(3;3)BK

origin: X-ray induced in *Dp(3;3)S2*.

references: Leicht and Bonner, 1988, *DIS* 67:54-56.

duplication	cytology	new order
<i>Dp(3;3)BK42</i>	<i>68E1-2;88B</i>	61A - 72D11 89E2 - 88B 68E2 - 100F
<i>Dp(3;3)BK43</i>	<i>72D;89A6-7</i>	61A - 72D11 89E2 - 89A6 72D - 100F
<i>Dp(3;3)BK51</i>	<i>87B6</i>	87B4 61A2 - 100F
<i>Dp(3;3)BK56</i>	<i>63D;87B15</i>	61A - 63D 87B4-87B15 61A2 - 100F
<i>Dp(3;3)BK63</i>	<i>61A;88D1-2</i>	61A 88D2 - 87B4 61A2 - 100F
<i>Dp(3;3)BK99</i>	<i>70D1;89D3-4</i>	61A - 72D11 89E2 - 89D3 71D1 - 100F
<i>Dp(3;3)BK113</i>	<i>70E6-8;89B16-22</i>	61A - 72D11 89E2 - 89B16 70E8 - 100F

Dp(3;3)bxd¹⁰⁰: see ***Tp(3;3)bxd¹⁰⁰***

Dp(3;3)bxd¹¹⁰: see ***Tp(3;3)bxd¹¹⁰***

Dp(3;3)C123.3

cytology: *Dp(3;3)88B-C;92D*.

references: Craymer.

Dp(3;3)C123.6

cytology: *Dp(3;3)89D;93A*.

references: Craymer.

Dp(3;3)C126

cytology: *Dp(3;3)78D;79B*; tandem repeat.

discoverer: Crosby.

references: Duncan 1982, *Genetics* 102: 49-70.

genetics: Contains two doses of *Pc⁺*.

Dp(3;3)CA44

cytology: *Dp(3;3)67C;71C*.

origin: γ ray induced.

discoverer: Ashburner.

Dp(3;3)D

origin: X ray induced.

references: Reuter, Gausz, Gyurkovics, Friede, Bang, Spierer, Hall, and Spierer, 1987, *Mol. Gen. Genet.* 210: 429-36.

duplication	cytology	genetics
<i>Dp(3;3)D1a</i>	<i>86A1;87C3-5</i>	lethal
<i>Dp(3;3)D2</i>	<i>86A1-5;86E9-F1</i>	lethal
<i>Dp(3;3)D7</i>	<i>85F8-16;87F2-7</i>	lethal
<i>Dp(3;3)D10</i>	<i>86C2-7;86E5-7</i>	slightly viable
<i>Dp(3;3)D26</i>	<i>86B4-C1;86D5-8</i>	slightly viable
<i>Dp(3;3)D45</i>	<i>86C2-7;86D8-E1</i>	viable

Dp(3;3)D1: Duplication (3;3) Duncan

cytology: *Dp(3;3)84A;85A*; tandem repeat.

references: Duncan, 1982, *Genetics* 102: 49-70.

genetics: Covers *Scr-Antp*. Enhances transformation phenotype of *ftz^{Ual}* mutants (Duncan, 1986, *Cell* 47: 297-309).

Dp(3;3)D3: Duplication (3;3) Denell

cytology: *Dp(3;3)83B;85D*; tandem repeat.

origin: γ ray induced.

references: Denell, 1976, *Genetics* 84: 193-210.

genetics: Covers *Tpl* region. Low fertility in presence of *Tpl* deficiency; lethal in combination with normal third chromosome.

****Dp(3;3)D5***

cytology: Unknown.

origin: γ ray induced.

references: Denell, 1976, *Genetics* 84: 193-210.

genetics: Recovered in combination with *Tpl* deficiency; fertility very low.

****Dp(3;3)D6***

cytology: Unknown.

origin: γ ray induced.

references: Denell, 1976, *Genetics* 84: 193-210.

genetics: Recovered in combination with *Tpl* deficiency; fertility very low.

Dp(3;3)D-U: Duplication (3;3) Denell-Ubx

cytology: *Dp(3;3)89B;90B;98B;98F*. Duplicated for both 89B-90B and 98B-98F.

new order:

61A – 98F|90B – 89B|98B – 100F.

origin: γ ray induced.

references: Denell, 1976, Genetics 84: 193-210.

genetics: Suppresses *Ubx*^{67b}.

Dp(3;3)dsx^{Drv3L}Antp^{BR}:
see **In(3R)dsx^{Drv3L}Antp^{BR}**

Dp(3;3)E2

cytology: *Dp(3;3)66D12-15;69E*.

references: Akam; Richards.

***Dp(3;3)E5**

cytology: Unknown.

origin: γ ray induced.

references: Denell, 1976, Genetics 84: 193-210.

genetics: Recovered in combination with *Tpl* deficiency; fertility very low.

Dp(3;3)E6: see *Dp(3;3)Tpl*

**Dp(3;3)E(var)88D: Duplication (3;3)
Enhancer of Variegation**

origin: *P*-element induced mutagenesis.

synonym: *Dp(3;3)E8 - Dp(3;3)E74* (Locke *et al.*).

references: Locke, Kotarski, and Tartof, 1988, Genetics 120: 181-98.

genetics: Enhance position-effect variegation in *In(1)w^{m4}*. Tandem duplications presumably revertable by unequal crossing over.

duplication	cytology
<i>Dp(3;3)E(var)88D#8</i>	88D4-6;88E4-F2
<i>Dp(3;3)E(var)88D#11</i>	88A5-12;88D6-10
<i>Dp(3;3)E(var)88D#22A</i>	88B7-C4;88E2-9
<i>Dp(3;3)E(var)88D#51</i>	88F2-88A4;88E2-9
<i>Dp(3;3)E(var)88D#74</i>	88A6-12;90E5-F2

**Dp(3;3)H⁺: Duplication (3;3)
Hairless-wild type**

cytology: *Dp(2;2)92E;96A* (Vässin).

origin: Recovered as *trans* suppressor of *H* from X-irradiated female.

discoverer: Klämbt.

synonym: *Dp(3;3)SuH²*.

references: Vässin, Vielmetter, and Campos-Ortega, 1985, J. Neurogenet. 2: 291-308.

genetics: Suppressor of *H²*.

Dp(3;3)M: Duplication (3;3) Mglinetz

origin: Recovered as apparent double recombinants from γ -irradiated *rucuca/+* males.

duplication	cytology	ref ^{α}
<i>Dp(3;3)M29</i>	66A;68F	1
<i>Dp(3;3)M30</i>	66E;74B	1
<i>Dp(3;3)M31</i>	62C;63A	1
<i>Dp(3;3)M33</i>	62C;63A	1
<i>Dp(3;3)M34</i>	63E;68E	1
<i>Dp(3;3)M35</i>	69B;75E	1
<i>Dp(3;3)M36</i>	70C;71A	1
<i>Dp(3;3)M37</i>	83C;95C	1
<i>Dp(3;3)M38</i>	92F;94D	1
<i>Dp(3;3)M39</i>	92E;95B	1
<i>Dp(3;3)M40</i>	89E;93A	1
<i>Dp(3;3)M41</i>	91A;92E	1
<i>Dp(3;3)M42</i>	88F;90E	1
<i>Dp(3;3)M43</i>	89D;90E	1
<i>Dp(3;3)M44</i>	93D;95A	1
<i>Dp(3;3)M45</i>	94F;96B	1
<i>Dp(3;3)M46</i>	93E;94F	1
<i>Dp(3;3)M47</i>	94B;96A	1

duplication	cytology	ref ^{α}
<i>Dp(3;3)M48</i>	91D;94D	1
<i>Dp(3;3)M49</i>	93B;95A	1
<i>Dp(3;3)M71</i>	68F;74C	2
<i>Dp(3;3)M72</i>	76E;78D	2
<i>Dp(3;3)M73</i>	91F;94D	2
<i>Dp(3;3)M74</i>	66B;68A	2
<i>Dp(3;3)M75</i>	86C;88A	2
<i>Dp(3;3)M76</i>	96A;97B	2
<i>Dp(3;3)M77</i>	98A;100C	2
<i>Dp(3;3)M78</i>	98F;100B	2

^{α} 1 = Mglinetz, 1972, Genetika (Moscow) 8(2): 82-92; 2 = Mglinetz, 1973, Genetika (Moscow) 9(3): 69-73.

Dp(3;3)M67C⁺: Duplication (3;3) Madrid Spain

origin: X ray induced. Recovered as *trans* suppressors of *M(3)67C⁴*; most are tandem repeats.

references: Moscoso del Prado and Ripoll, 1983, Genet. Res. 42: 59-63.

duplication	cytology	65F	<i>M(3)</i> 's covered		69E ⁵
			67C ⁴	67C ⁷	
<i>Dp(3;3)M67C*1</i>	64D-F;67D8-13	+	+	+	-
<i>Dp(3;3)M67C*2</i>	65F10-11;67E5-7	-	+		
<i>Dp(3;3)M67C*3</i>	65F10-11;67E;70A ^{α}	+	+		+
<i>Dp(3;3)M67C*4</i>	65D3-5;66C3-5;68F ^{β}	-	+	+	-
<i>Dp(3;3)M67C*5</i>	66E1-2;70A-B	-	+	+	+
<i>Dp(3;3)M67C*6</i>	67A5-9;67C5-10		+	+	
<i>Dp(3;3)M67C*7</i>	67C1-4;67D4-11		+	+	

^{α} New order: 61 - 67E|(65F11 - 70A)|67E - 100.

^{β} New order: 61 - 65D3|(66C5 - 68F)|65D5 - 100.

**Dp(3;3)M86D⁺: Duplication (3;3)
Minute-wild type**

origin: Recovered from X-irradiated *p^p* by females as *trans* suppressors of *M(3)86D*.

references: Kemphues, Raff, and Kaufman, 1983, Genetics 105: 345-56.

genetics: Carries wild-type allele of *M(3)86D*.

duplication	cytology
<i>Dp(3;3)M86D⁺-2^{α}</i>	85D1-4;87A5
<i>Dp(3;3)M86D⁺-20</i>	86B4;87E1
<i>Dp(3;3)M86D⁺-31</i>	85F1-4;86E3-5

^{α} Duplicated for β *Tub85D#(=\beta2t)*.

Dp(3;3)M95A⁺

origin: X ray induced in wild-type females. Recovered as *trans* suppressors of *M(3)95A²*.

synonym: *Dp(3;3)SuM3w*.

genetics: Cover and suppress *M(3)95A²*. When heterozygous, many of the duplications suppress crossing over.

duplication	cytology ^{α}	ref ^{β}
<i>Dp(3;3)M95A⁺1</i>	95A1	1
<i>Dp(3;3)M95A⁺1a</i>	93E;95C	2
<i>Dp(3;3)M95A⁺2</i>	94E6;95B	1
<i>Dp(3;3)M95A⁺3</i>	94E4;95D1	1
<i>Dp(3;3)M95A⁺4</i>	94A1;95A	1
<i>Dp(3;3)M95A⁺4a</i>	92E;94E	2
<i>Dp(3;3)M95A⁺5^{γ}</i>	95A1;96B1;97E	1
<i>Dp(3;3)M95A⁺6</i>	93F;96B	1
<i>Dp(3;3)M95A⁺7</i>	93D;95D	1
<i>Dp(3;3)M95A⁺8</i>	92E1;96A1	1
<i>Dp(3;3)M95A⁺9</i>	95A1;98B	1
<i>Dp(3;3)M95A⁺10</i>	91F;95A1	1
<i>Dp(3;3)M95A⁺11</i>	92E1;98A	1
<i>Dp(3;3)M95A⁺12</i>	93D5;99B1	1

duplication	cytology ^α	ref ^β
<i>Dp(3;3)M95A⁺13</i> ^δ	82F4-6;90E;98B	1
<i>Dp(3;3)M95A⁺13a</i>	94D;96A	2
<i>Dp(3;3)M95A⁺16</i>	94D;99E	2
<i>Dp(3;3)M95A⁺21</i>	93E;95C	1
<i>Dp(3;3)M95A⁺23</i>	94D;96E	1
<i>Dp(3;3)M95A⁺24</i>	92E;94E	1
<i>Dp(3;3)M95A⁺26</i>	94D;95E	1
<i>Dp(3;3)M95A⁺30</i>	92E;98C	1
<i>Dp(3;3)M95A⁺30a</i>	92E;98C	2
<i>Dp(3;3)M95A⁺33</i>	90A;94E	1
<i>Dp(3;3)M95A⁺33a</i>	90A;94E	2

^α Tandem duplications (except for #'s 5 and 13).
^β 1 = Roberts and Broderick, 1982, Genetics 102: 75-89; 2 = Väassin, Vielmetter, and Campos-Ortega, 1985, J. Neurogenet. 2: 291-308.
^γ New order: 61 - 97E|96B1 - 95A1|97E - 100.
^δ New order: 61 - 82F4|98B - 90E|82F6 - 100.

Dp(3;3)M96C⁺

genetics: Covers and suppresses *M(3)96C*.
references: González, Molina, Casal, and Ripoll.

duplication	cytology
<i>Dp(3;3)M96C⁺8</i>	96A20-25;96E
<i>Dp(3;3)M96C⁺9</i>	95B;97A
<i>Dp(3;3)M96C⁺10</i>	96B;96D
<i>Dp(3;3)M96C⁺13</i>	96B1-10;96E
<i>Dp(3;3)M96C⁺14</i>	94C7-9;98A
<i>Dp(3;3)M96C⁺16</i>	96A1-10;100F
<i>Dp(3;3)M96C⁺17</i>	96C1;96C5
<i>Dp(3;3)M96C⁺18</i>	96A20-25;99F1-4
<i>Dp(3;3)M96C⁺19</i>	94A1-5;96F
<i>Dp(3;3)M96C⁺20</i>	91B2-7;97F
<i>Dp(3;3)M96C⁺21</i>	94C7-9;97D

Dp(3;3)MS8:* see *Tp(3;3)MS8

Dp(3;3)Mtn⁺H22: Duplication (3;3) Metallothionein

cytology: *Dp(3;3)85E10;85E15* (based on *in situ* hybridization with a *Mtn* probe). Polytene chromosomes apparently normal.

origin: Spontaneous in population under selection for cadmium resistance.

discoverer: Otto and Maroni.

synonym: *Dp(3;3)Mtn⁺H22*.

references: Otto, Young, and Maroni, 1986, Proc. Nat. Acad. Sci. USA 83: 6025-29.

genetics: Homozygous viable and fertile. Shows increased tolerance to Cd ions. Can be induced to produce twice as much metallothionein RNA as wild type.

molecular biology: Tandem duplication of 2.2 kb including *Mtn⁺*. Duplicated segment starts 228 bp upstream of the transcription start site box and ends 1.4 kb downstream of the site corresponding to the end of the mRNA. Sequence analysis indicates almost exact duplication; 3' region of first repeated unit joined by a 6 bp segment to 5' region of the second unit (Otto *et al.*, 1986).

Dp(3;3)Mtn⁺H46

origin: Spontaneous in unselected laboratory strain carrying *cn* and *bw*.

references: Otto, Young, and Maroni, 1986, Proc. Nat. Acad. Sci. USA 83: 6025-29.

synonym: *Dp(3;3)Mtn^{H46}*.

molecular biology: Involves repeat of 4.6 kb of DNA which includes the entire *Mtn* transcription unit, the boundaries of the repeat being different from those of

Dp(3;3)Mtn⁺H22.

Dp(3;3)P5: Duplication (3;3) Pasadena

cytology: *Dp(3;3)89E1-2;90A*; tandem repeat.

discoverer: E.B. Lewis.

references: Duncan, 1982, Genetics 102: 49-70.

Duncan and Lewis, 1982, Developmental Order: Its Origin and Regulation (Subtelny and Green, eds.). Alan R. Liss, Inc., New York, pp. 533-34.

genetics: Covers BXC and surrounding material.

Dp(3;3)P6:* see *Tp(3;3)P26

Dp(3;3)P20:* see *Tp(3;3)P20

Dp(3;3)P26:* see *Tp(3;3)P26

Dp(3;3)S: Duplication (3;3) Synthetic

origin: Series of double duplications resulting from recombination between overlapping inversions [*In(3LR)*'s].

discoverer: Craymer.

duplication	new order	synonym
<i>Dp(3;3)S1</i> ^α	61 - 62A2 89E1 - 87B4 61A2 - 100F	<i>Dp(3;3)HR33^P Ubx^{UD}</i>
<i>Dp(3;3)S2</i> ^{βγ}	61 - 72D11 89E2 - 87B4 61A2 - 100F	<i>Dp(3;3)HR33^P bxd^{106D}</i>
<i>Dp(3;3)S3</i> ^γ	61 - 62A2 89E1 - 82A1 61A2 - 100F	<i>Dp(3;3)LD3^P Ubx^{UD}</i>
<i>Dp(3;3)S5</i>	61 - 69F 89D - 81F 67D - 100F	<i>Dp(3;3)C190^L P91^R</i>

^α Reference: Craymer, 1981, Genetics 99: 75-97.

^β Reference: Craymer, 1984, DIS 60: 234-36. Useful for selection of induced deficiencies and tandem duplication of segments in region 61A-72D.

^γ Genetics: Lethal when heterozygous over normal 3.

Dp(3;3)S2a

origin: X-ray-induced derivatives of a *Dp(3;3)S2* carrying *ru*, *Hn^{r3}-h²* and *app*.

discoverer: Craymer, 1981.

references: Craymer, 1984, DIS 60: 234-36.

duplication	cytology	new order
<i>Dp(3;3)S2a1</i>	70A-C;71A1	61-71A1 70A-C-100F
<i>Dp(3;3)S2a2</i>	66D;67D-E	61-67D 66D-100F
<i>Dp(3;3)S2a3</i>	67D9-11;68A1-2	61-68A1 67D13-100F
<i>Dp(3;3)S2a4</i>	88F;89E2 + Df(3L)72D11-E1;73A10-B1	61-72D11 89E2-88F 73B1-100F
<i>Dp(3;3)S2a5</i>	69D1-2;71D2-4	61-71D2 69D2-100F
<i>Dp(3;3)S2a8</i>	66E6-F1;69E7-F2	61-69E7 66F1-100F
<i>Dp(3;3)S2a9</i>	64C4-5;66E	61-66E 64C5-100F
<i>Dp(3;3)S2a11</i>	66B;70A	61-70A 66B-100F

Dp(3;3)S462:* see *Tp(3;3)S462

Dp(3;3)sbd¹⁰⁴:* see *Tp(3;3)sbd¹⁰⁴

Dp(3;3)SMG: Duplication (3;3) Semenova Mglinetz Glotoff

origin: γ ray induced.

references: Semenova, Mglinetz, and Glotoff, 1970, Genetika (Moscow) 6(4): 165-69.

Mglinetz and Semenova, 1970, Genetika (Moscow) 6(8): 86-94.

duplication	cytology
<i>Dp(3;3)SMG35</i>	88D;100B
<i>Dp(3;3)SMG37</i>	87C;91A
<i>Dp(3;3)SMG38</i>	62A;64B (inverted repeat)

Dp(3;3)st⁺g18: Duplication (3;3) scarlet - wild type

cytology: *Dp(3;3)72E1-2;74F4-75A1*.
new order:
 61 – 72E1|74F4 – 72E2|74F4 – 72E2|75A1 – 100.
origin: X ray induced.
references: Belote and McKeown, 1985, DIS 61: 33-34.
genetics: Carries *st⁺* in a tandem, inverted duplication.

Dp(3;3)Su⁸

cytology: *Dp(3;3)96A;96E-F*.
discoverer: Ripoll.
references: De la Concha, Dietrich, Weigel, and Campos-Ortega, 1988, Genetics 118: 499-508.
genetics: Duplication of *E(spl)⁺*.

Dp(3;3)Tpl: Duplication (3;3) Triplolethal

cytology: *Dp(3;3)83D;84B3*; reversed repeat.
origin: γ ray induced in *C(1)RM/T(Y;3)A109* female.
synonym: *Dp(3;3)E6;Dp(3;3)83D-E;Dp(3;3)21173*.
references: Denell, 1976, Genetics 84: 193-210.
 Keppy and Denell, 1979, Genetics 91: 421-41.
 Lucchesi and Roehrdanz, 1980, Genetics 95: 355-66.
genetics: Covers *Tpl* region (83D-E). Lethal over normal 3, viable and reasonably fertile over *Tpl*-deficient 3. Cell lethal in four doses (Lucchesi and Roehrdanz, 1980).
other information: Treatment of *Df(3R)83DE/Dp(3;3)Tpl* males with ethyl methanesulfonate produced three duplication chromosomes [*Dp(3;3)9k75-1*, *Dp(3;3)9k75-2*, and **Dp(3;3)19k75*] that are viable over normal 3's, but cytologically show the reversed repeat associated with *Dp(3;3)Tpl* (Keppy and Denell, 1979).

Dp(3;3)VV7: Duplication (3;3) V. Velissariou

cytology: *Dp(3;3)66E1;67A9*; reversed repeat.
new order:
 61A – 67A9|67A9 – 66E1|67A9 – 100.
origin: γ ray induced.

discoverer: Velissariou.

references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

Dp(3;4)h⁺⁴⁴: see Tp(3;4)h⁺⁴⁴

Dp(3;4)ry⁺: Duplication (3;4) rosy - wild type

cytology: *Dp(3;4)86D2-3;88B;101A-D;101F*. Appears as a ring in salivary preparations (Moscoso del Prado).
new order:
 |88B – 86D3|101F – 101A|. Tentative.
origin: X-ray-induced derivative of the 4^P3R^D element of *T(3;4)86D = T(3;4)86D2-3;101F*.
discoverer: E. H. Grell, 1960.

references: 1962, Z. Indukt. Abstamm. Vererbungslehre 93: 371-77.
 Hall and Kankel, 1976, Genetics 83: 517-35;
 Ish-Horowicz, Holden, and Gehring, 1977, Cell 121: 643-52.
genetics: Carries normal alleles of *cu*, *kar*, and *ry*. Covers *Df(3R)kar3J*. Shows tendency toward somatic elimination.

Dp(3;f)MS13: Duplication (3;f) Mglinetz Semenova

cytology: *Dp(3;f)61;80*.
origin: γ ray induced.
references: Mglinetz and Semenova, 1970, Genetika (Moscow) 6(8): 86-94.

Dp(3;f)SMG50: Duplication (3;f) Semenova Mglinetz Glotoff

cytology: *Dp(3;f)69E;99A*.
origin: γ ray induced.
references: Semenova, Mglinetz, and Glotoff, 1970, Genetika (Moscow) 6(4): 165-69.
 Mglinetz and Semenova, 1970, Genetika (Moscow) 6(8): 86-94.

INVERSIONS

In(1)-Df(1)ct: see ***In(1)ct-df***

In(1)2-4-1-1

cytology: *In(1)13E9-14;14C7-8*.
references: Kulkarni, Steinlauf, and Hall, 1988, *Genetics* 118: 267-85.

In(1)3C1^{65g}

cytology: *In(1)12D;19A*.
references: Gersh, 1967, *Genetics* 56: 309-19.

In(1)9A3

cytology: *In(1)8E;16F*.
origin: Induced with ethyleneimine.
references: Lim and Snyder, 1968, *Mutat. Res.* 6: 129-37.

In(1)24: see ***YSX.YL, In(1)24^LA2^R***
 in section on X-Y COMBINATIONS

In(1)26: see ***YSX.YL, In(1)26***
 in section on X-Y COMBINATIONS

In(1)62b12

cytology: *In(1)3B1;12F2-3*.
references: Judd, Shen, and Kaufman, 1972, *Genetics* 71: 139-56.
 Young and Judd, 1978, *Genetics* 88: 723-42.

In(1)65: Inversion (1) 65

cytology: *In(1)2C4-8;10A1-2*.
origin: X ray induced simultaneously with, but separated from, *T(1;3)65* in y.
references: Lindsley, Edington, and Von Halle, 1960, *Genetics* 45: 1649-70.
 Voelker, Greenleaf, Gyurkovics, Wisely, Huang and Searles, 1984, *Genetics* 107: 279-84.
genetics: Inseparable from y. About 1% nondisjunction and 21.8% recombination in *In(1)65/+* female; 25.9% nondisjunction and 19.7% recombination in *In(1)65/+Y* female (Grell, 1962, *Genetics* 47: 1737-54).

In(1)78b

cytology: *In(1)3A2-3;3C3-5*.
origin: X ray induced in *fa^{swb}* chromosome.
discoverer: Keppy.
references: Welshons and Welshons, 1985, *Genetics* 110: 465-77.
genetics: Male viable. Suppresses the *fa^{swb}* position effect but shows no mutant phenotype as a hemizygote, homozygote, or heterozygote with *Df(1)w67k30*. Distal break near z; proximal break between w and N.

In(1)94-2A

cytology: *In(1)1F-2A;5E-6A* (Lindsley).
origin: Derived by recombination from *C(1)94-2A*.
discoverer: Rosenfeld.
references: CP627.
genetics: Leads to partial stabilization of tandem ring compound X chromosome. Recoverable in derivative single ring, *R(1)9-4*. Exists in three interchangeable configurations in *C(1)TR* (e.g., Novitski and Braver, 1954, *Genetics* 39: 197-209).

In(1)123

cytology: *In(1)9E1;20F*.
discoverer: Nash.
genetics: Deficient for *ras*.

****In(1)272-13***

cytology: *In(1)1A6-B1;11A7-8;11F2-12A1;18A4-B1*.
new order:
 1A1 – 1A6|12A1 – 18A4|11A7 – 1B1|11A8 – 11F2|18B1 – 20.
origin: X ray induced.
discoverer: Demerec, 1940.
synonym: *In(1)l-272-13; Tp(1;1)l-272-13*.
references: Sutton, 1943, *Genetics* 28: 213.
genetics: Mutant for *sc* but not *ac* or *svr*. Male lethal.

****In(1)303-1***

cytology: *In(1)2B13-15;7B1-3;9D1-3*.
new order:
 1 – 2B13|9D1 – 7B3|2B15 – 7B1|9D3 – 20.
origin: X ray induced.
discoverer: Demerec.
synonym: *Tp(1;1)303-1*.
references: Hoover, 1938, *Z. Indukt. Abstamm. Vererbungsleh.* 74: 420-34 (fig.).
genetics: Nearly lethal.

****In(1)481***

cytology: *In(1)12E-F;14B*.
origin: X ray induced simultaneously with *Df(1)bb481*.
references: Lindsley, Edington, and Von Halle, 1960, *Genetics* 45: 1649-70.

In(1)601

cytology: *In(1)2B;16A1-2*.
origin: X ray induced.
synonym: *In(1)B^r*.
references: Brosseau, 1967, *DIS* 42: 38.
genetics: Associated with *B^{r601}*. Homozygous viable and fertile with eyes 1/2 to 2/3 normal size and resembling *L*.

In(1)1625

cytology: *In(1)3E2-3;20F*; includes nucleolus organizer.
references: Vyse and Nash, 1969, *Genet. Res.* 13: 281-87.
 Nash and Vyse, 1977, *Can. J. Genet. Cytol.* 19: 637-44;
genetics: Homozygous viable but with "weak" bristles when grown on yeast-sucrose or casein with RNA. Lethal when grown on casein medium alone.

In(1)A

inversion	cytology	origin	discoverer or ref ^α	male lethal?
<i>In(1)A1</i>	<i>16D;18D</i>	spont	5	
<i>In(1)A14</i>	<i>3B1-2;4E1</i>	X ray	3	+
<i>In(1)A74</i>	<i>1A7-8;1B8-9</i>	X ray	3	+(at 1B8-9)
<i>In(1)A78^β</i>	<i>11A1-2;20A</i>	X ray	2,3	+(at 11A1-2)
<i>In(1)A82^γ</i>	<i>12C6;14A8</i>	X ray	1,3	+
<i>In(1)A96</i>	<i>18F-19A;19B1-2</i>	X ray	2,3	+
<i>In(1)A97^β</i>	<i>10E1-2;11A1-2</i>	X ray	3	+(at 11A1-2)
<i>In(1)A99</i>	<i>7C;9A3 + 6A1-2;19C-D</i>	X ray	3	
<i>In(1)A99b^δ</i>	<i>1D3-E1;19D-E</i>	X ray	4	
<i>In(1)A101</i>	see <i>Tp(1;1)A101</i>			
<i>In(1)A141</i>	<i>11C3-4;19F1-2</i>	X ray	3	+

^α 1 = Craymer and Roy, 1980, *DIS* 55: 200-04; 2 = Konrad, Goralski, and Mahowald; 3 = Lefevre; 4 = Stone; 5 = Trench, 1981, *DIS* 56: 30.
^β Allele of *l(1)11Aa*.

γ Induced with $Df(1)A82 = Df(1)AD5-6;4E3$, but separable from it.
 δ Viability, fertility, and egg hatch good.

In(1)AB

cytology: *In(1)9F;13F1-10*.

discoverer: Bodeman.

references: Stone and Thomas, 1935, *Genetica* 17: 170-84.

genetics: Primary nondisjunction 0.5%, secondary 29.3%; recombination 18.2% in *In(1)AB/+* and 26.3% in *In(1)AB/+Y* female (Grell, 1962, *Genetics* 47: 1737-54). Stone and Thomas (1935) obtained 14.3% recombination in *In(1)AB/+*.

In(1)ac³: Inversion (1) achaete

cytology: *In(1)1B2-3;1B14-C1*.

origin: X ray induced.

discoverer: Dubinin, 1929.

synonym: *In(1)sc¹⁰*.

references: 1930, *Zh. Eksperim. Biol.* 6: 300-24.

1932, *J. Genet.* 25: 163-81.

1933, *J. Genet.* 27: 447.

Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, *Cell* 40: 327-38.

Villares and Cabrera, 1987, *Cell* 50: 415-24.

genetics: Associated with *ac³*.

molecular biology: Breakpoint at DNA coordinate 58.8 or 59.6 (Campuzano *et al.*, 1985).

In(1)AC2: Inversion (1) Adelalde Carpenter

cytology: *In(1)9D5-E1;13B5-6*.

origin: Spontaneous.

discoverer: A. Carpenter.

In(1)AM

cytology: *In(1)8C17-D1;16E2-3* (Hoover).

discoverer: Mackensen.

references: Stone and Thomas, 1935, *Genetica* 17: 170-84.

Hoover, 1938, *Z. Indukt. Abstamm. Vererbungsl.* 74: 420-34 (fig.).

genetics: Homozygous female sterile and therefore inversion used as an X chromosome balancer. Inversion departs slightly from wild-type phenotype in that eyes are rounded and slightly bulging. Total recombination 3.8% in *In(1)AM/+* (Stone and Thomas, 1935).

In(1)At: Inversion (1) Attenuated

cytology: *In(1)16A4-5;18C4-6;20A2-3* superimposed on *In(1)1B3-4;20F^L1B2-3;20F^R + In(1)4D7-E1;11F2-4*.

new order:

1A – 1B3|20F – 20A3|16A5 – 18C4|20A2 – 18C6|16A4 – 11F4|4E1 – 11F2|4D7 – 1B3|20F.

origin: X ray induced in *In(1)sc^{SIL}sc^{8R} + In(1)dl-49*.

discoverer: Valencia and Valencia, 1949.

synonym: *Tp(1;1)At*.

references: 1949, *DIS* 23: 64.

genetics: Associated with *At*. Male and homozygous female viable and fertile.

****In(1)B²⁶³⁻⁵: Inversion (1) Bar***

cytology: *In(1)15F9-16A1;16A7-B1;17A3-4*. Left break occurs between repeated regions associated with $Dp(1;1)B = Dp(1;1)15F9-16A1;16A7-B1$ (Kaufmann and Sutton).

new order:

1 – 16A7|17A3 – 16A1|17A4 – 20.

origin: X ray induced in *B*.

discoverer: Demerec, 33k.

references: Sutton, 1943, *Genetics* 28: 97-107.

genetics: *B* reversed; lethal; *un*, *vb*, *f*, *lh*, and *os^o* not affected.

****In(1)B²⁶³⁻²⁴***

cytology: *In(1)10C2-D1;12D2-E1;15F9-16A1;16A7-B1*; right breakpoint between first and second segments of B^iB^i triplication.

new order:

1 – 10C2|16A7 – 12E1|10D1 – 12D2|16A1 – 16A7|16A1 – 20.

origin: X ray induced in $Dp(1;1)B^iB^i = Dp(1;1)15F9-16A1;16A7-B1$.

discoverer: Demerec, 34a.

synonym: *Tp(1;1)B²⁶³⁻²⁴*.

references: Sutton, 1943, *Genetics* 28: 97-107.

genetics: Reversal of B^iB^i to wild type; *un*, *vb*, *f*, *lh*, and *os* not affected. Male lethal.

****In(1)B²⁶³⁻⁴⁷***

cytology: *In(1)16A2-4;20A2-3*.

origin: X ray induced.

discoverer: Demerec, 38d.

references: Sutton, 1943, *Genetics* 28: 97-107.

genetics: Position effect at *B*.

In(1)B^{M1}: Inversion (1) Bar of Muller

cytology: *In(1)16A2-5;20D-F* (Lefevre).

origin: X ray induced.

discoverer: Muller, 34e.

references: 1935, *DIS* 3: 29.

genetics: Position effect at *B*. *In(1)B^{M1}/0* males are *bb* and variegate for *pdf* (Schalet, 1969, *DIS* 44: 87). Primary nondisjunction 0.4 and secondary 18.5%; recombination 32% in *In(1)B^{M1}/+* and 35.4% in *In(1)B^{M1}/+Y* female (Grell, 1962, *Genetics* 47: 1737-54). Males reared at 18° show diffuse polytene X. (Lakhotia and Mishra, 1983, *Indian J. Exp. Biol.* 20: 643-51).

In(1)B^{M2}

cytology: *In(1)16A2-5;20F* (Sutton, 1943, *Genetics* 28: 97-107).

origin: X ray induced.

discoverer: Muller, 34e.

references: 1935, *DIS* 3: 29.

genetics: Position effect at *B*.

****In(1)B^{rv2}: Inversion (1) Bar-reversed***

cytology: *In(1)3F8-4A1;16A2-4*; right break in right section of $Dp(1;1)B = Dp(1;1)15F9-16A1;16A7-B1$.

new order:

1 – 3F8|16A2 – 16A1|16A7 – 4A1|16A4 – 20.

origin: X ray induced in $Dp(1;1)B$.

discoverer: Bishop, 1940.

references: Sutton, 1943, *Genetics* 28: 100.

genetics: Reversal of *B*.

****In(1)B^{rv3}***

cytology: *In(1)15F9-16A1;16A7-B1;20A5-F*; right break between segments of $Dp(1;1)B = Dp(1;1)15F9-16A1;16A7-B1$.

new order:

1 – 16A7|20A5 – 16A1|20F.

origin: X ray induced in *B*.

discoverer: Bishop, 1940.
references: Sutton, 1943, Genetics 28: 100.
genetics: *B* reversion.

In(1)B1-4

cytology: *In(1)1A7-8;1F3-4*.
origin: Induced by *I-R* interaction.
references: Pallison, 1981, Mol. Gen. Genet. 183: 123-29.
genetics: Male lethal. Deficient for *y* and *ac*.

In(1)bb^{Df}: Inversion (1) bobbed-Deficiency

cytology: *In(1)4D2-3;20F*; deficiency in 20F; two-thirds normal length at metaphase.

new order:

1 – 4D2|20F – 4D3|20F.

origin: X ray induced.

discoverer: Sivertzev-Dobzhansky and Dobzhansky, 31b.

references: 1933, Genetics 18: 173-92.

Sturtevant and Beadle, 1936, Genetics 21: 554-604.

genetics: Right breakpoint between *rb* and *rg*. Deficient for *bb*. *In(1)bb^{Df}/+* female produces about 2% exceptional sons from 4-strand double exchange within inverted segment. Secondary exceptions about 13%.

In(1)bb^{DfL}Cl^R: Inversion (1) bobbed-Deficiency Left Cl-Right

cytology: *In(1)4D2-3;20F^L4A5-B1;17A6-B1^R*; duplicated for 4B1-D2 and 17B1-20F.

origin: Recombinant containing left end of *In(1)bb^{Df}* and right end of *In(1)Cl*.

references: Sturtevant and Beadle, 1936, Genetics 21: 554-604.

genetics: Duplicated for *bi*, *rb*, *fu*, and *car* but not *ec*, *rg*, *f*, *os*, or *bb*. Survives as small male with less convex outer wing margins than normal and usually one or more notches at wing tips; sterile, has collapsed testes. Heterozygous female fertile, has slightly narrowed wings.

In(1)bb^{DfL}y^{4R}: Inversion (1) bobbed-Deficiency Left yellow-4 Right

cytology: *In(1)4D2-3;20F^L1A8-B1;18A3-4^R*; duplicated for 1B1-4D2 and 18A4-20F.

origin: Recombinant containing left end of *In(1)bb^{Df}* and right end of *In(1)y⁴*.

references: Sturtevant and Beadle, 1936, Genetics 21: 554-604.

genetics: Duplicated for *ac* through *rb* and *fu* through *car*. Heterozygous female has stubby posterior verticals and disarranged scutellars; outer wing margin less convex than normal; fair viability and fertility. Enhances expression of heterozygous *B*. Male lethal.

In(1)br¹⁰³: Inversion (1) broad

cytology: *In(1)2B3-4;3C1* (or *3C2-3*).

origin: Induced by ethyl methanesulfonate.

references: Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90.

genetics: Break in 2B3-4 associated with lethal effect.

****In(1)Br: Inversion (1) Bridged***

origin: X ray induced.

discoverer: Muller, 2713.

references: 1935, DIS 3: 29.

genetics: Associated with dominant mutant, *Br*. Crossing over suppressed to right of *v*, about normal to left.

In(1)C10 – In(1)C212

inversion	cytology	origin	discoverer	ref ^α
<i>In(1)C10</i>	3A;5E	EMS		1
<i>*In(1)C18^β</i>	3F;17A1-6	X ray	Roberts, 1964	2
<i>In(1)C88^γ</i>	5E4-7;20	X ray	Lefevre	
<i>In(1)C99^γ</i>	2E3;20	X ray	Lefevre	
<i>In(1)C146^β</i>	1F;14A	X ray	Roberts, 1965	2
<i>In(1)C171^γ</i>	3C5;20A	X ray	Lefevre	
<i>In(1)C206^{δe}</i>	8F;11A;16A	X ray	Roberts, 1965	2
<i>In(1)C212</i>	see <i>T(1;2)C212</i>			

^α 1 = Lim and Snyder, 1968, Mutat. Res. 6: 129-37; 2 = Roberts, 1970, Genetics 65: 429-48.

^β Eliminates *sc-f* recombination. Male fertile.

^γ Male lethal.

^δ 1 – 8F|16A1 – 11A1|8F – 11A|16A – 20.

^e Eleven percent recombination between *sc* and *f*. Male lethal.

In(1)CA32

cytology: *In(1)4F1-2;6A*.

origin: γ ray induced.

discoverer: Ashburner.

genetics: Associated with *osp²⁰¹*.

In(1)ci-x: Inversion (1) cubitus

cytology: *In(1)1A;20F*.

origin: Spontaneous.

references: Muller, 1934, DIS 2: 90.

Brosseau, 1969, DIS 44: 45.

genetics: Homozygous viable and fertile. Associated with *y^{ci-x}*, an extreme *y* allele. Enhances *ci*.

In(1)Cl: Inversion (1) Cl

cytology: *In(1)4A5-B1;17A6-B1* (Hoover, 1938, Z. Indukt. Abstamm. Vererbungsl. 74: 429).

origin: Spontaneous in a *sc t² v sl B* chromosome.

discoverer: Muller, 20j.

references: 1928, Genetics 13: 279-357.

Gershenson, 1935, J. Genet. 30: 115-25.

Sturtevant and Beadle, 1936, Genetics 21: 554-604.

genetics: Left break between *ec* and *bi*; right break between *os* and *fu*; *l(1)C* associated with left break (Muller). About 0.35% primary and 37% secondary exceptions. Total recombination about 1%.

other information: *In(1)Cl*, *sc l(1)C t² v sl B* is the *CIB* chromosome, described in the section on balancers.

In(1)Cl^Lbb^{DfR}: Inversion (1) Cl-Left bobbed-Deficiency Right

cytology: *In(1)4A5-B1;17A6-B1^L4D2-3;20F^R*; deficient for 4B1-D2 and 17B1-20F.

origin: Recombinant containing left end of *In(1)Cl* and right end of *In(1)bb^{Df}*.

references: Sturtevant and Beadle, 1936, Genetics 21: 554-604.

genetics: Deficient for *bi*, *rb*, *fu*, *car*, and *bb* but not *ec*, *rg*, *f*, or *os*. Both *bi* and *fu* lethal when heterozygous for *In(1)Cl^Lbb^{DfR}*. Heterozygous female extreme Minute [*M(1)18C*], with abnormal wing shape; ovaries normal but female sterile. Male lethal.

***In(1)Cl^Ly^{4R}*: Inversion (1) Cl-Left
yellow-4 Right**

cytology: *In(1)4A5-B1;17A6-B1^L1A8-B1;18A3-4^R*; duplicated for 1B1-4A5, deficient for 17B1-18A3.

origin: Recombinant containing left end of *In(1)Cl* and right end of *In(1)y⁴*.

references: Sturtevant and Beadle, 1936, Genetics 21: 554-604.

genetics: Duplicated for *ac* through *ec*; deficient for *fu* but not *f*, *vb*, *os*, or *car*. Heterozygous female has irregular acrostichal rows and wings smaller and with less-convex posterior margin than normal. Enhances expression of heterozygous *B*. Male lethal.

***In(1)cm-df*: Inversion (1)
carmine-deficiency**

cytology: *In(1)3D;6D + Df(1)6D;6F*.

origin: Derived from an unstable *X* carrying a lethal (*Uc-1^{UJD15}*).

synonym: *Df(1)cm-In*.

references: Johnson-Schlitz and Lim, 1987, Genetics 115: 701-09.

Lim, 1988, Proc. Nat. Acad. Sci. USA 85: 9153-57.

genetics: Deficient for *cm*. Produces *N* deficiencies at high rate.

molecular biology: Chromosome carries five *hobo* transposing elements (Lim, 1988).

***In(1)ct*: Inversion (1) cut**

origin: X ray induced.

genetics: All male lethal except for *In(1)ct^{43aH1}* and *In(1)ct^{J7}* (semilethal). *In(1)ct^{3a2}* and *In(1)ct^{J7}* give extreme *ct* in combination with *ct⁶*.

inversion	cytology	ref ^α
* <i>In(1)ct^{3a2}</i>	7B;20	2, 3
* <i>In(1)ct^{3b1}</i>	3A4-B1;7B2-5	2
* <i>In(1)ct^{12a2}</i>	4E2-3;7B2-4	1
* <i>In(1)ct^{13a1}</i>	7B2-3;19-20	2
* <i>In(1)ct^{14a3}</i>	7B2-3;20	2
* <i>In(1)ct^{14b2}</i>	3D2-5;7B2-4	2
<i>In(1)ct^{43aH1}</i>	4B1-4;7B4-C1 + 10D5-6;20F	7
* <i>In(1)ct^{268-13β}</i>	2E3-F1;2F2-3;7B2-3; 7B4-5;19A4-5;19A6-B1 superimposed on <i>R(1)1A3-4;19F-20A1</i> (2F1-2,7B3-4,19A5-6 missing)	4, 5
* <i>In(1)ct^{268-18γ}</i>	7B2-3;7B4-5;11D8-9 (7B3-4 missing)	5
* <i>In(1)ct^{268-20δ}</i>	6F11-7A1;7B3;10B11-12 (7A1-B5 missing)	5
* <i>In(1)ct²⁶⁸⁻²⁷</i>	3D6-E1;7B3-5	5
* <i>In(1)ct²⁶⁸⁻²⁷</i>	3D6-E1;7B3-5	5
* <i>In(1)ct^{J7}</i>	7B3-4;7D22	6
* <i>In(1)ct^{J12}</i>	7B3-4;7D4	6
* <i>In(1)ct^{J13}</i>	5A;7B3-4	6

^α 1 = De Frank; 2 = Hannah-Alava; 3 = Hannah-Alava, 1971, Mol. Gen. Genet. 113: 191-203; 4 = Hoover, 1937, Genetics 22: 634-40; 5 = Hoover, 1938, Z. Indukt. Abstamm. Vererbungsl. 74: 420-34; 6 = Lefevre and Johnson, 1973, Genetics 74: 633-46; 7 = Valencia, 1966, DIS 41: 58.

^β New order: |1A1 - 2E3|7B2 - 2F3 |19A4 - 7B5 |19B1 - 20.20 - 20A1|.

^γ New order: 1A - 7B2|11D8 - 7B5|11D9 - 20.

^δ New order: 1A - 6F11|10B11 - 7B6|10B12 - 20.

In(1)ct⁷⁸

cytology: *In(1)6A1-B1;6F1-2;7C1-2;19A1-4;* contains reversed repeat of 6F2 to 7C1.

new order:

1A1 - 6A1|19A1 - 6F2|6F2 - 7C1|
6F1 - 6B1|19A4 - 20.

origin: Derived from unstable *X* chromosome with an easily mobilized *gypsy* element at *ct*.

references: Lim, 1979, Genetics 93: 681-701.

genetics: Unstable.

***In(1)ct-df*: Inversion (1)
cut-deficiency**

cytology: *In(1)3D;6F + Df(1)6F;7C*.

origin: Derived from an unstable *X* carrying a lethal (*Uc-1^{UJD15}*).

synonym: *In-Df(1)ct*.

references: Johnson-Schlitz and Lim, 1987, Genetics 115: 701-09.

genetics: Deficient for *ct*.

***In(1)D1*: Inversion (1) from
deoxycytidine**

cytology: *In(1)13B;16A*.

origin: Induced by tritiated deoxycytidine.

discoverer: Kaplan, 1965.

references: 1966, DIS 41: 59.

genetics: Male lethal.

In(1)D30

cytology: *In(1)14C6-D1;15E-F*.

synonym: *In(1)l^{D30}*.

references: Ganetzky, 1984, Genetics 108: 897-911.

genetics: Distal break associated with *l(1)14Dc¹*.

***In(1)dl-49*: Inversion (1) delta-49**

cytology: *In(1)4D7-E1;11F2-4* [Painter; Hoover, 1938, Z. Indukt. Abstamm. Vererbungsl. 74: 420-34 (fig.)].

discoverer: Muller, 26k.

references: Muller and Stone, 1930, Anat. Record 47: 393-94.

Stone and Thomas, 1935, Genetica 17: 170-84.

Sturtevant and Beadle, 1936, Genetics 21: 554-604.

genetics: Left break between *rb* and *cv*; right between *wy* and *g*. Measures of recombination vary from 5.5% (Grell, 1962, Genetics 47: 1737-54) to about 15% (Sturtevant and Beadle, 1936); secondary exceptions from 33% (Grell, 1962) to 44% (Sturtevant and Beadle, 1936).

other information: Used as a balancer either with markers *y Hw m² g⁴* or *y w lz^s* with *Hw* and *lz^s* sterilizing homozygous female.

***In(1)dm75e*: Inversion (1) diminutive**

cytology: *In(1)3E;5E + Df(1)3C11;3E4*.

origin: X ray induced.

discoverer: Lefevre.

references: Craymer and Roy, 1980, DIS 55: 200-04.

genetics: Deficient for *dm*.

***In(1)dor^{var2}*: Inversion (1) deep orange**

cytology: *In(1)2A4-B1;12F-13F*.

new order:

1A - 2A4|12F - 2B1|13F - 20.

origin: γ ray induced.

references: Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS

58: 184-90.

genetics: Inversion males lethal with *Dp(1;f)18* or *Dp(1;f)112*, viable and normal with *Dp(1;f)101*, $y^{39b}Y(2)$, or $y^2Y67g24.2$. Females heterozygous for *BRC* mutants, *dor*, or *hfw* show strong position effect.

***In(1)dor*^{var7}**

cytology: *In(1)2B5-6;20* + additional rearrangements between *X* and 2.

origin: γ ray induced.

references: Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90.

genetics: Females heterozygous with *hfw*, *dor*, *BRC* mutants, and *sta* but not *l(1)2Bk* or *l(1)2Bl* show strong position effect.

In(1)drp: Inversion (1) droop wings

cytology: *In(1)12B;20B*.

origin: Spontaneous from *hi*.

discoverer: Ives, 48f.

synonym: *In(1)hi1; Inversion (1) droop*.

references: 1949, DIS 23: 58.

genetics: Associated with mutant droop wings. Male viable.

****In(1)dta: Inversion (1) delta wing***

cytology: *In(1)6B2-3;15E7-F2*.

origin: Induced by triethylenemelamine.

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 69.

genetics: Associated with *dta*. Female sterile.

In(1)EC238

cytology: *In(1)3D5-6;4C1*.

origin: Induced by ethyl methanesulfonate.

discoverer: Lefevre.

genetics: Male lethal. Allele of *dm*.

***In(1)elav*⁴**

cytology: *In(1)1A7;1B8*.

synonym: *In(1)elav*^{G3}.

references: Campos, Rosen, and White, 1986, Genetics 113: s27.

In(1)EN: Inversion (1) Entire

cytology: *In(1)1A;20F;20F*. At prophase, distal end carries a single heterochromatic segment about equal in size to chromosome 4; proximally, it carries a very short heterochromatic segment and as a second arm two larger heterochromatic segments (Lindsley).

new order:

20 – 1A|20C – 20F·20.

Tentative.

origin: Spontaneous opening out of *R(1)1*, *y*.

discoverer: Novitski.

references: 1949, DIS 23: 94-95.

Lindsley, 1958. Z. Indukt. Abstamm. Vererbungsl. 89: 103-22.

Lindsley and Novitski, 1959, Genetics 44: 187-96.

genetics: Entire chromosome, including the lethally mutable loci distal to *y*, inverted. Carries mutant alleles of *bb* at each end, which acting together produce *bb*⁺ phenotype. Male viable and fertile.

In(1)EN2

cytology: *In(1)1A3-4;19F-20A;20F*. Inferred from origin.

new order:

20 – 1A3|20A – 20F·20.

Tentative.

origin: Spontaneous opening of *R(1)2*, *y*⁺.

discoverer: Muller.

references: 1956, DIS 30: 140-41.

genetics: Entire chromosome inverted like *In(1)EN* but carries *y*⁺ rather than *y*.

****In(1)EN2B: Inversion (1) Entire 2 of Bender***

cytology: *In(1)1A3-4;19F-20A;20F*. Inferred from origin.

new order:

20 – 1A3|20A – 20F·20F.

Tentative.

origin: Spontaneous opening of *R(1)2*, *y* v.

discoverer: M. A. Bender, 55e6.

references: 1955, DIS 29: 69.

****In(1)exr: Inversion (1) extra venation***

cytology: *In(1)12E8-10;15D1-3*.

origin: Induced by triethylenemelamine.

discoverer: Fahmy, 1952.

references: 1958, DIS 32: 70.

genetics: Affects *exr*.

***In(1)f*^{67a}: Inversion (1) forked**

cytology: *In(1)12E;18B + In(1)15E;20*.

origin: X ray induced.

synonym: *Tp(1;1)f*^{67a}.

references: Becker, 1968, DIS 43: 59.

genetics: Mutant for *f*. Homo- and hemizygous viable.

***In(1)f*²⁵⁷⁻⁴**

cytology: *In(1)15F2-16A1;16D2-E1*.

origin: X ray induced.

discoverer: Demerec, 33j.

genetics: *f* affected.

In(1)FM3: Inversion (1) First Multiple

cytology: *In(1)3E-F;16A-B;19F-20F*; superimposed on *In(1)1B2-3;20F + In(1)4D7-E1;11F2-4*.

new order:

1A – 1B2|20F|16B – 19F|3F – 4D7|

11F2 – 4E1|11F4 – 16A|3E – 1B3|20F.

origin: X ray induced in *In(1)sc*⁸ + *In(1)dl-49*, $y^{31d} sc^8 dm B$.

discoverer: R. F. Grell, 1954.

references: Mislove and Lewis, 1954, DIS 28: 77.

genetics: Mutant for two lethals, one allelic to *l(1)1Ac* and therefore covered by y^+Y and the other covered by B^SY ; both *In(1)FM3/y^+Y/B^SY* and *In(1)FM3/B^SY y^+* males survive. The treated chromosome carried y^{31d} , but *In(1)FM3/y* variegates for yellow bristles.

other information: Used as a first chromosome balancer, described as *FM3* in the section on balancers.

In(1)FM4

cytology: *In(1)3C;4E-F* superimposed on *In(1)1B2-3;20F + In(1)4D7-E1;11F2-4*.

new order:

1A – 1B2|20F – 11F4|4E|3C – 4D7|

11F2 – 4F|3C – 1B3|20F.

origin: X ray induced in *In(1)sc*⁸ + *In(1)dl-49*, $y^{31d} sc^8$

dm B.

discoverer: R. F. Grell, 1954.

references: Mislove and Lewis, 1954, DIS 28: 77.

other information: Used as a first chromosome balancer, described as *FM4* in the section on balancers.

In(1)FM6

cytology: *In(1)15D-E;20F* superimposed on *In(1)1B2-3;20F + In(1)3C;4E-F + In(1)4D7-E1;11F2-4*.

new order:

1A – 1B2|20F|15E – 20F|15D – 11F4|4E|
3C – 4D7|11F2 – 4F|3C – 1B3|20F.

origin: X ray induced in *In(1)FM4*, $y^{31d} sc^8 dm B.$

discoverer: R. F. Grell, 55i.

references: Grell and Lewis, 1956, DIS 30: 70.

other information: Used as first chromosome balancer, described as *FM6* in the section on balancers.

In(1)FM7

cytology: *In(1)1B2-3;20F + In(1)4D7-E1;11F2-4 + In(1)15D-E;20A-E*. Salivaries of females heterozygous with *In(1)sc⁸ In(1)dl-49* show complete synapsis, with the delta-49 region homozygous and an inversion loop from 15D-E to the chromocenter.

new order:

1A1-1B2|20F-20A|15D-20A|15D-11F4|4E1-
11F2|4D7-1B3|20F.

origin: Crossing over in *FM6/In(1)sc⁸ In(1)dl-49* female.

synonym: *FM7a*.

references: Merriam, 1968, DIS 43: 64.

1969, DIS 44: 101.

other information: Used as a first chromosome balancer, described as *FM7* in the section on balancers.

In(1)FM7^LEN^R

origin: Single exchange between y^{+YSX} , *In(1)FM7* and *X-YLB^S*, *In(1)EN* to yield *YSX-YL*, *In(1)FM7^LEN^R* in which the euchromatin from the *X* terminus through *sc* is replaced by *YS*.

discoverer: Craymer.

synonym: $y^{+YSX-YLB^S}, In(1)FM7^LEN^{R}}$.

references: 1974, DIS 51: 21.

genetics: Males and homozygous females viable and fertile. Carries y^{+}, y, v^{Of} , and $B^{S}}$.

In(1)g¹⁷: Inversion (1) garnet

cytology: *In(1)12B10;19F* superimposed on *In(1)1B3-4;20F^L1B2-3;20F^R + In(1)4D7-E1;11F2-4*.

new order:

1A – 1B3|20F – 19F|12B10 – 19F|
12B10 – 11F4|4E1 – 11F2|4D7 – 1B3|20F.

origin: X ray induced in *In(1)sc^{SIL}sc^{8R} + In(1)dl-49*.

discoverer: Muller, Valencia, and Valencia, 1946-53.

references: Valencia, 1966, DIS 41: 58.

genetics: Associated with g^{17} .

In(1)g^w: Inversion (1) garnet-wild

cytology: Breakpoints unknown.

origin: X ray induced.

discoverer: Muller.

references: 1946, DIS 20: 67.

Chovnick, 1958, DIS 32: 88.

1961, Genetics 46: 493-507 (fig.).

genetics: Associated with g^{w} .

In(1)g^X: Inversion (1) garnet from X irradiation

cytology: *In(1)12;19-20*.

origin: X ray induced.

discoverer: Muller.

references: 1946, DIS 20: 67.

genetics: Mutant for *g*.

In(1)GA106

cytology: *In(1)2B6;20F*.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal. Mutant for *br*.

In(1)GE

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal.

inversion	cytology	genetics
<i>In(1)GE224</i>	3A;20F	<i>l(1)3Ac⁸³</i>
<i>In(1)GE228</i>	3B1;20A	<i>l(1)3Ba²⁴</i>
<i>In(1)GE246^α</i>	2A-B;3D5-6	<i>l(1)3Da²</i>
<i>In(1)GE257</i>	3B3;20F	<i>l(1)3Ba²⁵</i>

^α Complex.

In(1)GEM224: see T(1;2)GEM64

In(1)GJ4

cytology: *In(1)1B6-7;3C1-2*.

references: Gelbart, 1971, Ph.D. Thesis, Univ. of Wisconsin.

genetics: Associated with *Dp(1;1)GJ4*.

In(1)GJ5

cytology: *In(1)1B;10A*.

references: Gelbart, 1971, Ph.D. Thesis, Univ. of Wisconsin.

genetics: Associated with *Dp(1;1)GJ5*.

In(1)Gr^{Z+}: Inversion (1) Green

cytology: *In(1)3C;12B-C*.

origin: X ray induced.

references: Green, 1967, Biol. Zentralbl. 86: 211-20.

genetics: Eye color wild type.

In(1)HA46

cytology: *In(1)7B1-2;8C-D*.

origin: X ray induced.

references: Lefevre and Leeds, 1983, Genetics 104: s45-46.

genetics: Male lethal, but strong *ct* over *ct⁶*. Viable with *ct¹* or *Df(1)RF19*, showing strong *kf²* phenotype.

In(1)HC

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal.

inversion	cytology	lethal at	genetics
<i>In(1)HC146</i>	11A1-2;12A	11A1-2, 12A	<i>l(1)11Aa⁷</i>
<i>In(1)HC206</i>	1E1-2;2A1-2	2A1-2	<i>l(1)2Ae⁴</i>
<i>In(1)HC207</i>	2C3;7B1	2C3	
<i>In(1)HC293</i>	6E1;7F1-2	7F1-2	

***In(1)hi: Inversion (1) high**

origin: Spontaneous in *hi*.
discoverer: Ives.
references: Hinton, Ives, and Evans, 1952, *Evolution* 6: 19-28.
genetics: Male lethal.

inversion	cytology
*In(1)hi2	1F;20
*In(1)hi3	4D;20
*In(1)hi4	4C;20
*In(1)hi5	1F;20
*In(1)hi7	12E;20
*In(1)hi8	3C;20
*In(1)hi9	8F;20
*In(1)hi10	4E2-3;8A1-2
*In(1)hi11 ^{αβ}	5C;7E;20A-F
*In(1)hi12	1C3;20
*In(1)hi13	4E;20

^α Synonym: *Tp(1;1)hi11*.

^β New order: 1-5C|7E-20A|7E-5C|20A-F.

In(1)hi1: see *In(1)drp*

***In(1)Hv: Inversion (1) Hooked veins**

cytology: Breakpoints unknown.
discoverer: Tanaka, 35a4.
references: 1937, DIS 8: 11.
genetics: Associated with *Hv*.

In(1)Hw²: Inversion (1) Hairy wing

cytology: *In(1)1A2-3;1A8-B1;1B2-3*.
new order:
 1A1-1A2|1B2-1A3|1B1-20.
origin: Spontaneous derivative of *Dp(1;1)Hw* = *Dp(1;1)1A8-B1;1B2-3*.
discoverer: Nichols-Skoog, 35a9.
genetics: Associated with *Hw*².

In(1)Hw^{49C}

cytology: *In(1)1B;2B3-4*.
references: Belyaeva, Protopopov, Baricheva, Semeshin, Izquierdo, and Zhimulev, 1987, *Chromosoma* 95: 295-310.
genetics: Inversion has weak *br* effect.
molecular biology: Right break within *BRC* at coordinate 115 kb (Sampedro, Galcerán, and Izquierdo, 1989, *Mol. Cell. Biol.* 9: 3588-91).

In(1)IW1: Inversion (1) Inoue Watanabe

cytology: *In(1)5A;16E*.
origin: Spontaneous in natural population in Japan.
references: Inoue and Watanabe, 1969, *Jpn. J. Genet.* 54: 69-82.

In(1)IW2

cytology: *In(1)5E;10C*.
origin: Spontaneous in a natural population in Japan.
references: Inoue and Watanabe, 1969, *Jpn. J. Genet.* 54: 69-82.

In(1)JA9

cytology: *In(1)7A3;20F*.
origin: X ray induced.
discoverer: Lefevre.
references: Nicklas and Cline, 1983, *Genetics* 103: 619-31.
genetics: Male lethal. Associated with *l(1)7Aa*².

In(1)JA11

cytology: *In(1)7B3-4;7E5*.
origin: X ray induced.
discoverer: Lefevre.

In(1)JC

origin: X ray induced.
discoverer: Lefevre.
genetics: Male lethal.

inversion	cytology	genetics
<i>In(1)JC31</i>	1E5;3A1-2	<i>l(1)3Ac</i> ⁸⁸
<i>In(1)JC43</i>	see <i>T(1;4)JC43</i>	
<i>In(1)JC46</i>	3A4;4E	<i>l(1)3Ac</i> ⁸⁹
<i>In(1)JC111</i>	2C3;9A2-3	<i>l(1)2Cb</i> ⁷

***In(1)K2: Inversion (1) of Krivshenko**

cytology: *In(1)6A;9A-B*.
origin: Spontaneous.
discoverer: Krivshenko, 54c24.
references: 1956, DIS 30: 75.
genetics: Homozygous viable.

In(1)l3Ac²⁷: Inversion (1) lethal

cytology: *In(1)3A3-4;6B2-3*.
origin: X ray induced.
discoverer: Judd, 62g31.
synonym: *In(1)g31*.
references: Judd, Shen, and Kaufman, 1972, *Genetics* 71: 139-56.
genetics: Associated with *l(1)3Ac*²⁷.

In(1)l3Ba¹

cytology: *In(1)3B1;12F2-3*.
origin: X ray induced.
discoverer: Judd, 62b12.
genetics: Associated with *l(1)3Ba*¹.

**In(1)l-272-13*: see *In(1)272-13*

In(1)l-D30: see *In(1)D30*

In(1)l-v: Inversion (1) lethal variegated

origin: X ray induced.
references: Lindsley, Edington, and Von Halle, 1960, *Genetics* 45: 1649-70.
genetics: Variegated Y-suppressed lethal; male fertile.

inversion	cytology
<i>In(1)l-v59</i>	3-4;20F
<i>In(1)l-v132</i>	4E;20F
<i>In(1)l-v139</i>	3C6-7;XR het
* <i>In(1)l-v146</i>	5-6;20F
<i>In(1)l-v227</i>	1-2;20F
<i>In(1)l-v231</i> ^α	1C-D;20F

^α Dimitri and Pisano, 1989, *Genetics* 122: 793-800.

In(1)Lü1: Inversion (1) Lüning

cytology: Breakpoints in proximal half of the X.
references: Lüning, Lake, and Linde, 1984, *Hereditas* 100: 247-57.
genetics: Homozygous females show low frequency of primary nondisjunction. Heterozygous females show five-fold increase in primary nondisjunction and excess of XO exceptional male offspring. Recombination enhanced outside the inversion and in region of distal break in homozygous females (Lake and Cederberg, 1984, *Hereditas*

itas 101: 79-84).

In(1)Lü2

cytology: *In(1)3E;4C*.
discoverer: Lünig.

****In(1)lz*: Inversion (1) lozenge**

origin: X ray induced.
discoverer: Green and Green.
references: 1956, Z. Indukt. Abstamm. Vererbungsl. 87: 708-21.
genetics: Mutant for spectacted-like alleles of *lz*.

inversion	cytology ^α
* <i>In(1)lz1</i>	8D;20F
* <i>In(1)lz2</i>	8D;20F
* <i>In(1)lz3</i>	4D;8D
* <i>In(1)lz4</i>	8A2-B1;8D
* <i>In(1)lz5</i>	8D;18F2-19A1
* <i>In(1)lz6</i>	8D;9B12-C1
* <i>In(1)lz7</i>	8D;20F

^α Hannah.

****In(1)lz^{SB}*: Inversion (1) lozenge-spectacted of Bishop**

cytology: *In(1)8;20* (Green).
origin: X ray induced.
discoverer: Bishop.
references: Oliver, 1947, Texas Univ. Publ. 4720: 167-84.
genetics: Associated with *lz^{SB}*.

****In(1)lza***

cytology: *In(1)3E;3F;9E;9F-10A*; inferred from Mackensen's figure; bands in 3E-F and 9E-F missing.
new order: 1 - 3E|9E - 3F|10A1 - 20.
synonym: *Df(1)Del lz a*.
references: Mackensen, 1935, J. Hered. 26: 163-74 (fig.).
genetics: Mutant or deficient for *v* but not *lz* or *ras*. No clue to reason for *lz* appearing in name.

***In(1)m^{38c}*: Inversion (1) miniature**

cytology: *In(1)10E1-2;13B* (Craymer).
origin: X ray induced.
discoverer: Griffin, 1938.

***In(1)m^K*: Inversion (1) miniature of Krivshenko**

cytology: *In(1)10E4-5;20F*. In mitotic chromosomes, right breakpoint is near juncture of heterochromatic elements *hC* and *hD* to the left of the *NO* but to the right of right breakpoint of *In(1)sc⁴* (Cooper, 1959, Chromosoma 10: 535-88).
origin: X ray induced.
discoverer: Krivshenko, 5513.
synonym: *In₁(1)^K*.
references: 1956, DIS 30: 75.
 Wargent, 1971, DIS 47: 91.
 Wargent and Hartman-Goldstein, 1974, Heredity 33: 317-26.
 Hartman-Goldstein and Wargent, 1975, Chromosoma 52: 349-62.
genetics: Variegated for *m*; variegation increased in strain carrying *In(2LR)Rev^B* (Wargent and Hartman-Goldstein, 1974) as is heterochromatization of the *m^K* chromosome in this strain (Hartman-Goldstein and Wargent, 1975).

In(1)M20

cytology: *In(1)2B9-11;3C4-6*.
origin: Induced by methyl methanesulfonate.
references: Lim and Snyder, 1974, Genet. Res. 24: 1-10.
 Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90.
genetics: Carries *l(1)3Ae²²*.

In(1)mei269

cytology: *In(1)3A-B;9E*.
references: Baker and Carpenter, 1972, Genetics 71: 255-86.
genetics: Associated with *mei-269*. Suppresses nearly all recombination between *y* and *v*.

***In(1)Mg*: Inversion (1) Mglinetz**

references: Mglinetz, 1968, Genetika (Moscow) 4(8): 81-86.

inversion	cytology	origin ^α
<i>In(1)Mg83</i>	5C;8F	1
<i>In(1)Mg91</i>	1C;4B	1
<i>In(1)Mg92</i>	8F;11E	1
<i>In(1)Mg93</i>	4D;20	2
<i>In(1)Mg94</i>	14B;16A	2
<i>In(1)Mg95</i>	12A;20	2
<i>In(1)Mg96</i>	2D;14F	2

^α 1 = γ ray induced; 2 = Induced by ³²P feeding.

***In(1)MKS*: Inversion (1) Medvedeva Kupert Savvateeva**

origin: X ray induced.
references: Medvedeva, Kupert, and Savvateeva, 1985, DIS 61: 214.
genetics: Male lethal.

inversion	cytology
<i>In(1)MKS45</i>	9A;18A
<i>In(1)MKS47</i>	9A1;20E-F
<i>In(1)MKS78</i>	14B;20C-D

***In(1)Mud*: Inversion (1) Muddled**

cytology: *In(1)3C3-4;5A6-B1*.
discoverer: Grell.
genetics: Associated with dominant mutant, *Mud*.

***In(1)N*: Inversion (1) Notch**

molecular biology: Lesions identified on physical map of *N* (Artavanis-Tsakonas *et al.*, 1983, 1984; Grimwade *et al.*, 1985). 3C7 breakpoint of *In(1)N^{76b8}* chosen as coordinate 0.

inversion	cytology	origin	ref ^α	genetics
<i>In(1)N^{66h26}</i>	3C1-2;3C7-8	mutable w ^β	1, 2, 3, 4, 5, 6, 7	wN rsr ⁺ vr ⁺
<i>In(1)N^{76b8}</i>	3C7-8;3C9-10	γ ray	1, 2, 3, 6	N
<i>In(1)N^{77c17}</i> γ	1D2-E1;3C7-9	X ray	1, 2	N
<i>In(1)N⁸⁰⁹</i>	3C6-7;3C7-9	revertant	3	
<i>In(1)N^{81k6}</i> δ	3C6-9;13A12-B2	X ray	1, 3	N
<i>In(1)N^{81k10}</i>	3C5-9;20A3-F	X ray	2, 3	N
<i>In(1)N⁸¹¹¹</i>	3C5-9;20A3-F	X ray	2, 3	N
<i>In(1)N⁸¹¹⁵</i>	3C5-9;20A3-F	X ray	2, 3	N
<i>In(1)N⁸¹¹⁹</i>	3C3-D3;20A3-F	X ray	2, 3	N

^α 1 = Artavanis-Tsakonas, Muskavitch, and Yedvobnick, 1983, Proc. Nat. Acad. Sci. USA 80: 1977-81; 2 = Artavanis-Tsakonas, Grimwade, Harrison, Markopoulou, Muskavitch, Schlasinger-Bryant, Wharton, and Yedvobnick, 1984, Dev. Biol. 4: 233-54. 3 = Grimwade, Muskavitch, Welshons, Yedvobnick, and Artavanis-Tsakonas, 1985, Dev. Biol. 107: 503-19; 4 = Keppy and Welshons,

1980, Chromosoma 76: 191-200; 5 = Welshons, 1974, Genetics 76: 775-94; 6 = Welshons and Keppy, 1981, Mol. Gen. Genet. 181: 319-24; 7 = Welshons and Welshons, 1985, Genetics 110: 465-77.

β Discoverer: Green (1967, Genetics 56: 467-82). Stable *N* but can regain mutability ($N \rightarrow N^+$) by reinversion (Welshons and Keppy, 1981; Welshons and Welshons, 1985). Highly mutable FB elements thought to be cause of the instability (Grimwade *et al.*, 1985).

γ Discoverer, cytology: Welshons.

δ Discoverer: Muskavitch; cytology: Welshons.

***In(1)N²¹⁸**

cytology: *In(1)3C;20*; position of right breakpoint with respect to centromere of ring not determined.

origin: X ray induced in *R(1)2*.

discoverer: Barigozzi.

references: 1942, Rev. Biol. (Perugia) 34: 59-72.

genetics: Variegates for *w* and *ec* but not *pn* or *cv*. Seems to carry intermediate allele of *N*.

In(1)N²⁶⁴

origin: X ray induced.

inversion	cytology	ref ^{α}	genetics
*In(1)N²⁶⁴⁻⁷β	3C6-7;3C8-9;8C5-7; 3C7-8 missing (Hoover)	1	<i>rst</i> ⁺ <i>fa</i> ⁻ <i>sp</i> ⁻ <i>dm</i> ⁺
*In(1)N²⁶⁴⁻⁴⁸γ	1B6-7;1B10-11;3C7-8; 1B7-10 missing (Hoover)	1	<i>rst</i> ⁺ <i>fa</i> ⁻ <i>dm</i> ⁺
*In(1)N²⁶⁴⁻⁵²	3C3-5;20B2-C1	1	variegates for <i>rst-bi</i> , not for <i>w</i> or <i>peb</i>
*In(1)N²⁶⁴⁻⁵⁷	3C9-11;20F (Hoover)	2	<i>rst</i> <i>fa</i> ⁺ <i>N</i> <i>dm</i> ⁺
*In(1)N²⁶⁴⁻⁷¹	3C6-7;20D-F (Sutton)	2	<i>rst</i> ⁺ <i>N</i> <i>dm</i> ⁺
*In(1)N²⁶⁴⁻⁸⁴	3C6-7;20A-F (Sutton)	2	variegates for <i>fa-dm</i> , not for <i>rst</i> or <i>bi</i>
*In(1)N²⁶⁴⁻¹⁰⁸δ	3C3-5;3E7-8;20A4-5; 3C5-E7 missing	1	<i>w</i> ⁺ <i>sp</i> ⁻ <i>ec</i> ⁺
*In(1)N²⁶⁴⁻¹¹²	3C6-7;3F5-6 (Sutton)	1	<i>w</i> ⁺ <i>dm</i> ⁺ <i>ec</i> ⁺
*In(1)N²⁶⁴⁻¹¹⁶	2C8-10;3C7-9 (Sutton)	3	<i>pn</i> ⁺ <i>w</i> ⁺ <i>rst</i> ⁺ <i>dm</i> ⁺

α 1 = Demerec; 2 = Demerec, 1941, Proc. Int. Congr. Genet. 7th, pp. 99-103.

β new order: 1 - 3C6|8C5 - 3C9|8C7 - 20.

γ new order: 1 - 1B6|3C7 - 1B11|3C8 - 20.

δ new order: 1 - 3C3|20A4 - 3E8|20A5 - 20F.

In(1)N^{M7}

cytology: *In(1)10B-C;20*.

references: Reuter, Hoffman, and Wolff, 1983, Biol. Zentralbl. 102: 281-98.

In(1)N^P: Inversion (1) Notch from ³²P

cytology: *In(1)3C;8E* (Darby).

origin: Induced by ³²P.

discoverer: Bateman, 1950.

references: 1950, DIS 24: 55.

1951, DIS 25: 77.

In(1)N66

cytology: *In(1)7A7-8;11A3-5*.

discoverer: Lefevre.

references: Kulkarni and Hall, 1987, Genetics 115: 461-75.

genetics: Fails to complement *cac*.

In(1)N83

cytology: *In(1)7F10;9A4*.

origin: Lefevre.

genetics: Mutant for *gg*⁶.

***In(1)Nel-A: Inversion (1) of Nel**

cytology: *In(1)12A;18D*.

origin: Spontaneous.

discoverer: Nel.

***In(1)Nel-B**

cytology: *In(1)11A;12F*.

origin: Spontaneous.

discoverer: Nel.

***In(1)ney: Inversion (1) narrow eye**

cytology: *In(1)10A;16D*.

origin: X ray induced.

discoverer: Becker, 1950.

references: 1952, DIS 26: 69.

genetics: Associated with *ney*.

In(1)oc: Inversion (1) ocelliless

cytology: *In(1)7F1-2;8A1-2*.

origin: X ray induced.

discoverer: Bedichek, 30c15.

references: Spradling, Waring, and Mahowald, 1979, Cell 16: 609-16.

Spradling and Mahowald, 1981, Cell 27: 203-09.

genetics: Mutant for *oc*. Homozygous females sterile, but sterility may not be due to mutation at *oc* since *oc*¹/*oc* females are fertile (Lefevre). *Cp36* and *Cp38* are included within and 1-3 kb from the distal breakpoint of the inversion. Synthesis of chorion proteins *s36-1* and *s36-2* much reduced in homozygotes (Spradling *et al.*, 1979). Abnormal ultrastructure of chorion described by Johnson and King (1974, J. Insect Morphol. Embryol. 3: 385-95).

molecular biology: Sequences containing both breakpoints as well as adjacent sequences cloned. Inversion shows drastically altered pattern of differential replication of sequences surrounding 7F1-2; the 40 kb of DNA lying distal to the 7F breakpoint do not amplify as in the normal X; the remaining 50 kb, now adjacent to 8A, show somewhat reduced amplification. Sequences proximal to the 8A breakpoint do amplify in *In(1)oc*-containing follicle cells. Region 7F seems to contain a replication origin stimulated to additional rounds of replication.

In(1)omb^{H31}: Inversion (1)

optomotor blind

cytology: *In(1)4C4-7;12D2-E1*.

origin: Induced by ethyl methanesulfonate.

references: Heisenberg, Wonneberger, and Gotz, 1978, J. Comp. Physiol. 124: 287-96.

Blondeau and Heisenberg, 1982, J. Comp. Physiol. 145: 321-29.

Heisenberg, 1982.

genetics: Associated with *omb*¹, a mutant showing an optic lobe defect producing abnormal tracking behavior in homozygotes and in flies heterozygous with *Df(1)RC40*.

In(1)ovo⁵: Inversion (1) ovo

cytology: *In(1)4E1-2;5A1-6*.

discoverer: Oliver.

synonym: *In(1)ovo*^{D1rG5}.

genetics: Mutant for *ovo*.

***In(1)para^{lk4}*: Inversion (1) paralytic**

cytology: *In(1)13E9-14;14C7-8*.
 references: J.C. Hall.
 genetics: Mutant for *para*.

***In(1)pdf*: Inversion (1) podfoot**

cytology: *In(1)16B;19F-20A*.
 origin: X ray induced.
 discoverer: Welshons, 57h6.
 references: 1960, DIS 34: 54.
 genetics: Associated with *pdf*.

***In(1)pn*: Inversion (1) prune**

origin: X ray induced.
 references: Ilyina, Sorokin, Belyaeva, and Zhimulev, 1980, DIS 55: 205.
 genetics: Mutant for *pn*.

inversion	cytology
<i>In(1)pn¹</i>	see <i>Tp(1;2)pn¹</i>
<i>In(1)pn³⁶</i>	see <i>Tp(3;1)pn³⁶</i>
<i>In(1)pn⁴⁵</i>	<i>2D1-2;20A</i>

***In(1)PS*: Inversion (1) Paik Sung**

origin: Naturally occurring inversions in Korea.
 references: Paik and Sung, 1980, DIS 45: 120.

inversion	cytology
<i>In(1)PS1</i>	<i>1D;3F</i>
<i>In(1)PS2</i>	<i>8C;18B</i>
<i>In(1)PS3</i>	<i>10B;12B</i>
<i>In(1)PS4</i>	<i>13F;16E</i>

****In(1)Pub*: Inversion (1) Pub**

discoverer: P. Farnsworth.
 references: Lefevre, 1954, DIS 28: 75.
 genetics: Associated with *Pub*. Called inversion because of reduction in crossing over; less than 1% combination with *spl* and about 10% with *B*.

***In(1)r^{70b}*: Inversion (1) rudimentary**

cytology: *In(1)7B;15A1-2*.
 origin: X ray induced.
 discoverer: Lefevre.
 references: Rawls and Porter, 1979, Genetics 93: 143-61.
 genetics: Associated with *r^{70b}*, a non-complementing *r* allele.

In(1)r^{hd}

origin: Induced by hybrid dysgenesis.
 genetics: Mutant for *r*.

inversion	cytology
<i>In(1)r^{hd1-2}</i>	<i>11A1;15A1</i>
<i>In(1)r^{hd1-3}</i>	<i>7F1-3;15A1</i>
<i>In(1)r^{hd1-4}</i>	<i>5D1;15A1</i>
<i>In(1)r^{hd2-1}</i>	<i>7E1-2;15A1</i>
<i>In(1)r^{hd5-2}</i>	<i>9F4;15A1</i>

****In(1)r^K*: Inversion (1) rudimentary of Krivshenko**

cytology: Proximal break in heterochromatin.
 discoverer: Krivshenko.
 references: Agol, 1936, DIS 5: 7.
 genetics: Mutant for *r*.

In(1)RA35

cytology: *In(1)6E;7F1*.
 discoverer: Lefevre.
 references: King, Mohler, Riley, Storto, and Nicolazzo, 1986, Dev. Genet. 7: 1-20.

***In(1)rb^{D1}*: Inversion (1) ruby**

cytology: *In(1)4C6-8;20B-C*.
 origin: X ray induced.
 references: Banga, Bloomquist, Brodberg, Pye, Larrivee, Mason, Boyd, and Pak, 1986, Chromosoma 93: 341-46.
 genetics: Mutant for *rb*. Male lethal.

In(1)RB10-18

cytology: *In(1)10F;18D*.
 origin: Hybrid dysgenesis from *RB18* (chromosome with a 2.0 kb *Bam*HI - *Eco*RI fragment carrying *tra⁺* inserted in X at 18D).
 references: McKeown, Belote, and Baker, 1987, Cell 48: 489-99.
 genetics: Carries *tra⁺*, making *tra* female flies with the inversion fully female in phenotype and fertility.

In(1)RF19*: see *Df(1)RF19

In(1)RF19*: see *T(1;2)RF19

In(1)RF24

cytology: *In(1)13A6;19E*.
 origin: X ray induced.
 discoverer: Lefevre.
 genetics: Male lethal.

****In(1)rg⁷*: Inversion (1) rugose**

cytology: *In(1)4E;7A* (J.I. Valencia).
 origin: X ray induced.
 discoverer: Cantor, 46d20.
 genetics: Mutant for *rg*.

****In(1)rg^P***

cytology: *In(1)3C;4E* (Darby).
 origin: Induced by ³²P.
 discoverer: Bateman.
 references: 1951, DIS 25: 77-78.
 genetics: Mutant for *rg*.

***In(1)rst³*: Inversion (1) roughest**

cytology: *In(1)3C3-4;20F*. Right breakpoint about one-fourth the distance between the heterochromatic-euchromatic junction and the centromere, approximately between heterochromatic segments *hC* and *hD* (Cooper, 1959, Chromosoma 10: 535-88).
 origin: X ray induced.
 discoverer: Gruneberg, 33116.
 references: 1935, DIS 3: 27.
 1935, J. Genet. 31: 163-84 (fig.).
 1937, J. Genet. 34: 169-89.
 Spofford, 1969, Genetics 62: 555-71.

genetics: Left breakpoint between *w* and *rst*; right breakpoint to the left of *bb* [Emmens, 1937, J. Genet. 34: 191-202 (fig.); Kaufmann, 1942, Genetics 27: 537-49 (fig.)]. Variegates for *rst* with extent of mutant eye tissue decreased by *Su(var)* (Spofford, 1969); in *X/O* male variegates for *w* (Gersh, 1963, DIS 37: 81). Precise reinversions of *In(1)rst³* accompanied by reversion of phenotype reported to occur spontaneously (Gruneberg, 1935) and after X irradiation of oocytes (Novitski, 1961,

Genetics 46: 711-17) but not after irradiation of sperm (Kaufmann, 1942).

***In(1)rstl: Inversion (1) roughestlike**

cytology: Breakpoints unknown.
origin: X ray induced.
discoverer: Oliver, 29d3.
references: 1935, DIS 3: 28.
genetics: Associated with *rstl*.

In(1)S: Inversion (1) of Sinitskaya

cytology: *In(1)6F;10F10-11A1* (Brosseau, 1967).
origin: X ray induced simultaneously with *In(1)sc^{SI}*.
discoverer: Sinitskaya.
references: Muller and Prokofyeva, 1934, Dokl. Akad. Nauk SSSR, n.s. 4: 74-83.
 Slizynski, 1948, DIS 22: 77.
 Brosseau, 1967, DIS 42: 41.
other information: *In(1)sc^{SI}* + *In(1)S* used as crossover suppressors in certain balancers, e.g., *Basc*.

In(1)S39

cytology: *In(1)1D1-2;1E1*.
genetics: Mutant for *su(w^a)*.

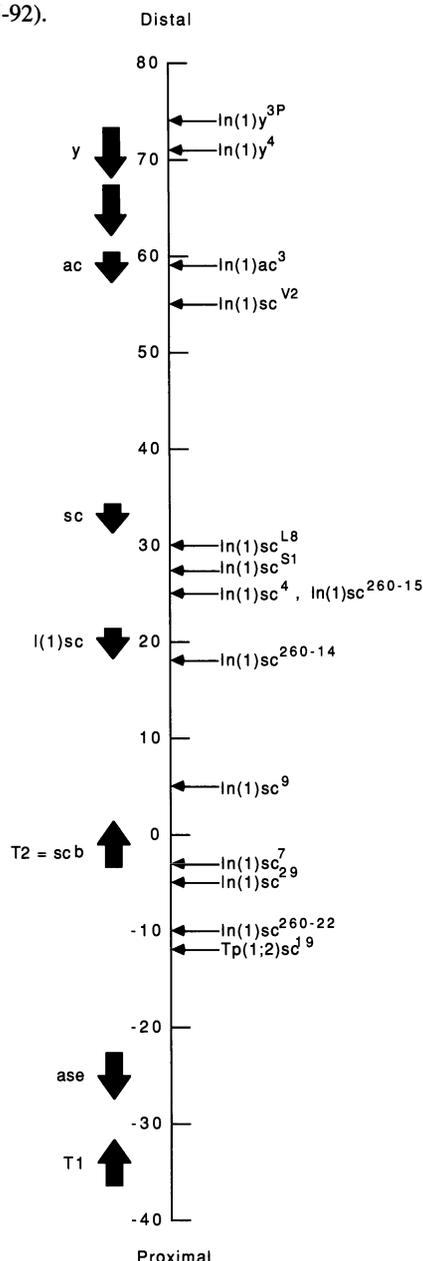
In(1)sc¹

cytology: *In(1)1B3-4;6D8*.
genetics: Mutant for *sc*.

In(1)sc⁴: Inversion (1) scute

cytology: *In(1)1B3-4;20F*. In mitotic chromosomes, the right break is to the right of and near the euchromatic-heterochromatic juncture in the heterochromatic segment h26 as defined by Gatti and Pimpinelli.
origin: X ray induced in a y chromosome.
discoverer: Agol, 1928.
references: 1929, Zh. Eksp. Biol. Med. 5: 86-101.
 1931, Genetics 16: 254-66.
 Serebrovsky and Dubinin, 1930, J. Hered. 21: 259-65.
 Sturtevant and Beadle, 1936, Genetics 25: 554-604.
 Muller and Raffel, 1940, Genetics 25: 541-83.
 Schalet, Lefevre, and Singer, 1970, DIS 45: 165.
 Schalet and Lefevre, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol 1b, pp. 847-902.
 Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, Cell 40: 327-38.
genetics: Mutant at *sc*; also carries *y*. Left break placed to the right of the α subdivision of the *sc* locus and to the left of *l(1)sc* and the β subdivision (García-Bellido, 1979, Genetics 91: 491-520). Right break in the proximal heterochromatin between *su(f)* and *bb* (Schalet *et al.*, 1970). Proximal heterochromatin carries the *ABO-X* to *CR* region as well as the *NO* (Yedvobnick, Krider, and Levine, 1980, Genetics 95: 661-72). Maternal and zygotic interactions between *abo* and *In(1)sc⁴* (Malva, Labella, Manzi, Salzano, Lavorgna, De Ponti, and Graziani, 1985, Genetics 111: 487-94). *In(1)sc⁴/+* female produces about 6% exceptional males from 4-strand double exchange. Secondary exceptions about 4%.
molecular biology: Left breakpoint between DNA coordinates +24.0 and +25.8 (Campuzano *et al.*, 1985). 0 is an arbitrarily chosen *EcoRI* site in the *sc β* segment of *ASC*; positive values to the left (Carramolino, Ruiz-Gómez, Guerro, Campuzano, and Modolell, 1982, EMBO J.

1: 1185-92).



Molecular map of y and sc inversion breakpoints (after Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, Cell 40: 327-38 and González, Romani, Cubas, Modolell, and Campuzano, 1989, EMBO J. 8: 3553-62).

In(1)sc^{4L}sc^{8R}: Inversion (1) scute-4 Left scute-8 Right

cytology: *In(1)1B3-4;20F^L1B2-3;20F^R*; duplication for 1B3; mitotic chromosomes deficient for the proximal portion of h26 through the distal portion of h32 according to Gatti and Pimpinelli. About 0.6 the length of a normal X at metaphase.
origin: Recombinant containing left end of *In(1)sc⁴* and right end of *In(1)sc⁸*.
discoverer: Gershenson.
synonym: *Df(1)bb-G*.
references: 1933, J. Genet. 28: 297-313.
 1933, Biol. Zh. (Moscow) 2: 145-59, 419-24.
 Peacock, 1965, Genetics 51: 573-83.

Ritossa and Spiegelman, 1965, Proc. Nat. Acad. Sci. USA 53: 739-45.

Tartof, 1971, Science 171: 294-97.

Malva, Graziani, Boncinelli, Polito, and Ritossa, 1972, Nature (London), New Biol. 239: 135-37.

Tartof, 1973, Genetics 73: 613-34.

Parry and Sandler, 1974, Genetics 77: 735-39.

Peacock, Miklos, and Goodchild, 1975, Genetics 79: 613-34.

Procnier and Tartof, 1978, Genetics 88: 67-79.

genetics: Duplicated for the *sc* locus, carrying both *sc*⁴ and *sc*⁸; deficient for *ABO-X*, *CR* and all the ribosomal RNA-complementary DNA at the *bb* locus of a normal X (Ritossa and Spiegelman, 1965; Tartof, 1971). *In(1)sc*⁴*sc*⁸/*Ybb*⁺ males and heterozygous *In(1)sc*⁴*sc*⁸ females have one *NO* (on the Y and on the normal X) and show a *bb*⁺ phenotype. *In(1)sc*⁴*sc*⁸/*Ybb*⁻ males and *XO* males carrying *In(1)sc*⁴*sc*⁸ are lethal. Magnification or increase in rDNA multiplicity may take place in *In(1)sc*⁴*sc*⁸/*Ybb* males, changing their phenotype from *bb* to *bb*⁺ (Tartof, 1971, 1973; Malva *et al.*, 1972). *In(1)sc*⁴*sc*⁸/*Y* males frequently show correlated meiotic drive and nondisjunction (Peacock, 1965; Peacock *et al.*, 1975), resulting in the recovery of more nullto than *In(1)sc*⁴*sc*⁸/*Y* gametes and more *sc*⁴*sc*⁸ than *Y* gametes. Distorted ratios result from sperm dysfunction which is proportional to the chromosome content of the nucleus (McKee, 1984, Genetics 106: 403-22). The extent of these meiotic irregularities is dependent on Y chromosome(s) present (Peacock *et al.*, 1965) and the temperature at which meiosis occurs (Zimmering, 1963, Genetics 48: 133-38). *In(1)sc*^{4L}*sc*^{8R}/*T(Y;A)* and *In(1)sc*^{4L}*sc*^{8R}/*mal*⁺*Y* males sterile (McKee and Lindsley, 1987, Genetics 116: 399-407).

***In(1)sc*^{4L}*sc*^{9R}: Inversion (1) scute-4 Left scute-9 Right**

cytology: *In(1)IB3-4;20F*^L*1B3-4;18C1*^R. Duplicated for 18C1-20.

origin: Recombinant containing left end of *In(1)sc*⁴ and right end of *In(1)sc*⁹.

discoverer: Muller.

references: 1935, Genetics 17: 237-52.

García-Bellido and Santamaria, 1978, Genetics 88: 469-86.

García-Bellido, 1979, Genetics 91: 491-520.

genetics: Deficient for *l(1)sc*. Mutant for *Hw*. Duplicated for loci to right of 18C1. Male lethal but viable in presence of *Dp(1;2)sc*¹⁹ = *Dp(1;2)1B1-2;1B4-7;25-26*.

Only affects larval nervous system (Villares and Cabrera, 1987, Cell 50: 415-24).

***In(1)sc*^{4L}*sc*^{L8R}: Inversion (1) scute-4 Left scute of Levy 8 Right**

cytology: *In(1)IB3-4;20F*^L*1B3-4;20F*^R. Mitotic chromosomes deficient for the proximal part of h26 through h30 (Gatti and Pimpinelli).

origin: Recombinant containing left end of *In(1)sc*⁴ and right end of *In(1)sc*^{L8}.

references: Muller, Raffel, Gershenson, and Prokofyeva-Belgovskaya, 1937, Genetics 22: 87-93.

genetics: Deficient for *bb* and the *NO*.

***In(1)sc*^{4L}*sc*^{S1R}: Inversion (1) scute-4 Left scute of Sinitskaya 1 Right**

cytology: *In(1)IB3-4;20F*^L*1B3-4;20F*^R; deficient for the proximal part of h26 through the majority of h32 (Gatti and Pimpinelli). X at metaphase.

origin: Recombinant containing left end of *In(1)sc*⁴ and right end of *In(1)sc*^{S1}.

references: Muller, Raffel, Gershenson, and Prokofyeva-Belgovskaya, 1937, Genetics 22: 87-93.

genetics: Deficient for *bb*. Behavior in meiosis of the male like that of *In(1)sc*^{4L}*sc*^{8R}.

***In(1)sc*^{4L}*sc*^{V2R}**

cytology: *In(1)IB3-4;20F*^L*1B2-3;20F*^R. Deficient for the proximal part of h26 through the distal half of h29 (Gatti and Pimpinelli).

origin: Single recombinant between *In(1)sc*^{4L}*sc*^{8R} and *In(1)sc*^{V1}.

discoverer: Lindsley.

genetics: X-Y disjunction normal; *In(1)sc*^{4L}*sc*^{V2R}/*T(Y;A)* and *In(1)sc*^{4L}*sc*^{V2R}/*mal*⁺*Y* males fertile (McKee and Lindsley, 1987, Genetics 116: 399-407). Carries sufficient ribosomal cistrons to support survival and *bb*⁺ phenotype of *XO* male. Duplicated for *sc* α region.

***In(1)sc*^{4L}*y*^{3PR}: Inversion (1) scute-4 Left yellow-3P Right**

cytology: *In(1)IB3-4;20F*^L*1A;20F*^R; duplicated for 1B1-3.

origin: Recombinant containing left end of *In(1)sc*⁴ and right end of *In(1)y*^{3P}.

references: García-Bellido, 1979, Genetics 91: 491-520.

genetics: Fully viable, *ac*⁺ and *sc*⁺ with weak *Hw* phenotype.

***In(1)sc*^{4L}*y*^{4R}: Inversion (1) scute-4 Left yellow-4 Right**

cytology: *In(1)IB3-4;20F*^L*1A8-B1;18A3-4*^R; duplicated for 1B1-3 and 18A4-20F.

origin: Recombinant containing left end of *In(1)sc*⁴ and right end of *In(1)y*⁴.

references: Sturtevant and Beadle, 1936, Genetics 21: 554-604.

genetics: Duplicated for the loci of *ac*, *sc*, *car*, and *M(1)18C*; carries *y*¹ distally and the *y* locus interrupted by *In(1)y*⁴ proximally. Both male and female look normal.

***In(1)sc*⁷**

cytology: *In(1)IB3-4;6D8* (Lefevre).

origin: X ray induced in a *w*^a chromosome.

discoverer: Dubinin, 1929.

references: 1930, Zh. Eksp. Biol. Med. 6: 300-24.

Serebrovsky and Dubinin, 1930, J. Hered. 21: 259-65.

Dubinin, 1933, J. Genet. 27: 443-64.

Sturtevant and Beadle, 1936, Genetics 21: 554-604.

García-Bellido, 1979, Genetics 91: 491-520.

Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, Cell 40: 327-38.

genetics: Mutant for *sc*. Normal disjunction and 33% recombination in *In(1)sc*⁷/*+* female, 26% secondary nondisjunction and 27% recombination in *In(1)sc*⁷/*+**Y* female (Grell, 1962, Genetics 47: 1737-54). *w*^a removable from the inversion by double crossing over in triploid.

molecular biology: Left breakpoint between DNA coordi-

ates -1.8 and -5.9 (Campuzano *et al.*, 1985). 0 is an arbitrarily chosen *EcoRI* site in the $sc\beta$ segment of *ASC*; positive values to the left (Carramolino, Ruiz-Gómez, Guerro, Campuzano, and Modolell, 1982, *EMBO J.* 1: 1185-92).

In(1)sc⁸

cytology: *In(1)1B2-3;20F*. Mitotic chromosomes show break in proximal heterochromatin extremely close to the centromere in proximal part of h32 (Gatti and Pimpinelli). *NO* near left end of X.

origin: X ray induced.

discoverer: Sidorov, 1929.

references: 1931, *Zh. Exper. Biol. Med.* 7: 28-40.

1936, *Biol. Zentralbl.* 5: 1-26.

Noujdin, 1935, *Zool. Zh.* 14: 317-52.

Patterson and Stone, 1935, *Genetics* 20: 172-78.

Sturtevant and Beadle, 1936, *Genetics* 21: 554-604.

Baker, 1971, *Proc. Nat. Acad. Sci. USA* 68: 2472-76.

García-Bellido, 1979, *Genetics* 91: 491-520.

Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Vilares, Boronat, and Modolell, 1985, *Cell* 40: 327-38.

genetics: Weak mutant for *sc* and shows a *Hw* effect; variegates for *ac*, *y*, and probably *l(1)IAc* (Hess, 1962, *Verh. Dtsch. Zool. Ges., Zool. Anz., Suppl.* 26: 87-92) in *X/O* male. Left break between *ac* and *sc* because induced deficiencies for the terminal uninverted portion of *In(1)sc⁸* are deficient for *y* and *ac* but not *sc⁸* (Patterson and Stone, 1935) and because *In(1)sc^{8Lsc^{4R}}* is deficient for *sc* (Sturtevant and Beadle, 1936); [*i.e.*, for the α subdivision of the *sc* locus (García-Bellido)]. Right break between *bb* and centromere because deficiencies for terminal genes are frequently deficient for *bb* (Patterson, 1933, *Genetics* 18: 32-52) as is *In(1)sc^{4Lsc^{8R}}* (Gershenson, 1933, *J. Genet.* 28: 297-313; Sturtevant and Beadle, 1936). *In(1)sc^{8/+}* female produces about 3% exceptional sons from 4-strand double crossing over within the inversion and about 8.7% recombination. *In(1)sc^{8/+Y}* female produces 19% secondary nondisjunction and 12% recombination (Sturtevant and Beadle, 1936; Grell, 1962, *Genetics* 47: 1737-54). *In(1)sc^{8/0}* partially male lethal (Baker, 1971; Johnson, Harger, and Holm, 1979, *Genetics* 92: s54-55); variegates for *y*; *ac* enhanced.

molecular biology: Left breakpoint between DNA coordinates 46.8 and 47.9 (Campuzano *et al.*, 1985). 0 is an arbitrarily chosen *EcoRI* site in the $sc\beta$ segment of *ASC*; positive values to the left (Carramolino, Ruiz-Gómez, Guerro, Campuzano, and Modolell, 1982, *EMBO J.* 1: 1185-92).

In(1)sc^{8L}EN^R: Inversion (1) scute-8 Left Entire Right

cytology: *In(1)1B2-3;20F^L1A;20;20F^R*.

origin: Recombinant containing left end of *In(1)sc⁸* and right end of *In(1)EN*.

references: Lindsley, 1958, *Z. Indukt. Abstamm. Vererbungsl.* 89: 103-22.

genetics: Carries *l(1)IA⁺* alleles, *y⁺* (or *y^{31d}*), and *ac⁺* distally and *l(1)IA⁺* alleles, *y*, and *ac⁺* proximally on long arm plus heterochromatic short arm of *In(1)EN*. Carries *bb⁺* in distal heterochromatin derived from *In(1)sc⁸* and a mild allele of *bb* in proximal heterochromatin derived from *In(1)EN*.

In(1)sc^{8L}sc^{4R}: Inversion (1) scute-8 Left scute-4 Right

cytology: *In(1)1B2-3;20F^L1B3-4;20F^R*; deficient for 1B3 and duplicated for proximal portion of h26 through majority of h32. About 1.4 times the length of a normal X at metaphase.

origin: Recombinant containing left end of *In(1)sc⁸* and right end of *In(1)sc⁴*.

references: Sturtevant and Beadle, 1936, *Genetics* 21: 554-604.

García-Bellido, and Santamaria, 1978, *Genetics* 88: 469-86.

García-Bellido, 1979, *Genetics* 91: 491-520.

genetics: Deficient for α subset of *sc* locus; duplicated for *bb⁺* and the *NO*. Homozygous female lethal; *In(1)sc^{8Lsc^{4R}/+}* female often has crippled legs. Male survives rarely and is extreme *sc* and slight *ac* (García-Bellido, 1979). Remains immobile after hatching.

In(1)sc^{8L}sc^{9R}

cytology: *In(1)1B2-3;20F^L1B3-4;18C1^R*.

origin: Recombinant containing left end of *In(1)sc⁸* and right end of *In(1)sc⁹*.

references: García-Bellido and Santamaria, 1978, *Genetics* 88: 469-86.

García-Bellido, 1979, *Genetics* 91: 491-520.

genetics: Male lethal. Deficient for α subset of *sc* locus and *l(1)sc*; duplicated for loci to right of 18C1. Mutant for *Hw*.

In(1)sc^{8L}sc^{L8R}: Inversion (1) scute-8 Left scute of Levy 8 Right

cytology: *In(1)1B2-3;20F^L1B3-4;20F^R*; deficient for 1B3; mitotic chromosomes duplicated for h31 through majority of h32 (Gatti and Pimpinelli).

origin: Recombinant containing left end of *In(1)sc⁸* and right end of *In(1)sc^{L8}*.

references: Muller, Raffel, Gershenson, and Prokofyeva-Belgovskaya, 1937, *Genetics* 22: 87-93.

genetics: Duplicated for the majority of the α subset of the *sc* locus.

In(1)sc^{8L}sc^{S1R}: Inversion (1) scute-8 Left scute of Sinitskaya 1 Right

cytology: *In(1)1B2-3;20F^L1B3-4;20B-D1^R*; deficient for 1B3.

origin: Recombinant containing left end of *In(1)sc⁸* and right end of *In(1)sc^{S1}*.

genetics: Deficient for the majority of the α subset of the *sc* locus. A few extreme *sc* males survive.

In(1)sc^{8L}y^{3PR}: Inversion (1) scute-8 Left yellow-3 of Patterson Right

cytology: *In(1)1B2-3;20F^L1A;20F^R*; duplicated for 1A-B2.

origin: Recombinant containing left end of *In(1)sc⁸* and right end of *In(1)y^{3P}*.

references: Muller, 1935, *J. Hered.* 26: 469-78.

García-Bellido, 1979, *Genetics* 91: 491-520.

genetics: Duplicated for *y* and *ac* loci; shows variable *Hw* phenotype in the mesopleura (García-Bellido, 1979). Not deficient for *bb*. Male viable.

***In(1)sc^{8L y 4R}*: Inversion (1) scute-8
Left yellow-4 Right**

cytology: *In(1)1B2-3;20F^L1A8-B1;18A3-4^R*; duplicated for 1B1-2 and 18A4-20F.

origin: Recombinant containing left end of *In(1)sc⁸* and right end of *In(1)y⁴*.

references: Sturtevant and Beadle, 1936, *Genetics* 21: 554-604.

genetics: Duplicated for *ac*, *car*, *M(1)18C*, and *bb*. Both male and female survive and show *Hw* effect of *In(1)sc⁸*.

In(1)sc⁹

cytology: *In(1)1B3-4;18C1* (Lefevre).

origin: X ray induced.

discoverer: Levit, 1929.

references: 1930, Wilhelm Roux's *Arch. Entwickl.-lungsmech. Organ.* 122: 770-83.

Norton and Valencia, 1965, *DIS* 40: 40.

García-Bellido, 1979, *Genetics* 91: 491-520.

Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, *Cell* 40: 327-38.

genetics: Mutant for *sc*. Left break to right of the α region of the *sc* locus and *l(1)sc*; inferred from observation that *In(1)sc^{4L sc 9R}* is lethal in male unless *Dp(1;2)sc¹⁹* is present (Muller, 1935). Right break right of *sby*, *smd*, and *coc* and left of *car*, as shown by the deficiency for *sby*, *smd*, and *coc* of *In(1)y^{4L sc 9R}* (Norton and Valencia, 1965).

molecular biology: Breakpoint between DNA coordinates +5.6 and +4.7 (Campuzano *et al.*, 1985). 0 is an arbitrarily chosen *EcoRI* site in the *sc β* segment of *ASC*; positive values to the left (Carramolino, Ruiz-Gómez, Guerro, Campuzano, and Modolell, 1982, *EMBO J.* 1: 1185-92).

In(1)sc¹⁰: see *In(1)ac³*

In(1)sc²⁹

cytology: *In(1)1B;13A2-5* (Raffel).

discoverer: Agol, 1930.

references: Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, *Cell* 40: 327-38.

genetics: Mutant at *sc*. Left break to right of *l(1)sc* (Muller).

molecular biology: Breakpoint between DNA coordinates -6.1 and -6.4 (Campuzano *et al.*, 1985). 0 is an arbitrarily chosen *EcoRI* site in the *sc β* segment of *ASC*; positive values to the left (Carramolino, Ruiz-Gómez, Guerro, Campuzano, and Modolell, 1982, *EMBO J.* 1: 1185-92).

****In(1)sc^{52c}***

origin: Spontaneous.

discoverer: Green, 52c.

references: 1952, *DIS* 26: 63.

genetics: Mutant for *sc* and *su(s)* [*i.e.*, *su(s)^{52c}*]. Inversion inferred from failure of *sc^{52c}* to recombine with *ras* or *v*.

****In(1)sc⁹⁰***

cytology: *In(1)1B4-7;1D2-E1*; inferred from Goldat's fig. 2.

origin: X ray induced derivative of *sc⁶*.

discoverer: Goldat.

references: 1936, *Biol. Zentralbl.* 5: 803-12.

genetics: Mutant for *sc*.

In(1)sc²⁶⁰⁻¹⁴

cytology: *In(1)1B2-3;11D3-8*.

origin: X ray induced.

discoverer: Sutton, 39b.

references: 1943, *Genetics* 28: 210-17.

Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, *Cell* 40: 327-38.

genetics: Mutant for *sc* but not *y*, *ac*, or *svr*.

molecular biology: Breakpoint between DNA coordinates +17.6 and +16.4 (Campuzano *et al.*, 1985). 0 is an arbitrarily chosen *EcoRI* site in the *sc β* segment of *ASC*; positive values to the left (Carramolino, Ruiz-Gómez, Guerro, Campuzano, and Modolell, 1982, *EMBO J.* 1: 1185-92).

In(1)sc²⁶⁰⁻²²

cytology: *In(1)1B2-3;1E2-3*.

origin: X ray induced.

discoverer: Sutton, 39f.

references: 1943, *Genetics* 28: 210-17.

Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, *Cell* 40: 327-38.

genetics: Mutant for *sc* but not *y*, *ac*, or *svr*.

molecular biology: Breakpoint between DNA coordinates -10.9 and -11.5 (Campuzano *et al.*, 1985). 0 is an arbitrarily chosen *EcoRI* site in the *sc β* segment of *ASC*; positive values to the left (Carramolino, Ruiz-Gómez, Guerro, Campuzano, and Modolell, 1982, *EMBO J.* 1: 1185-92).

****In(1)sc^A*: Inversion (1) scute of Agol**

discoverer: Agol.

references: 1936, *DIS* 5: 7.

genetics: Mutant for *sc*; semilethal. Genetically, appears to extend from *sc* to near *r* (54.5).

***In(1)sc^{J1}*: Inversion (1)
scute of Jacobs-Muller**

cytology: *In(1)1A4-5;1B4-5*.

discoverer: Jacobs-Muller.

references: Muller, 1932, *Proc. Intern. Congr. Genet.* 6th., Vol. 1: 225.

Muller, Prokofyeva, and Raffel, 1935, *Nature* (London) 135: 253-55.

genetics: Mutant for *sc* and *l(1)1Ac*.

***In(1)sc^{L8}*: Inversion (1) scute of Levy**

cytology: *In(1)1B3-4;20F*; inferred from genetic data. In mitotic chromosomes, right break is between h30 and h31 of Gatti and Pimpinelli. *NO* near left end of *X*.

discoverer: Levy, 1932.

references: Muller, Raffel, Gershenson, and Prokofyeva-Belgovskaya, 1937, *Genetics* 22: 87-93.

Muller and Raffel, 1938, *Genetics* 23: 160.

Raffel and Muller, 1940, *Genetics* 25: 541-83.

Baker, 1971, *Proc. Nat. Acad. Sci. USA* 68: 2472-76.

García-Bellido, 1979, *Genetics* 91: 491-520.

Johnson, Harger, and Holm, 1979, *Genetics* 92: s54-55.

Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, *Cell* 40: 327-38.

genetics: Mutant for *sc*. Left break in α segment of the *sc* locus. Right break to right of *bb* because *In(1)sc^{4L sc L8R}* deficient for *bb* (Muller *et al.*, 1937). Homozygous females sterile (Pucket and Snyder, 1973) *In(1)sc^{L8/0}* almost completely male lethal (Baker, 1971; Pucket and Snyder, 1973, *Genetics* 74: s221) but cell viable in

gynandromorphs [Pyati, 1976, *Genetika* (Moscow) 12: 75-81]. Lethality of *XO* males believed to be due to position-effect inactivation of the ribosomal cistrons and other genes affecting viability (Baker, 1971); maternal effect suppression of this lethality reported (Johnson *et al.*, 1979).

molecular biology: Breakpoint between DNA coordinates +30.9 and +28.8 (Campuzano *et al.*, 1985). 0 is an arbitrarily chosen *EcoRI* site in the *sc* β segment of *ASC*; positive values to the left (Carramolino, Ruiz-Gómez, Guerro, Campuzano, and Modolell, 1982, *EMBO J.* 1: 1185-92).

***In(1)sc^{L8L}sc^{4R}*: Inversion (1) scute of
Levy 8 Left scute-4 Right**

cytology: *In(1)1B3-4;20F^L1B3-4;20F^R*. Mitotic chromosomes duplicated for proximal half of h26 through h30 of Gatti and Pimpinelli.

origin: Recombinant containing left end of *In(1)sc^{L8}* and right end of *In(1)sc⁴*.

references: Muller, Raffel, Gershenson, and Prokofyeva-Belgovskaya, 1937, *Genetics* 22: 87-93.

genetics: Duplicated for *bb* and the *NO*.

***In(1)sc^{L8L}sc^{8R}*: Inversion (1) scute of
Levy 8 Left scute-8 Right**

cytology: *In(1)1B3-4;20F^L1B2-3;20F^R*; duplicated for 1B3 and mitotic chromosomes, deficient for h31 and majority of h32 (Gatti and Pimpinelli).

origin: Recombinant containing left end of *In(1)sc^{L8}* and right end of *In(1)sc⁸*.

references: Muller, Raffel, Gershenson, and Prokofyeva-Belgovskaya, 1937, *Genetics* 22: 87-93.

genetics: Duplicated for α segment of the *sc* locus but not *bb*. Survives as *X/0* male and homozygous female.

In(1)sc^{L8L}sc^{9R}

cytology: *In(1)1B3-4;20F^L1B3-4;18C1^R*.

origin: Recombinant containing left end of *In(1)sc^{L8}* and right end of *In(1)sc⁹*.

references: García-Bellido, 1979, *Genetics* 91: 491-520.

genetics: Male lethal. Deficient for *l(1)sc¹*; duplicated for loci to right of 18C1. Mutant for *Hw*.

molecular biology: Deficient for 23.2 kilobases of DNA in *sc* region: coordinates 5.6 to 28.8 (Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, *Cell* 40: 327-38). 0 is an arbitrarily chosen *EcoRI* site in the *sc* β segment of *ASC*; positive values to the left (Carramolino, Ruiz-Gómez, Guerro, Campuzano, and Modolell, 1982, *EMBO J.* 1: 1185-92).

***In(1)sc^{L8L}sc^{S1R}*: Inversion (1) scute
of Levy 8 Left scute
of Sinitskaya 1 Right**

cytology: *In(1)1B3-4;20F^L1B3-4;20F^R*; mitotic chromosome deficient for h31 and majority of h32 (Gatti and Pimpinelli).

origin: Recombinant containing left end of *In(1)sc^{L8}* and right end of *In(1)sc^{S1}*.

references: Muller, Raffel, Gershenson, and Prokofyeva-Belgovskaya, 1937, *Genetics* 22: 87-93.

genetics: Homozygous viable and fertile; does not affect expression of variegation of *bw^A*.

***In(1)sc^{S1}*: Inversion (1)
scute of Sinitskaya**

cytology: *In(1)1B3-4;20F*; inferred from genetic identity of left break with that of *In(1)sc⁴* and of right break with that of *In(1)sc⁸*. In mitotic chromosomes, right break is in proximal end of h32 of Gatti and Pimpinelli.

origin: X ray induced simultaneously with *In(1)S*.

discoverer: Sinitskaya, 34c.

references: Muller and Prokofyeva, 1934, *Dokl. Akad. Nauk SSSR* 4: 74-83.

Muller and Raffel, 1938, *Genetics* 23: 160.

Raffel and Muller, 1940, *Genetics* 25: 541-83.

Crew and Lamy, 1940, *J. Genet.* 39: 273-83.

Baker, 1971, *Proc. Nat. Acad. Sci. USA* 68: 2472-76.

Nix, 1973, *Biochem. Genet.* 10: 1-12.

García-Bellido, 1979, *Genetics* 91: 491-520.

Johnson, Harger, and Holm, 1979, *Genetics* 92: s54-55.

Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, *Cell* 40: 327-38.

genetics: Mutant for *sc*. Left break between the α segment of *sc* and *l(1)sc*. Right break to right of *bb* (Muller, Raffel, Gershenson, and Prokofyeva-Belgovskaya, 1937, *Genetics* 22: 87-93). Homozygous female sterile (Pucket and Snyder, 1973, *Genetics* 74: s221).

In(1)sc^{S1}/0 almost completely male lethal (Baker, 1971; Pucket and Snyder, 1973), the adult survivors extreme scute and almost immotile, with reduction in rRNA (Nix, 1973). In gynandromorphs, however, *In(1)sc^{S1}/0* cells viable [Pyati, 1976, *Genetika* (Moscow) 12: 75-81].

molecular biology: Breakpoint between DNA coordinates +28.2 and +26.9 (Campuzano *et al.*, 1985). 0 is an arbitrarily chosen *EcoRI* site in the *sc* β segment of *ASC*; positive values to the left (Carramolino, Ruiz-Gómez, Guerro, Campuzano, and Modolell, 1982, *EMBO J.* 1: 1185-92).

***In(1)sc^{S1L}sc^{4R}*: Inversion (1) scute
of Sinitskaya 1 Left
scute-4 Right**

cytology: *In(1)1B3-4;20F^L1B3-4;20F^R*; duplicated for h26 through the majority of h32 (Gatti and Pimpinelli).

About 1.4 times the length of a normal X at metaphase.

origin: Recombinant containing left end of *In(1)sc^{S1}* and right end of *In(1)sc⁴*.

references: Ritossa and Spiegelmann, 1965, *Proc. Nat. Acad. Sci. USA* 53: 737-45.

Krider and Plaut, 1972, *J. Cell. Biol.* 11: 675-83.

Yedvobnick, Krider, and Levine, 1980, *Genetics* 95: 661-72.

genetics: Duplicated for *ABO-X*, *CR*, *bb⁺* and the *NO*. (Krider and Plaut, 1972; Yedvobnick *et al.*, 1980).

Viable in *XO* males. *abo* females heterozygous for *In(1)sc^{S1L}sc^{4R}* produce *XO* offspring that do not show disproportionate replication of rDNA (Yedvobnick *et al.*, 1980). Carries twice the normal amount of DNA that is complementary to *Drosophila* ribosomal RNA (Ritossa and Spiegelmann, 1965).

***In(1)sc^{S1L}sc^{8R}*: Inversion (1) scute
of Sinitskaya 1 Left
scute-8 Right**

cytology: *In(1)1B3-4;20F^L1B2-3;20F^R*; duplicated for 1B3.

origin: Recombinant containing left end of *In(1)sc^{S1}* and right end of *In(1)sc⁸*.

genetics: Duplicated for *sc α* .

In(1)sc^{S1L}sc^{9R}

cytology: *In(1)1B3-4;20F^L1B3-4;18C1^R*.
origin: Recombinant containing left end of *In(1)sc^{S1}* and right end of *In(1)sc⁹*.
references: García-Bellido, 1979, Genetics 91: 491-520.
genetics: Male lethal. Deficient for *l(1)sc*; duplicated for loci to right of 18C1. Mutant for *Hw*.

In(1)sc^{S1L}sc^{L8R}: Inversion (1) scute of Sinitskaya 1 Left scute of Levy 8 Right

cytology: *In(1)1B3-4;20F^L1B3-4;20F^R*; chromosomes duplicated for h31 plus majority of h32 (Gatti and Pimpinelli).
origin: Recombinant containing left end of *In(1)sc^{S1}* and right end of *In(1)sc^{L8}*.
references: Muller, Raffel, Gershenson, and Prokofyeva-Belgovskaya, 1937, Genetics 22: 87-93.

In(1)sc^{SJ}

cytology: *In(1)1B3-4;2E3*.
origin: X ray induced.
references: Rowan, 1968, DIS 43: 61.
genetics: Male lethal. Associated with *sc^{SJ}*.

In(1)sc^{V2}

cytology: *In(1)1B2-3;20F*. Proximal breakpoint in heterochromatic segment h29 (Gatti and Pimpinelli).
origin: γ ray induced.
discoverer: J.I. Valencia, 23h46.
references: Muller and Valencia, 1947, DIS 21: 70. Cooper, 1959, Chromosoma 10: 535-88. Lindsley, Appels, and Hilliker, 1982, DIS 58: 99-100. Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, Cell 40: 327-38.
genetics: Mutant for *ac-sc*. Left break between *ac* and *sc* and right break in the middle of the region containing the ribosomal cistrons as indicated by *in situ* hybridization and by the survival as *XO* males of both *In(1)sc^{4L}sc^{V2R}* and *In(1)sc^{V2L}sc^{8R}* (*bb⁺* in phenotype) (Lindsley *et al.*, 1982). *XO* males semi-lethal (Baker, 1971, Proc. Nat. Acad. Sci. USA 68: 2472-76; Johnson, Harger, and Holm, 1979, Genetics 92: s54).
molecular biology: Breakpoint between DNA coordinates +55.7 and +54.3 (Campuzano *et al.*, 1985). 0 is an arbitrarily chosen *EcoRI* site in the *sc β* segment of *ASC*; positive values to the left (Carramolino, Ruiz-Gómez, Guerro, Campuzano, and Modolell, 1982, EMBO J. 1: 1185-92).

In(1)sc^{V2L}sc^{8R}

cytology: *In(1)1B2-3;20F^L1B2-3;20F^R*.
origin: Single recombinant between *In(1)sc^{V2}* and *In(1)sc^{4L}sc^{8R}*.
discoverer: Lindsley.
genetics: *X-Y* disjunction normal; *In(1)sc^{V2L}sc^{8R}/T(Y;A)* and *In(1)sc^{V2L}sc^{8R}/mal⁺Y* males fertile (McKee and Lindsley, 1987, Genetics 116: 399-407). Carries sufficient ribosomal cistrons to support survival and *bb⁺* phenotype of *In(1)sc^{V2L}sc^{8R}/w^ebb^l* females.

****In(1)sd²: Inversion (1) scalloped***

origin: X ray induced.
discoverer: Panshin, 33g7.

references: 1935, DIS 3: 28.

genetics: Mutant for *sd*. Crossing over inhibited.

In(1)sd^{58d}

origin: γ ray induced.
discoverer: Ives, 58d14.
references: 1961, DIS 35: 46. Simpson, Lawrence, and Maschat, 1981, Dev. Biol. 84: 206-11.
genetics: Mutant for *sd*. Not a translocation genetically; reduces recombination between *ras* and *f* by 80%.

In(1)sdx: Inversion (1) spreadex

origin: X ray induced.
discoverer: Muller.
references: 1965, DIS 40: 35.
genetics: Associated with *sdx*.

In(1)Si: Inversion (1) Simmons

origin: Selected as sex-linked recessive lethals from P-M dysgenic crosses.
references: Simmons, Raymond, Culbert, and Laverty, 1984, Genetics 107: 49-63.

inversion	cytology	location of lethal ^{α}
<i>In(1)SI1</i>	2B;14D1-2;19D1-2	14B13-15A9
<i>In(1)SI2</i>	11A2-6;14D1-2	19A1-20E
<i>In(1)SI3</i>	14C7-D1;19C2-D1	14B13-15A9
<i>In(1)SI4</i>	14C7-D1;19C2-D1	14B13-15A9
<i>In(1)SI5</i>	14C8-D2;19C2-6	
<i>In(1)SI6</i>	14D;19C	14B13-15A9
<i>In(1)SI7</i>	14D;19C	14B13-15A9
<i>In(1)SI8</i>	14D;19C	14B13-15A9
<i>In(1)SI9</i>	14D1-2;19A-D	14B13-15A9
<i>In(1)SI10</i>	14D1-2;19C2-5	14B13-15A9
<i>In(1)SI11</i>	14D1-2;19C-E	14B13-15A9
<i>In(1)SI12</i>	14D2-3;19C	14B13-15A9
<i>In(1)SI13</i>	14D2-E1;18F2-5	19A1-20E

^{α} Determined by deficiency testing against *Df(1)r75c = Df(1)14B13;15A9* and *Df(1)mal3 = Df(1)19A1-2;20E-F*.

In(1)SMG: Inversion (1) Semenova Mglinetz Glotoff

origin: γ ray induced.
references: Semenova, Mglinetz, and Glotoff, 1970, Genetika (Moscow) 6(4): 165-69.

inversion	cytology
<i>In(1)SMG1</i>	2B;5C
<i>In(1)SMG2</i>	1A;3C
<i>In(1)SMG3^{α}</i>	4;17B

^{α} Includes deficiency ?

In(1)St: Inversion (1) Stalker

origin: Naturally occurring inversions.
references: Stalker, 1976, Genetics 82: 323-47.

inversion	cytology
<i>In(1)St-A</i>	12A;18F
<i>In(1)St-B</i>	6D;11A
<i>In(1)St-C</i>	15A;18B
<i>In(1)St-D^{α}</i>	4D;17B

^{α} Stalker, unpublished.

***In(1)stx: Inversion (1) streakex**

origin: X ray induced.
discoverer: Muller, 26k30.
references: 1935, DIS 3: 30.
genetics: Associated with stx.

In(1)sx: Inversion (1) sexcombless

cytology: *In(1)11D4-6;14B8-9 + In(1)11E2-6;15E2-4.*
new order:
 1 – 11D4|14B9 – 15E2|11E2 – 11D6|
 14B8 – 11E6|15E4 – 20.
origin: X ray induced.
discoverer: Muller, 261.
references: Mukherjee, 1963, DIS 38: 62 (fig.).
 Reinhardt and Sánchez, 1982, Wilhelm Roux's Arch.
 Dev. Biol. 191: 264-69.
genetics: Associated with mutant sx, which is male sterile.

In(1)Sxl: Inversion (1) Sex lethal

cytology: *In(1)6F-7A;20.*
discoverer: Cline.
genetics: Mutant for Sxl.

***In(1)Th1: Inversion (1) Thymidine**

cytology: *In(1)12C;16C.*
origin: Induced by ingested H³-thymidine.
discoverer: Kaplan.
genetics: Male lethal.

**In(1)Uc: Inversion (1)
 Unstable chromosome**

origin: Derived from unstable X chromosome (termed *Uc*)
 which contains a readily mobilized *gypsy* element at *ct*.
references: Lim, 1979, Genetics 93: 681-701.
 Johnson-Schlitz and Lim, 1987, Genetics 115: 701-09.
genetics: Unstable.

inversion	cytology
<i>In(1)Uc3C-6F</i>	<i>3C10-D1;6F1-2</i>
<i>In(1)Uc6D-10F</i>	<i>6D-E1;10F-11A1</i>
<i>In(1)Uc6F-7B</i>	<i>6F2-5;7B5-C1</i>
<i>In(1)Uc6F-7C</i>	<i>6F1-2;7C1-2</i>
<i>In(1)Uc6F-10F</i>	<i>6F1-2;10F-11A1</i>
<i>In(1)Uc6F-11B</i>	<i>6F1-2;11B4-7</i>
<i>In(1)Uc6F-12E</i>	<i>6F1-2;12E2-8</i>
<i>In(1)Uc7C-9A</i>	<i>7C1-4;9A1-4</i>
<i>In(1)Uc7C-10F</i>	<i>7C1-4;10F-11A1</i>

In(1)V: Inversion (1) Valencia

origin: X ray induced.
references: Valencia, 1970, DIS 45: 37.

inversion	cytology	genetics
<i>In(1)V7-7</i>	<i>3C9-10;16C10;18B2-3</i>	semilethal
<i>In(1)V7-12</i>	<i>5B;11A7</i>	lethal
<i>In(1)V10-7</i>	<i>3A-B;16C</i>	lethal

Y-suppressed lethal.

In(1)VA288

cytology: *In(1)17E-F;19E.*
origin: Induced with ethyl methanesulfonate.
discoverer: Lefevre.

In(1)vao: Inversion (1) varied outspread

cytology: *In(1)18C5-6;19E7-8.*
origin: Induced by triethylenemelamine.
discoverer: Fahmy, 1953.

references: 1959, DIS: 33: 94.

genetics: Mutant for *vao*; variegated for an eye color, possibly *car*. Male sterile.

In(1)VE891

cytology: *In(1)2B6-7;20A.*
origin: Induced with ethyl methanesulfonate.
discoverer: Lefevre.
genetics: Male lethal.

***In(1)w²⁵⁸⁻⁵²: Inversion (1) white**

cytology: *In(1)3C;8E11-F1*
origin: X ray induced.
discoverer: Demerec, 40a.
genetics: Mutant for *w* and *rst* but not for *spl*, *lz*, *dvr*, or *flp*.

In(1)w^{-64d}

cytology: *In(1)3B1-2;20F + Df(1)3B1-2;3C2-3* (Lefevre).
origin: X ray induced.
references: Lefevre, 1968, DIS 43: 62-63; 165.
 Kaufman, Shannon, Shen and Judd, 1975, Genetics
 79: 265-82.

genetics: Male lethal. When covered by *Dp(1;3)w^{vco}*,
 males variegate for *w* and *rst* and show loss of bristles.
 Effect on bristles more extreme in *XO* males.
Dp(1;3)w^{m49a} also covers *In(1)w^{-64d}*.

molecular biology: Left breakpoint in a 1.6 kb restriction
 fragment at approximately 90 kilobases distal to the *w^a*
copia insertion (Bargiello and Young, 1984, Proc. Nat.
 Acad. Sci. USA 81: 2142-46; Pirrotta, Hadfield, and Pre-
 torius, 1983, EMBO J. 2: 927-34).

In(1)w^{-68b15} - In(1)w^{-71a27.1}

origin: X ray induced in *In(1)z^{+64b9}=In(1)3C1-2;12B8-9.*
references: Sorsa, Green, and Beerman, 1973, Nature
 (London), New Biol. 245: 34-37.

inversion	cytology	new order	genetics
<i>In(1)w^{-68b15}</i>	<i>3C1;11F3;12B8</i>	1A – 3C1 11F3 – 12B8 11F2 – 3C1 12B9 – 20	male viable; <i>z^v w⁻</i>
<i>In(1)w^{-70L26.3}</i>	<i>4C;12A + Df(1)3B;3C1 + Df(1)12A;12B8-9</i>	1A – 3B 12B 4C – 12A 4C – 3C2 12B9 – 20	male lethal; <i>crm⁺ w⁻ g⁺</i>
<i>In(1)w^{-70L26.8}</i>	<i>3C1;12B1;12B8</i> (reversion)	1A – 3C1 12B1 – 12B8 12A10 – 3C2 12B9 – 20	male viable; <i>z^v w⁻</i>
<i>In(1)w^{-71a9.3}</i>	<i>1D1;3C1</i>	1A – 1C5 3C1 – 1D1 12B8 – 3C2 12B9 – 20	male viable; <i>z^v w⁻</i>
<i>In(1)w^{-71a27.1}</i>	<i>3C1;11A1;12B8</i>	1A – 3C1 11A1 – 12B8 10F11 – 3C2 12B9 – 20	male viable <i>z^v w⁻</i>

In(1)w^{DZL-1}

cytology: *In(1)2F6;3C1-2.*
origin: In a *w^{DZL/w^{DZL}}* female. Presumed to be a rear-
 rangement generated by *w^{DZL}*.
references: Bingham, 1980, Cold Spring Harbor Symp.
 Quant. Biol. 45(2): 519-25.
genetics: *In(1)w^{DZL-1/w^{DZL}}* females have wild-type eye
 color. Viable and fertile in homo- and hemizygotes.

In(1)w^{DZL-3}

cytology: *In(1)3C1-2;3E3-7.*
origin: In a *w^{DZL/w^{DZL}}* female. Presumed to be a rear-
 rangement generated by *w^{DZL}*.
references: Bingham, 1980, Cold Spring Harbor Symp.
 Quant. Biol. 45(2): 519-25.
genetics: *In(1)w^{DZL-3/w^{DZL}}* females have wild-type eye
 color. Viable and fertile in homo- and hemizygotes.

***In(1)w^G: Inversion (1) white of Goldschmidt**

cytology: *In(1)3C;3D-E* (Kodani).
origin: X ray induced in *In(1)y^G = In(1)IA;1C3-4*.
discoverer: Goldschmidt.
references: 1945, Univ. Calif. (Berkeley) Publ. Zool 49: 522.

In(1)w^{m4}: Inversion (1) white-mottled

cytology: *In(1)3C1-2;20F* (Lefevre, 1976). Right breakpoint in mitotic chromosomes is to the left of *bb* in region h28 (Gatti and Pimpinelli). Appels and Hilliker (1982, Genet. Res. 39: 149-56) place the breakpoint at the distal border of the rDNA sequences.

origin: X ray induced.
discoverer: Muller, 1929.

references: 1930, J. Genet. 22: 299-334.

Lefevre, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 31-66.

Spofford and De Salle, 1978, DIS 53: 204.

Pirrotta, Hadfield, and Pretorius, 1983, EMBO. J. 2: 927-34.

Tartof, Hobbs, and Jones, 1984, Cell 37: 869-78.

genetics: Variegated for *w*. Left break to the left of *w*.

molecular biology: Left (3C1-2) breakpoint at -24.5 kb in the restriction map of the *w* locus (0 point at site of the *copia* insertion in *w^a*); right (20F) breakpoint near 5' end of a Type I mobile element (Tartof *et al.*, 1984).

other information: A strongly variegating line has been designated *In(1)w^{m4h}* (Reuter, Werner, and Hoffmann, 1982, Chromosoma 85: 539-51; Reuter and Szidonya, 1983, Chromosome 88: 277-85).

In(1)w^{m4L}rst^{3R}: Inversion (1) white-mottled 4 Left roughest-3 Right

cytology: *In(1)3C1-2;20F^L3C3-4;20F^R*; deficient for 3C2-3.

origin: Recombinant containing left end of *In(1)w^{m4}* and right end of *In(1)rst³*.

references: Lefevre and Wilkins, 1966, Genetics 53: 175-87.

Lefevre, 1968, DIS 43: 141.

Lefevre and Green, 1972, Genetics 36: 391-412.

genetics: Deficient for *w*; position effect on *rst* (Lefevre, 1968). Males lethal, with occasional *w rst* escapers at 29°; lethality covered by *Dp(1;2)51b*.

In(1)w^{m4L}sc^{4R}

cytology: *In(1)3C1-2;20F^L1B3-4;20F^R*; carries distal heterochromatin of *In(1)w^{m4}* and proximal heterochromatin of *In(1)sc⁴*.

origin: Recombinant containing left end of *In(1)w^{m4}* and right end of *In(1)sc⁴*.

references: Yedvobnick, Krider, and Levine, 1980, Genetics 95: 667-721.

genetics: Variegated for *w*; duplicated for the *ABO-X* to *CR* region and for *l(1)sc* through *w*; carries *bb⁺*.

In(1)w^{m4L}w^{m51bR}

cytology: *In(1)3C1-2;20F^L3C1-2;20F^R*. Right break just proximal to the *NO*.

origin: Recombinant containing left end of *In(1)w^{m4}* and right end of *In(1)w^{m51b}*.

references: Appels and Hilliker, 1982, Genet. Res. 39: 149-56.

genetics: Variegated for *w*. Carries *ABO-X* and *CR*; deficient for the majority, if not all, of the ribosomal cistrons.

In(1)w^{m4L}w^{mJR}: Inversion (1) white-mottled 4 Left white-mottled of Jonsson Right

cytology: *In(1)3C1-2;20F^L3C2-3;20F;102C^R* deficient for 3C2 and for an undetermined portion, including the centromere, of the base of the *X*.

new order:

1 - 3C1|20F - 3C3|102C - 101A.

origin: Recombinant containing left end of *In(1)w^{m4}* and right end of *In(1)w^{mJ}*, which is part of *T(1;4)w^{mJ} = T(1;4)3C2-3;20F;102C*.

references: Lefevre, 1963, DIS 37: 49-50.

Lefevre and Wilkins, 1966, Genetics 53: 175-87.

genetics: Deficient for white; males viable and therefore not deficient for any lethal. Also deficient for proximal heterochromatin, including *bb* (*XO* lethal).

In(1)w^{m4rv}

origin: X ray induced in *In(1)w^{m4}*.

references: Tartof, Hobbs, and Jones, 1984, Cell 37: 869-78.

genetics: Eyes *w⁺* (even when revertants reared at 17°).

inversion	cytology	new order
<i>In(1)w^{m4rv5}</i>	<i>3C1-2;4A;20A</i>	1A - 3C1 20 - 4A 3C2 - 4A 20
<i>In(1)w^{m4rv6}</i>	<i>1F;3C1-2;20A</i>	1A - 1F 3C2 - 20 3C1 - 1F 20
<i>In(1)w^{m4rv26}</i>	<i>3C1-2;6B;20A</i>	1A - 3C1 20 - 6B 3C2 - 6B 20

In(1)w^{m51b}

cytology: *In(1)3C1-2;20F*; right break proximal to the *NO* (Gersh), at the junction between h29 and h30 (Gatti and Pimpinelli). Appels and Hilliker (1982, Genet. Res. 39: 149-56) place the breakpoint at the proximal border of the rDNA sequences.

origin: X ray induced.

discoverer: Baker, 51b19.

references: CP627.

Spofford and De Salle, 1978, DIS 53: 204.

Tartof, Hobbs, and Jones, 1984, Cell 37: 869-78.

genetics: Variegated for *w* and *rst*. Recombinant carrying left end of *In(1)w^{m51b}* and right end of the 4-centric element of *T(1;4)w^{mJ} = T(1;4)3C2-3;20F;102C* is white eyed and male viable, indicating that *In(1)w^{m51b}*, like *In(1)w^{m4} = In(1)3C1-2;20F*, is broken between 3C1 and 2 (Gersh).

molecular biology: Left (3C1-2) breakpoint is at -21 kb of the restriction map of the *w* locus (0 point at site of the *copia* insertion in *w^a*); right (20F) breakpoint is at a mobile element (Tartof *et al.*, 1984).

In(1)w^{m51bL}w^{m4R}

cytology: *In(1)3C1-2;20F^L3C1-2;20F^R*.

origin: Recombinant containing left end of *In(1)w^{m51b}* and right end of *In(1)w^{m4}*.

references: Appels and Hilliker, 1982, Genet. Res. 39: 149-56.

genetics: Variegated for *w* and *rst*. Carries *ABO-X* and *CR*; duplicated for the majority, if not all, of the ribosomal cistrons.

In(1)w^{m53j}

cytology: *In(1)1A;3C3-5;20;20F;20F*. Inferred from origin.

new order:

20 – 3C5|20F|1A – 3C3|20.

Tentative.

origin: X ray induced in *In(1)EN = In(1)1A;20F;20F*.

discoverer: Bender, 53j.

references: 1955, DIS 29: 69.

genetics: Variegated for *w*.

In(1)w^{m54l}

cytology: *In(1)3C3-5;20F*.

origin: Neutron induced.

discoverer: Mickey, 54l3.

references: 1963, DIS 38: 29.

genetics: Variegated for *w*.

In(1)w^{m55b}

cytology: *In(1)1A3-4;3C3-5;20;19F-20A1;20A1-F*.

Inferred from origin. Appears as a rod in metaphase.

new order:

20F – 3C5|20A1|1A4 – 3C3|20F.20.

Tentative.

origin: X-ray-induced derivative of *R(1)2 = R(1)1A3-4;20F* opened in inverted order.

discoverer: M. A. Bender, 55b28.

references: 1955, DIS 29: 69.

genetics: Variegated for *w*.

In(1)w^{mb}: Inversion (1) white-mottled of Barnes

cytology: *In(1)3C2-3;20F*.

origin: X ray induced.

discoverer: Barnes, 1965.

references: Lefevre, 1968, DIS 43: 62-63.

genetics: Variegated for *w* in both males and females as in *In(1)w^{md}*. Variegated for *rst* and *vt* in *XO* males.

In(1)w^{mJ}: see T(1;4)w^{mJ}

In(1)w^{mJL}rst^{3R}: Inversion (1) white-mottled of Jonsson Left roughest-3 Right

cytology: *In(1)3C2-3;20F^L3C3-4;20F^R*; deficient for 3C3.

origin: Recombinant carrying left end of *In(1)w^{mJ}*, which is part of *T(1;4)w^{mJ}*, and right end of *In(1)rst³*.

references: Lefevre, 1963, DIS 37: 49-50.

Lefevre and Wilkins, 1966, Genetics 53: 175-87.

genetics: Male lethal. Variegated for *w* and *rst*.

In(1)w^{mMc}: Inversion (1) white-mottled of McLean

cytology: *In(1)3C1-2;20F*; inferred from genetic data.

origin: X ray induced.

discoverer: McLean.

references: Muller, 1946, DIS 20: 68.

Tartof, Hobbs, and Jones, 1984, Cell 37: 869-78.

genetics: Variegates for *w* and *rst*. Complementary single recombinants between *In(1)w^{md} = In(1)3C1-2;20F* and *In(1)w^{mMc}* are viable, fertile, and *bb⁺*. Left breakpoints therefore identical and right breakpoints on the same side of *bb*.

molecular biology: Left (3C1-2) breakpoint is at -21 kb in the restriction map of the *w* locus (0 point at site of the

copied insertion in *w^a*); right (20F) breakpoint near the 3' end of a Type I mobile element that is inserted in a ribosomal RNA gene (Tartof *et al.*, 1984).

In(1)w^{vc}: Inversion (1) white-variegated of Catcheside

cytology: *In(1)3C1-2;20F* superimposed on *R(1)1A3-4;20F*.

new order:

|1A4-3C1|20F-3C2|20F|. Position of centromere indeterminate.

origin: X ray induced in *R(1)2*.

discoverer: Catcheside.

references: Hinton, 1955, Genetics 40: 952-61.

genetics: Variegates for *w*, *rst*, *spl*, and *N* but not *y*. *X/Y* male viability reduced; *X/Y/Y* male more viable. Characterized by variable degree of instability manifested by production of gynandromorphs, *X/O* males, and dominant lethals. An extreme example gave 140 females, 106 gynandromorphs, 181 *X/O* males, and 868 dominant lethals among 1295 putative ring/rod zygotes. Small ring-shaped duplications are generated infrequently (analysis by Hinton, 1955). Behavior of rod derivatives of *In(1)w^{vc}* (Hinton, 1957, Genetics 42: 55-65) suggests generation of dicentric through sister-strand fusion rather than exchange. Fusion postulated to occur in heterochromatin of the 3C1|20F reunion point of *In(1)w^{vc}*. Mitotic abnormalities in cleavage of *In(1)w^{vc}* embryos described (Hinton, 1959, Genetics 44: 923-31). Chromosome tends to become stable in stocks. Viability and fertility correlated with stability. Widely used in mosaic analysis and fate mapping (*e.g.*, García-Bellido and Merriam, 1969, J. Exp. Zool. 170: 61-76).

In(1)w^w

cytology: *In(1)3B2-C1;4B4-C1*.

origin: Spontaneous in a mutable *z w^{zmmz}* strain.

references: Kalish, 1970, Mol. Gen. Genet. 107: 336-50. 1980, DIS 55: 206-07.

genetics: Mutant for *w*. Reverts to *w^{zmmz}* with reinversion of *In(1)w^w*.

In(1)y^{3P}: Inversion (1) yellow-3 of Patterson

cytology: *In(1)1B1-2;20* (Muller and Prokofyeva, 1935) or *In(1)1A8-B1;20F*. Heterochromatic break proximal to h29.

origin: X ray induced.

discoverer: Patterson, 31e25.

references: Muller, 1935, Genetica 17: 237-52.

Muller and Prokofyeva, 1935, Proc. Nat. Acad. Sci. USA 21: 16-26.

Sidorov, 1936, Biol. Zh. (Moscow) 5: 3-26.

García-Bellido, 1979, Genetics 91: 491-520.

Carramolino, Ruiz-Gómez, Guerrero, Campuzano, and Modolell, 1982, EMBO J. 1: 1185-91.

Biessmann, 1985, Proc. Nat. Acad. Sci. USA 82: 7369-73.

Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, Cell 40: 327-38.

genetics: Variegated for *y* and, to a lesser extent, for *Hw*. Genetic breaks between *l(1)1Ac* and *y* and between *bb* and centromere. Inversion slightly longer than *In(1)sc⁸*.

molecular biology: The left breakpoint defines the distal limit of the *y* coding region. On the DNA map of Campuzano *et al.*, 1985, this breakpoint is at +73 and on the

DNA map of Biessmann, 1985, it lies between +36.5 and +38.3; in both maps, positive values are to the left of the origin.

In(1)y^{3PL}sc^{4R}

cytology: *In(1)1A;20^L1B3-4;20F^R*.

origin: Recombinant containing left end of *In(1)y^{3P}* and right end of *In(1)sc⁴*.

references: García-Bellido and Santamaria, 1978, Genetics 88: 469-86.

García-Bellido, 1979, Genetics 91: 491-520.

genetics: Male lethal. Carries *Hw* and the region covering *l(1)sc*. Deficient for *y*, *ac*, and the α segment of *sc*.

molecular biology: Deficiency includes coding sequences for transcripts *T4*, *T5*, and *T6* (Campuzano, Carramolino, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, Cell 40: 327-48).

In(1)y^{3PL}sc^{8R}: Inversion (1) yellow-3 of Patterson Left scute-8 Right

cytology: *In(1)1A;20^L1B2-3;20F^R*; deficient for 1A-B2.

origin: Recombinant containing left end of *In(1)y^{3P}* and right end of *In(1)sc⁸*.

references: Muller, 1935, J. Hered. 26: 469-78.

Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, Cell 40: 327-38.

Chia, Howes, Martin, Meng, Moses, and Tsubota, 1986, EMBO J. 5: 3597-3605.

genetics: Deficient for *y* and *ac* but not *l(1)IAc*, *sc*, or *bb*. Male viable.

molecular biology: Deletes sequences encoding transcripts *T5* and *T6*.

In(1)y^{3PL}sc^{9R}

cytology: *In(1)1A;20^L1B3-4;18C1^R*.

origin: Recombinant containing left end of *In(1)y^{3P}* and right end of *In(1)sc⁹*.

references: García-Bellido and Santamaria, 1978, Genetics 88: 469-86.

García-Bellido, 1979, Genetics 91: 491-520.

genetics: Male lethal. Deficient for *y*, *ac*, *sc α* , and *l(1)sc*. Mutant for *Hw*. Duplicated for *car-bb*.

In(1)y^{3PL}sc^{51R}

cytology: *In(1)1A;20^L1B3-4;20F^R*.

origin: Recombinant containing left end of *In(1)y^{3P}* and right end of *In(1)sc⁵¹*.

references: García-Bellido and Santamaria, 1978, Genetics 88: 469-86.

García-Bellido, 1979, Genetics 91: 491-520.

genetics: Male lethal. Deficient for *y*, *ac*, and *sc α* ; retains *l(1)sc*. Male spots in gynandromorphs deficient for *ac* and *sc*.

In(1)y⁴

cytology: *In(1)1A8-B1;18A3-4* (Norton and Valencia, 1965, DIS 40: 40).

origin: X ray induced.

discoverer: Serebrovsky.

references: Dubinin and Friesen, 1932, Biol. Zentralbl. 52: 147-62.

Sturtevant and Beadle, 1936, Genetics 21: 554-604.

Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Boronat, and Modolell, 1985, Cell 40: 327-38.

genetics: Mutant for *y*. Right break within the *y* locus (Biessmann, 1985, Proc. Nat. Acad. Sci. USA 82: 7369-

73); left break between *fu* on left and *sby*, *smd*, and *coc* on right; shown by deficiency of *In(1)y^{4L}sc^{9R}* for *ac*, *sc α* , *l(1)sc*, *sby*, *smd*, and *coc* but not *l(1)IAc* or *fu* (Norton and Valencia, 1965). *In(1)y⁴/+* female produces about 2% exceptional sons from 4-strand double exchange in the inverted regions; *In(1)y⁴/+Y* female produces about 7% secondary exceptions (Sturtevant and Beadle, 1936).

molecular biology: Left breakpoint between DNA coordinates 70.6 and 71.6 [to right of breakpoint of *In(1)y^{3P}* (Campuzano *et al.*, 1985)].

In(1)y^{4L}sc^{4R}: Inversion (1) yellow-4 Left scute-4 Right

cytology: *In(1)1A8-B1;18A3-4^L1B3-4;20F^R*; deficient for 1B1-3 and 18A4 through the distal portion of h26.

origin: Recombinant containing left end of *In(1)y⁴* and right end of *In(1)sc⁴*.

references: Sturtevant and Beadle, 1936, Genetics 21: 554-604.

genetics: Deficient for *ac*, *sc*, *car*, and *M(1)18C* but not *os* or *bb*. Heterozygous female Minute and poorly viable but fertile. Male lethal.

In(1)y^{4L}sc^{8R}: Inversion (1) yellow-4 Left scute-8 Right

cytology: *In(1)1A8-B1;18A3-4^L1B2-3;20F^R*; deficient for 1B1-2 and 18A4 through the majority of h32.

origin: Recombinant containing left end of *In(1)y⁴* and right end of *In(1)sc⁸*.

references: Sturtevant and Beadle, 1936, Genetics 21: 554-604.

genetics: Deficient for *ac*, *car*, *M(1)18C*, and *bb* but not *svr*; carries the distal half of the *y* locus, *y⁴*. Heterozygous female Minute, fertile, and poorly viable. Male lethal.

In(1)y^{4L}sc^{9R}: Inversion (1) yellow-4 Left scute-9 Right

cytology: *In(1)1A8-B1;18A3-4^L1B2-3;18B8-9^R*; deficient for 1B1-2 and 18A4-B8.

origin: Recombinant containing left end of *In(1)y⁴* and right end of *In(1)sc⁹*.

references: Norton and Valencia, 1965, DIS 40: 40.

genetics: Deficient for *ac*, *sc α* , *l(1)sc*, *sby*, *smd*, and *coc* but not *l(1)IAc* or *fu-pph*. Carries the distal half of the *y* locus. Male lethal.

****In(1)y⁵***

cytology: *In(1)1A-B;14D* (Muller and Raffel).

discoverer: Patterson.

genetics: Mutant for *y*. Recessive lethal associated with right breakpoint.

In(1)y^{65f4}

cytology: *In(1)1B2-3;1C1*.

origin: X ray induced.

references: Lefevre, 1968, Genetics 60: 196-97. 1970, DIS 45: 32.

genetics: Phenotype *y²*-like, with bristles dark and body slightly darker than *y²*. Fertile in males and females.

In(1)y^A: Inversion (1) yellow of Alexandrov

references: Alexandrov, Ankina and Alexandrova, 1985,

DIS 61: 212-13.

inversion	cytology	origin α	genetics β
<i>In(1)y^{A72d3}</i>	1B1-2;20A	2	y
<i>In(1)y^{A74b51}</i> γ	1B1-2;20D	1	y ^{c4}
<i>In(1)y^{A74c166}</i>	1B4-9;9D	1	y ^{c4}
<i>In(1)y^{A74e}</i>	1B1-2;20D	2	y
<i>In(1)y^{A76b94}</i>	1B1-2;16C8	2	y ^{td}
<i>In(1)y^{A79b21}</i>	1B1-2;16D	2	y
<i>In(1)y^{A79d2}</i>	1B1-2;20A-B	2	y
<i>In(1)y^{A83fXL}</i>	1B1-2;20E-F	3	y

α 1 = caffeine and γ rays; 2 = γ rays; 3 = X rays.
 β Phenotype resembles allele indicated.
 γ Additional rearrangements between X and 3.

****In(1)y^G*: Inversion (1) yellow of Goldschmidt**

cytology: *In(1)IA;1C3-4* (Kodani).

origin: Spontaneous.

discoverer: Goldschmidt.

synonym: *In(1)y^{px bl}*.

genetics: Mutant for y.

In(1)y^{R10}

cytology: *In(1)IA5-B1;20*.

origin: X ray induced.

references: Roberts, 1974, Mutat. Res. 22: 139-44.

genetics: Phenotype dark yellow with dark hairs and darkish wing. Male viable and fertile.

In(1)y^{R20}

cytology: *In(1)IA6-8;1E2-3*.

origin: X ray induced.

references: Roberts, 1974, Mutat. Res. 22: 139-44.

genetics: Mutant for y, ac, and probably separate lethal. Males lethal with or without y⁺Y or *l(1)IAc⁺Y*.

***In(1)z⁺*: Inversion (1) zeste wild**

origin: X ray induced. Recovered as z reversion in genotype ordinarily exhibiting z phenotype.

inversion	cytology	ref α
<i>In(1)z⁺ 1</i>	3C1;3F5-6	1
<i>In(1)z⁺ 2</i>	3C1-2;7A	1
<i>In(1)z⁺ 7G3</i>	3C3-5;14B4-C1;20A3	2
<i>In(1)z⁺ 8</i>	3C1-3;20D-F	1
<i>In(1)z⁺ 11E7</i>	3C2-5;17C4-D1;20A3	2
<i>In(1)z⁺ 28</i>	2B-C;3C	1
<i>In(1)z⁺ 30</i>	3C1-2;5D	1
<i>In(1)z⁺ 38</i>	3C;10C-D	1
<i>In(1)z⁺ 48</i>	3C1-2;4D-5D	1
<i>In(1)z⁺ 50</i>	3C1-2;20B-C	1
<i>In(1)z⁺ 51</i>	1F3-4;3B4-C1;12B-D;20	1

α 1 = Gelbart, 1971, Ph.D. Thesis, Univ. of Wisconsin; 2 = Gans, 1953, Bull. Biol. Fr. Belg., Suppl. 38: 1-90.

In(1)z^{+64b9}

cytology: *In(1)3C1-2;12B9-10*; faint band to right of 3C1 consisting of leftmost border of 3C2 plus material from 12B9 (Sorsa, 1973).

origin: X ray induced in z male carrying triplication of w.

references: Green, 1967, Biol. Zentralbl. 86: 211-20.

Arcos-Terán and Beerman, 1968, Chromosoma 25: 377-91.

Sorsa, 1973, Cold Spring Harbor Symp. Quant. Biol. 38: 601-08.

Sorsa, Green, and Beerman, 1973, Nature (London), New Biol. 245: 34-37.

genetics: Males fertile, with wild-type eye pigmentation.

molecular biology: Molecular breakpoint localized

approximately 10 kb to the right of the w^a copia insert (Goldberg, Paro, and Gehring, 1982, EMBO. J. 1: 93-98).

In(1)z^{e(bx)}

cytology: *In(1)3A;4F*.

origin: γ ray induced.

synonym: *In(1)e(bx)*.

references: Lewis, 1959, DIS 33: 96.

Kaufman, Tasaki, and Suzuki, 1973, Genetics 75: 299-321.

genetics: Associated with z^{e(bx)}. Homozygotes show slightly variegated eye color (Lewis, 1959); heterozygotes over z are z.

molecular biology: Left breakpoint localized to a 4 kilobase BamHI fragment near the origin of a 160 kb walk (Gunaratne, Matsukhani, Lipari, Liou, Martindale, and Goldberg, 1986, Proc. Nat. Acad. Sci. USA 83: 701-05).

****In(1)z2*: Inversion (1) zeste**

cytology: *Dp(1;1)2C10-D1;4D2-4;18F-19A*; duplicated for 2D1-4D2.

new order:

1 - 4D2|18F - 2D1|19A - 20.

origin: X ray induced.

discoverer: Gans.

references: 1953, Bull. Biol. Fr. Belg., Suppl. 38: 1-90.

***In(1)ZP*: Inversion (1) Zacharopoulou Pelecanos**

cytology: *In(1)IA;3D*.

origin: Spontaneous in natural population in Greece.

references: Zacharopoulou, 1974, DIS 51: 52-53.

Zacharopoulou and Pelecanos, 1980, Genetica 54: 105-11.

***In(1)ZWD*: Inversion (1) Zeste White Disrupter**

origin: γ ray induced.

references: Smolik-Utlaut, and Gelbart, 1987, Genetics 116: 285-98.

inversion	cytology	male phen.	female phen.
<i>In(1)ZWD2</i>	3C1-2;10D	w ⁺ eyes	w ⁺ eyes
<i>In(1)ZWD3</i>	3C1-2;3F	w ⁺ ec eyes	brick red, ec eyes
<i>In(1)ZWD4</i>	3C1-2;4E	w ⁺ , rough eyes; sterile	
<i>In(1)ZWD6</i>	see <i>T(1;3)ZWD6</i>		
<i>In(1)ZWD7</i>	3C1-2;3E	w ⁺ eyes	mottled eyes
<i>In(1)ZWD8</i>	3C;18F + <i>Df(1)18F;20</i>	lethal	
<i>In(1)ZWD10</i>	see <i>Tp(1;3)ZWD10</i>		

In₁(1)^K: see *In(1)m^K*

***In(1LR)l-v139*: Inversion (1LR) lethal-variegated**

cytology: *In(1LR)3C6-7*.

origin: X ray induced.

discoverer: Lindsley, Edington, and Von Halle.

references: 1960, Genetics 45: 1649-70.

Gersh, 1965, Genetics 51: 477-80 (fig.).

genetics: Variegated for w, rst, and a lethal; requires two Y chromosomes for survival; X/Y/Y male fertile. Recombinant carrying left end of the X^{P4D} element of *T(1;4)w^{m5} = T(1;4)3C3-4;101F1-2* and right end of *In(1LR)l-v139* variegates for w but not for rst or the lethal.

***In(1LR)sc²⁶⁰⁻²⁵*: Inversion (1LR) scute**

cytology: *In(1LR)1B2-3*.
origin: X ray induced.
discoverer: Sutton, 1940.
synonym: *Tp(1;1)sc²⁶⁰⁻²⁵*.
references: 1940, Genetics 25: 628-35 (fig.).
genetics: Mutant for *sc*; variegates for *y* and *ac* but not *svr*. Genetic tests indicate loci of *l(1)IAc*, *y*, and *ac* are located at the base of *X* to the right of *bb*. Sutton judged it to be a transposition of 1A1-B2 into the proximal heterochromatin, but since this requires three breaks with one to the left of 1A1, a pericentric inversion is deemed more probable. Recombination between *In(1LR)sc²⁶⁰⁻²⁵* and a normal sequence yields *Df(1)sc²⁶⁰⁻²⁵* = *Df(1)1B2-3* and *Dp(1;1)sc²⁶⁰⁻²⁵* = *Dp(1;1)1B2-3*. The deficiency is deficient for *l(1)IAc*, *y*, and *ac* (Sutton, 1940).

***In(1LR)sc^{V1}*: Inversion (1LR) scute of Valencia**

cytology: *In(1LR)1A8-C3*; inferred from genetic results.
origin: γ ray induced.
discoverer: J. I. Valencia, 46h23.
synonym: *Inp(1)sc^{V1}* (*Inp* symbolized a pericentric inversion).
references: Muller and Valencia, 1947, DIS 21: 69-70.
genetics: Mutant for *ac* and *sc*. A single exchange between *In(1LR)sc^{V1}* and a normal *X* chromosome produces one recombinant with the left end of *In(1LR)sc^{V1}* that is deficient for the tip of *X*, *Df(1)sc^{V1}*, and one with the right end of *In(1LR)sc^{V1}* that is duplicated for the tip of *X*, *Dp(1;1)sc^{V1}*. Left break between *ac* and *M(1)1B* based on observation that *Dp(1;1)sc^{V1}* is duplicated for *ac*, and *Df(1)sc^{V1}* is deficient for *ac* but not *M(1)1B*. Right break in *XR*. *Dp(1;1)sc^{V1}* carrying *y* in normal position and *y⁺* in duplicated region provides an excellent marker system for right end of the *X*.

In(Y)

origin: Induced in *y⁺Y* by X rays (Brosseau, 1960) or by γ rays (M. Schwartz); and in *B^SYy⁺* by X rays.
genetics: Male sterile; mutant sterility factors indicated in table below. Presence of inversions indicated by examination of prophase or metaphase figures.

inversion	cytology	ref ^α	genetics
<i>In(YL)A49</i>		4	<i>kl-2⁻</i>
<i>In(YL)B2^β</i>	<i>h9;h15</i>	1, 3	<i>kl-5⁻-kl-3⁻</i>
<i>In(YL)B3</i>	<i>h1-2;h3</i>	1, 3	<i>kl-5⁻</i>
<i>In(YL)D43</i>	<i>h2;h9</i>	2	
<i>In(YL)G11</i>	<i>h7;h10</i>	2	
<i>In(YL)S15^γ</i>	<i>h7;h11</i>	3	<i>kl-3⁻-kl-1⁻</i>
<i>In(YL)S16^δ</i>	<i>h7;h10-11 + h13;h17-18</i>	3	<i>kl-3⁻-kl-2⁻</i>
<i>In(YLS)A33</i>		4	male sterile
<i>In(YLS)B1</i>	<i>h14-15;h19</i>	1, 3	<i>kl-1⁻</i>
<i>In(YLS)S18</i>	<i>h10-11;h20</i>	3	<i>kl-2⁻</i>
<i>In(YLS)S19</i>	<i>y⁺Xhj;h21-22</i>	3	<i>ks-1⁻</i>
<i>In(YS)S17</i>	<i>h21;h25</i>	3	<i>ks-1⁻-ks-2⁻</i>

^α 1 = Brosseau, 1960, Genetics 45: 257-74; 2 = Gatti; 3 = Gatti and Pimpinelli, 1983, Chromosoma 83: 349-73. 4 = Hazelrigg, Fornili, and Kaufman, 1982, Chromosoma 87: 535-59.

^β Chromosome includes *Df(YL)B2* = *Df(YL)h3;h8*.

^γ Mutant located proximal to inversion?

^δ Chromosome carries two inversions.

***In(2)bw^R*: *Inversion (2) brown-Rearranged**

origin: X ray induced.
references: Slatis, 1955, Genetics 40: 5-23.

inversion	cytology
<i>*In(2)bw^{R18}</i>	<i>40F-41A;59E4-F1</i>
<i>*In(2)bw^{R35}</i>	<i>40F-41A;59D11-E1</i>
<i>*In(2)bw^{R45}</i>	<i>40F-41A;59E3-4</i>
<i>*In(2)bw^{R47}</i>	<i>40-41;59D11-E1</i>
<i>*In(2)bw^{R56}</i>	<i>40F;41A;59D-E</i>
<i>*In(2)bw^{R67}</i>	<i>40F-41A;59E4-F1</i>
<i>*In(2)bw^{R73}</i>	<i>40-41A;59E4-F1</i>
<i>*In(2)bw^{R79}</i>	<i>40F-41A;59F2-3</i>

***In(2)C*: Inversion (2) Crossover suppressor**

origin: X ray induced.
references: Roberts, 1970, Genetics 65: 429-48.

inversion	cytology	genetics
<i>In(2)C56</i>	<i>40-41;59B</i>	homozygous lethal; recombination between <i>b</i> and <i>sp</i> much reduced
<i>In(2)C113</i>	<i>40-41;46D</i>	homozygous lethal; recombination between <i>b</i> and <i>sp</i> reduced
<i>In(2)C162</i>	<i>36B-C;40-41</i>	homozygous viable; recombination between <i>al</i> and <i>b</i> much reduced
<i>In(2)C224</i>	<i>25E;40-41</i>	homozygous lethal; recombination between <i>al</i> and <i>b</i> virtually eliminated
<i>In(2)C282</i>	<i>31E;40-41</i>	homozygous viable; recombination between <i>al</i> and <i>b</i> much reduced

In(2)DTD43*: see *T(2;3)DTD43

In(2)H164

cytology: *In(2)40-41;44F*.
origin: X ray induced.
references: Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84.
genetics: Associated with *T(Y;2)H164=T(Y;2)YS;25E*.

***In(2)TE34Cc*: Inversion (2) Transposing Element**

cytology: *In(2)34C4-5;40-41*.
origin: γ ray induced in *TE34Cc*.
discoverer: Angel.
synonym: *In(2)TE94Z*.
genetics: Variegates for *w⁺*.

In(2)TE35A

origin: γ ray induced except for *In(2)TE35A-1*, which is spontaneous.
synonym: *In(2)TE146Z*.

inversion	cytology	discoverer	genetics
<i>In(2)TE35A-1</i>	<i>35B;40-41</i>		
<i>In(2)TE35A-101</i>	<i>35B;40-41</i>		associated with <i>pu-l(2)35Dg</i>
<i>In(2)TE35A-219</i>	<i>35B;40-41</i>	Samkange	

In(2)TE94R*: see *In(2)TE34Cc

***In(2)TE146Z*: see *In(2)TE35A* Eye color mottled.**

In(2L)47

cytology: *In(2L)37A2-B1;38A6-C1*.

***In(2L)53d**

cytology: *In(2L)25A;29F*.
origin: Neutron induced.
discoverer: Mickey, 53d4.
references: 1963, DIS 38: 29.
other information:

In(2L)75c

cytology: *In(2L)27D1-5;35A1-2;35D4-7*.
new order:
 21 – 27D1|35A1–27D5|35A2– 60;
 deficient for 35A1-D7.
origin: X ray induced.
references: Woodruff and Ashburner, 1979, Genetics 92: 117-32.
 Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.
 Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux’s Arch. Dev. Biol. 193: 267-82.
genetics: Deficiency uncovers *wb – lace*.

In(2L)75c^LC158.1^R

cytology: *In(2L)27D1-2;35A1-2^L26D1-2;35B3^R*;
 deficient for 35A2-B3; duplicated for 26D2-27D1.
new order:
 21 – 27D1|35A1 – 26D2|35B3–60F.
origin: Recombinant between left end of *In(2L)75c* and right end of *In(2L)C158.1*.
synonym: *Dp(2;2)C75RL;Df(2L)C75RL*.
references: Woodruff and Ashburner, 1979, Genetics 92: 117-32.
 Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.
genetics: Deficient for *wb - Adh*. Duplication does not suppress *Sp*.

In(2L)75c^LC163^R

cytology: *In(2L)27D1-2;35A1-2^L27D1-2;35E1-2^R*;
 deficient for 35A2-E1.
origin: Recombinant between the left end of *In(2L)75c^LC158^R* and the right end of *In(2L)C163*.
discoverer: Ashburner.
genetics: Deficient for *wb – lace*.

In(2L)A

cytology: *In(2L)26A;33E*.
origin: Naturally occurring inversion.
discoverer: Oshima and Watanabe.
references: 1965, DIS 40: 88.
 Watanabe, 1967, Mem. Fac. Sci. Kyushu Univ., Ser. E: 159-82.
 Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.
 Mettler, Voelker, and Mukai, 1977, Genetics 87: 169-76.
 Inoue and Watanabe, 1979, Jpn. J. Genet. 54: 69-82.

In(2L)AL1: Inversion (2L) Ashburner Lemeunier

cytology: *In(2L)22A;26A*.
origin: Spontaneous in a natural population.
references: Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

In(2L)Arp1: Inversion (2L) Aristapediid

cytology: *In(2L)49A12-B3;49E5-F1*.
origin: Hybrid dysgenesis.

references: Adler, 1984, Genetics 107: s1.
genetics: Mutant for *Arp* in heterozygotes. Embryonic recessive lethal at proximal breakpoint.

In(2L)Arp2

cytology: *In(2L)49A12-B3;49E5-F1*.
origin: Hybrid dysgenesis.
references: Adler, 1984.
genetics: Not identical to *In(2L)Arp1* (based on crosses to *vg* deletions). Recessive lethals at proximal and distal breakpoints.

In(2L)ast: Inversion (2L) asteroid

cytology: *In(2L)21E1-3;40*.
origin: γ ray induced.
references: Roberts, Brock, Rudden, and Evans-Roberts, 1985, Genetics 109: 145-56.
genetics: Mutant for *ast*.

***In(2L)ast^{rv2}: Inversion (2L) asteroid-reverted**

cytology: *In(2L)21E2-3;31*.
origin: X ray induced in *ast*.
discoverer: E. B. Lewis, 1942.
references: 1945, Genetics 30: 158.
genetics: Partial reversion of *ast*.

In(2L)AWI

origin: Accumulated on second chromosomes during repeated backcrossing, these second chromosomes coming from a lethal-carrying chromosome *AW* derived in 1967 from a wild-type cage population.
references: Yamaguchi and Mukai, 1974, Genetics 78: 1209-21.

inversion	cytology
<i>In(2L)AWI1</i>	22D;30B
<i>In(2L)AWI2</i>	22E;24C
<i>In(2L)AWI3</i>	23B;29F
<i>In(2L)AWI4</i>	23E;26B
<i>In(2L)AWI5</i>	25C;32C
<i>In(2L)AWI6</i>	26A;30D
<i>In(2L)AWI7</i>	26A;31B ^α
<i>In(2L)AWI8</i>	26A;33B
<i>In(2L)AWI9</i>	27D;29F
<i>In(2L)AWI10</i>	29F;33B
<i>In(2L)AWI11</i>	33A;37B
<i>In(2L)AWI12</i>	33B;35F
<i>In(2L)AWI13</i>	34A;36A
<i>In(2L)AWI14</i>	35B;36A

^α Superimposed on *In(2L)AWI8*.

In(2L)b: Inversion (2L) black

inversion	cytology	origin	ref ^α	genetics
<i>In(2L)b 79d5</i>	34D4;35B10	neutrons	1	lethal
<i>In(2L)b 80c2</i>	34C7;34D6-7	γ ray	2	<i>b</i>
<i>In(2L)b 81f3</i>	34D2-4;35B10	neutrons	1	<i>b</i> ; homozygous viable
<i>In(2L)b 81i7</i>	34D2-4;40F	γ ray	1	lethal
<i>In(2L)b 82c44</i>	34D4;40F	neutrons	1	<i>b</i> ; homozygous viable but sterile
<i>In(2L)b 83b2</i>	34D4;35B10	γ ray	1	lethal
<i>In(2L)b 85f2</i>	33A1-2;35E3-4 + <i>Df(2L)34D2-4;</i> <i>34E6-F1</i>	neutrons	1	lethal

^α 1 = Alexandrov and Alexandrova, 1986, DIS 63: 159-61; 2 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

In(2L)bib8: Inversion (2L) big brain

discoverer: Campos-Ortega.
cytology: *In(2L)30A9;30F*.

In(2L)C127

cytology: *In(2L)23C;32A*.
origin: X ray induced.
discoverer: Roberts, 1965.
references: 1970, Genetics 65: 429-48.
genetics: Homozygous viable. Recombination between *a* and *b* virtually eliminated.

In(2L)C158

cytology: *In(2L)26D1-2;35B3*.
origin: Spontaneous in a natural population from Finland.
references: Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.
Woodruff and Ashburner, 1979, Genetics 92: 117-32.
Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-195.
genetics: Region 35 breakpoint mapped between *Adh* and *l(2)35Bb* (Ashburner), but breakpoint not associated with lethal. Heterozygote *Df(2L)75cIn(2L)C158* viable and fertile (Woodruff and Ashburner, 1979).

In(2L)C158^LSco^{rv11R}

cytology: *In(1)26D1-2;35B3^L24C3-9;35D1-2^R*; duplicated for 24C9 to 26D1; deficient for 35B3-D1.
origin: Recombinant between left end of *In(2L)C158* and right end of *In(2L)Sco^{rv11}*.
synonym: *Dp(2;2)C158.1^LSco^{rv11R};Df(2L)C158.1^LSco^{rv11R}*.
references: Velissariou and Ashburner, 1980, Chromosoma 77: 13-27.
Ashburner, Tsubota, and Woodruff, 1982, Genetics 102: 401-20.
genetics: Duplicated for *M(2)24F* and carries both *Sgs1^c* and *Sgs1^e* (Velissariou and Ashburner, 1980); deficient for *l(2)35Bb - l(2)35Da*.

In(2L)C158^LSco^{rv17R}

cytology: *In(1)26D1-2;35B3^L25D3;35D1^R*; duplicated for 25D7 to 26D1-2; deficient for 35B3-D1.
new order:
21 – 26D1|35B3 – 25D7|35D1 – 60.
origin: Recombinant between left end of *In(2L)C158* and right end of *In(2L)Sco^{rv17}*.
synonym: *Dp(2;2)C158.1^LSco^{+R17R};Df(2L)C158.1^LSco^{+R17R}*.
references: Velissariou and Ashburner, 1980, Chromosoma 77: 13-27.
genetics: Deficient for *l(2)35Bb - l(2)35Da*.

In(2L)C163.41

cytology: *In(2L)27D1-2;35E1-2*.
origin: Spontaneous in a natural population in Oklahoma.
references: Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.
Woodruff and Ashburner, 1979, Genetics 92: 117-32.
Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.
genetics: Proximal breakpoint maps between *l(2)35Cd* and *l(2)35Ea*.

In(2L)C163^LC158^R

cytology: *In(1)27D1-2;35E1-2^L26D1-2;35B3^R*; duplicated for 26D2-27D1 and for 35B3-35E1.
origin: Recombinant between left end of *In(2L)C163* and

right end of *In(2L)C158*.

discoverer: Littlewood.
synonym: *Dp(2;2)C163.41^LC158.1^R*.
references: Ashburner, 1982, Genetics 101: 447-59.
genetics: Dominant enhancer of *H*. Proximal duplication (region 35) covers *l(2)35Bb - lace*.

In(2L)C236

cytology: *In(2L)22B;25F*.
origin: X ray induced.
discoverer: Roberts, 1965.
references: 1970, Genetics 65: 429-48.
genetics: Homozygous lethal. Recombination between *a* and *b* reduced.

In(2L)C263

cytology: *In(2L)24C;25F;26F*; 25F-26F missing.
new order:
21 – 24C|25F – 24C|26F – 60.
origin: X ray induced.
references: Roberts, 1970, Genetics 65: 429-48.
genetics: *In(2L)C263/SM1* and *In(2L)C263/SM5* females nearly sterile. Recombination reduced in *2L*. Homozygous lethal.

In(2L)CA

origin: γ ray induced.
discoverer: Ashburner.

inversion	cytology
<i>In(2L)CA26</i>	29D2;34D4
<i>In(2L)CA38</i>	22A1;34A1-2
<i>In(2L)CA39</i>	24E-F;28A-B
<i>In(2L)CA51^α</i>	29E;34A7-11;36A1-2;40

^α new order: 21 – 29E|34A11 – 36A1|34A7 – 29E|40; 40|36A – 40.

In(2L)CA11

cytology: *In(2L)21D;36F*.
origin: γ ray induced in sperm along with *In(3L)jz³=In(3L)70D6-7;75D3-8 + In(3L)73D3-5;80-81*.
discoverer: Velissariou.
references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.
Velissariou and Ashburner, 1981, Chromosoma 84: 173-85.

In(2L)CA38

cytology: *In(2L)22A1;34A1*.
origin: γ rays.
references: Ashburner. Associated with *cosI*.

In(2L)Cy: Inversion (2L) Curly

cytology: *In(2L)22D1-2;33F5-34A1*. Breakpoints close to those of *In(2L)t* and thus often confused with it (Ashburner and Lemeunier, 1976).
origin: Naturally occurring inversion.
discoverer: Ward, 21f.
references: 1923, Genetics 8: 276-300.
Sturtevant, 1931, Carnegie Inst. Washington Publ. No. 421: 20.
Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.
genetics: Exists with and without *Cy*. Homozygous viable without *Cy*. Crossing over in *In(2L)Cy/+* heterozygote

greatly reduced in *2L* (also see *In(2R)Cy*).

***In(2L)Cy^{LtR}*: Inversion (2L) Curly-Left t-Right**

cytology: *In(2L)22D1-2;33F5-34A1^L22D3-E1;34A8-9^R*.

Deficient for 22D2-6 and 34A1-8.

origin: Recombinant carrying left end of *In(2L)Cy* and right end of *In(2L)t*.

discoverer: Bridges.

references: Morgan, Bridges, and Schultz, 1937, Year Book - Carnegie Inst. Washington 36: 300-1.

genetics: Acts as suppressor of *S*.

In(2L)Cy^{RC1}

cytology: *In(2L)23B;24B + In(2L)22D3-E1;34A8-9*.

origin: X ray induced in *In(2L)t*, *Cy Roi*.

synonym: *In(2L)Cy^{+RC1}*.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou and Woodruff, 1980, DIS 55: 193-95.

genetics: *Cy* revertant.

In(2L)D6*: see *T(Y;2)D6

In(2L)D219*: see *T(Y;2)D219

***In(2L)dl^H*: Inversion (2L) dorsal**

cytology: *In(2L)36C;37B-C* (just distal to *Ddc*).

synonym: *dl⁴⁰²⁸*.

references: Steward, McNally, and Schedl, 1984, Nature (London) 311: 262-65.

Steward, Ambrosio, and Schedl, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 223-28.

genetics: Mutant for *dl* over *Df(2L)TW119* (deficient for 36C). Homozygous lethal.

molecular biology: 36C breakpoint localized on the molecular map (Steward, McNally, and Schedl, 1984; Steward, Ambrosio, and Schedl, 1985).

In(2L)dl^T

cytology: *In(2L)21E-F;36C*.

synonym: *dl¹⁴²⁸*.

references: Steward, McNally, and Schedl, 1984, Nature (London) 311: 262-65.

Steward, Ambrosio, and Schedl, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 223-28.

genetics: Mutant for *dl* over *Df(2L)TW119* (deficient for 36C). Homozygous lethal.

molecular biology: 36C breakpoint localized on the molecular map (Steward, McNally, and Schedl, 1984; Steward, Ambrosio, and Schedl, 1985).

***In(2L)dp^{olvr}*: Inversion (2L) dumpy-oblique lethal vortex Ruffled**

cytology: *In(2L)25A;25B3-4*.

origin: X ray induced.

discoverer: Schultz, 33a25.

genetics: Mutant at *dp*. Homozygous lethal.

In(2L)dp^{w18}

discoverer: Craymer.

genetics: Mutant for *dp*.

In(2L)dp-1

cytology: *In(2L)25A2-3;28C7-D3*.

origin: γ ray induced.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.

genetics: Mutant for *dp*.

In(2L)dp-2

cytology: *In(2L)22B;25A2-8*.

origin: γ ray induced.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.

genetics: Mutant for *dp*.

***In(2L)dpp*: Inversion (2L) decapentaplegic (W.M. Gelbart)**

genetics: Mutant for *dpp*.

allele	class	origin	cytology	ref ^{α}
<i>In(2L)dpp¹</i>	d-III	hobo	<i>In(2L)22E2-3;22F2-3</i>	1,4
<i>In(2L)dpp²</i>	d-III	X-ray	<i>In(2L)22F1-2;28B</i>	1,4
<i>In(2L)dpp⁶</i>	d-III	X-ray	<i>In(2L)22F1-3;24F2-6</i>	1,3,4
<i>In(2L)dpp⁸</i>	d-III	X-ray	<i>In(2L)22A1-2;22F1-2</i>	1,3,4
<i>In(2L)dpp¹⁰</i>	d-III	X-ray	<i>In(2L)22E4-F1;23E2-4</i>	1,3,4
<i>In(2L)dpp¹²</i>	d-V	X-ray	<i>In(2L)22E2-3;22F2-3</i>	1,3,4
<i>In(2L)dpp¹⁷</i>	d-III	X-ray	<i>In(2L)22F1-3;27E</i>	1,4
<i>In(2L)dpp¹⁸</i>	d-III	X-ray	<i>In(2L)22F1-2;36C4-6</i>	1
<i>In(2L)dpp⁴¹</i>	d-III	γ -ray	<i>In(2L)22F2-4;54F</i>	1
<i>In(2L)dpp⁴²</i>	d-V	γ -ray	<i>In(2L)22F1-3;22F3-4</i>	1
<i>In(2L)dpp⁵⁰</i>	d-II	γ -ray	<i>In(2L)22F2-3;27C</i>	1
<i>In(2L)dpp⁶⁰</i>	d-III	γ -ray	<i>In(2L)21E;22F</i>	1
<i>In(2L)dpp⁶⁵</i>	d-V	γ -ray	<i>In(2L)22F;34C;40</i>	1
<i>In(2L)dpp^{H86}</i>	Hin?	γ -ray	<i>In(2L)22F;26C;35D-E</i>	1
<i>*In(2L)dpp^{ho40}</i>	d-II	X-ray	<i>In(2L)21D4-E1;22E2-3</i>	1,2,4
<i>In(2L)dpp^{s4}</i>	shv-inc	X-ray	<i>In(2L)21B1-C1;22F1-2</i>	1
<i>In(2L)dpp^{s5}</i>	shv-inc	X-ray	<i>In(2L)21E1-2;22F1-2</i>	1
<i>In(2L)dpp^{s11}</i>	shv-p	γ -ray	<i>In(2L)22F1-2;31C-D</i>	1
<i>In(2L)dpp^{s12}</i>	shv-lc	γ -ray	<i>In(2L)22F1-2;24A</i>	1
<i>In(2L)dpp^{s20}</i>	shv-lc	γ -ray	<i>In(2L)22B1-2;22F1-2</i>	1
<i>In(2L)dpp^{s21}</i>	shv-inc	γ -ray	<i>In(2L)22A1-3;22F1-2</i>	1
<i>In(2L)dpp^{s22}</i>	shv-p	γ -ray	<i>In(2L)22F1-2;35C-D</i>	1
<i>In(2L)dpp^{t24}</i>	t	X-ray	<i>In(2L)22F1-2;58B</i>	1
<i>In(2L)dpp^{t63}</i>	t	γ -ray	<i>In(2L)22F;39C-D</i>	1
<i>In(2L)dpp^{Tg}</i>	Tg	X-ray	<i>In(2L)21C;22F</i>	1

^{α} 1 = Gelbart; 2 = Lewis, 1945, Genetics 30: 137-66; 3 = Segal and Gelbart, 1985, Genetics 109: 119-43; 4 = Spencer, Hoffmann, and Gelbart, 1982, Cell 28: 451-61.

In(2L)DTD

inversion	cytology	ref ^{α}
<i>In(2L)DTD27</i>	21B;40	1, 2
<i>In(2L)DTD94</i>	21F;40F	2
<i>In(2L)DTD104</i>	32E-F;41A + <i>In(2L)23D-E;28D</i>	2

^{α} 1 = Craymer, 1984, DIS 60: 81-82; 2 = Gelbart.

***In(2L)E(SD)3*: Inversion (2L) Enhancer of Segregation distorter**

cytology: *In(2L)29E-30A;34A-35B*.

origin: γ ray induced in *SD5*.

synonym: *In(2L)SD^{+R3}*.

references: Ganetzky, 1977, Genetics 86: 321-55.

genetics: Associated with *Df(2L)E(SD)3*, which is apparently responsible for the partial reversion of *SD*.

In(2L)EJ

cytology: *In(2L)23A3;27B2*.

origin: Spontaneous in a natural population.

discoverer: Vallaso.

***In(2L)el⁹* : Inversion (2L) elbow**

cytology: *In(2L)34A2-3;35A3-4*.
origin: γ rays.
discoverer: Johnson.
references: Ashburner.
genetics: Associated with *el⁹*.

In(2L)el18

cytology: *In(2L)35B;36C2-11 + Df(2L)el18*.
origin: γ rays.
references: Ashburner.
genetics: Deficient for *wb - l(2)35Ea*.

In(2L)Epa: Inversion (2L) Epaulet

cytology: *In(2L)25A1-4;35D1-2*.
origin: γ rays.
discoverer: Harrington.
genetics: Associated with *Cos¹⁰*.

In(2L)ho⁴⁰ : see *In(2L)dpp^{ho40}*

In(2L)ID

cytology: *In(2L)31B;38C*.
origin: In second chromosome of isogenic line *ID* derived from Ames I, Oregon-R-C lab stock.
references: Kidwell and Kidwell, 1975, J. Hered. 66: 367-75.

In(2L)IW: Inversion (2L) Inoue Watanabe

origin: Spontaneous in natural population in Japan.
references: Inoue and Watanabe, 1979, Jpn. J. Genet. 54: 69-82.

inversion	cytology
<i>In(2L)IW3</i>	21A;30E
<i>In(2L)IW4</i>	26A;31F
<i>In(2L)IW5^α</i>	28C;32C

^α Synonym: *In(2L)W*.

In(2L)JHI

origin: Accumulated on second chromosomes during repeated backcrossing. These second chromosomes come from a lethal-carrying chromosome *JH* derived in 1967 from a wild-type cage population.
references: Yamaguchi and Mukai, 1974, Genetics 78: 1209-21.

inversion	cytology
<i>In(2L)JHI1</i>	21C;26F
<i>In(2L)JHI2</i>	22C;26A
<i>In(2L)JHI3</i>	22F;26A
<i>In(2L)JHI4</i>	22F;26A
<i>In(2L)JHI5</i>	24A;29F
<i>In(2L)JHI6</i>	24D;26B
<i>In(2L)JHI7</i>	25E;26B
<i>In(2L)JHI8</i>	26A;27C
<i>In(2L)JHI9</i>	26A;27D
<i>In(2L)JHI10</i>	26A;27E
<i>In(2L)JHI11</i>	26A;28B
<i>In(2L)JHI12</i>	26A;29B
<i>In(2L)JHI13</i>	26A;29C
<i>In(2L)JHI14</i>	26A;29D
<i>In(2L)JHI15</i>	26A;29E
<i>In(2L)JHI16</i>	26A;29E
<i>In(2L)JHI17</i>	26A;29E
<i>In(2L)JHI18</i>	26A;29F
<i>In(2L)JHI19</i>	26A;30D
<i>In(2L)JHI20</i>	26A;31F
<i>In(2L)JHI21</i>	26A;31D
<i>In(2L)JHI22</i>	26A;32A

inversion	cytology
<i>In(2L)JHI23</i>	26A;32D
<i>In(2L)JHI24</i>	26A;33B
<i>In(2L)JHI25</i>	26A;34A
<i>In(2L)JHI26</i>	26A;34A
<i>In(2L)JHI27</i>	26A;35A
<i>In(2L)JHI28^α</i>	26A;35F
<i>In(2L)JHI29</i>	26A;38D
<i>In(2L)JHI30</i>	26B;28D
<i>In(2L)JHI31</i>	26B;29C
<i>In(2L)JHI32</i>	26E;29B
<i>In(2L)JHI33</i>	26F;32D
<i>In(2L)JHI34</i>	27B;27C
<i>In(2L)JHI35</i>	27B;29C
<i>In(2L)JHI36</i>	27C;34C
<i>In(2L)JHI37^β</i>	27D;28F
<i>In(2L)JHI38</i>	27D;29D
<i>In(2L)JHI39</i>	27D;34C
<i>In(2L)JHI40</i>	28B;30A
<i>In(2L)JHI41</i>	28B;34D
<i>In(2L)JHI42</i>	28C;31E
<i>In(2L)JHI43</i>	28D;30B
<i>In(2L)JHI44</i>	29B;34D
<i>In(2L)JHI45</i>	29C;34A
<i>In(2L)JHI46</i>	29D;43C
<i>In(2L)JHI47</i>	29E;30F
<i>In(2L)JHI48</i>	29E;35C
<i>In(2L)JHI49</i>	29E;35F
<i>In(2L)JHI50</i>	29F;34A
<i>In(2L)JHI51</i>	30A;36B
<i>In(2L)JHI52</i>	30B;34A
<i>In(2L)JHI53</i>	30B;38A
<i>In(2L)JHI54</i>	30B;39E
<i>In(2L)JHI55</i>	30B;39F
<i>In(2L)JHI56</i>	30C;36D
<i>In(2L)JHI57</i>	31B;34A
<i>In(2L)JHI58</i>	31B;34D
<i>In(2L)JHI59</i>	31B;38E
<i>In(2L)JHI60</i>	31C;36D
<i>In(2L)JHI61</i>	32B;40D
<i>In(2L)JHI62</i>	32C;36C
<i>In(2L)JHI63</i>	32D;34A
<i>In(2L)JHI64^γ</i>	32D;34A
<i>In(2L)JHI65</i>	32D;36A
<i>In(2L)JHI66</i>	34E;38D
<i>In(2L)JHI67^δ</i>	34A;35B
<i>In(2L)JHI68</i>	34A;39A
<i>In(2L)JHI69</i>	34D;38F
<i>In(2L)JHI70</i>	34E;38E
<i>In(2L)JHI71</i>	35A;40D
<i>In(2L)JHI72</i>	35F;39E

^α Superimposed on *In(2L)JHI62*.

^β Superimposed on *In(2L)JHI38*.

^γ Superimposed on *In(2L)JHI26*.

^δ Superimposed on *In(2L)JHI64*.

In(2L)K

cytology: *In(2L)22D;26A*.

discoverer: Oshima and Watanabe.

references: 1965, DIS 40: 88.

Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

In(2L)Ka

cytology: *In(2L)22D;24A*.

origin: Spontaneous in a natural population in Korea.

references: Choi, 1977, DIS 52: 88.

1977, Genetika 47: 155-60.

In(2L)KA

cytology: *In(2L)22A;26B*.

origin: Spontaneous in natural populations in Korea.

references: Paik, 1986, DIS 63: 167.

In(2L)Kb

cytology: *In(2L)32B;34D*.
origin: Spontaneous in a natural population in Korea.
references: Choi, 1977, DIS 52: 88.
 1977, Genetika 47: 155-60.

In(2L)KB

cytology: *In(2L)37D;40A*.
origin: Spontaneous in a natural population in Korea.
references: Paik, Hong, and Sung, 1969, DIS 44: 67.
 Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

In(2L)KC - In(2L)KG

origin: Spontaneous in natural populations in Korea.
references: Paik, 1986, DIS 63: 167.

inversion	cytology
<i>In(2L)KC</i>	24C-D;36B-C
<i>In(2L)KD</i>	26B;29B-C
<i>In(2L)KE</i>	26C;29B-C
<i>In(2L)KF</i>	26C-D;32B-C
<i>In(2L)KG</i>	28A-B;32D

In(2L)L67: see ***T(Y;2)L67***

In(2L)L135: see ***T(Y;2)L135***

In(2L)lt^{G5}

cytology: *In(2L)24F-25A;40*.
discoverer: Craymer.

****In(2L)lt^m*: Inversion (2L) light-mottled**

origin: X ray induced.
discoverer: Hessler, 1957.
references: 1958, Genetics 43: 395-403.
genetics: Variegated for *lt*.

inversion	cytology
* <i>In(2L)lt m2</i>	22F-23A;40B-F
* <i>In(2L)lt m20</i>	32C;40B-F
* <i>In(2L)lt m26</i>	27C;40B-F

In(2L)M

cytology: *In(2L)30E;37A*.
origin: Spontaneous in a natural population in Japan.
references: Watanabe and Oshima, 1966, Ann. Rpt. Nat. Inst. Genetics Japan 16: 17-35.
 Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

****In(2L)M1: Inversion (2L) of Mourad***

cytology: *In(2L)38E;40F*.
origin: Spontaneous.
references: Mourad and Mallah, 1960, Evolution 14: 166-70.
 Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

****In(2L)M2***

cytology: *In(2L)21F;33A*.
origin: Spontaneous.
discoverer: Mourad and Mallah.
references: 1960, Evolution 14: 166-70.
 Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

In(2L)Mg: In (2L) Mglinetz

inversion	cytology	origin ^α	ref ^β
<i>In(2L)Mg1</i>	22A;26B	1	2
<i>In(2L)Mg81</i>	31C;40	1	1
<i>In(2L)Mg82</i>	37E;38E	1	1
<i>In(2L)Mg84</i>	24E;36E	1	1
<i>In(2L)Mg85</i>	22D;40	1	1
<i>In(2L)Mg86</i>	27C;37F	1	1
<i>In(2L)Mg87</i>	22D;40	2	1
<i>In(2L)Mg88</i>	22F;40B	2	1
<i>In(2L)Mg89</i>	21D;24D	2	1
<i>In(2L)Mg90</i>	25E;31D	2	1
<i>In(2L)Mg98</i>	26E;31E	1	1

^α 1 = γ ray induced; 2 = ³²P feeding.

^β 1 = Mglinetz, 1968, Genetika (Moscow) 4(8): 81-86; 2 = Mglinetz, 1971, Genetika (Moscow) 7(8): 108-14.

In(2L)noc²: Inversion (2L) no-ocelli

cytology: *In(2L)35B1-2;36D3*.
origin: Induced by ethyl methanesulfonate.
discoverer: Tsubota.
references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou and Walker, 1981, DIS 56: 186-91.
 Ashburner, Tsubota, and Woodruff, 1982, Genetics 102: 401-20.
 Ashburner, Aaron, and Tsubota, 1982, Genetics 102: 421-35.
 Chia, Karp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706.
 Gubb, Roote, Harrington, McGill, Durrant, Shelton, and Ashburner, 1985, Chromosoma 92: 116-23.
genetics: Associated with *noc²*.

In(2L)NS: Inversion (2L) from Nova Scotia

cytology: *In(2L)23E2-3;35F1-2* (Bridges and Li in Morgan, Bridges, and Schultz, 1936, Year Book - Carnegie Inst. Washington 35: 292).
origin: Naturally occurring inversion.
discoverer: Sturtevant, 13i.
synonym: CIII; C2L.
references: Sturtevant, 1919, Carnegie Inst. Washington Publ. No. 278: 305-41.
 Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.
 Mettler, Voelker and Mukai, 1977, Genetics 87: 169-76.
genetics: Crossing over in *2L* greatly reduced; none between *S* and *b*; 0.3% between *b* and *pr*. Small inter-chromosomal effect at proximal end of *X* in *In(2L)NS* and *In(2R)NS/+*. (Valentin, 1972, Hereditas 72: 243-54).

In(2L)osp²²: Inversion (2L) outspread

cytology: *In(2L)35B3;38D3-5*.
origin: Induced by ethyl methanesulfonate.
discoverer: Detwiler.
references: Chia, Karp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706.
genetics: Associated with *osp²²*.
molecular biology: Position of distal breakpoint localized to a 2.4 kilobase fragment close to the origin of a 165 kb walk.

In(2L)osp⁵⁹

cytology: *In(2L)35B3;38B3-6*.
origin: γ ray induced.
discoverer: Detwiler.

references: Chia, Karp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706.
genetics: Associated with *osp*⁵⁹.
molecular biology: Position of distal breakpoint localized to a 2.4 kilobase fragment close to the origin of a 165 kb walk.

In(2L)PA - In(2L)PC: Inversion (2L) Pipkin

origin: Naturally occurring inversions.
references: Pipkin, Franklin-Springer, Law, and Labega, 1976, J. Hered. 67: 258-66.

inversion	cytology
In(2L)PA	23A;31C-D on <i>In(2L)t</i> = <i>In(2L)22D3-E1;34A8-9</i>
In(2L)PB	33A;35C-D
In(2L)PC	25F;34B-F

In(2L)pr⁴⁰: Inversion (2L) purple

cytology: *In(2L)21D-E;38B*.
references: Reuter and Wolff, 1981, Mol. Gen. Genet. 182: 516-19.

In(2L)PS: Inversion (2L) Paik Sung

origin: Naturally occurring inversions in Korea.
references: Paik and Sung, 1980, DIS 55: 120.

inversion	cytology
In(2L)PS5	22A;26B
In(2L)PS6	22A;33B
In(2L)PS7	22B;25C
In(2L)PS8	23B;25E-F
In(2L)PS9	23E;33E
In(2L)PS10	24A;31F
In(2L)PS11	25B;28C
In(2L)PS12	26A;31A
In(2L)PS13	26A;34E
In(2L)PS14	37A;40A
In(2L)PS15	31F;35D
In(2L)PS16	31F;36F
In(2L)PS17	37A;40A
In(2L)PS18	37E;39E

In(2L)R145: see **T(Y;2)R145**

In(2L)R147: see **T(Y;2)R147**

In(2L)RBR

origin: Spontaneous [accumulated on second chromosome lines during repeated backcrossing of males heterozygous for each second chromosome line over *In(2LR)bw*^{V1} to *In(2LR)bw*^{V1}/*SM1* females].

references: Yamaguchi, Cardellino, and Mukai, 1976, Genetics 83: 409-22.

inversion	cytology
In(2L)RBR2	28E;37E
In(2L)RBR3	22A;22F
In(2L)RBR8	23A;33B
In(2L)RBR24	22A;30B + 23F;35B
In(2L)RBR26	32C;33E
In(2L)RBR37	see <i>T(2;3)RBR37-1</i>
In(2L)RBR40	29F;35F
In(2L)RBR50	22E;33E
In(2L)RBR68-1	22A;23A
In(2L)RBR68-2	32E;36A
In(2L)RBR70	22E;28E
In(2L)RBR93	26A;34A
In(2L)RBR109	21D;34D + 26D;34D
In(2L)RBR115	21E;34B + 28E;34B
In(2L)RBR118	22D;34A

inversion	cytology
In(2L)RBR132	21C;34D + 31E;34D
In(2L)RBR141	30E;38F

In(2L)S14: Inversion (2L) Segal

cytology: *In(2L)26A;28C*.
origin: γ ray induced with *T(2;4)shv*^{S14}.
references: Segal and Gelbart, 1985, Genetics 109: 119-43.

In(2L)Sco^{rv}: Inversion (2L) Scutoid - revertant

origin: X ray induced reversions of *Sco* in *Tp(2;2)Sco*.
references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.
 Ashburner, Detwiler, Tsubota, and Woodruff, 1983, Genetics 104: 405-31.
 Ashburner and Harrington, 1984, Chromosoma 89: 329-37.

inversion	cytology	genetics
In(2L)Sco^{rv2}	35D1-2;36D3	includes <i>noc-l(2)35Cb</i> ; homozygous lethal
In(2L)Sco^{rv4} α	six breaks	<i>noc⁻-l(2)35Cb⁻</i>
*In(2L)Sco^{rv5} β	35D1-2;38A3-8	
In(2L)Sco^{rv8}	34C1-2;35D1-2	homozygous lethal as pharate adults; lethal with <i>Sco</i>
In(2L)Sco^{rv11} γ	24C3-9;35D1-2	homozygous semi-lethal as pharate adults; escapers extreme <i>Sco</i>
In(2L)Sco^{rv17} γ	25D3-7;35D1-2	homozygous lethal
In(2L)Sco^{rv21}	35D1-2;36E1-2	homozygous semi-viable and <i>Sco</i>
In(2L)Sco^{rv24}	34B1-2;35D1-2	homozygous lethal
In(2L)Sco^{rv26} δ	35D1-2;40	semilethal with <i>sna</i> ^{HG31}

α Deletion of 35B1-35D inferred from genetic evidence. new order: 21 - 28F1|33A2 - 35A4|28F5 - 32F4|37A2 - 38F6|35D2 - 36F11|39A2 - 60.
 Tentative according to Ashburner and Harrington, 1984. synonym: *Df(2L)Sco-rv4*. Left break mapped to the DNA (McGill, Chia, Karp, and Ashburner, 1988, Genetics 119: 641-61).
 β Induced with *T(Y;2)CA13* and *In(2LR)CA14*.
 γ Right break mapped to the DNA (McGill, Chia, Karp, and Ashburner, 1988, Genetics 119: 647-61).
 δ Heterochromatic break in 40 assumed on basis of inability to recover autosynaptic derivatives.

In(2L)Sco^{rv17L}C158^R

cytology: *In(2L)25D3-7;35D1-2^L26D1-2;35B3^R*;
 deficient for 25D7-26D1 and duplicated for 35B3-D1.

origin: Recombinant product between left end of *In(2L)Sco^{rv17}* and right end of *In(2L)C158*.

references: Velissariou and Ashburner, 1980, Chromosoma 77: 13-27.

Ashburner, 1982, Genetics 101: 447-59.

genetics: Deficient for *cl* but not *Sgs-I*; duplicated for *l(2)35Bb - l(2)35Da*. Dominant enhancer of *H*.

In(2L)SD-N3: Inversion (2L) Segregation Distorter

cytology: *In(2L)29E-30A;34A-B*.

origin: X ray induced.

references: Ganetzky, 1977, Genetics 86: 321-55.

In(2L)SD-RA: Inversion (2L)
Segregation Distorter-Ranna

cytology: *In(2L)32A-C;35B-C*.
origin: Spontaneous in a natural population in Ranna, Sicily.
references: Trippa, Loverre, and Cicchetti, 1980, *Genetics* 95: 399-412.
genetics: Carries *Sd-RA* and *fs(2)TLM*. Homozygous viable but sterile in males and females.

In(2L)SD-YT236

cytology: *In(2L)35;39*.
origin: X ray induced in *SD-72* chromosome.
references: Yamazaki and Thompson, 1973, *Jpn. J. Genet.* 48: 217-29.
genetics: *k* value = .8537 - .9929.

In(2L)SD-YT258

cytology: *In(2L)22F;30B*.
origin: X ray induced in *SD-72* chromosome.
references: Yamazaki and Thompson, 1973, *Jpn. J. Genet.* 48: 217-29.
genetics: *k* value = .8700 - .9753.

In(2L)shv^S: Inversion (2L)
shortvein of Segal

references: Segal and Gelbart, 1985, *Genetics* 109: 119-43.
genetics: Associated with *shv*. Lethal over *shv* except for *In(2L)shv^{S11}/shv* which is viable with mutant phenotype.

inversion	cytology	origin
<i>In(2L)shv^{S2}</i>	<i>21B1-3;22F1-2</i>	X ray
<i>In(2L)shv^{S4}</i>	<i>21B6-C1;22F1-2</i>	X ray
<i>In(2L)shv^{S5}</i>	<i>21E1-2;22F1-2</i>	X ray
<i>In(2L)shv^{S11}</i>	<i>22F1-2;31C-D</i>	γ ray
<i>In(2L)shv^{S12}</i>	<i>22F1-2;24A</i>	γ ray
<i>In(2L)shv^{S20}</i>	<i>22B1-2;22F1-2</i>	γ ray
<i>In(2L)shv^{S21}</i>	<i>22A1-3;22F1-2</i>	γ ray
<i>In(2L)shv^{S22}α</i>	<i>22F1-2;35B5-10</i>	γ ray

α Deficient for *l(2)35Cb* (Ashburner).

In(2L)SLNT

cytology: *In(2L)35E-F;36C-D*.
origin: Spontaneous in a natural population in Italy.
references: Sandler, Lindsley, Nicoletti, and Trippa, 1968, *Genetics* 60: 525-58.
genetics: Associated with *mei-S82*.

In(2L)SMG4: Inversion (2L)
Semenova Mglinetz Glotoff

cytology: *In(2L)22E;36F*.
origin: γ ray induced.
references: Semenova, Mglinetz, and Glotoff, 1970, *Genetika (Moscow)* 6(4): 165-69.

In(2L)SMG7

cytology: *In(2L)23E;40D*.
origin: γ ray induced.
references: Semenova, Mglinetz, and Glotoff, 1970, *Genetika (Moscow)* 6(4): 165-69.

In(2L)St: Inversion (2L) Stalker

origin: Naturally occurring inversions.

references: Stalker, 1976, *Genetics* 82: 323-47.

inversions	cytology
<i>In(2L)St-A</i>	<i>26A;33E</i>
<i>In(2L)St-C</i>	<i>27E;31A</i>
<i>In(2L)St-D</i>	<i>24D;26F</i>
<i>In(2L)St-E</i>	<i>31B;34E</i>
<i>In(2L)St-F</i>	<i>25E;30C</i>
<i>In(2L)St-G</i>	<i>30A;34A</i>
<i>In(2L)St-H</i>	<i>26A;29F</i>

In(2L)Su(z)40

cytology: *In(2L)21F;22F-23A*.
references: Gelbart, 1971, Ph.D. Thesis, Univ. of Wisconsin.

In(2L)t: Inversion (2L) t

cytology: *In(2L)22D3-E1;34A8-9* (Bridges and Li in Morgan, Bridges, and Schultz, 1936, Year Book - Carnegie Inst. Washington 35: 292).
origin: Naturally occurring inversion.
discoverer: Bridges, 21a30.
synonym: *In(2L)B* (Oshima and Watanabe, 1965, DIS 40: 88); *In(2L)C* (Mukai, Mettler, and Chigusa, 1970, DIS 45: 77).
references: Sturtevant, 1931, Carnegie Inst. Washington Publ. No. 421: 20.
 Mukai, Watanabe, and Yamaguchi, 1974, *Genetics* 77: 771-93.
 Inoue and Watanabe, 1979, *Jpn. J. Genet.* 54: 69-82.

genetics: Includes *Gpdh* and *Mdh-1*; *Adh* proximal to right breakpoint (Mukai and Voelker, 1977, *Genetics* 86: 175-85).

other information: Found in many natural populations (e.g., Warters, 1944, Texas Univ. Publ. 4445: 129-74; Oshima and Watanabe, 1965, DIS 40: 88; Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57; Pipkin, Franklin-Springer, Law and Lubega, 1976, J. Hered. 67: 258-66; Stalker, 1976, *Genetics* 82: 323-47; Choi, 1977, DIS 52: 88; Mettler, Voelker, and Mukai, 1977, *Genetics* 87: 169-76; Knibb, 1982, *Genetica* 58: 213-21). Inversion in N.C. population formerly thought to be *In(2L)Cy* rather than *In(2L)t* (Mukai, Mettler, and Chigusa, 1971, Proc. Nat. Acad. Sci. USA 68: 1065-69).

In(2L)T

cytology: *In(2L)30F;36D*.
origin: Spontaneous in a natural population.
references: Yang, Kojima, and Kovarik, 1971, DIS 47: 71-72.
 Yang and Kojima, 1972, Univ. Tex. Publ. 7213: 229-36.
 Langley, Tobari, and Kojima, 1974, *Genetics* 78: 921-36.
 Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

In(2L)T4

cytology: *In(2L)30F;36D*.
references: Lyttle.

In(2L)TE35A: Inversion (2L)
Transposing Element

origin: γ ray induced.

synonym: *In(2L)TE146Z*.

inversion	cytology	discov ^α	ref ^β	genetics
<i>In(2L)TE35A-1</i>	35B;39A-B	1	1	
<i>In(2L)TE35A-3</i>	35B1;40	1	1	
<i>In(2L)TE35A-8</i>	35B1-2;35C4	2	1	
<i>In(2L)TE35A-9</i>	35B1-2;40	1	1	variegates for <i>w</i> ⁺
<i>In(2L)TE35A-11</i>	35B1-2;40	2	1	variegates for <i>w</i> ⁺
<i>In(2L)TE35A-13</i>	35B1-2;40 + Df	1	1, 2	<i>pu</i> ⁻ - <i>l(2)35Dg</i> ⁻
<i>In(2L)TE35A-16</i>	35B1-2;40	2		
<i>In(2L)TE35A-27</i>	35B1-2;38C1-2	2	1	
<i>In(2L)TE35A-53</i>	35B1-2;40	4	1	
<i>In(2L)TE35A-55</i>	35B1-2;36C3-11	4	1	
<i>In(2L)TE35A-56</i>	35B1-2;40	4	1	
<i>In(2L)TE35A-63</i>	35B1-2;40			
<i>In(2L)TE35A-210</i>	28B12-D1-2;35B	3	1	
<i>In(2L)TE35A-220</i>	35B;39D	3	1	
<i>In(2L)TE35A-225</i>	35B;40	3	1	
<i>In(2L)TE35A-227</i>	40;72B-C + <i>In(2L)35B;40</i> + <i>In(3R)81;88B</i>		1	

^α 1 = Durrant; 2 = Roote; 3 = Samkang; 4 = Wilkins.

^β 1 = Ashburner; 2 = Gubb, Roote, Harrington, Durrant, Shelton, and Ashburner, 1985, Chromosoma 92: 116-23.

In(2L)TE35A-210^LC163.41^R

cytology: *Df(2L)35B1-3;35E1-2 + Dp(2;2)27D1-2;28B3-D1*.

origin: Recombinant between *In(2L)TE35A-210* and *In(2L)C163.41*.

discoverer: Roote.

synonym: *In(2L)TE146(Z)GR210^LC163.41^R*.

In(2L)TE35BC

origin: γ ray induced in *TE35BC*.

discoverer: Shelton.

synonym: *In(2L)TE36R*.

references: Ashburner.

genetics: Variegates for *w*⁺.

inversion	cytology
<i>In(2L)TE35BC-4</i>	26A;35B;40-41
<i>In(2L)TE35BC-7</i>	35B;40-41
<i>In(2L)TE35BC-10</i>	35B;40-41

In(2L)TE36R: see *In(2L)TE35BC*

In(2L)TE146Z: see *In(2L)TE35A*

In(2L)TE146(Z)GR210^LC163.41^R:
see *In(2L)TE35A-210^LC163.41^R*

In(2L)Tg: Inversion (2L) Tegula

cytology: *In(2L)21C;22E-F*.

origin: X ray induced.

discoverer: E. B. Lewis, 1962.

references: Mora, 1963, DIS 38: 32.

Spencer, Hoffmann, and Gelbart, 1982, Cell 29: 451-61.

genetics: Associated with *Tg*. *In(2L)Tg/dpp* same phenotype as *In(2L)Tg/+*.

In(2L)TW47

cytology: *In(2L)37A2-B1;38A6-C1*.

origin: X ray induced in chromosome carrying *Tf1*.

references: Wright, Hodgetts, and Sherald, 1976, Genetics 84: 267-85.

In(2L)V11-3

cytology: *In(2L)22A;40E*.

origin: X ray induced.

references: Valencia, 1970, DIS 45: 37-38.

genetics: Homozygous viable and fertile.

In(2L)V12

cytology: *In(2L)21F;29B-C*.

origin: X ray induced simultaneously with *T(2;3)V12-1-6*.

references: Valencia, 1970, DIS 45: 37-38.

genetics: Lethal.

In(2L)VV2: Inversion (2L) V. Velissariou

cytology: *In(2L)22B;27A*.

origin: X ray induced.

discoverer: Velissariou.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.

In(2L)VV3

cytology: *In(2L)25E1-E2;30D*.

origin: X ray induced.

discoverer: Velissariou.

In(2L)W

cytology: *In(2L)28C;32C*.

origin: Naturally occurring inversion.

references: Inoue and Watanabe, 1979, Jpn. J. Genet. 54: 83-96.

Inoue, Watanabe, and Watanabe, 1984, Evolution 38: 753-65.

In(2L)wg^P: Inversion (2L) wingless

cytology: *In(2L)28A1-3;32E-F* (Lefevre, 1976).

origin: X ray induced.

synonym: *In(2L)wg^P*.

references: Baker, 1987, EMBO J. 6: 1765-77.

genetics: Mutant for *wg*. Pupal lethal.

molecular biology: Breakpoint at 28A1-3 mapped to the region between +16.4 to 17.1 kb (0 is *Hind* III site).

In(2L)Z4: Inversion (2L) Zacharopoulou

cytology: *In(2L)26A;33E*.

origin: Spontaneous in a natural population of Patras, Greece.

references: Zacharopoulou, 1976, DIS 51: 110.

In(2L)ZP: Inversion (2L)

Zacharopoulou Pelecanos

origin: Naturally occurring inversions in Greece.

references: Zacharopoulou and Pelecanos, 1980, Genetica 54: 105-11.

inversion	cytology
<i>In(2L)ZP2</i>	30C;34D
<i>In(2L)ZP3</i>	26A;30F
<i>In(2L)ZP4</i>	30F;39A
<i>In(2L)ZP5</i>	23A;29E
<i>In(2L)ZP6</i>	33A;37D

****In(2LR)40d***

cytology: *In(2LR)26D;41A-B*.

origin: X ray induced.

discoverer: T. Hinton and Atwood, 49d.

references: Demerec, Kaufmann, Sutton, and Fano, 1941, Year Book - Carnegie Inst. Washington 40: 225-34.

Hinton, 1942, DIS 16: 48.

Hinton, 1955, Genetics 40: 224-34.

genetics: Variegated for a dominant dark eye color and irregular facets; more extreme at low temperature. Homozygous lethal. Certain stocks containing *In(2LR)40d* fail to grow on media lacking RNA or adenine (Hinton, Ellis, and Noyes, 1951, Proc. Nat. Acad. Sci. USA 37: 293-99). This was true at pH 7.0 but not at pH 5.0 (Ellis, 1959, Physiol. Zool. 32: 29-39).

In(2LR)102

cytology: *In(2LR)26A;51C + In(2R)41;57A*.

new order:

21 – 26A|51C – 41|57A – 51C|26A – 41|57A – 60.

origin: X ray induced in *ds^w sp²*.

discoverer: R. F. Grell, 53k.

references: Kramer and Lewis, 1956, J. Heredity 47: 132-36.

Grell and Lewis, 1956, DIS 30: 71.

other information: Useful as a balancer.

In(2LR)429

discoverer: Gelbart.

inversion	cytology
<i>In(2LR)429.20</i>	22E1-2;41A-C
<i>In(2LR)429.31</i> ^α	21D;26F;41;57C
<i>In(2LR)429.41</i>	22D1-2;41A
<i>In(2LR)429.49</i>	28A;41A
<i>In(2LR)429.75</i>	29B1-2;41
<i>In(2LR)432.3</i>	23C;41A
<i>In(2LR)432.6</i>	22F3-23A1;41A
<i>In(2LR)432.41</i>	23A1-2;41A
<i>In(2LR)434.14</i>	26A1-2;41
<i>In(2LR)434.37</i>	see T(2;3)434.37
<i>In(2LR)434.38</i>	33A;56D
<i>In(2LR)434.59</i>	23E1-2;41
<i>In(2LR)434.60</i>	34D;41A
<i>In(2LR)434.93</i>	27D1-2;41A-B
<i>In(2LR)434.96</i>	24E;41
<i>In(2LR)434.125</i>	22A;41A-C + T(2;3)35F;84E9

^α new order: 21A – 21D|41 – 57C|26F – 21D|41 – 26F|57C – 60F.

****In(2LR)a^{M60}*: Inversion (2LR) arc of Meyer**

cytology: Breakpoints unknown.

origin: X ray induced.

discoverer: Meyer, 60f.

references: 1963, DIS 37: 50.

genetics: Associated with *a^{M60}*.

In(2LR)A379*: see *Df(2L)A379

***In(2LR)ade2^B*: Inversion (2LR) adenosine 2**

cytology: *In(2LR)26B;40–41;57B–C*.

new order: 21-26|40-26|57-41|57-60 (tentative).

origin: γ ray induced.

synonym: *In(2LR)ST*.

references: Tiong, Keizer, Nash, Bleskan, and Patterson, 1989, Biochem. Genet. 27: 333-48.

genetics: Mutant for *ade2^B*.

In(2LR)al^B

cytology: *In(2LR)21C1-2;41C-D*.

origin: X ray induced.

references: Korochkina and Golubovsky, 1978, DIS 53: 197-200.

Golubovsky, Kulakov, and Korochkina, 1978, Genetika

(Moscow) 14(2): 294-305.

genetics: Homozygous lethal; associated with *al^B*. Heterozygotes with *Df(2)al* survive very poorly; escapers have reduced arista, very broad thoraces, arched wings, incomplete veins, and large eyes (Korochkina and Golubovsky, 1978).

****In(2LR)al^{M60}*: Inversion (2LR) aristaless of Meyer**

origin: X ray induced.

discoverer: Meyer, 60f.

references: 1963, DIS 37: 50.

genetics: Mutant for *al*. Homozygous lethal. Inversion inferred from crossing over inhibition in *2L* and *2R*.

In(2LR)al^V

cytology: *In(2LR)21B-C1;41*.

origin: X ray induced.

discoverer: E. B. Lewis, 1940.

references: 1945, Genetics 30: 137-66.

genetics: Variegated for *al*. Variegation suppressed by extra Y so that *al^V/al* becomes wild type (Korochkina and Golubovsky, 1978, DIS 53: 197). Homozygous lethal.

***In(2LR)AL*: Inversion (2LR) Ashburner Lemeunier**

origin: Naturally occurring inversions.

references: Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

inversion	cytology
<i>In(2LR)AL4</i>	31E;41
<i>In(2LR)AL5</i>	32E;43E
<i>In(2LR)AL6</i>	36A;49D

***In(2LR)b81a*: see *Tp(2;2)b81a*.**

In(2LR)B107*: see *T(Y;2)B107

In(2LR)B185

cytology: *In(2LR)34A;51A;53C–D*.

origin: X ray induced.

references: Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84.

genetics: Homozygous lethal. Associated with, but separable from, *T(Y;2)B185*.

In(2LR)BEL

cytology: *In(2LR)36D;46F + In(2LR)30C;47E*.

origin: Spontaneous in a natural population in Belinga, Gabon.

discoverer: David.

references: Ashburner, 1972, DIS 49: 34.

Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

genetics: Homozygous lethal.

***In(2LR)bw^R*: Inversion (2LR) brown-Rearranged**

origin: X ray induced.

discoverer: Slatis.

references: 1955, Genetics 40: 5-23.

inversion	cytology	associated with
<i>In(2LR)bw^{R3}</i> ^α	40F;51F;55E;57E;58D8-9	<i>bw^{R8}</i>

inversion	cytology	associated with
* <i>In(2LR)bw</i> ^{R20}	40D;59D5-6	<i>bw</i> ^{R20}
* <i>In(2LR)bw</i> ^{R55} β	24D-E1;42E + <i>In(2)40F-41A;59D4-5</i>	<i>bw</i> ^{R55}
α	new order: 21 – 40F 55E – 51F 57E – 55E 57E – 59D8 51F – 40F 59D9 – 60.	
β	new order: 21 – 24D 42E – 41A 59D4 – 42E 24E1 – 40F 59D5 – 60.	

***In(2LR)bw*^{V1}: Inversion (2LR) brown-Variegated**

cytology: *In(2LR)21C8-D1;60D1-2 + In(2LR)40F;59D4-E1.*

new order:

21A – 21C8|60D1 – 59E1|40F – 59D4|40F – 21D1|60D2 – 60F.

origin: X ray induced.

discoverer: Muller, 1929.

synonym: *Ins(2LR)Pm*: *Inversion (2LR) Plum*.

references: 1930, J. Genet. 22: 299-334 (fig.).

Glass, 1934, J. Genet. 28: 69-112 (fig.).

1934, Am. Naturalist 68: 107-14.

Bridges, 1937, Cytologia (Tokyo), Fujii Jub., Vol. 2: 745-55.

Holm and Chovnick, 1976, Genetics 81: 293-311.

Craymer, 1981, DIS 95: 75-97.

Genetics 81: 293-311.

genetics: Mutant for *ds* which is included as *ds*^{33k} in the *In(2LR)21C8-D1;60D1-2* component (Craymer, 1981); variegated for *lt*, *bw*, *mi*, and *abb*. *al*⁴ arose after origin. Expression of *bw*^{V1} suppressed by extra Y (Holm and Chovnick, 1976) or extra chromosome 4 (Lindsley). Double crossovers in 2L but not 2R fairly frequent. Single exchange in region 21D1-40F of 2L between *In(2LR)bw*^{V1} and a normal sequence produces a recombinant carrying left end of normal chromosome 2, which is duplicated for 21A1-C8 and deficient for 60D2-F5. Heterozygote for this recombinant poorly viable, fertile, brown-Variegated, Minute, and dwarf with pebbled arc wings; deficient for locus of *M(2)60E*. Reciprocal recombinant deficient for 21A1-C8 and duplicated for 60D2-F5; heterozygote poorly viable, fertile, *bw*⁺, Minute and giant; deficient for *al* and *M(2)21C*. Inversion heterozygote shows interchromosomal effect (Valentin, 1972, Hereditas 72: 243-54).

***In(2LR)bw*^{V29I}**

origin: X ray induced.

discoverer: Van Atta.

references: 1932, Genetics 17: 637-59.

genetics: Variegated for *bw*. Breaks most probably just to the left of centromere and near *bw*.

***In(2LR)bw*^{V30k1}**

origin: X ray induced.

discoverer: Van Atta.

references: 1932, Genetics 17: 637-59.

genetics: Variegated for *bw*. Breaks most likely just to the left of centromere and near *bw*.

***In(2LR)bw*^{V32g}**

cytology: *In(2LR)40F;59E.*

origin: X ray induced.

discoverer: Dobzhansky, 32g6.

synonym: *In(2LR)Pm*²: *Inversion (2LR) Plum-2*.

references: Schultz and Dobzhansky, 1934, Genetics

19: 344-64.

Schultz, 1936, Proc. Nat. Acad. Sci. USA 22: 27-33.

Hiraizumi, 1969, Jpn. J. Genet. 44: 97-103.

genetics: Variegated for *bw*; variegation suppressed by extra Y (Hiraizumi, 1969). Homozygous lethal.

In(2LR)C251: Inversion (2LR) Crossover suppressor

cytology: *In(2LR)36F;57B.*

origin: X ray induced.

discoverer: Roberts, 1965.

references: 1970, Genetics 65: 429-48.

genetics: Homozygous viable. Recombination reduced in 2R.

In(2LR)CA: Inversion (2LR) Cambridge

discoverer: Ashburner.

inversion	cytology	origin	associated with	ref ^α
<i>In(2LR)CA10</i>	21A;58B	γ ray	<i>Df(2L)TE35A-8</i>	2
<i>In(2LR)CA14</i>	23A;46E	X ray	<i>In(2L)Sco</i> ^{rv5}	1
<i>In(2LR)CA22</i>	40;57B	γ ray	<i>TE35BC-32</i>	3
<i>In(2LR)CA29</i>	36B;57A5-10	X ray	<i>T(Y;2)A38</i>	4

^α 1 = Ashburner; 2 = Gubb, Roote, Harrington, McGill, Durrant, Shelton, and Ashburner, 1985, Chromosoma 92: 116-23. 3 = Gubb, Shelton, Roote, McGill, and Ashburner, 1984, Chromosoma 91: 54-64; 4 = Lyttle.

In(2LR)D

cytology: *In(2LR)36F;49B.*

discoverer: Oshima and Watanabe.

references: 1965, DIS 40: 88.

Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

In(2LR)D20^Lnoc^{4R}

cytology: *In(2LR)34E4-F2;35B1-2;41A*; Deficient for 34F2-35B1.

origin: Recombinant via autosynaptic elements between *LS(2)D20* and *DS(2)noc*⁴.

discoverer: Gubb.

genetics: Deficient for *rk-noc*.

In(2LR)DA3

cytology: *In(2LR)26D;53E.*

origin: X ray induced in *SD-NH2*.

discoverer: Lyttle.

In(2LR)dp: see T(2;3)dp

In(2LR)dpp: Inversion (2LR) decapentaplegic (W. M. Gelbart)

genetics: Mutant for *dpp*.

allele	class	origin	cytology	ref ^α
<i>In(2LR)dpp</i> ²³	d-III	X-ray	<i>In(2LR)22F1-2;41A</i>	1,3
<i>In(2LR)dpp</i> ²⁶	d-V	X-ray	<i>In(2LR)22F1-2;47A1-4</i>	1,3
<i>In(2LR)dpp</i> ³¹	d-III	EMS	<i>In(2LR)22F1-3;41C-D</i>	1
<i>In(2LR)dpp</i> ³⁵	d-V	X-ray	<i>In(2LR)22F1-2;42A2-8</i>	1,2
<i>In(2LR)dpp</i> ³⁶	d-III	X-ray	<i>In(2LR)22F1-3;35E</i>	1
<i>In(2LR)dpp</i> ⁶⁷	d-V	γ-ray	<i>In(2LR)21F;22F</i>	1
<i>In(2LR)dpp</i> ⁶⁸	d-III	γ-ray	<i>In(2LR)22F1-2;23D;51D</i>	1
<i>In(2LR)dpp</i> ⁷⁵	d-V	γ-ray	<i>In(2LR)22F1-2;58D</i>	1
<i>In(2LR)dpp</i> ^{H45}	Hin-Df	γ-ray	<i>In(2LR)22F1-3;52F</i>	2
<i>In(2LR)dpp</i> ^{s15}	shv-lc	γ-ray	<i>In(2LR)22F1-2;59B</i>	1
<i>In(2LR)dpp</i> ^{s17}	shv-lc	γ-ray	<i>In(2LR)22F1-2;41A</i>	1
<i>In(2LR)dpp</i> ^{t24}	t	X-ray	<i>In(2LR)22F1-2;58B</i>	1

^α 1 = Gelbart; 2 = Irish and Gelbart, 1987, Genes Dev. 1: 869-79; 3 = Spencer, Hoffmann, and Gelbart, 1982, Cell 28: 451-61.

**In(2LR)ds^{33k}*: see *In(2LR)bw^{V1}*

***In(2LR)DTD: Inversion (2LR)*
Disrupter of Transvection
at Decapentaplegic**

origin: X ray induced with exception of γ -ray-induced *In(2LR)DTD22.18* and *In(2LR)DTD22.59*

references: Gelbart, 1982, Proc. Nat. Acad. Sci. USA 79: 2636-40; Gelbart (not published).

genetics: Disrupts transvection at *dpp*.

inversion	cytology	synonym
<i>In(2LR)DTD4</i>	32E1-2;41A-C	<i>In(2LR)429.20</i>
<i>In(2LR)DTD8</i>	23D1-2;41A	<i>In(2LR)429.41</i>
<i>In(2LR)DTD11</i>	28A;41A	<i>In(2LR)429.49</i>
<i>In(2LR)DTD22.18</i>	35B;41;42A;58F	
<i>In(2LR)DTD22.59</i>	35C;46A	
<i>In(2LR)DTD24</i>	26C1-2;41A	
<i>In(2LR)DTD25.59</i>	35B1-3;48C6-8	
<i>In(2LR)DTD32</i>	26A1-2;41	<i>In(2LR)434.14</i>
<i>In(2LR)DTD42</i>	23E1-2;41	<i>In(2LR)434.59</i>
<i>In(2LR)DTD43</i>	34D;41A	<i>In(2LR)434.60</i>
<i>In(2LR)DTD51</i>	27D1-2;41A-B	<i>In(2LR)434.93</i>
<i>In(2LR)DTD52</i>	24E;41	<i>In(2LR)434.96</i>
<i>In(2LR)DTD55</i>	32A1-2;41A	
<i>In(2LR)DTD86</i>	33B1-2;41A	
<i>In(2LR)DTD99</i>	40F;53F	
<i>In(2LR)DTD109</i>	25E;41A	
<i>In(2LR)DTD111</i>	29E;41A-C	
<i>In(2LR)DTD114</i>	26A;45D	
<i>In(2LR)DTD116</i>	26A;41A	
<i>In(2LR)DTD124</i>	24D1-2;41A	
<i>In(2LR)DTD125</i>	31E;41A	
<i>In(2LR)DTD128</i>	35C;46A	

In(2LR)E1

cytology: *In(2LR)34A;58F*.

discoverer: Ising.

In(2LR)el⁶

cytology: *In(2LR)35B1-3;57C3-9* (Ashburner).

origin: Induced by ethyl methanesulfonate.

discoverer: Angel.

genetics: Associated with *el⁶*.

In(2LR)Eni: Inversion (2R) Enigma

cytology: Pericentric inversion.

origin: Recombination between *Dp(2;2)Eni/F(2R)* and standard strain.

references: Fitz-Earle, 1979, Genetics 91: s34.

genetics: *Dp(2;2)Eni/F(2R)* and additional free arms produced again through recombination.

In(2LR)f6: Inversions (2LR) from free arms 6

cytology: *In(2LR)39D3-E1;48F6-49A1*.

origin: X ray induced as a translocation between the 2^{PY}D element of *T(Y;2)B238 = T(Y;2)YS;41* and *F(2R)VH1*. Subsequently reconstituted as *In(2LR)f6*.

references: Craymer, 1984, DIS 60: 234-36.

In(2LR)Gla: Inversion (2LR) Glazed

cytology: *In(2LR)27D;51E* superimposed on *In(2L)22D3-E1;34A8-9* (Woodruff and Ashburner, 1979, Genetics 92: 117-32).

new order:

21 – 22D3|34A8 – 27D|51E – 34A9|22E1 – 27D|51E – 60.

origin: X ray induced in chromosome containing *In(2L)t = In(2L)22D3-E1;34A8-9*.

genetics: Associated with *Gla*. Effective crossover suppressor; no single or double crossovers recovered to

the left of *c* (Alexander, 1952, Texas Univ. Publ. 5204: 219-26). Orange Malpighian tubules in larvae and adults heterozygous with *cn* (Ashburner).

In(2LR)JHI

origin: Accumulated on second chromosomes during repeated backcrossing. These second chromosomes come from a lethal- carrying chromosome *JH* derived in 1967 from a wild-type cage population.

references: Yamaguchi and Mukai, 1974, Genetics 78: 1209-21.

inversion	cytology
<i>In(2LR)JHI1</i>	22F;60A
<i>In(2LR)JHI2</i>	24D;56F
<i>In(2LR)JHI3</i>	26A;56D
<i>In(2LR)JHI4</i>	26A;59D
<i>In(2LR)JHI5</i>	29B;50B
<i>In(2LR)JHI6</i>	33B;60B

In(2LR)KB

origin: Spontaneous in natural populations in Korea.

references: Paik, 1986, DIS 63: 167.

In(2LR)L^{rv5}: Inversion (2LR) Lobe-revertant

cytology: *In(2LR)26F;50F-51A*.

origin: X ray induced.

synonym: *In(2LR)L^{+R5}*.

references: Baker and Ridge, 1980, Genetics 94: 383-423.

genetics: Revertant of *L*.

In(2LR)L2

cytology: *In(2LR)36C;51D*.

discoverer: Ising.

In(2LR)It^m: Inversion (2LR) light-mottled

origin: X ray induced.

discoverer: Hessler, 1957.

references: 1958, Genetics 43: 395-403.

genetics: Variegated for *It*.

inversion	cytology
<i>In(2LR)It^{m3}</i>	see <i>T(2;4)It^{m3}</i>
* <i>In(2LR)It^{m9}</i>	40B-F;56E
<i>In(2LR)It^{m12}</i>	40B-F;60D
* <i>In(2LR)It^{m22}</i>	40B-F;59D
* <i>In(2LR)It^{m25}</i>	40B-F;57C-D
* <i>In(2LR)It^{m33}</i>	40B-F;58E.

In(2LR)It^{G10}: Inversion (2LR) light

cytology: *In(2LR)40;59F3*.

origin: X ray induced in *cn bw* chromosome.

discoverer: Craymer.

references: Brittnacher and Ganetzky, 1989, Genetics 121: 739-50.

genetics: Variegation for *It*.

In(2LR)It^{G16}

cytology: *In(2LR)40;60E4*.

origin: X ray induced in *cn bw* chromosome.

discoverer: Craymer.

references: Brittnacher and Ganetzky, 1989, Genetics 121: 739-50.

genetics: Variegation for *It*. Mglinetz, 1971, Genetika (Moscow) 7(8): 108-14.

In(2LR)M9

cytology: *In(2LR)37B;38F;47F*; deficient for 37B-38F.
origin: X ray induced.
references: Nakamura, 1973, Ph.D. Thesis, Paris - Sud.
 Nakamura, 1978, Mol. Gen. Genet. 159: 285-92.
genetics: Homozygous lethal.

In(2LR)Mg: Inversion (2LR) Mglinetz

inversion	cytology	origin α	ref β
<i>In(2R)Mg3</i>	40;57C	γ ray	2
<i>In(2LR)Mg4</i>	37C;55E	1	2
<i>In(2LR)Mg97</i>	33E;46B	1	1
<i>In(2LR)Mg99</i>	35B;45A	2	1
<i>In(2LR)Mg100</i>	36C;46C	2	1
<i>In(2LR)Mg101</i>	39E;60E	2	1
<i>In(2R)Mg104</i>	40;48D	γ ray	1
<i>In(2R)Mg110</i>	40;57A	32 P feeding	1
<i>In(2R)Mg111</i>	40;56A	32 P feeding	1
<i>In(2LR)Mg199</i>	26B;42E	2	1

α 1 = γ ray induced; 2 = 32 P feeding.
 β 1 = Mglinetz, 1968, Genetika (Moscow) 4(8): 81-86; 2 = Mglinetz, 1971, Genetika (Moscow) 7(8): 108-14.

In(2LR)net¹⁸: Inversion (2LR) net

cytology: *In(2LR)21B3-4;42C-D1*; small deficiency at distal end.
new order:
 21A - 21B3|42C - 21B4|42D1 - 60.
origin: X ray induced along with *T(2;3)net¹⁸* = *T(2;3)27E-F;65A*.
references: Golubovsky, Kulakov, and Korochkina, 1978, Genetika 14(2): 294-305.
 Korochkina and Golubovsky, 1978, DIS 53: 197-200.
genetics: Deficient for *net* and *l(2L)gl* but not *al*. Homozygous lethal and Minute.

In(2LR)noc⁴

cytology: *In(2LR)35B1-2;41*.
origin: γ ray induced.
discoverer: Harrington.
references: Ashburner, Tsubota, and Woodruff, 1982, Genetics 102: 401-20.
 Ashburner, Aaron, and Tsubota, 1982, Genetics 102: 421-35.
 Chia, Karp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706.
 Gubb, Roote, Harrington, McGill, Durrant, Shelton, and Ashburner, 1985, Chromosoma 92: 116-23.
genetics: Associated with *noc⁴*. Autosynaptic derivatives [*LS(2)noc⁴*; *DS(2)noc⁴*] recoverable (Gubb).

In(2LR)noc^{4L} Sco^{rv9R}

cytology: *In(2LR)35B1-2;35D1-2;41*; deficient for 35B2-D1.
origin: Recombinant between autosynaptics, *LS(2)noc⁴* and *DS(2)Sco^{rv9}*.
discoverer: Gubb.
synonym: *Df(2L)noc^{4L} Sco^{+R9R}*.
genetics: Deficient for *noc - l(2)35Da*. Presumably aneuploid for interval between region 41 breakpoints of parental inversions.

In(2LR)noc⁷

cytology: *In(2LR)35A1-4;40;46B1-2;48C6 + T(Y;2)60D7-F*.
new order:
 21A - 35A1|46B2 - 48C6|40 - 46B1|

35A4 - 40|48C6 - 60D7|Y;
 Y|60D7 - 60F.

origin: γ ray induced.
discoverer: Harrington.
references: Ashburner, Aaron, and Tsubota, 1982, Genetics 102: 421-35.
 Chia, Karp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706.
 Gubb, Roote, Harrington, McGill, Durrant, Shelton, and Ashburner, 1985, Chromosoma 92: 116-23.
genetics: Associated with *noc⁷*.

In(2LR)O: Inversion (2LR) of Oster

cytology: *In(2LR)30E-F;50C10-D1* superimposed on *In(2L)22D1-2;33F5-34A1 + In(2R)42A2-3;58A4-B1* (Lindsley).
new order:
 20 - 22D1|33F5 - 30F|50D1 - 58A4|
 42A2 - 34A1|22D2 - 30E|50C10 - 42A3|
 58B1 - 60.
origin: X ray induced in *In(2L)Cy + In(2R)Cy, Cy dp^{lv} pr cn²*.

references: Oster, 1956, DIS 30: 145.
molecular biology: 50C-D breakpoint distal to *mam*; between coordinates +25 and +29 on the chromosome walk including *mam* (Yedvobnick, Smoller, Young, and Mills, 1988, Genetics 118: 483-97).
other information: Used as a balancer for chromosome 2, described as *CyO* in the section on balancers.

In(2LR)Pm: see *In(2LR)bw^{V1}*

In(2LR)Pm²: see *In(2LR)bw^{V32g}*

In(2LR)Pu^{Ly}: Inversion (2LR) Punch of Lytle

cytology: *In(2LR)40B-F;57C4-6*.
origin: γ ray induced in *SD-Roma*.
discoverer: Lytle.
synonym: *In(2LR)R-3*.
references: Mackay and O'Donnell, 1983, Genetics 105: 35-53.
 O'Donnell, Boswell, Reynolds, and Mackay, 1989, Genetics 121: 273-80.
genetics: Associated with *lt* at 40B-F and *Pu^{Ly}* at 57B-C. Dominant, variegated eye color. Homozygous lethal.

In(2LR)px^{52g}: Inversion (2LR) plexus

cytology: *In(2LR)30A;58E*.
origin: X ray induced in *cn crs*.
discoverer: Iyengar and Meyer, 52g.
references: 1956, DIS 30: 73.
 Meyer, 1956, DIS 30: 81.
 1958, DIS 32: 83.
 Craymer, 1980, DIS 55: 197-200.
 1981, Genetics 99: 75-97.
genetics: Mutant for *px*. Pericentric inversion with breakpoints between *dp* and *b* and between *px* and *sp*. Homozygous female is fertile but male is sterile. Male genitalia rotated. Sterility factor not allelic to *ab* and not covered by duplication in *bw^{+Y}* (as is *crs*, the male sterility factor present in original chromosome).

In(2LR)Px⁴: Inversion (2LR) Plexate

cytology: *In(2LR)22A3-B1;60B-C^L;21C8-D1;60D1-2^R + In(2R)42A2-3;58A4-B1*; deficient for 60B-D1 and duplicated for 21D1-22A3.

new order:

21A – 22A3|60B – 58B1|42A3 – 58A4|
42A2 – 21D1|60D2 – 60F.

origin: Synthetic. This chromosome is a recurrent product of recombination in region 33F-40F between *In(2LR)21C8-D1;60D1-2* from *In(2LR)bw^{VI}* [*In(2LR)21C8-D1;60D1-2* + *In(2LR)40F;59D4-E1*] and *In(2LR)22A3-B1;60B-C* from *SM1* [*In(2L)Cy* = *In(2L)22D1-2;33F5-34A1* + *In(2LR)22A3-B1;60B-C* + *In(2R)Cy* = *In(2R)42A2-3;58A4-B1*]. Recombinant carries tip of 2L and *In(2R)Cy* from *SM1* and tip of 2R and most of 2L from *In(2LR)bw^{VI}*. The reciprocal recombinant is *In(2LR)S^{56f}*.

discoverer: Thompson.

references: Burdick, 1956, DIS 30: 69.

genetics: Deficient for *bs*, *ba*, *Pin*, *Px*, and probably *sp*; duplicated for *S*.

In(2LR)RBR

origin: Spontaneous [accumulated on second chromosome lines during repeated backcrossing of males heterozygous for each second chromosome line over *In(2LR)bw^{VI}* to *In(2LR)bw^{VI}/SM1* females].

references: Yamaguchi, Cardellino, and Mukai, 1976, Genetics 83: 409-22.

inversion	cytology
<i>In(2LR)RBR7</i>	34A;50B
<i>In(2LR)RBR25^{αβ}</i>	26E;29B;35D;46C;49D;56D
<i>In(2LR)RBR36</i>	30E;42C
<i>In(2LR)RBR49^{αγ}</i>	22A;26A + 26E;34A;57C;58E
<i>In(2LR)RBR63</i>	38A;57B
<i>In(2LR)RBR78</i>	28C;59B + 32E;60E
<i>In(2LR)RBR91</i>	30B;45E
<i>In(2LR)RBR100</i>	33E;50B
<i>In(2LR)RBR104</i>	22A;59C
<i>In(2LR)RBR116</i>	31C;57E
<i>In(2LR)RBR120</i>	35B;58E
<i>In(2LR)RBR144</i>	29B;42C
<i>In(2LR)RBR146</i>	38F;42C
<i>In(2LR)RBR147</i>	35D;57B

^α Overlapping rearrangements.

^β New order unknown.

^γ new order: 21 – 22A|26A – 22A|26A – 26E|58E – 57C|
(26E – 34A)|57C – 34A|58E – 60.

In(2LR)Rev: Inversion (2LR) Revolute

cytology: *In(2LR)40F;52D10-E1* (Bridges and Li in Morgan, Bridges, and Schultz, 1936, Year Book - Carnegie Inst. Washington 35: 293).

origin: X ray induced.

discoverer: Dobzhansky, 31b5.

references: Valentin, 1972, Hereditas 72: 243-54. 1978, Hereditas 89: 263-64.

genetics: Variegated for *It* and *Rev*. Heterozygotes show no interchromosomal effect on distal X recombination (Valentin, 1978).

In(2LR)Rev^B: Inversion (2LR) Revolute of Bridges

cytology: *In(2LR)40;52D5*.

origin: Spontaneous.

discoverer: Bridges, 36e22.

synonym: *In(2LR)Rvd*.

references: Morgan, Bridges, and Schultz, 1936, Year Book - Carnegie Inst. Washington 35: 293. Valentin, 1978, Hereditas 89: 263-64. Wargent, 1972, DIS 49: 50.

Wargent and Hartman-Goldstein, 1974, Heredity 33: 317-26.

genetics: Variegated for *Rev* and *It* (Wargent, 1972, DIS 49: 50-51). Variegation enhanced by low temperature; suppressed by extra Y chromosome (Wargent). Heterozygotes show no interchromosomal effect on distal X recombination (Valentin, 1978).

In(2LR)S^{56f}: Inversion (2LR) Star

cytology: *In(2L)22D1-2;33F5-34A1* + *In(2LR)21C8-D1;60D1-2^L22A3-B1;60B-C^R* + *In(2LR)40F;59D4-E1*; deficient for 21D1-22A3 and duplicated for 60B-D1.

new order:

21A – 21C8|60D1 – 50E1|40F – 59D4|40F – 34A1|
22D1 – 33F5|22D1 – 22B1|60C – 60F.

origin: Synthetic. This chromosome is a recurrent product of recombination in region 33F-40F between *In(2LR)21C8-D1;60D1-2* from *In(2LR)bw^{VI}* [*In(2LR)21C8-D1;60D1-2* + *In(2LR)40F;59D4-E1*] and *In(2LR)22A3-B1;60B-C* from *SM1* [*In(2L)Cy* = *In(2L)22D1-2;33F5-34A1* + *In(2LR)22A3-B1;60B-C* + *In(2R)Cy* = *In(2R)42A2-3;58A4-B1*]. Recombinant carries the tip of 2L and *In(2LR)40F;59D4-E1* from *In(2LR)bw^{VI}* and the tip of 2R and *In(2L)Cy* from *SM1*. Reciprocal recombinant is *In(2LR)Px⁴*.

discoverer: Thompson.

references: Burdick, 1956, DIS 30: 69.

genetics: Deficient for *S*; duplicated for *Px*.

In(2LR)S³²⁵

cytology: *In(2LR)21D2-3;21D3-E2;21E2-3;41F*.

new order:

21A – 21E2|41 – 21D3|41 – 60.
Tentative.

origin: X ray induced in *Dp(2;2)S* = *Dp(2;2)21D2-3;21E2-3*.

discoverer: E. B. Lewis.

genetics: Break in 2L either in or between duplicated segments of *Dp(2;2)S*. *In(2LR)S³²⁵/ds^W* shows a *ds⁻/ds^W* phenotype (Craymer, 1981, Genetics 99: 75-97).

****In(2LR)S^K: Inversion (2LR) Star of Krivshenko***

cytology: Breakpoints near ends of 2L and 2R.

discoverer: Krivshenko.

references: 1936, DIS 5: 8.

genetics: Associated with *S^K*.

In(2LR)S^{Ly}: Inversion (2LR) Star of Lyttle

cytology: *In(2LR)21E-F;41*.

origin: X ray induced in *SD-Roma*.

discoverer: Lyttle.

genetics: Associated with *S^{Ly}* which is located at 21E-F and shows an *S* phenotype but is somewhat more variable than *S*.

In(2LR)Sco^{rv1}: Inversion (2LR) Sco-revertant

cytology: *In(2LR)35D1-2;44C4-5* (Ashburner).

origin: X ray induced in *Tp(2;2)Sco*.

synonym: *Sco^{+R1}*; *l(2)br29^{Sco+R1}*.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.

Craymer, 1981, Genetics 99: 75-97.

Ashburner, Detwiler, Tsubota, and Woodruff, 1983, Genetics 104: 405-31.

Ashburner and Harrington, 1984, Chromosoma 89: 329-37.

Gubb, Roote, Harrington, McGill, Durrant, Shelton, and Ashburner, 1985, Chromosoma 92: 116-23.

Gubb, Roote, McGill, Shelton, and Ashburner, 1986, Genetics 112: 551-75.

genetics: *Sco* revertant. Homozygous lethal in pupal stage. Lethal over *Sco* and most *Sco* revertants, but semilethal with *Sco*^{rv27}. Also semilethal with *l(2)35Ba* alleles and with some *el* and *noc* alleles. Lethality maps between left breakpoints of *Df(2L)A446* and *Df(2L)A267*. Can be converted to autosynaptic form, *AS(2)Sco*^{rv1} (Craymer, 1981). Associated with warped wing and pale scutellum phenotypes (Ashburner *et al.*, 1983).

molecular biology: Left DNA breakpoint at -67.1 to -68.3 kb, the origin of the map being the *EcoRI* site immediately distal to *Adh* (Chia, Karp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706).

other information: Revertants of the *Sco*^{rv1} lethal interaction with *noc*^{TE35A} include *Tp(2;2)Sco*^{rv1}-*DV1* (*viz*) and seven other derivatives, which are not further rearranged, designated *DV4*, *DV5*, and *DV6* (EMS induced) and *DV9*, *DV10*, and *DV11* (γ ray induced) (Ashburner, unpublished).

***In(2LR)Sco*^{rv1L}*TE35A-14*^R**

cytology: *In(2LR)35D1-2;44C3-5*^L*35B1-2;43B3-C1*^R.

origin: Recombinant via autosynaptics between *LS(2)Sco*^{rv1} and *DS(2)TE35A-14*.

discoverer: Roote.

synonym: *In(2LR)Sco*^{rv1L}*TE146(Z)SR14*^R.

***In(2LR)Sco*^{rv1L}*TE35A-15*^R**

cytology: *In(2LR)35D1-2;44C2-3*^L*35B1-2;44D-E*^R.

origin: Recombinant via autosynaptics between *LS(2)Sco*^{rv1} and *DS(2)TE35A-15*.

discoverer: Roote.

synonym: *In(2LR)Sco*^{rv1L}*TE146(Z)GR15*^R.

***In(2LR)Sco*^{rv9}**

cytology: *In(2LR)35D1-2;41*.

origin: X ray induced in *Tp(2;2)Sco*.

synonym: *Sco*^{+R9}.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.

Craymer, 1981, Genetics 99: 75-97.

Ashburner, Detwiler, Tsubota, and Woodruff, 1983, Genetics 104: 405-31.

Ashburner and Harrington, 1984, Chromosoma 89: 329-37.

genetics: *Sco* revertant. Almost lethal when homozygous, most dying as pharate adults; escapers *Sco*. Can be converted to autosynaptic form, *LS(2)Sco*^{rv9}/*DS(2)Sco*^{rv9} (Craymer, 1981).

molecular biology: Left break mapped to the DNA (McGill, Chia, Karp, and Ashburner, 1988, Genetics 119: 647-61).

In(2LR)SD72

cytology: *In(2LR)39D-E;42A*.

references: Lewis, 1962, DIS 36: 87.

Yamasaki and Thompson, 1973, Jpn. J. Genet. 48: 217-29.

Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

Ganetzky, 1977, Genetics 86: 321-55.

genetics: Associated with *SD-72*.

In(2LR)SD-YT186

cytology: *In(2LR)26;56F*.

origin: X ray induced in a *SD-72* chromosome.

references: Yamasaki and Thompson, 1973, Jpn. J. Genet. 48: 217-29.

genetics: *k* value: .4885 - .7778.

In(2LR)shv*^{S15}: *Inversion (2LR) short vein of Segal

cytology: *In(2LR)22F1-2;59B*.

origin: γ ray induced.

references: Segal and Gelbart, 1985, Genetics 109: 119-43.

genetics: Associated with break in *shv* region at 22F1-2. Heterozygotes with *shv* are lethal, with *dpp* alleles are lethal or wild type.

***In(2LR)shv*^{S17}**

cytology: *In(2LR)22F1-2;41A*.

origin: γ ray induced.

references: Segal and Gelbart, 1985, Genetics 109: 119-43.

genetics: Associated with break in *shv* region at 22F1-2. Heterozygotes with *shv* are lethal, with *dpp* alleles are mutant or wild type.

In(2LR)SM1: Inversion (2LR) Second Multiple

cytology: *In(2LR)22A3-B1;60B-C* superimposed on *In(2L)22D1-2;33F5-34A1 + In(2R)42A2-3;58A4-B1*.

new order:

21 - 22A3|60B - 58B1|42A3 - 58A4|42A2 - 34A1|22D2 - 33F5|22D1 - 22B1|60C - 60F.

origin: X ray induced in *In(2L)Cy + In(2R)Cy*.

references: R. F. Grell, 1953, DIS 27: 58.

genetics: The pericentric inversion, *In(2LR)22A3-B1;60B-C*, enhances balancing power of *In(2L)Cy + In(2R)Cy* since it causes the single crossover between the two *Cy* inversions to yield complementary products that are dominant lethal. Heterozygote shows interchromosomal effect; used to increase crossing-over and gene conversion in *ry* region (Chovnick, Ballantine, and Holm, 1971, Genetics 69: 179-209); also causes non-disjunction of compound autosomes (Lindsley and Grell, 1969, Genetics 61: 69-78).

other information: Used as a balancer for chromosome 2, described as *SM1* in the section on balancers.

***In(2LR)SM1-bw*^{V1}**

cytology: *In(2L)22D1-2;33F5-34A1 + In(2LR)22A3-B1;60B-C + In(2LR)40F;59D4-E1*.

new order:

21A - 22A3|60B - 59E1|40F - 59D4|40F - 34A1|22D2 - 33F5|22D1 - 22B1|60C - 60F.

origin: An *SM1* chromosome in which a double crossover replaces *In(2R)Cy* and the centromere with the inner pericentric inversion [*In(2LR)40F;59D4-E1*] from *In(2LR)bw*^{V1}.

references: Kaufmann and Gay, 1970, DIS 45: 81.

genetics: Mutant for *Cy* and *bw*^{V1}.

In(2LR)SM5

cytology: *In(2L)21D2-3;36C + In(2L)29C-E;40F + In(2R)42D;53C;58F* superimposed on *In(2L)22D1-2;33F5-34A1 + In(2LR)22A3-B1;60B-C + In(2R)42A2-3;58A4-B1*. Duplicated for regions 42A3-D and 58B1-F.

new order:

21A – 21D2|36C – 40F|29C – 22D2|34A1 – 36C|
21D3 – 22A3|60B – 58B1|42A3 – 42D|42D – 42A3|
58B1 – 58F|53C – 42D|53C – 58A4|42A2 – 40F|
29E – 33F5|22D1 – 22B1|60C – 60F.

origin: X ray induced in several steps in *In(2LR)SM1*.

discoverer: R. F. Grell, 1955.

references: Mislove and Lewis, 1955, DIS 29: 75.

genetics: Variegated for *lt* owing to *In(2L)29C-E;40F*.

Carries *ds*⁵⁵ (Craymer, 1980, DIS 55: 197-200).

In(2LR)SM5/M(2)58F lethal (C. Hinton); probably related to break in 58F.

other information: Excellent balancer for all of chromosome 2, described as *SM5* in the section on balancers.

In(2LR)SM6

cytology: *In(2LR)SM1 = In(2LR)22A3-B1;60B-C + In(2LR)O = In(2LR)30E-F;50C10-D1*, both inversions superimposed on *In(2L)Cy = In(2L)22D1-2;33F5-34A1 + In(2R)Cy = In(2R)42A2-3;58A4-B1*.

new order:

21 – 22A3|60B – 58B1|42A3 – 50C10|
30E – 22D|34A1 – 42A2|58A4 – 50D1|
30F – 33F5|22D1 – 22B1|60C – 60F.

origin: Recombination between chromosomes derived from *SM1* and *CyO*, *Roi* chromosomes.

discoverer: Craymer.

genetics: Carries *al*² *Cy dp*^{lv1} *cn*^{2P} *sp*² and either *pr* (*SM6a*) or *Roi*(*SM6b*).

other information: Used as a balancer for chromosome 2, described as *SM6* in the section on balancers.

In(2LR)SMG8

cytology: *In(2LR)22D;42*.

new order:

21 – 22A3|60B – 58B1|42A3 – 50C10|
30E – 22D|34A1 – 42A1|58A4 – 50D1|

origin: γ ray induced.

references: Semenova, Mglinetz, and Glotoff, 1970, *Genetika* (Moscow) 6: 165-69.

In(2LR)SMG9

cytology: *In(2LR)35B;57F*.

origin: γ ray induced.

references: Semenova, Mglinetz, and Glotoff, 1970, *Genetika* (Moscow) 6: 165-69.

In(2LR)St: Inversion (2LR) Stalker

cytology: *In(2LR)29D;45B-C*.

origin: Spontaneous in a natural population in Texas.

references: Stalker, 1976, *Genetics* 82: 323-47.

In(2LR)TE35A

origin: γ ray induced in *TE35A*.

synonym: *In(2LR)TE146*.

inversion	cytology	origin	discov ^α	genetics
<i>In(2LR)TE35A-4</i> ^β	35B1-2;43B3-C1	spont	2	<i>pk</i>
<i>In(2LR)TE35A-6</i> ^γ	35B1-2;41	γ rays	1	<i>wv</i>
<i>In(2LR)TE35A-14</i>	35B1-2;43B3-C1	spont	2	
<i>In(2LR)TE35A-15</i> ^δ	35B1-2;44D-E	γ rays	2	
<i>In(2LR)TE35A-50</i>	35B1-2;40-41	γ rays	1	

inversion	cytology	origin	discov ^α	genetics
<i>In(2LR)TE35A-205</i>	<i>In(2LR)35B;60B8-13 + T(Y;3)CA49 + T(2;3)CA48</i>	γ rays	3	
<i>In(2LR)TE35A-222</i>	<i>In(2LR)35B;41</i>	γ rays	3	
<i>In(2LR)TE35A-223</i>	<i>In(2LR)35B;41C</i>	γ rays	3	
<i>In(2LR)TE35A-226</i>	<i>In(2LR)35B;47B10-14</i>	γ rays	3	
α	1 = Ashburner; 2 = Roote; 3 = Samkange.			
β	Reference: Gubb, Roote, McGill, Shelton, and Ashburner, 1986, <i>Genetics</i> 112: 551-75.			
γ	Recoverable in autosynaptic form, <i>AS(2)TE35A-6</i> (Gubb).			
δ	Not suppressed by <i>z</i> .			

In(2LR)TE35A-6^LSco^{rv9R}

cytology: *In(2LR)35B1-2;41^L35D1-2;41^R*; deficient for 35B2-D1.

origin: Recombinant via autosynaptics between *In(2LR)TE35A-6* and *In(2LR)Sco^{rv9}*.

discoverer: Gubb.

synonym: *In(2LR)TE146-6^LSco^{R+9R}*.

genetics: Deficient for *noc - l(2)35Da*.

In(2LR)TE35A-14^LTE35A-4^R

cytology: *In(2LR)35B1-2;43B3-C1^L35B1-2;43B3-C1^R*; deficient for 42F-43A.

new order:

21 – 35B1|43B3 – 43A|42F – 35B2|43C1 – 60.

origin: Recombinant via autosynaptics between

In(2LR)TE35A-14 and *In(2LR)TE35A-4*.

synonym: *In(2LR)TE146(Z)SR14^LTE146(Z.SR36)SZ4^R*.

references: Ashburner.

genetics: Deficient for *pk*.

In(2LR)TE35A-15^LSco^{rv1R}

cytology: *In(2LR)35B1-2;44D-E^L35D1-2;44C4-5^R*. Deficient for 35B1-D2; duplicated for 44C5-D.

origin: Recombinant via autosynaptics of *LS(2)TE35A-15* and *DS(2)Sco^{rv1}*.

synonym: *In(2LR)TE146(Z)GR15^LSco^{rv1R}*.

references: Ashburner.

In(2LR)TE146: see *In(2LR)TE35A*

In(2LR)U: Inversion (2LR) Upturned

cytology: *In(2LR)40;53A* (Bridges, Year Book - Carnegie Inst. Washington 35: 293).

origin: X ray induced.

discoverer: Ball, 32a27.

references: 1935, DIS 3: 17.

genetics: Associated with *U*.

In(2LR)V26

cytology: *In(2LR)26F;41A*.

origin: X ray induced.

references: Valencia, 1970, DIS 45: 37-38.

genetics: Homozygous lethal.

In(2LR)vg: Inversion (2LR) vestigial

references: Alexandrov and Alexandrova, 1987, DIS 66: 185-87.

inversion	cytology	origin ^α	phenotype under 25°
<i>In(2LR)vg^{74b1}</i>	37F-38A1;49D2-E1	caffeine + γ ray	lethal
<i>In(2LR)vg^{74c4}</i>	22A5-B1;49D2-E	caffeine + γ ray	<i>vg^{nw}</i>
<i>In(2LR)vg^{77d1}</i>	25C-D;49D2-E1	MeV	lethal

inversion	cytology	origin α	phenotype under 25°
<i>In(2LR)vg^{79a}</i>	34B2-C1;49B12-C3 + <i>Df(2R)49D1;49E2</i>	MeV	lethal
<i>In(2LR)vg^{79h4}</i>	24D;49D1-E1	γ ray	lethal
<i>In(2LR)vg⁸¹¹¹⁸</i>	36C4-D1;49D2-F1	γ ray	vg
<i>In(2LR)vg^{82c14}</i>	36C-D;49D2-E	252 Cf	vg <i>nw</i>
<i>In(2LR)vg^{82c61}</i>	24E2-F1;49D2-E7	252 Cf	lethal

α MeV = 0.35-0.85 MeV neutrons.

In(2LR)vsh

cytology: *In(2LR)21B;58A + In(2R)52;56*.

origin: X ray induced.

discoverer: Ashburner.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou and Woodruff, 1980, DIS 55: 193-95.

In(2R)41-47

cytology: *In(2R)41A;47A*.

origin: X ray induced simultaneously with *T(1;2)B^{bd}*.

discoverer: Bridges.

references: Morgan, Bridges, and Schultz, 1936, Year Book - Carnegie Inst. Washington 35: 291.

genetics: Probably not separable from *T(1;2)B^{bd} = T(1;2)16A1-2;48C2-3*.

In(2R)434

discoverer: Gelbart.

inversion	cytology
<i>In(2R)434.13</i>	42D-E;58C-E
<i>In(2R)434.39</i>	41A;50E-F
<i>In(2R)434.87</i>	41A;44B-C
<i>In(2R)434.99</i>	41A;57E-F

In(2R)AA21

cytology: *In(2R)56D-E;58E-F + Df(2R)56F;57D-12*.

new order: 41-56D|58E - 57D12|56F-56E|58F-60F.

origin: γ ray induced.

references: O'Donnell, Boswell, Reynolds, and Mackay, 1989, Genetics 121: 273-80.

genetics: Deficient for *Pu* and *tud*.

In(2R)AL: Inversion (2L)

Ashburner Lemeunier

origin: Naturally occurring inversions.

references: Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

inversion	cytology	source
<i>In(2R)AL7</i>	49B;56B	Cameroun
<i>In(2R)AL8</i>	50F;53D	Melbourne, Australia
<i>In(2R)AL9</i>	53A;54D-E	Wales
<i>In(2R)AL10</i>	53B;55C	Reunion Island

In(2R)Arp: Inversion (2L) Aristapedioid

origin: Hybrid dysgenesis.

references: Adler, 1984, Genetics 107: s1.

genetics: Homozygous lethal in embryo. Heterozygotes show partial transformation of arista into tarsus and partial loss of large medial thoracic bristles.

molecular biology: *In situ* hybridization with *P* element

probe shows *P* element at both ends of the inversions.

inversion	cytology
<i>In(2R)Arp1</i>	49A12-B1;49E7-F1
<i>In(2R)Arp2</i>	49A12-B3;49E7-F1
<i>In(2R)Arp3</i>	49A12-B3;49E7-F1

In(2R)AWI

origin: Accumulated on second chromosomes during repeated backcrossing to balancer chromosomes, these second chromosomes coming from a lethal-carrying chromosome *AW* derived in 1967 from a wild-type cage population.

references: Yamaguchi and Mukai, 1974, Genetics 78: 1209-21.

inversion	cytology
<i>In(2R)AWI1</i>	42F;50C
<i>In(2R)AWI2</i>	42E;50E
<i>In(2R)AWI3</i>	43A;45D
<i>In(2R)AWI4</i>	50A;60B
<i>In(2R)AWI5</i>	57C;59D

In(2R)B1

cytology: *In(2R)41A;53E*.

discoverer: Ising.

In(2R)B46

cytology: *In(2R)53D2-E1;57B6-D11*.

synonym: *In(2R)t^{B46}*.

references: Schüpbach and Wieschaus, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 302-17.

genetics: Associated with *tud^{B46}*.

In(2R)Ba^B

cytology: *In(2R)48D-E;60E5-6* (Kennison).

origin: X ray induced.

discoverer: Botas.

synonym: *Antp-1, Art³*.

references: Cohen, Brönnner, Küttner, Jürgens, and Jäckle, 1989, Nature 338: 432-34.

genetics: Associated with *Ba^B*.

In(2R)Bld: see T(1;2)Bld

**In(2R)bw^A: Inversion (2R) brown-Auburn*

cytology: *In(2R)41;59D*.

origin: X ray induced.

discoverer: Dubinin.

synonym: *In(2R)Pm^{D1}*.

references: 1936, Biol. Zh. (Moscow) 5: 851-66.

genetics: Variegated for *bw* and *mi*; variegation for *bw* dominant to *bw*. Dubinin claims brown-Variegated effect exists at both ends of the inversion.

other information: Ninety-one secondary rearrangements derived from irradiation of *In(2R)bw^A* analyzed by Dubinin.

**In(2R)bw^{AL}Cy^R: Inversion (2R) brown-Auburn Left Curly-Right*

cytology: *In(2R)41;59D^L42A2-3;58A4-B1^R*; deficient for 41-42A2 and duplicated for 58B1-59D.

origin: Recombinant carrying left end of *In(2R)bw^A* and right end of *In(2R)Cy*.

references: Dubinin, 1936, Biol. Zh. (Moscow) 5: 851-66.

genetics: Variegated for *bw*; Minute, presumably owing to *Df(2R)M41A*. Wings divergent with incised inner mar-

gins.

***In(2R)bw^{R32}: Inversion (2R) brown-Rearranged**

cytology: *In(2R)41A;59D*.
 origin: X ray induced.
 discoverer: Slatis.
 references: 1955, Genetics 40: 5-23.
 genetics: Associated with *bw*^{R32}.

***In(2R)bw^{R33}**

cytology: *In(2R)41;59D-E*.
 origin: X ray induced.
 discoverer: Slatis.
 references: 1955, Genetics 40: 5-23.
 genetics: Associated with *bw*^{R33}.

In(2R)bw^V: Inversion (2R) brown-Variiegated

phenotype: Heterozygotes exhibit brown variegation;
 homozygous lethal in embryonic stage [e.g.,
In(2R)bw^{De2} (Tsai, 1955, Genetics 40: 601)].

inversion	cytology	origin	discoverer	ref	α
<i>In(2R)bw^{V2}</i>		X ray	Harris, 1929	3, 4, 8	
<i>*In(2R)bw^{V7}</i>		X ray	Winchester, 1932	5, 11	
<i>In(2R)bw^{V30k}</i>		X ray	VanAtta, 30k10	10	
<i>In(2R)bw^{V34k} β</i>	<i>In(2R)41;59E</i> on <i>In(2R)Cy =</i> <i>In(2R)42A2-3;58A4-B1</i>	X ray	Oliver, 34k22	9	
<i>*In(2R)bw^{V40b}</i>	<i>In(2R)41A-B;59D-E</i>	X ray	T. Hinton, 40b	1	
<i>In(2R)bw^{V54a}</i>	<i>In(2R)41A-B;59D4-9</i>	γ ray	Micky, 54a6	7	
<i>*In(2R)bw^{V54b}</i>	<i>In(2R)41A;60D9-11</i> γ	neutrons	Micky, 54b12	7	
<i>*In(2R)bw^{V54c}</i>	<i>In(2R)41;59E1</i>	neutrons	Yanders, 54c5	7	
<i>In(2R)bw^{VDe1}</i>	<i>In(2R)41B2-C1;59E2-4</i>	X ray	Demerec, 33i28	2	
<i>In(2R)bw^{VDe2}</i>	<i>In(2R)41A-B;59D6-E1</i>	X ray	Demerec, 33j14	2	
<i>*In(2R)bw^{VI}</i>	<i>In(2R)41A;59D</i>	spont	Ives, 38i13	6	

α 1 = Atwood, 1942, DIS 16: 47; 2 = Bridges, 1937, Cytologia Fujii Jub., vol. 2: 745-55; 3 = Glass, 1933, J. Genet. 28: 69-112; 4 = Glass, 1934, Am. Nat. 68: 107-14; 5 = Glass, 1939, DIS 12: 47; 6 = Ives, 1950, DIS 24: 58; 7 = Mickey, 1963, DIS 38: 29; 8 = Muller, 1930, J. Genet. 28: 299-334; 9 = Oliver, 1937, DIS 7: 19; 10 = VanAtta, 1932, Genetics 17: 637-59; 11 = Winchester, 1938, DIS 9: 23.
 β Reciprocal recombinants between *In(2R)bw^{V34k}* and a normal second chromosome are reciprocally duplicate and deficient for 41-42A2 and 58B-E.
 γ Unlikely that right breakpoint at 60D9-11; perhaps at 59D9-11.

In(2R)bw^{VDe1L}Cy^R: Inversion (2R) brown-Variiegated of Demerec 1 Left Curly-Right

cytology: *In(2R)41B2-C1;59E2-4^L42A2-3;58A4-B1^R*;
 deficient for 41C1-42A2 and duplicated for 58B1-59E2.
 origin: Recombinant carrying left end of *In(2R)bw^{VDe1}*
 and right end of *In(2R)Cy*.
 genetics: Deficient for *Df(2R)M41A* but not *rl* or *M(2)p*;
 duplicated for *M(2)58F*, *bw*, and *mi*.

In(2R)bw^{VDe2L}Cy^R: Inversion (2R) brown-Variiegated of Demerec 2 Left Curly-Right

cytology: *In(2R)41A-B;59D6-E1^L42A2-3;58A4-B1^R*;
 deficient for 41B-42A2 and duplicated for 58B1-59D6.
 origin: Recombinant carrying left end of *In(2R)bw^{VDe2}*
 and right end of *In(2R)Cy*.
 genetics: Duplicated for *M(2)58F* and *bw* but not *mi*; carries *Df(2R)M41A* and *Dip-A* (Voelker and Langley, 1978, Genetica 49: 233-36) but not *rl* or *M(2)p*.

In(2R)C2

cytology: *In(2R)41C;57B*.
 discoverer: Ising.

In(2R)C72: Inversion (2R) Crossover suppressor

cytology: *In(2R)50E;57F;60D*.
 new order:
 21 – 50E|57F – 60D|57F – 50E|60D – 60F.
 origin: X ray induced.
 discoverer: Roberts and D. Stewart, 1964.
 references: Roberts, 1970, Genetics 65: 429-48.
 genetics: Homozygous viable. Recombination between *b*
 and *sp* sharply reduced.

In(2R)C129

cytology: *In(2R)43F;56E*.
 origin: X ray induced.
 discoverer: Roberts, 1965.
 references: 1970, Genetics 65: 429-48.
 genetics: Homozygous lethal. Recombination between *b*
 and *sp* reduced.

In(2R)CA40

cytology: *In(2R)54F;59A*.
 origin: γ ray induced simultaneously with *Df(2L)TE35A-3*.
 discoverer: Ashburner.

In(2R)CA41

cytology: *In(2R)41;59D1-4*.
 origin: γ ray induced.
 discoverer: Ashburner.

In(2R)Cy: Inversion (2R) Curly

cytology: *In(2R)42A2-3;58A4-B1* (Bridges and Li in Morgan, Bridges, and Schultz, 1936, Year Book - Carnegie Inst. Washington 35: 292).
 origin: Spontaneous.
 discoverer: L. Ward, 21f.
 references: 1923, Genetics 8: 276-300.
 Sturtevant, 1931, Carnegie Inst. Washington Publ. No. 421: 20.
 Graubard, 1932, Genetics 17: 81-105.
 Ramel and Valentin, 1965, Hereditas 54: 193-207.
 1968, DIS 43: 140-41.
 Valentin, 1972, Hereditas 72: 243-54.
 genetics: Left breakpoint between *ap* and *pk*. Homozygous viable and fertile. Crossing over in 2R strongly reduced. Carries *cn*² in most laboratory stocks. Interchromosomal effect on crossing over and primary non-disjunction greater when in *cis* with *In(2L)Cy* than in *trans* (Valentin, 1972).
 other information: Used in combination with *In(2L)Cy* as a balancer for chromosome 2.

In(2R)Cy^Lbw^{VDe1R}: Inversion (2R) Curly-Left brown-Variiegated of Demerec 1 Right

cytology: *In(2R)42A2-3;58A4-B1^L41B2-C1;59E2-4^R*;
 duplicated for 41C1-42A2 and deficient for 58B1-59E2.
 origin: Recombinant carrying left end of *In(2R)Cy* and
 right end of *In(2R)bw^{VDe1}*.
 genetics: Deficient for *M(2)58F*, *bw*, and *mi*; duplicated
 for *Df(2R)M41A* but not *rl* or *M(2)p*.

***In(2R)Cy^Lbw^{VDe2R}*: Inversion (2R) Curly-Left brown-Variegated of Demerec 2 Right**
cytology: *In(2R)42A2-3;58A4-B1^L41A-B;59D6-E1^R*; duplicated for 41B-42A2 and deficient for 58B1-59D6.
origin: Recombinant carrying left end of *In(2R)Cy* and right end of *In(2R)bw^{VDe2}*.
genetics: Deficient for *M(2)58F* and *bw* but not *mi*; duplicated for *Df(2R)M41A* but not *rl* or *M(2)p*.

In(2R)Cy^LC129^R
cytology: *In(2R)42A2-3;58A4-B1^L43F;56E^R*. Deficient for 41B-42A2 and 58A4-59E1.
origin: Recombination between *In(2R)Cy* and *In(2R)C129*.
references: Nix, 1973, Mol. Gen. Genet. 120: 309-18.
other information: May be used in combination with *In(2L)t* as a balancer for chromosome 2.

***In(2R)en²⁸*: Inversion (2R) engrailed**
cytology: Complex rearrangement, including *Df(2R)47B3;47B9-14 + In(2R)47B9-14;48A1-2 + Df(2R)48A1-2;48B-C1*.
origin: X ray induced.
references: Eberlein, 1982, Genetics 100: s21-22. Eberlein and Russell, 1983, Dev. Biol. 100: 227-37.
genetics: Homozygous lethal and cell lethal. When heterozygous over *en¹* or *en³⁰*, *In(2R)en²⁸* survives to adult stage and shows weak *en* phenotype.

In(2R)en⁴⁸
cytology: *In(2R)47F;48A3-4*.
origin: X ray induced.
discoverer: Kornberg.
references: Kuner, Nakanishi, Ali, Drees, Gustavson, Theis, Kauvar, Kornberg, and O'Farrell, 1985, Cell 42: 309-16. Drees, Ali, Soeller, Coleman, Poole, and Kornberg, 1987, EMBO J. 6: 2803-09.
genetics: Homozygous lethal. Associated with *en⁴⁸*.
molecular biology: Right breakpoint 10-11 kb toward the centromere from the insertion site of the *en¹* transposition. (Kuner *et al.*, 1985).

In(2R)en^{C2}
cytology: *In(2R)47A;48A*.
origin: Induced by ethyl methanesulfonate.
references: Kornberg, 1981, Proc. Nat. Acad. Sci. USA 78: 1095-99. Drees, Ali, Soeller, Coleman, Poole, and Kornberg, 1987, EMBO J. 6: 2803-09.
genetics: Homozygous lethal; semilethal over *en⁷*; *en* phenotype over *en* showing duplicated antennal segments and fused leg segments.
molecular biology: Breakpoint 14 kb upstream of the *en* transcripts (Drees *et al.*, 1987).

In(2R)G: Inversion (2R) Gallup
cytology: *In(2R)50C20;54B1* (Vassin).
origin: Spontaneous.
discoverer: Ives.
references: 1957, DIS 31: 83.
genetics: Associated with *mam¹*. Crossing over in 2R reduced to about 13%.
molecular biology: 50C breakpoint between coordinates -27.3 and -25.5 in the region of *mam* (Yedvobnick, Smoller, Young, and Mills, 1988, Genetics 118: 483-97).

In(2R)HNI: Inversion(2R) Hiraizumi Nakazima
cytology: *In(2R)55E;60E*.
origin: Spontaneous in a wild population in Japan.
references: Hiraizumi and Nakazima, 1965, DIS 40: 72. Hartl, 1980, Genetics 80: 539-47.
other information: Carried along with *In(2R)NS* in *SD*-bearing chromosome.

In(2R)IW6: Inversion (2R) Inoue Watanabe
cytology: *In(2R)42E;47C*.
origin: Spontaneous in a natural population in Japan.
references: Inoue and Watanabe, 1979, Jpn. J. Genet. 54: 69-82.

In(2R)IW7
cytology: *In(2R)51B;59B*.
origin: Spontaneous in a natural population in Japan.
references: Inoue and Watanabe, 1979, Jpn. J. Genet. 54: 69-82.

In(2R)JHI
origin: Accumulated on second chromosomes during repeated backcrossing to balancer chromosome. These second chromosomes come from a lethal-carrying chromosome *JH* derived in 1967 from a wild-type cage population.
references: Yamaguchi and Mukai, 1974, Genetics 78: 1209-21.

inversion cytology

<i>In(2R)JHI1</i>	42C;47F
<i>In(2R)JHI2</i>	46C;57C
<i>In(2R)JHI3</i>	50C;59A
<i>In(2R)JHI4</i>	51B;52C
<i>In(2R)JHI5</i>	51E;56C
<i>In(2R)JHI6</i>	52A;59C
<i>In(2R)JHI7</i>	54D;59C
<i>In(2R)JHI8</i>	54E;59D
<i>In(2R)JHI9</i>	56A;57B
<i>In(2R)JHI10</i>	57B;59D
<i>In(2R)JHI11</i>	57F;59D

In(2R)K: Inversion (2R) Kalle
cytology: *In(2R)50B;55B*.
origin: X ray induced.
references: Ytterborn, 1968, Hereditas 59: 49-62. Valentin, 1972, Hereditas 72: 243-54.
genetics: Homozygous lethal.

In(2R)K3
cytology: *In(2R)42A;57C*.
discoverer: Ising.

In(2R)KA - In(2R)KB
origin: Spontaneous in natural populations in Korea.
references: Paik, 1986, DIS 63: 167.

In(2R)Kc
cytology: *In(2R)55E;57D*.
origin: Spontaneous in a natural population in Korea.
references: Choi, 1977, DIS 52: 88. 1977, Genetica 47: 155-60.

In(2R)Kd
cytology: *In(2R)57C;58E*.
origin: Spontaneous in a natural population in Korea.
references: Choi, 1977, DIS 52: 88. 1977, Genetica 47: 155-60.

In(2R)KOG: Inversion (2L) of Knibb, Oakeshott, and Gibson

origin: Spontaneous in natural populations.
references: Knibb, Oakeshott, and Gibson, 1981, Genetics 98: 833-47.

inversion	cytology
<i>In(2R)KOG-C</i>	51D;59F-60A
<i>In(2R)KOG-D</i>	42A;53A-B
<i>In(2R)KOG-E</i>	50C;57A-B

In(2R)Kr^{UR1}: Inversion (2R) Krüppel of Urs Rosenberg

cytology: *In(2R)58A;60F3*.
origin: X ray induced in *Bl If* chromosome.
references: Preiss, Rosenberg, Kienlin, Seifert, and Jäckle, 1985, Nature (London) 313: 27-32.
genetics: Mutant for *Kr*. Incomplete revertant for *If*, the phenotype depending on the genetic background.
molecular biology: Four-kilobase deletion within the cloned *Kr* region.

In(2R)L

cytology: *In(2L)43F;49F*. Probably same as *In(2R)L* of Choi (1977).
origin: Spontaneous in a natural population of Japan (Watanabe and Oshima, 1965) and Korea (Choi, 1977).
references: Watanabe and Oshima, 1965, Ann. Rpt. Nat. Inst. Genetics Japan 15: 18.
 Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.
 Choi, 1977, DIS 52: 88.
 1977, Genetica 47: 155-60.

In(2R)L5

cytology: *In(2R)44A;53D*.
discoverer: Ising.

In(2R)LK1

cytology: *In(2R)41A-B;57F2-58A1*.
origin: X ray induced in a chromosome carrying *ix*.
references: Keltner and Puro, 1978, DIS 53: 170.
genetics: Homozygous viable. Males fertile and females *ix*.

In(2R)M: Inversion (2R) of Mourad

cytology: *In(2R)54F1-55A1;58F-59A*.
origin: Spontaneous.
discoverer: Mourad and Mallah.
references: 1960, Evolution 14: 166-70.
 Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

In(2R)mam

origin: Induced by hybrid dysgenesis in a Harwich second chromosome which has *P*-elements at 42C-D; 50C-D, and 57B.
references: Yedvobnick, Smoller, Young, and Mills, 1988, Genetics 118: 483-97.
genetics: Mutant for *mam*.

inversion	synonym	cytology ^α
<i>In(2R)mam⁴²</i>	HD3/1	42C-D;50C-D
<i>In(2R)mam⁴⁵</i>	HD11/2	50C-D;57B
<i>In(2R)mam⁴⁶</i>	HD13/6	42C-D;50C-D

^α *P*-element at 50C-D.

In(2R)Mg: Inversion (2R) Mglinetz

inversion	cytology	origin	ref ^α
<i>In(2R)Mg2</i>	42B;46B	γ ray	2
<i>In(2R)Mg102</i>	45E;56B	γ ray	1
<i>In(2R)Mg103</i>	52A;57C	γ ray	1
<i>In(2R)Mg105</i>	43A;54E	γ ray	1
<i>In(2R)Mg106</i>	49E;52F	γ ray	1
<i>In(2R)Mg107</i>	54E;57D	γ ray	1
<i>In(2R)Mg108</i>	43E;60B	γ ray	1
<i>In(2R)Mg109</i>	42A;60B	γ ray	1
<i>In(2R)Mg119</i>	52E;60A	32 P feeding	1

^α 1 = Mglinetz, 1968, Genetika (Moscow) 4(8): 81-86; 2 = Mglinetz, 1971, Genetika (Moscow) 7(8): 108-14.

In(2R)N2G

cytology: *In(2R)50C-D;54D*.
origin: Spontaneous in wild populations.
references: Ives, 1947, Evolution 1: 42-47.
genetics: *mam* allele (Yedvobnick, Smoller, Young, and Mills, 1988, Genetics 118: 483-97).

In(2R)NC: Inversion (2R) from North Carolina

cytology: *In(2R)46A;49F*.
origin: Spontaneous in *In(2R)NS*.
references: Mettler, Voelker, and Mukai, 1977, Genetics 87: 169-76.

In(2R)NS: Inversion (2R) from Nova Scotia

cytology: *In(2R)52A2-B1;56F9-13* (Bridges and Li in Morgan, Bridges, and Schultz, 1936, Year Book - Carnegie Inst. Washington 35: 292-3).
origin: Naturally occurring inversion.
discoverer: Sturtevant, 13i (as a crossover suppressor).
synonym: *In(2R)A* (Stalker, 1976, Genetics 82: 323-47); *In(2R)C* (Oshima and Watanabe, 1965, DIS 40: 88).
references: Sturtevant, 1919, Carnegie Inst. Washington Publ. No. 278: 305-41.
 1931, Carnegie Inst. Washington Publ. No. 421: 1-27.
 Mukai, Watanabe, and Yamaguchi, 1974, Genetics 77: 771-93.
 Inoue and Watanabe, 1979, Jpn. J. Genet. 54: 69-82.
genetics: Crossing over reduced to about 1.5% between centromere and inversion and to about 0.1% between inversion and tip of chromosome. Includes *Amy* (Mukai *et al.*, 1974).
other information: Found in many natural populations (e.g., Warters, 1944, Texas Univ. Publ. 4445: 129-74; Oshima and Watanabe, 1965, DIS 40: 88; Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57; Stalker, 1976, Genetics 82: 523-47; Choi, 1977, DIS 52: 88; Mettler, Voelker and Mukai, 1977, Genetics 87: 169-76; Knibb, 1982, Genetika 58: 213-21). Found in most *SD* chromosomes (Watanabe and Oshima, 1970, Genetics 64: 93-106).

In(2R)PA – In(2R)PF: Inversion (2R) Pipkin

origin: Naturally occurring inversions.
references: Pipkin, Franklin-Springer, Law, and Lubega, 1976, J. Hered. 67: 258-66.

inversion	cytology
<i>In(2R)PA</i>	49E;56C
<i>In(2R)PB</i>	50B;57A
<i>In(2R)PC</i>	51A;57C-D
<i>In(2R)PD</i>	53B;57A-B

inversion	cytology
<i>In(2R)PE</i>	53F;57A
<i>In(2R)PF</i>	55F-56F;59B-C

***In(2R)Pcl^{W4}*: Inversion (2R) Polycomblike**

cytology: *In(2R)55A;57A*.

origin: γ ray induced.

discoverer: Williams.

references: Sato, Russell, and Denell, 1983, Genetics 105: 357-70.

Sato, Hayes, and Denell, 1984, Dev. Genet. 4: 185-98.

genetics: Mutant for *Pcl*. Some homozygotes do not complete head involution (Sato *et al.*, 1984).

***In(2R)pk⁷⁸*: Inversion (2R) prickle**

cytology: *In(2R)42C1-7;43F5-8;59F5-8*; deficient for 42C7 - 43F5.

new order:

21A - 42C1|59F5 - 43F8|59F8 - 60.

origin: X ray induced.

references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

genetics: Deficient for *pk*, *pwn*, *spla*, and *cn*.

In(2R)Pm^{D1}: see *In(2R)bw^A*

***In(2R)PS*: Inversion (2R) Paik Sung**

origin: Naturally occurring inversions in Korea.

references: Paik and Sung, 1980, DIS 55: 120.

inversion	cytology
<i>In(2R)PS19</i>	42A;60A
<i>In(2R)PS20</i>	42D;60F
<i>In(2R)PS21</i>	42E;43A
<i>In(2R)PS22</i>	43B;46E
<i>In(2R)PS23</i>	47C;54D
<i>In(2R)PS24</i>	47E;55E
<i>In(2R)PS25</i>	51F;60D
<i>In(2R)PS26</i>	54B;59C
<i>In(2R)PS27</i>	56D;59B

***In(2R)Pu^K*: Inversion (2R) Punch of Krivshenko**

cytology: *In(2R)41;57E-F*.

origin: X ray induced.

discoverer: Krivshenko, 53k24.

synonym: *In(2R)Pm^K*.

references: 1954, DIS 28: 75.

genetics: Associated with *Pu^K* (Rowan). *Pu^K/Pu²* is lethal.

***In(2R)Pu^{r1}*: Inversion (2R) Punch-recessive**

cytology: *In(2R)57C3-4;57D13-E1*.

origin: Spontaneous.

discoverer: Ives.

references: O'Donnell, 1982, Genetics 100: s52.

Mackay and O'Donnell, 1983, Genetics 105: 35-53.

Mackay, Reynolds, and O'Donnell, 1985, Genetics 111: 885-904.

O'Donnell, Boswell, Reynolds, and Mackay, 1989, Genetics 121: 273-80.

genetics: Mutant for *Pu^{r1}* which shows a recessive eye color phenotype. Homozygous lethal.

In(2R)RBR

origin: Spontaneous [accumulated on second chromosome lines during repeated backcrossing of males heterozygous for each second chromosome line over *In(2LR)bw^{V1}* to *In(2LR)bw^{V1}/SM1* females].

references: Yamaguchi, Cardellino, and Mukai, 1976, Genetics 83: 409-22.

inversion	cytology
<i>In(2R)RBR7</i>	58E;59B
<i>In(2R)RBR13</i>	43A;52F
<i>In(2R)RBR24</i>	44B;57C
<i>In(2R)RBR31-1</i>	44D;47D
<i>In(2R)RBR31-2</i>	50A;57B
<i>In(2R)RBR66</i>	51F;57F
<i>In(2R)RBR69</i>	50F;56C
<i>In(2R)RBR77</i>	51B;52B
<i>In(2R)RBR93</i>	49E;57E
<i>In(2R)RBR100</i>	50B;58B
<i>In(2R)RBR117</i>	42A;59E
<i>In(2R)RBR135</i>	44E;47C
<i>In(2R)RBR137</i>	51E;57A
<i>In(2R)RBR143</i>	49D;50F
<i>In(2R)RBR145</i>	43F;54B
<i>In(2R)RBR148</i>	49B;56C

In(2R)SD5

cytology: *In(2R)45C-F;49A*.

origin: A component of the *SD-5* chromosome isolated at Madison, Wisconsin.

references: Lewis, 1962, DIS 36: 87.

In(2R)SD72-240

cytology: *In(2R)43;53*.

origin: X ray induced in *SD-72* chromosome.

references: Yamazaki and Thompson, 1973, Jpn. J. Genet. 48: 217-29.

In(2R)SMG10

cytology: *In(2R)52A;58B*.

origin: γ ray induced.

references: Semenova, Mglinetz, and Glotoff, 1970, Genetika (Moscow) 6(4): 165-69.

In(2R)SMG11

cytology: *In(2R)55F;60F*.

origin: γ ray induced.

references: Semenova, Mglinetz and Glotoff, 1970, Genetika (Moscow) 6(4): 165-69.

***In(2R)St*: Inversion (2R) Stalker**

origin: Naturally occurring inversions

references: Stalker, 1976, Genetics 82: 323-47.

inversion	cytology
<i>In(2R)St-A</i>	52A;56F
<i>In(2R)St-B</i>	49B;60B
<i>In(2R)St-C</i>	48F-49A;60E
<i>In(2R)St-D</i>	49A;51D
<i>In(2R)St-E</i>	45D;49B
<i>In(2R)St-F</i>	53C;55F
<i>In(2R)St-H</i>	50F;54B
<i>In(2R)St-J</i>	55F;60E
<i>In(2R)St-K</i>	50A;54B
<i>In(2R)St-L</i>	42A;45B
<i>In(2R)St-M</i>	44C;47E
<i>In(2R)St-N</i>	49F;51D
<i>In(2R)St-O</i>	49B;51D
<i>In(2R)St-P</i>	43E;47D
<i>In(2R)St-Q</i>	54B;60F

In(2R)TA

cytology: *In(2R)50A;53B*.
origin: Spontaneous in a natural population in Brownsville, Texas.
references: Yang, Kojima, and Kovarik, 1971, DIS 47: 71-72.
 Langley, Tobari, and Kojima, 1974, Genetics 78: 921-36.

In(2R)top: Inversion (2R) torpedo

cytology: *In(2R)57A;57F*.
origin: γ ray induced.
references: Price, Clifford, and Schüpbach, 1989, Cell 56: 1085-92.
genetics: Mutant for *Egfr*^{f36}. Homo- and hemizygotes embryonic lethals.
molecular biology: Right break in the large intron separating exon 2 from exon 3 of *Egfr*; also has a 2.5 kb deletion at the 57F breakpoint.

In(2R)vg: Inversion (2R) vestigial

references: Alexandrov and Alexandrova, 1987, DIS 66: 185-87.

inversion	cytology	origin	phenotype under 25°
<i>In(2R)vg 72a1</i>	44C2-5;49D2-E1	γ ray	lethal
<i>In(2R)vg 78a1</i>	41D-E;49D3-E1	γ ray	vg ^{no}
<i>In(2R)vg 78a2</i>	49D3-E;56E	γ ray	vg
<i>In(2R)vg 78j3</i>	41D2-E1;49D2-E1	γ ray	lethal
<i>In(2R)vg 78k3</i>	49D2-E1;59D4-8	γ ray	lethal
<i>In(2R)vg 79b4</i>	41C-D;49D2-E1	MeV	lethal
<i>In(2R)vg 79d3</i>	41A;49D2-E2	MeV	lethal
<i>In(2R)vg 79d4</i>	41E;49D2-E1	MeV	vg ^{nw}
<i>In(2R)vg 79d7</i>	41D-E;49D3-F1	MeV	vg ^{nw}
<i>In(2R)vg 79f2</i>	49D2-E1;49E7-F13	NaF + γ ray	vg ^{nw}
<i>In(2R)vg 79h5</i>	49D2-F;50A2-3	γ ray	vg
<i>In(2R)vg 79h6</i>	41E-F;49D2-E1 + 42B2-3;57F-58A1	γ ray	vg ^{nw}
<i>In(2R)vg 79h7</i>	49D2-7;49F10-13	γ ray	vg ^{nw}
<i>In(2R)vg 81b1</i>	48C4-D1;49D2-E	γ ray	lethal
<i>In(2R)vg 81c28</i>	41D-E;49D3-E7	γ ray	vg ^{nw}
<i>In(2R)vg 81c41d</i>	49C2;49F14	γ ray	lethal
<i>In(2R)vg 83b39</i>	49D2-E;51D2-6	γ ray	vg ^{nw}
<i>In(2R)vg 83c3</i>	41C-D;49D2-E1	γ ray	vg ^{nw}
<i>In(2R)vg 83c43</i>	49C2-3;49D2-E	γ ray	vg ^{nw}
<i>In(2R)vg 83d4</i>	48E2-F1;49D2-E1	γ ray	vg ^{nw}
<i>In(2R)vg 83f38</i>	49D;59D-E	γ ray	lethal
<i>In(2R)vg 83f-XD</i>	48E2-F1;49D2-E	X ray	vg ^{nw}
<i>In(2R)vg 83l2b</i>	41D-E;49D2-E1	MeV	lethal
<i>In(2R)vg 84f51</i>	44F2-45A1;49D2-E1	X ray	vg ^{nw}
<i>In(2R)vg 84hXC</i>	48E6-F1;49D2-E1	γ ray	lethal
<i>In(2R)vg 84hXD</i>	41B-C;49D2-E1	γ ray	vg ^{nw}
<i>In(2R)vg 85c</i>	41B-C;49F1 + Df(2R)49B12-C1;49E2-F1	MeV	lethal

^α MeV = 0.35-0.85 MeV neutrons.

In(2R)vg^U: Inversion (2R) vestigial-Ultra

cytology: *In(2R)49C1-2;50C1-2* (Ratty and Lindsley, 1964, DIS 38: 30).
origin: γ ray induced.
discoverer: Ives, 55131.
references: 1956, DIS 30: 72-73.
genetics: Associated with vg^U. Homozygous lethal.

In(2R)vg^W: Inversion (2R) vestigial-wingless

cytology: *In(2R)47F15-48A12;49E4-5* (Ashburner).
origin: X ray induced.
references: Williams and Bell, 1988, EMBO J. 7: 1355-63.

genetics: Associated with vg.

In(2R)Z: Inversion (2R) Zacharopoulou

origin: Naturally occurring inversions in Greece.
references: Zacharopoulou, 1974, DIS 51: 110.

inversion	cytology
<i>In(2R)Z5</i>	47F;60A
<i>In(2R)Z6</i>	52A;60A
<i>In(2R)Z7</i>	54C;60D

In(2R)ZP: Inversion (2R)

Zacharopoulou Pelecanos

origin: Naturally occurring inversions in Greece.
references: Zacharopoulou and Pelecanos, 1980, Genetica 54: 105-11.

inversion	cytology
<i>In(2R)ZP7</i>	51D;59F
<i>In(2R)ZP9</i>	53A;56F
<i>In(2R)ZP10</i>	54A;57A
<i>In(2R)ZP11</i>	53E;55E

In(3)B20 - In(3)H140

origin: X ray induced along with the corresponding T(Y;3)^s.

references: The Seattle-La Jolla Drosophila Laboratories, 1971, DIS 47, Suppl. Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84.

inversion	cytology
<i>In(3)B20</i>	65F;80-81
<i>In(3)B68</i>	80-81;87A
<i>In(3)B81</i>	80-81;87-88
<i>In(3)B154</i>	64C-D;80-81
<i>In(3)G24</i>	80-81;85D-E
<i>In(3)H140</i>	66F;80-81

In(3)Brd: Inversion (3) Bearded

cytology: *In(3)71A1-2;80-81*; position of right break with respect to centromere undermined.
origin: Three γ ray-induced inversions with similar breakpoints isolated as *Brd*⁴, *Brd*⁵, and *Brd*⁶.
discoverer: Posakony.
genetics: Homozygous lethal; less extreme than *Brd*¹.

In(3)C: Inversion (3) Crossover suppressor

origin: X ray induced.
discoverer: Roberts, 1964.
references: 1970, Genetics 65: 429-48.
genetics: Homozygous lethal. Recombination as indicated in table.

inversion	cytology	reduced recombination
<i>In(3)C41</i>	80-81;91E-F	st-ca
<i>In(3)C229</i>	67B;80-81	ve-st
<i>In(3)C289</i>	80-81;93E	ve-st

In(3)h^{m2}: Inversion (3) hairy

cytology: *In(3)66D6-10;80C-81F*.
origin: X ray induced.
references: Ingham, Pinchin, Howard, and Ish-Horowicz,

1985, Genetics 111: 463-86.

Howard, Ingham, and Rushlow, 1988, Genes Dev. 2: 1037-46.

genetics: Homozygous lethal. Variegates for *h* when heterozygous with *h*.

***In(3)p^{100.48}: Inversion (3) pink**

cytology: *In(3)80-81;85A6-B1*; position of left breakpoint with respect to centromere not determined.

origin: X ray induced.

discoverer: Alexander.

references: Ward and Alexander, 1957, Genetics 42: 42-54.

genetics: Mutant for *p*.

***In(3)p^{100.88}**

cytology: *In(3)80-81;94D11-E1*; position of left breakpoint with respect to centromere not determined.

origin: X ray induced.

discoverer: Alexander.

references: Ward and Alexander, 1957, Genetics 42: 42-54.

genetics: Mutant for *p*; mutation independent of breakpoint.

In(3)pb²⁸

cytology: *In(3LR)80-81;84A4-5*; position of left break with respect to centromere undetermined.

origin: X ray induced.

references: Pultz, Diederich, Cribbs, and Kaufman, 1988, Genes Dev. 2: 901-20.

genetics: Inversion of the *ANTC* sequences into heterochromatin; variegates for *pb*.

In(3)st^{K21}: Inversion (3) scarlet

cytology: *In(3)73A3-4;80-81*.

discoverer: Belote and McKeown.

genetics: Mutant for *st*.

***In(3L)100.307**

cytology: *In(3L)62E2-4;64C2-4*.

origin: X ray induced simultaneously with *e^{100.307}*.

discoverer: Alexander.

references: Ward and Alexander, 1957, Genetics 42: 42-54.

In(3L)100r

cytology: Inversions with one break in 3L euchromatin break point (ebp) and the other in region 80 proximal to break in *Dp(1;3)N²⁶⁴⁻¹⁰⁰ = Dp(1;3)3B4-C1;4B4-5;80*.

new order:

61 – ebp|80|(3C1 – 4B4)|80 – ebp|80 – 100.

origin: X ray induced in *Dp(1;3)N²⁶⁴⁻¹⁰⁰*.

references: Gersh, 1959, Genetics 44: 163-72.

phenotype: Partial reversion of white mottling as result of separation of duplication from centromere region.

inversion	cytology
<i>In(3L)100r2</i>	76A4-B1;80
<i>In(3L)100r8</i>	73F1-74A1;80
<i>In(3L)100r11</i>	65A1-B1;80

In(3L)A: see *In(3L)P*

In(3L)A82

cytology: *In(3L)70D-F;79B-C*.

origin: X ray induced simultaneously with *T(Y;3)A82 = T(Y;3)Xhy⁺;98F*.

references: Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84.

In(3L)AL11: Inversion (3L)

Ashburner Lemeunier

cytology: *In(3L)62E;68A*.

origin: Spontaneous in a natural population.

references: Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

In(3L)AL12

cytology: *In(3L)69F;75C-D*.

origin: Spontaneous in a natural population.

references: Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

In(3L)AM44: Inversion (3L) Amherst

cytology: *In(3L)61A;62C-D*.

origin: Spontaneous in a natural population in Massachusetts collected by Ives in 1965 (Ashburner, 1972, DIS 49: 34).

references: Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

genetics: Homozygous viable.

***In(3L)Apt: Inversion (3L) Apart**

cytology: Breakpoints unknown.

origin: X ray induced.

discoverer: Belgovsky, 34e23.

references: 1935, DIS 3: 27.

genetics: Associated with *Apt*.

In(3L)B: see *In(3L)M*

In(3L)B141 - In(3L)R150

origin: X ray induced along with the corresponding *T(Y;3)s*.

references: The Seattle-La Jolla Drosophila Laboratories, 1971, DIS 47, Suppl.

Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, David, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84.

inversion	cytology
<i>In(3L)B141</i>	64E;68A
<i>In(3L)H138</i>	66B;66F
<i>In(3L)R150</i>	64D;71B?

***In(3L)Bit: Inversion (3L) Bitten**

cytology: Breakpoints unknown.

origin: X ray induced.

discoverer: Lefevre, 48g5.

references: 1949, DIS 23: 58.

genetics: Associated with *Bit*.

In(3L)BK160

cytology: *In(3L)73F;75B*.

references: Leicht and Bonner, 1988, DIS 67: 54-56.

In(3L)Brd: Inversion (3L) Bearded

origin: γ ray induced derivatives of *Brd*¹..

discoverer: Posakony.

genetics: Mutant for *Brd*.

inversion	cytology	synonym	comments
<i>In(3L)Brd</i> ²	71A;71F	<i>In(3L)Brd</i> ^{m0}	lethal; severe eye defect; > <i>Brd</i> ¹
<i>In(3L)Brd</i> ³	70D-E;71A	<i>In(3L)Brd</i> ^{m1}	lethal; severe eye defect; > <i>Brd</i> ¹

In(3L)C: Inversion (3L) Crossover suppressor

origin: X ray induced.

genetics: Homozygous lethal. Recombination between *ve* and *st* reduced or virtually eliminated.

inversion	cytology	discoverer	ref ^α
<i>In(3L)C90</i>	62B;80C	Roberts and Stewart, 1964	1, 2
<i>In(3L)C299</i>	63C;80	Roberts, 1965	2
<i>In(3L)C302</i>	63A;71A	Roberts, 1965	2

^α 1 = Marsh, 1978, DIS 53: 155-56; 2 = Roberts, 1970, Genetics 65: 429-48.

In(3L)CA1

cytology: *In(3L)68C1-5;79D4*.

origin: Spontaneous.

discoverer: Richards.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.

In(3L)CA31

cytology: *In(3L)65F;69A*.

origin: γ ray induced.

discoverer: Ashburner.

In(3L)CA55

cytology: *In(3L)70A;70D*.

origin: γ ray induced.

discoverer: Ashburner.

In(3L)D: Inversion (3L) Dichaete

cytology: *In(3L)69D3-E1;70C13-D1* (Bridges).

origin: Spontaneous.

discoverer: Bridges, 15a3.

references: Morgan, Bridges, and Schultz, 1937, Year Book - Carnegie Inst. Washington 36: 301.

genetics: Associated with *D*.

In(3L)E: see *In(3L)P*

In(3L)F: see *In(3L)M*

In(3L)Fd: Inversion (3L) Federova

cytology: *In(3L)62;67*.

origin: Recombination in *In(3LR)DcxF*, separating *In(3L)Fd* from *In(3LR)CxD*.

references: Puro and Novitski, 1982, DIS 58: 126.

genetics: Homozygous lethal. No phenotypic effects in heterozygotes.

In(3L)Fz³: Inversion (3L) frizzled

cytology: *In(3L)70D6-7;75D3-8 + In(3)73D3-5;80-81*.

new order:

60 – 70D6|(75D3 – 73D5|80 – 75D8|70D7 – 73D3)|81 – 100.

origin: γ ray induced simultaneously with *In(2L)CA11 = In(2L)21D;36F*.

discoverer: Velissariou.

references: Ashburner, Angel, Detwiler, Faithfull, Gubb,

Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

Velissariou and Ashburner, 1981, Chromosoma 84: 173-85.

genetics: Associated with *fz*³.

In(3L)fz⁴

cytology: *In(3L)70D6-7;80-81 + In(3LR)79F;87D-E*.

new order:

60 – 70D6|(81 – 87D|79F – 70D7|80 – 79F)|87E – 100.

origin: γ ray induced simultaneously with *T(2;3)CA15 = T(2;3)42F;100F*.

discoverer: Velissariou.

references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

Velissariou and Ashburner, 1981, Chromosoma 84: 173-85.

genetics: Associated with *fz*⁴.

In(3L)fz^{CT8CX1}

cytology: *In(3L)61C;69A;70D4-7*.

new order:

61A – 61C|69A – 70D4|69A – 61C|70D7 – 100.

origin: Induced by hybrid dysgenesis in *fz^{CT8C}*.

discoverer: Adler.

genetics: Strong *fz* phenotype. Homozygous lethal.

In(3L)fz^{K21}

cytology: *In(3L)70D4-7;75A5-B12*.

origin: γ ray induced.

discoverer: Adler.

genetics: Strong *fz* phenotype. Homozygous lethal. Viable over all other *fz* alleles.

In(3L)h: Inversion (3L) hairy

inversion	cytology	origin	ref ^α	genetics
* <i>In(3L)h</i> ¹⁰	61A2-3;66D	X ray	5	mutant for <i>h</i>
* <i>In(3L)h</i> ¹¹	66D11-12;80C	X ray	5	mutant for <i>h</i>
* <i>In(3L)h</i> ¹⁶	66D14-E1;80F	X ray	2, 3, 4	homoz. semilethal; <i>h</i> ¹⁶ / <i>h</i> variegated
<i>In(3L)h</i> ⁴⁰	66D10-15;72C	hybrid dysgenesis	2	homoz. lethal
<i>In(3L)h</i> ^{42^β}	65B1-2;66D6-10	X ray	1, 2	homoz. lethal
<i>In(3L)h</i> ⁴⁴	66D5-11;80C	X ray	1	mutant for <i>h</i>

^α 1 = Howard, Ingham, and Rushlow, 1988, Genes Dev. 2: 1037-46; 2 = Ingham, Pinchin, Howard, and Ish-Horowicz, 1985, Genetics 111: 463-86; 3 = Jeffery, 1971, DIS 47: 37; 4 = Jeffery, 1979, Genetics 91: 105-25; 5 = Ward and Alexander, 1957, Genetics 42: 42-54. See references for synonyms.

^β Molecular biology: 66D break of *In(3L)h*⁴² and *In(3L)h*⁴⁴ located at -5.15 to -2.5 kb and -9.35 to -6.8 kb, respectively, on the DNA map of *h* (Howard *et al.*, 1988); "+" values to the right, "-" values to the left. The coordinate system places 0 at the start of transcription (Rushlow).

In(3L)HR

origin: X ray induced.

discoverer: Ashburner.

genetics: Suppresses crossing over between *h* and *ri*.

inversion	cytology	ref ^α	more genetics
<i>In(3L)HR2</i>	62E;78B	1	homozygous viable
<i>In(3L)HR10</i>	64C;75C	1	homozygous lethal
<i>In(3L)HR13^β</i>	75F;80	1	homozygous lethal
<i>In(3L)HR15</i>	64D2-5;68C4-9	1, 2	homozygous viable, fertile and wild type
<i>In(3L)HR27</i>	76C3-4;80B2-4	1, 2	homozygous viable, fertile; variegated

inversion	cytology	ref ^α	more genetics
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eye color
^α 1 = Ashburner, 1972, DIS 49: 34; 2 = Craymer, 1981, Genetics 99: 75-97.
^β Induced simultaneously with *In(3R)HR13=In(3R)81;98E*.

In(3L)IW: Inversion (3L) Inoue Watanabe

origin: Naturally occurring inversions in Japan.
references: Inoue and Watanabe, 1979, Jpn. J. Genet. 54: 69-82.

inversion	cytology
<i>In(3L)IW7</i>	66C;69E
<i>In(3L)IW8</i>	66C;77D
<i>In(3L)IW9</i>	68F;75C
<i>In(3L)IW10</i>	70F;75F

In(3L)K: Inversion (3L) Korea

origin: Naturally occurring inversions in Korea.
references: Choi, Ha, and Kim, 1984, DIS 60: 76.

inversion	cytology
<i>In(3L)Kd</i>	65A;67C
<i>In(3L)Ke</i>	71B;80C
<i>In(3L)Kf</i>	64E;76A

In(3L)KA

cytology: *In(3L)66C;71B-C*.
origin: Spontaneous in natural populations in Korea.
references: Paik, 1986, DIS 63: 167.

In(3L)KOG

origin: Spontaneous in natural populations of Australasia.
references: Knibb, Oakeshott, and Gibson, 1981, Genetics 98: 833-47.

inversion	cytology
<i>In(3L)KOG-H</i>	62C;68A
<i>In(3L)KOG-I</i>	62A-B;69A

In(3L)L17

cytology: *In(3L)68B;73F*.
origin: X ray induced simultaneously with, but independently of, *T(Y;3)L17 = T(Y;3)h16;86A*.
references: Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84.

In(3L)L20: see T(Y;3)L20

****In(3L)M: Inversion (3L) of Mourad***

cytology: *In(3L)66D;71D*.
origin: Spontaneous.
synonym: *In(3L)B = In(3L)66C;70B* (Warters, 1944, Texas Univ. Publ. 4445: 129-74) and *In(3L)F = In(3L)66C;71B* (Oshima and Watanabe, 1965, DIS 40: 88) probably the same.
references: Mourad and Mallah, 1960, Evolution 14: 166-70.
 Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.
 Tantawy, Mourad, and Masry, 1977, Egypt. J. Genet. Cytol. 6(2): 370-74.
 Inoue and Watanabe, 1979, Jpn. J. Genet. 54: 69-82.

In(3L)Mg: Inversion (3L) Mglinetz

inversion	cytology	origin ^α	ref ^β
<i>In(3L)Mg58</i>	64E;78D	1	2
<i>In(3L)Mg62</i>	62A;67B	1	3
<i>In(3L)Mg63</i>	67E;71C	1	3
<i>In(3L)Mg64</i>	72C;80	1	3
<i>In(3L)Mg112</i>	66B;73F	1	1
<i>In(3L)Mg113</i>	66B;80	1	1
<i>In(3L)Mg114</i>	69D;73F	1	1
<i>In(3L)Mg115</i>	71E;79B	2	1
<i>In(3L)Mg116</i>	62C;80	2	1
<i>In(3L)Mg117</i>	67A;80	2	1
<i>In(3L)Mg127</i>	62B;80	2	1

^α 1 = γ ray induced; 2 = ³²P feeding.
^β 1 = Mglinetz, 1968, Genetika (Moscow) 4(8): 81-86; 2 = Mglinetz, 1972, Genetika (Moscow) 8(2): 82-92; 3 = Mglinetz, 1973, Genetika (Moscow) 9(3): 69-75.

In(3L)P: Inversion (3L) Payne

cytology: *In(3L)63B8-11;72E1-2* on Bridges revised map (Craymer, 1981, 1984).

origin: Naturally occurring inversion.

discoverer: Payne, 17g.

synonym: *In(3L)A* (Warters, 1944, Texas. Univ. Publ. 4445: 129-74); *In(3L)E* (Watanabe, 1967, Mem. Fac. Sci. Kyushu Univ. Ser E: 159-78; Inoue and Watanabe, 1979, Jpn. J. Genet. 54: 69-82).

references: Payne, 1918, Indiana Univ. Studies 5 No. 36: 1-45.

1924, Genetics 9: 327-42.

Sturtevant, 1931, Carnegie Inst. Washington Publ. No. 421: 18.

Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

Craymer, 1981, Genetics 99: 75-97.

1984, DIS 60: 234-36.

genetics: Homozygous viable, although it often contains lethals of independent origin.

other information: Often associated with *In(3R)P*. Useful as a balancer for *3L*. Allows only about 0.02% crossing over between *ru* and *st*. Balancers contain recessive lethals or *Me*. Balancers for all of chromosome 3 made by combining *In(3L)P* with *In(3R)P* or *In(3R)C*. Found in many wild populations (e.g., Warters, 1944, Texas Univ. Publ. 4445: 129-174; Oshima and Watanabe, 1965, DIS 40: 88; Stalker, 1976, Genetics 82: 323-47; Mettler, Voelker, and Mukai, 1977, Genetics 87: 169-76; Knibb, 1982, Genetics 58: 213-21).

In(3L)P8: Inversion (3L) Pasadena 8

cytology: *In(3L)63A-C;78F* (Craymer).

origin: X ray induced.

discoverer: E.B. Lewis.

In(3L)PA - In(3L)PI: Inversion (3L) Pipkin

origin: Naturally occurring inversion.

references: Pipkin, Franklin-Springer, Law, and Lubega, 1976, J. Hered. 67: 258-66.

inversion	cytology
<i>In(3L)PA</i>	63A;67B
<i>In(3L)PB</i>	63F-64A;67B
<i>In(3L)PC</i>	65B;69F
<i>In(3L)PD</i>	65E-F;72C
<i>In(3L)PE</i>	66D;71C
<i>In(3L)PF</i>	63A;72F
<i>In(3L)PG</i>	68A;73A-B

inversion	cytology
<i>In(3L)PH</i>	68F;76D-E
<i>In(3L)PI</i>	63F;66A

In(3L)Pc: Inversion (3L) Polycomb

cytology: *In(3L)76C;78B + In(3L)78C-D;78E5-6* (Tiong).
origin: γ ray induced.
discoverer: Tiong.
genetics: Associated with *Pc*²⁶.

In(3L)pers: Inversion (3L) persimmon

cytology: *In(3L)63C2-5;73B2-5*.
origin: X ray induced.
discoverer: Demerec, 3712.
references: 1941, DIS 14: 40.
genetics: Associated with *pers*.

In(3L)PS: Inversion (3L) Paik Sung

origin: Naturally occurring inversions in Korea.
references: Paik and Sung, 1980, DIS 55: 120.

inversion	cytology
<i>In(3L)PS28</i>	61F;67E
<i>In(3L)PS29</i>	62A;63C
<i>In(3L)PS30</i>	65E;67D
<i>In(3L)PS31</i>	66D;73B
<i>In(3L)PS32</i>	69C;77C
<i>In(3L)PS33</i>	71E;75E
<i>In(3L)PS34</i>	72F;78B

In(3L)rg17

cytology: *In(3L)62B2-7;63F*.
origin: γ ray induced.
references: Sliter, Henrich, Tucker, and Gilbert, 1989, Genetics 123: .if n .bp

In(3L)SMG: Inversion (3L) Semenova Mglinetz Glotoff

origin: γ ray induced.
references: Semenova, Mglinetz, and Glotoff, 1970, Genetika (Moscow) 6(4): 165-69.

inversion	cytology
<i>In(3L)SMG12</i>	67C;77
<i>In(3L)SMG13</i>	63F;75C
<i>In(3L)SMG14</i>	67B;67E
<i>In(3L)SMG15</i>	63A;73E
<i>In(3L)SMG16</i>	65;70C
<i>In(3L)SMG17</i>	61F;72F
<i>In(3L)SMG18</i>	61C;67D
<i>In(3L)SMG19</i>	70C;75D
<i>In(3L)SMG20</i>	62B;66A

327-36.

****In(3L)Spr: Inversion (3L) Spread***

cytology: Breakpoints unknown.
origin: X ray induced.
discoverer: Oliver, 32k21.
references: 1935, DIS 4: 15.
genetics: Associated with *Spr*.

In(3L)st⁵: Inversion (3L) scarlet

cytology: *In(3L)73A2-3;80C*.
origin: γ ray induced.
references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou and

Walker, 1981, DIS 56: 186-91.

Velissariou and Ashburner, 1981, Chromosoma 84: 173-85.

genetics: Associated with *st*⁵.

In(3L)St: Inversion (3L) Stalker

origin: Naturally occurring inversions.
references: Stalker, 1976, Genetics 82: 323-47.

inversion	cytology
<i>In(3L)St-A</i>	60F-61A;67B
<i>In(3L)St-C</i>	65A;73B
<i>In(3L)St-D</i>	65E;66E
<i>In(3L)St-E</i>	66C;71C
<i>In(3L)St-F</i>	67C-D;76A
<i>In(3L)St-G</i>	64B;68F-69A
<i>In(3L)St-H</i>	62E;70C
<i>In(3L)St-J</i>	61C;64C
<i>In(3L)St-K</i>	75C;76F
<i>In(3L)St-L</i>	66C;70F
<i>In(3L)St-M</i>	64C;66F
<i>In(3L)St-N</i>	77B;79E

In(3L)std11: Inversion (3L) scarlet transformer doublesex

cytology: *In(3L)72E1-2;73A11-B1;73D1-4*; deficient for 73B1-D1 (Belote and McKeown).

new order:

61 – 72E1|73A11 – 72E2|73D4 – 100.

origin: X ray induced.

discoverer: M. Hoffmann.

genetics: *st*⁺ phenotype; deficient for *Abl*, *l(3)73Bc*, and *Dab*.

In(3L)TB

cytology: *In(2L)70B;75A*.

origin: Spontaneous in natural population in Brownville, Texas.

references: Yang, Kojima, and Kovarik, 1971, DIS 47: 71-72.

Langley, Tobari, and Kojima, 1974, Genetics 78: 921-36.

****In(3L)th²⁹³: Inversion (3L) thread***

cytology: *In(3L)72A2-B1;76A4-B1;79A4-B1*.

new order:

61 – 72A2|79A4 – 76B1|72B1 – 76A4|79B1 – 100.

origin: X ray induced.

discoverer: Alexander.

synonym: *Tp(3;3)th^{100.293}*.

references: Ward and Alexander, 1957, Genetics 42: 42-54.

genetics: Mutant for *th*.

In(3L)th^{SS108}

cytology: *In(3L)63F3-5;72A3-4 + In(3L)68F5;73F*.

new order:

61A – 63F3|72A3 – 68F5|73F – 72A4|

63F5 – 68F5|73F – 100.

origin: X ray induced.

discoverer: S. Smith.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.

genetics: Associated with *th^{SS108}*.

In(3L)V

cytology: *In(3L)68F;75C*.
origin: Naturally occurring inversion.
references: Inoue and Watanabe, 1979, Jpn. J. Genet. 54: 69-82.
 Inoue, 1979, Jpn. J. Genet. 54: 83-96.

In(3L)V4-14: Inversion (3L) Valencia

cytology: *In(3L)70B;80B*. Includes a possible deficiency in 70C.
origin: X ray induced.
references: Valencia, 1970, DIS 45: 37-38.
genetics: Homozygous lethal.

In(3L)V13-1

cytology: *In(3L)70B;71E-F*.
origin: X ray induced simultaneously with *T(2;3)35B;96E*.
references: Valencia, 1970, DIS 45: 37-38.
genetics: Homozygous lethal.

In(3L)VV11: Inversion (3L) V. Velissariou

cytology: *In(3L)64C4-8;69F3-7*.
origin: γ ray induced.
references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou and Walker, 1981, DIS 56: 186-91.

In(3L)VV14

cytology: *In(3L)75C1-3;75F1-5*.
origin: γ ray induced.
references: Velissariou, 1981(?), Ph.D. Thesis, Cambridge University.
genetics: Homozygous viable.

In(3L)W^{rv16}: Inversion (3L) Wrinkled

cytology: *In(3LR)70F1-2;75C3-4*.
origin: X ray induced.
synonym: *In(3L)W^{+R16}*.
references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.
genetics: Revertant of W.

In(3L)Y

cytology: *In(3L)68F;75C*.
origin: Spontaneous in natural population in Japan.
references: Inoue and Watanabe, 1979, Jpn. J. Genet. 54: 69-82.
 Inoue, Watanabe, and Watanabe, 1984, Evolution 38: 753-65.

In(3L)Yt: Inversion (3L) Ytterborn

cytology: *In(3L)70C;76D*.
origin: Spontaneous.
references: Ytterborn, 1968, Hereditas 59: 49-62.

In(3L)Z8: Inversion (3L) Zacharopoulou

cytology: *In(3L)66A;79B*.
origin: Spontaneous in natural population in Greece.
references: Zacharopoulou, 1974, DIS 51: 110.

In(3L)Z9

cytology: *In(3L)69C;72C*.
origin: Spontaneous in natural population in Greece.
references: Zacharopoulou, 1974, DIS 51: 110.

In(3L)ZP: Inversion (3L)

Zacharopoulou Pelecanos

origin: Naturally occurring inversions in Greece.
references: Zacharopoulou and Pelecanos, 1980, Genetika 54: 105-11.

inversion	cytology
<i>In(3L)ZP13</i>	69A;72B
<i>In(3L)ZP14</i>	68F;73B
<i>In(3L)ZP15</i>	65E;73B
<i>In(3L)ZP16</i>	61C;62C
<i>In(3L)ZP17</i>	66D;71D
<i>In(3L)ZP18</i>	73B;79D
<i>In(3L)ZP19</i>	65A;69A
<i>In(3L)ZP20</i>	66B;70A
<i>In(3L)ZP21</i>	69E;78D

In(3LR)79i

cytology: *In(3LR)67B11-C1;87D5-13*.
origin: Spontaneous.
discoverer: Duncan, 79i.
references: Craymer.
genetics: Homozygous lethal.

In(3LR)100r

cytology: Inversions with one break in 3R euchromatin (ebp) and the other in region 80 proximal to *Dp(1;3)3B4-C1;4B4-5;80*.
new order:
 61 – 80|(3C1 – 4B4)|80|ebp – 80|ebp – 100.
origin: X ray induced in *Dp(1;3)N²⁶⁴⁻¹⁰⁰*.
references: Gersh, 1959, Genetics 44: 163-72.
phenotype: Partial reversion of white mottling owing to separation of duplication from centromere region.

inversion	ebp
<i>In(3LR)100r1</i>	96B1-3
<i>*In(3LR)100r3</i>	86C1-D1
<i>*In(3LR)100r7</i>	99B-C1
<i>In(3LR)100r27</i>	96B3-5

In(3LR)201 - In(3LR)238

origin: X ray induced in Canton-S.
discoverer: Ou.
genetics: Transvects *Cbx Ubx/+*. Also see table.

inversion	cytology	ref ^a	genetics
<i>In(3LR)201</i>	71;82	1	
<i>In(3LR)202</i>	68F-69A;81F	1	
<i>In(3LR)206</i>	65E10-12;85A1-3 (B.S. Baker)	2	homozygous viable, fertile, wild type
<i>In(3LR)207</i>	3L;81F	1	
<i>In(3LR)208</i>	61E2-3;86C1-2 (Craymer)	2	
<i>In(3LR)209</i>	62F;89D (Craymer)	2	homozygous lethal
<i>In(3LR)215</i>	63E;86D (Craymer)	2	
<i>In(3LR)216^B</i>	63E3-6;80-81; 84A1-2 (B.S. Baker; Craymer)	1	
<i>In(3LR)217</i>	71B2-4;81F1	2	
<i>In(3LR)220</i>	63C5-D1;85E-F (Craymer)	2	
<i>In(3LR)222</i>	62A10-12;85F10-12 (Craymer)	2	homozygous viable
<i>In(3LR)223</i>	64D5-E1;84E8-10 (B.S. Baker)	2	
<i>In(3LR)224</i>	69E7-F1;83B7-C1	2	homozygous viable, fertile, wild type
<i>In(3LR)225</i>	77E1;88E2-3 (Craymer)	2	
<i>In(3LR)226</i>	64A10-B1;87E2-4 (Craymer)	2	
<i>In(3LR)228</i>	76;81	1	
<i>In(3LR)229</i>	61F7-62A1;88B (Craymer)	2	
<i>In(3LR)230</i>	67F2-68A1;84E2-7	2	
<i>In(3LR)231</i>	68A;83B	1	

inversion	cytology	ref ^α	genetics
<i>In(3LR)232</i>	67F3-4;84D12-14 (B.S. Baker)		
<i>In(3LR)234</i>	67D6-8;88A10-B1	2	homozygous viable, fertile, wild type
<i>In(3LR) 238</i>	80D-F;89B (Craymer)	2	
	^α 1 = Craymer; 2 = Craymer, 1984, DIS 60: 234-36.		
	^β new order: 61 - 63E3 81 - 84A1 80 - 63E6 84A2 - 100.		

In(3LR)A114

cytology: *In(3LR)80;92A2-B1* (E.B. Lewis).
origin: X ray induced simultaneously with, but independently of, *T(Y;3)A114 = T(Y;3)Xhy⁺;61F*.
references: The Seattle-La Jolla Drosophila Laboratories, 1971, DIS 47, Suppl. Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84. Craymer, 1984, DIS 60: 234-36.
genetics: Homozygous lethal (Craymer, 1981, Genetics 99: 75-97).

In(3LR)A200: see ***T(Y;3)A200***

In(3LR)Antp^{PW}

cytology: *In(3R)71F;84B1-2*.
references: Garber, Kuroiwa, and Gehring, 1983, EMBO J. 2: 2027-36. Schneuwly and Gehring, 1985, Dev. Biol. 108: 377-86.
genetics: Mutant for *Antp^{PW}*.

In(3LR)Antp^{PW}

cytology: *In(3LR)71F;84B1-2*.
origin: Induced by methoxydiethylnitrosoamine.
discoverer: Pinchin.
references: Garber, Kuroiwa, and Gehring, 1983, EMBO J. 2: 2027-36.
genetics: Mutant for *Antp*.
molecular biology: Right breakpoint localized to a clone approximately 100 kilobases proximal to the left breakpoint of *In(3R)Hu*, the origin of the *ANTC* walk of Garber *et al.*

In(3LR)Antp^{rv6}: Inversion (3LR) Antennapedia-revertant

cytology: *In(3LR)79D1-2;84A4-B2*.
origin: X ray induced in *Antp^{Ns}*.
synonym: *In(3LR)Antp^{Ns+R6}*.
references: Duncan and Kaufman, 1975, Genetics 80: 733-52.
genetics: Revertant of *Antp^{Ns}*. Homozygous lethal. Also lethal with *Antp^B* and *Antp^{Scx}*.

In(3LR)Antp^{rv16}

cytology: *In(3LR)75A-B;82B-C + In(3R)80C;84A4-B2*.
new order: 61 - 75A|(82B - 80C|84A4 - 82C|75B - 80C)|84B2 - 100.
origin: γ ray induced.
synonym: *Antp^{Ns+R16}*.
references: Duncan and Kaufman, 1975, Genetics 80: 733-52.
genetics: Revertant of *Antp^{Ns}*. Lethal homozygous and with *Antp^B*.

In(3LR)Antp^{rvC4}

cytology: *In(3LR)75B;84B1-2*.
origin: Induced by ethyl methanesulfonate.
synonym: *In(3LR)Antp^{Ns+RC4}*.
references: Struhl, 1981, Nature (London) 292: 635-38. Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, Cell 35: 763-76.
genetics: Revertant of *Antp^{Ns}*. Homozygous lethal and lethal over *Df(3R)4SCB* (Scott *et al.*, 1983).
molecular biology: Right breakpoint approximately 120 kb proximal to left breakpoint of *In(3R)Hu* (Garber, Kuroiwa, and Gehring, 1983, EMBO J. 2: 2027-36) and 128 kb to the right of the proximal breakpoint of *Tp(3;3)Dfd^{rvX16}* (Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, Cell 35: 763-76).

In(3LR)Antp^W

cytology: *In(3LR)75C;84B1-2*.
origin: γ ray induced.
references: Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, Cell 35: 763-76.
genetics: Mutant for *Antp*. Homozygous lethal.
molecular biology: Right breakpoint 158 kb distal to the left breakpoint of *Tp(3;3)Dfd^{rvX16}*.

In(3LR)B130: see ***T(Y;3)B130***

In(3LR)B158

cytology: *In(3LR)76A;93B-94A*.
origin: X ray induced.
references: Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84. Craymer, 1984, Genetics 108: 573-87.
genetics: Homozygous lethal (Craymer, 1981, Genetics 99: 75-97). Associated with, but separable from, *T(Y;3)B158*.

In(3LR)B238

cytology: *In(3LR)80D-80F;88A10-B1*.
origin: X ray induced.
references: Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, and Gould-Somero, 1972, Genetics 71: 157-84.
genetics: Induced simultaneously with *T(Y;2)B238*.

In(3LR)BK133

cytology: *In(3LR)80A;89A*.
references: Leicht and Bonner, 1988, DIS 67: 54-56.

In(3LR)BTD: Inversion (3LR) Bithorax Transvection Disrupter

origin: γ ray induced.
references: Smolik-Utlaut and Gelbart, 1987, Genetics 116: 285-98.
genetics: Disrupts transvection effects at the BXC.

inversion	cytology
<i>In(3LR)BTD2</i>	62D-E;84B
<i>In(3LR)BTD7</i>	76A;87A;92B ^α
<i>In(3LR)BTD11</i>	80;88B

^α New order: 61 - 76A|87A - 76A|92B - 87A|92B - 100 (tentative).

***In(3LR)bx_d*: Inversion (3LR) bithoraxoid**

origin: X ray induced.

references: Craymer, 1981, Genetics 99: 75-97.

genetics: Mutant for *bx_d*.

inversion	cytology	discoverer
<i>In(3LR)bx_d</i> ¹⁰⁶	72D11-E1;89E2-3	E.B. Lewis
<i>In(3LR)bx_d</i> ¹¹³ α	69C3-4;89E2-3	E.B. Lewis
<i>In(3LR)bx_d</i> ¹⁹⁴ β	80;89E	Shaw

α Apparently associated with small deletion of bithorax material at 89E; 3R breakpoint 7-10 kilobases proximal to distal breakpoint of *In(3R)Cb_x*^{rv1} (Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, Science 221: 23-29).

β Synonym: *In(3LR)bx_d*⁹²; *In(3LR)bx_d*^{19409.2X}.

***In(3LR)by*³⁶: Inversion (3LR) blistery**

cytology: *In(3LR)66;89B;96*.

references: Kempthues, Raff, and Kaufman, 1983, Genetics 105: 345-56.

genetics: Mutant for *by*.

***In(3LR)by*⁴⁴**

cytology: *In(3LR)65;81*.

references: Kempthues, Raff, and Kaufman, 1983, Genetics 105: 345-56.

genetics: Mutant for *by*.

***In(3LR)C*: Inversion (3LR) Crossover suppressor**

origin: X ray induced.

discoverer: Roberts, 1964, 1965.

references: 1970, Genetics 65: 429-48 [for all except *In(3LR)C267*, for which see Craymer, 1981, Genetics 99: 75-97].

inversion	cytology	genetics
<i>In(3LR)C35</i>	64B;89E	homozygous lethal; recombination practically eliminated between <i>ve</i> and <i>st</i> and reduced between <i>st</i> and <i>ca</i>
<i>In(3LR)C117</i>	64D;89B	homozygous lethal; <i>ve</i> - <i>st</i> and <i>st</i> - <i>ca</i> recombination reduced
<i>In(3LR)C165</i>	64C;83C	homozygous viable; recombination practically eliminated between <i>ve</i> and <i>st</i>
<i>In(3LR)C175</i>	65C;95E	homozygous lethal; recombination practically eliminated between <i>ve</i> and <i>st</i> and between <i>st</i> and <i>ca</i>
<i>In(3LR)C190</i>	69F;89D	homozygous lethal; recombination reduced between <i>ve</i> and <i>st</i> and between <i>st</i> and <i>ca</i>
<i>In(3LR)C267</i> ^{α}	74F;88D	
<i>In(3LR)C269</i>	78C;98F	homozygous viable; recombination practically eliminated between <i>st</i> and <i>ca</i>
<i>In(3LR)C334</i> ^{β}	67E;86D;91F	homozygous lethal; recombination reduced between <i>ve</i> and <i>st</i> and between <i>st</i> and <i>ca</i>

α Originally associated with *T(2;3)C267* (CP627).

β new order: 61 - 67E|86D - 67E|91F - 86D|91F - 100.

In(3LR)CA18

cytology: *In(3LR)80;98B4-8*.

origin: γ ray induced.

discoverer: Ashburner.

genetics: Induced with *In(2LR)noc*⁷.

In(3LR)CA25

cytology: *In(3LR)61A-B;92B*.

origin: γ ray induced; occurred simultaneously with partial loss of *TE35BC-33*.

discoverer: Ashburner.

references: Gubb, Shelton, Roote, McGill, and Ashburner, 1984, Chromosoma 91: 54-64.

***In(3LR)Cx_D*: Inversion (3LR) Crossover Suppressor Dichaete**

cytology: *In(3LR)71F;85C* + *In(3LR)80;84A;93F* superimposed on *In(3L)69D3-E1;70C13-D1*.

new order:

61A - 69D3|70C13 - 69E1|70D1 - 71F|85C - 84A|80 - 84A|93F - 85C|71F - 80|93F - 100 (Bridges).

origin: X ray induced in *In(3L)D*.

discoverer: Oliver.

synonym: *CxD*; *Dcx*.

references: Glass, 1933, J. Genet. 28: 70.

Federova, 1937, Dokl. Acad. Nauk SSSR 14: 135-38.

genetics: Carries *D* (separates from other inversions with frequency of 0.2%). Crossing over strongly reduced in chromosome 3 except distal half of 3L; virtually no crossing over between *st* and *e*.

other information: Name easily confused with what has been called *C(3)x*, which appears to be *In(3L)P* + *In(3R)P* (Lewis, 1956, DIS 30: 130).

***In(3LR)Dcx_F*: Inversion (3LR) Dichaete crossover suppressor of Federova**

cytology: *In(3L)62;67* superimposed on *In(3L)69D3-E1;70C13-D1* + *In(3LR)71F;85C* + *In(3LR)80;84A;93F*. From Federova's drawings (1937), there appears to be an inversion from about 62 to 67 in addition to a complex rearrangement, presumably *In(3LR)Cx_D*.

new order:

61 - 62|67 - 62|67 - 69D3|70C13 - 69E1|70D1 - 71F|85C - 84A|80 - 84A|93F - 85C|71F - 80|93F - 100.

origin: X ray induced in *In(3LR)Cx_D*.

discoverer: Federova.

synonym: *In(3LR)Cx_F*; *Dcx_F*; *CxF*, *D*.

references: 1937, Dokl. Acad. Nauk SSSR 14: 135-38.

Craymer, 1980, DIS 55: 197-200.

genetics: Carries *ru*, *h* and *D*. Crossing over strongly inhibited throughout chromosome 3.

***In(3LR)DI*^{KX12} Inversion (3LR) Delta**

cytology: *In(3LR)67C-D;91F;92B*.

new order:

61 - 67C|91F - 67D|92B-100. Lacks 91F - 92B.

origin: X ray induced.

discoverer: Vässin.

references: Campos-Ortega.

genetics: Associated with *DI*.

***In(3LR)dsx*³⁴: Inversion (3LR) doublesex**

cytology: *In(3LR)71F1-2;84E1-2*. Break near distal edge of 84E1-2.

origin: X ray induced in *dsx*^M.

discoverer: Baker, Hoff, Kaufman, Wolfner, and Hazelrigg, 1991, Genetics 127: 125-38.

synonym: *In(3LR)dsx*^{Mas+R13}.

genetics: Revertant of *dsx*^M.

***In(3LR)dsx*³⁶**

cytology: *In(3LR)76A5-7;84E1-4*.
origin: X ray induced in *dsx*^M.
discoverer: Baker, Hoff, Kaufman, Wolfner, and Hazelrigg, 1991, Genetics 127: 125-38.
synonym: *In(3LR)dsx*^{Mas+R23}.
genetics: Revertant of *dsx*^M.

***In(3LR)E(spl)*^{rv1}: Inversion (3LR) Enhancer of split-revertant**

cytology: *In(3LR)96F-97A,99B*.
origin: X ray induced.
references: Lehmann, Jiménez, Dietrich, and Campos-Ortega, 1983, Wilhelm Roux's Arch. Dev. Biol. 192: 62-74.
genetics: Revertant of *E(spl)*.

***In(3LR)f*: Inversion (3LR) free arms**

origin: X ray induced as reciprocal translocation between *F(3L)* and *F(3R)*. Replacement of the centromere regions with that of a normal chromosome by double recombination converts the translocation into a pericentric inversion.
discoverer: Craymer, 1981.
references: 1984, DIS 60: 217-18 and 234-36.

inversion	cytology
<i>In(3LR)f1</i>	74D;86E1-2
<i>In(3LR)f7</i> ^{α β γ}	75A1-2;90A1-5
<i>In(3LR)f9</i>	68C9;84F9
<i>In(3LR)f13</i>	63C5-D1;84D4-7
<i>In(3LR)f19</i> ^{α γ}	62A,98A

^α Homozygous viable, fertile, and phenotypically wild type.
^β Wings may be somewhat flimsy, but otherwise wild type.
^γ Subsequently reconstituted as *In(3LR)* by double-crossover centromere replacement from a normal third chromosome.

***In(3LR)fkf*: Inversion (3LR) fork head**

cytology: *In(3LR)80;98D2-3*.
origin: X ray induced.
references: Jürgens and Weigel, 1988, Roux's Arch. Dev. Biol. 197: 345-54.
genetics: Weak *fkf* phenotype.

***In(3LR)gv*^U: Inversion (3LR) grooved**

cytology: *In(3LR)69C1-2;81*.
origin: γ ray induced.
synonym: *In(3LR)gs*^U.
references: Akam, Roberts, Richards, and Ashburner, 1978, Cell 13: 215-25.
genetics: Mutant for *gv*. *eyg/gv*^U flies show *eyg* phenotype; *gv/gv*^U flies show *eyg* and *gv* phenotype. Homozygous lethal.

***In(3LR)h*^{R15}**

cytology: *In(3LR)66D14-E1;85B5-C12*.
origin: X ray induced.
synonym: *In(3LR)h*⁺¹⁵.
references: Jeffery, 1979, Genetics 91: 105-25.
 Ingham, Pinchin, Howard, and Ish-Horowitz, 1985, Genetics 111: 463-86.
genetics: Homozygous lethal.
molecular biology: Breakpoint located within cloned region of *h* (Holmgren, 1984, EMBO J. 3: 569-73).

In(3LR)H159*:** see ***T(Y;3)H159

In(3LR)H175*:** see ***T(Y;3)H175

***In(3LR)Hab*^{rvC51}:** see ***In(3LR)iab2*^{C51}**

****In(3LR)Hi*: Inversion (3LR) Hirsute**

cytology: *In(3LR)71A;91F*.
origin: X ray induced.
discoverer: Bishop, 1939.
genetics: Associated with *Hi*.

In(3LR)HR

origin: X ray induced.
discoverer: Ashburner.
genetics: Suppresses crossing over between *h* and *ri*.

inversion	cytology	ref ^α	more genetics
<i>In(3LR)HR5</i> ^β	67F;88E + 75C;98C	1	homozygous lethal
<i>In(3LR)HR6</i>	67F;83E	1	homozygous lethal
<i>In(3LR)HR8</i> ^γ	70B;78C + 72C-E;83A	1	homozygous lethal
<i>In(3LR)HR20</i>	64C;84D	1	homozygous lethal
<i>In(3LR)HR32</i>	62E;81	1	homozygous lethal
<i>In(3LR)HR33</i> ^δ	61A1-2;87B2-4	1, 2	homozygous viable, fertile, and <i>gvl</i> -like, often overlapping wild type

^α 1 = Ashburner, 1972, DIS 49: 34; 2 = Craymer, 1981, Genetics 99: 75-97.

^β new order: 61A - 67F|88E - 75C|98C - 88E|67F - 75C|98C - 100F.

^γ new order: 61A - 70B|78C - 83A|72C - 70B|78C - 72E|83A - 100F.

^δ No puffing in 61A2-3 in homozygous larvae.

***In(3LR)HR33*^{L bxd}^{106R}**

cytology: *In(3LR)61A1-2;87B2-4^L72D11-E1;89E2-3^R*; deficient for 61A2-72D11 and 87B4-89E2.
origin: Recombinant carrying the left end of *In(3LR)HR33* and the right end of *In(3LR)bxd*¹⁰⁶.
discoverer: Craymer.
references: 1984, DIS 60: 234-36.
genetics: Lethal over normal 3; viable over *Dp(3;3)S2*.

***In(3LR)iab2*^{C51}: Inversion (3LR) infraabdominal 2**

cytology: *In(3LR)80;89E*.
origin: X ray induced in *Hab*.
discoverer: Crosby.
synonym: *In(3LR)Hab*^{rvC51}.
references: Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.
genetics: Associated with *iab2*. Revertant of *Hab*.
molecular biology: 3R breakpoint 43 kb distal to distal breakpoint of *In(3R)Cbx*^{rv1}.

***In(3LR)iab3*⁴**

cytology: *In(3LR)80C;85A;89E1-4*; complex inversion.
origin: Induced by ethyl methanesulfonate.
synonym: *In(3LR)Uab*⁴; *In(3LR)Sab3*^{Uab}.
references: Lewis, 1978, Nature (London) 276: 565-70.
genetics: Associated with *Uab*⁴.
molecular biology: Right breakpoint at 58.5 to 61.5 kb distal to distal breakpoint of *In(3R)Cbx*^{rv1}.

***In(3LR)iab3*³³**

cytology: *In(3LR)74A;89E*.
origin: X ray induced.
discoverer: R.H. Baker.
synonym: *iab3*^{35250.33}.
genetics: Associated with *iab*³.

***In(3LR)iab3*^{Uab}:** see ***In(3LR)iab3*⁴**

***In(3LR)iab4*¹⁶⁶**

cytology: *In(3LR)79D-E;89E*.
origin: X ray induced.
discoverer: Von der Ahe.
synonym: *Mcp*^{rev31166.1}.
references: Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 432: 81-96.
genetics: Associated with *iab4*⁴.
molecular biology: DNA breakpoint at +76 to +83 kb.

***In(3LR)iab7*^{MX2}**

cytology: *In(3LR)64A;89A;89E*.
origin: X ray induced.
discoverer: Casanova.
synonym: *In(3LR)AbdB*^{MX2}.
references: Sánchez-Herrero, Vernos, Marco, and Morata, 1985, Nature (London) 313: 108-13.
 Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.
genetics: Associated with *iab7*^{MX2}. Viable over *Df(3R)P9*, showing transformation of the fifth, sixth and seventh abdominal segments to the fourth.
molecular biology: DNA breakpoint 139.5-142 kb distal to the distal breakpoint of *In(3R)Cbx*^{rv1}.

In(3LR)IC67-280

cytology: *In(3LR)66D;89E*.
origin: X ray induced.
references: Brosseau, 1969, DIS 44: 45.
genetics: Homozygous lethal.

In(3LR)IC67-356

cytology: *In(3LR)63B3-4;86E*.
origin: X ray induced.
references: Brosseau, 1969, DIS 44: 45.
genetics: Homozygous lethal.

In(3LR)IW18: Inversion (1) Inoue Watanabe

cytology: *In(3LR)62A;88C*.
origin: Spontaneous in a natural population in Japan.
references: Inoue and Watanabe, 1979, Jpn. J. Genet. 54: 69-82.

****In(3LR)K: Inversion (3LR) of Krivshenko***

cytology: *In(3LR)61C6-7;100A-B*; only the left end recovered.
new order: 100F – 100B|61C7 – 100.
origin: X ray induced in oocytes.
genetics: Result of a pericentric inversion followed by an exchange or of a translocation between 3L of one chromatid and 3R of its sister or homolog.

In(3LR)KB

cytology: *In(3LR)68C;91D*.
origin: Spontaneous in natural populations in Korea.
references: Paik, 1986, DIS 63: 167.

In(3LR)L130: see T(Y;3)L130

In(3LR)LD: Inversion (3LR) of L. DeJongh

origin: X ray induced in *bx*^{34e} and selected for bithorax transvection over *Ubx*.
discoverer: DeJongh.

inversion	cytology	ref ^α	genetics
<i>In(3LR)LD1</i> ^β	61B-C3;64C2-9;87B-C	2	homozygous viable, female sterile

inversion	cytology	ref ^α	genetics
<i>In(3LR)LD3</i>	61F7-62A2;81F5-82A1	2	homozygous viable, fertile, wild type
<i>In(3LR)LD4</i>	62;84	1	
<i>In(3LR)LD5</i> ^β	62;70A;75-77;79F;84D	1	
<i>In(3LR)LD6</i>	62A10-B1;85A2-3	2	homozygous viable fertile, wild type
<i>In(3LR)LD9</i>	64;86	1	
<i>In(3LR)LD11</i>	67;86 but complex	1	
<i>In(3LR)LD12</i>	64;81F-82A	1	
<i>In(3LR)LD13</i>	70;85	1	homozygous viable
<i>In(3LR)LD17</i>	74;85	1	
<i>In(3LR)LD19</i>	76;84	1	homozygous viable
<i>In(3LR)LD27</i>	80;81?	1	
<i>In(3LR)LD30</i>	64-65;81F	1	
<i>In(3LR)LD31</i>	67C-D;81F	1	

^α 1 = Craymer; 2 = Craymer, 1981, Genetics 99: 75-97.
^β new order:
In(3LR)LD1 = 61A – 61B|87B – 64C9|61C3 – 64C2|87C – 100F.
In(3LR)LD5 = 61A – 62|70A – 75|79F – 84D|70A – 62|84D – 100F.
 75-77 unaccounted for.

In(3LR)M6: Inversion (3LR) Multiple 6

cytology: *In(3LR)75C;94A*.
references: Craymer, 1984, DIS 60: 234-36.
other information: Inversion component of balancers *TM6*, *TM6B* and *TM6C*.

In(3LR)M54c: Inversion (3LR) Minute-54c

cytology: *In(3L)73A9-10;75D7-E1 + In(3LR)61C2-3;80C4-5;93B4-5;100B8-9*.
new order: 61A – 61C2|93B5 – 100B8|80C5 – 93B4|80C4 – 75E1|73A10 – 75D7|73A9 – 61C3|100B9 – 100F.
 Also carries an inversion with unspecified breakpoints in the region between 61C3 and 73A9.
origin: Neutron induced.
discoverer: Mickey, 54c10.
references: 1963, DIS 38: 29.
genetics: Mutant or deficient for *st* and an unidentified Minute.

In(3LR)Mg: Inversion (3LR) Mglinetz

inversion	cytology	origin	ref ^α
<i>In(3LR)Mg17</i>	63A;97A	γ ray	2
<i>In(3LR)Mg19</i>	80;98A	γ ray	2
<i>In(3LR)Mg57</i>	67D;90A	γ ray	2
<i>In(3LR)Mg59</i>	80;93D	γ ray	2
<i>In(3LR)Mg60</i>	80;93D	γ ray	2
<i>In(3LR)Mg61</i>	76D;82D	γ ray	2
<i>In(3LR)Mg66</i>	61A;96F	γ ray	3
<i>In(3LR)Mg69</i>	80;83D	γ ray	3
<i>In(3LR)Mg120</i>	78F;83D	γ ray	1
<i>In(3LR)Mg121</i>	67B;97A	32 P feeding	1
<i>In(3LR)Mg122</i>	61C;98A	32 P feeding	1
<i>In(3LR)Mg123</i>	73F;83F	32 P feeding	1
<i>In(3LR)Mg124</i>	65D;87D	32 P feeding	1
<i>In(3LR)Mg125</i>	67B;92F	32 P feeding	1
<i>In(3LR)Mg128</i>	73F;99A	32 P feeding	1
<i>In(3LR)Mg129</i>	63A;98D	32 P feeding	1
<i>In(3LR)Mg130</i>	69C;97C	32 P feeding	1
<i>In(3LR)Mg131</i>	72C;85E	32 P feeding	1
<i>In(3LR)Mg132</i>	66C;85D	32 P feeding	1
<i>In(3LR)Mg134</i> ^β	80;98A		
<i>In(3LR)Mg135</i> ^β	80;86C		
<i>In(3LR)Mg142</i> ^β	80;95A		
<i>In(3LR)Mg144</i> ^β	80;96A		

^α 1 = Mglinetz, 1968, Genetika (Moscow) 4(8): 81-86; 2 = Mglinetz, 1972, Genetika (Moscow) 8(2): 82-92; 3 = Mglinetz, 1973, Genetika (Moscow) 9(3): 69-75.

^β See Ashburner as reference.

***In(3LR)p^{XT117}*: Inversion (3LR) pink**

cytology: *In(3LR)62E;85A-B*.

origin: X ray induced.

references: Lehmann and Nüsslein-Volhard, 1987, Dev. Biol. 119: 402-07.

genetics: Mutant for *p*.

***In(3LR)P*: Inversion (3LR) Pasadena**

discoverer: E.B. Lewis.

inversion	cytology	origin ^α	ref ^β	genetics
<i>In(3LR)P3</i>	75B12-13;85D18-27	1	1	
<i>In(3LR)P10</i>	65C5-9;81 (Craymer)	1		
<i>In(3LR)P13</i>	63A;81F	1		
<i>In(3LR)P21</i>	65A;87F-88A	1		
<i>In(3LR)P30</i>	64C7-9;82A2-4	1	4	homozygous viable, fertile, wild type
<i>In(3LR)P35^Y</i>	65E;83D-E	1	1, 3, 5	
<i>In(3LR)P41</i>	64A5-7;88D6-8	1	4	homozygous viable, fertile, wild type
<i>In(3LR)P42</i>	70F1-2;81F1	1	4	homozygous viable, fertile, wild type
<i>In(3LR)P88^δ</i>	61A1-2;89C2-4 (Craymer)	1	2, 6	<i>ss^a bx⁺</i>
<i>In(3LR)P91</i>	67C10-D1;81F	2	4	homozygous lethal
<i>In(3LR)P93</i>	64B10-12;81	2	4	homozygous lethal

^α 1 = Induced by X rays; 2 = Induced by fast neutrons from nuclear detonation.

^β 1 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91; 2 = CP627; 3 = Craymer and Roy, 1980, DIS 55: 197-200; 4 = Craymer, 1981, Genetics 99: 75-97; 5 = Lewis, 1956, DIS 30: 76-77; 6 = Struhl, 1982, Genetics 102: 737-49.

^γ Induced simultaneously with, but subsequently separated from, *T(2;3)Sb^V*.

^δ Originally described as deficient for *ss*; however Struhl (1982) contends that it carries an extreme *ss^a* allele.

***In(3LR)pb¹⁶*: Inversion (3LR) proboscipedia**

cytology: *In(3LR)66B;84A4-5*.

origin: X ray induced.

references: Pultz, Diederich, Cribbs, and Kaufman, 1988, Genes Dev. 2: 901-20.

genetics: Null mutant for *pb* in the *ANTC*.

***In(3LR)pcv*: Inversion (3LR) posterior crossvein**

cytology: *In(3LR)65B-C;92A*.

origin: X ray induced.

references: Puro, 1982, DIS 58: 205-08.

genetics: Associated with *pcv*. Homozygous viable and fertile.

In(3LR)R92

cytology: *In(3LR)75C-D;85D-E*.

discoverer: E.B. Lewis.

references: Merriam and García-Bellido, 1972, Mol. Gen. Genet. 115: 302-13.

***In(3LR)S1*: Inversion (3LR) Segal**

cytology: *In(3LR)71B6-C1;97A1-2*.

origin: γ ray induced with *T(2;3)shv^{S1}*.

references: Segal and Gelbart, 1985, Genetics 109: 119-43.

In(3LR)S24

cytology: *In(3LR)63B;85D*.

origin: γ ray induced with *T(2;3)shv^{S24}*.

references: Segal and Gelbart, 1985, Genetics 109: 119-43.

***In(3LR)sep*: Inversion (3LR) separated**

cytology: *In(3LR)65D2-3;85F2-4* (Craymer, 1981). 85F puff associated with left end of inversion.

discoverer: Muller.

references: Lewis, 1951, DIS 25: 108-09.

Craymer, 1981, Genetics 99: 75-97.

genetics: Mutant for *sep*. Also carries *ri* and *p^P*, which can be removed only with great difficulty.

***In(3LR)SMG21*: Inversion (3LR)**

Seminova Mglinetz Glotoff

cytology: *In(3LR)65A;95E*.

origin: γ ray induced.

references: Semenova, Mglinetz, and Glotoff, 1970, Genetika (Moscow) 6(4): 165-69.

In(3LR)SMG32

cytology: *In(3LR)64F;100F*.

origin: γ ray induced.

references: Semenova, Mglinetz, and Glotoff, 1970, Genetika (Moscow) 6(4): 165-69.

***In(3LR)st^{a27}*: Inversion (3LR) scarlet**

cytology: *In(3LR)73A3-4;87D13-14*.

origin: X ray induced.

references: McKeown, Belote, and Baker, 1987, Cell 48: 489-99.

discoverer: Belote and McKeown.

genetics: Mutant for *st*.

molecular biology: Left-most breakpoint is coordinate 0 of the walk in region 73AB of McKeown and Belote ("+" values to the right, "-" values to the left).

***In(3LR)St*: Inversion (3LR) Stalker**

cytology: *In(3LR)77D;88D*.

origin: Spontaneous in a natural population.

references: Stalker, 1976, Genetics 82: 323-47.

***In(3LR)Sta*: Inversion (3LR) Stigmata**

cytology: *In(3LR)79D;94A* (Lewis).

origin: X ray induced in *Tp(3;2)P10*.

discoverer: E.B. Lewis, 1978.

references: Craymer, 1981, Genetics 99: 75-97. 1984, DIS 60: 234-36.

genetics: Associated with *Sta*. Homozygote occasionally survives. Gelbart, 1971, Ph.D. Thesis, Univ. of Wisconsin.

***In(3LR)TI^{rv18}*: Inversion (3LR) Toll-revertant**

cytology: *IN(3LR)74A;97D*.

origin: X ray induced.

synonym: *In(3LR)TI^{5BRXV}*.

references: Anderson, Jürgens, and Nüsslein-Volhard, 1985, Cell 42: 779-89.

genetics: Revertants of the female sterility of *Tl*; trans heterozygotes viable.

***In(3LR)TM1*: Inversion (3LR) Third Multiple**

cytology: *In(3L)63C;72E1-2 + In(3LR)69E;91C + In(3R)89B;97D*.

new order:

61 – 63C|72E1 – 69E|91C – 97D|89B – 72E2|
63C – 69E|91C – 89B|97D – 100.

origin: Derived from *T(2;3)Melri*, presumably by a double crossover with exchanges in regions 72E2-80 and 81-89B, which replaced the *T(2;3)* breakpoint in 3 with *ri*.

discoverer: E. B. Lewis.

references: 1949, DIS 23: 92.
1953, DIS 27: 58.

genetics: Carries *Me*, *ri*, and *sbd*¹.

other information: Used as a balancer for chromosome 3, described as *TM1* in the section on balancers.

In(3LR)TM3

cytology: *In(3LR)71C;94D-F + In(3LR)76C;93A + In(3LR)79E;100C* superimposed on 3^{PX^D} from *T(1;3)IA8-B1;61A1-2 + In(3LR)65E;85E + In(3R)92D1-E1;100F2-3*.

new order:

1A1 – 1A8|61A2 – 65E|85E – 79E|100C – 100F2|
92D1 – 85E|65E – 71C|94D – 93A|76C – 71C|
94F – 100C|79E – 76C|93A – 92E1|100F3 – 100F5.

origin: Induced by repeated irradiation of the 3^{PX^D} element of *T(1;3)sc*²⁶⁰⁻²⁰, which carried *In(3LR)sep + In(3R)C, y⁺ ri p sep bx*^{34e e^s}.

discoverer: E. B. Lewis.

references: Mitchell, 1958, Cold Spring Harbor Symp. Quant. Biol. 23: 279-90.
Lewis, 1960, DIS 34: 51.

other information: Used as a balancer for chromosome 3, described as *TM3* in the section on balancers.

In(3LR)TM6

cytology: *In(3LR)M6 = In(3LR)75C;94A* superimposed on *In(3L)P = In(3L)63B8-11;72E1-2 + In(3LR)P88 = In(3LR)61A1-2;89C2-4 + In(3R)C = In(3L)92D1-E1;100F2-3*.

new order:

61A1|89C2 – 75C|94A – 100F2|92D1 – 89C4|
61A2 – 63B8|72E1 – 63B11|72E2 – 75C|
94A – 92E1|100F3 – 100F5.

origin: X ray induced.

discoverer: E. B. Lewis and F. Bacher, 66i.

genetics: Homozygous lethal. Deficiency for *ss* but not *bx* associated with *In(3LR)P88*.

other information: Used as a balancer for chromosome 3. Described as *TM6* in section on balancers.

In(3LR)TM6B

cytology: *In(3LR)M6 = In(3LR)75C;94A* superimposed on *In(3L)P = In(3L)63B8-11;72E1-2 + In(3LR)HR33 = In(3LR)61A1-2;87B2-4 + In(3R)Hu = In(3R)84B1-2;84F4;86C7-8 + In(3R)C = In(3R)92D1-E1;100F2-3*.

new order:

61A1|87B2 – 86C6|84F1 – 85C5|84B6 – 84F1|
84B3 – 75C|94A – 100F2|92D1 – 87B4|61A2 – 63B8|
72E1 – 63B11|72E2 – 75C|94A – 92E1|100F3 – 100F5.

references: Craymer, 1984, DIS 60: 234-36.

genetics: Resembles *In(3LR)TM6* except for substitution of *In(3LR)HR33* for *In(3LR)P88* and addition of *Hu*. Usually carries *D*³ or *Tb* with combinations of *e*, *ca*, *h* and *Hn*^P.

other information: Used as balancer for chromosome 3. Described as *TM6B* in section on balancers.

In(3LR)TM6C

cytology: Same as *In(3LR)TM6B* except for absence of *In(3LR)Hu = In(3R)84B1-2;84F4;86C7-8*.

references: Craymer, 1984, DIS 60: 234-36.

genetics: Homozygotes viable but with development delayed. Stocks may carry *cu* and *Sb* in addition to the *TM6B* markers.

other information: Used as balancer for chromosome 3 [less effective for centromere region than *In(3LR)TM6B*]. Described as *TM6C* in section on balancers.

In(3LR)TM8

origin: X ray induced in *In(3L)C90 In(3R)C/+* males carrying *l(3)DTS4* in 3L.

references: Marsh, 1978, DIS 53: 155-56.

genetics: Temperature-sensitive dominant lethal, showing very few viable and fertile escapers from the DTS phenotype.

other information: Used as a dominant temperature-sensitive balancer for chromosome 3. Described as *TM8* in the section on balancers.

In(3LR)TM9

origin: X ray induced in *In(3L)C90 In(3R)C/+* males carrying *l(3)DTS4* in 3L.

references: Marsh, 1978, DIS 53: 155-56.

genetics: Temperature-sensitive dominant lethal, showing very few viable and fertile escapers from the DTS phenotype.

other information: Used as a dominant temperature-sensitive balancer for chromosome 3. Described as *TM9* in the section on balancers.

*In(3LR)Uab*⁴: see *In(3LR)iab3*⁴

In(3LR)Ubx: Inversion (3LR) Ultrabithorax

origin: X ray induced.

inversion	cytology	discov	ref ^α	genetics
<i>In(3LR)Ubx</i> ^{16Rβ}	79D;89B (E.B. Lewis)	Ramey	2	<i>Ubx</i>
<i>In(3LR)Ubx</i> ^{42T}	70D;89E		1	<i>Ubx</i>
<i>In(3LR)Ubx</i> ^{101γ}	80;89D9-E1	E.B. Lewis	3	<i>Ubx</i>
<i>In(3LR)Ubx</i> ^{130δ}	61A-C;74;89D-E; 93B;96A	E.B. Lewis	3	<i>Ubx</i> ; homoz. lethal
<i>In(3LR)Ubx</i> ¹⁹⁶	80;89E;90D1	R.H. Baker		<i>Ubx</i>
<i>In(3LR)Ubx</i> ^A	see <i>T(3;4)Ubx</i> ^A	Schalet		<i>Ubx</i>
<i>In(3LR)Ubx</i> ^{R42}	70D;89E	Ramey		<i>Ubx</i>
<i>In(3LR)Ubx</i> ^{Uε}	62A2-3;89E1-2	Bacher	2	extreme <i>Ubx</i>

^α 1 = Craymer; 2 = Craymer, 1981, Genetics 99: 75-97; 3 = CP627.

^β Synonym: *In(3LR)Cbx*^{rvR17.16R}.

^γ Synonym: *In(3LR)Bxl*¹⁰¹ (Lewis, 1949, DIS 23: 59).

^δ New order: 61A|96B-93B|89D-74|61C-74|89E-93B|96A-100.

^ε Synonym: *In(3LR)Ubx*³⁰⁰; *In(3LR)Ubx*^{P300}. Homozygote viable with *Ubx*⁺ duplication and has slightly altered venation pattern.

In(3LR)V: Inversion (3LR) Valencia

origin: X ray induced.

references: Valencia, 1970, DIS 45: 37-38.

inversion	cytology	genetics
<i>In(3LR)V5-25</i>	69C;92E-F	homozygous viable and fertile
<i>In(3LR)V9-10</i>	66D;99F	homozygous lethal
<i>In(3LR)V10-7</i>	80C;97A-B	low viability in homozygotes; male sterile
<i>In(3LR)V12-1</i>	68D-E;94E	homozygous lethal

***In(3LR)W^{rv8}*: Inversion (3LR)
Wrinkled-revertant****cytology:** *In(3LR)75C3-D2;86D1-2*.**origin:** X ray induced.**synonym:** *In(3LR)W^{+R8}*.**references:** Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.**genetics:** Revertant of *W*.***In(3LR)Z2*****cytology:** *In(3LR)67E;98F*.**origin:** Spontaneous in a natural population in Greece.**references:** Zacharopoulou, 1974, DIS 51: 52-53.Zacharopoulou and Pelecanos, 1980, *Genetica* 54: 105-11.***In(3LR)Z3*****cytology:** *In(3LR)71F;90D*.**origin:** Spontaneous in a natural population in Greece.**references:** Zacharopoulou, 1974, DIS 51: 52-53.Zacharopoulou and Pelecanos, 1980, *Genetica* 54: 105-11.***In(3R)221*****cytology:** *In(3R)82F;96* (E.B. Lewis).**origin:** X ray induced in Canton-S.**discoverer:** S. Ou.**references:** Craymer, 1984, DIS 60: 234-36.**genetics:** Associated with, but separable from, *T(2;3)221*.****In(3R)300.96*****cytology:** *In(3R)89F2-90A1,99B2-4*.**origin:** X ray induced simultaneously with *e^{300.96}*.**discoverer:** Alexander.**references:** Ward and Alexander, 1957, *Genetics* 42: 42-54.**genetics:** Carries an independent mutant for *e*. Homozygous viable but male sterile.***In(3R)429.10*: see *T(3;4)429.10******In(3R)429.18*: see *T(2;3;4)429.18******In(3R)1000*****cytology:** *In(3R)81F;90C-D*.**references:** E.B. Lewis.***In(3R)A60*: see *T(Y;3)A60******In(3R)A7132*****cytology:** *In(3R)96C3-9;96E5-12;97A3-4*.**new order:**

61 – 96C3|96E5 – 96C9|97A4 – 100

(deficient between 96E5 and 97A4).

origin: X ray induced reversion of *E(spl)*.**discoverer:** Knust and Ziemer.**genetics:** Deficient for *E(spl)*.***In(3R)AFA*****cytology:** *In(3R)86C4-5;93D6-7* (Scalenghe and Ritossa, 1976).**origin:** X ray induced.**references:** Scalenghe and Ritossa, 1976, *Atti. Accad. Naz. Lincei* 13: 439-528.

D'Alessandro, Ritossa, and Scalenghe, 1977, DIS

52: 46.

Caggese, Caizze, Morea, Scalenghe, and Ritossa, 1979, *Proc. Nat. Acad. Sci. USA* 76: 2385-89.**genetics:** Homozygous viable.***In(3R)AL: Inversion (3R)
Ashburner Lemeunier*****origin:** Naturally occurring inversions.**references:** Ashburner and Lemeunier, 1976, *Proc. R. Soc. London, B* 193: 137-57.

inversion cytology

In(3R)AL13 82A;87C*In(3R)AL14* 84E;99E*In(3R)AL15* 85E;91A*In(3R)AL16* 88E;93F*In(3R)AL17* 92E;97E***In(3R)Antp^{73b}*****cytology:** *In(3R)84B1-2;84C5-6* (Hazelrigg and Kaufman, 1983). Cytology difficult in *In(3R)Antp^{73b}/+*.**origin:** Spontaneous.**discoverer:** Green.**references:** Kaufman, Lewis, and Wakimoto, 1980, *Genetics* 94: 115-33.Garber, Kuroiwa, and Gehring, 1983, *EMBO J.*

2: 2027-36.

Hazelrigg and Kaufman, 1983, *Genetics* 105: 581-600.Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, *Cell* 35: 763-76.Cavener, Corbett, Cox, and Whetton, 1986, *EMBO J.*

5: 2939-48.

genetics: Mutant for *Antp*. Homozygous lethal and lethal with *Df(3R)Antp17* and *Df(3R)Scr* (Kaufman *et al.*, 1980).**molecular biology:** Proximal breakpoint about 80 kilobases to the left of the proximal breakpoint of *In(3R)Hu* on the DNA map of Garber *et al.*, 1983, and 178-182 kilobases to the right of the proximal breakpoint of *Tp(3;3)Dfd^{rvX16}* on the DNA map of Scott *et al.*, 1983 ("+" values to left in walk of Garber *et al.* and to right in walk of Scott *et al.*). Distal breakpoint at about +56 kb on the molecular map of Cavener *et al.*, 1986. *Antp* transcripts found in head, some of them being fusion RNAs between *Antp* and *l(3)84Cd*, a gene normally expressed in the head (Frischer, Hagen, and Garber, 1986, *Cell* 47: 1017-23).***In(3R)Antp^{a74}*****cytology:** *In(3R)84B1-2;87C*.**references:** Abbott and Kaufman, 1983, *Genetics* 104: s1. Bender, Turner, and Kaufman, 1987, *Dev. Biol.*

119: 418-32.

genetics: Mutant for *Antp*.***In(3R)Antp^B: Inversion (3R)
Antennapedia of Bacon*****cytology:** *In(3R)84B1-2;85E*.**origin:** X ray induced.**discoverer:** Bacon, 50g.**references:** Lewis, 1956, DIS 30: 76.Kaufman, Lewis, and Wakimoto, 1980, *Genetics* 84: 115-33.Garber, Kuroiwa, and Gehring, 1983, *EMBO J.*

2: 2027-36.

Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe,

and Kaufman, 1983, Cell 35: 763-76.

genetics: Mutant for *Antp*.

molecular biology: Left breakpoint about 100 kilobases to the left of the proximal breakpoint of *In(3R)Hu* on DNA map of Garber *et al.*, 1983, or 138 kilobases distal to the left-most breakpoint of *Tp(3;3)Dfd^{rvX16}* (Scott *et al.*, 1983).

In(3R)Antp^{BL dsx^{Drv3R}}

cytology: *In(3R)84B1-2;85E^L;84E1-2;85D25-E1^R*; deficient for 84B2-E1 and duplicated for 85D25-E.

origin: Recombination between *In(3R)Antp^B* and *In(3R)dsx^{Drv3}*.

synonym: *In(3R)Antp^{BL dsx^{D+R3R}}*

references: Kaufman, Lewis, and Wakimoto, 1980, Genetics 94: 115-33.

genetics: Mutant for *Antp* and *roe*.

In(3R)Antp^{CB}

cytology: *In(3R)84B1-2;99F-100A*.

origin: X ray induced.

discoverer: Black.

references: Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, Cell 35: 763-76.

genetics: Mutant for *Antp*. Homozygous lethal.

molecular biology: Left breakpoint 198 kilobases distal to the proximal breakpoint of *Tp(3;3)Dfd^{rvX16}* (Scott *et al.*, 1983).

In(3R)Antp^{LC}: Inversion (3R)

Antennapedia of Le Calvez

cytology: *In(3R)84B1-2;91F-92A* (Garber *et al.*, 1983).

origin: Neutron induced.

discoverer: Le Calvez.

references: 1948, Bull. Biol. France Belg. 82: 97-113 (fig.).

Garber, Kuroiwa, and Gehring, 1983, EMBO J. 2: 2027-36.

genetics: Associated with *Antp^{LC}*.

molecular biology: Left breakpoint about 100 kilobases proximal to proximal breakpoint of *In(3R)Hu* (Garber *et al.*, 1983).

In(3R)Antp^R: Inversion (3R)

Antennapedia of Rappaport

cytology: *In(3R)84B1-2;85F* (Garber *et al.*, 1983).

origin: X ray induced.

discoverer: Rappaport, 1963.

references: Falk, 1964, DIS 39: 60.

Denell, 1973, Genetics 75: 279-97.

Lewis, Kaufman, Denell and Talerico, 1980, Genetics 95: 367-81.

Garber, Kuroiwa, and Gehring, 1983, EMBO J. 2: 2027-36.

genetics: Associated with *Antp^R*.

molecular biology: Left breakpoint about 100 kilobases to the left of left breakpoint of *In(3R)Hu* (Garber *et al.*, 1983).

In(3R)Antp^{r-A74}: Inversion (3R)

Antennapedia-recessive

cytology: *In(3R)84B2;84C*.

origin: X ray induced.

synonym: *In(3R)antp^{A74}*.

references: Abbott and Kaufman, 1986, Genetics 114: 919-42.

genetics: Recessive lethal with dominant phenotype.

In(3R)Antp^{r-s2}

cytology: *In(3R)80;84B1-2*. [Either *In(3LR)* or proximal breakpoint in 81F?]

origin: X ray induced.

synonym: *In(3R)antp^{s2}*.

references: Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, Cell 35: 763-76.

Abbott and Kaufman, 1986, Genetics 114: 919-42.

genetics: Recessive lethal without dominant phenotype.

molecular biology: Right breakpoint 170 – 174 kb to the right of the left-most breakpoint of *Tp(3;3)Dfd^{rvX16}* (Scott *et al.*, 1983).

In(3R)Antp^{RM}

cytology: *In(3R)82E1;84B1-2*.

origin: X ray induced.

references: Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, Cell 35: 763-76.

genetics: Mutant for *Antp*. Homozygous lethal.

molecular biology: Right breakpoint 167-170 kb to the right of the left-most breakpoint of *Tp(3;3)Dfd^{rvX16}* (Scott *et al.*, 1983).

In(3R)Antp^{rv}: Inversion (3R)

Antennapedia-revertant

origin: X ray induced except for *In(3R)Antp^{rv1P}*, which is spontaneous.

genetics: Revertant of *Antp*. Homozygous lethal.

inversion	cytology	synonym	ref ^α
<i>In(3R)Antp^{rv1β}</i>	81F;90B-C	<i>Antp^{Ns+R1}</i>	5
<i>In(3R)Antp^{rv1Pγ}</i>	84B;84D + <i>Tp(3;3)</i>	<i>Antp^{+R1}</i>	5, 7
<i>In(3R)Antp^{rv2δ}</i>	84B1-2;84D1-2	<i>Antp^{+R2}</i>	5, 7
<i>In(3R)Antp^{rv5}</i>	see <i>T(2;-3)Antp^{rv5}</i>	<i>Antp^{73b+RX5}</i>	6
<i>In(3R)Antp^{rv9}</i>	84B1-2;84D1-2	<i>Antp^{73b+RX9}</i>	6
<i>In(3R)Antp^{rv25ε}</i>	81;84B1-2;85A	<i>Antp^{Ns+R25}</i>	1, 2, 3, 7, 8
<i>In(3R)Antp^{rv72ζ}</i>	84B2-6;84D5-8 (B.S. Baker)	<i>Antp^{Ns+R72}</i>	1, 2, 3, 4, 7, 8
<i>In(3R)Antp^{rv73b}</i>	84B2-6;84C8;85C3-8 deficient for 84B6-C8.	<i>Antp^{Ns+R73}</i>	
<i>*In(3R)Antp^{rv85}</i>	81;84B1-2	<i>Antp^{Ns+R85}</i>	3
<i>In(3R)Antp^{rv96}</i>	see <i>T(Y;3)Antp^{rv96}</i>		

^α 1 = Denell, 1972, DIS 48: 45; 2 = Denell, 1972, Mutat. Res. 15: 221-23; 3 = Denell, 1973, Genetics 75: 279-97; 4 = Denell, 1978, Genetics 90: 277-89; 5 = Duncan and Kaufman, 1975, Genetics 80: 733-52; 6 = Hazelrigg and Kaufman, 1983, Genetics 105: 581-600; 7 = Lewis, Kaufman, Denell, and Talerico, 1980, Genetics 95: 367-81. 8 = Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, Cell 35: 763-76.

^β Partial revertant. Discoverer: Duncan and Kaufman.

^γ Part of the inverted material transposed to 87B distal to *cu*. Discoverer: M.M. Green.

^δ Listed as deficiency in references; later found to be an inversion (Kaufman).

^ε 84B breakpoint 120-128 kilobases distal to the proximal breakpoint of *Tp(3;3)Dfd^{rvX16}* on molecular map of Scott *et al.*, 1983.

^ζ Originally described as a deficiency (Denell, 1972, 1973).

In(3R)Antp^{Scx}: see *In(3R)Scx*

In(3R)ats: Inversion (3R) abnormal tarsi

cytology: *In(3R)89C;96A21-25*.

references: Sierra and Comendador, 1984, DIS 60: 244-45.

genetics: Abnormalities of tarsi. Males and females sterile. Viability reduced.

In(3R)B

origin: X ray induced along with *T(Y;3)B*.
references: The Seattle-La Jolla Drosophila Laboratories, 1971, DIS 47, Suppl. Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84.

inversion	breakpoints
<i>In(3R)B172</i>	93B-C;99A
<i>In(3R)B229</i>	94E-F;97C-D

In(3R)BTD7

cytology: *In(3R)87A;92B + In(3LR)76A;87A*.
references: Smolnik-Utlaut and Gelbart, 1987, Genetics 116: 285-98.
genetics: Disrupts *BXC* transvection effects.

***In(3R)bx^d*¹⁸³: Inversion (3R) bithoraxoid**

cytology: *In(3R)89C;89E*.
origin: Induced by ethyl methanesulfonate.
discoverer: E.B. Lewis.
synonym: *bx^d*^{17758.83}.
genetics: Mutant for *bx^d*.

In(3R)C: Inversion (3R)

Crossover suppressor

cytology: *In(3R)92D1-E1;100F2-3* (Bridges and Li in Morgan, Bridges, and Schultz, 1937, Year Book - Carnegie Inst. Washington 36: 301).
origin: Naturally occurring inversion.
discoverer: Sturtevant, 13f.
synonym: *C3, CIIIIE; In(3R)E; In(3R)H*.
references: 1913, Science 37: 990-92. 1917, Proc. Nat. Acad. Sci. USA 3: 555-58. 1926, Biol. Zentr. 46: 697-702. 1931, Carnegie Inst. Washington Publ. No. 421: 1-27. Muller, 1918, Genetics 3: 422-99.
genetics: Homozygous viable. Crossing over in *3R* reduced to 1% between centromere and *ss*, to 0.2% between *ss* and *e*; no crossovers between *e* and tip of *3R* recovered except for rare doubles within inversion.
other information: First inversion demonstrated genetically (Sturtevant, 1926). Used as a balancer for the region from *D1* to *3R* tip. Balancers contain *Sb, e, l(3)a*, or *l(3)e*. Balancer for all of chromosome 3 made by combining with *In(3L)P*. Found in wild populations (e.g., Oshima and Watanabe, 1965, DIS 40: 88; Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57; Stalker, 1976, Genetics 82: 323-47; Choi, 1977, DIS 52: 88; 1977, Genetica 47: 155-60).

In(3R)C13 - In(3R)C289

origin: X ray induced.

inversion	cytology	ref ^α
<i>In(3R)C13</i> ^β	94E;98D	2
<i>In(3R)C41</i>	81E;91E-F	1
<i>In(3R)C67-79</i>	85F;97F	1
<i>In(3R)C67-327</i>	82D;98E	1
<i>In(3R)C133</i>	93F;97C-D	3
<i>In(3R)C208</i>	91B;96B	3
<i>In(3R)C289</i>	81F;93E	1

^α 1 = Ashburner; 2 = Morrison, 1972, DIS 49: 37; 3 = Roberts, 1970, Genetics 65: 429-48.

^β Reduces crossing over between *Pr* and *Dr* to < 0.1%.

In(3R)ca^V*: see *T(2;3)ca^V

In(3R)CA

origin: γ ray induced.
discoverer: Ashburner.

inversion	cytology
<i>In(3R)CA23</i> ^α	81;98C
<i>In(3R)CA33</i> ^α	81;98C
<i>In(3R)CA35</i>	92;97
<i>In(3R)CA37</i>	81;84E
<i>In(3R)CA52</i>	81;99B

^α same?

In(3R)Camel*: see *In(3R)iab6^G

In(3R)Cbx²: Inversion (3R) Contrabithorax

cytology: *In(3R)89E;91C-E*.
discoverer: Kreber.

In(3R)Cbx³: Inversion (3R) Contrabithorax-like

cytology: *In(3R)89A;89E1-2*.
origin: X ray induced.
discoverer: Akam.
references: Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, Science 221: 23-29.
genetics: Associated with *Cbx³*. Viable over *BXC* deficiencies.
molecular biology: Right breakpoint between 103 and 110 kb to the left of the right breakpoint of *In(3R)Cbx^{rv1}*.

In(3R)Cbx^{rv1}

cytology: *In(3R)87E1-2;89E1-2*.
origin: X ray induced in a *Cbx* chromosome.
discoverer: Kaufman.
synonym: *In(3R)Cbx^{+R1}*.
references: Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983a, Science 221: 23-29. Bender, Spierer, and Hogness, 1983b, J. Mol. Biol. 168: 17-33.
genetics: Reversion of *Cbx*; *Ubx* phenotype.
molecular biology: The 89E1-2 (right or distal) breakpoint is the origin (coordinate 0) of the *BXC* walk of Bender *et al.*, 1983a; "+" values to the right, "-" values to the left. The origin of the *ry Ace* walk is approximately 6.5 kilobases distal to the 87E breakpoint (Bender *et al.*, 1983b).

In(3R)Cbx^{Twt}

cytology: *In(3R)87E-F;89E1-2*.
origin: X ray induced.
discoverer: Abbott.
references: Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, Science 221: 23-29.
genetics: Associated with *Cbx^{Twt}*. Viable over *BXC* deficiencies.
molecular biology: Right breakpoint between 103 and 110 kb to the left of the right breakpoint of *In(3R)Cbx^{rv1}*.

In(3R)CS: Inversion (3R) Canton-S

cytology: *In(3R)93C-D;99A*.
origin: Spontaneous in Canton-S stock.
references: Sequeira, Nelson, and Szauter, 1989, Genetics 123: 511-24.
genetics: Distal breakpoint alters banding of *ca^{nd4}*/+ in salivaries.

In(3R)cu^{5J}

cytology: *In(3R)84F3-10;86D6-10* (B.S. Baker).
origin: X ray induced.
discoverer: Holden.
references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou and Walker, 1981, DIS 56: 186-91.
genetics: Mutant for *cu*.

In(3R)CW: Inversion (3R) C of Warters

cytology: *In(3R)86B;92F*.
origin: Spontaneous in a natural population.
references: Warters, 1944, Univ. Texas Publ. 4445: 129-74.
 Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

In(3R)Dipr: Inversion (3R) Distal into proximal

cytology: *In(3R)84D;84F*.
origin: X ray induced.
references: Kerridge, 1981, Mol. Gen. Genet. 184: 319-23.
genetics: Associated with *rn^D*.

In(3R)DI^B: Inversion (3R) Delta-Barish

cytology: *In(3R)91B1-2;92A1-2* (Lehmann and Vässin).
discoverer: Schultz, 1933.
references: Vässin and Campos-Ortega, 1987, Genetics 16: 433-45.
genetics: Mutant for *DI*.

In(3R)DI^{KX15}

cytology: *In(3R)89A;92A1-2*.
origin: X ray induced.
discoverer: Vässin.
references: Vässin and Campos-Ortega, 1987, Genetics 16: 433-45.
genetics: Associated with *DI*.

In(3R)DI^{Sp}

cytology: *In(3R)92A1-2;92A12-13*.
origin: Spontaneous.
discoverer: Knust.
references: Vässin and Campos-Ortega, 1987, Genetics 16: 433-45.
genetics: Associated with *DI*.

In(3R)dsx^{Drv}: Inversion (3R) doublesex-Dominant revertant

origin: X ray induced in *dsx^D*.
genetics: Revertant of *dsx^D*.

inversion	cytology ^α	synonym	ref ^β
<i>In(3R)dsx²³</i>	84E1-2;85D22-E1	<i>In(3R)dsx^{D+R3}</i>	1,2
<i>In(3R)dsx²⁶</i>	84E1-2;92C3-6	<i>In(3R)dsx^{D+R7}</i>	1
<i>In(3R)dsx²⁷γ</i>	84E1-2;92C3-6	<i>In(3R)dsx^{D+R9}</i>	1

^α Breakpoints revised by B.S. Baker.
^β 1 = Baker, Hoff, Kaufman, Wolfner, and Hazelrigg, 1991, Genetics 127: 125-38; 2 = Duncan and Kaufman, 1975, Genetics 80: 733-52.
^γ Not distinguishable cytologically from *In(3R)dsx^{Drv7}*.

In(3R)dsx^{Mrv}

origin: X ray induced in *dsx^M*.
discoverer: Baker and Hoff.

genetics: Revertant of *dsx^M*.

inversion	cytology	synonym
<i>In(3R)dsx³¹ α</i>	84D9-10 (distal edge); 84E1-2 (proximal edge)	<i>In(3R)dsx^{Mas+R4}</i>
<i>In(3R)dsx³⁵ α</i>	84A;84E1-2	<i>In(3R)dsx^{Mas+R18}</i>
<i>In(3R)dsx⁴⁸</i>	83D5-E1 (interband); 85A3-4 (interband) ^β	<i>In(3R)dsx^{Mas+R48}</i>

^α Baker, Hoff, Kaufman, Wolfner, and Hazelrigg, 1991, Genetics 127: 125-38.
^β Deficient for 84D-85A3.

In(3R)e: Inversion (3R) ebony

genetics: Mutant for *e*. Homozygous viable.

inversion	cytology	origin	ref ^α
<i>In(3R)e^{100.25}</i>	<i>In(3R)90B1-2;92A1-2</i>	X ray	3
<i>In(3R)e^{D12}</i>	<i>In(3R)92E12-13;93D6-7</i>	X ray	1
<i>In(3R)e^{H3}</i>	<i>In(3R)81;93D2-3</i>	X ray	2
<i>In(3R)e^{N24}</i>	<i>In(3R)90F;93D</i>		

^α 1 = D'Alessandro, Ritossa, and Scalenghe, 1977, DIS 52: 46; 2 = Henikoff, 1980, DIS 55: 61-62; 3 = Ward and Alexander, 1957, Genetics 42: 42-54.

In(3R)E: see *In(3R)C*

In(3R)E(spl)^{rv1}

cytology: Breakpoint in 96E-F in common with that of *In(3R)E(spl)^{rv2}*.
synonym: *In(3R)E(spl)^{+R1}*.
references: Lehmann, Jiménez, Dietrich, and Campos-Ortega, 1983, Wilhelm Roux's Arch. Dev. Biol. 192: 62-74.
genetics: Revertant of *E(spl)*.

In(3R)E(spl)^{rv2}

cytology: Breakpoint in 96E-F in common with that of *In(3R)E(spl)^{rv1}*.
synonym: *In(3R)E(spl)^{+R2}*.
references: Lehmann, Jiménez, Dietrich, and Campos-Ortega, 1983, Wilhelm Roux's Arch. Dev. Biol. 192: 62-74.
genetics: Revertant of *E(spl)*.

In(3R)EJ

cytology: *In(3R)85A4;89A10*.
origin: Spontaneous in a natural population.
discoverer: Vallasso.

In(3R)GC

origin: γ ray induced.

inversion	cytology	ref ^α	genetics
<i>In(3R)GC18</i>	81F;93C	2	
<i>In(3R)GC21</i>	91A;93C	2	
<i>In(3R)GC23^β</i>	81F;93D	1, 2	homo- and hemizygous viable with rough eyes
<i>In(3R)GC25</i>	93E;94A	2	

^α 1 = Mohler and Pardue, 1982, Chromosoma 86: 457-67; 2 = Mohler and Pardue, 1984, Genetics 106: 249-65.
^β Chromosome does not usually exhibit 93D puff. Transcription and *in situ* hybridization of 93D sequences occurs outside the inversion.

In(3R)hb^{D1}: Inversion (3R) hunchback-Dominant

cytology: *In(3R)85A;88C-D* (B.S. Baker).
origin: Induced by ethyl methanesulfonate.
discoverer: Bacher.
synonym: *In(3R)Rg-pbx*, *Inversion (3R) Regulator of Post-bithorax* (E.B. Lewis).

references: Lewis, 1968, Proc. XII Int. Congr. Genet. 2: 96-97.
Kiger, 1973, J. Theoret. Biol. 40: 455-67.
García-Bellido and Lewis, 1976, Dev. Biol. 45: 400-10.
Bender, Turner, and Kaufman, 1987, Dev. Biol. 119: 418-32.
genetics: Associated with *hb^D* (= *Rg-pbx*) which shows a variable transformation of the halteres to wing tissue. Homozygous lethal.

In(3R)hb^{D2}

cytology: *In(3R)84B;85A*.
origin: γ ray induced.
discoverer: Posakony.
genetics: Homozygous lethal. Associated with *hb^{D2}*. Lethal over *hb*; a few escapers.

In(3R)hp: see *In(3R)P*

In(3R)HR13

cytology: *In(3R)81;98E*.
references: Ashburner, 1972, DIS 49: 34.

In(3R)Hu: Inversion (3R) Humeral

cytology: Double inversion - *In(3R)84B3-6;84F1-2;86C5-6* (B.S. Baker) or *84B1-2;84F4;86C7-8* (Hazelrigg and Kaufman, 1983).

new order:

61A – 84B3|84F1 – 84B6|
86C5 – 84F2|86C6 – 100 (B.S. Baker).

origin: X ray induced.

discoverer: Ruch, 1931.

references: Kaufman, Lewis, and Wakimoto, 1980, Genetics 94: 115-33.

Garber, Kuroiwa, and Gehring, 1983, EMBO J. 2: 2027-36.

Hazelrigg and Kaufman, 1983, Genetics 105: 581-600.

Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, Cell 35: 763-76.

Kuroiwa, Hafen, and Gehring, 1984, Cell 37: 825-31.

genetics: Associated with *Hu*. Homozygotes survive (but at low frequency) until third instar (Hazelrigg and Kaufman, 1983).

molecular biology: Left-most (proximal) breakpoint is the point of initiation (coordinate 0) of the walk of Garber *et al.*, 1983 ("+" values to the left, "-" values to the right) which is about 255 kilobases distal to the 0 coordinate of Scott *et al.*, 1983 ("+" values to the right, "-" values to the left).

In(3R)iab2^{S3}

cytology: *In(3R)89E;93F*.

origin: γ ray induced.

discoverer: Tiong.

synonym: *In(3R)abda^{S3}*.

references: Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.

Tiong, Bone, and Whittle, 1985, Mol. Gen. Genet. 200: 335-42.

genetics: Associated with *iab2*. Homozygous viable and almost wild type; causes partial transformation of second abdominal segment into the first in hemizygotes.

molecular biology: Left breakpoint between 24 and 28 kilobases to the right of the right breakpoint of *In(3R)Cbx^{rv1}*.

In(3R)iab4¹⁶⁶

cytology: *In(3R)79D-E;89E*.

origin: X ray induced in *Mcp*.

discoverer: Von Der Ahe.

synonym: *Mcp^{rv31166.1}*.

references: Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.

genetics: Associated with *iab4*. *In(3R)iab4/Df(3R)P9* adults show fourth abdominal segment transformed into third; also lack gonads.

molecular biology: Right breakpoint 76-83 kb distal to the right breakpoint of *In(3R)Cbx^{rv1}*.

In(3R)iab6^G

cytology: *In(3R)89B;89E3-4*.

origin: X ray induced.

discoverer: Gausz.

synonym: *Camel*.

references: Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.

genetics: Associated with *iab6^G*. Heterozygotes have longitudinal furrow on the notum; also a dominant partial *iab5* phenotype. *iab6^G/+* males lack pigment on fifth tergite and have some bristles on the sixth sternite. *iab6^G/Df* shows transformations of the fifth, sixth, and seventh segments toward the fourth and a more extensive longitudinal furrow, extending into the abdomen and causing a fusion problem in the tergites.

molecular biology: Right breakpoint 113-121 kilobases to the right of the right breakpoint of *In(3R)Cbx^{rv1}*.

In(3LR)iab6^{Spth}*: see *In(3LR)iab7^{Spth}

In(3R)iab7^{SGA}

cytology: *In(3R)88C4-7;89E1-2*.

origin: X ray induced.

discoverer: Gyurkovics.

synonym: *SGA62*.

references: Awad, Gausz, Gyurkovics, and Párducz, 1981, Acta Biol. Acad. Sci. Hung. 32: 219-28.

Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.

genetics: Associated with *iab7*. Recessive transformation of sixth and seventh abdominal segment into fifth. In heterozygotes, shows tumor-like growths of sixth tergite tissue on back of head; also partial transformation of sixth segment into fifth.

molecular biology: Right breakpoint 133-139.5 kb distal to the right breakpoint of *In(3R)Cbx^{rv1}*.

In(3R)iab7^{Spth}

cytology: *In(3R)89A;89E*.

origin: X ray induced.

discoverer: Kempfues.

synonym: *In(3R)iab6^{Spth}*.

references: Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.

genetics: Associated with *iab7*. Recessive transformation of sixth and seventh abdominal segment into fifth. In heterozygotes, shows tumor-like growths of sixth tergite tissue on back of head; also partial transformation of sixth segment into fifth.

molecular biology: Right breakpoint 133 to 139.5 kilobases to the right of the right breakpoint of *In(3R)Cbx^{rv1}*.

In(3R)iab9^{rvTab}

cytology: *In(3R)89E;90A + In(3R)89E;90D*.
new order:
 61 – 89E|90A – 90D|90A – 89E|90D – 100.
origin: X ray induced in *In(3R)iab9*.
synonym: *In(3R)Tab^{rv107}*.
references: Celniker and Lewis, 1987, Genes and Development 1: 111-23.
genetics: Revertant of the *iab9^{Tab}* phenotype. When hemizygous, shows stronger transformation of the eighth ventral setal band toward that of the seventh than does *iab9/Df(3R)P9*; also, abnormal posterior spiracles and a rudimentary ninth ventral setal band.
molecular biology: Lesion associated with the 89E breakpoint lies between 185 and 187 kb on the composite restriction map of the BXC (Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, Science 221: 23-29).

In(3R)iab9^{Tab}

cytology: *In(3R)89E;90D*.
origin: X ray induced in *Mcp Sab²* chromosome.
synonym: *In(3R)Tab*.
references: Celniker and Lewis, 1984, Genetics 107: s17. Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96. Celniker and Lewis, 1987, Genes and Development 1: 111-23.
phenotype: Associated with *iab9^{Tab}*. Homozygous lethal and lethal over *Df(3R)P9*, the embryos showing transformation of segments A8 towards A7 or A6 and A7 towards A6. Also, ninth setal belt appears as group of denticles behind eighth setal belt. *iab9/+* flies show abdominal tissue in two sets of longitudinal stripes on the mesonotum.
molecular biology: Left breakpoint 188 kb to the right of the right breakpoint of *In(3R)Cbx^{rv1}*.

In(3R)IC67-79

cytology: *In(3R)85F;97F*.
origin: X ray induced.
references: Brosseau, 1969, DIS 44: 45.
genetics: Homozygous lethal.

In(3R)IC67-327

cytology: *In(3R)82D;98E*.
origin: X ray induced.
references: Brosseau, 1969, DIS 44: 45.
genetics: Homozygous viable and fertile. Associated with recessive rough eye phenotype.

In(3R)IW: Inversion (3R) Inoue Watanabe

origin: Naturally occurring inversions in Japan.
references: Inoue and Watanabe, 1979, Jpn. J. Genet. 54: 69-82.

inversion	cytology
<i>In(3R)IW11</i>	86E;89B
<i>In(3R)IW12</i>	86F;98F
<i>In(3R)IW13</i>	87B;100F
<i>In(3R)IW14</i>	87D;93A
<i>In(3R)IW15</i>	88E;93A
<i>In(3R)IW16</i>	92C;96A
<i>In(3R)IW17</i>	93E;97D

In(3R)J

cytology: *In(3R)96E;98F*.
origin: Naturally occurring inversion.
discoverer: Oshima and Watanabe.
references: 1965, DIS 40: 88. Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57. Choi, 1977, DIS 52: 88. 1977, Genetica 47: 155-60.

****In(3R)K: Inversion (3R) of Kodani***

cytology: *In(3R)86F1-87A1;96F11-97A1*.
origin: Spontaneous.
discoverer: Kodani.
references: Mourad and Mallah, 1960, Evolution 14: 166-70. Yang and Kojima, 1972, Univ. Tex. Publ. Zool. No. 7213: 229-36. Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

In(3R)K-

origin: Spontaneous in natural populations in Korea.
references: Choi, Ho, and Kim, 1984, DIS 60: 76.

inversion	cytology
<i>In(3R)K-g</i>	87F;91F
<i>In(3R)K-oa</i>	90B-C;97C + 92D;100F (overlapping)

In(3R)KA - In(3R)KK

origin: Spontaneous in natural populations in Korea.
references: Paik, 1986, DIS 63: 167.

inversion	cytology
<i>In(3R)KA</i>	82E-F;99E-F
<i>In(3R)KB</i>	83D-E;86D
<i>In(3R)KC</i>	83C-D;93A
<i>In(3R)KD</i>	84D;94D-E
<i>In(3R)KE</i>	85C;88F
<i>In(3R)KF</i>	86B;87B-C
<i>In(3R)KG</i>	86D-E;97C
<i>In(3R)KH</i>	88C;96D
<i>In(3R)KI</i>	88D;94A
<i>In(3R)KJ</i>	90D;93B
<i>In(3R)KK</i>	92E;97C

In(3R)kar: Inversion (3R) karmoisin

genetics: Mutant or deficient for *kar*.

inversion	cytology	ref ^α
<i>In(3R)kar^{3Hβ}</i>	81;87C7-D1 + 84E;98F + 89B;94A	4
<i>In(3R)kar^{Dγ}</i>	81F1;87C8	1
<i>In(3R)kar^{H4}</i>	87C8;89E	1
<i>In(3R)kar^{H6}</i>	81;87B6;87C8; deficient for 87B6-C8	1
<i>In(3R)kar^{IG27}</i>	87B3-5;87D6-12;99E1-F1; deficient for 87B3-5 to 87B6-12.	2,3

^α 1 = Henikoff, 1979, Genetics 93: 105-15; 2 = Hilliker, Clark, Chovnick, and Gelbart, 1980, Genetics 95: 95-110; 3 = Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72; 4 = Holden.
^β 61A – 81|87C7 – 84E|98F – 94A|89B – 94A|89B – 87D1|81 – 84E|98F – 100F.
^γ Homozygotes show reduced viability and male sterility; eyes vary in color from orange to brown. Heterozygotes with *In(3R)kar^{H4}* fully viable.

In(3R)KOG

origin: Spontaneous in natural populations of Australasia.
references: Knibb, Oakeshott, and Gibson, 1981, Genetics 98: 833-47.

inversion	cytology
<i>In(3R)KOG-L</i>	85D;93B-C
<i>In(3R)KOG-M</i>	87C;95A
<i>In(3R)KOG-N</i>	93D;98F
<i>In(3R)KOG-O</i>	95D;98F

In(3R)lab⁹: Inversion (3R) labial

cytology: *In(3R)84A1-2;84E*.
origin: X ray induced.
discoverer: Abbott.
synonym: *In(3R)lab^{a76}*.
references: Pultz, Diederich, Cribbs, and Kaufman, 1988, Genes. Dev. 2: 901-20.
genetics: Deficient for *lab⁹*.

In(3R)LD29

cytology: *In(3R)82;84*.
origin: X ray induced; detected by *bx* transvection effect.
discoverer: DeJongh.
references: Craymer.
genetics: Associated with a *T(2;3)*.

In(3R)LD30

cytology: *In(3R)83;99*.
origin: X ray induced; detected by *bx* transvection effect.
discoverer: DeJongh.
references: Craymer.

In(3R)M: Inversion (3R) of Mourad

cytology: *In(3R)86F;100E*.
origin: Spontaneous.
discoverer: Mourad and Mallah.
references: 1960, Evolution 14: 166-70.
 Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

In(3R)Mg: Inversion (3R) Mglinetz

inversion	cytology	origin	ref ^α
<i>In(3R)Mg18</i>	82D;99F + <i>Dp(3;3)82D;86(?)</i>	γ ray	2
<i>In(3R)Mg20</i>	89E;93A	γ ray	2
<i>In(3R)Mg21</i>	87E;97A	γ ray	2
<i>In(3R)Mg56</i>	87B;88F	γ ray	2
<i>In(3R)Mg67</i>	83C;85B	γ ray	3
<i>In(3R)Mg68</i>	84F;87A	γ ray	3
<i>In(3R)Mg70</i>	96A;100D	γ ray	3
<i>In(3R)Mg133</i>	86C;90D	γ ray	1
<i>In(3R)Mg136</i>	91D;98B	γ ray	1
<i>In(3R)Mg137</i>	81F;89D	γ ray	1
<i>In(3R)Mg138</i>	87B;88A	γ ray	1
<i>In(3R)Mg139</i>	86E;98F	32 P feeding	1
<i>In(3R)Mg140</i>	83D;96A	32 P feeding	1
<i>In(3R)Mg141</i>	88E;90F	32 P feeding	1
<i>In(3R)Mg143</i>	89F;96A	32 P feeding	1
<i>In(3R)Mg145</i>	82F;92B	32 P feeding	1
<i>In(3R)Mg146</i>	84B;97A	32 P feeding	1
<i>In(3R)Mg147</i>	84B;97A	32 P feeding	1
<i>In(3R)Mg148</i>	93A;97A	32 P feeding	1
<i>In(3R)Mg157</i>	87C;98F	γ ray	1

^α 1 = Mglinetz, 1968, Genetika (Moscow) 4(8): 81-86; 2 = Mglinetz, 1972, Genetika (Moscow) 8(2): 82-92; 3 = Mglinetz, 1972, Genetika (Moscow) 9(3): 69-75.

In(3R)ML193

cytology: *In(3R)93E;100F + T(2;3)52E1;80-81*.
origin: X ray induced.
references: Mukhina and Zhimulev, 1980, DIS 55: 209.

In(3R)Mo: Inversion (3R) from Missouri

cytology: *In(3R)93D;98F2-3* (Bridges and Li in Morgan, Bridges, and Schultz, 1936, Year Book - Carnegie Inst. Washington 35: 293).

origin: Naturally occurring inversion.
discoverer: Sturtevant, 1924.
synonym: *CIIR-K2; In(3R)A; In(3R)I*.
references: 1931, Carnegie Inst. Washington Publ. No. 421: 6-7.
 Inoue and Watanabe, 1979, Jpn. J. Genet. 54: 69-82.
genetics: Crossing over reduced in heterozygote to about 5% between centromere and *sr* and 0.3% between *sr* and *ca*.
other information: Found in natural populations (e.g., Warters, 1944, Texas Univ. Publ. 4445: 129-74; Oshima and Watanabe, 1965, DIS 40: 88; Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57; Choi, 1977, DIS 52: 88; 1977, Genetica 47: 155-60).

In(3R)Msc: see *In(3R)Scr^{Msc}*

In(3R)Na: Inversion (3R) from Naples

cytology: *In(3R)86F2-3;96F11-97A1;97A2-5*. 97A1-2 missing.
origin: Spontaneous.
discoverer: Carfagna and Nicoletti, 1960.
references: 1963, DIS 38: 32.
genetics: Carries a lethal, which may be separable from the inversion (or the deficiency for 97A1-2 may be the lethal).
other information: Breakpoints similar to those of *In(3R)K = In(3R)86F1-87A1;96F11-97A1* and may be the same.

****In(3R)Nel-D: Inversion (3R) of Nel***

cytology: *In(3R)86D;97A*.
origin: Spontaneous in natural population.
discoverer: Nel.
other information: Possibly the same as *In(3R)K = In(3R)86F1-87A1;96F11-97A1*.

In(3R)neur^{XK2}: Inversion (3R) neuralized

cytology: *In(3R)85C;87D5-14;90E-F*.
origin: X ray induced.
discoverer: Knust.
references: Campos-Ortega.
genetics: Reverted for enhancing effect on *spl* of *E(spl)*.

****In(3R)p^{100.290}: Inversion (3R) pink***

cytology: *In(3R)85B3-4;85D12-15*.
origin: X ray induced.
discoverer: Alexander.
references: Ward and Alexander, 1957, Genetics 42: 42-54.
genetics: Mutant for *p*.

In(3R)p⁴¹⁹

cytology: *In(3R)84D4-6;86A3*.
origin: X ray induced.
references: Kempthues, Raff, and Kaufman, 1983, Genetics 105: 345-56.
genetics: Mutant for *p*.

In(3R)P: Inversion (3R) of Payne

cytology: *In(3R)89C-D;96A* (Inoue and Watanabe, 1979).
origin: Widespread in natural populations.
discoverer: Payne, 17g.
synonym: *In(3R)G* (Watanabe, 1967, Mem. Fac. Sci. Kyushu, Univ. E4: 159-78); *In(3R)hp* (Craymer).
references: 1918, Indiana Univ. Studies 5, No. 36: 1-45. 1924, Genetics 9: 327-42. Sturtevant, 1931, Carnegie Inst. Washington Publ. No. 421: 1-27.

Inoue and Watanabe, 1979, Jpn. J. Genet. 54: 69-82.
genetics: Crossing over reduced in heterozygous females to 1% between *p* and *sr*, none between *sr* and *ro*, and 0.5% between *ro* and *ca*.

other information: Widespread in laboratory stocks and is part of the balancers, *LVM* and *C(3)x*. Also found in many wild populations (e.g., Warters, 1944, Texas Univ. Publ. 4445: 129-74; Oshima and Watanabe, 1965, DIS 40: 88; Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57; Stalker, 1976, Genetics 82: 323-47; Choi, 1977, DIS 52: 88; 1977, Genetica 47: 155-60; Knibb, 1982, Genetica 58: 213-21).

In(3R)P18 - In(3R)P(Pc³): Inversion (3R) Pasadena

origin: X ray induced.

inversion	cytology	ref ^α
<i>In(3R)P18</i> ^β	81F;91F-92A	1, 3
<i>In(3R)P33</i>	89B;98B	2
<i>In(3R)P110</i>	81F;98F-99A	2
<i>In(3R)P(Pc³)</i> ^γ	85D3-6,88C-D	4

^α 1 = Chovnick, 1973, Genetics 75: 123-31; 2 = Craymer; 3 = Craymer, 1984, DIS 60: 81-82; 4 = Lewis, 1980, DIS 55: 207-08.

^β Discoverer: E.B. Lewis.

^γ Discoverer: E.B. Lewis. Induced along with *Pc³*, which is separable from the inversion. Breakpoints revised by B.S. Baker, 1985.

In(3R)pb: In(3R) proboscipedia

origin: X ray induced.
genetics: Null mutant for *pb*.

inversion	cytology	references ^α
<i>In(3R)pb</i> ²⁴	84A4-5;84D	2
<i>In(3R)pb</i> ²⁵	84A4-5;80-81	2
<i>In(3R)pb</i> ²⁶	84A4-5;85D	2
<i>In(3R)pb</i> ³⁶	84A1-2;87B or 84A4-5;87A-B	1,2

^α 1 = Bender, Turner, and Kaufman, 1986, Dev. Biol. 119: 418-32; 2 = Pultz, Diederich, Cribbs, and Kaufman, 1988, Genes Dev. 2: 901-20.

In(3R)PB - In(3R)PN: Inversion (3R) Pipkin

origin: Naturally occurring inversions.
references: Pipkin, Franklin-Springer, Law, and Lubega, 1976, J. Hered. 67: 258-66.

inversion	cytology
<i>In(3R)PB</i>	86E-F;98E
<i>In(3R)PC</i>	84F;86D
<i>In(3R)PC208</i>	91A-B;96A-B
<i>In(3R)PD</i>	86F;96A-B
<i>In(3R)PE</i>	88C;96E
<i>In(3R)PG</i>	89D;96A-B
<i>In(3R)PH</i>	89B;96A
<i>In(3R)PI</i>	90D;96A-B
<i>In(3R)PJ</i>	88F;98E-F
<i>In(3R)PL</i>	89C;93D;96A
<i>In(3R)PM</i>	86E-F;89E;95B;96F

inversion	cytology
<i>In(3R)PN</i>	86E-F;92E;96F

In(3R)PS: Inversion (3R) Paik Sung

origin: Naturally occurring inversions in Korea.
references: Paik, 1979, Korean J. Genet. 1: 18-27. Paik and Sung, 1980, DIS 55: 120.

inversion	cytology
<i>In(3R)PS31</i>	83C;85B
<i>In(3R)PS32</i>	86F;96A
<i>In(3R)PS33</i>	87B;92F
<i>In(3R)PS34</i>	87F;90F
<i>In(3R)PS35</i>	88C;98F
<i>In(3R)PS36</i>	88C-D;93C
<i>In(3R)PS37</i>	88D;90F
<i>In(3R)PS38</i>	88D;94A
<i>In(3R)PS39</i>	91C;93B

In(3R)R24

cytology: *In(3R)91B-C;94C-E;98F*.
origin: X ray induced.
references: Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84.
genetics: Associated with *T(Y;3)R24*.

In(3R)rn: see T(2;3)rn

In(3R)ry⁵⁴

cytology: *In(3R)81;87D8-12*.
origin: X ray induced.
references: Lefevre, 1971, DIS 46: 40. Clark and Chovnick, 1986, Genetics 114: 819-40.
genetics: Variegates for *ry* and *pic*, but not for *snk* (Clark and Chovnick, 1986). *ry⁵⁴* homozygotes almost completely lethal (CP627).

In(3R)ry^{ps11136}

cytology: *In(3R)81;87D*.
origin: γ ray induced.
references: Chovnick, McCarron, Clark, Hilliker, and Rushlow, 1980, Development and Neurobiology of Drosophila (Siddiqi, Babu, Hall and Hall, eds.). Plenum Press, New York and London, pp. 3-23. Rushlow and Chovnick, 1984, Genetics 108: 589-602. Rushlow, Bender, and Chovnick, 1984, Genetics 108: 603-15. Clark and Chovnick, 1986, Genetics 114: 819-40.
genetics: Variegates for *ry*. Mutant for *l(3)87Df*. Lethal homozygous or when heterozygous with certain *ry* deficiencies. Also produces mutant effects on *snk* and *pic* (Clark and Chovnick, 1986). Purine sensitive when heterozygous with noncomplementing *ry* alleles.
molecular biology: Right breakpoint at about 175 kb to the left of the origin of walk in the *ry* region; this origin is an arbitrary point which is in turn 6.5 kb to the right of the left breakpoint of *In(3R)Cbx^{rv1}*.

In(3R)sbd: Inversion (3R) stubbloid

origin: X ray induced.
references: Spillman and Nöthiger, 1978, DIS 53: 164-65.

genetics: Homozygous lethal in first larval instar.

inversion	cytology
<i>In(3R)sbd</i> ¹²	88B2-C1;89B3-16
<i>In(3R)sbd</i> ¹⁷	81;89B10-12
<i>In(3R)sbd</i> ²¹	86D2-E1;89B3-12

***In(3R)Scm*^{K1}: Inversion (3R)
Sex combs on midleg**

cytology: *In(3R)85F;89AB*.

origin: EMS induced.

discoverer: Kennison.

genetics: Associated with *Scm*^{K1}.

***In(3R)Scr*^{Msc}: Inversion (3R)
Sex comb reduced**

cytology: *In(3R)84B1-2;84F1-2*.

origin: Spontaneous.

synonym: *In(3R)Msc*.

references: Tokunaga, 1966, DIS 41: 57.

Denell, 1973, Genetics 75: 279-97.

Lewis, Wakimoto, Denell, and Kaufman, 1980, Genetics 95: 383-97.

Sato, Russell, and Denell, 1983, Genetics 105: 357-70.

Kuroiwa, Hafen, and Gehring, 1984, Cell 37: 825-31.

genetics: Dominant *Scr* phenotype (Sato *et al.*, 1983).

Recessive lethal (Duncan and Kaufman, 1975, Genetics 80: 733-52).

molecular biology: Left breakpoint 62-64 kilobases distal to the proximal breakpoint of *Tp(3;3)Dfd*^{rvX16} (Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, Cell 35: 763-76).

***In(3R)Scr*^{Msc-T1}**

cytology: *In(3R)84A1-2;84A6-B1* (Tiong).

origin: γ ray induced in a *mwh tuh-2* stock.

discoverer: Tiong.

synonym: *Msc*^{T1}.

genetics: *Dfd*⁺ *Scr*⁻ *ftz*⁺ *Antp*⁺.

***In(3R)Scr*^{MscL} *Antp*^{BR}**

cytology: *In(3R)84B1-2;84F1-2*^L *84B1-2;85E*^R; deficient for 84F2-85E and for part of 84B1-2.

origin: Recombination between *In(3R)Scr*^{Msc} and *In(3R)Antp*^B.

synonym: *In(3R)Msc*^L *Antp*^{BR} + *Df(3R)Msc*^L *Antp*^{BR} or *Df(3R) β 2t*.

references: Kempthues, Raff, and Kaufman, 1983, Genetics 105: 345-56.

genetics: Associated with *Antp*^B and *Scr*^{Msc}. Deficient for *β Tub85D* and *M(3)85E*.

***In(3R)Scx*^{Wrv5}: Inversion (3R)
Sex combs extra**

cytology: *In(3R)80-81;84B1-2*. [Break in 81 if indeed *In(3R)*].

origin: X ray induced.

synonym: *In(3R)Scx*^{W+RX5}; *In(3R)Antp*^{Scx-rv5}.

references: Hazelrigg and Kaufman, 1983, Genetics 105: 581-600.

genetics: Incomplete revertant of *Scx*^W. Homozygous lethal.

molecular biology: Right breakpoint 45-52 kilobases to the right of the left-most breakpoint of *Tp(3;3)Dfd*^{rv16} (Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, Cell 35: 763-76).

In(3R)SGA62

cytology: *In(3R)88C4-7;89E1-2*.

origin: X ray induced.

references: Awad, Gausz, Gyurkovics, and Párduez, 1981, Acta Biol. Acad. Sci. Hung. 32: 219-28.

Kuhn and Packert, 1988, Dev. Biol. 125: 8-18.

genetics: Associated with *iab7*^{SGA}, a dominant mutation that transforms the occipital region of the head and the eye disc into abdominal and sometimes genital tissue.

***In(3R)SMG*: Inversion (3R)
Semenova Mglinetz Glotoff**

origin: γ ray induced.

references: Semenova, Mglinetz, and Glotoff, 1970, Genetika (Moscow) 6(4): 165-69.

inversion	cytology
<i>In(3R)SM22</i>	83D;94E
<i>In(3R)SM23</i>	82F;92D
<i>In(3R)SM24</i>	87E;95A
<i>In(3R)SM25</i>	92B;98C
<i>In(3R)SM26</i>	81;92A
<i>In(3R)SM27</i>	88;97A
<i>In(3R)SM28</i>	97A;100A
<i>In(3R)SM29</i>	87E;94A
<i>In(3R)SM30</i>	88A;94A
<i>In(3R)SM31</i>	92F;95C
<i>In(3R)SM33</i>	85C;88B
<i>In(3R)SM34</i>	83E;86A

****In(3R)sr*^{3.2}: Inversion (3R) stripe**

cytology: *In(3R)90D1-E1;93B-E*.

origin: X ray induced.

discoverer: Alexander, 1959.

references: 1960, Genetics 45: 1019-22.

genetics: Mutant for *sr*.

***In(3R)St*: Inversion (3R) Stalker**

origin: Naturally occurring inversions.

references: Stalker, 1976, Genetics 82: 323-47.

inversion	cytology
<i>In(3R)St-C</i>	87A;100F
<i>In(3R)St-E</i>	86E;97C
<i>In(3R)St-F</i>	86D;88E-F
<i>In(3R)St-G</i>	96F;100B
<i>In(3R)St-H</i>	90B;98A
<i>In(3R)St-J</i>	92D;93F
<i>In(3R)St-K</i>	86E;87F
<i>In(3R)St-L</i>	84D;86C
<i>In(3R)St-M</i>	89A;99C
<i>In(3R)St-O</i>	84E;87C
<i>In(3R)St-P</i>	96C;98E
<i>In(3R)St-Q</i>	93D;98B
<i>In(3R)St-R</i>	86B;87F

****In(3R)su(pr)*: Inversion (3R)
suppressor of purple**

cytology: Breakpoints unknown.

origin: Spontaneous.

discoverer: Stern, 27c2.

synonym: *su*^{S-pr}.

references: 1929, Z. Indukt. Abstamm. Vererbungslehre 52: 373-89.

1934, DIS 1: 35.

genetics: Associated with *su(pr)*.

In(3R)TC

cytology: *In(3L)84D;91E*.

origin: Spontaneous in a natural population in Brownsville, Texas.

references: Yang, Kojima, and Kovarik, 1971, DIS 47: 71-72.

Langley, Tobar, and Kojima, 1974, Genetics 78: 921-36.

Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

In(3R)TI^{rv}: Inversion (3R) Toll-revertant

references: Anderson, Jürgens, and Nüsslein-Volhard, 1985, Cell 42: 779-89.

genetics: Revertants of the female sterility of *Tl*; *trans* heterozygotes viable and produce dorsalized embryos as maternal effect.

inversion	cytology	origin	synonym
<i>In(3R)TI^{rv5}</i>	97D;98F	X ray	<i>In(3R)TI^{RXJ}</i>
<i>*In(3R)TI^{rv6}</i>	97D;99E-F	X ray	<i>In(3R)TI^{RXZ}</i>
<i>In(3R)TI^{rv12}</i>	97D;99B	EMS	<i>In(3R)TI^{5BREN}</i>
<i>In(3R)TI^{rv17}</i>	97A;97B;97D ^α	EMS	<i>In(3R)TI^{5BREW}</i>
<i>In(3R)TI^{rv19}</i>	97D;98C-D	EMS	<i>In(3R)TI^{9QRE}</i>

^α Deficient for 97E-F.

In(3R)tll: Inversion (3R) tailless

cytology: *In(3R)85F10-86A1;100A6-B1*.

origin: X ray induced.

synonym: *tll²*.

references: Strecker, Kongswan, Lengyel, and Merriam, 1986, Dev. Biol. 113: 64-76.

phenotype: Mutant for *tll*. When in heteroallelic combination with *tll¹*, embryos lack telson but have normal eighth abdominal denticle belt and pharyngeal ridges.

In(3R)Ubx: Inversion (3R) Ultrabithorax

genetics: Associated with *Ubx* alleles.

inversion	cytology	origin	synonym	discov or ref ^α
<i>In(3R)Ubx^{6Q}</i>	89E3-4;96F;97A		<i>Ubx^{21560.6Q}</i>	7
<i>In(3R)Ubx^{7L}</i>	89E1-2;96A	X ray	<i>Ubx^{19286.7L}</i>	
<i>In(3R)Ubx^{16N}</i>	89E;99	X ray	<i>Ubx^{19286.16N}</i>	7
<i>In(3R)Ubx^{30A}</i>	89E1-2;91B	X ray	<i>Ubx^{3966.30}</i>	
<i>In(3R)Ubx⁸⁰</i>	87F-88A;89E	EMS	<i>Ubx^{16800.752}</i>	1
<i>In(3R)Ubx¹²⁵</i>	81;89E	X ray		5
<i>In(3R)Ubx⁹⁶¹</i>	89E;96			
<i>In(3R)Ubx^{G2}</i>	89E;96A1-7	X ray	<i>Ubx¹⁹²⁸⁶</i>	5
<i>In(3R)Ubx^K</i>	89E;92A	X ray	<i>Cbx^{+R1}</i>	3
<i>In(3R)Ubx^{KM5^β}</i>	88B;89E1-2	X ray	<i>Ubx^{12.5}</i>	4
<i>In(3R)Ubx^{KM22^β}</i>	88F;89E1-2	X ray	<i>Ubx^{5.22}</i>	4
<i>In(3R)Ubx^{P18}</i>	81F;91F-92			5
<i>In(3R)Ubx^{R5}</i>	89E;92A	X ray	<i>Cbx^{rvR17.5E}</i>	6
<i>In(3R)Ubx^{R6}</i>	87;89E	X ray	<i>Cbx^{rvR17.6F}</i>	6
<i>In(3R)Ubx^{R14}</i>	87D-E;89E	X ray	<i>Cbx^{rvR17.14P}</i>	2, 6
<i>In(3R)Ubx^{R20}</i>	87B-D;89E	X ray	<i>Cbx^{rvR17.20V}</i>	2, 6
<i>In(3R)Ubx^X</i>	89E;98B-C	X ray		5

^α 1 = Bacher, 66h;

2 = Bender, Spierer, and Hogness, 1983, J. Mol. Biol. 168: 17-33;

3 = Kaufman; 4 = Kerridge and Morata, 1982, J. Embryol. Exp.

Morphol. 68: 211-34; 5 = Lewis; 6 = Ramey; 7 = Smit, 72i.

^β Homozygotes lethal; heterozygotes weak *Ubx*.

In(3R)UF

cytology: *In(3R)84E;91C*.

origin: Spontaneous in a natural population in Egypt.

references: Mourad and Mallah, 1960, Evolution 14: 166-70.

Mourad, Tantawy and Masry, 1972, Egyptian J. Genet. Cytol. 1: 141-48.

Ashburner and Lemeunier, 1976, Proc. R. Soc. London, B 193: 137-57.

In(3R)V: Inversion (3R) Valencia

origin: X ray induced.

references: Valencia, 1970, DIS 45: 37-38.

inversion	cytology	genetics
<i>In(3R)V9-8</i>	81-82A;89A-B	lethal
<i>In(3R)V16</i>	93F;99C +	detrimental
	<i>T(2;3)60B-C;84A</i>	
<i>In(3R)V103</i>	85A;96E +	lethal
	<i>T(2;3)58F;67B4</i>	

In(3R)Vno: see *Tp(3;3)iab6^{Vno}*

In(3R)VV10: Inversion (3R) V. Velissariou

cytology: *In(3R)94C;99F*.

origin: γ ray induced.

discoverer: Velissariou.

references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

***In(3R)W: Inversion (3R) of Warters**

cytology: *In(3R)86B;92F*.

origin: Naturally occurring inversion.

discoverer: Warters.

references: 1944, Texas Univ. Publ. 4445: 129-74.

In(3R)ZP: Inversion (3R)

Zacharopoulou Pelecanos

origin: Naturally occurring inversions in Greece.

references: Zacharopoulou and Pelecanos, 1980, Genetics 54: 105-11.

inversion	cytology
<i>In(3R)ZP24</i>	91B;97C
<i>In(3R)ZP25</i>	87D;92C
<i>In(3R)ZP26</i>	96A;98B
<i>In(3R)ZP27</i>	90F;94E
<i>In(3R)ZP28</i>	84F;91D
<i>In(3R)ZP29</i>	88A;93C
<i>In(3R)ZP30</i>	94A;98C

Inp(1)sc^{VI}: see *In(1LR)sc^{VI}*

Ins(2LR)Pm: see *In(2LR)bw^{VI}*

RINGS

R(1)1: Ring (1)

cytology: *R(1)1A;20B-C*; salivary chromosomes show deficiency for most of 1A and a duplication for 20C-D [Schultz and Catcheside, 1937, *J. Genet.* 35: 315-20 (fig.)]. Ring shaped in metaphase.

new order:

|1A – 20·20F – 20C|

origin: Spontaneous from *C(1)RM*, *y* female.

discoverer: L. V. Morgan, 1922.

synonym: X^c ; X^0 .

references: 1926, *Proc. Nat. Acad. Sci. USA* 12: 180-81. 1933, *Genetics* 18: 250-83.

genetics: Carries *y*. Male and homozygous female have reduced viability; *X/0* male lethal (Schultz, 1941, *Proc. Int. Congr. Genet.*, 7th., pp. 257-62). Somewhat unstable, tending to be eliminated during mitosis. Shows about 5 times as much somatic crossing over as rod *X* (Brown, Walen, and Brosseau, 1962, *Genetics* 47: 1573-79). Crossing over reduced in ring/rod heterozygote; only double crossovers recovered. Exceptional males result from 4-strand double crossing over in *R(1)1/+* female.

other information: May open out into a rod [e.g., *In(1)EN*] spontaneously in stock.

R(1)2

cytology: *R(1)1A3-4;19F-20A1*; salivary chromosomes deficient for 1A1-3 and reported to be duplicated for all of region 20 (Schultz and Catcheside, 1937; Viniika *et al.*, 1971), but duplication was not confirmed by Falk (1973). Ring shaped in metaphase.

new order:

|1A4 – 20·20F – 20A1|

origin: Spontaneous as a detachment of *C(1)RM*, y^+ .

discoverer: Beadle, 34b (ring nature discovered by Boche).

synonym: X^{c2} .

references: Schultz and Catcheside, 1937, *J. Genet.* 35: 315-20.

Viniika, Hannah-Alava, and Arajarvi, 1971, *Chromosoma* 36: 34-45.

Falk, R, 1973, *DIS* 50: 144-45.

genetics: Carries y^+ . More viable than *R(1)1*; *X/0* male survives. Ordinarily, ring elimination less than 1% (Battacharya, 1950, *Proc. R. Soc. Edinburgh*, B 64: 199-215; Braver and Blount, 1950, *Genetics* 35: 98), but nearly 20% of the first progeny of 11-day-old females crossed to ring-bearing males are gynandromorphs (Hannah, 1955, *Z. Indukt. Abstamm. Vererbungslehre* 86: 600-21). Number of male nuclei in eggs of gynandromorphs varies greatly, suggesting that loss of the ring *X* may occur more than once and that it may take place as late as the ninth cleavage (Zalokar, 1980, *Cell* 19: 133-41). Crossing over reduced in ring/rod heterozygote; only double crossovers recovered. Exceptional males result from 4-strand double exchange in *R(1)2/+* female.

other information: Ring may open out spontaneously in stock [e.g., *In(1)EN2*].

R(1)5A

cytology: Ring shaped in mitotic figures.

origin: Derived from *C(1)TM5A* by meiotic exchange between arms.

references: Pasztor, 1967, *DIS* 42: 107.

1971, *Genetics* 68: 245-58.

genetics: Mitotically unstable, producing gynandromorphs and *XO* males. Duplication of *X* required for male survival; *R(1)5A/0* and *R(1)5A/Y* males not recovered; however, *R(1)5A*-bearing males survive in combination with B^{SY} or $su(f)^+Y$.

R(1)9-1

cytology: Ring shaped in mitotic figures. Early prophase shows heterochromatic constitution (proceeding from normally proximal euchromatin, across the centromere to the normally distal euchromatin) to be as follows: a large segment, a well-defined constriction, a large segment, a constriction, a small segment, the centric constriction, a small segment.

origin: Regular product of exchange in *C(1)TMB^S9-1*.

references: Lindsley and Sandler, 1965, *Genetics* 51: 223-45 (fig.).

genetics: Carries *y*. *R(1)9-1/0* male survives. On basis of origin, *R(1)9-1* is euchromatically but not heterochromatically identical with *R(1)1*.

R(1)9-4

cytology: Ring shaped in mitotic figures. Early prophase shows heterochromatic constitution (proceeding from normally proximal euchromatin, across the centromere to the normally distal euchromatin) to be as follows: a large segment, a constriction, a small segment, the centric constriction, a small segment.

origin: Regular product of exchange in *C(1)TMB^S9-4*.

references: Lindsley and Sandler, 1965, *Genetics* 51: 223-45 (fig.).

genetics: Carries *y*. *R(1)9-4/0* male viable. Based on origin, *R(1)9-4* euchromatically but not heterochromatically identical with *R(1)1*.

R(1)63

cytology: Ring shaped in mitotic figures. Early prophase shows heterochromatic constitution (proceeding from the normally proximal euchromatin, across the centromere to the normally distal euchromatin) to be as follows: two large segments separated by an ill-defined constriction, a constriction, a small segment, the centric constriction, a small segment.

origin: Regular product of exchange in *C(1)TM2*.

references: Lindsley and Sandler, 1965, *Genetics* 51: 223-45 (fig.).

genetics: Carries *y*. *R(1)63/0* male survives. Based on origin, *R(1)63* is euchromatically but not heterochromatically identical with *R(1)1*; however, survival of *R(1)63/0* males suggests otherwise.

R(1)94-2A1

cytology: *R(1)1A;1F-2A;5E-6A;17F-18A;20*; duplicated for 1A-F and 18A-20.

new order:

|1A – 5E|1F – 1A|20·20 – 6A|18A – 20|

origin: Spontaneous product of *C(1)94-2A*. Possibly a product of breakage of double second-anaphase bridge formed by exchange between the arms of the compound.

discoverer: Armentrout, 1964.

***R(1)C1**

cytology: Ring shaped in mitotic figures.
origin: Spontaneous derivative of *In(1)sc*^{8L}*EN*^R; arose by recombination between distal heterochromatic segment of *In(1)sc*⁸ and heterochromatic short arm of *In(1)EN*.
discoverer: Lindsley, 1950.
references: 1958, Z. Indukt. Abstamm. Vererbungsl. 89: 103-22.
genetics: Carries *y*. On basis of origin, *R(1)C1* is euchromatically identical with *R(1)I*, but it must be different heterochromatically since *R(1)C1/0* male viable.

R(1)Ddc⁺

cytology: *R(1)IA3-4;19F-20A1 In(1)3C1-2;20F + Ddc⁺* insert (near *sn*).
origin: Double crossover between *R(1)2*, *In(1)w^{vc}* and rod-X chromosome with *P*-element mediated *Ddc⁺* insert.
synonym: *Ddc⁺-Ring-X*.
references: Gailey, Bordne, Vallés, Hall, and White, 1987, Genetics 115: 305-11.
genetics: Shows instability characteristic of *R(1)2*, *In(1)w^{vc}*. Used to generate *Ddc* mosaics.

R(1)I-v459

cytology: *R(1)3D-F*.
origin: Associated with *T(1;2;3)I-v459*.

R(1)y⁴: Ring (1) yellow

cytology: *R(1)IA8-B1;18A3-4*; deficient for 1A and duplicated for 18-20.
new order: |1B1 – 20·20 – 18A4|
origin: Regular product of exchange within inversion in *C(1)SB*; which is a *C(1)RM* heterozygous for *In(1)y⁴ = In(1)IA8-B1;18A3-4*.
discoverer: Sturtevant and Beadle.
references: 1936, Genetics 21: 554-604.
 Novitski and Sandler, 1956, Genetics 41: 194-206.
genetics: Deficient for loci distal to *y*, duplicated for *car-bb*. Heterozygous female survives; male lethal, owing to deficiency for *l(1)IA* loci.

R(1;Y)EN

cytology: Complete *X* and *Y* chromosomes joined together in a ring.

origin: X-ray-induced exchange in tandem metacentric carrying a normal *X* attached to one *Y* arm and an inverted *X* to the other arm without loss of *Y* fertility factors.

synonym: *C(1;Y)R, EN*.

references: Fowler, 1971, DIS 46: 74.

Novitski and Childress, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 487-503.

Novitski and Puro, 1978, DIS 53: 205.

genetics: Filicidal except in certain genetic backgrounds. Not obviously unstable. Neither the *X* nor the *Y* in the ring lack any essential viability or fertility genes.

R(Y)

Described in section on *Y* derivatives.

R(2)Bl: see *Dp(2;f)Bl*

R(2)rl⁺: see *Dp(2;f)rl⁺*

R(3)S1: Ring (3) Synthetic

cytology: Ring shaped in mitotic figures. Duplicated for 89C-92D and deficient for terminal chromatin of 61A and 100F.

new order: |61A – 100F|92D – 89C|

origin: Synthesized from *In(3LR)P88 + In(3R)C* and a structurally normal *3* by crossing over in both the 61-89 and the 92E-100F region.

references: Craymer, 1984, DIS 60: 80-81.

genetics: Duplication of 89C-92D compensated by *Df(3R)P47 = Df(3R)89D;92A; R(3)S1/Df(3R)P47* flies have minor phenotypic abnormalities and are sterile or nearly so; a few fertile crossovers carrying *Df(3R)P47* in the ring are fertile.

other information: Ring useful for inserting markers into *In(3R)C*.

R(3L)

cytology: Ring involving all of *3L*.

origin: Byproduct of synthesis of *3R3L.3R* chromosome by X ray.

references: Novitski and Puro, 1978, DIS 53: 205.

TRANSLOCATIONS

***T(1;?)sc²⁶⁰⁻²³: Translocation (1;?) scute**

cytology: T(1;?)1B2-3; position of second break not determined.

origin: X ray induced.

discoverer: Sutton, 1939.

references: 1943, Genetics 28: 210-17.

genetics: Mutant for *sc* but not *y* or *svr*.

T(1;Y)

Table I: Translocations with breaks in X euchromatin.

cytology: X breaks based on salivary analysis (exceptions indicated in notes); Y breaks based on genetic data.

origin: X ray induced. Translocations with unlettered designations induced by Nicoletti; those with lettered designations F, G and P-W (except P12) induced by Kennison; the remainder induced by Merriam and colleagues.

genetics: Male viability and fertility indicated in table.

translocation	cytology	break in	ref ^α	male viable ?	T(1;Y)0 fertile ?
*T(1;Y)1	16F;YL	y ⁺ Y	3,4	+	
T(1;Y)2 ^β	5E;11F;19F;YS	y ⁺ Y	3,4	+	
T(1;Y)3	3E;YS	y ⁺ Y	3,4	-	
T(1;Y)4	11A;YL	y ⁺ Y	3,4	+	
T(1;Y)6	11D;YS	y ⁺ Y	3,4	-	
*T(1;Y)8	4B;YL	y ⁺ Y	3,4	+	
*T(1;Y)9 ^β	2C;19F;YS	y ⁺ Y	3,4	+	
T(1;Y)10	3E;YL	y ⁺ Y	3,4	+	
*T(1;Y)11	19F;YS	y ⁺ Y	3,4	+	
*T(1;Y)13	7D;YL	y ⁺ Y	3,4	-	
T(1;Y)14	19F;YS	y ⁺ Y	3,4	+	
*T(1;Y)15	14F;YL	y ⁺ Y	3,4	-	
T(1;Y)16	4C;YL	y ⁺ Y	3,4	-	
*T(1;Y)18	19F;YS	y ⁺ Y	3,4	+	
T(1;Y)19	17A;YL	y ⁺ Y	3,4	+	
T(1;Y)20	11A;YL	y ⁺ Y	3,4	+	
T(1;Y)21	1F;YL	y ⁺ Y	3,4	+	
T(1;Y)22	19E;YS	y ⁺ Y	3,4	+	
*T(1;Y)100	13F;YS	B ^S Y	3,4	-	
T(1;Y)101	19E;YS	B ^S Y	3,4	+	
T(1;Y)102	7D;YL	B ^S Y	3,4	+	
T(1;Y)103	19F;YS	B ^S Y	3,4	-	
T(1;Y)104	3D;YL	B ^S Y	3,4	+	
T(1;Y)105	19F;YS	B ^S Y	3,4	+	
T(1;Y)106	16A;YL	B ^S Y	3,4	+	
T(1;Y)107 ^γ	3C8-12;YL	B ^S Y	3,4,7	+	
T(1;Y)108	5D;YL	B ^S Y	3,4	-	
T(1;Y)111 ^δ	3C;YL	B ^S Y	3,4	-	
T(1;Y)112	15A;YL	B ^S Y	3,4	-	
*T(1;Y)113	20A;YS	B ^S Y	3,4	+	
T(1;Y)114	3C;YL	B ^S Y	3,4	-	
T(1;Y)115	8F;YS	B ^S Y	3,4	-	
*T(1;Y)117	17A;YL	B ^S Y	3,4	+	
T(1;Y)118	16E;YL	B ^S Y	3,4	+	
T(1;Y)119	19F;YS	B ^S Y	3,4	+	
*T(1;Y)120	17E;YS	B ^S Y	3,4	-	
T(1;Y)122	20A;YS	B ^S Y	3,4	+	
*T(1;Y)123	19F;YS	B ^S Y	3,4	+	
T(1;Y)124	9F;YL	B ^S Y	3,4	+	
T(1;Y)125	15D;YL	B ^S Y	3,4	+	
T(1;Y)128 ^γ	3C;YL	B ^S Y	3,4,7	-	
T(1;Y)129	11A;YL	B ^S Y	3,4	+	
T(1;Y)131	6E;YS	B ^S Y	3,4	+	
T(1;Y)132	19F;YS	B ^S Y	3,4	+	
T(1;Y)133	19E;YS	B ^S Y	3,4	+	
*T(1;Y)135	18C;YL	B ^S Y	3,4	+	
T(1;Y)137	19F;YS	B ^S Y	3,4	+	
*T(1;Y)139	20A;YS	B ^S Y	3,4	+	

translocation	cytology	break in	ref ^α	male viable ?	T(1;Y)0 fertile ?
T(1;Y)140 ^γ	3C8-12;YL	B ^S Y	3,4,7	+	
T(1;Y)141	19E;YS	B ^S Y	3,4	+	
T(1;Y)142	13E;YL	B ^S Y	3,4	+	
T(1;Y)145	11B;YS	B ^S Y	3,4	+	
T(1;Y)147	8F;YS	B ^S Y	3,4	+	
*T(1;Y)148	2D;YL	B ^S Y	3,4	+	
T(1;Y)149	6E;YL	B ^S Y	3,4	+	
T(1;Y)150	3F;YS	B ^S Y	3,4	+	
T(1;Y)151	19F;YS	B ^S Y	3,4	+	
T(1;Y)152	13A;YL	B ^S Y	3,4	+	
T(1;Y)155	7B;YS	B ^S Y	3,4	-	
T(1;Y)156	7D;YL	B ^S Y	3,4	+	
*T(1;Y)157	14F;YL	B ^S Y	3,4	+	
*T(1;Y)158	11A;YL	B ^S Y	3,4	-	
T(1;Y)159	18A;YL	B ^S Y	3,4	+	
T(1;Y)164 ^γ	3C;YL	B ^S Y	3,4,7	-	
*T(1;Y)169	11D;YS	B ^S Y	3,4	+	
T(1;Y)240	14A;YL	B ^S Y	3,4	+	
*T(1;Y)290 ^β	1A;20A;YS	B ^S Y	3,4	-	
T(1;Y)A1	12E;YL	B ^S Yy ⁺	2	+	
T(1;Y)A2	3F;YS	B ^S Yy ⁺	2	+	+
T(1;Y)A4	11A;YS	B ^S Yy ⁺	2	+	+
T(1;Y)A8	12F;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B5	7C;YL	B ^S Yy ⁺	5	-	
T(1;Y)B6	14E-F;YS	B ^S Yy ⁺	5	±	
*T(1;Y)B7	12B-C;Y	B ^S Yy ⁺	5	-	
T(1;Y)B8	16D;YL	B ^S Yy ⁺	5	-	
T(1;Y)B9	2A;YS	B ^S Yy ⁺	5	-	
T(1;Y)B10	16C3-5;YL	B ^S Yy ⁺	2,5,6	+	+
T(1;Y)B12	20;Y	B ^S Yy ⁺	5	+	+
*T(1;Y)B13	15D;YL	B ^S Yy ⁺	5	+	+
T(1;Y)B16	11B;YS	B ^S Yy ⁺	5	-	
T(1;Y)B17	7C;YS	B ^S Yy ⁺	2,5	+	+
T(1;Y)B18	16C;YL	B ^S Yy ⁺	2,5,6	+	+
T(1;Y)B24	12E;YL	B ^S Yy ⁺	2,5,6	+	+
T(1;Y)B25	15C;YS	B ^S Yy ⁺	2,5	+	+
T(1;Y)B26	9C;YS	B ^S Yy ⁺	2,5	+	+
T(1;Y)B28	13D;YS	B ^S Yy ⁺	2,5,6	+	+
T(1;Y)B29	4B;YL	B ^S Yy ⁺	2,5,6	+	+
T(1;Y)B31 ^e	20A1-3;YS	B ^S Yy ⁺	2,5	+	+
T(1;Y)B32	12D-E;YL	B ^S Yy ⁺	2,5	+	+
T(1;Y)B34	20A5-B1;YL	B ^S Yy ⁺	2,5	+	+
T(1;Y)B35	15A;YS	B ^S Yy ⁺	2,5,6	+	+
T(1;Y)B36	5C;YS	B ^S Yy ⁺	2,5,6	+	+
T(1;Y)B37	15C;YL	B ^S Yy ⁺	2,5	+	+
T(1;Y)B38	20F1;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B39	11E-12A;YS	B ^S Yy ⁺	2,5,6	+	+
T(1;Y)B41	20B1-3;YL	B ^S Yy ⁺	2	+	+
T(1;Y)B43	20A1-3;YL	B ^S Yy ⁺	2,5	+	+
T(1;Y)B44	11A6-7;YL	B ^S Yy ⁺	2,5,6	+	+
T(1;Y)B45	11A6-7;YS	B ^S Yy ⁺	2,5	+	+
T(1;Y)B47	15C;YL	B ^S Yy ⁺	2,5	+	+
T(1;Y)B48	1A;3F;YS	B ^S Yy ⁺	5	+	+
T(1;Y)B49	11C-D;YL	B ^S Yy ⁺	2,5	+	+
T(1;Y)B50 ^e	18B9-10;YL	B ^S Yy ⁺	2,5	+	+
T(1;Y)B51	12F;YL	B ^S Yy ⁺	2,5	+	+
T(1;Y)B52	9A;YL	B ^S Yy ⁺	2,5	+	+
T(1;Y)B53	11A6-7;YS	B ^S Yy ⁺	2,5	+	+
T(1;Y)B54	12E;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B55 ^c	16F3-4;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B56	4F13-5A1;YL	B ^S Yy ⁺	2	+	+
T(1;Y)B57	9A1-2;YL	B ^S Yy ⁺	2	+	+
T(1;Y)B59	11C1-2;YL	B ^S Yy ⁺	2	+	+
T(1;Y)B60	9B;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B61 ^e	20B1-3;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B62 ^e	3D5-E1;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B67	10E;YL	B ^S Yy ⁺	2	+	+
T(1;Y)B75	16C-D;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B78	16C-D;YL	B ^S Yy ⁺	2	+	+
T(1;Y)B87	11A6-7;YL	B ^S Yy ⁺	2	+	+
T(1;Y)B88	11E-F;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B89	12B9-C1;YL	B ^S Yy ⁺	2	+	+
T(1;Y)B90	15A;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B91	20A5-B1;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B92 ^e	18A5-D1;YL	B ^S Yy ⁺	2	+	+

CHROMOSOMES - TRANSLOCATIONS

translocation	cytology	break in	ref ^α	male viable ?	T(1;Y)/O fertile ?	translocation	cytology	break in	ref ^α	male viable ?	T(1;Y)/O fertile ?
T(1;Y)B93 ^E	19F5-20A1;YL	B ^S Yy ⁺	2	+		T(1;Y)B171		B ^S Yy ⁺	1	+	
T(1;Y)B94	20A5-B1;YL	B ^S Yy ⁺	2	+		T(1;Y)B172 ^E	18A1-5;YL	B ^S Yy ⁺	2	+	
T(1;Y)B95	20A1-3;YL	B ^S Yy ⁺	2	+		T(1;Y)B173 ^E	19F2;YL	B ^S Yy ⁺	2	+	
T(1;Y)B96	20A1-3;YS	B ^S Yy ⁺	2	+		T(1;Y)B188	12F6-7;Y	B ^S Yy ⁺	1		
T(1;Y)B97	20B1-3;YL	B ^S Yy ⁺	2	+		T(1;Y)C1	complex	B ^S Yy ⁺	5	-	
T(1;Y)B99	20A1-3;YL	B ^S Yy ⁺	2	+		T(1;Y)C2	13E-F;YL	B ^S Yy ⁺	5	-	
T(1;Y)B100	7F5-10;YL	B ^S Yy ⁺	2	+	+	T(1;Y)C4	4C;YL	B ^S Yy ⁺	5	±	
T(1;Y)B101 ^E	19F1;YL	B ^S Yy ⁺	2	+		T(1;Y)C5	complex	B ^S Yy ⁺	5	-	
T(1;Y)B102	16C-D;YL	B ^S Yy ⁺	2	+		T(1;Y)C7	17A5-6;Y	B ^S Yy ⁺	5	+	-
T(1;Y)B103	20B1-3;YS	B ^S Yy ⁺	2	+		T(1;Y)C10	3E;7F;Y	B ^S Yy ⁺	5	+	+
T(1;Y)B104	9A1-2;YL	B ^S Yy ⁺	2	+		T(1;Y)D1	8C;YS	B ^S Yy ⁺	2	+	
T(1;Y)B105	10A;YS	B ^S Yy ⁺	2	+		T(1;Y)D2	8C;YS	B ^S Yy ⁺	2	+	
T(1;Y)B106	1C;YL	B ^S Yy ⁺	2	+	+	T(1;Y)D7 ^E	3C;YS	B ^S Yy ⁺	2	±	+
T(1;Y)B107 ^E	18E1-19D1;YL	B ^S Yy ⁺	2	+		T(1;Y)D8	11A;YL	B ^S Yy ⁺	2	+	
T(1;Y)B108	20A1-3;YL	B ^S Yy ⁺	2	+		T(1;Y)D9	11A6-7;YL	B ^S Yy ⁺	2	+	
T(1;Y)B109	20A1-3;YL	B ^S Yy ⁺	2	+		T(1;Y)D10	5D5-6;YS	B ^S Yy ⁺	2	+	
T(1;Y)B110	4D;YL	B ^S Yy ⁺	2	+	+	T(1;Y)D12	7C;YL	B ^S Yy ⁺	2	+	
T(1;Y)B111	11C;YL	B ^S Yy ⁺	2	+	+	T(1;Y)D15 ^E	18E1-19D1;YL	B ^S Yy ⁺	2	+	+
T(1;Y)B112	11A6-7;YL	B ^S Yy ⁺	2	+	+	T(1;Y)D16	?	B ^S Yy ⁺	1	+	
T(1;Y)B114	1F;YS	B ^S Yy ⁺	2	+	+	T(1;Y)D17	4B;YS	B ^S Yy ⁺	2	+	
T(1;Y)B115	16C-D;YL	B ^S Yy ⁺	2	+		T(1;Y)F22	20A5-B1;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B116	14C;YS	B ^S Yy ⁺	2	+	+	T(1;Y)G20	2B;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B117	13C-D;YL	B ^S Yy ⁺	2	+		T(1;Y)J1	13A;YL	B ^S Yy ⁺	5	+	+
T(1;Y)B118	12F;YL	B ^S Yy ⁺	2	+		T(1;Y)J2a	9B;YS	B ^S Yy ⁺	2,5,6	+	+
T(1;Y)B119 ^E	5A6-9;YS	B ^S Yy ⁺	2	+	+	T(1;Y)J6	20;Y	B ^S Yy ⁺	5	+	+
T(1;Y)B120	20A1-3;YL	B ^S Yy ⁺	2	+		T(1;Y)J8	8C;YL	B ^S Yy ⁺	2,5,6	+	+
T(1;Y)B121 ^E	20B1-3;YL	B ^S Yy ⁺	2	+		T(1;Y)J9	2C;YS	B ^S Yy ⁺	5	-	
T(1;Y)B122		B ^S Yy ⁺	1	+		T(1;Y)J10	17D-E;YS	B ^S Yy ⁺	2,5	+	+
T(1;Y)B123	7B;YS	B ^S Yy ⁺	2,5	+			(complex)				
T(1;Y)B124	12F;YL	B ^S Yy ⁺	2	+		T(1;Y)J11	20;Y	B ^S Yy ⁺	5	-	
T(1;Y)B125	15A;YS	B ^S Yy ⁺	2	+	+	T(1;Y)J14	1D;Y	B ^S Yy ⁺	5	-	
T(1;Y)B126 ^E	3C;YL	B ^S Yy ⁺	2	+	+	T(1;Y)J100 ^E	3C;YL	B ^S Yy ⁺	2	+	+
T(1;Y)B127	14A-B;YL	B ^S Yy ⁺	2	+	+	T(1;Y)J101	11E-F;YL	B ^S Yy ⁺	2	+	
T(1;Y)B128	12F6-7;YS	B ^S Yy ⁺	2	+		T(1;Y)J102	5A1-3;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B130	20A5-B1;YS	B ^S Yy ⁺	2	+		T(1;Y)J103	7B-C;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B131	2B;YL	B ^S Yy ⁺	2	+	+	T(1;Y)J104	6E;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B132	17A9-12;YS	B ^S Yy ⁺	2	+		T(1;Y)J105	6E;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B133	12E;YL	B ^S Yy ⁺	2	+	+	T(1;Y)J106	11E-F;YL	B ^S Yy ⁺	2	+	
T(1;Y)B134	20F1;YS	B ^S Yy ⁺	2	+		T(1;Y)J107	20A1-3;YL	B ^S Yy ⁺	2	+	+
T(1;Y)B135	9A;YL	B ^S Yy ⁺	2	+		T(1;Y)J108	4D;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B136	12C;YS	B ^S Yy ⁺	2	+	+	T(1;Y)J110	20A1-3;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B137	16F;YS	B ^S Yy ⁺	2	+	+	T(1;Y)L1a	12E;YL	B ^S Yy ⁺	5	+	+
T(1;Y)B138	7F6-8;YL	B ^S Yy ⁺	2	+		T(1;Y)L2a	9B;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B139	7F9-10;YL	B ^S Yy ⁺	2	+	+	T(1;Y)L4	17B-C;YL	B ^S Yy ⁺	5,6	+	+
T(1;Y)B140	16C-D;YL	B ^S Yy ⁺	2	+		T(1;Y)L5	16F;YL	B ^S Yy ⁺	5	+	+
T(1;Y)B141	3F4-5;YL	B ^S Yy ⁺	2	+		T(1;Y)M1	14A;Y	B ^S Yy ⁺	5	-	
T(1;Y)B142	15C;YS	B ^S Yy ⁺	2	+	+	T(1;Y)N3	20A5-B1;YL	B ^S Yy ⁺	2	+	+
T(1;Y)B143	13D;YL	B ^S Yy ⁺	2	+		T(1;Y)N5 ^E	19D1-F1;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B144	16F;YS	B ^S Yy ⁺	2	+		T(1;Y)O15	2B;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B145 ^E	20A1-3;YL	B ^S Yy ⁺	2	+		T(1;Y)P3	20;Y	B ^S Yy ⁺	5	+	+
T(1;Y)B146	12E9;YL	B ^S Yy ⁺	2	+		T(1;Y)P5	3C;Y	B ^S Yy ⁺	5	-	
T(1;Y)B147	16F;YS	B ^S Yy ⁺	2	+		T(1;Y)P6	18B;YL	B ^S Yy ⁺	5	±	
T(1;Y)B148	10B;YS	B ^S Yy ⁺	2	+		T(1;Y)P7a	11C-D;Y	B ^S Yy ⁺	5	-	
T(1;Y)B149	10A1-2;YL	B ^S Yy ⁺	2,8	+		T(1;Y)P8	18A;Y	B ^S Yy ⁺	5	+	-
T(1;Y)B150 ^E	19D1-F1;YL	B ^S Yy ⁺	2	+		T(1;Y)P11	18E1-19D1;YL	B ^S Yy ⁺	2	+	+
T(1;Y)B151	14C;YS	B ^S Yy ⁺	2	+	+	T(1;Y)P12	13F1-2;YS	B ^S Yy ⁺	2,5	+	+
T(1;Y)B152	9C;YL	B ^S Yy ⁺	2	+	+	T(1;Y)P21	15E;YL	B ^S Yy ⁺	5	+	+
T(1;Y)B153 ^E	2B8-10C;YS	B ^S Yy ⁺	2	±		T(1;Y)P22	3A;YS	B ^S Yy ⁺	5	-	
T(1;Y)B154	19F5-20A1;YL	B ^S Yy ⁺	2	+		T(1;Y)Q6	20A1-3;YL	B ^S Yy ⁺	2	+	+
T(1;Y)B155	20A1-3;YL	B ^S Yy ⁺	2	+		T(1;Y)R1 ^E	16F;YL	B ^S Yy ⁺	2	+	-
T(1;Y)B156	16D;YL	B ^S Yy ⁺	2	+		T(1;Y)R2	8D10-12;YL	B ^S Yy ⁺	2	+	-
T(1;Y)B157	20A1-3;YL	B ^S Yy ⁺	2	+		T(1;Y)R4	15A9-11;YS	B ^S Yy ⁺	1	+	-
T(1;Y)B158	10A;YL	B ^S Yy ⁺	2	+		T(1;Y)R8	20A1-3;YL	B ^S Yy ⁺	2	+	-
T(1;Y)B159	16C-D;YS	B ^S Yy ⁺	2	+	+	T(1;Y)R20	16C-D;YL	B ^S Yy ⁺	2	+	-
T(1;Y)B160		B ^S Yy ⁺	1	+		T(1;Y)R30 ^E	3C;YL	B ^S Yy ⁺	2	+	+
T(1;Y)B161 ^E	18E1-4;YS	B ^S Yy ⁺	2	+		T(1;Y)R34 ^E	18E1-4;YL	B ^S Yy ⁺	2	+	-
T(1;Y)B162	14A-B;YS	B ^S Yy ⁺	2	+	+	T(1;Y)R36 ^E	3C6;YL	B ^S Yy ⁺	2	+	-
T(1;Y)B163	4C-D;YS	B ^S Yy ⁺	2	+		T(1;Y)R44	7C;YL	B ^S Yy ⁺	2	+	-
T(1;Y)B164	18A1-5;YS	B ^S Yy ⁺	2	+		T(1;Y)S10	20A1-3;YS	B ^S Yy ⁺	2	+	+
T(1;Y)B165	18A1-5;YS	B ^S Yy ⁺	2	+		T(1;Y)S15	11B;YS	B ^S Yy ⁺	5	-	
T(1;Y)B166	12A-B;YS	B ^S Yy ⁺	2	+		T(1;Y)S19	14A3-5;YL	B ^S Yy ⁺	2	+	-
T(1;Y)B167	9B;YS	B ^S Yy ⁺	2	+		T(1;Y)S23	20;Y	B ^S Yy ⁺	5	+	+
T(1;Y)B168	9B;YL	B ^S Yy ⁺	2	+	+	T(1;Y)S27	7C;YL	B ^S Yy ⁺	2	+	-
T(1;Y)B169	20A1-3;YL	B ^S Yy ⁺	2	+		T(1;Y)S29	13B4-6;YS	B ^S Yy ⁺	2	+	-
T(1;Y)B170	8A;YS	B ^S Yy ⁺	2	+		T(1;Y)S30 ^E	20A1-3;YS	B ^S Yy ⁺	2	+	-

THE GENOME OF *DROSOPHILA MELANOGASTER*

translocation	cytology	break in	ref ^α	male viable ?	<i>T(1;Y)0</i> fertile ?
<i>T(1;Y)S31</i> ^E	18E1-19D1;YL	<i>B</i> ^S <i>Yy</i> ⁺	2	+	-
<i>T(1;Y)S33</i>	20;Y	<i>B</i> ^S <i>Yy</i> ⁺	5	+	+
<i>T(1;Y)S34</i>		<i>B</i> ^S <i>Yy</i> ⁺	1	+	-
<i>T(1;Y)S39</i>	11C;YL	<i>B</i> ^S <i>Yy</i> ⁺	2	+	-
<i>T(1;Y)T9</i>	12F2-5;YL	<i>B</i> ^S <i>Yy</i> ⁺	2	+	+
<i>T(1;Y)T12</i>	[prox. to v];YL	<i>B</i> ^S <i>Yy</i> ⁺	1	+	+
<i>T(1;Y)T16</i>	14B2-5;YS	<i>B</i> ^S <i>Yy</i> ⁺	2	+	+
<i>T(1;Y)V3</i>		<i>B</i> ^S <i>Yy</i> ⁺	1	+	+
<i>T(1;Y)V7</i> ^ζ	16F4-8;YS	<i>B</i> ^S <i>Yy</i> ⁺	2	+	+
<i>T(1;Y)V16</i>	6E1-3;YL	<i>B</i> ^S <i>Yy</i> ⁺	2	+	+
<i>T(1;Y)V29</i>	16F4-8;YL	<i>B</i> ^S <i>Yy</i> ⁺	2	+	+
<i>T(1;Y)V35</i>	5B3-9;YL	<i>B</i> ^S <i>Yy</i> ⁺	2	+	+
<i>T(1;Y)V46Df</i>	20A1-3;YL	<i>B</i> ^S <i>Yy</i> ⁺	2	+	+
<i>T(1;Y)V59</i>	11F-12A;YL	<i>B</i> ^S <i>Yy</i> ⁺	2	+	+
<i>T(1;Y)V61</i>	11D2-5;YS	<i>B</i> ^S <i>Yy</i> ⁺	2	+	+
<i>T(1;Y)V64</i>	20A1-3;YL	<i>B</i> ^S <i>Yy</i> ⁺	2	+	+
<i>T(1;Y)W14</i> ^E	5A9-13;YL	<i>B</i> ^S <i>Yy</i> ⁺	2	+	+
<i>T(1;Y)W15</i>		<i>B</i> ^S <i>Yy</i> ⁺	1	+	+
<i>T(1;Y)W16</i> ^E	19F1;YS	<i>B</i> ^S <i>Yy</i> ⁺	2	+	+
<i>T(1;Y)W17</i> ^E	3C;YL	<i>B</i> ^S <i>Yy</i> ⁺	2	+	+
<i>T(1;Y)W23</i>	13E1-7;YL	<i>B</i> ^S <i>Yy</i> ⁺	2	+	+
<i>T(1;Y)W31</i>	7E2-3;YS	<i>B</i> ^S <i>Yy</i> ⁺	2	+	+
<i>T(1;Y)W32</i> ^ζ	16F3-4;YS	<i>B</i> ^S <i>Yy</i> ⁺	2	+	+

^α 1 = Merriam; 2 = Merriam, Yamamoto, Stewart, Rahman, and Nicholau; 3 = Nicoletti and Lindsley, 1960, DIS 34: 95-97; 4 = Nicoletti and Lindsley, 1960, Genetics 45: 1705-22; 5 = Stewart and Merriam, 1973, DIS 50: 167-69; 6 = Stewart and Merriam, 1975, Genetics 79: 635-47; 7 = Young and Judd, 1978, Genetics 88: 723-42; 8 = Zhimulev, Belyaeva, Pokholkova, Kochneva, Fomina, Bgatov, Khudyakov, Patzевич, Semeshin, Baricheva, Aizenzon, Kramers, and Eeken, 1981, DIS 56: 192-96.

^β New order:
 $T(1;Y)2 = 1 - 5E|YS^P - YL;$
 $20 - 19F|11F - 5E|19F - 11F|YS^D.$
 $*T(1;Y)9 = 1A - 2C|YS^P - YL;$
 $20 - 19F|2C - 19F|YS^D.$
 $*T(1;Y)290 = 1A|YS^P - YL;$
 $20F - 20A|1A - 20A|YS^D.$

^γ 3C break between *N* and *dm* (Young and Judd, 1978).
^δ Variiegates for *w* and *N* (Nicoletti and Lindsley, 1960).
^ε Break locations inferred from crosses to deletions or lethals.

^ζ Molecular biology: *T(1;Y)B55* maps between +33.4 and +34.9 kb and *T(1;Y)W32* maps between +95.2 and +98.7 kb (Kamb, Iverson, and Tanouye, 1987, Cell 50: 405-13). These and *T(1;Y)V7* display leg shaking owing to their disruption of the *Sh* locus (Tanouye, Ferrus, and Fujita, 1981, Proc. Nat. Acad. Sci. USA 78: 6548-52).

Table II: Translocations with breaks in X heterochromatin.

cytology: Breakpoints on the cytogenetic map of the *Y* (Gatti and Pimpinelli, 1983, Chromosoma 88: 349-73) and the *X* heterochromatin (Gatti and Pimpinelli, unpublished) inferred from the cytogenetic data of Kennison (1981, Genetics 98: 529-48) or, in a few cases, determined by Bonaccorsi. *X* breakpoints designated *h26-h29* are distal to *bb*, those designated *h29* are within *bb* and those designated *h29-h33* are proximal to *bb*. *Y* breaks between *B^SXh* and *h17* are in *YL*; those between *h18* and *Xhy⁺* are in *YS* with *bb* occupying *h20*.

origin: γ ray induced using *B^SYy⁺*.

discoverer: Kennison.

genetics: Male viable. Fertility of males indicated in table.

translocation	cytology ^α	ref ^β	male fertility
<i>T(1;Y)E1</i>	<i>h26-h29;h11</i>	1,2	+
<i>T(1;Y)E5</i>	<i>h29;B^S-h1</i>	2	+
<i>T(1;Y)E12</i>	<i>h29;B^S-h1</i>	2	+
<i>T(1;Y)E15</i>	<i>h26-h29;h12-13</i>	1,2	+
<i>T(1;Y)E17</i>	<i>h29-h33;B^S-h1</i>	2	+
<i>T(1;Y)F6</i>	<i>h29-h33;B^S-h1</i>	2	+

translocation	cytology ^α	ref ^β	male fertility
<i>T(1;Y)F12</i>	<i>h29;h15-h17</i>	2	+
<i>T(1;Y)F14</i>	<i>h26-h29;h15-h17</i>	2	+
<i>T(1;Y)F15</i>	<i>h29;h15-h17</i>	2	+
<i>T(1;Y)G1</i>	<i>h29-h33;h18-h20</i> or <i>h26-h29;h20-h21</i>	2	+
<i>T(1;Y)G7</i>	<i>h29-h33;h24-y⁺</i>	2	+
<i>T(1;Y)G8</i>	<i>h26-h29;h11-h12</i>	1,2	+
<i>T(1;Y)G15</i>	<i>h26-h29;B^S-h1</i>	2	+
<i>T(1;Y)G22</i>	<i>h29;B^S-h1</i>	2	+
<i>T(1;Y)G24</i>	<i>h26-h29;B^S-h1</i>	2	+
<i>T(1;Y)G25</i>	<i>h26-h29;h13</i>	1,2	+
<i>T(1;Y)G29</i>	<i>h29-h33;h18-h20</i> or <i>h26-h29;h20-h21</i>	2	+
<i>T(1;Y)G30</i>	<i>h29;B^S-h1</i>	2	+
<i>T(1;Y)J2</i>	<i>h29-h33;h11-h13</i>	2	+
<i>T(1;Y)K1</i>	<i>h29;B^S-h1</i>	2	+
<i>T(1;Y)L1</i>	<i>h29-h33;h20</i> or <i>h29;h20-h25</i>	2	+
<i>T(1;Y)L2</i>	<i>h29-h33;h2-h3</i>	2	<i>kl-5</i>
<i>T(1;Y)N1</i>	<i>h29-h33;h18-h20</i> or <i>h26-h29;h20-h21</i>	2	+
<i>T(1;Y)N10</i>	<i>h29-h33;h25-y⁺</i>	2	+
<i>T(1;Y)N12</i>	<i>h26-h29;h25-y⁺</i>	2	+
<i>T(1;Y)N13</i>	<i>h26-h29;h18-h20</i>	2	+
<i>T(1;Y)N14</i>	<i>h26-h29;h18-h20</i>	2	+
<i>T(1;Y)N16</i>	<i>h29;B^S-h1</i>	2	+
<i>T(1;Y)N18</i>	<i>h26-h29;h2-h3</i>	2	<i>kl-5</i>
<i>T(1;Y)N20</i>	<i>h29;h2-h3</i>	2	<i>kl-5</i>
<i>T(1;Y)N29</i>	<i>h29-h33;h11</i>	1,2	+
<i>T(1;Y)N30</i>	<i>h29-h33;h25-y⁺</i>	2	+
<i>T(1;Y)P1</i>	<i>h29-h33;h25-y⁺</i>	2	+
<i>T(1;Y)P7</i>	<i>h29-h33;h11</i>	1,2	+
<i>T(1;Y)P9</i>	<i>h29;B^S-h1</i>	2	+
<i>T(1;Y)R5</i>	<i>h29-h33;h10</i>	2	<i>kl-2</i>
<i>T(1;Y)R12</i>	<i>h26-h29;h25-y⁺</i>	2	+
<i>T(1;Y)R17</i>	<i>h29-h33;h4-h7</i>	2	+
<i>T(1;Y)R19</i>	<i>h29-h33;h10</i>	1,2	<i>kl-2</i>
<i>T(1;Y)R21</i>	<i>h29-h33;h2-h3</i>	2	<i>kl-5</i>
<i>T(1;Y)R29</i>	<i>h29-h33;h2-h3</i>	2	<i>kl-5</i>
<i>T(1;Y)R36</i>	<i>h29;h2-h3</i>	2	<i>kl-5</i>
<i>T(1;Y)R39</i>	<i>h29-h33;h8-h9</i>	2	<i>kl-3</i>
<i>T(1;Y)R43</i>	<i>h29-h33;h15-h17</i>	2	+
<i>T(1;Y)S1</i>	<i>h29-h33;h8-h9</i>	2	<i>kl-3</i>
<i>T(1;Y)S2</i>	<i>h26-h29;h8-h9</i>	2	<i>kl-3</i>
<i>T(1;Y)S7</i>	<i>h29-h33;h11-h13</i>	2	+
<i>T(1;Y)S13</i>	<i>h26-h29;h11-h13</i>	2	+
<i>T(1;Y)S20</i>	<i>h29-h33;h2-h3</i>	2	<i>kl-5</i>
<i>T(1;Y)S24</i>	<i>h29-h33;h8-h9</i>	2	<i>kl-3</i>
<i>T(1;Y)S25</i>	<i>h26-h29;h2-h3</i>	2	<i>kl-5</i>
<i>T(1;Y)S28</i>	<i>h29-h33;h11-h13</i>	2	+
<i>T(1;Y)S32</i>	<i>h29-h33;h4-h7</i>	2	+
<i>T(1;Y)S38</i>	<i>h29;h4-h7</i>	2	+
<i>T(1;Y)T2</i>	<i>h29-h33;h2-h3</i>	2	<i>kl-5</i>
<i>T(1;Y)T4</i>	<i>h29;h2-h3</i>	2	<i>kl-5</i>
<i>T(1;Y)T6</i>	<i>h29-h33;h24-h25</i>	2	<i>ks-2</i>
<i>T(1;Y)T10</i>	<i>h26-h29;h21-h23</i>	2	<i>ks-1</i>
<i>T(1;Y)T11</i>	<i>h29-h33;h2-h3</i>	2	<i>kl-5</i>
<i>T(1;Y)T13</i>	<i>h29-h33;h8-h9</i>	2	<i>kl-3</i>
<i>T(1;Y)T17</i>	<i>h26-h29;h24-h25</i>	2	<i>ks-2</i>
<i>T(1;Y)T20</i>	<i>h26-h29;h25-y⁺</i>	2	+
<i>T(1;Y)T21</i>	<i>h26-h29;h21-h23</i>	2	<i>ks-1</i>
<i>T(1;Y)V4</i>	<i>h29-h33;h25-y⁺</i>	2	+
<i>T(1;Y)V8</i>	<i>h26-h29;h24</i>	2	+
<i>T(1;Y)V13</i>	<i>h29-h33;B^S-h1</i>	2	+
<i>T(1;Y)V14</i>	<i>h26-h29;h18-h20</i>	2	+
<i>T(1;Y)V23</i>	<i>h29-h33;h25-y⁺</i>	2	+
<i>T(1;Y)V24</i>	<i>h29-h33;h4-h7</i>	2	+
<i>T(1;Y)V25</i>	<i>h29-h33;h25-y⁺</i>	2	+
<i>T(1;Y)V27</i>	<i>h26-h29;B^S-h1</i>	2	+
<i>T(1;Y)V30</i>	<i>h29-h33;h10</i>	2	<i>kl-2</i>
<i>T(1;Y)V31</i>	<i>h29-h33;h13</i>	1,2	+
<i>T(1;Y)V32</i>	<i>h29-h33;h18-h20</i> or <i>h26-h29;h20-h21</i>	2	+
<i>T(1;Y)V33</i>	<i>h29-h33;h21-h23</i>	2	<i>ks-1</i>
<i>T(1;Y)V36</i>	<i>h29-h33;h25-y⁺</i>	2	+

translocation	cytology α	ref β	male fertility
T(1;Y)V43	<i>h26-h29;h12-13</i>	1,2	+
T(1;Y)V47	<i>h26-h29;h4-h7</i>	2	+
T(1;Y)V54	<i>h29-h33;h21-h23</i>	2	<i>ks-1</i>
T(1;Y)V57	<i>h29-h33;h8-h9</i>	2	<i>kl-3</i>
T(1;Y)V63	<i>h26-h29;h11-h12</i>	1,2	+
T(1;Y)W1	<i>h29-h33;h21-h23</i>	2	<i>ks-1</i>
T(1;Y)W2	<i>h26-h29;B^S-h1</i>	2	+
T(1;Y)W3	<i>h29-h33;h15-h17</i>	2	+
T(1;Y)W8	<i>h29-h33;B^S-h1</i>	2	+
T(1;Y)W9	<i>h26-h29;h21-h23</i>	2	-
T(1;Y)W19	<i>h29-h33;h20-h21</i>	2	+
T(1;Y)W28	<i>h29-h33;h15-h17</i>	2	+
T(1;Y)W29	<i>h29-h33;h18-h20 or h26-h29;h20-h21</i>	2	+
T(1;Y)W30	<i>h26-h29;B^S-h1</i>	2	+
T(1;Y)W33	<i>h29-h33;h15-h17</i>	2	+
T(1;Y)Z2	<i>h26-h29;h11-h13</i>	2	+
T(1;Y)Z3	<i>h26-h29;h21-h23</i>	2	<i>ks-1</i>
T(1;Y)Z6	<i>h29-h33;h14</i>	1,2	<i>kl-1</i>
T(1;Y)Z13	<i>h29-h33;h21-h23</i>	1,2,3	<i>ks-1</i>
T(1;Y)Z14	<i>h29-h33;h25-y⁺</i>	2	+

α The heterochromatic proximal part of the X has been divided by Gatti into 9 sections, *h26-h34*, distinguishable in neuroblast chromosomes by special staining techniques; the heterochromatic Y had previously been divided (Gatti and Pimpinelli, 1983) into 25 sections, *h1-h25*. The normal position of the X centromere is at *h33-h34* and of the Y centromere at *h17-h18*.

β 1 = Hardy, Lindsley, Livak, Lewis, Sivertsen, Joslyn, Edwards, and Bonaccorsi, 1984, *Genetics* 107: 591-610; 2 = Kennison, 1981, *Genetics* 98: 529-48; 3 = Kennison, 1983, *Genetics* 103: 219-34.

T(1;Y)1E: see **Dp(1;Y)1E**

T(1;Y)2E: see **Dp(1;Y)2E**

T(1;Y)B132

cytology: *T(1;Y)17A9-12*.

origin: X ray induced.

references: Baumann, Krah-Jentgens, Müller, Müller-Holtkamp, Seidel, Kecskemethy, Casal, Ferrus, and Pongs, 1987, *EMBO J.* 6: 3419-29.

genetics: No *Sh* phenotype.

T(1;YS)118-2b: see **X.YSYL118-2b**

T(1;YS)A3: see **X.YSA3**

***T(1;Y;2)**

origin: X ray induced.

discoverer: Nicoletti.

references: Nicoletti and Lindsley, 1960, *Genetics* 45: 1705-22. 1960, *DIS* 34: 95-97.

translocation	cytology	new order α	genetics
*T(1;Y;2)7	<i>14F;YS + YL;36C</i>	1	male lethal
T(1;Y;2)17	<i>7B;YL;39</i>	2	male viable but sterile
T(1;Y;2)109β	<i>3C;YL;40-41</i>		male viable but sterile
*T(1;Y;2)110	<i>19D;55F + YS;45F</i>	3	male viable but sterile
*T(1;Y;2)130β	<i>11F;YL;40-41</i>		male viable but sterile
*T(1;Y;2)146	<i>7D;YL + 20A;57F</i>	4	male viable but sterile
*T(1;Y;2)153	<i>17A;YS;35D</i>	5	male lethal
*T(1;Y;2)160β	<i>17C;YS;40-41</i>		male viable but sterile

α 1 = 1 - 14F^PYS^P - YL^P 36C - 21; 20 - 14F^PYS^D; YL^D 36C - 60.

2 = 1 - 7B^P39 - 60; 20 - 7B^PYL^D; YS - YL^P 39 - 21.

3 = 1 - 19D^P55F - 45F^PYS^P - YL; 20 - 19D^P55F - 60;

YS^D 45F - 21.
4 = 1 - 7D^PYL^P - YS;
20F - 20A^P57F - 60;
YL^D 7D - 20A^P57F - 21.
5 = 1 - 17A^P35D - 60;
20 - 17A^PYS^D;
YL - YS^P 35D - 21.

β Involvement of chromosome 2 inferred from genetic data; rearrangement not cytologically observable in polytene chromosome preparations.

T(1;Y;2)C8

cytology: *T(1;Y;2)12E;Y;26*.

origin: X ray induced in *B^SYy⁺*.

references: Stewart and Merriam, 1973, *DIS* 50: 167-69.

genetics: Lethal.

T(1;Y;2;3)B46

cytology: *T(1;Y;2;3)12E;Y;2R;3R*.

origin: X ray induced in *B^SYy⁺*.

references: Stewart and Merriam, 1973, *DIS* 50: 167-69.

genetics: Viable, fertile.

***T(1;Y;3)**

origin: X ray induced.

discoverer: Nicoletti.

references: Nicoletti and Lindsley, 1960, *Genetics* 45: 1705-22. 1960, *DIS* 34: 95-97.

translocation	cytology	new order α	genetics
*T(1;Y;3)5	<i>11D;YL + 14F;72</i>	1	male viable but sterile
T(1;Y;3)121	<i>6F;YS;86D</i>	2	male lethal
T(1;Y;3)127	<i>19F;YS;85E</i>	3	male viable but sterile
*T(1;Y;3)134	<i>12E;YS + 19E;62A</i>	4	male viable but sterile
*T(1;Y;3)136	<i>7A;YL;70C</i>	5	male viable but sterile
*T(1;Y;3)138	<i>11A;YL;84B</i>	6	male viable but sterile
T(1;Y;3)143	<i>12A;YL + 3F;69C</i>	7	male viable, sterile with free Y, lethal without
T(1;Y;3)144	<i>15E;YL;74D</i>	8	male viable, sterile with free Y, lethal without
T(1;Y;3)154β	<i>10B1-2;YS;97F11</i>	9	male viable but sterile
T(1;Y;3)161	<i>17A;YL;94</i>	10	male lethal

α 1 = 1 - 11D^PYL^P - YS;
20 - 14F^P72 - 61;
YL^D 11D - 14F^P72 - 100.

2 = 1 - 6F^PYS^P - YL;
20 - 6F^P86D - 100;
YS^D 86D - 61.

3 = 1 - 19F^P85E - 61;
20 - 19F^PYS^D;
YL - YS^P 85E - 100.

4 = 1 - 12E^PYS^P - YL;
20F - 19E^P62A - 61;
YS^D 12E - 19E^P62A - 100.

5 = 1 - 7A^P70C - 100;
20 - 7A^PYL^D;
YS - YL^P 70C - 61.

6 = 1 - 11A^P84B - 61;
20 - 11A^PYL^D;
YS - YL^P 84B - 100.

7 = 1 - 3F^P69C - 100;
20 - 12A^PYL^D;
YS - YL^P 12A - 3F^P69C - 61.

8 = 1 - 15E^P74D - 100;
20 - 15E^PYL^D;
YS - YL^P 74 - 61.

9 = 1 - 10A^P97A - 61;
20 - 10A^PYS^D;
YL - YS^P 97A - 100.

10 = 1 - 17A^P94 - 61;
20 - 17A^PYL^D;
YS - YL^P 94 - 100.

^β Other references: Zhimulev, Belyaeva, Khudyakov, and Pokholkova, 1980, DIS 55: 211; Zhimulev, Pokholkova, Bgatov, Semeshin, and Belyaeva, 1981, Chromosoma 82: 25-40; Zhimulev, Semeshin, and Belyaeva, 1981, Chromosoma 82: 9-23.

T(1;Y;3)J4

cytology: *T(1;Y;3)11A;Y;87*.
origin: X ray induced in *B^SYy⁺*.
references: Stewart and Merriam, 1973, DIS 50: 167-69.
genetics: Lethal.

T(1;Y;3)V6

cytology: *T(1;Y;3)1A;14B;18B1;YL;63A*.
origin: X ray induced in *B^SYy⁺*.
references: Merriam, Stewart, Nicholau, Yamamoto, and Rahman.
genetics: Male viable.

T(1;Y;3)V17

cytology: *T(1;Y;3)h29-h30;h11-12;75F-76A* (*Y* breaks from Gatti and Pimpinelli, 1983, Chromosoma 88: 349-73).
new order:
 1 – h29|h12 – Xhy⁺;
 h34 – h30|76A – 61;
 XhB^S – h11|76A – 100.
origin: γ ray induced in *B^SYy⁺*.
references: Kennison, 1981, Genetics, 98: 529-48.
 Hardy, Lindsley, Livak, Lewis, Sivertsen, Joslyn, Edwards, and Bonaccorsi, 1984, Genetics 107: 591-610.
genetics: Male viable and fertile.

T(1;Y;3)W27

cytology: *T(1;Y;3)h29-h30;h9-h10;91A + In(Y)h10;h20* (*Y* breaks from Gatti and Pimpinelli, 1983, Chromosoma 88: 349-73).
new order:
 1 – h29|h20 – h10|h20 – Xhy⁺;
 h34 – h30|91A – 100;
 XhB^S – h9|91A – 61.
origin: γ ray induced in *B^SYy⁺*.
references: Kennison, 1981, Genetics, 98: 529-48.
 Hardy, Lindsley, Livak, Lewis, Sivertsen, Joslyn, Edwards, and Bonaccorsi, 1984, Genetics 107: 591-610.
genetics: Male viable and fertile. Males carrying *3R^PY^D* and *X^P3R^D* from *T(1;Y;3)W27* and *Y^PX^D* from *T(1;Y)E15* are deficient for *kl-2* and flanking sequences (Hardy *et al.*, 1984).

T(1;Y;4)116

cytology: *T(1;Y)14D;YS + T(1;4)9C;101*.
new order:
 1 – 9C|101;
 20 – 14D|YS^D;
 YL – YS^P|14D – 9C|102.
origin: X ray induced in *B^SY*.
discoverer: Nicoletti.
references: Nicoletti and Lindsley, 1960, Genetics 45: 1705-22.
 1960, DIS 34: 95-97.
genetics: Male viable and sterile.

T(1;Y;4)B^S: Translocation (1;Y;4) Bar of Stone

cytology: A derivative of *T(1;4)B^S* in which the X centromere has been replaced by a Y chromosome whose other arm (presumably YL) is derived from *y⁺Y*.
references: Novitski and Peacock, 1970, DIS 45: 95-96.
genetics: Males are viable with low fertility.

T(1;Y;A)V63

cytology: *T(1;Y;A)h26-h29;h11-12;A* (*Y* breaks from Gatti and Pimpinelli, 1983, Chromosoma 88: 349-73).
new order:
 1 – h26|h12 – Xhy⁺;
 h34 – h29|A^D;
 XhB^S – h11|A^P.
origin: γ ray induced in *B^SYy⁺*.
references: Kennison, 1981, Genetics, 98: 529-48.
 Hardy, Lindsley, Livak, Lewis, Sivertsen, Joslyn, Edwards, and Bonaccorsi, 1984, Genetics 107: 591-610.
genetics: Male viable and fertile.

T(1;2)1.10

cytology: *T(1;2)4A3-4;24B*.
origin: X ray induced.
discoverer: Nüsslein-Volhard.
synonym: *T(1;2)odd^{1.10}*.
references: Nüsslein-Volhard, Kluding, and Jürgens, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 145-54.
genetics: Male fertile. Second-chromosome breakpoint between *odd* and *slp*; neither gene mutant. The crossing scheme described should not have yielded X-autosome translocations.

T(1;2)2A4

cytology: *T(1;2)11B;33E*.
origin: Induced by ethyleneimine.
references: Lim and Snyder, 1968, Mutat. Res. 6: 129-37.

***T(1;2)7**

origin: X ray induced.
discoverer: Bonner, 1931.
references: Dobzhansky, 1935, Z. Indukt. Abstamm. Vererbungsl. 68: 134-62.
genetics: X broken between *rb* and *cv*; chromosome 2 to the right of *sp*. Male and heterozygous female viable and fertile; homozygous female poorly viable and sterile. *X^P2^D* recoverable as an aneuploid segregant that is duplicated for the loci of *y* through *rb* but is not demonstrably deficient for 2R markers; nothing written to indicate that it is deficient for *M(2)c*.

T(1;2)26: see *Tp(1;2)26*

T(1;2)27A2

cytology: *T(1;2)8E;57B*.
origin: Induced by ethyleneimine.
references: Lim and Snyder, 1968, Mutat. Res. 6: 129-37.

T(1;2)63i: see *Tp(1;2)v⁺63i*

T(1;2)65b: see *Tp(1;2)v^{65b}*

***T(1;2)106**

origin: X ray induced.
discoverer: Sturtevant, 1930.
genetics: Break in X chromosome near centromere to right of *f*; break in chromosome 2 near centromere, probably in 2L. Male fertile; homozygous female viable and fertile.

Crossing over and disjunction for both chromosomes *X* and 2 normal in *T(1;2)106/+* female. *T(1;2)106/+Y* female shows nondisjunction of *X*'s.

****T(1;2)271b***

cytology: *T(1;2)3C3-7;40*; inferred from figs. 15A, G, and H of Mackensen (1935).

origin: X ray induced.

discoverer: Patterson.

synonym: *Df(1)Del271b*.

references: 1932, Am. Nat. 66: 193-206.

Mackensen, 1935, J. Heredity 26: 163-74 (fig.).

genetics: Mutant for *N*.

T(1;2)429.44

cytology: *T(1;2)2B12-14;30B1-2*.

discoverer: Gelbart.

T(1;2)A18

cytology: *T(1;2)2A4;60E8-9*.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal. Mutant for *br*.

****T(1;2)A61b***

cytology: *T(1;2)15F*; breakpoint in chromosome 2 at unknown position in left arm, which also carries an inversion. Breakpoint in *X* inferred from fig. 17G of Mackensen (1935).

references: Mackensen, 1935, J. Heredity 26: 163-74 (fig.).

genetics: Mutant for *f*.

T(1;2)A64

cytology: *T(1;2)2B7;42*.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal. Mutant for *br*.

T(1;2)A1125: see *Tp(1;2)rb⁺71g*

****T(1;2)B^{48g}*: Translocation (1;2) Bar**

cytology: *T(1;2)15F-16A1;33B* superimposed on *In(1)1B3-4;19F-20C1*.

new order:

1A1 - 1B3|19F - 16A1|33B - 60;

20F - 20C1|1B4 - 15F|33B - 21.

origin: X ray induced in *In(1)sc⁴*.

discoverer: Yu, 48g.

genetics: Position effect at *B*. Male sterile.

***T(1;2)B^{bd}*: Translocation (1;2) Bar-baroid**

cytology: *T(1;2)16A1-2;48C2-3* + *In(2R)41A;47A* (Bridges in Morgan, Bridges, and Schultz, 1936, Year Book - Carnegie Inst. Washington 35: 291).

new order:

1 - 16A1|48C2 - 47A|41A - 47A|41A - 21;

20 - 16A2|48C3 - 60.

origin: X ray induced simultaneously with *In(2R)41-47*.

discoverer: Dobzhansky, 31b5.

references: 1932, Genetics 17: 369-92.

genetics: Recessive position effect for *B*. Translocation and inversion probably not separable.

T(1;2)B27

origin: Induced in a *Sh¹⁴* chromosome.

references: Baumann, Krah-Jentgens, Müller, Müller-Holtkamp, Seidel, Kecskemethy, Casal, Ferrus, and

Pongs, 1987, EMBO J. 6: 3419-29.

genetics: Associated with modified *Sh* phenotype.

molecular biology: Maps between 42 and 43 kb proximal to the start site of the *Sh* chromosomal walk.

***T(1;2)Ba¹⁸*: Translocation (1;2) Brista (J. Kennison)**

cytology: *T(1;2)3C3-7;60D-E* (J. Kennison).

origin: Spontaneous.

discoverer: Williams.

synonym: *Art; Dll^w; Ba^w*.

references: Cohen, Bronner, Küttner, Jürgens, and Jackle, 1989, Nature 338: 432-34.

molecular biology: 60D-E located on molecular map (Cohen et al., 1989).

genetics: Associated with *Ba¹⁸*. 2^{PX^D} element is duplicated for the loci of *y* and *w*, deficient for *M(2)60E* and *Kr* and has the dominant *Ba* phenotype.

***T(1;2)bi^{D2}*: Translocation (1;2) bifid**

cytology: *T(1;2)4C5-6;46B5-7*.

origin: X ray induced.

references: Banga, Bloomquist, Brodberg, Pye, Larrivee, Mason, Boyd, and Pak, 1986, Chromosoma 93: 341-46.

genetics: Associated with *bi*, *Qd*, and *omb*. Male lethal.

***T(1;2)Bld*: Translocation (1;2) Blond**

cytology: *T(1;2)1C3-4;60B12-13* + *In(2R)42A2-3;58A4-B1*.

new order:

1A - 1C3|60B12 - 58B1|42A3 -

58A4|42A2 - 21;

20 - 1C4|60B13 - 60F5.

origin: Spontaneous in *In(2R)Cy*.

discoverer: Burkart, 1930.

references: 1931, Rev. Fac. Argon. Vet. Univ. Buenos Aires 7: 393-491.

Burkart and Stern, 1933, Z. Indukt. Abstamm. Vererbungsl. 64: 310-25.

Bridges, 1937, Cytologia (Tokyo), Fujii Jub., Vol. 2: 745-55.

Morgan, Bridges, and Schultz, 1938, Year Book - Carnegie Inst. Washington 37: 307.

Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

genetics: Associated with *Bld*. Both aneuploid segregants survive. The 2^{PX^D} element is duplicated for *y*, *ac*, *sc*, *Hw*, *svr*, *su(s)*, *l(1)1Ab*, *su(b)*, and *M(1)1B* and deficient for *sp*, *bs*, *ba*, *Pin*, and *M(2)60E*; heterozygote extreme Plexate and slight Minute with small dark body and slow development; viability low; male sterile; female slightly fertile. X^{P2^D} is reciprocally duplicate deficient; heterozygous female Blond and extreme Minute [*M(1)1B*] with short, broad, occasionally downward-curved wings; ecloses 3-4 days late; male lethal, with embryos showing no organized ventral nerve cord structure but only some cell clusters in the brain lobes (Jiménez and Campos-Ortega, 1979, Nature 282: 310-12).

***T(1;2)C*: Translocation (1;2) Crossover suppressor**

origin: X ray induced; selected on the basis of reduced recombination in one chromosome arm.

discoverer: Roberts, 1964, 1965.

references: 1970, Genetics 65: 429-48.

translocation	cytology	new order ^α	male viable?	male fertile?	Xover reduced in
T(1;2)C6	12E;40-41;60B	1	-	-	2R
T(1;2)C20	12E;30B		+	-	2L
T(1;2)C54	12E;32F		-	-	2L
T(1;2)C60	20;52B		+	+	2R
T(1;2)C171	12A;40-41		+	-	X
T(1;2)C176	20;40-41 + <i>In(1)8C-D;18D</i>	2	-	-	X
T(1;2)C179	9A;49A + <i>In(1)5C;20</i>	3	-	-	X
T(1;2)C183	12E;40-41 + <i>In(2L)24C;30A</i>	4	-	-	2L
T(1;2)C256	2A;40-41 + <i>In(1)7E;17A;18B</i>	5	-	-	X
T(1;2)C261	14C;40-41		+	-	X
T(1;2)C262	11A;18A;40-41	6	-	-	X
T(1;2)C314	5D;40-41 + 9D;51D + 20;56F	7	+	-	X, 2R
T(1;2)C324^β	15F;20;30A	8	-	-	2L
T(1;2)C349	6C;47D + <i>In(1)2E;20</i>	9	-	-	X
T(1;2)C357	20;56F		+	-	2R

- ^α 1 = 1 - 12E|41 - 60B|40 - 21;
20 - 12E|60B - 60F.
2 = 1 - 8C|18D - 8D|18D - 20|41 - 60;
20|40 - 21 (tentative).
3 = 1 - 5C|20 - 9A|49A - 21;
20|5C - 9A|49A - 60.
4 = 1 - 12E|40 - 60;
20 - 12E|40 - 30A|24C - 30A|24C - 21 (tentative).
5 = 1 - 2A|41 - 60;
20 - 18B|17A - 18B|7E - 17A|7E - 2A|40 - 21.
6 = 1 - 11A|41 - 60;
20 - 18A|11A - 18A|40 - 21 (tentative).
7 = 1 - 5D|41 - 51D|9D - 5D|40 - 21;
20|56F - 51D|9D - 20|56F - 60 (tentative).
8 = 1 - 15F|20 - 15F|30A - 60;
20|30A - 21.
9 = 1 - 2E|20 - 6C|47D - 21;
20|2E - 6C|47D - 60.
^β Mutant or deficient for *f*.

T(1;2)C33 - T(1;2)C238

origin: X ray induced.
discoverer: Lefevre.
genetics: Male lethal.

translocation	cytology	mutant for
T(1;2)C33	2E3;41D	<i>kz</i>
T(1;2)C123	2B6;54A	<i>br</i>
T(1;2)C238	19E1-2;35A-B	<i>leg</i>

T(1;2)cl^{CA1}: Translocation (1;2) clot

cytology: *T(1;2)IDE;25D7-E4*.
origin: X ray induced.
discoverer: Velissariou.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.
Velissariou and Ashburner, 1980, Chromosoma 77: 13-27.

genetics: Associated with *cl^{CA1}*.

T(1;2)Clv³: Translocation (1;2) Cloven

cytology: *T(1;2)11A7-8;27E2-3* (Kirschbaum).
origin: X ray induced in *Binsc* chromosome.
references: Maroni, 1968, DIS 43: 60.
genetics: Mutant for *Clv*. Male semi-lethal and sterile.

***T(1;2)ct^{7a1}: Translocation (1;2) cut**

cytology: *T(1;2)7B*; other breakpoints not recorded.
origin: X ray induced in *R(1)2*.
discoverer: Hannah, 1947.
genetics: Mutant for *ct*; male lethal.

***T(1;2)ct²⁶⁸**

origin: X ray induced.
genetics: Male lethal.

translocation	cytology	discoverer ^α	genotype
*T(1;2)ct²⁶⁸⁻¹⁷	7B2-5;41E2-4 (Hoover)	1	<i>scp⁺ ct sn⁺</i>
*T(1;2)ct²⁶⁸⁻²⁴	7B2-5;41F6-42A1	2	<i>scp⁺ ct sn⁺</i>
*T(1;2)ct²⁶⁸⁻²⁶	7B3-C1;36E	2	<i>scp⁺ ct sn⁺</i>
*T(1;2)ct^{268-32^β}	1E-F;3D-E;7B2-5;46 (Hoover)	1	<i>fa⁺ dm⁺ scp⁺ ct sn⁺</i>
*T(1;2)ct²⁶⁸⁻³³	7B2-5;41E (Hoover)	1	<i>ct sn⁺</i>
*T(1;2)ct²⁶⁸⁻⁴¹	7B2-5;37C2-3 (Sutton)	1	<i>cm⁺ ct sn⁺</i>

^α 1 = Demerec; 2 = Hoover.
^β New order: 1A - 1E|3E - 7B2|46 - 21;
20 - 7B5|3D - 1F|46 - 60.

T(1;2)ct^{J1}

cytology: *T(1;2)7B3-4;60E*.
origin: X ray induced.
references: Lefevre and Johnson, 1973, Genetics 74: 633-45.
genetics: Male lethal but somewhat viable with *ct^{71g}*.

T(1;2)ct^{J5}

cytology: *T(1;2)7B3-4;40*.
origin: X ray induced.
references: Lefevre and Johnson, 1973, Genetics 74: 633-45.
genetics: Male lethal but viable with *ct^{71g}*.

***T(1;2)D1: Translocation (1;2) from deoxycytidine**

cytology: *T(1;2)6F;26C*.
origin: Induced by tritiated deoxycytidine.
discoverer: Kaplan, 1965.
references: 1966, DIS 41: 59.
genetics: Male lethal.

T(1;2)D2

cytology: *T(1;2)8B;46B*.
origin: Induced by tritiated deoxycytidine.
discoverer: Kaplan, 1965.
references: 1966, DIS 41: 59.
genetics: Male lethal.

T(1;2)DEB: Translocation (1;2) diepoxybutane

origin: Induced by diepoxybutane.
references: Denell, Lim, and Auerbach, 1978, Mutat. Res. 49: 219-24.
genetics: Male fertile.

translocation	cytology
T(1;2)DEB1	5B;25E-F
T(1;2)DEB2	5C;22E
T(1;2)DEB3	20F;36D
T(1;2)DEB4	20F;40-41
T(1;2)DEB5	1C;56C
T(1;2)DEB6	20F;25A
T(1;2)DEB7	20F;37D

***T(1;2)dor^{var7}: Translocation (1;2) deep orange-variegated**

cytology: T(1;2)2B8-9;40-41; + T(1;2)6F-7A;60C + In(2LR)35B;45A.

new order:

1 - 2B8|40F - 35B|45A - 41A|2B9 - 6F
|60C - 45A|35B - 21;
20F - 7A|60C - 60F.

synonym: In(1)dor^{var7} (Belyaeva et al., 1982).

references: Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, D15 58: 184-90.

Zhimulev, Belyaeva, Fomina, Protopopov, and Bolshakov, 1986, Chromosoma 94: 492-504.

Demakova and Belyaeva, 1988, DIS 67: 19-20.

Demakova, Belyaeva, and Zhimulev, 1988, DIS 67: 19-20.

genetics: l(1)2Ad, l(1)2Ae, l(1)2Af, BRC, dor, and hfw translocated to 40F show position-effect variegation whose spread and type is affected by the source (maternal or paternal) of the rearrangement and the temperature (Demakova and Belyaeva, 1988).

T(1;2)dpp¹⁵: Translocation (1;2) decapentaplegic

cytology: T(1;2)20;22F1-2.

origin: X ray induced.

discoverer: Spenser (Gelbart's lab).

genetics: d-III dpp mutant.

T(1;2)dpp⁴⁹

cytology: T(1;2)h33-34;22F. Cytology difficult; perhaps broken in XR.

origin: γ ray induced.

discoverer: Irish.

references: Gelbart.

genetics: d-V dpp mutant.

***T(1;2)ef: Translocation (1;2) elfin**

cytology: T(1;2)14C8-D1;2R.

origin: Induced by triethylenemelamine (CB. 1246).

discoverer: Fahmy, 1952.

references: 1959, DIS 33: 86.

genetics: Mutant for ef. Male sterile.

***T(1;2)f²⁵⁷⁻²²**

cytology: T(1;2)4D2-3;8F;15E4-F1;39E;41F-42A super-imposed on Dp(1;1)15F9-16A1;16A7-B1.

new order:

1 - 4D2|(8F - 15E4)|41F - 39E|(4D3 - 8F)|39E - 21;
20 - 16A1|16A7 - 16A1|16A7 - 15F1|42A - 60.

origin: X ray induced in y BⁱBⁱ.

discoverer: Demerec, 36c.

genetics: Mutant for f but B unaffected. Male lethal.

T(1;2)FN107: see Tp(1;2)sn⁺72d

T(1;2)GA

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal.

translocation	cytology	mutant for
T(1;2)GA1	2E-F;41	
T(1;2)GA26	2E3;23	kz

translocation	cytology	mutant for
T(1;2)GA113	2C;49A	

T(1;2)GE204

cytology: T(1;2)3D3-4;60.

origin: X ray induced.

discoverer: Lefevre.

genetics: Hemizygous lethal.

T(1;2)GE214

cytology: T(1;2)20A-B;27-28.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal. Allele of l(1)20Ac.

T(1;2)GEM224

cytology: T(1;2)11A4;11C3;40-41.

new order:

1 - 11A4|11C3 - 11A4|41 - 60;
21 - 40|11C3 - 20.

discoverer: Lefevre.

genetics: Mutant for fw (Kulkani and Hall, 1987, Genetics 115: 461-75).

T(1;2)GF326

cytology: T(1;2)1D4;36C.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal. Allele of l(1)1Dc.

T(1;2)gl⁺: Translocation (1;2) giant larvae-wild type

cytology: 21A1-C1 [location of l(2)gl] translocated to X tip.

references: Cline, 1976, Genetics 83: s16.

T(1;2)HA37

cytology: T(1;2)3A4;32A1-2.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal. Allele of l(1)3Ac.

***T(1;2)HC221**

cytology: T(1;2)2B11;22B.

origin: X ray induced.

discoverer: Lefevre.

genetics: Hemizygous lethal. Wild type over dor mutant; lethal over dor²⁵ (Lefevre).

T(1;2)HC282

cytology: T(1;2)2C1-2;32B.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal. Not covered by Dp(1;3)w^{vco} or y²Y67g (Lefevre).

T(1;2)HF326

cytology: T(1;2)3C7;27E.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal. N allele.

T(1;2)HG370

cytology: T(1;2)3E3-4;A (complex).

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal. Allele of *slc*.

***T(1;2)Hw^{bap}*: Translocation (1;2)
Hairy-wing-bristly
abdominal pleura**

cytology: *T(1;2)1B;21B* (Cline, 1984).

discoverer: Cline.

references: Roseland and Schneiderman, 1979, Wilhelm Roux's Arch. Dev. Biol. 186: 235-65.

Cline, 1984, Genetics 107: 231-77.

1988, Genetics 119: 829-62.

genetics: Mutant for *Hw^{bap}*. Deficient for *al* but not *net* (Cline).

T(1;2)K1: see *Dp(1;2)K1*

T(1;2)KC16

cytology: *T(1;2)2D5;52F*.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal. Mutant for *csw*.

T(1;2)KW

cytology: *T(1;2)20;38C1* (Beermann). Heterochromatin translocated to *Ddc* region.

origin: γ ray induced.

references: Bishop and Wright, 1987, Genetics 115: 477-91.

***T(1;2)l-v*: Translocation (1;2) lethal-variegated**

origin: X ray induced.

discoverer: Lindsley, Edington, and Von Halle.

references: 1960, Genetics 45: 1649-70.

genetics: Selected as X chromosomes lethal or semilethal in XO males, but viable in XY males. Male sterile except for *T(1;2)l-v135* which was not tested.

translocation	cytology
<i>T(1;2)l-v25</i> ^{α}	19-20;40-41
<i>T(1;2)l-v75</i>	19-20;41
<i>T(1;2)l-v129</i>	18B;41
<i>T(1;2)l-v135</i> ^{β}	18-19;41
<i>T(1;2)l-v150</i>	16-17;40
<i>T(1;2)l-v219</i> ^{γ}	10A1-2;40

^{α} Position of breakpoint in chromosome 2 with respect to centromere not determined.

^{β} Induced simultaneously with **T(2;3)l35 = *T(2;3)37;85A*.

^{γ} Other references: Zhimulev, Belyaeva, Pokholkova, Kochneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baritcheva, Aizenzon, Kramers, and Eeken, 1981, DIS 56: 192-96; Zhimulev, Pokholkova, Bgatov, Semeshin, and Belyaeva, 1981, Chromosoma 82: 25-40.

T(1;2)L124

cytology: *T(1;2)21C;XR*.

origin: γ ray-induced detachment of *C(1)RM*, $y^2 su(w^a)$ $w^a bb$ by the *Y^{P2D}* element of *T(Y;2)L124*.

discoverer: Kennison.

genetics: 21A-C appended to right arm of X chromosome.

***T(1;2)lt*: Translocation (1;2) light**

cytology: *T(1;2)20C-D;40F*.

origin: X ray induced in chromosome carrying *eq*.

discoverer: Schultz.

genetics: Variegated for *lt*.

****T(1;2)lz*: Translocation (1;2) lozenge**

cytology: *T(1;2)8D12-E1;33A-B* (Hannah).

origin: X ray induced.

discoverer: Green and Green.

references: 1956, Z. Indukt. Abstamm. Vererbungslehre 7: 708-21.

genetics: Mutant for *lz*.

***T(1;2)M*: Translocation (1;2) Mglinetz**

references: Mglinetz, 1968, Genetika (Moscow) 4(8): 81-86.

translocation	cytology	origin
<i>T(1;2)M151</i>	17F;44D	γ rays
<i>T(1;2)M154</i>	11E;51B	γ rays
<i>T(1;2)M156</i>	9A;47E	γ rays
<i>T(1;2)M158</i>	10F;23C	³² P feeding

***T(1;2)N²⁶⁴*: Translocation (1;2) Notch**

origin: X ray induced.

discoverer: Demerec.

translocation	cytology	ref ^{α}	variegates for
<i>*T(1;2)N²⁶⁴⁻⁹</i>	{3C;41}	1	<i>rst N dm w</i> ^{β} <i>a(2) M(2)A1a</i>
<i>T(1;2)N²⁶⁴⁻¹⁰</i> ^{γ}		1	<i>rst N dm</i>
<i>*T(1;2)N²⁶⁴⁻²³</i>	3C8-9;41A (Demerec and Hoover)	2	<i>rst fa</i>
<i>*T(1;2)N²⁶⁴⁻²⁴</i>	3C8-9;40F (Demerec)	2	<i>w rst fa</i>
<i>*T(1;2)N²⁶⁴⁻⁵³</i> ^{δ}	3C6-7;34C7-D1	2	
<i>*T(1;2)N²⁶⁴⁻⁵⁹</i>	3C8-9;40F (Hoover)	2	<i>w rst spl</i>
<i>*T(1;2)N²⁶⁴⁻⁶²</i>	3C7-8;41A-B (Sutton)	2	<i>w rst fa</i>
<i>*T(1;2)N²⁶⁴⁻⁶⁶</i>	3C6-7;41 + 7C9-D1;53F (Hoover)	1	<i>w rst fa dm ec</i>
<i>*T(1;2)N²⁶⁴⁻⁶⁹</i> ^{e}	3C7-8;44C4-5 (Demerec)	2	
<i>*T(1;2)N²⁶⁴⁻⁸²</i>	3C3-4;41A + 20A;57 (Demerec)	1	<i>w rst fa dm</i>

^{α} 1 = CP627; 2 = Demerec, 1941, Proc. Int. Congr. Genet. 7th, pp. 99-103.

^{β} w variegation occurs at low temperatures.

^{γ} XY male lethal; XYY male survives.

^{δ} Carries mutant alleles of *rst*, *fa*, *dm*.

^{e} Carries mutant allele of *N*.

T(1;2)NSt: see *Tp(1;2)w-ec*

****T(1;2)N4***

cytology: *T(1;2)3A5;55A*.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal. Allele of *l(1)3Ac*.

***T(1;2)OR*: Translocation (1;2) from Oak Ridge**

origin: X ray induced; recovered by virtue of pseudolinkage in daughters of irradiated wild-type males.

discoverer: Warters, 1959, 1961.

translocation	cytology	genetics
<i>T(1;2)OR6</i> ^{α}	2A;60D	male viable, fertile; homozygous female viable
<i>T(1;2)OR7</i>	3A;41E	XY male lethal; with <i>B^Sw⁺Y</i> , male viable but sterile
<i>T(1;2)OR8</i> ^{β}	20;40-41	male viable, fertile; homozygous female viable
<i>T(1;2)OR11</i>	14F;41	variegated for a lethal; XY male viable but sterile
<i>T(1;2)OR14</i>	18D;46B	male quite inviable; rare survivor has unexpanded wings and crossed scutellars.
<i>T(1;2)OR15</i>	11B;60E	male viable but sterile
<i>T(1;2)OR17</i>	3C;37C	male viable but sterile with <i>Y</i> or <i>B^Sw⁺Y</i>
<i>T(1;2)OR18</i> ^{γ}	20B;30E	male viable but sterile

translocation	cytology	genetics
T(1;2)OR20	16C;43B	male lethal (lethal appeared later)
T(1;2)OR21	19E;32D	male lethal
T(1;2)OR22	8D;22B	male lethal
T(1;2)OR23	6B;40	male viable but sterile
T(1;2)OR25	1B;38E	male viable but sterile
T(1;2)OR26	15A;41	male viable but sterile; XO male has melanotic, distended wings
T(1;2)OR27	16D;34B	male viable but sterile
T(1;2)OR28	3B;39E	male viable but sterile with Y or B ^S _w ⁺ Y
T(1;2)OR29	8D;40	male viable but sterile
T(1;2)OR30 ^β	20;40-41	male viable but sterile
T(1;2)OR32 ^δ	20;54A	male viable but sterile
T(1;2)OR33	17;47A	male viable but sterile
T(1;2)OR36 ^β	20;40-41	male viable but sterile
T(1;2)OR37	10A;50C	male viable but sterile
T(1;2)OR38	20B;50A	male lethal
T(1;2)OR39	1D;46E	male viable but sterile
T(1;2)OR41	12D;25E	male lethal
T(1;2)OR42	12F;58F	male lethal
T(1;2)OR43	15E;40D	male viable but sterile, with small rough eyes (<i>un</i> ?)
T(1;2)OR44	16F;28F	male viable but sterile
T(1;2)OR45 ^β	7D;40-41	male viable but sterile
T(1;2)OR47	19E;53B	male viable but sterile
T(1;2)OR48	17A;31F	male viable but sterile
T(1;2)OR49 ^β	20;40-41	male viable but sterile
T(1;2)OR50	19C;33F	male viable, fertile; homozygous female weakly viable
T(1;2)OR51	8D;41F	male lethal (lethal originated after translocation)
T(1;2)OR52	4F;41A	male viable but sterile
T(1;2)OR54	19E;32E	male viable but sterile with slightly curled wings
T(1;2)OR55	20A;44D	male viable but sterile
T(1;2)OR56	18F;47D	male lethal
T(1;2)OR58 ^{βγε}	11B;40-41 + 19C;30B	male lethal
T(1;2)OR59 ^δ	19E;57B	male lethal
T(1;2)OR60	10A;52D	male viable but sterile
T(1;2)OR61	18D;31F	male viable but sterile
T(1;2)OR62 ^{βε}	8F;17F;40-41	male lethal
T(1;2)OR64	11A;53F	male lethal
T(1;2)OR65	7A;40	male lethal
T(1;2)OR66	8B;23C	male viable, fertile
T(1;2)OR67 ^ε	12B;41 + 20A;45B	male viable, fertile
T(1;2)OR68 ^ε	16E;41 + Tp(2;2)25E;33A;40	male viable but sterile
T(1;2)OR69	3C;41C	variegated for lethal; male sterile with Y or B ^S _w ⁺ Y
T(1;2)OR72 ^{εζ}	19E;29F + In(2LR)24F;54B	male viable but sterile
T(1;2)OR73 ^δ	19E;57E	male viable but sterile
T(1;2)OR74 ^δ	19E;56C	male viable but sterile
T(1;2)OR75	12E;32B	male lethal
T(1;2)OR78 ^γ	19E;30B	male viable but sterile
T(1;2)OR82	13D;30B	male viable but sterile
T(1;2)OR83	12A;22B	male lethal
T(1;2)OR84	3C;38E	male viable but sterile with Y or B ^S _w ⁺ Y
T(1;2)OR85	10;38	male viable but sterile
T(1;2)OR86	11A;32B	male viable but sterile

α 2^PX^D element can replace one chromosome 2, producing a deficiency for tip of 2R; resulting progeny Minute since M(2)60E in the deficiency.

β Positions of second-chromosome breakpoint(s) with respect to centromere not determined.

γ Male hyperploid for X^P2L^D element survives, has outstretched wings.

δ Male hyperploid for X^P2R^D element survives.

ε T(1;2)OR58 = 1 - 11B|40 - 30B|19C - 11B|41 - 60; 20 - 19C|30B - 21.

T(1;2)OR62 = 1 - 8F|40 - 60; 20 - 17F|8F - 17F|40 - 21 (tentative).

T(1;2)OR67 = 1 - 12B|41 - 45B|20A; 21 - 41|12B - 20A|45B - 60.

T(1;2)OR68 = 1 - 16E|41 - 40|(25E - 33A)|40 - 33A|25E - 21; 20 - 16E|41 - 60 (tentative).

T(1;2)OR72 = 1 - 19E|29F - 54B|24F - 21; 20 - 19E|29F - 24F|54B - 60.

ζ Male hyperploid for X^P2L^M2R^D element survives.

T(1;2)para^{lk1}: Translocation (1;2) paralytic

cytology: T(1;2)14C7-D1;41A.

references: J.C. Hall.

genetics: Mutant for *para*.

T(1;2)pn20: Translocation (1;2) prune

cytology: T(1;2)2E1-2;40C1-2.

references: Slobodyanyuk and Serov, 1983, Mol. Gen. Genet. 191: 372-77.

genetics: Mutant for *pn*.

T(1;2)pn40

cytology: T(1;2)2E1-2;41A.

origin: X ray induced.

references: Ilyina, Sorokin, Belyaeva, and Zhimulev, 1980, DIS 55: 205.

Slobodyanyuk and Serov, 1983, Mol. Gen. Genet. 191: 372-77.

genetics: Mutant for *pn*. Homozygous lethal.

T(1;2)RC45

cytology: T(1;2)3A4;3C1-2;59D.

new order:

1 - 3A4|3C1 - 3A4|59D - 21;

20 - 3C2|59D - 60.

origin: X ray induced.

discoverer: Lefevre.

references: Young and Judd, 1978, Genetics 88: 723-42.

genetics: 3C break between *w* and *rst*; not lethal. Allele of *l(1)3Ac*.

T(1;2)RC66

cytology: T(1;2)1E4;58E.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal.

***T(1;2)ret: Translocation (1;2) reticulated**

cytology: T(1;2)20A5-B2;2R.

origin: Induced by L-p-N,N-di-(2-chloroethyl)-aminophenylalanine (CB. 3025).

discoverer: Fahmy, 1953.

references: 1958, DIS 32: 73.

genetics: Associated with *ret*; male sterile.

T(1;2)RF19

cytology: T(1;2)20;2L + Df(1)7A4;7B2-3 + In(1)6A1;19E8.

origin: X ray induced.

discoverer: Lefevre.

references: Johnson and Judd, 1979, Genetics 92: 485-502.

genetics: Associated with Df(1)RF19 and In(1)RF19.

T(1;2)RF45

cytology: T(1;2)19E;20E-F;52A.

new order:

1 - 19E|52A - 21;

20F|19E - 20E|52A - 60.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal. Allele of *run* (Lefevre).

T(1;2)S25: Translocation (1;2) of Segal

cytology: *T(1;2)20?;50F*.
origin: γ ray induced with *T(2;3)shv*^{S25}.
references: Segal and Gelbart, 1985, Genetics 109: 119-43.

T(1;2)S76

cytology: *T(1;2)3A4;42A*.
origin: X ray induced.
discoverer: Lefevre.
genetics: Male lethal. Allele of *l(1)3Ac*.

****T(1;2)sc*¹¹⁵: Translocation (1;2) scute**

cytology: *T(1;2)1A6-B1;25F*; inferred from fig. 3 of Goldat.
origin: X ray induced derivative of *sc*⁶.
discoverer: Goldat.
references: 1936, Biol. Zh. (Moscow) 5: 803-12.
genetics: Mutant for *sc*.

****T(1;2)sc*²⁶⁰⁻¹⁷**

cytology: *T(1;2)1B2-3;31C*.
origin: X ray induced.
discoverer: Sutton, 39d.
references: 1943, Genetics 28: 210-17.
genetics: Mutant for *sc* but not *y*, *ac*, or *svr*.

***T(1;2)sc*^{S2}: Translocation (1;2) scute of Sinitskaya**

cytology: *T(1;2)1B4-7;60C-E*.
discoverer: Sinitskaya, 1934.
references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.
genetics: Mutant for *sc*. *X* chromosome broken to the right of *l(1)sc* in same place as right breakpoint of *Tp(1;2)sc*¹⁹ and *2R* broken between *sp* and *M(2)60E* (Muller). Aneuploid segregants *2^PX^D* and *X^P2R^D* should survive.
molecular biology: *X* breakpoint between left breakpoints of *In(1)sc*⁹ and *In(1)sc*⁷ (Campuzano, Carramolino, Cabrera, Ruiz-Gómez, Villares, Bononat and Modell, 1985, Cell 49: 327-38).

***T(1;2)shv*^{S26}: Translocation (1;2) short vein**

cytology: *T(1;2)1D;22F1-2*.
origin: γ ray induced.
references: Segal and Gelbart, 1985, Genetics 109: 119-43.
genetics: Associated with *shv*. Lethal with *shv* and *l(2)DPP* alleles.

T(1;2)SP1: Translocation (1;2) from São Paulo

origin: γ ray induced. Selected as X-linked male-sterile mutations in a *y w X* chromosome.
discoverer: Lindsley and Musatti, 1961.

translocation	cytology	genetics
<i>T(1;2)SP1</i>	<i>8B;41</i>	variegated for a lethal
<i>T(1;2)SP4</i>	<i>20;40-41</i>	
<i>T(1;2)SP10</i> ^{α}	<i>10D4-6;50D5-7</i>	
<i>T(1;2)SP16</i>	<i>20;40-41</i>	
<i>T(1;2)SP18</i>	<i>1A;56A</i>	male fertile
<i>T(1;2)SP19</i>	<i>20;40-41</i>	homozygous female fertile
<i>T(1;2)SP20</i>	<i>20;40-41</i>	
<i>T(1;2)SP31</i> ^{β}	<i>20;56B</i>	
* <i>T(1;2)SP33</i>	<i>14;41</i>	
<i>T(1;2)SP36</i>	<i>20;40-41</i>	
<i>T(1;2)SP42</i>	<i>20;40-41</i>	
<i>T(1;2)SP43</i>	<i>16A;60C</i>	
<i>T(1;2)SP48</i>	<i>15F;35A</i>	

translocation	cytology	genetics
<i>T(1;2)SP49</i>	<i>12;40-41</i>	
<i>T(1;2)SP50</i> ^{γ}	<i>20;29-30</i>	
<i>T(1;2)SP51</i>	<i>20;40-41</i>	
<i>T(1;2)SP52</i>	<i>12E;57F</i>	
<i>T(1;2)SP55</i> ^{δ}	<i>1A;41 + 4B;30B + In(1)12D;14B</i>	male lethal
<i>T(1;2)SP58</i>	<i>10A;34A</i>	
<i>T(1;2)SP60</i>	<i>17E;35A</i>	
<i>T(1;2)SP61</i>	<i>18F;47D</i>	
<i>T(1;2)SP64</i>	<i>3C;28C</i>	male sterile with <i>Y</i> or <i>B^S_w + Y</i>
<i>T(1;2)SP67</i>	<i>20;40-41</i>	
<i>T(1;2)SP69</i>	<i>7C;41</i>	
<i>T(1;2)SP71</i>	<i>20;40-41</i>	
* <i>T(1;2)SP75</i>	<i>8C;35D</i>	
<i>T(1;2)SP77</i>	<i>9A;41</i>	
<i>T(1;2)SP81</i> ^{γ}	<i>20;24F-25A</i>	
<i>T(1;2)SP84</i>	<i>4C;42C</i>	
<i>T(1;2)SP87</i>	<i>9A4-B1;58A3-4</i>	
<i>T(1;2)SP88</i> ^{γ}	<i>20;32F-33A</i>	
* <i>T(1;2)SP89</i>	<i>4E;35A</i>	mutant for <i>rg?</i>
<i>T(1;2)SP93</i> ^{γ}	<i>18C-D;22A-B</i>	
<i>T(1;2)SP94</i>	<i>14B-C;23F</i>	
<i>T(1;2)SP96</i>	<i>20;40-41</i>	
* <i>T(1;2)SP97</i>	<i>9E-F;35A-B</i>	
<i>T(1;2)SP102</i>	<i>16A;41</i>	
* <i>T(1;2)SP106</i>	<i>6B;40</i>	
<i>T(1;2)SP110</i>	<i>13A;57E</i>	
<i>T(1;2)SP111</i>	<i>20;41-41</i>	

α See Lefevre, 1970, DIS 45: 39.
 β Male hyperploid for *X^P2R^D* element survives.
 γ Male hyperploid for *X^P2L^D* element survives.
 δ New order: *1A|41 - 30B|4B - 1A|41 - 60; 20 - 14B|12D - 14B|12D - 4B|30B - 21*.

****T(1;2)Sy: Translocation (1;2) Stubby***

origin: Spontaneous.
discoverer: Ives, 34j31.
genetics: Associated with *Sy*. Male sterile. Probably reciprocal translocation with breaks near the base of *X* and *2L*.

T(1;2)TE35A-217: Translocation (1;2) Transposing Element

cytology: *T(1;2)20;35A-B*.
origin: γ ray induced in *TE35A*.
discoverer: Samkange (Ashburner's lab).
synonym: *T(1;2)TE146-217*.

****T(1;2)v*²⁶⁷⁻⁴**

cytology: *T(1;2)11A7-8;36* (Sutton).
origin: X ray induced.
discoverer: Hoover, 35i.
genetics: Mutant for *v* (breakpoint not at *v* locus). Semi-lethal. *ras*, *dwx*, *sbr*, *m*, *dy*, and *fw* not affected.

T(1;2)V: Translocation (1;2) Valencia

origin: X ray induced.
references: Valencia, 1970, DIS 45: 37.

translocation	cytology	genetics
<i>T(1;2)V9-2</i>	<i>6A2-3;38F</i>	male sterile
<i>T(1;2)V11-2</i>	<i>20;40-41</i>	male fertile, homozygous viable
<i>T(1;2)V12-1</i>	<i>11E-F;25C-D</i>	male sterile
<i>T(1;2)V12-2</i>	<i>12C9;40-41A</i>	male sterile
<i>T(1;2)V101</i>	<i>20A(C-D?);60F5</i>	male sterile
<i>T(1;2)V153</i>	<i>20A3-4;56F5</i>	male sterile
<i>T(1;2)V154</i>	<i>12A3;40-41</i>	lethal
<i>T(1;2)V161</i>	<i>8B4;40</i>	lethal

T(1;2)VE614

cytology: *T(1;2)7B1-2;2L*.
origin: Induced by ethyl methanesulfonate.
references: Lefevre and Leeds, 1983, *Genetics* 104: s45-46.
genetics: Shows strong *kf*² phenotype but has no *ct* effect.

T(1;2)VE715

cytology: *T(1;2)19E;27A*.
origin: X ray induced.
discoverer: Lefevre.
genetics: Male lethal. *unc* allele.

T(1;2)VE901

cytology: *T(1;2)2B;25E-F*.
origin: X ray induced.
discoverer: Lefevre.
genetics: Male lethal. Mutant for *br*.

***T(1;2)w^{13G2}: Translocation (1;2) white**

cytology: *T(1;2)3C3-5;41;56F*; also inversion in 2R.
new order:
 1 – 3C3|41 – 56F|41 – 21;
 20 – 3C5|56F – 60.
 Probable.
origin: X ray induced.
discoverer: Gans.
genetics: Variegated for *w*.

T(1;2)w^{64d}: see *Tp(1;2)w-ec*

T(1;2)w^{70k18.1}

cytology: *T(1;2)3C1;12B2-9;39E2*.
new order:
 1 – 3C1|39E3 – 60;
 21 – 39E2|12B2 – 3C2|12B9 – 20.
origin: X ray induced in *In(1)z^{64b9}*.
references: Sorsa, Green, and Beermann, 1973, *Nature* (London) *New Biology* 245: 34-37.
genetics: Male viable with white eyes.

T(1;2)w^{+51b7}: see *T(1;2)51b*

***T(1;2)w^{m53e}: Translocation (1;2) white-mottled**

cytology: *T(1;2)3C3-4;20A2-3;58F8-59A1*.
new order:
 1 – 3C3|58F8 – 21;
 20F – 20A3|3C4 – 20A2|59A1 – 60.
origin: Neutron induced.
discoverer: Mickey, 53e11.
synonym: *T(X-2)In^{X and 3}*.
references: 1963, *DIS* 38: 29.
genetics: Variegated for *w*.

***T(1;2)w^{m258-34}**

cytology: *T(1;2)3C3-5;41A* (Demerec and Hoover).
origin: X ray induced.
discoverer: Demerec, 38b.
genetics: Variegated for *w* but not *rst*, *fa*, or *dm*. Male viable.

***T(1;2)w^{m258-36}**

cytology: *T(1;2)3C6-7;41A-B + T(1;2)4C2-3;41F5-6*.
new order:
 1 – 3C6|(41B – 41F5)|4C3 – 20;
 21 – 41A|(3C7 – 4C2)|41F6 – 60.

Insertions said to be in inverted order but not specified with respect to centromere or numerical order.

origin: X ray induced.

discoverer: Demerec, 38b.

references: Sutton, 1940, *Genetics* 25: 534-40 (fig.).

genetics: Variegated for *w* and *rst* but not *pn*, *fa*, or *dm*. Male viable. Cytology predicts that each element of the translocation should survive as aneuploid but not so recorded.

***T(1;2)w^{m258-37}**

cytology: *T(1;2)3C3-4;40-41A* (Sutton).

origin: X ray induced.

discoverer: Demerec, 33j.

genetics: Variegated for *w* but not *kz*, *rst*, *fa*, or *dm*.

***T(1;2)w^{m258-39}**

cytology: *T(1;2)3C3-5;40E-F* (Demerec and Hoover).

origin: X ray induced.

discoverer: Demerec, 38e.

genetics: Variegated for *w* but not *pn*, *rst*, *fa*, or *dm*. Male viable.

***T(1;2)w^{m258-40}**

cytology: *T(1;2)3C3-5;41* (Demerec and Hoover).

origin: X ray induced.

discoverer: Demerec, 38e.

genetics: Variegated for *w* and *rst* but not *pn*, *kz*, *fa*, or *dm*.

***T(1;2)w^{MD1}: Translocation (1;2)**

white-mottled of Dubinin

cytology: *T(1;2)3B;19-20;21F*.

new order:

1 – 3B|21F – 60;
 20|3B – 19|21F – 21A.

origin: X ray induced.

discoverer: Dubinin.

references: Sacharov, 1936, *Biol. Zh. (Moscow)* 5: 293-302.

***T(1;2)w^{VD4}: Translocation (1;2)**

white-variegated of Demerec

cytology: *T(1;2)3D6-E1;40F* (Schultz).

origin: X ray induced.

discoverer: Demerec, 33k2.

genetics: Variegated for *N*, *rst*, *w*, and *dm*. *X/Y* male survives only rarely as *rst* with mottled eye color; *X/Y/Y* male more viable, slightly *rst*, and sterile. Variegation for *lt* in *X/X/Y* female.

T(1;2)(w-ec)^{64d}: see *Tp(1;2)w-ec*

T(1;2)w-ec: see *Tp(1;2)w-ec*

T(1;2)X9^{ts}

cytology: *T(1;2)2D1-2;56A1-2*.

origin: γ ray induced.

references: Kaufman and Suzuki, 1974, *Can. J. Genet. Cytol.* 16: 579-92.

genetics: Male fertile. Associated with temperature-sensitive lethal in males. *X/X* females and *X/w⁺Y* males are viable and normal at 22° and 29°; *X/Y* males at 29° are lethal or show escaper phenotype of net-like wing veins, etched abdominal tergites, and sexcombs on meso- and metathoracic legs.

***T(1;2)y²⁶⁰⁻¹³: Translocation (1;2) yellow**

cytology: *T(1;2)1A4-5;36D*.
origin: X ray induced.
discoverer: Sutton, 1939.
references: 1943, *Genetics* 28: 210-17.
genetics: Mutant for *y*.

T(1;2)y^{A76b37}

cytology: *T(1;2)1B1-2;60F*.
origin: γ ray induced.
references: Alexandrov, Ankina, and Alexandrova, 1985, *DIS* 61: 212-13.
genetics: Mutant for a *y*^{ca}-like allele.

T(1;2)y^{A79d}

cytology: *T(1;2)1A6-B2;23E5*.
origin: Neutron induced.
references: Alexandrov, Ankina, and Alexandrova, 1985, *DIS* 61: 212-13.
genetics: Mutant for *y*.

T(1;2)y^{R7}

cytology: *T(1;2)1A6-B1;49F*.
origin: X ray induced.
references: Roberts, 1974, *Mutat. Res.* 22: 139-44.
genetics: Body color yellow. Male viable but sterile.

***T(1;2)y^{v1}: Translocation (1;2) yellow-variegated**

cytology: *T(1;2)1A;39*.
origin: X ray induced.
discoverer: Schultz, 33a11.
genetics: Variegated for *y*.

T(1;2)z⁺: Translocation (1;2) zeste-wild type

origin: X-ray-induced rearrangements in *z*-bearing X chromosomes that interfere with the *z-w* interaction.
genetics: *z*⁺ phenotype.

translocation	cytology	ref ^α
T(1;2)z⁺3a	3C2-3;chromosome 2	2
T(1;2)z⁺4E6	3C3-5;40A	1
T(1;2)z⁺6E8	3E2-6;25E + X distal;80-81	
T(1;2)z⁺14E9	3B2-C1;19B2-C1;24D4-E1	1
T(1;2)z⁺20	3C1-2;43E	2
T(1;2)z⁺23	3C1;3D;40	2
T(1;2)z⁺29	3C1;37C + <i>Dp(1;2)2E1;3B4-C1;22D2-3</i>	
T(1;2)z⁺35	3C6-7;23A4-B1	2
T(1;2)z⁺36	3C3;54F-55A	2
T(1;2)z⁺43	3C1-2;37F	2
T(1;2)z⁺49	3C;55D-E	2
T(1;2)z⁺53	3B3-4;45A-D	2
T(1;2)z⁺57^β	3C1-3;41A-B;54B-C	

^α 1 = Gans, 1953, *Bull. Biol. Fr. Belg. Supp.* 38: 1-90; 2 = Gelbart, 1971, Ph.D. Thesis, Univ. of Wisconsin.
^β New order: 1 - 3C1|41A - 21;
 20 - 3C3|54B - 41B|54C - 60.

T(1;2)ZWD1

cytology: *T(1;2)3C2-5;45C*.
origin: γ -ray-induced.
references: Smolik-Utlaut and Gelbart, 1987, *Genetics* 116: 285-98.
genetics: Moles *w*⁺ and sterile.

T(1;2)ZWD12

cytology: *T(1;2)2A;3C1-2;221A;22A;43F*.
origin: γ -ray-induced.
references: Smolik-Utlaut and Gelbart, 1987, *Genetics* 116: 285-98.
genetics: Moles *w*⁺ and sterile.

***T(1;2;3)58i**

origin: X ray induced.
discoverer: Imazumi.
references: 1961, *DIS* 35: 87-88.
 1962, *DIS* 36: 80.
 1962, *Cytologia* 27: 212-28 (fig.).
genetics: Distal one-third of 2L appended to X chromosome as short arm. Also *T(2;3)* with 2R broken between *cn* and *vg* and 3L broken between *se* and *st*. Male lethal in embryo.

***T(1;2;3)100r20**

cytology: *T(2;3)35B2-3;40;80* superimposed on *Dp(1;3)3B4-C1;4B4-5;80*.
new order:
 21 - 35B2|4B4 - 3C1|80 - 100;
 60 - 40|35B3 - 40|80 - 61.
origin: X ray induced in *Dp(1;3)N²⁶⁴⁻¹⁰⁰*.
discoverer: Gersh, 1959.
synonym: **T(2;3)100r20*.
references: 1959, *Genetics* 44: 163-72.
genetics: Selected as a partial reversion from white-mottled.

T(1;2;3)220

cytology: *T(1;2;3)14A;50A;75C*.
new order:
 1 - 14A|50A - 21;
 20 - 14A|75C - 61;
 60 - 50A|75C - 100.
origin: X ray induced.
references: Lindsley, Edington, and Von Halle, 1960, *Genetics* 45: 1649-70.
genetics: Male viable and sterile.

***T(1;2;3)A149**

cytology: *T(1;2;3)19B1-2;20;48F;81*. Complex.
origin: X ray induced.
discoverer: Lefevre.
genetics: Male lethal.

***T(1;2;3)b^{75.1}: Translocation (1;2;3) black**

cytology: *T(1;2;3)1B7-8;42E;62B9*.
new order:
 61 - 62B9|1B7-8 - 20;
 21 - 42E|1B7-8 - 1A;
 60 - 42E|62B9 - 100.
origin: X ray induced.
references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, *DIS* 55: 193-95.
genetics: Mutant for *b* (induced by independent event).

T(1;2;3)by³⁵: Translocation (1;2;3) blistery

cytology: Break in 3 at 85D7-12 (used to localize *by*); other breaks not given.
origin: X ray induced.
references: Kempthues, Raff, Raff, and Kaufman, 1980, *Cell* 21: 445-51.

genetics: Mutant for *by*.

**T(1;2;3)C232: Translocation (1;2;3)
Crossover suppressor**

cytology: T(2;3)35D;71E; additional presence of T(1;2)20;40-41 or T(1;3)20;80-81 inferred from genetic data.

origin: X ray induced.

discoverer: Roberts, 1965.

references: 1970, Genetics 65: 429-48.

genetics: Male viable and fertile; homozygous female lethal. Recombination reduced in 2L.

T(1;2;3)C312

cytology: T(2;3)32C;87E; additional presence of T(1;2)20;40-41 or T(1;3)20;80-81 inferred from genetic data.

origin: X ray induced.

discoverer: Roberts, 1965.

references: 1970, Genetics 65: 429-48.

genetics: Male sterile. Recombination reduced in 2L.

***T(1;2;3)ct²⁶⁸⁻⁴⁰: Translocation (1;2;3) cut**

cytology: T(1;2;3)7D2-3;10A5-6;21B-C;28-29;40-41;75B-C;87D;88C;92; new order not determined.

origin: X ray induced.

discoverer: Demerec, 1938.

references: Sutton, 1940, Genetics 25: 534-40 (fig.).

genetics: Mutant at *ct* but not *scp*, *cm*, *sn*, *v*, *sbr*, *dy*, *g*, *ty*, *na*, *pl*, *sd*, or *mc*. Male lethal.

**T(1;2;3)DEB1: Translocation (1;2;3)
diepoxybutane**

cytology: T(1;2;3)20F;24F;88F.

origin: Induced by diepoxybutane.

references: Denell, Lim, and Auerbach, 1978, Mutat. Res. 49: 219-24.

T(1;2;3)Din: Translocation (1;2;3) Dinty

cytology: T(1;3)3C;63A + T(2;3)39D;73A (Lindsley).

new order:

1A - 3C|63A - 73A|39D - 60;

20 - 3C|63A - 61;

21 - 39D|73A - 100.

origin: X ray induced.

discoverer: Braver, 55a.

references: 1955, DIS 29: 70.

Pollock, 1963, DIS 38: 50.

genetics: Associated with *Din*. Male viable and fertile.

The two translocations should be easily separable, and *Din* is, in all probability, associated with only one.

***T(1;2;3)l-v216: Translocation (1;2;3)
lethal-variegated**

origin: X ray induced.

references: Lindsley, Edington, and Von Halle, 1960, Genetics 45: 1649-70.

genetics: Variegated for a lethal; male sterile.

T(1;2;3)l-v459

cytology: T(1;2;3)3D-F;XR;50;80-81.

new order:

1A - 3D|50 - 21;

3F - 20F|;

XR-80 - 61;

60 - 50-81 - 100.

Tentative.

Postulated that centromere of chromosome 3 split or double with one half capped by 2R^D and the other by XR^D X^P in the form of a ring.

origin: X ray induced.

references: Lindsley, Edington, and Von Halle, 1960, Genetics 45: 1649-70.

genetics: Variegated for a lethal; male fertile.

T(1;2;3)ML140

cytology: T(1;2;3)20;40-41;80-81.

origin: X ray induced.

references: Mukhina and Zhimulev, 1980, DIS 55: 209.

genetics: Homozygous lethal.

***T(1;2;3)N²⁶⁴⁻⁷⁴: Translocation (1;2;3) Notch**

cytology: T(1;2;3)3C10-11;20D-E;40C-D;92E6-8; 20D-E break claimed to be to the left of the nucleolus organizer (Sutton).

new order:

1 - 3C10|40D - 60;

20F - 20E|40C - 21;

61 - 92E6|20D - 3C11|92E8 - 100.

origin: X ray induced.

discoverer: Demerec, 1938.

references: Sutton, 1940, Genetics 25: 534-40 (fig.).

genetics: Variegates for *w*, *rst*, and *N* but not *kz*, *pn*, or *dm*.

***T(1;2;3)N²⁶⁴⁻⁸⁷**

cytology: T(1;2;3)3C7-9;10A2-B1;45F-46A;59F-60A;97C-D;100E-F (Sutton).

new order:

1 - 3C7|97D - 100E|59F - 46A|10B1 - 20;

21 - 45F|3C9 - 10A2|60A - 60F;

61 - 97C|100F.

origin: X ray induced.

discoverer: Demerec, 1939j.

references: Sutton, 1940, Genetics 25: 534-40.

genetics: Carries a mutant allele of *N* and normal alleles of *w*, *rst*, and *dm*.

**T(1;2;3)OR9: Translocation (1;2;3)
from Oak Ridge**

origin: X ray induced. Selected on the basis of pseudolinkage in daughters of treated wild-type males.

discoverer: Warters, 1961.

translocation	cytology	new order ^α	genetics
T(1;2;3)OR9	19-20;49F;81F	1	male lethal
T(1;2;3)OR10	18A;41;73F	2	male viable but sterile
T(1;2;3)OR12	3A;41 + 7E;78F	3	male viable but sterile with Y or B ^S w ⁺ Y
T(1;2;3)OR14	5E;21D;62C	4	male viable, fertile; homozygous female viable
T(1;2;3)OR16	1A;57D + 29;72E	5	male viable but sterile
T(1;2;3)OR17 ^B	20;40-41;61F		male viable but sterile
T(1;2;3)OR23	14C;27D;87B	6	male lethal
T(1;2;3)OR24	14B;39D + 2C;80C + 29;87A	7	male lethal
T(1;2;3)OR25 ^{γδ}	19E;29B;80-81	8	male viable but sterile
T(1;2;3)OR26	2D;56F + 3F;96B	9	male viable, fertile
T(1;2;3)OR31 ^ε	20;92A + 38D;87E	10	male viable but sterile
T(1;2;3)OR34	18F;84B + 28B;75F + 44C;63A	11	male lethal

^α 1 = 1 - 19|81F - 61; 20|49F - 60; 21 - 49F|81F - 100.
2 = 1 - 18A|73F - 100; 20 - 18A|41 - 60;

- 21 - 41|73F - 61.
 3 = 1 - 3A|41 - 21;
 20 - 7E|78F - 61;
 60 - 41|3A - 7E;78F - 100.
 4 = 1 - 5E|21D - 60;
 20 - 5E|62C - 61;
 21A - 21D|62C - 100.
 5 = 1A|57D - 21;
 20|72E - 61;
 60 - 57D|1A - 20|72E - 100.
 6 = 1 - 14C|87B - 61;
 20 - 14C|27D - 21;
 60 - 27D|87B - 100.
 7 = 1 - 2C|80C - 87A|19 - 14B|39D - 21;
 20 - 19|87A - 100;
 60 - 39D|14B - 2C|80C - 61.
 8 = 1 - 19E|80 - 100;
 20 - 19E|29B - 21;
 60 - 29B|80 - 61 (tentative).
 9 = 1 - 2D|56F - 21;
 20 - 3F|96B - 100;
 60 - 56F|2D - 3F|96B - 61.
 10 = 1 - 20|92A - 87E|38D - 60;
 20|92A - 100;
 21 - 38D|87E - 61.
 11 = 1 - 18F|84B - 75F|28B - 21;
 20 - 18F|84B - 100;
 60 - 44C|63A - 75F|28B - 44C|63A - 61 (tentative).

- β Neither breakpoints in X and 2 with respect to centromere nor new order determined.
 γ Position of breakpoint in chromosome 3 with respect to centromere not determined; therefore new order ambiguous.
 δ Hyperploid male, presumably carrying $X^P 2L^D$, survives.
 ε Male hyperploid for $X^P 3R^D$ element survives.

T(1;2;3)S53

cytology: *T(1;2;3)2D5;57E;86B*.
origin: X ray induced.
discoverer: Lefevre.
genetics: Male lethal. *csw* allele.

T(1;2;3)sc²⁶⁰⁻¹⁸: Translocation (1;2;3) scute

cytology: *T(1;2)1A6-B1;41D-E + T(1;3)7A2-B1;80C*.
new order:
 1A1 - 1A6|41D - 21;
 20 - 7B1|80C - 61;
 60 - 41E|1B1 - 7A2|80C - 100.

origin: X ray induced.
discoverer: Sutton, 39d.
references: 1943, Genetics 28: 210-17.
genetics: Mutant for *sc* but not *y*, *ac*, or *svr*. Male sterile.

***T(1;2;3)sc²⁶⁰⁻²⁹**

cytology: *T(1;2;3)1A6-B1;22A-B;34A-B;75C-E*.
new order:
 1A1 - 1A6|34A - 22B|34B - 60;
 20 - 1B1|75C - 61;
 21 - 22A|75E - 100.

origin: X ray induced.
discoverer: Sutton, 40a.
references: 1943, Genetics 28: 210-17.
genetics: Mutant for *sc* but not *y*, *ac*, or *svr*.

***T(1;2;3)sc^{P1}: Translocation (1;2;3) scute of Panshin**

discoverer: Panshin, 1934.
genetics: Mutant for *sc*.

T(1;2;3)SP: Translocation (1;2;3) from São Paulo

origin: γ ray induced. Selected as male-sterile mutations in γw X chromosome.
discoverer: Lindsley and Musatti, 1961.

translocation	cytology	new order ^α	genetics
T(1;2;3)SP3^β	20;23A-B;96B	1	male lethal
T(1;2;3)SP5	6 break rearrangement: 2R (2 breaks), 3L (1 break), 3R (2 breaks)	2	
T(1;2;3)SP6^γ	20;40-41;80-81		
T(1;2;3)SP8	5;17F;44B;90A	3	
*T(1;2;3)SP25^δ	19;54;86	4	
T(1;2;3)SP29	10E-11A;40;60D;64D	5	
T(1;2;3)SP40	4-5;50A;80 + 40;86	6	variegated for a lethal
T(1;2;3)SP57^ε	20;40-41;75A	7	
T(1;2;3)SP65	18A;39E;76A	8	

- α 1 = 1 - 20|96B - 61;
 20|23A - 21;
 60 - 23B|96B - 100.
 2 = $X^D 2R^P - 2L$;
 $X^P X^M | 3R^M | 3L^D$;
 $2R^D | 3L^P - 3R^P | 3R^D$.
 3 = 1 - 5|17F - 20;
 21 - 44B|5 - 17F|90A - 100;
 61 - 90A|44B - 60.
 4 = 1 - 19|54 - 21;
 20 - 19|86 - 100;
 60 - 54|86 - 61.
 5 = 1 - 10E|40 - 60D|64D - 61;
 20 - 11A|60D - 60F;
 21 - 40|64D - 100.
 6 = 1 - 4|80 - 86|40 - 21;
 20 - 5|50A - 60;
 61 - 80|50A - 40|86 - 100 (tentative).
 7 = 1 - 20|75A - 100;
 20|40 - 21;
 60 - 40|75A - 61
 8 = 1 - 18A|76A - 100;
 20 - 18A|39E - 21;
 60 - 39E|76A - 61.
 β Male hyperploid for $X^P 2L^D$ element survives.
 γ Neither breakpoints with respect to centromere nor new order determined.
 δ Male hyperploid for $X^P 3R^D$ element apparently survives.
 ε Breakpoint in chromosome 2 inferred from genetic data.

T(1;2;3)TE35A-100

cytology: *T(1;2;3)?;25F;35B;40;?*.
new order:
 21 - 25F|het;
 het|35B - 40|het;
 het|25F - 35B|het;
origin: γ ray induced.
discoverer: Samkange.
synonym: *T(1;2;3)TE146-100*.

***T(1;2;3)v^{L8}**

cytology: *T(1;3)3E5;62D + T(1;2)9D3-4;10A1-2;25E-F*.
new order:
 1 - 3E5|62D - 100;
 20 - 10A2|25E - 21;
 60 - 25F|9D3 - 3E5|62D - 61. Deficient for 9D4-10A1.
origin: X ray induced.
references: Lefevre, 1969, Genetics 63: 589-600.
genetics: Deficient for *ras-v*.

***T(1;2;3)w^{m258-44}: Translocation (1;2;3) white-mottled**

cytology: *T(1;2;3)3C3-4;4D2-E1;56E1-F1;80D* (Sutton).

new order:

1 – 3C3|80D – 100;
20 – 4E1|80D – 61;
21 – 56E1|(3C4 – 4D2)|56F1 – 60.

origin: X ray induced.

discoverer: Demerec, 38k.

genetics: Variegated for *w* but not *pn*, *rst*, or *fa*. *T(1;2;3)w^{m258-44}* may be separated into *T(1;3)w^{m258-44}* = *T(1;3)3C3-4;4D2-E1;80D*, which is deficient for 3C4 through 4D2 (i.e., *Df(1)w^{m258-44}* = *Df(1)3C3-4;4D2-E1*), and *Dp(1;2)w^{m258-44}* = *Dp(1;2)3C3-4;4D2-E1;56E1-F1*, which is duplicated for the same region. The deficiency includes the loci of *fa*, *dm*, *M(1)3E*, *ec*, *M(1)4BC*, *bi*, *peb*, and *rb* but not *rst* or *rg*. 3C3-4 breakpoint inconsistent with genetic data on *rst*. *Dp(1;2)w^{m258-44}* should be viable.

***T(1;2;3)wy²⁷⁴⁻²: Translocation (1;2;3) wavy**

cytology: *T(1;2)8F-9A;20A-B;26B-D* + *T(1;3)11D-E;65C-D* (Sutton).

new order:

1 – 8F|26D – 60;
20F – 20B|9A – 11D|65C – 61;
21 – 26B|20A – 11E|65D – 100.

origin: X ray induced.

discoverer: Demerec, 34a.

genetics: Mutant for *wy* but not *fw*, *dy*, *g*, or *s*. Male lethal.

T(1;2;3)y^{R1}: Translocation (1;2;3) yellow of Roberts

cytology: *T(1;2;3)1B4-8;12F;21D2;41;97F*.

origin: X ray induced.

synonym: *T(1;2;3)y-1*.

references: Roberts, 1974, *Mutat. Res.* 22: 139-44.

genetics: Mutant for *y*. Male viable and fertile.

T(1;2;3)y^{R8}

cytology: *T(1;2;3)1A6-B1;47A;67D*.

origin: X ray induced.

synonym: *T(1;2;3)y-8*.

references: Roberts, 1974, *Mutat. Res.* 22: 139-44.

genetics: Mutant for *y* and a male lethal not covered by *y⁺Y* or *l(1)IAc⁺Y*.

T(1;2;3)z⁺12E7

cytology: *T(1;2)2B12;3C2-5;22A1;24D2* + *T(1;3)2B13;80-81*.

references: Gans, 1953, *Bull. Biol. Fr. Belg. Supp.* 38: 1-90.

genetics: Phenotype *z⁺*.

T(1;2;3)z⁺16G1

cytology: *T(1;2;3)3B2-C1;46F2-3;70A*.

references: Gans, 1953, *Bull. Biol. Fr. Belg. Supp.* 38: 1-90.

genetics: Phenotype *z⁺*.

T(1;2;3;4)l-v454: Translocation (1;2;3;4) lethal-variegated

cytology: *T(1;2;3)12B;22-23;81* + *T(2;4)44F;101F*.

new order:

1 – 12B|81 – 61;
20 – 12B|22 – 21;

60 – 44F|101F – 101A;

100 – 81|23 – 44F|101F – 102.

origin: X ray induced.

discoverer: Lindsley, Edington, and Von Halle.

references: 1960, *Genetics* 45: 1649-70.

genetics: Associated with *l(1)v454*. Male sterile.

T(1;2;4)429.28

cytology: *T(1;2;4)5B;56D-E;101B-C*.

new order:

1A – 5B|56D – 21A;
60F – 56E|101B – 101A;
102F – 101C|5B – 20F.

discoverer: Gelbart.

***T(1;2;4)A12: Translocation (1;2;4) from Austin**

cytology: *T(1;2;4)1B-C;7A;7B;13B1-5;101-102*; breakpoints in chromosomes 2 and 4 not determined (Mackensen, 1935, *Texas Univ. Publ.* 4032: frontispiece).

new order:

1A – 1B|13B5 – 20;
21 – ?|(7A – 7B)|? – 60;
101|((1C – 7A)|(7B – 13B1))|102.

origin: X ray induced.

discoverer: Patterson, Stone, Bedicheck, and Suche.

references: Stone, 1934, *Genetica* 16: 506-19.

Mackensen, 1935, *J. Heredity* 26: 163-74.

Patterson, Stone, and Bedicheck, 1935, *Genetics* 20: 259-79.

1937, *Genetics* 22: 407-26.

genetics: A section from between *sc* and *br* on the left to between *g* and *sd* on the right is inserted into chromosome 4. The *ct* locus, but not *cm*, *sn*, or *oc*, is deleted from the insertion [i.e., *Df(1)A12* = *Df(1)7A;7B*] and inserted into chromosome 2 [i.e., *Dp(1;2)A12* = *Dp(1;2)7A;7B*]. Female hyperploid for the *X^{PX^D}* element [i.e., *Dp(1;f)A12* = *Dp(1;f)1B-C;13B1-5*] survives and is claimed to be fertile. Female hyperploid for *X^M* [i.e., *Dp(1;4)A12* = *Dp(1;4)1B-C;7A;7B;13B1-5;101-102*] occasionally survives and is sterile.

***T(1;2;4)N²⁶⁴⁻⁸⁵: Translocation (1;2;4) Notch**

cytology: *T(1;2;4)3B4-C1;6A2-B1;60A4-5;101F-102A* [Sutton, 1940, *Genetics* 25: 534-40 (fig.)] (complex).

new order:

1 – 3B4|60A4 – 21;
20 – 6B1|60A5 – 60F;
101A – 101F|(3C1 – 6A2)|102A – 102F.

origin: X ray induced.

discoverer: Demerec, 39d.

genetics: Variegates for *w*, *rst*, *fa*, *dm*, *rg*, *cx*, *cv*, *rux*, and *vs* but not *pn*, *ec*, *bi*, *peb*, or *rb*. Carries normal allele of *ci* (Stern). *Dp(1;4)N²⁶⁴⁻⁸⁵* = *Dp(1;4)3B4-C1;6A2-B1;101F-102A* viable in both sexes and sterile in male. Complementary *Df(1)N²⁶⁴⁻⁸⁵* inviable.

T(1;2;4)OR24: Translocation (1;2;4) from Oak Ridge

cytology: *T(1;2)3C;38A* + *T(1;4)11A;102C*.

new order:

1 – 3C|38A – 60;
20 – 11A|102C – 102F;
21 – 38A|3C – 11A|102C – 101A.

origin: X ray induced.

discoverer: Warters, 1961.

genetics: Male viable and sterile. Produces a hyperploid

female that may carry the X^{P4D} element.

***T(1;2;4)w^{mbw}V: Translocation (1;2;4)
white-mottled
brown-Variigated**

cytology: T(1;2)12F3-4;59C4-5 + T(1;4)3C3-4;101E4-5.

new order:

1 - 3C3|101E4 - 101A;

20 - 12F4|59C5 - 60;

21 - 59C4|12F3 - 3C4|101E5 - 102F.

origin: Neutron induced.

discoverer: Mickey, 53f15.

references: 1963, DIS 38: 30.

genetics: Variegated for *w*. Also claimed to variegate for *bw*, which is unusual since the T(1;2) is completely euchromatic.

***T(1;2;4)w^{VD2}: Translocation (1;2;4)
white-variegated
of Demerec**

cytology: T(1;2;4)3C4-5;18F;38;101A-C (Schultz).

new order:

1 - 3C4|101C - 102F;101A;

20 - 18F|3C5 - 18F|38 - 21;

60 - 38|101A;101C-102F.

Tentative.

origin: X ray induced.

discoverer: Demerec, 33k27.

genetics: Variegated for *w* but not *rst* in male and for *w* and occasionally *rst* in female. Absence of effect on *ci* a criterion for postulating break in 4L. Fly hyperploid for the 4^{PX^D} element survives.

T(1;2;4)y^{R29}

cytology: T(1;2;4)1E4-F1;20;26A;35B-C;101.

origin: X ray induced.

references: Roberts, 1974, Mutat. Res. 22: 139-44.

genetics: Mutant for *y* although breakpoint given is proximal to *y*. Male viable but sterile.

T(1;2;4)z⁺4E7

cytology: T(1;2;4)3C7-9;56C4-D1;102A.

references: Gans, 1953, Bull. Biol. Fr. Belg. Supp. 38: 1-90.

genetics: Phenotype *z*⁺.

T(1;3)2B7

cytology: T(1;3)2B7;84A4-6.

references: Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelova, and Zhimulev, 1980, Chromosoma 81: 281-306.

***T(1;3)3**

origin: X ray induced.

discoverer: Bonner, 1931.

references: Dobzhansky, 1935, Z. Indukt. Abstamm. Vererbungsl. 68: 143-46.

genetics: X chromosome broken between *rb* and *rg*; 3R broken to the right of *ca*. Male and homozygous and heterozygous females viable and fertile. Crossing over in heterozygous female nearly absent in left end of X, rises to about normal around *ct*, and may be increased at right end. Crossing over in chromosome 3 in translocation heterozygote normal between *e^s* and *ro* and reduced to two-thirds normal between *ro* and *ca*. Male carrying the 3^{PX^D} element in place of a normal 3 nearly lethal;

female has narrow wings, occipital bristles, and branched posterior veins. Crossing over between normal X chromosomes about one-third of normal at left end in duplication-bearing female and nearly normal to right of *ct*.

T(1;3)3Ac⁵⁹

cytology: T(1;3)3A4-6;94F.

origin: Induced by triethylenemelamine.

synonym: T(1;3)zw1²¹⁴.

references: Lim and Snyder, 1974, Genet. Res. 24: 1-10.

genetics: Associated with l(1)3Ac⁵⁹ (= l(1)zw1²¹⁴).

T(1;3)3C1

cytology: T(1;3)3C1;81-82.

origin: X ray induced in In(1)w^{m51BL_wmJR}.

references: Gersh, 1967, Genetics 56: 309-19.

genetics: Males semiviable and sterile, sometimes M-like.

T(1;3)16

cytology: T(1;3)11F1-2;97D3-4.

origin: γ ray induced simultaneously with In(3LR)Antp^{rv16}.

references: Duncan and Kaufman, 1975, Genetics 80: 733-52.

T(1;3)16A4

cytology: T(1;3)9E;80 + T(1;3)14F;66A.

new order:

1 - 9E|80 - 66A|14F - 9E|80 - 100;

20 - 14F|66A - 61.

origin: Induced by ethyleneimine.

references: Lim and Snyder, 1968, Mutat. Res. 6: 129-37.

genetics: Homozygous lethal.

T(1;3)20A4

cytology: T(1;3)17F;83A;85D.

origin: Induced by ethyleneimine.

references: Lim and Snyder, 1968, Mutat. Res. 6: 129-37.

genetics: Homozygous lethal.

T(1;3)29A2

cytology: T(1;3)2F;91A.

origin: Induced by ethyleneimine.

references: Lim and Snyder, 1968, Mutat. Res. 6: 129-37.

genetics: Homozygous lethal.

T(1;3)33

cytology: T(1;3)20F;91F5-9.

origin: X ray induced.

references: Ashburner.

*T(1;3)54a: see Tp(1;3)w⁺54a4

*T(1;3)54c: see Tp(1;3)w⁺54c10

T(1;3)64b: see Tp(1;3)w⁺64b

***T(1;3)65**

cytology: T(1;3)16-17;79D.

origin: X ray induced.

discoverer: Lindsley, Edington, and Von Halle.

references: 1960, Genetics 45: 1649-70.

genetics: Male viable and sterile.

T(1;3)67k: see Tp(1;3)w⁺67k

T(1;3)71b: see Tp(1;3)f⁺71b

T(1;3)74c: see Tp(1;3)v⁺74c

***T(1;3)102**

origin: X ray induced.
discoverer: Sturtevant, 1930.
references: Dobzhansky, 1932, Biol. Zentr. 52: 495.
genetics: Breakpoint in X chromosome between *bb* and centromere; break in 3L between *ru* and *se*. Crossing over in 3L greatly reduced. Male and homozygous female fertile. Male and female hyperploid for the X^P3L^D element survive and are fertile; duplicated for locus of *ru* but not *se*, *h*, *car*, or *bb*.

***T(1;3)143-3**

origin: X ray induced.
discoverer: Neuhaus.
synonym: *T(1;3)Dell143-3*.
references: Belgovsky, 1941, DIS 15: 16.
genetics: Two breaks in the X chromosome (Gershenson), one between *sc* and *br* and another near the centromere (Belgovsky, 1941). A break in chromosome 3 is between *st* and the centromere of 3. The *y*⁺ and *sc*⁺ loci are then attached to the proximal end of 3L and the distal end of 3L is attached to the centromere of X. Bulk of the X chromosome is lost.

***T(1;3)260-21**

cytology: *T(1;3)6C;70E-F*.
origin: X ray induced simultaneously with *In(1)y*²⁶⁰⁻²¹.
discoverer: Sutton, 1939.
references: 1943, Genetics 28: 210-17.

T(1;3)429.17

cytology: *T(1;3)1B;11B;19C-D;67D;77C-D;94D-E;99A*.
new order:
 1A – 1B|11B – 19C|99A – 94E|77C – 67D|19D – 20F;
 61A – 67D|77D – 94D|1B – 11B|99A – 100F.
discoverer: Gelbart.

T(1;3)A59: see *Tp(1;3)f⁺71b*

T(1;3)A125

cytology: *T(1;3)18F-19A;83C + In(1)19E-20*.
origin: X ray induced.
discoverer: Lefevre.
genetics: Male lethal. Allele of *l(1)20Ac*.

T(1;3)Apx: Translocation (1;3) Antennapedix

cytology: *T(1;3)20D;79F;84B*.
references: Stepshin and Ginter, 1970, Genetika (Moscow) 6(9): 101-09.
genetics: Mutant for *Apx*. Homozygous lethal and lethal over *In(3R)Antp^B* [Stepshin and Ginter, 1972, Genetika (Moscow) 8(8): 98-104]. *Apx* clearly an *Antp* allele.

T(1;3)Aw^{m609e}: see *T(1;3)w^{m609e}*

***T(1;3)B⁵⁸¹: Translocation (1;3) Bar**

cytology: *T(1;3)16A;88F*.
origin: X ray induced.
discoverer: E.B. Lewis, 5814.
references: Ogaki, 1960, DIS 34: 97.
 1960, Jpn. J. Genet. 35: 282.
genetics: Position effect at *B*. Male sterile.

***T(1;3)Bb: Translocation (1;3) Bubble**

cytology: *T(1;3)13E;84F* (Morgan, Bridges, and Schultz, 1937, Year Book - Carnegie Inst. Washington 36: 301).
origin: X ray induced.

discoverer: King, 32d.

genetics: Associated with *Bb*. Male sterile.

T(1;3)bi^{D1}: Translocation (1;3) bifid

cytology: *T(1;3)4C5-6;65C3-5*.

origin: X ray induced.

references: Banga, Bloomquist, Brodberg, Pye, Larrivee, Mason, Boyd, and Pak, 1986, Chromosoma 93: 341-46.

genetics: Associated with *bi*, *Qd*, and *omb*. Male lethal.

T(1;3)bx⁶⁵⁷: Translocation (1;3) bithoraxoid

cytology: *T(1;3)X;81?;89E*.

origin: Induced by ethyl methanesulfonate.

discoverer: E.B. Lewis.

synonym: *bx^{16765.7}*.

genetics: Associated with *bx⁶⁵⁷*.

T(1;3)C: Translocation (1;3) Crossover suppressor

origin: X ray induced.

discoverer: Roberts, 1964, 1965.

references: 1970, Genetics 65: 429-48.

translocation	cytology	genetics
<i>T(1;3)C48^α</i>	<i>In(1)10E-F;18C-D + T(1;3)20;80-81</i>	male lethal; crossing over reduced in X
<i>T(1;3)C151^β</i>	<i>9D;80-81</i>	male viable but sterile; crossing over reduced in X
<i>T(1;3)C160^β</i>	<i>14B;80-81</i>	male lethal; crossing over reduced in X
<i>T(1;3)C195</i>	<i>11D;71A-B</i>	male viable and fertile; homozygous female viable; crossing over reduced in X
<i>T(1;3)C250^α</i>	<i>In(1)9F;15D-E + T(1;3)20;80-81</i>	male viable and fertile; homozygous female viable; crossing over reduced in X
<i>T(1;3)C291^γ</i>	<i>16C;20;87F;98E</i>	male viable and fertile; homozygous female viable; crossing over reduced in X
<i>T(1;3)C300^δ</i>	<i>12C;61F;66E;68D</i>	male dies in 3rd larval instar; crossing over reduced in 3L
<i>T(1;3)C315</i>	<i>20;70F</i>	male fertile; homozygous female lethal; crossing over reduced in 3L
<i>T(1;3)C329^β</i>	<i>3F;80-81</i>	male viable and sterile; crossing over reduced in X

^α Presence of *T(1;3)20;80-81* inferred from genetic data.

^β Position of breakpoint in chromosome 3 with respect to centromere not determined.

^γ New order: 1 – 16C|98E – 87F|(16C – 20)|87F – 61; 20|98B – 100.

^δ New order: 1 – 12C|68D – 100; 61 – 61F|66E – 61F|68D – 66E|12C – 20.

T(1;3)C212

cytology: *T(1;3)1A7;2C3;80*.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal.

T(1;3)C243

cytology: *T(1;3)20A3;75C*.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal.

T(1;3)CA17

cytology: *T(1;3)20F;86F*.

origin: γ ray induced.

references: Ashburner.

genetics: Associated with *b^{80jl}*.

T(1;3)CA45

cytology: *T(1;3)20?;97E*.
origin: γ ray induced.
references: Ashburner.
genetics: Induced simultaneously with *Df(2L)b84a2*.

***T(1;3)ct²⁶⁸⁻⁵: Translocation (1;3) cut**

cytology: *T(1;3)7B2-3;90C4-D1*.
origin: X ray induced.
discoverer: Demerec, 33k.
genetics: Mutant for *ct* but not *scp* or *sn*.

***T(1;3)ct²⁶⁸⁻²¹**

cytology: *T(1;3)7B3-4;7B4-5;96F*.
new order:
 1A – 7B3|96F – 61;
 20 – 7B5|96F – 100;
 deficient for 7B4.
origin: X ray induced.
discoverer: Hoover, 35i.
genetics: Mutant for *ct* but not *scp* or *sn*. Male lethal.

***T(1;3)ct²⁶⁸⁻³⁶**

cytology: *T(1;3)7B2-C1;66F* (Sutton).
origin: X ray induced.
discoverer: Demerec, 39j.
genetics: Mutant for *ct*. Male lethal.

T(1;3)ct^J

origin: X ray induced.
references: Lefevre and Johnson, 1973, *Genetics* 74: 633-45.
genetics: Male lethal.

translocation	cytology
<i>T(1;3)ct^{J2}α</i>	7B3-4;96A
<i>T(1;3)ct^{J9}</i>	7B3-4;86D-E
<i>T(1;3)ct^{J11}</i>	7B3-4;95F

α Viable over *ct^{71g}*. Not covered by *Dp(1;3)sn^{13a1}*.

***T(1;3)cu^{100.69}: Translocation (1;3) curled**

cytology: *T(1;3)6B1-C1;88A4-B1*.
origin: X ray induced.
discoverer: Alexander.
references: Ward and Alexander, 1957, *Genetics* 42: 42-54.
genetics: Mutant for *cu*.

T(1;3)D3

cytology: *T(1;3)4F;62A*.
origin: Induced by tritiated deoxycytidine.
discoverer: Kaplan, 1965.
references: 1966, DIS 41: 59.
genetics: Male lethal.

T(1;3)DEB: Translocation (1;3) diepoxybutane

origin: Induced by diepoxybutane. Selected by pseudolinkage in patroclinous sons of treated males.
references: Denell, Lim, and Auerbach, 1978, *Mutat. Res.* 49: 219-24.
genetics: Male fertile.

translocation	cytology
<i>T(1;3)DEB1</i>	20F;80-81
<i>T(1;3)DEB2</i>	20F;97
<i>T(1;3)DEB3</i>	20F;80-81
<i>T(1;3)DEB4</i>	20F;80-81

translocation	cytology
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<i>T(1;3)DEB5</i>	
<i>T(1;3)DEB6</i>	20F;62B
<i>T(1;3)DEB7</i>	20F;80-81

T(1;3)Del 143-3: see *T(1;3)143-3*

T(1;3)dsx: Translocation (1;3) doublesex

origin: X ray induced.
references: Baker, Hoff, Kaufman, Wolfner, and Hazelrigg, 1991, *Genetics* 127: 125-38.
genetics: Revertant of *dsx^M*.

translocation	cytology	synonym
<i>T(1;3)dsx³³</i>	18D3-5;20F;80-81;84E1-2	<i>T(1;3)dsx^{M+R12}</i>
<i>T(1;3)dsx⁴⁴</i>	20F;84D13-E8	<i>T(1;3)dsx^{M+R46}</i>

T(1;3)e^{H2}: Translocation (1;3) ebony of Henikoff

cytology: *T(1;3)20;93D*.
origin: X ray induced.
references: Henikoff, 1980, DIS 55: 61-62.
genetics: Variegated for *e* in XX and XXY females and XY males; XX flies more ebony than XXY or XY flies. Mutant for *e* in X0 males. Homozygous lethal.

***T(1;3)f²⁵⁷⁻²⁹: Translocation (1;3) forked**

cytology: *T(1;3)15F5-16A1;64*.
origin: X ray induced.
discoverer: Bishop, 40l.
genetics: Mutant for *f* but not *M(1)15D*, *B*, or *os*. Male viable and sterile.

T(1;3)FA11

cytology: *T(1;3)20F;84D-87D*.
references: Lehmann and Nüsslein-Volhard, *Dev. Biol.* 119: 402-17.

T(1;3)FA62

cytology: *T(1;3)19-20;84F-90D*.
references: Lehmann and Nüsslein-Volhard, *Dev. Biol.* 119: 402-17.

T(1;3)FC8

cytology: *T(1;3)20;84D-85D*.
origin: X ray induced.
references: Lehmann and Nüsslein-Volhard, 1987, *Dev. Biol.* 119: 402-17.
 Agnel, Kerridge, Vola, and Rabinow, 1989, *Genes Dev.* 3: 85-95.

***T(1;3)fd: Translocation (1;3) furled**

cytology: *T(1;3)7A;86E* superimposed on *In(3R)89C;96A* (Darby).
new order:
 1 – 7A|86E – 61;
 20 – 7A|86E – 89C|96A – 89C|96A – 100.
origin: Induced by ³²P in *In(3R)P*.
discoverer: Bateman, 1949.
references: 1950, DIS 24: 54.
 1951, DIS 25: 77.
genetics: Associated with *fd*.

T(1;3)GA

origin: X ray induced.
discoverer: Lefevre.
genetics: Male lethal.

translocation	cytology	mutant for
T(1;3)GA25	3E;80A-B	
T(1;3)GA79	2F-3A;82C	phl
T(1;3)GA91	1F4;99A	
T(1;3)GA119	1E1;93A	

T(1;3)GE222

cytology: T(1;3)2B6;83E.
origin: X ray induced.
discoverer: Lefevre.
genetics: Male lethal. Mutant for *br*.

T(1;3)GE245

cytology: T(1;3)3C7;96. Region from 80-96 complex.
origin: X ray induced.
discoverer: Lefevre.
genetics: Male lethal. *N* allele.

***T(1;3)H: Translocation (1;3) Hairless**

discoverer: Efromson.
references: Kamshilov, 1933, Biol. Zh. (Moscow) 2: 161-83.
genetics: Break in X chromosome to the left of *w*; *3R* broken near *H*.

T(1;3)h⁺⁰⁶

cytology: T(1;3)1B10-C1;66D14-E1.
origin: X ray induced.
references: Jeffery, 1979, Genetics 91: 105-25.
genetics: Male viable but sterile. Variegates for *h*.

T(1;3)HA14

cytology: T(1;3)19E;80.
origin: X ray induced.
discoverer: Lefevre.
genetics: Male lethal. *run* allele.

T(1;3)JA29

cytology: T(1;3)1E5;85A.
origin: X ray induced.
discoverer: Lefevre.
genetics: Male lethal.

T(1;3)JC59

cytology: T(1;3)2F6-3A1;92F-93A.
origin: X ray induced.
discoverer: Lefevre.
genetics: Male lethal. Mutant for *phl*.

T(1;3)JC153: see *Tp(1;3)JC153*

T(1;3)JK: Translocation (1;3) Jim Kennison

cytology: T(1;3)Xh;80.
origin: γ ray induced.
discoverer: Kennison.

translocation	genetics
T(1;3)JK1	male viable and fertile; variegates for <i>Pc</i> ⁴
T(1;3)JK2	male lethal
T(1;3)JK3	male lethal
T(1;3)JK4	male semi-lethal
T(1;3)JK5	male viable, but sterile
T(1;3)JK6	male viable and fertile

T(1;3)K2: see *Dp(1;3)K2*

***T(1;3)KA12**

cytology: T(1;3)19E;95D.
origin: X ray induced.
discoverer: Lefevre.
genetics: Male lethal. Allele of *l(1)19Ec*.

***T(1;3)l-184: Translocation (1;3) lethal**

cytology: T(1;3)18A;81.
origin: X ray induced.
references: Lindsley, Edington, and Von Halle, 1960, Genetics 45: 1649-70.
genetics: Associated with **l(1)184*.

T(1;3)l-v3: Translocation (1;3) lethal-variegated

origin: X ray induced.
references: Lindsley, Edington, and Von Halle, 1960, Genetics 45: 1649-70.
genetics: Variegated for a lethal; male sterile. Survival of X/Y males much greater than that of X/O males.

translocation	cytology
T(1;3)l-v3	4A;81
T(1;3)l-v163 ^{α}	17A-B;80-81
*T(1;3)l-v252 ^{β}	
*T(1;3)l-v361 ^{β}	19-20;80-81
T(1;3)l-v453 ^{α}	12D;80-81
T(1;3)l-v455 ^{γ}	3C;81
T(1;3)l-v463 ^{β}	19-20;81-82

^{α} Position of chromosome 3 breakpoint with respect to centromere not determined.
 ^{β} Position of breakpoints with respect to centromere not determined.
 ^{γ} Also variegated for *w*.

T(1;3)L1: see *T(1;3)v^{L1}*

***T(1;3)lz²⁶⁸⁻²⁹: Translocation (1;3) lozenge**

cytology: T(1;3)8D8-9;81F.
origin: X ray induced.
discoverer: Hoover, 38d.
genetics: Mutant for *lz* and independently for *ct* but not *sn*, *t*, *dvr*, *flp*, or *ras*. *T(1;3)lz²⁶⁸⁻²⁹/lz* female fertile. Male lethal.

T(1;3)M: Translocation (1;3) Mglinetz

translocation	cytology	origin	ref ^{α}
T(1;3)M50	20;87E	γ ray	2
T(1;3)M149	8C;86A	γ ray	1
T(1;3)M150	6B;90E	γ ray	1
T(1;3)M152	12A;91F	γ ray	1
T(1;3)M153	7F;100A	γ ray	1
T(1;3)M155	3A;97A	γ ray	1
T(1;3)M159	3F;84E	32 P feeding	1
T(1;3)M160	19D;99B	32 P feeding	1
T(1;3)M161	3A;64A	32 P feeding	1
T(1;3)M162	10B;67E	32 P feeding	1
T(1;3)M163	8E;71E	32 P feeding	1

^{α} 1 = Mglinetz, 1968, Genetika (Moscow) 4(8): 81-86; 2 = Mglinetz, 1972, Genetika (Moscow) 8(2): 82-92.

***T(1;3)N^{34b}: Translocation (1;3) Notch**

origin: X ray induced.
discoverer: Oliver, 34b3.
references: 1937, DIS 7: 19.
genetics: Carries mutant allele of *N* and normal alleles of *w* and *ec*.
other information: Reported as suspected of being a

T(1;3); basis of suspicion not given.

T(1;3)N^{81k9}

cytology: *T(1;3)3C6-9;3L* (Welshons).

origin: X ray induced.

discoverer: Muscavitch.

references: Grimwade, Muskavitch, Welshons, Yedvobnick, and Artavanis-Tsakonas, 1985, *Dev. Biol.* 107: 503-19.

genetics: Mutant for *N*.

molecular biology: Molecular breakpoint 24.2 to 22.6 kb to the left of the *In(1)N^{76b8}* breakpoint (Grimwade *et al.*, 1985).

****T(1;3)N²⁶⁴***

origin: X ray induced.

discoverer: Demerec.

translocation	cytology	new order α	ref β	genetics
* <i>T(1;3)N²⁶⁴⁻²⁹</i> γ	3D4-5;80 (Hoover)		1	<i>rst^v fa^v dm^v</i>
* <i>T(1;3)N²⁶⁴⁻³⁴</i>	3C3-5;70C2-3 (Hoover)		1	<i>N</i>
* <i>T(1;3)N²⁶⁴⁻⁵⁵</i>	3D4-5;80F9-81F1		1	<i>w^v rst^v fa^v dm^v</i>
* <i>T(1;3)N²⁶⁴⁻⁵⁶</i>	3D4-5;80 (Sutton)			<i>w^v N^v</i> (probably)
* <i>T(1;3)N²⁶⁴⁻⁶⁴</i>	3E5-6;80C-F (Hoover)		1	<i>w^v rst^v fa^v dm^v</i>
* <i>T(1;3)N²⁶⁴⁻⁶⁵</i>	2B10-16;3D4-5; 81E;96C4-5	1		<i>w^v rst^v fa^v dm^v</i>
* <i>T(1;3)N²⁶⁴⁻⁷⁰</i>	3C4-5;80D-F + 6F2-7A1; 100B2-3	2	2	<i>w^v rst^v fa^v dm^v</i>
* <i>T(1;3)N²⁶⁴⁻⁸³</i>	3C6-7;12F2-4;79E2-3 + <i>In(3R)81;88</i>	3	1	<i>N</i>
* <i>T(1;3)N²⁶⁴⁻¹⁰⁴</i>	3C7-9;87D1-E1 + <i>In(1)B4-5;18-19</i>	4		<i>svr N</i>
* <i>T(1;3)N²⁶⁴⁻¹²¹</i>	3C7-9;81F;86B6-C1	5		<i>N</i>
α	1 = 1 - 2B10 (81F - 96C4) 3D5 - 20; 61 - 81F (2B16 - 3D4) 96C5 - 100. 2 = 1 - 3C4 80F - 100B2 6F2 - 3C5 80D - 61; 20 - 7A1 100B3 - 100F. 3 = 1 - 3C6 12F2 - 3C7 79E3 - 81 88 - 81 88 - 100; 20 - 12F3 79E2 - 61. 4 = 1A1 - 1B4 18 - 3C9 87D1 - 61; 20 - 19 1B5 - 3C7 87E1 - 100. 5 = 1 - 3C7 81F - 86B6 81F - 61; 20 - 3C9 86C1 - 100.			
β	1 = Demerec, 1941, <i>Proc. Int. Congr. Genet.</i> 7th, pp. 99-103; 2 = Sutton, 1940, <i>Genetics</i> 25: 534-40.			
γ	<i>X/Y</i> male lethal; <i>X/Y/Y</i> male occasionally survives.			

T(1;3)N3

cytology: *T(1;3)1E1;71C1* and other rearrangements (complex).

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal.

T(1;3)N72

cytology: *T(1;3)7D;61F*.

origin: X ray induced.

discoverer: Lefevre.

references: Digan, Haynes, Mozer, Dawid, Forquignon, and Gans, 1986, *Dev. Biol.* 114: 161-69.

genetics: Allele of *fs(1)h*. Male lethal.

molecular biology: Lesion associated with the 7D breakpoint lies between 50.6 and 51.4 kb.

T(1;3)npr1: Translocation (1;3) nonpupariating 1

cytology: *T(1;3)2B5;61F3-4*.

origin: Induced by DEB.

synonym: *npr1⁷*; *npr^{JTD3}*.

references: Kiss, Beaton, Tardiff, Fristrom, and Fristrom, 1988, *Genetics* 118: 247-59.

genetics: Mutant for *npr1*.

T(1;3)O6

origin: X ray induced.

discoverer: Oliver, 34d24.

genetics: Mutant for *ec*. Break in 3L between *ru* and *h*.

Break in *X* not determined. Male and homozygous female viable and fertile.

T(1;3)OR: Translocation (1;3) from Oak Ridge

origin: X ray induced. Selected by virtue of pseudolinkage displayed by daughters of treated males.

discoverer: Warters, 1961.

translocation	cytology	new order α	genetics
<i>T(1;3)OR1^{\beta}</i>	5A;20;66B;79E	1	male lethal
<i>T(1;3)OR6</i>	4D;87F		male viable but sterile
<i>T(1;3)OR7</i>	14D;91E		male viable but sterile
<i>T(1;3)OR9</i>	6D;66B		variegated for a lethal; males sterile with small rough eyes
<i>T(1;3)OR11</i>	18F;84B		male viable but sterile
<i>T(1;3)OR12^{\gamma}</i>	2B6-13;84A + 18D;98F-99A (Becker)	2	male viable but sterile
<i>T(1;3)OR13</i>	15A;84E		male viable but sterile
<i>T(1;3)OR14</i>	17A;80B		variegated for a lethal; male sterile
<i>T(1;3)OR15</i>	18D;88A		male viable but sterile; male lethal acquired later
<i>T(1;3)OR17^{\beta}</i>	19E;67C		male viable, fertile; homozygous female viable
<i>T(1;3)OR18</i>	19B;80A		male viable but sterile
<i>T(1;3)OR19</i>	12E;75F		male viable but sterile
<i>T(1;3)OR21^{\beta}</i>	19E;61F		male viable but sterile; XO male lethal
<i>T(1;3)OR22</i>	6C;98C		male viable, fertile; homozygous female viable
<i>T(1;3)OR23^{\delta}</i>	20;80-81		male viable but sterile
<i>T(1;3)OR24</i>	12F;80B		male viable but sterile
<i>T(1;3)OR25^{\gamma}</i>	20B;99B		male viable but sterile
<i>T(1;3)OR28</i>	11A;80C		male lethal
<i>T(1;3)OR29</i>	16F;84B		male viable but sterile
<i>T(1;3)OR30^{\beta}</i>	19E;65D		male viable but sterile
<i>T(1;3)OR31</i>	10A;68D		male viable but sterile
<i>T(1;3)OR32</i>	16A;71B		male viable but sterile
<i>T(1;3)OR33</i>	13E;62F		male virtually lethal
<i>T(1;3)OR34</i>	3A;65A		male viable but sterile with Y or $B^S w^+ Y$
<i>T(1;3)OR35</i>	19E;75C		male viable but sterile
<i>T(1;3)OR36</i>	7D;62A;87E	3	male viable but sterile
<i>T(1;3)OR37</i>	3C;97F		male viable, fertile; homozygous female viable
<i>T(1;3)OR38^{\beta}</i>	18D;61D		male viable but sterile
<i>T(1;3)OR39</i>	6B-F;75C		male viable but sterile
<i>T(1;3)OR40</i>	6F;62D + 16B;20;84F	4	male viable but sterile
<i>T(1;3)OR41</i>	9F;98E		male viable but sterile
<i>T(1;3)OR43^{\gamma}</i>	20A;97D		male viable but sterile
<i>T(1;3)OR45</i>	17A;61D		male viable but sterile
<i>T(1;3)OR46</i>	12C;80A		variegated for a lethal; male sterile
<i>T(1;3)OR47^{\gamma}</i>	20;93D		male viable, fertile; homozygous female viable
<i>T(1;3)OR49</i>	11A;66D		male viable but sterile
<i>T(1;3)OR51</i>	12D;97A		male lethal
<i>T(1;3)OR52</i>	19E;70C;83F	5	male viable but sterile
<i>T(1;3)OR54</i>	12F;83A		male lethal
* <i>T(1;3)OR55</i>	11C;67C		male viable but sterile
* <i>T(1;3)OR57</i>	3E;5B;61C	6	male lethal even with $B^S w^+ Y$
<i>T(1;3)OR59^{\delta}</i>	20;80-81		male lethal
<i>T(1;3)OR60</i>	4B;88A		male lethal
<i>T(1;3)OR62</i>	10F;88C		male viable but sterile

translocation	cytology	new order α	genetics
<i>T(1;3)OR63</i>^{δ}	20;80-81		male sterile
<i>T(1;3)OR66</i>	3F;71E		male lethal even with $B^S w^+ Y$
<i>T(1;3)OR67</i>	4C;73C		male viable but sterile
<i>T(1;3)OR69</i>^{δ}	20;80-81		male viable but sterile
<i>T(1;3)OR71</i>	20;71D		male viable but sterile
<i>T(1;3)OR72</i>^{δ}	20;80-81		variegated for a lethal; male sterile

- α 1 = 1-5A|79E-66B|5A-20|79E-100;
20|66B-61.
2 = 1-2B6|84A-98F|18D-2B13|84A-61;
20-18D|99A-100 (tentative).
3 = 1-7D|62A-87E|62A-61;
20-7D|87E-100.
4 = 1-6F|62D-84F|20-16B|84F-100;
20|16B-6F|62D-61.
5 = 1-19E|83F-70C|83F-100;
20-19E|70C-61 (tentative).
6 = 1-3E|5B-3E|61C-100;
20-5B|61C-61A;
 β Male hyperploid for $X^P 3L^D$ survives.
 γ Male hyperploid for $X^P 3R^D$ survives.
 δ Positions of breakpoints with respect to centromere not determined.

***T(1;3)os^{bdw}*: Translocation (1;3) outstretched small eye-bending wings**

cytology: *T(1;3)16E;80C* (Nicoletti).
origin: X ray induced.
discoverer: Halfer, 1960.
genetics: Associated with *os^{bdw}*. Male sterile.

***T(1;3)P104*: Translocation (1;3) Pasadena**

cytology: *T(1;3)19-20;87F-88A*.
origin: X ray induced.
discoverer: E.B. Lewis.

***T(1;3)pn¹²*: Translocation (1;3) prune**

cytology: *T(1;3)2E1-2;98A1-2*.
origin: X ray induced.
references: Ilyina, Sorokin, Belyaeva, and Zhimulev, 1980, DIS 55: 205.
genetics: Mutant for *pn*. Male lethal.

T(1;3)pn³⁶

cytology: *Tp(3;1)2E1-2;61A;62C1-2*.
new order:
1A|62C3-100;
61A-62C2|2E1-1A|2E2-20.
Tentative.
origin: X ray induced.
references: Ilyina, Sorokin, Belyaeva, and Zhimulev, 1980, DIS 55: 205.
genetics: Mutant for *pn*.

***T(1;3)rb^{D1}*: Translocation (1;3) ruby**

cytology: *T(1;3)4C6-7;3R*.
origin: X ray induced.
references: Banga, Bloomquist, Brodberg, Pye, Larrivee, Mason, Boyd, and Pak, 1985, Chromosoma 93: 341-46.
genetics: Associated with *rb*. Male lethal.

T(1;3)RF29

cytology: *T(1;3)3D;7D4;93F-94A* + complex autosomal rearrangement.
origin: X ray induced.
discoverer: Lefevre.
genetics: Male lethal. Mutant for *dm*. Female fertile over

FM6 (Lefevre).

****T(1;3)rst*: Translocation (1;3) roughest**

origin: X ray induced.
discoverer: Ball, 32b25.
genetics: Associated with *rst*. Breakpoints in X chromosome near *w* and *bb*; position of breakpoint in chromosome 3 unknown.

***T(1;3)sc²⁶⁰⁻¹⁵*: Translocation (1;3) scute**

cytology: *T(1;3)1B4-5;71C-D*.
origin: X ray induced.
discoverer: Demerec, 381.
references: Sutton, 1943, Genetics 28: 210-17.
genetics: Mutant for *sc* but not *y* or *ac*. Male sterile.

****T(1;3)sc²⁶⁰⁻²⁰***

cytology: *T(1;3)1A8-B1;61A1-2*.
origin: X ray induced.
discoverer: Sutton, 39e.
references: 1943, Genetics 28: 210-17.
genetics: Mutant for *sc* but not *y*, *ac*, or *svr*. Male and homozygous female viable and fertile. The two halves of the translocation are recoverable independently. The X^{P3D} element should be deficient for *y* and *ac* but carry *sc²⁶⁰⁻²⁰*; it presumably is male lethal but survives in heterozygous female. The 3^{PX^D} element carries normal alleles of *y* and *ac* but not *sc²⁶⁰⁻²⁰* or *svr⁺* and should also carry normal alleles of *ve* and *ru*.

***T(1;3)sc^{J4}*: Translocation (1;3) scute of Jacobs-Muller**

cytology: *T(1;3)1B;3A3-C2;61A* (inferred from genetic tests); 1B-3A3 lost.
new order:
1A1-1B|61A-100;
20-3C2|61A.
origin: X ray induced.
discoverer: Jacobs-Muller.
references: Muller, 1932, Proc. Intern. Congr. Genet., 6th., Vol. 1: 225.
1934, DIS 2: 60.

genetics: The section of the X chromosome from 1B through 3A was presumably inserted elsewhere in the genome; it subsequently separated from the rest of the configuration and was lost. Base of the X, presumably capped by the undemonstrable terminus of $3L$ (i.e., X^{P3D}), is deficient for the tip of X through *z* and may be stocked in combination with a duplication for the tip of X, such as the 4^{PX^D} element from *T(1;4)w^{m5}* or *Dp(1;f)z⁹*. The 3^{PX^D} segregant carries normal alleles of *l(1)Ac*, *y*, and *ac* but is not demonstrably deficient for $3L$ factors since it is homozygous viable. y^+ localizes about 4 units to the left of *ru*. *T(1;3)sc^{J4}* restores viability of *In(1)sc^{V2}/0* males (Baker, 1971, Proc. Nat. Acad. Sci. USA 68: 2472-76).

****T(1;3)sc^K*: Translocation (1;3) scute of Krivshenko**

discoverer: Krivshenko.
references: Agol, 1936, DIS 5: 7.
genetics: Mutant for *sc*. Three-break rearrangement with X^D translocated to $3L^P$, $3L^D$ translocated to $3R^P$ and $3R^D$ translocated to X^P .

T(1;3)sc^{K3}

cytology: *T(1;3)1B2-3;61A1-2*.
origin: X ray induced.
discoverer: Krivshenko, 53j29.
references: 1959, DIS 33: 95-96.
genetics: Mutant for *sc*. Male fertile. Two halves of the translocation recoverable separately. $3^P X^D$ element is viable homozygous, although males are somewhat infrequent. $X^P 3^D$ is inviable in males and homozygous females but survives in heterozygous females.

T(1;3)sc^{KA8}

cytology: *T(1;3)1B4-5;98+*.
discoverer: Lefevre.
references: Carramolino, Ruiz-Gomez, Guerrero, Cam-puzano, and Modolell, 1982, EMBO J. 1: 1185-91.
genetics: Mutant for *sc*.
molecular biology: X breakpoint between coordinates +13.7 and +16.7 (Carramolino *et al.*, 1982).

T(1;3)Sh^{LC}: Translocation (1;3) Shaker

cytology: *T(1;3)16F1-2;80*.
origin: X ray induced.
references: Tanouye, Ferrus, and Fujita, 1981, Proc. Nat. Acad. Sci. USA 78: 6548-52.
 Papazian, Schwartz, Tempel, Jan and Jan, 1987, Science 237: 749-53.
genetics: Associated with *Sh¹⁵*.
molecular biology: Maps to a *HindIII-EcoRI* fragment between +54.1 and +59.1 kb (Kamb, Iverson, and Tanouye, 1987, Cell 50: 405-13) or +53 to +55 kb (Bau-man, Krah-Jentgens, Müller, Müller-Holtkamp, Seidal, Keckemethy, Casal, Ferrus, and Pongs, 1987, EMBO J. 6: 3419-29).

T(1;3)slu: Translocation (1;3) sluggish

cytology: *T(1;3)2E;97A10*.
origin: γ ray induced.
references: Sharma, 1977, Experientia 33: 171-73.
genetics: Males (semifertile) show abnormal phototactic behavior and light-dependent homosexual activity. Homozygous females sluggish and sterile. No crossing over reported in heterozygous females. Mutant phenotype recessive.

T(1;3)SMG: Translocation (1;3) Semenova Mglinetz Glotoff

cytology: *T(1;3)11A;70E*.
origin: γ ray induced.
references: Semenova, Mglinetz, and Glotoff, 1970, Genetika (Moscow) 6(4): 165-69.

T(1;3)SP: Translocation (1;3) from São Paulo

origin: γ ray induced. Selected as male-sterile mutations in a *y w X* chromosome.
discoverer: Lindsley and Musatti, 1961.
genetics: Male viable but sterile.

translocation	cytology
* <i>T(1;3)SP2</i>	20;90E
<i>T(1;3)SP11</i>	20;75B
<i>T(1;3)SP13^α</i>	20;80-81
<i>T(1;3)SP14^α</i>	20;80-81
* <i>T(1;3)SP15^β</i>	20;67
<i>T(1;3)SP21</i>	1B;83F
<i>T(1;3)SP22^α</i>	20;80-81

translocation	cytology
<i>T(1;3)SP26^α</i>	20;80-81
<i>T(1;3)SP34</i>	8A;84A
<i>T(1;3)SP37</i>	8F;64E
<i>T(1;3)SP38^γ</i>	10B10-12;85A8-12
<i>T(1;3)SP41</i>	3E;67C-D
<i>T(1;3)SP44^α</i>	20;80-81
<i>T(1;3)SP46</i>	11;98
<i>T(1;3)SP53</i>	12;92
<i>T(1;3)SP54^β</i>	20;67B
<i>T(1;3)SP59</i>	20;83C
<i>T(1;3)SP62</i>	20;89A
<i>T(1;3)SP63^β</i>	20;65
<i>T(1;3)SP68</i>	11A;80-81
<i>T(1;3)SP70^α</i>	20;80-81
* <i>T(1;3)SP73^δ</i>	20;89E
<i>T(1;3)SP79</i>	13D;64A
* <i>T(1;3)SP80^ε</i>	18C;100A
<i>T(1;3)SP82</i>	5B-C;81
<i>T(1;3)SP85^ζ</i>	16B;80-81
<i>T(1;3)SP90</i>	18D;68A
<i>T(1;3)SP96</i>	
<i>T(1;3)SP99</i>	12E;64E
<i>T(1;3)SP109</i>	
<i>T(1;3)SP112</i>	11B;85D
<i>T(1;3)SP122</i>	11E;92E
<i>T(1;3)SP123</i>	

- α Positions of breakpoints with respect to centromere not determined.
- β Male hyperploid for $X^P 3L^D$ element survives.
- γ Lefevre, 1970, DIS 45: 39.
- δ Mutant for *Ubx*.
- ϵ Male hyperploid for $X^P 3R^D$ element survives.
- ζ Position of chromosome 3 breakpoint with respect to centromere not determined.

T(1;3)Tab^{rv175}: Translocation (1;3) Transabdominal-reverted

cytology: *T(1;3)20;89E + In(3R)89F;90D*.
origin: X ray induced in *In(3R)Tab*.
references: Celniker and Lewis, 1987, Genes and Development 1: 111-23.
genetics: Revertant of the dominant *Tab* phenotype. When hemizygous, shows stronger transformation of the eighth ventral setal band toward that of the seventh than does *Tab/Df(3R)P9*; also, abnormal posterior spiracles and a rudimentary ninth ventral setal band.

T(1;3)Th1: Translocation (1;3) from Thymidine

cytology: *T(1;3)12C;65B*.
origin: From male treated with 3H -thymidine as larva.
discoverer: Kaplan.
genetics: Male lethal.

T(1;3)Uab⁵: Translocation (1;3) Ultraabdominal

cytology: *T(1;3)1F;89E3-4*.
origin: Induced by ethyl methanesulfonate.
references: Lewis, 1978, Nature 276: 565-70.
genetics: Phenotype associated with the distal part of the translocation; break in 89E to left of *Uab⁵* and to right or left of *iab2*.

T(1;3)Ubx: Translocation (1;3) Ultrabithorax

genetics: Associated with *Ubx*.

translocation	cytology	origin	discov.	synonym
<i>T(1;3)Ubx^{8A}</i>	5B;89E1-2			<i>Ubx^{21560.8A}</i>
<i>T(1;3)Ubx^{R49}</i>	20F;89E	X ray	Ramey	<i>Cbx^{revR17.49A}</i>
<i>T(1;3)Ubx^{X-A}</i>	20;89E			

T(1;3)v: Translocation (1;3) vermilion

cytology: *T(1;3)10A1-2;93B7-10* (Lefevre, 1970).
origin: X ray induced in a chromosome carrying *v*.
discoverer: Anderson, 1924.
references: 1925, Papers Mich. Acad. Sci. 5: 355-66.
 1926, Papers Mich. Acad. Sci. 7: 273-78.
 1929, Z. Indukt. Abstamm. Vererbungsl. 51: 397-411.
 Lefevre, 1970, DIS 45: 39.
 Zhimulev, Belyaeva, Khudyakov, and Pokholkova, 1980, DIS 55: 211.
 Zhimulev, Pokholkova, Bgatov, Semeshin, and Belyaeva, 1981, Chromosoma 82: 25-40.
genetics: Inseparable from *v*. Male viable and sterile. Male sterility associated with loss of central pair of microtubules from sperm-tail axoneme [Kiefer, 1973, Genetic Mechanisms of Development (F.H. Ruddell, ed.). Academic Press, New York, London, San Francisco, pp. 47-102]. Primary nondisjunction occurs with a frequency of about 2% in heterozygous females; secondary nondisjunction is 23%. Crossing over is reduced near *v* but approaches normal on both ends of the X.

T(1;3)v^{L1}

cytology: *T(1;3)3E1-2;90C9-11 + Df(1)10A1-2;10A4-5* (separable).
origin: X ray induced.
synonym: *T(1;3)L1*.
references: Lefevre, 1969, Genetics 63: 589-600.
 Zhimulev, Belyaeva, Pokholkova, Kochneva, Fomina, Bgatov, Khudyakov, Patzevich, Semeshin, Baricheva, Aizenzon, Kramers, and Eeken, 1982, DIS 58: 210-14.
genetics: Deficient for *v-l(1)10Ac*.

T(1;3)v^{L13}

cytology: *T(1;3)9B13-14;10A1-2;64F*.
origin: X ray induced.
references: Lefevre, 1969, Genetics 63: 589-600.
genetics: Mutant for *v*. Male lethal.

T(1;3)V5-2: Translocation (1;3) Valencia

cytology: *T(1;3)13A8-9;84E*.
origin: X ray induced.
references: Valencia, 1970, DIS 45: 37-38.
genetics: Male sterile.

T(1;3)V105

cytology: *T(1;3)12D3;8I*.
origin: X ray induced.
references: Valencia, 1970, DIS 45: 37-38.
genetics: Male sterile.

****T(1;3)Vel: Translocation (1;3) Velvet***

origin: X ray induced in *In(1)sc⁸*.
discoverer: Patterson.
references: 1934, DIS 2: 10.
genetics: Associated with *Vel*. Homozygous viable and fertile.

T(1;3)w^{DZL}: Translocation (1;3) white

cytology: *T(1;3)3C1-2;3L*.
origin: In a *w^{DZL/w^{DZL}}* female. Presumed to be a rearrangement generated by *w^{DZL}*.
references: Bingham, 1980, Cold Spring Harbor Symp. Quant. Biol. 45(2): 519-25.
genetics: *T(1;3)w^{DZL/w^{DZL}}* females have wild-type eye color.

****T(1;3)w^{m1}: Translocation (1;3) white-mottled***

origin: X ray induced.
discoverer: Muller, 1927.
references: 1930, J. Genet. 22: 299-334.
genetics: Variegated for *w* and *N*. *X/Y* males lethal; *X/Y/Y* males viable and sterile.
other information: First recorded case of variegated position effect.

****T(1;3)w^{m2}***

origin: X ray induced.
discoverer: Patterson.
references: Muller, 1930, J. Genet. 22: 299-334.
genetics: Variegated for *w*. Male sterile.

****T(1;3)w^{m258-32}***

cytology: *T(1;3)3C3-5;8I* (Demerec and Hoover).
origin: X ray induced.
discoverer: Demerec, 37I.
genetics: Variegated for *w* but not *rst*, *fa*, or *dm*. Male viable.

****T(1;3)w^{m258-44}***

cytology: *T(1;3)3C3-4;4D2-E1;80D*; deficient for 3C4-4D2.
origin: Aneuploid segregant from *T(1;2;3)w^{m258-44}/+*.

****T(1;3)w^{m258-54}***

cytology: *T(1;3)3B2-C1;19F2-20A1;20E;63C7-8*.
new order:
 1 - 3B2|63C8 - 100;
 20F|19F2 - 3C1|20A1 - 20E|63C7 - 61.
origin: X ray induced.
discoverer: Sutton, 40e.
genetics: Variegated for *w* and *rst* but not *pn* or *spl*. Male lethal.

T(1;3)w^{m264-58} : see *T(1;3)N²⁶⁴⁻⁵⁸*

****T(1;3)w^{m609e}***

cytology: *T(1;3)3C2-3;100C3-7*.
origin: X ray induced.
discoverer: Patterson.
synonym: *T(1;3)Aw^{m609e}*.
references: Griffen and Stone, 1938, Genetics 23: 149.
genetics: Variegated for *w*. Seems likely that the rearrangement is more complicated because a euchromatic-euchromatic translocation would not be expected to produce variegation.

T(1;3)w^{mSp} : see *Tp(1;3)w^{m49a}*

T(1;3)W^{rv19}: Translocation (1;3) Wrinkled-reverted

cytology: *T(1;3)18F3-5;75C3-7*.
origin: X ray induced.
discoverer: Faithfull.
references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou and Woodruff, 1980, DIS 55: 193-95.
genetics: Induced revertant of *W*.

T(1;3)X1^{ts}

cytology: *T(1;3)8C1-2;91D1-2*.
origin: γ ray induced.
references: Kaufman and Suzuki, 1974, Can. J. Genet. Cytol. 16: 579-92.
genetics: Temperature-sensitive lethal. Male sterile at all

temperatures. Lethal phenotype maps (by duplications) to 7C9-9D4.

T(1;3)X3^{ts}

cytology: *T(1;3)10E1-2;92F12-13.*

origin: γ ray induced.

references: Kaufman and Suzuki, 1974, *Can. J. Genet. Cytol.* 16: 579-92.

genetics: Temperature-sensitive lethal, leaky at 29°. Male sterile at all temperatures.

T(1;3)y^{67k5}: Translocation (1;3) yellow

cytology: *T(1;3)1B1-2;98C.*

origin: X ray induced.

discoverer: Lefevre, 67k5.

references: 1970, *DIS* 45: 32.

genetics: Male viable, fertile, and y.

****T(1;3)y²⁶⁰⁻¹¹***

cytology: *T(1;3)1B2-3;85F1-5.*

origin: X ray induced.

discoverer: Sutton, 39a.

references: 1943, *Genetics* 28: 210-17.

genetics: Mutant for y but not *ac*, *sc*, or *svr*. Male viable and sterile.

****T(1;3)y²⁶⁰⁻²¹***

cytology: *T(1;3)6C;70E-F + In(1)1A6-7;5D8-E1.*

new order:

1A1 – 1A6|5D8 – 1A7|5E1 – 6C|70F – 100;
20 – 6C|70E – 61.

origin: X ray induced.

discoverer: Sutton, 1939.

references: 1943, *Genetics* 28: 210-17.

genetics: Mutant for y but not *sc*. Male lethal.

T(1;3)y^{A78J}

cytology: *T(1;3)1B1-2;82A1.*

origin: γ ray induced.

references: Alexandrov, Ankina, and Alexandrova, 1985, *DIS* 61: 212-13.

genetics: Mutant for y.

T(1;3)y^{R19}

cytology: *T(1;3)1A5-1B1;87A.*

origin: X ray induced.

references: Roberts, 1974, *Mutat. Res.* 22: 139-44.

genetics: Male viable, fertile, and y.

T(1;3)y^{R26}

cytology: *T(1;3)17C;70C.*

origin: X ray induced.

references: Roberts, 1974, *Mutat. Res.* 22: 139-44.

genetics: Male viable but sterile. Body color y although break at proximal end of the X, independent of y locus.

T(1;3)z⁺: Translocation (1;3) zeste-wild type

origin: X-ray-induced rearrangements of z-bearing X chromosomes that interfere with z-w interaction.

genetics: z⁺ phenotype.

translocation	cytology	ref ^α
<i>T(1;3)z⁺7E8</i>	<i>3C6-7;75A2-3;85F-86A;93C-93E</i>	1
<i>T(1;3)z⁺9</i>	<i>3C;84A</i>	2
<i>T(1;3)z⁺12G2</i>	<i>3C1-2;93F2-4</i>	1
<i>T(1;3)z⁺13E8</i>	<i>2F2;3C3-5;13D2-F4;96F2-4;97A3-B1</i>	1
<i>T(1;3)z⁺13</i>	<i>3C2-3;85D-E</i>	2
<i>T(1;3)z⁺14</i>	<i>3C1-2;75B-C</i>	2

translocation	cytology	ref ^α
<i>T(1;3)z⁺15</i>	<i>1B4-9;83F2-4 + In(1)4D-5A;12A-B</i>	2
<i>T(1;3)z⁺19G3</i>	<i>3C1-2;71D + other autosomal breaks</i>	1
<i>T(1;3)z⁺21</i>	<i>3C1-2;4C4-6;72A-F + T(1;3)8A-F;75-76</i>	2
<i>T(1;3)z⁺22</i>	<i>3C3-5;71A-C</i>	2
<i>T(1;3)z⁺26</i>	<i>3C3-5;80-81</i>	2

^α 1 = Gans, 1953, *Bull. Biol. Fr. Belg. Supp.* 38: 1-90; 2 = Gelbart, 1971, Ph.D. Thesis, Univ. of Wisconsin.

T(1;3)z7: Translocation (1;3) zeste

cytology: *T(1;3)3D3-4;100D1-2.*

origin: X ray induced in z w⁺ X chromosome.

references: Gans, 1953, *Bull. Biol. Fr. Belg. Suppl.* 38: 1-90.

Jack and Judd, 1979, *Proc. Nat. Acad. Sci. USA* 76: 1368-72.

genetics: Eye color zeste in z w⁺ males carrying the duplication.

T(1;3)zw1²¹⁴: see *T(1;3)3Ac⁵⁹*

T(1;3)ZWD

origin: γ -ray-induced in a z¹-bearing X chromosome.

references: Smolik-Utlaut and Gelbart, 1987, *Genetics* 116: 285-98.

translocation	cytology	genetics
<i>T(1;3)ZWD6^α</i>	<i>3C;67F + 20;90D + In(1)11E;19E</i>	w ⁺ sterile males
<i>T(1;3)ZWD9</i>	<i>3C1;98D</i>	w ⁺ males, z females
<i>T(1;3)ZWD15^β</i>	<i>3C1-2;75F + 11A;88E</i>	male lethal

New order: 1A – 3C|67F – 90D|20 – 19E|11E – 19E|11E – 3C|67F – 61A; 20|90D – 100F.

New order: 1A – 3C1|75F – 88E|11A – 3C2|75F – 61A; 20 – 11A|88E – 100F.

****T(1;3;4)A: Translocation (1;3;4) from Austin***

origin: X ray induced.

discoverer: Patterson, Stone, Bedichek, and Suche.

synonym: *T(1;4)3A.*

references: Painter and Stone, 1935, *Genetics* 20: 327-41.

****T(1;3;4)A96b***

discoverer: *T(1;3;4)3C3-7;10;101F*; break in chromosome 3 not determined (Mackenson, 1935).

new order:

1 – 3C3|chrom3^P;
20 – 3C7|101F – 102F;
chrom3^D|101F – 101A.

origin: X ray induced.

discoverer: Mackensen.

references: 1935, *J. Heredity* 26: 163-74 (fig.).

genetics: Variegated for w.

T(1;3;4)R45

cytology: *T(1;3;4)X;61C3-4;101A-C.*

references: Gelbart, 1971, Ph. D. Thesis, Univ. of Wisconsin.

T(1;3;4)Tab^{rv114}

cytology: *T(1;3;4)20;89E;101 + In(3R)89E;90D.*

origin: X ray induced in *In(3R)Tab*.

references: Celniker and Lewis, 1987, *Genes and Development* 1: 111-23.

genetics: Revertant of the dominant *Tab* phenotype. When

hemizygous, shows stronger transformation of the eighth ventral setal band toward that of the seventh than does *Tab/Df(3R)P9*; also, abnormal posterior spiracles and a rudimentary ninth ventral setal band.

molecular biology: Lesion associated with the 89E break-point lies between 183 and 184 kb.

T(1;4)1: see *T(1;4)B^S*

**T(1;4)1*: see *Tp(1;4)A1*

**T(1;4)3A*: see *T(1;3;4)A*

T(1;4)4: see *T(1;4)B^S*

T(1;4)4C3

cytology: *T(1;4)4C3;101-102*.

discoverer: Lindsley.

references: Hawley, 1980, *Genetics* 94: 625-46.

****T(1;4)231b***

origin: X ray induced.

discoverer: Patterson.

references: Patterson and Painter, 1931, *Science* 73: 530-31.

Patterson, 1932, *Am. Nat.* 66: 193-206.

1932, *Genetics* 17: 38-59.

genetics: Variegated for *N* and *w*. Left end of *X* from *sc* to *ec* attached to chromosome 4.

T(1;4)A: Translocation (1;4) Austin

origin: X ray induced.

translocation	cytology	discoverer	ref ^α
* <i>T(1;4)A2</i>	<i>X-4</i>		2,5,10
* <i>T(1;4)A3</i>	10% of <i>X</i> transferred to 4	Patterson, 301	2,4,6,7
* <i>T(1;4)A4</i>	13F6-14A1		2,3,8,9,10,11
* <i>T(1;4)A5</i>	<i>X-4</i>		2,5,12
* <i>T(1;4)A6</i>	<i>X-4</i>		2,5,12
* <i>T(1;4)A7</i>	<i>X-4</i>		2,5,12
* <i>T(1;4)A8</i>	11A6-7		2,8,9,10,11,12
<i>T(1;4)A9</i>	5A1-4		2,8,9,10,11,12
* <i>T(1;4)A10</i>	1A5-6;102A2-4		2,10,12,13
* <i>T(1;4)A11</i>	<i>X-4</i>		1,2,5,12
<i>T(1;4)A13</i>	18C5-D1		2,8,9,10,11,12
<i>T(1;4)A14</i>	<i>X-4</i>		1,2,5,12
<i>T(1;4)A17^β</i>	7F5-8A2	Mickey	2,9,11

^α 1 = Brown, 1940, *Texas Univ. Publ.* 4032: 65-72; 2 = CP627; 3 = Mackensen, 1935, *J. Hered.* 26: 163-74; 4 = Muller and Painter, 1932, *Z. Indukt. Abstamm. Vererbungsl.* 62: 316-65; 5 = Painter and Stone, 1935, *Genetics* 20: 327-41 (fig.); 6 = Patterson, 1932, *Genetics* 17: 38-59; 7 = Patterson and Painter, 1931, *Science* 73: 530-31; 8 = Patterson, Stone, and Bedichek, 1935, *Genetics* 20: 259-79 (fig.); 9 = Patterson, Stone, and Bedichek, 1937, *Genetics* 22: 407-26; 10 = Patterson, Stone, Bedichek, and Suche, 1934, *Am. Nat.* 68: 359-69; 11 = Pipkin, 1940, *Texas Univ. Publ.* 4032: 126-56; 12 = Stone, 1934, *Genetica* 16: 506-20; 13 = Stone and Griffen, 1940, *Texas Univ. Publ.* 4032: 208-17;

^β *X* chromosome broken between *t* and *lz*, although the reported cytological breakpoint is to the left of this interval. The $4^P X^D$ element survives when added to either a normal diploid female or a triploid intersex genotype; at least in the latter, the product is a fertile female. The complementary $X^P 4^D$ is virtually lethal when added to a diploid female genotype but produces partially fertile females when added to a triploid intersex genotype.

T(1;4)A4: see *T(1;4)B^S*

T(1;4)A5: see *T(1;4)w^{m5}*

T(1;4)B^{68f}

cytology: *T(1;4)15F;101* (chromocenter).

origin: X ray induced.

references: Brosseau, 1969, *DIS* 44: 45.

genetics: Associated with *B^{68f}*, a moderate allele of *Bar*. Male viable but sterile.

T(1;4)B^S: Translocation (1;4) Bar of Stone

cytology: *T(1;4)15F9-16A1;16A7-B1;102F* (Griffen, 1941, *Genetics* 26: 154-55).

new order:

1 – 16A7|102F – 101F;

20 – 16A1|102F.

origin: X ray induced in *Dp(1;1)B = Dp(1;1)15F9-16A1;16A7-16B1*.

discoverer: Stone, 1931.

synonym: *T(1;4)1; T(1;4)4; T(1;4)A4*.

references: 1934, *Genetica* 16: 506-20.

Novitski, 1970, *DIS* 45: 87.

Childress and Hartl, 1972, *Genetics* 71: 417-27.

genetics: Position effect at *B* more extreme than in *Dp(1;1)B*. Homozygous female viable and fertile. Male also viable but with reduced fertility (Novitski, 1970). The $X^P 4^D$ segregant carries no known markers from chromosome 4 and *B^S* through *bb⁺* from *X*. Female hyperploid for this element viable and fertile. Hyperploid male poorly viable and sterile.

other information: Used by Stern in cytological demonstration of crossing over (1931, *Biol. Zentr.* 51: 547-87). $X^P 4^D$ from *T(1;4)B^S* used by Lindsley and Sandler [1963, *Methodology in Basic Genetics* (W. J. Burdette, ed.). Holden-Day, Inc., pp. 390-403] in construction of compound-generating *B^S* duplications. Reciprocal products of meiosis in male not recovered with equal frequencies (Novitski and Sandler, 1957, *Proc. Nat. Acad. Sci. USA* 43: 318-24; Zimmering, 1960, *Genetics* 45: 1253-68; Zimmering and Barbour, 1961, *Genetics* 46: 1253-60; Zimmering and Perlman, 1962, *Can. J. Genet. Cytol.* 4: 333-36; Novitski, 1970, *DIS* 45: 87).

T1;4)e15

cytology: *T(1;4)13C;101-102*.

references: McKee, 1987, *Genetics* 116: 409-13.

****T(1;4)HF346***

cytology: *T(1;4)2C1-2;101*.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal.

T(1;4)JC43

cytology: *T(1;4)3B1-2;3E3-4;102D* (Lefevre).

new order:

1A – 3B1|102D – F;

101A – 102D|3E3 – 3B2|3E4 – 20.

origin: X ray induced.

discoverer: Lefevre.

references: Young and Judd, 1978, *Genetics* 88: 723-42.

Smith and Konopka, 1981, *Mol. Gen. Genet.* 183: 243-51.

Reddy, Zehring, Wheeler, Pirotta, Hadfield, Hall, and Rosbach, 1984, *Cell* 38: 701-10.

genetics: Male lethal. *X* break near *per*; disrupts normal expression of gene (Reddy *et al.*, 1984).

molecular biology: 3B1-2 breakpoint is approximately 100 kb distal to the *w^a copia* insertion point (Pirotta,

Hadfield, and Pretorius, 1983, EMBO J 2: 927-34; Reddy *et al.*, 1984).

T(1;4)l-v11: Translocation (1;4) lethal-variegated

cytology: *T(1;4)15;101*.

origin: X ray induced.

references: Lindsley, Edington, and Von Halle, 1960, Genetics 45: 1649-70.

genetics: Variegated for a lethal. Male fertile.

T(1;4)m⁵: see *T(1;4)w^{m5}*

****T(1;4)M-pro: Translocation (1;4) Minute-producer***

discoverer: Bridges, 33d26.

synonym: *M-pro: Minute-producer*.

genetics: Minutes produced are haplo-4's. The translocation causes nondisjunction of chromosome 4 centromeres (L. V. Morgan, 1940, DIS 13: 51).

T(1;4)N²⁶⁴: Translocation (1;4) Notch

origin: X ray induced.

discoverer: Demerec.

translocation	cytology	genetics
<i>T(1;4)N²⁶⁴⁻¹² αβ</i>	<i>3C6-7;101F</i> (Sutton)	mutant for <i>N</i> ; variegates for <i>w</i> , <i>rst</i> ; <i>ci</i> ⁺ shows weakened dominance
* <i>T(1;4)N²⁶⁴⁻²⁰ γ</i>	<i>3C4-5;3C7-8;101F</i> [3C5-7 missing (Sutton)]	deleted for <i>N</i> ; variegates for <i>w</i> , <i>rst</i> ; <i>ci</i> ⁺ shows weakened dominance
* <i>T(1;4)N²⁶⁴⁻¹¹³ δ</i>	<i>3C10-D1;101</i> (102 missing)	variegates for <i>w</i> , <i>N</i> ; carries normal <i>ci</i> allele (Stern)

α Synonym: *T(1;4)N^{a8}*.

β References: Demerec, 1941, Proc. Int. Congr. Genet., 7th, pp. 99-103; Judd, 1955, DIS 29: 126-27.

γ New order: 1 – 3C4|101F;
20 – 3C8|101F – 102.

δ New order: 1 – 3C10|101;
20 – 3D1|?.

Proximal portion of X chromosome considered to be terminally deficient although it occasionally appears to be capped by a small nucleolus-like structure (Sutton, 1940, Genetics 25: 628-35). Not clear that a reciprocal translocation between X and short arm of 4 was adequately ruled out.

T(1;4)N^{a8}: see *T(1;4)N²⁶⁴⁻¹²*

T(1;4)NO: Translocation (1;4) Nucleolus Organizer

cytology: *T(1;4)20F;101-102*.

references: Atwood, 1969, Genetics 61: 319-27.

Komma and Endow, 1986, Genetics 114: 859-74.

genetics: *NO* translocated to 4.

other information: Useful as source of rDNA in *bb*⁻ stocks.

T(1;4)pn^{2a}: Translocation (1;4) prune

cytology: *T(1;4)2D5-6;101F*.

origin: X ray induced.

references: Ilyina, Sorokin, Belyaeva, and Zhimulev, 1980, DIS 55: 205.

Slobodyanyuk and Serov, 1983, Mol. Gen. Genet. 191: 372-77.

genetics: Mutant for *pn*. Male lethal.

T(1;4)RF43

cytology: *T(1;4)3A2-3;101-102* (complex).

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal. Allele of *l(1)3Ac*.

****T(1;4)RF60***

cytology: *T(1;4)3C;101-102*.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal. *N* allele.

****T(1;4)sc¹⁰⁻²: Translocation (1;4) scute***

cytology: *T(1;4)1A;102F + T(1;4)1D;101F*; two reciprocal *l-4* translocations, according to Schultz.

origin: X ray induced in *In(1)ac³*.

discoverer: Sturtevant, 1930.

references: 1934, Proc. Nat. Acad. Sci. USA 20: 515-18. 1936, Genetics 21: 444-66.

genetics: Mutant for *sc*. Virtually male lethal. X chromosome broken between *M(1)1B* and *br*; chromosome 4 broken proximal to *ci*. According to Schultz, both X and 4 also have breaks distal to all known loci and their termini are interchanged. *4^PX^D* carries X-chromosome loci from y through *M(1)1B* and was used extensively by Sturtevant (1934, 1936) in his studies on preferential segregation. The *X^{P4D}* element survives in the heterozygous female but is an extreme Minute owing to deficiency for *M(1)1B* and is rarely fertile.

T(1;4)sc^H: Translocation (1;4) scute of Hackett

cytology: *T(1;4)1B4-C3;101-102*; inferred from genetic results.

origin: γ ray induced.

discoverer: Hackett, 46a.

references: Muller and Valencia, 1947, DIS 21: 70.

García-Bellido, 1979, Genetics 91: 491-520.

genetics: Relatively viable and *sc* (García-Bellido, 1979).

Two halves of the translocation may be recovered separately. *X^{P4D}* is deficient for y and *sc* but not *M(1)1B* and carries *ey⁺*. *4^PX^D* covers *Df(1)sc¹⁹* and therefore carries a normal allele of *l(1)sc*. *T(1;4)sc^H/ci* is *ci⁺*.

molecular biology: X-chromosome breakpoint between coordinates +5.9 and +6.4 (Carramolino, Ruiz-Gómez, Guerrero, Campuzano, and Modolell, 1982, EMBO J. 10: 1185-91).

T(1;4)Sidky

cytology: *T(1;4)13C;101-102*.

references: McKee, 1987, Genetics 116: 409-13.

T(1;4)V46

cytology: *T(1;4)7D10-11;101F-102A*.

origin: X ray induced.

references: Valencia, 1970, DIS 45: 37-38.

genetics: Recessive lethal associated with 4.

**T(1;4)w¹³*: see *Tp(1;4)A1*

T(1;4)w^{70L26.5}: Translocation (1;4) white

cytology: *T(1;4)3C1-2;12B8-9;102F*. Ring-like chromomere complex in 3C1; remnants of band material between 3C1 and 3C2 missing (Sorsa, 1973).

new order:

1 – 3C1|102F – 101;

102F|12B8 – 3C2|12B9 – 20.

origin: X ray induced in $In(1)z^{+64b9} = In(1)3C1-2;12B8-9$.
references: Sorsa, 1973, Cold Spring Harbor Symp. Quant. Biol. 38: 601-08.
 Sorsa, Green, and Beermann, 1973, Nature (London) New Biol. 245: 34-37.
genetics: Homo- and hemizygous viable with white eyes.

***T(1;4)w²⁵⁸⁻⁴³**

cytology: $T(1;4)3C3-5;102F4-5$ (Demerec).
origin: X ray induced.
discoverer: Demerec, 38k.
genetics: Mutant for *w* but not *kz*, *pn*, *rst*, or *fa*. Male lethal.

***T(1;4)w^{m3}**: see $T(1;4)A3$

T(1;4)w^{m5}: Translocation (1;4) white-mottled

cytology: $T(1;4)3C2;101F1-2$ (Pipkin and Chakrabarty, 1975, Genetics 80: s64). Duplication 1.9 times the length of 4 at metaphase.
origin: X ray induced.
discoverer: Muller, 1929.
synonym: $T(1;4)m^5$: Translocation (1;4) mottled-5; $T(1;4)A5$.
references: 1930, J. Genet. 22: 299-334.
 Bolen, 1931, Am. Nat. 65: 417-22.
 Grell and Day, 1970, Chromosoma 31: 434.
genetics: Variegates for *w* and *ci* [Dubinin, Sokolov, and Tiniakov, 1935, Biol. Zh. (Moscow) 4: 716-20]. X chromosome broken between $l(1)3C3$ and *rst*, and chromosome 4 broken to the left of *ey*. $4^P X^D$ added to a normal male genome produces males with 20% normal viability that are weakly fertile; added to a diploid female genome, it produces a fertile hyperploid genome; and added to a triploid intersex genome, it is virtually lethal. $X^P 4^D$ is inviable when added to a male genome, is virtually lethal when added to a female genome, and produces rather fertile hypotriploid females when added to a triploid intersex genome (Pipkin, 1940, Texas Univ. Publ. 4032: 126-56). Griffen and Stone (1940, Texas Univ. Publ. 4032: 190-200) produced and studied a number of X-ray-induced derivatives of $T(1;4)w^{m5}$.

***T(1;4)w^{m11}**

cytology: $T(1;4)3C3-4;101A-D$.
origin: X ray induced.
discoverer: Panshin.
references: Panshin and Khvostova, 1938, Biol. Zh. (Moscow) 7: 359-80.
 Panshin, 1938, Nature 142: 837.
 1941, DIS 15: 33-34.
genetics: Variegated for *w* but not *ci*. First rearrangement to involve, and therefore to demonstrate, existence of 4L. Panshin and Khvostova [1938; Panshin, 1938, Biol. Zh. (Moscow) 7: 837-65] produced and studied a number of X-ray-induced derivatives of $T(1;4)w^{m11}$.

T(1;4)w^{m258-18}

cytology: $T(1;4)3C4-5;101$.
origin: X ray induced.
discoverer: Demerec, 33k.
references: Demerec and Slizynska, 1937, Genetics 22: 641-49.
genetics: Variegated for *w* and *rst* but not *pn*, *fa*, *dm*, or *ec*. Also variegated for *ci* (Stern). Male and homozygous

female viable and fertile. X chromosome broken between *rst* and *vt* (Gersh, 1965, Genetics 51: 477-80). The $4^P X^D$ element survives as a duplication.

T(1;4)w^{m258-21}

cytology: $T(1;4)3E5-6;101F$ (Demerec and Hoover).
origin: X ray induced.
discoverer: Demerec, 1934.
synonym: $T(1;4)w^{vD3}$: Translocation (1;4) white-variegated of Demerec.
references: Hartmann-Goldstein, 1967, Genet. Res. 10: 143-59.
 Cowell and Hartmann-Goldstein, 1980, Chromosoma 79: 329-40.
 Kornher and Kauffman, 1986, Chromosoma 94: 205-16.
genetics: Variegates for *w*, *fa*, *spl*, *N*, *Sgs-4*, *dm*, and $M(1)3E$ but not *ec* or *bi*. Also variegates for *ci* (Gersh). Low temperature increases variegation of larval Malpighian tubules and also heterochromatic appearance of the transposed euchromatin (Hartmann-Goldstein, 1967). Cytological compaction of the *Sgs-4* locus (3C11-12) enhanced at low temperature (17°C) and *Sgs-4* protein and transcript reduced in translocation heterozygotes at this temperature (Kornher and Kauffman, 1986). Males usually lethal; survivors probably *X/Y/Y*. Cell lethal in *X/0* tissue in gynandromorphs (Judd, 1953, DIS 27: 95).

***T(1;4)w^{m258-31}**

cytology: $T(1;4)3C3-5;102F4-17$ (Demerec and Hoover).
origin: X ray induced.
discoverer: Demerec, 37l.
genetics: Variegated for *w* but not *rst*. Male viable.

***T(1;4)w^{m258-53}**

cytology: $T(1;4)3C1-2;101E-F$; distal part of chromosome 4 lost. Sutton thought it a terminal deficiency of X. Evidence that chromosome 4 is involved seems equivocal, especially since, according to events postulated, the original mottled fly should have been haplo-4. Alternative interpretation is translocation between X and Y in *X/Y* sperm.
new order:
 1A – 3C1|101E – 101A;
 20 – 3C2|?.
origin: X ray induced.
discoverer: Demerec, 39l.
references: Sutton, 1940, Genetics 25: 628-35.
genetics: Variegated for *w* but not *pn*, *rst*, or *spl*. Male viable. Translocation-bearing fly carries two normal fourth chromosomes.

T(1;4)w^{mA}: Translocation (1;4) white-mottled from Austin

cytology: $T(1;4)3C2-3;101A2-3$.
origin: X ray induced.
discoverer: Stone.
references: Griffen and Stone, 1939, Genetics 24: 73.
 1940, Texas Univ. Publ. 4032: 201-7 (fig.).
genetics: Variegated for *w*. Male viable and fertile. Second demonstration of the existence of a left arm on chromosome 4.

***T(1;4)w^{MD3}*: Translocation (1;4) white-mottled of Dubinin**

cytology: *T(1;4)3C;101*.

discoverer: Dubinin.

references: Sacharov, 1936, Biol. Zh. (Moscow) 5: 293-302.

genetics: Variegated for *w*.

***T(1;4)w^{MDV4}*: Translocation (1;4) white-mottled of Dubinin and Volotov**

cytology: *T(1;4)3C3-7;3D;101A-D*; 3C-3D missing; inferred from genetic data and from figs. 5-7 of Sacharov (1936) which indicate that the break in chromosome 4 is in the left arm.

new order:

1 - 3C3|101D - 102F;

20 - 3D|101A.

discoverer: Dubinin and Volotov.

references: Sacharov, 1936, Biol. Zh. (Moscow) 5: 293-302 (fig.).

genetics: Deficient for *N*; variegated for *w*. Male lethal. Since the 4^{PX^D} element of *T(1;4)w^{MDV4}* survives as a duplication and carries *w^{MDV4}*, the left break in the X chromosome is between *w* and *N*.

***T(1;4)w^{mJ}*: Translocation (1;4) white-mottled of Jonsson**

cytology: *T(1;4)3C2-3;20F;102C*.

new order:

1 - 3C2|20F - 3C3|102C - 101A;

20|102C - 102F.

origin: X ray induced.

discoverer: Jonsson, 61i28.

references: Lefevre, 1963, DIS 37: 49.

Lefevre and Wilkins, 1966, Genetics 53: 175-87.

genetics: Variegated for *w*. The X^{P4^D} element of the translocation has become separated from the 4^{PX^D} element and lost. The 4^{PX^D} element is viable as an *X/Y* male but lethal as an *X/O* male, probably owing to deficiency for *bb*. Additional evidence for appreciable deficiency for proximal X heterochromatin is virtually random disjunction of X and Y chromosomes. 4^{PX^D} carries *ci⁺* but not *ey⁺*. The variegation of white is unorthodox because heterochromatin has been moved to the white locus rather than white moved into proximal heterochromatin.

***T(1;4)w^{mK}*: Translocation (1;4) white-mottled of Krist**

cytology: Distal tip of X translocated to centromere of 4.

origin: X ray induced.

references: Breugel, 1972, Genetica 43: 25-42.

genetics: Variegates for *w*. Most of the homozygous females and hemizygous males have mottled eyes with large spots; a few have almost wild-type or pale eyes without large spots. More pigment at 25° than at 16°.

****T(1;4)w^{mMed}*: Translocation (1;4) white-mottled of Medvedev**

discoverer: Medvedev, 1934.

genetics: Variegated for *w* and probably *rst*. Arose in *w^a* and therefore has light eye color.

T(1;4)w^{vD3}: see *T(1;4)w^{m258-21}*

T(1;4)y^{A74c40}

cytology: *T(1;4)1B1-2;101F*.

origin: Induced with caffeine and γ rays.

references: Alexandrov, Ankina, and Alexandrova, 1985, DIS 61: 212-13.

genetics: Mutant for *y*.

***T(1;4)z^{20G1}*: Translocation (1;4) zeste**

discoverer: *T(1;4)3C1-2;102F2-4*; genetic data more in accord with breakpoint in 3C2-3 than 3C1-2.

origin: X ray induced in a chromosome carrying *z*.

discoverer: Gans.

references: 1953, Bull. Biol. France Belg. Suppl. 38: 1-90 (fig.).

Gersh, 1963, DIS 37: 80.

genetics: Suppresses *z*. The 4^{PX^D} element is poorly viable when added to male genome but viable and fertile in female; duplicated for *w* but does not cover lethality of *Df(1)w^{m4L}rst^{3R}* = *Df(1)3C1-2;3C3-4* (Gersh, 1963).

T(1;4)z⁺

origin: X-ray-induced rearrangements of *z*-bearing X chromosomes that interfere with *z-w⁺* interaction.

genetics: *z⁺* phenotype.

references: Gelbart, 1971, Ph.D. Thesis, Univ. of Wisconsin.

translocation	cytology
<i>T(1;4)z⁺ 12</i>	<i>3C6-7;101A</i>
<i>T(1;4)z⁺ 31</i>	<i>3C3-5;101B-C</i>
<i>T(1;4)z⁺ 34</i>	<i>3C2-3;102F</i>
<i>T(1;4)z⁺ 54</i>	<i>3C;3F;102D-E</i>

T(1;A)A7

cytology: *T(1;A)2D;?*. Autosomal break in heterochromatin.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal.

T(1;A)GF332

cytology: *T(1;A)3E5;?*. Autosomal break in heterochromatin.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal.

**T(1;A)pn-ec*: see *Tp(1;2)w⁺62g*

T(1;A)RF52

cytology: X break in 3E; autosomes complex.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal.

T(X.2)In^{x and 3}: see **T(1;2)w^{m53e}*

T(X^{c2};2)26: see *Tp(1;2)26*

T(Y;2) - T(Y;2;3)

Table I: *T(Y;2)a2-T(Y;2;3)rc77*

origin: γ ray induced.

discoverer: Lyttle.

synonym: *T(Y;2)A2-T(Y;2)RC77*.

genetics: Male fertile with normal X.

CHROMOSOMES - TRANSLOCATIONS

translocation	cytology	new order α	break in β	<i>Df(1)bb158/T(Y;2)</i> male fertility	translocation	cytology	new order α	break in β	<i>Df(1)bb158/T(Y;2)</i> male fertility
T(Y;2)a2 ^{γe}	<i>h11-h13;41A-B</i>		4	-	T(Y;2)re4	21C		1b	
T(Y;2)a15 ^γ	<i>B^SYy⁺;35A4-B1</i>		1b		T(Y;2)re14	40-41		1b	
T(Y;2)a18	<i>B^SYy⁺;36B</i>		1b			+ <i>In(2L)34D;37F</i>			
T(Y;2)a21	<i>B^SYy⁺;36B-C</i>		1b		T(Y;2)re21	40-41		1b	
T(Y;2)a35	<i>B^SYy⁺;35C-E</i>		1b		T(Y;2)re32	42F		1b	
T(Y;2)a38	<i>B^SYy⁺;35B-40</i>		1c		T(Y;2)re39	49A		1b	
T(Y;2)b6	<i>Ybb^L;35B-D</i>		7		T(Y;2)sl4	YS;40A-F		1a	
T(Y;2)b8	<i>Ybb^L;35C1-D1</i>					(prox. to <i>lt</i>)			
T(Y;2)b10 ^δ	<i>YL;36D2-3</i>		1		T(Y;2)t21	40-41		4	-
T(Y;2)b12	30F		1		T(Y;2)t22 ^δ	<i>YL;36B-C</i>		4	-
T(Y;2)b13 ^δ	YS;57A		1		T(Y;2)t41 ^δ	<i>41B-C + In(2L)25A;?</i>		3	
T(Y;2)b14	41B-C		1		T(Y;2;3)b11	<i>42A-B;86A-B;92E</i>	5	1	
T(Y;2)b15	24C-D		1		T(Y;2;3)b21	<i>YS;57B + 24E-F;63A</i>	6	1	
T(Y;2)b20	<i>YS;40-41;47B</i>	1	1		T(Y;2;3)b22	<i>40E-F;91F</i>		1	
T(Y;2)b23	<i>YS;42A;60A-B</i>	2	1		T(Y;2;3)cb15	<i>YL;21A1-3;80B3;87A-B</i>		5	-
T(Y;2)b24	<i>Y;36C + 40E--F;44B</i>	3	1		T(Y;2;3)cb19	<i>28C;40-41;87A6</i>		5	-
T(Y;2)c5 ^δ	38B1-2		4		T(Y;2;3)cb23	<i>24C;60A;88F</i>		5	
T(Y;2)c79	<i>B^SYy⁺;35A</i>		4b		T(Y;2;3)cb45	<i>Y;44C + 37D;79B</i>	7	5	-
T(Y;2)cb2	<i>YS;27C;36F</i>		5	+(semi)	T(Y;2;3)cb47	<i>Y;97D + 52D;</i>	8	5	-
T(Y;2)cb3	<i>YL;30F</i>		5	-		<i>55C-D + 42D;86C</i>			
T(Y;2)cb4	30B		5	-	T(Y;2;3)ra24	<i>38B-C;93B-D</i>	9	1b	-
T(Y;2)cb5 ^{δe}	<i>h25;41</i>		5	-	T(Y;2;3)ra34	<i>YL;25F;97F</i>	10	1b	+
T(Y;2)cb6	40-41		5	-	T(Y;2;3)rc17	<i>YS;43A-B;62E</i>		4a	+
T(Y;2)cb7	40-41		5	-	T(Y;2;3)rc77	<i>YS;30B;73F-74A + 26CD;</i>	11	4a	+
T(Y;2)cb8	<i>57C-D</i>		5	-		<i>53C-D + 55D-E;</i>			
T(Y;2)cb9	35F		5	-		<i>60E-F + 61A;80</i>			
T(Y;2)cb10	35E		5	-					
T(Y;2)cb11	<i>YL;55B</i>		5	-	α				
T(Y;2)cb13	<i>36A7-10</i>		5	-		<i>1 = Y^D or P^D 47B - 41 47B - 60F;</i>			
T(Y;2)cb14	distal to B on YL;		5	+		<i>Y^P or D^P 40 - 21A.</i>			
	distal to all bands					<i>2 = YL-YS^P 60A - 42A 60B - 60F;</i>			
	on 2L or 2R					<i>YS^D 42A - 21A.</i>			
T(Y;2)cb16 ^e	<i>h9-h10</i> ; distal to all		5	+		<i>3 = Y^D 36C - 40E 44B - 40F 44B - 60F;</i>			
	bands on 2L or 2R					<i>Y^P 36B - 21A.</i>			
T(Y;2)cb18	<i>36D-E</i>		5	-		<i>4 = Y^D 55E - 30E 25A - 21A;</i>			
T(Y;2)cb21	55F		5	-		<i>Y^P 30D - 25B 55E - 60F.</i>			
T(Y;2)cb22	34D		5	-		<i>5 = Y^D 42A - 21A;</i>			
*T(Y;2)cb23	<i>24C;60A;88F</i>		5	-		<i>Y^P 86B - 92E 42B - 60F;</i>			
T(Y;2)cb24	35A-B		5	-		<i>61A - 86A 92E - 100F.</i>			
T(Y;2)cb25	<i>YL (in B marker);</i>		5	-		<i>6 = YL-YS^P 57B - 60F;</i>			
	<i>41A (prox. to all</i>					<i>YS^D 57B - 24F 63A - 61A;</i>			
	<i>Hoechst S33258 bands)</i>					<i>21A - 24E 63A - 100F.</i>			
T(Y;2)cb31	40-41		5	-		<i>7 = Y^D 44C - 37D 79B - 61A;</i>			
T(Y;2)cb34	40-41		5	-		<i>Y^P 44C - 60F;</i>			
T(Y;2)cb36	34A		5	-		<i>21A - 37D 79B - 100F.</i>			
T(Y;2)cb37	33F		5	-		<i>8 = Y^D 97D - 86C 42D - 21A;</i>			
T(Y;2)cb38	25D		5	-		<i>Y^P 97D - 100F;</i>			
T(Y;2)cb42	42A		5	-		<i>61A - 86C 42D - 52D 55C - 52D 55C - 60F.</i>			
T(Y;2)cb44	40-41		5	-		<i>9 = Y^D 93B - 61A;</i>			
T(Y;2)cb48	<i>25A-B;30D-F;55E</i>	4	5	-		<i>Y^P 38B - 21A;</i>			
T(Y;2)da4	40-41		4			<i>60F - 38C 93D - 100F.</i>			
T(Y;2)da5	40-41		4			<i>10 = Y^D 25F - 60F;</i>			
T(Y;2)da6	40-41		4			<i>Y^P 97F - 100F;</i>			
T(Y;2)e2	<i>Ybb^L;35D1-4;35E1-2</i>		7			<i>21A - 25F 97F - 61A.</i>			
T(Y;2)e24	<i>Ybb^L;36B-C</i>					<i>11 = YL-YS^P 30B - 26C 53D - 55D 60F;</i>			
T(Y;2)l1	59C		3			<i>YS^D 73F - 61A 80 - 74A 30B - 51F 56E - 55E 60E - 56F 52A - 53C 26D - 21A;</i>			
T(Y;2)m1	<i>25D-E;56B-C</i>		4			<i>61A 80 - 100F.</i>			
T(Y;2)m3	40-41 +		2a		β				
	<i>In(2L)27A;38A</i>					<i>1 = SD-Roma; 1a = SD-Roma carrying In(2LR)S^L = In(2LR)21E-F;41;</i>			
T(Y;2)ra6	48B-C		1b	-		<i>1b = SD-Roma carrying In(2LR)Pu^L = In(2LR)40B;57B-C; 1c = SD-Roma</i>			
T(Y;2)ra8	<i>YL;40B-F</i>		1b	-		<i>carrying In(2LR)Pu + In(2LR)36B;57A5-10;</i>			
T(Y;2)ra18	36B		1b	-		<i>2 = SD-36; 2a = SD-79;</i>			
T(Y;2)ra23	55C-E		1b	-		<i>3 = SD-72;</i>			
T(Y;2)ra35	35C-E		1b	-		<i>4 = SD-NH₂; 4a = SD-NH₂ with In(2LR)DA3 = In(2LR)26D;53E +</i>			
T(Y;2)ra46	48E		1b	+		<i>In(2LR)39D-E;42A + In(2R)52A;56F + In(2R)55E;60;</i>			
T(Y;2)ra55	YS;21F		1b	+		<i>5 = cn bw (Madison, WI. Stock);</i>			
T(Y;2)rb9	25F		1b	+		<i>6 = T(Y;2)L141;</i>			
T(Y;2)rc5	<i>YL;38B1-2</i>		4a	-		<i>7 = Ybb^L.</i>			
T(Y;2)rc22	40-41		4a	+	γ				
T(Y;2)rc30	24F		4a	-		Associated with <i>lace</i> .			
T(Y;2)rc43	<i>YS;40-41</i>		4a	-	δ				
T(Y;2)rc45	28C5-9		4a	-		References: Lyttle, 1986, Genetics 114: 203-16 [T(Y;2)b10]; Lyttle, 1984,			
T(Y;2)rc52	29F		4a	+(semi)		Genetics 106: 423-34 [T(Y;2)t22]; Lyttle, 1977, Genetics 86: 413-45			
T(Y;2)rc58	38A		4a	-		[T(Y;2)t41].			
T(Y;2)rc71	36B-C		4a	-		ϵ			
T(Y;2)rd4	37C-D		1b	+(semi)		ζ			

Table II: T(Y;2)A24-T(Y;2)S14
origin: X ray induced using *B^SYy⁺*.

THE GENOME OF *DROSOPHILA MELANOGASTER*

genetics: Many are male sterile in combination with deficiencies for *Xh*.

translocation	cytology $\alpha\beta$	ref γ	<i>X/T(Y;2)</i> male fert. β	translocation	cytology $\alpha\beta$	ref γ	<i>X/T(Y;2)</i> male fert. β
				T(Y;2)D225	YS;40	5, 9	-
				T(Y;2)G10	YS;41	5, 9	-
				T(Y;2)G20	<i>Xhy</i> ⁺ ;31C-D	3, 5, 9	+
				T(Y;2)G53	h3;50A	3, 5, 9	-
				T(Y;2)G74	h7;34C3	1, 3, 5, 9, 11	(<i>kl3</i> ⁻ - <i>kl5</i> ⁻)
T(Y;2)A24	(YS); 46A	3, 5, 9	-	T(Y;2)G100	<i>Xhy</i> ⁺ ;56D	3, 5, 7, 9	+
*T(Y;2)A33	56F	5, 9	+	T(Y;2)G105	<i>Xhy</i> ⁺ ;25F	3, 5, 7, 9	+
T(Y;2)A62	<i>B</i> ^S <i>Xh</i> ;36C1-2 (Ashburner)	3, 5, 9	+	T(Y;2)G113	YS;40	5, 9	+
T(Y;2)A77 δ	h19;23F;25A	3, 5, 9	+	T(Y;2)G120	h24;24A	1, 3, 5, 9	+
T(Y;2)A80	h21;35A3-4 + <i>Df</i> (YL) <i>B</i> ^S <i>Xh</i> ;h7	1, 5, 9	-	T(Y;2)G146	h23;23B-C	1, 3, 5, 9	+
T(Y;2)A87	YL;40 (or 38A-B)	5, 8, 9	+	T(Y;2)H52	<i>Xhy</i> ⁺ ;28B4-C1 + <i>Df</i> (YL)h3;h16	3, 5, 9	(+)
*T(Y;2)A96	YL;59D + <i>In</i> (3R)65F;80-81	5, 9	+	T(Y;2)H54	YL;40 (or 39D-E)	5, 8, 9	-
*T(Y;2)A97	34A	5, 9	-	T(Y;2)H56	<i>Xhy</i> ⁺ ;22B	3, 5, 9	(-)
T(Y;2)A107	YL;40	5, 8, 9	+	T(Y;2)H69	h21;26A7-8	3, 5, 9	-
T(Y;2)A109	YL;83E	5, 9	+		(Knipple and MacIntyre, 1984)		
T(Y;2)A111	h14;28D + <i>Df</i> (YL)h5;h11	3, 5, 9	-	T(Y;2)H116	h3;24F5-6	1, 3, 5, 9	-
T(Y;2)A120 δ	h16;h21;57E	3, 5, 9	-	T(Y;2)H118	YL;40	5, 8, 9	+
T(Y;2)A128	YL;40	5, 9	-	T(Y;2)H121	h16;26B + <i>Df</i> (YL)h3;h13	3, 5, 9	(+)
*T(Y;2)A139	36D-E	5, 9	-	T(Y;2)H124	YS;40	5, 9	-
T(Y;2)A145	h25;29F	3, 5, 9	+	T(Y;2)H131	YL;40	5, 8, 9	-
T(Y;2)A146	<i>Xhy</i> ⁺ ;60F	3, 5, 9	+	T(Y;2)H136	h3;44C	3, 5, 9	+
T(Y;2)A159	YS;40	5, 9	+	T(Y;2)H137	h25;60D	3, 5, 9	+
T(Y;2)A160	YS;60B-C	5, 9	+	T(Y;2)H143	<i>B</i> ^S <i>Xh</i> ;h14;59F; deficient for <i>B</i> ^S <i>Xhj</i> -h13	3, 5, 9	(+)
T(Y;2)A161	<i>Xhy</i> ⁺ ;21B	3, 5, 9	+	T(Y;2)H144	h3;47F	3, 5, 9	-
T(Y;2)A162	h21;31E-F	3, 5, 9	-	T(Y;2)H149	h21;54F	3, 5, 9	-
T(Y;2)A165	YL;55E-F	5, 9	-	T(Y;2)H151	YS;25F3	5, 9	+
T(Y;2)A169	h13;55B	3, 5, 9	+	T(Y;2)H158	<i>Xhy</i> ⁺ ;58D	3, 5, 9	+
T(Y;2)A171	h3;27D	3, 5, 9	-	T(Y;2)H164	YS;25E + <i>In</i> (2)40-41;44F	5, 9	+
T(Y;2)A183	48D-E	2		T(Y;2)H165 δ	<i>Xhy</i> ⁺ ;21E;25D	3, 5, 9	+
T(Y;2)B3	YS;40-41	4	-	T(Y;2)H174	YS;37D12-E12 (Ashburner)	5, 9	-
T(Y;2)B4	h21;36C(<i>Df</i>)	3, 5, 9	-	T(Y;2)J30	YS;40	5, 9	+
T(Y;2)B5	YS;21C-D	5, 9	+	T(Y;2)J43	YS;41	5, 9	-
T(Y;2)B13	22E-F + <i>Df</i> (YL)B13	4	-	T(Y;2)J45	YL;40-41;49E	9	+
T(Y;2)B24	h23;43B	3, 5, 9	(+)	T(Y;2)J59	<i>Xhy</i> ⁺ ;43A + <i>Df</i> (YL)h3;h16	3, 5, 9	(+)
T(Y;2)B26	<i>B</i> ^S <i>Xh</i> ;43E-F	3, 5, 9	(-)	T(Y;2)J69	h21;22A	3, 5, 9	-
T(Y;2)B63	YS;41	5, 9	+	T(Y;2)J70	h11;26B + <i>In</i> (Y)h3;h21	3, 5, 9	-
T(Y;2)B66	<i>Xhy</i> ⁺ ;28C	3, 5, 9	(-)	T(Y;2)J118	h21;22D + <i>Df</i> (YL)h3;h10	1, 3, 5, 9	-
T(Y;2)B80 δ	<i>Xhy</i> ⁺ ;h21;60F	3, 5, 9	+	T(Y;2)J122	YS;23E1-2	1, 5, 9	-
T(Y;2)B92	h11;33B	3, 5, 9	(-)	T(Y;2)J131	YL;59D	5, 9	+
T(Y;2)B104	<i>B</i> ^S <i>Xh</i> ;28D	3, 5, 9	+	T(Y;2)J136	<i>B</i> ^S <i>Xh</i> ;26F5-7 + <i>Df</i> (YL)h3;h9	3, 5, 9	+
*T(Y;2)B106	h21;60B	3, 5, 9	-	T(Y;2)J146	h14;21B	3, 5, 9	+
T(Y;2)B107 δ e	<i>B</i> ^S <i>Xh</i> ;29C;47E	3, 5, 9	+	*T(Y;2)J157	YL;60?	5, 9	+
T(Y;2)B110	h3;38C	3, 5, 9	-	T(Y;2)J160	YS;33E-34A	5, 9	+
T(Y;2)B112	h25D;22C	3, 5, 9	+	T(Y;2)J163	h23;57F	3, 5, 9	-
T(Y;2)B135	h23;42A-B	3, 5, 9	+	T(Y;2)J165	h21;35C4-5	1, 3, 5, 9, 11	-
T(Y;2)B137	<i>Xhy</i> ⁺ ;25D6-7	1, 3, 5, 9	+	T(Y;2)J166	h25D;31D-E	3, 5, 9	+
T(Y;2)B177	YL;41	5, 9	+	T(Y;2)L11	h25D;60C	3, 5, 9	+
T(Y;2)B184	h25D;56C	3, 5, 9	(-)	T(Y;2)L23	h21;45F	3, 5, 9	-
T(Y;2)B185 δ	YS;60F + 34A;51A;53C-D	5, 9	+	T(Y;2)L26	h25D;24C	3, 9	+
T(Y;2)B190	YS;40 (or 39C)	5, 8, 9	+	T(Y;2)L52	h3;30F	3, 5, 9	(+)
T(Y;2)B196	YS;40	5, 9	+	T(Y;2)L62 δ	<i>Xhy</i> ⁺ ;56E-F	3, 5, 6, 7, 9	(-)
T(Y;2)B199	YL;40	5, 8, 9	-	T(Y;2)L67 δ	<i>Xhy</i> ⁺ ;25E;40	3, 5, 9	-
T(Y;2)B202	h20;59A-B	3, 5, 9	+	T(Y;2)L107	YL;57B	5, 9	-
T(Y;2)B209	YL;40 (or 39D-E)	5, 8, 9	-	T(Y;2)L110	h3;50C	3, 5, 9	+
T(Y;2)B210	h25D;36A2-6 (Ashburner)	3, 5, 9	+	T(Y;2)L116	<i>Xhy</i> ⁺ ;58A	3, 9	(-)
*T(Y;2)B212	33F-34A	5, 9	+	T(Y;2)L124	<i>Xhy</i> ⁺ ;21C	3, 5, 9	+
T(Y;2)B214	<i>Xhy</i> ⁺ ;36A2-6 (Ashburner)	3, 5, 9	+	T(Y;2)L126	YS;40	9	+
T(Y;2)B224	<i>Xhy</i> ⁺ ;33F-34A	3, 5, 9	+	T(Y;2)L134	YS;40	5, 9	+
T(Y;2)B228	YS;60F	5, 9	+	T(Y;2)L135 δ	YS;31F;40	5, 9	-
T(Y;2)B236	<i>B</i> ^S <i>Xh</i> ;h16;25D-E(<i>Df</i>)	3, 5, 9	-	T(Y;2)L137 δ	YS;43E;60A	5, 9	+
T(Y;2)B238	YS;41	5, 9	+	T(Y;2)L138	<i>Xhy</i> ⁺ ;39C	3, 5, 8, 9	+
T(Y;2)B242	h14;36C	3, 5, 9	-	T(Y;2)L139 η	<i>Xhy</i> ⁺ ;56E	3, 5, 6, 7, 9	+
T(Y;2)B251	YS;40 (or 39D-E)	5, 8, 9	-	T(Y;2)L140	YL;40	5, 9	-
T(Y;2)D6	<i>Xhy</i> ⁺ ;24C3;24C4-6;24E1-2;25D2-3; 25D6-7 (complex; includes inversion and deficiency in 2L).	1, 3, 5, 9, 10	+	T(Y;2)L141 η	<i>Xhy</i> ⁺ ;56F	3, 5, 6, 7, 9	+
T(Y;2)D19	h3;48E	3, 5, 9	-	T(Y;2)P8	h21;24D	3, 5, 9	-
T(Y;2)D20	YL;40	5, 9	-	T(Y;2)P42	YS;40	5, 9	+
T(Y;2)D70	YS;21D	5, 9	+	T(Y;2)P51	h21;25D6-7	1, 3, 5, 9	(+)
T(Y;2)D106	h21;26B	3, 5, 9	(+)	T(Y;2)P57 θ	h9-10;38B	3, 5, 9	-
T(Y;2)D110	<i>B</i> ^S <i>Xh</i> ;25D1-2	3, 5, 9	(-)	T(Y;2)P58	<i>Xhy</i> ⁺ ;35D5-8	1, 3, 5, 9, 11	+
T(Y;2)D211	<i>B</i> ^S <i>Xh</i> ;h3;26B3-5 (Knipple and MacIntyre, 1984)	3, 5, 9	+	T(Y;2)P59	<i>Xhy</i> ⁺ ;59B	3, 5, 9	+
T(Y;2)D212	YL;33F	5, 9	-				
T(Y;2)D217	YL;35D	5, 9	-				
T(Y;2)D219 δ	YL;35F-36A;36E-37A	5, 9	-				
T(Y;2)D222	YS;25F-26A1	5, 9	-				

translocation	cytology $\alpha\beta$	ref γ	X/T(Y;2) β male fert.
T(Y;2)R9	YS;40	5, 9	+
T(Y;2)R14¹	h3;53A-B	3, 5, 9	-
T(Y;2)R15	Xhy ⁺ ;35B9-C1	1, 3, 5, 9, 11	+
T(Y;2)R50	h1-2;28B	3, 5, 9	-
T(Y;2)R85	YS;40	5, 9	-
T(Y;2)R93	h21;57A	3, 5, 9	-
*T(Y;2)R103	YS;60E-F	5, 9	+
T(Y;2)R104	Xhy ⁺ ;58A	3, 5, 9	+
T(Y;2)R116	YL;40	5, 8, 9	+
T(Y;2)R123	YL;60F	5, 9	-
T(Y;2)R124	h3;55E	3, 5, 9	-
T(Y;2)R127	h21;24F-25A	3, 5, 9	-
T(Y;2)R131	27E-F	2	
T(Y;2)R136	h3;h7;22D; deficient for h4-h6	3, 5, 9	-
T(Y;2)R145^{\delta}	YS;23F;40	5, 9	-
T(Y;2)R146	YL;40	5, 9	+
T(Y;2)R147^{\delta}	B ^S Xhj;23;27E	3, 5, 9	+
T(Y;2)R155	h10-h11;43C	3, 5, 9	-
T(Y;2)R158	Xhy ⁺ ;32F + Df(YL)B ^S Xhj;h10	3, 5, 9	+
*T(Y;2)S14	YL;56F	5, 9	

α Y breakpoints in the terminology of Gatti and Pimpinelli, 1983, whenever available.

β Parentheses used for data from Lindsley *et al.*, 1972, that differ from data from Gatti and Pimpinelli, 1983.

γ 1 = Ashburner and Velissariou, 1980, DIS 55: 196; 2 = Baker, 1980, DIS 55: 197; 3 = Gatti and Pimpinelli, 1983, Chromosoma 88: 349-73; 4 = Hazelrigg, Fornili, and Kaufman, 1982, Chromosoma 87: 535-59; 5 = Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, and Gould-Somero, 1972, Genetics 71: 157-84; 6 = Procnier and Dunn, 1978, Cell 15: 1087-93; 7 = Procnier and Tartof, 1976, Nature 263: 255-57; 8 = Siegel, 1981, DIS 56: 123-24; 9 = The Seattle-La Jolla Drosophila Laboratories, 1971, DIS 47, Suppl.; 10 = Velissariou and Ashburner, 1980, Chromosoma 77: 13-27; 11 = Woodruff and Ashburner, 1972, Genetics 92: 117-32.

δ New order:

- T(Y;2)A77 = B^SXh - h19|23F - 25A|23F - 21; Xhy⁺ - h19|25A - 60.
- T(Y;2)A120 = B^SXh - h16|h21 - h16|57E - 60; Xhy⁺ - h21|57E - 21.
- T(Y;2)B80 = B^SXh - h21|Xhy⁺ - h21|60F; Xhy⁺|60F - 21.
- T(Y;2)B107 = B^SXh|29C - 47E|29C - 21; Xhy⁺ - B^SXh|47E - 60.
- T(Y;2)B185 = YL·YS^P|60F; YS^D|60F - 53D|51A - 53C|34A - 51A|34A - 21.
- T(Y;2)D219 = YL^D|36F - 60; YS·YL^P|36E - 36A|35F - 21.
- T(Y;2)H165 = B^SXh - Xhy⁺|21E - 21A; Xhy⁺|28D - 21E|28D - 60.
- T(Y;2)L67 = B^SXh - Xhy⁺|25E - 40|25E - 21; Xhy⁺|40 - 60.
- T(Y;2)L135 = YL^D·YS^F|31F - 40|31F - 21; YS|40 - 60.
- T(Y;2)L137 = YL·YS^P|60A - 60F; YS^D|43E - 60A|43E - 21.
- T(Y;2)R145 = YL·YS^P|23F - 40|23F - 21; YS^D|40 - 60.
- T(Y;2)R147 = B^SXh|27E - 60 YS·YL|23 - 27E;|23 - 21.

ϵ Synonym: T(Y;2)Kr.

δ Broken within the 5S RNA gene cluster (Procnier and Tartof, 1976; Procnier and Dunn, 1978).

η Broken just outside the 5S RNA gene cluster, proximal for T(Y;2)L139 and distal for T(Y;2)L141 (Procnier and Dunn, 1978). Completely male sterile in combination with Df(1)bb158 (Lyttle).

θ Suppresses Y loop associated with kl-3 (Bonaccorsi and Gatti).

¹ Cytology corrected by Davis and MacIntyre, 1988, Genetics 120: 755-66.

T(Y;2)1 – T(Y;2)16

origin: X ray induced.

references: Novitski and Ehrlich, 1970, DIS 45: 102.

translocation	cytology	induced in
T(Y;2)1	56E	Y
T(Y;2)7	57D	Y
T(Y;2)11-11N	34A	y ⁺ Y
T(Y;2)11-26A	36F	y ⁺ Y
T(Y;2)12-4A	34A	y ⁺ Y
T(Y;2)16	59F	Y

T(Y;2)2.31

cytology: T(Y;2)25C;38B.

new order:

Y^P|25C - 21;

Y^D|38B - 25C|38B - 60.

origin: X ray induced.

discoverer: Nüsslein-Volhard.

synonym: T(Y;2)odd^{2.31}.

references: Nüsslein-Volhard, Kluding, and Jürgens, 1985,

Cold Spring Harbor Symp. Quant. Biol. 50: 145-54.

genetics: Second-chromosome breakpoint between *slp* and *mid*; neither gene mutant.

T(Y;2)4.25

cytology: T(Y;2)24D.

origin: X ray induced.

discoverer: Nüsslein-Volhard.

synonym: T(Y;2)odd^{4.25}.

references: Nüsslein-Volhard, Kluding, and Jürgens, 1985,

Cold Spring Harbor Symp. Quant. Biol. 50: 145-54.

genetics: Second-chromosome breakpoint between *slp* and *mid*; neither gene mutant.

***T(Y;2)21E**

cytology: T(Y;2)21D4-E1.

discoverer: Schultz.

references: Lewis, 1945, Genetics 30: 137-66.

genetics: Not mutant for *S* or *ast*. Chromosome 2 broken between *ds* and *S*. Both Y^{P2D} and 2^PY^D recoverable in aneuploid progeny.

***T(Y;2)54a**

cytology: T(Y;2)YL;59C4-6.

discoverer: Mickey, 54a.

references: 1959, Texas Univ. Publ. 5914: 99-105.

1963, DIS 38: 30.

genetics: Variegated for *bw*. Male fertile. Male hyperploid for Y^{P2D} survives, is not variegated, and is sterile.

T(Y;2)60D-F: see *In(2LR)noc*⁷

T(Y;2)429 - T(Y;2)434

origin: X ray induced.

discoverer: Gelbart.

translocation	cytology
T(Y;2)429.39	25E-F
T(Y;2)434.78	25E1-4
T(Y;2)434.86	27C1-2
T(Y;2)434.89	35D-E
T(Y;2)434.105	32A1-4

T(Y;2)A

cytology: T(Y;2)40F-41A1; placed in 2R by Whittinghill (1937, DIS 8: 82-84).

origin: X ray induced.

discoverer: Dobzhansky, 1929.

references: 1930, Biol. Zentr. 50: 671-85.

1932, Z. Indukt. Abstamm. Vererbungsl. 60: 235-86.

genetics: Break between *pr* and *tk*. *rl*, *M(2)41A*, *stw*, *ap*, *msf*, *tk*, and *ltd* not affected.

***T(Y;2)A3: Translocation (Y;2) from Austin**

origin: X ray induced.

discoverer: Stone.

genetics: Variegated for *bw*.

***T(Y;2)B: Translocation (Y;2) Bar**

cytology: *T(Y;2)40F-41A1*; placed in 2R by Whittinghill (1937, DIS 8: 82). *Y* breakpoint at telomeric end of *YS*, as indicated by fluorescence analysis (Halfer, Tiepolo, Barigozzi, and Fraccaro, 1972, Chromosoma 39: 43-44).

origin: X ray induced.

discoverer: Dobzhansky, 1929.

references: 1930, Biol. Zentr. 50: 671-85.

1932, Z. Indukt. Abstamm. Vererbungsl. 60: 235-86.

genetics: Lethal in combination with *Df(2R)M41A10* and shows an extreme *rl* phenotype with *rl*. *stw*, *ap*, *msf*, *tk*, and *ltd* not affected.

T(Y;2)B^{SV5}: Translocation (Y;2) Bar of Stone Variegated

cytology: *T(Y;2)YL;41*.

discoverer: Craymer.

T(Y;2)B^{SV14}

cytology: *T(Y;2)YL;80*.

discoverer: Craymer.

T(Y;2)bw^{Drv18}

cytology: *In(2L)59B1-2*.

references: P. Simpson.

***T(Y;2)bw^{R27}: Translocation (Y;2) brown-Rearranged**

cytology: *T(Y;2)59D11-E1*.

origin: X-ray-induced derivative of *bw*.

discoverer: Slatis.

references: 1955, Genetics 40: 5-23.

genetics: Associated with *bw^{R27}*.

***T(Y;2)bw^{R57}**

cytology: *T(Y;2)59D5-6*.

origin: X ray induced.

discoverer: Slatis.

references: 1955, Genetics 40: 5-23.

genetics: Associated with *bw^{R57}*.

T(Y;2)C

cytology: *T(Y;2)40F-41A1*; placed in 2R by Whittinghill (1937, DIS 8: 82-84).

origin: X ray induced.

discoverer: Dobzhansky, 1929.

references: 1930, Biol. Zentr. 50: 671-85.

1932, Z. Indukt. Abstamm. Vererbungsl. 60: 235-86.

genetics: Does not affect *rl*, *M(2)41A*, *stw*, *ap*, *msf*, *tk*, or *ltd*.

T(Y;2)CA

translocation	cytology	origin	discov or ref ^α	induced with
<i>T(Y;2)CA13</i>	40-41	X ray	2	<i>In(2L)Sco^{rv5}</i>
<i>T(Y;2)CA19</i>	60D7-8	γ ray	3	<i>In(2LR)noc⁷</i>

translocation	cytology	origin	discov or ref ^α	induced with
<i>T(Y;2)CA27</i>	34B1-2	γ ray	4	
<i>T(Y;2)CA42</i>	40-41	γ ray	1	<i>Df(2L)TE35A-6</i>
<i>T(Y;2)CA50</i>	24F	γ ray	1	<i>Df(2L)TE35A-219</i>

^α 1 = Ashburner; 2 = Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95; 3 = Gubb, Roote, Harrington, McGill, Durrant, Shelton, and Ashburner, Chromosoma 92: 116-23; 4 = Roote.

T(Y;2)CyO: Translocation (Y;2) Curly Oster

cytology: Translocation between *Y* and *CyO*, *l(2)DTS513^{DTS}*. Cytology not given.

origin: X ray induced.

references: Wright and Green, 1974, DIS 51: 108-09.

genetics: *Cy* males carry temperature-sensitive lethal. Useful for constructing virginator stocks.

T(Y;2)D: see *T(Y;2;3)D*

T(Y;2)dp^{61d}: Translocation (Y;2) dumpy

origin: X ray induced.

discoverer: Thompson, 61d.

genetics: Mutant for *dp*.

***T(Y;2)dp^{w2}: Translocation (Y;2) dumpy-warped**

origin: X ray induced.

discoverer: Schalet, 55k.

references: Carlson and Schalet, 1956, DIS 30: 71.

Carlson, 1958, DIS 32: 117-18.

genetics: Variegated for *dp*.

T(Y;2)dpp¹⁶: Translocation (Y;2) decapentaplegic

cytology: *T(Y;2)22F1-2*.

origin: X ray induced.

discoverer: Spencer.

references: Gelbart.

genetics: *d-III dpp* mutant.

T(Y;2)DTD50

cytology: *T(Y;2)35D-E*.

discoverer: Gelbart.

genetics: Disrupts transvection at *dpp*.

T(Y;2)E

cytology: *T(Y;2)36D2-3* (Whittinghill, 1937, DIS 8: 82-84).

origin: X ray induced.

discoverer: Dobzhansky, 1929.

references: 1930, Biol. Zentr. 50: 671-85.

1932, Z. Indukt. Abstamm. Vererbungsl. 60: 235-86.

genetics: Male fertile; *Df(1)sc^{4L}sc^{8R}/T(Y;2)E* male is sterile.

T(Y;2)el⁴: Translocation (Y;2) elbow

cytology: *T(Y;2)35B1*. *Y* arm not determined.

origin: Induced by ethyl methanesulfonate.

discoverer: Harrington.

references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

genetics: Associated with *el⁴*. Thought to be mutant, but not deficient, for *el* since *el⁴/el* is like *el/el* but less extreme than *el/Df*.

***T(Y;2)Elp²: Translocation (Y;2) Ellipse**

cytology: T(Y;2)57C9-D5.
origin: X ray induced.
discoverer: Angel.
references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.
genetics: Ellipse-like eye phenotype; allelism with *Egfr^{El}* not tested before translocation lost.

T(Y;2)en^{SF32}: Translocation (Y;2) engrailed

cytology: T(Y;2)48A.
origin: X ray induced.
references: Kuner, Nakanishi, Ali, Drees, Gustavson, Theis, Kauvar, Kornberg, and O'Farrell, 1985, Cell 42: 309-16.
genetics: Mutant for *en*; a lethal allele.
molecular biology: Located at -2 kb on the molecular map of *en* ("- values to left, "+" values to right of *en* which is at position 0).

T(Y;2)F: see T(Y;2;3)F

T(Y;2)GT1

cytology: T(Y;2)35A4-B1.
origin: γ ray induced.
discoverer: Durrant.
genetics: Deficient for *noc-osp*.

T(Y;2)GT2

cytology: T(Y;2)35A1-2.
origin: γ ray induced.
discoverer: Durrant.
genetics: Deficient for *l(2)35Aa-pu*.

T(Y;2)IC68-5

cytology: T(Y;2)41F;53B.
new order:
Y^P|53B – 60F;
Y^D|41F – 53B|41F – 21.

origin: X ray induced.
references: Brosseau, 1969, DIS 44: 45.
genetics: Male fertile; shows dominant rough eye phenotype (allelism not determined).

T(Y;2)J^{rv99}: Translocation (Y;2) Jammed-revertant

cytology: {T(Y;2)31B-F}. Autosomal breakpoint assumed to be at or near *J*.
origin: X ray induced in *J* males.
synonym: *J-der-99*.
references: Salas and Lengyel, 1984, DIS 60: 243-44.
genetics: Revertant of *J*.

T(Y;2)J-D: Translocation (Y;2) J of Dobzhansky

cytology: T(Y;2)40F-41A1;57F1-2 (Whittinghill, 1937, DIS 8: 82-84).
new order:
Y^D or P|40F – 21;
Y^P or D|57F1 – 41A1|57F2 – 60.
origin: X ray induced.
discoverer: Dobzhansky, 1929.
references: 1930, Biol. Zentr. 50: 671-85. 1932, Z. Indukt. Abstamm. Vererbungsl. 60: 235-86.
genetics: Does not affect *rl*, *M(2)41A*, *stw*, *ap*, *msf*, *tk*, or *ltd*.

T(Y;2)JL11

cytology: T(Y;2)YS;56F + *In(2LR)21A;57F*.
new order:
YL.YS|56F – 57F|21A;
60F – 57F|21A – 56F|YSy⁺.
origin: γ-ray-induced 2° translocation from T(Y;2)L141 = T(Y;2)56F.
references: Mackay and O'Donnell, 1983, Genetics 105: 35-53. Lyttle, 1984, Genetics 106: 423-34. Mackay, Reynolds, and O'Donnell, 1985, Genetics 111: 885-904.
genetics: Partially male fertile. *Pu⁺* translocated to YS. Normal guanosine triphosphate cyclohydrolase (GTP CH) activity (Mackay *et al.*, 1985).

T(Y;2)JL12

cytology: T(Y;2)YS;56F + *In(2LR)21A;59D*.
new order:
YL.YS|56F – 59D|21A;
60F – 59D|21A – 56F|YS.
origin: γ-ray-induced 2° rearrangement on T(Y;2)L141 = T(Y;2)56F.
references: Lyttle, 1984, Genetics 106: 423-34.

T(Y;2)Kr: see T(Y;2)B80

T(Y;2)Kr^{AK1}

cytology: T(Y;2)60F3-5.
origin: X ray induced in *If* chromosome.
references: Preiss, Rosenberg, Kienlin, Seifert, and Jäckle, 1985, Nature 313: 27-32.
genetics: Mutant for *Kr*. Incomplete revertant for *If* (phenotype dependent on chromosomal background).
molecular biology: Entire cloned *Kr* region (from -5 to +34) translocated onto *Y*; *EcoRI* site at origin of chromosomal walk of Preiss *et al.*, 1985.

T(Y;2)MK2

cytology: T(Y;2)25E1-3.
discoverer: Kotarski.

T(Y;2)odd^{2.31}: see *Tp(2;Y)odd^{2.31}*

T(Y;2)odd^{4.13}: see *Tp(2;Y)odd^{4.13}*

T(Y;2)odd^{4.25}: see *Tp(2;Y)odd^{4.25}*

T(Y;2)prd^{5.12}: see *Tp(2;Y)prd^{5.12}*

T(Y;2)SD: Translocation (Y;2) Segregation Distorter

origin: X ray induced in *SD-72* males carrying *B^SYy⁺* in translocations T(Y;2)SD-NE1, -NE2, and -NE3 and a normal *Y* in the other translocations.
genetics: Associated with *SD*. *K* values tested.

translocation	cytology	synonym	ref ^α
T(Y;2)SD-L	58	T(Y;2)SD L ²	2
T(Y;2)SD-L1	26B9-10	T(Y;2)SD72, T-1	1
T(Y;2)SD-L22	36B-C	T(Y;2)SD72, T-22	1
T(Y;2)SD-L23	39B-C	T(Y;2)SD72, T-23	1
T(Y;2)SD-L24	41	T(Y;2)SD72, T-24	1
T(Y;2)SD-L25	41 [within <i>In(2LR)SD72</i> = <i>In(2LR)39-40;42A</i>]	T(Y;2)SD72, T-25	1
T(Y;2)SD-L31	40-41	T(Y;2)SD72, T-31	1
T(Y;2)SD-L32	38A	T(Y;2)SD72, T-32	1
T(Y;2)SD-L41	41B-C + <i>In(2LR)25A;41B-C</i>	T(Y;2)SD72, T-41	1
T(Y;2)SD-NE1	31D	T(Y;2)SD, EM106	1, 3

translocation	cytology	synonym	ref ^α
<i>T(Y;2)SD-NE2</i>	37B	<i>T(Y;2)SD, j-4</i>	1, 3
<i>T(Y;2)SD-NE3</i> ^β	42A	<i>T(Y;2)SD, EM135</i>	1, 3
<i>T(Y;2)SD-NE4</i>	44D	<i>T(Y;2)SD, CB-1c</i>	1, 3
<i>T(Y;2)SD-YT1</i>	42	<i>T(Y;2)SD72-92</i>	4
<i>T(Y;2)SD-YT2</i>	31	<i>T(Y;2)SD72-132</i>	4
<i>T(Y;2)SD-YT3</i>	37	<i>T(Y;2)SD72-343</i>	4
<i>T(Y;2)SD-YT4</i>	57	<i>T(Y;2)SD72-398</i>	4

^α 1 = Lyttle, 1977, Genetics 86: 413-45; 2 = Lyttle, 1979, Genetics 91: 339-57; 3 = Novitski and Ehrlich, 1970, DIS 45: 102; 4 = Yamazaki and Thompson, 1973, Jpn. J. Genet. 48: 217-29.

^β Suppresses *SD* strongly.

T(Y;2)TE34Ca: Translocation (Y;2) Transposing Element

cytology: *T(Y;2)35D5-7*. *Y* arm not determined.
origin: γ ray induced as partial revertant of *TE34Ca* of Ising.
discoverer: Gubb.
synonym: *T(Y;2)TE60*^{V1}.
references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.
genetics: Variegates for the w^+ of the transposing element. Male fertile.
other information: Cytology not consistent with the insertion site of the transposing element.

T(Y;2)TE35A

origin: γ ray induced in *TE35A*.
synonym: *T(Y;2)TE146Z*.

translocation	cytology	<i>T(Y;2)TE146Z</i> discov ^α
<i>T(Y;2)TE35A-18</i>	35B1-2	1
<i>T(Y;2)TE35A-51</i>	35B1-2	3
<i>T(Y;2)TE35A-102</i>	35B	2
<i>T(Y;2)TE35A-201</i>	35B	2
<i>T(Y;2)TE35A-204</i>	35B	2
<i>T(Y;2)TE35A-206</i>	35B [+ small Dp?]	2
<i>T(Y;2)TE35A-211</i>	35B + <i>ln(3R)CA52</i>	2
<i>T(Y;2)TE35A-213</i>	35B	2

^α 1 = Roote; 2 = Samkange; 3 = Wilkins.

T(Y;2)TW20: Translocation (Y;2) Ted Wright

cytology: *T(Y;2)38A6-C1*.
origin: X ray induced.
references: Wright, Hodgetts, and Serald, 1976, Genetics 84: 267-85.
genetics: Deficient for *pr*; carries *Tft* and *l(2)74i*.

T(Y;2)TW124

cytology: *T(Y;2)45A2-E1*.
origin: X ray induced in *cn bw*.
references: Wright, Hodgetts, and Serald, 1976, Genetics 84: 267-85.
genetics: Variegated for *pr*.

T(Y;2)w⁺Y

Described as w^+Y (see *Y DERIVATIVES* in SPECIAL CHROMOSOMES section).

T(Y;2;3)A147

cytology: *T(Y;3)93F + T(2;3)60B;61A*.
origin: X ray induced.
references: Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972,

Genetics 71: 157-84.

T(Y;2;3)b: see *T(Y;2) - T(Y;2;3)*

T(Y;2;3)b10-1

cytology: *T(Y;2)YL;36D2-3 + T(2;3)36C10-D1;62A-B*.

new order:

YL^D|36D3 – 60;
 YS·YL^F|36D2 – 36D1|62A – 61;
 21 – 36C10|62B – 100.

origin: X-ray-induced derivative of *T(Y;2)b10*; male fertile in combination with *Dff(1)bb158*.

references: Lyttle, 1984, Genetics 106: 423-34.

genetics: 36C10-D1 breakpoint apparently inactivates *rdo*, since *T(Y;2;3)b10-1/rdo* flies exhibit a strong reduced-ocelli phenotype. Males carrying the *Y*-chromosome element capped by 36D1-2 plus region 61 are viable and fertile; therefore *Y*-chromosome breakpoint distal to *kl-5*. Females carrying remaining two elements of the translocation are marked with *B* and are sterile, presumably owing to heterozygous deficiency for region 61. This genotype, which is deficient also for 36D1-2 is deficient for *Arr1*, by *in situ* hybridization, and *I(2)36Db* but not *ninaD*; whether *kel* is included not determined (Hardy).

T(Y;2;3)Coi^{rv1}: Translocation (Y;2;3) Coiled-revertant

cytology: *T(Y;2)22B8 + T(2;3)32F;89E-F + Tp(3;3)64C;71A;75C*.

new order:

Y|21 – 22B8;
 60 – 32F|89F – 100;
 61 – 64C|71A – 75C|64C – 71A|75C – 89E|32F – 22B8|Y.

origin: X ray induced.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.

genetics: Revertant of *Coi*.

****T(Y;2;3)D***

cytology: *T(Y;2;3)29F-30A1 + T(2;3)34C;78F + Df(2R)41A;41C + Df(3L)61E2-F1;62A4-6*. May also carry small inverted segment in region 41 (Whittinghill, 1937, DIS 8: 82-84).

new order:

Y^D|30A1 – 34C|78F – 100;
 Y^F|29F – 21;
 60 – 41C|41A – 34C|78F – 62A6|61E2 – 61A.

origin: X ray induced.

discoverer: Dobzhansky, 1929.

synonym: *T(Y;2)D*.

references: 1930, Biol. Zentr. 50: 671-85.

1932, Z. Indukt. Abstamm. Vererbungsl. 60: 235-86.

genetics: Deficient for *M(2)41A* and *stw* (but not *rl*, *ap*, *msf*, *tk*, or *ltd*) in chromosome 2 and for *ru*, *aa*, and *ve* [but not *su(ve)* or *R*] in chromosome 3. The *Y^{F2D}* element survives in hyperploids.

T(Y;2;3)F

origin: X ray induced.

discoverer: Dobzhansky, 1929.

synonym: *T(Y;2)F*.

references: 1930, Biol. Zentr. 50: 671-85.

1932, Z. Indukt. Abstamm. Vererbungsl. 60: 235-86.

genetics: Break in 2R to right of *sp*.

T(Y;2;3)G20

cytology: *T(Y;2;3)YS;31C-D;81F;82F.*

new order:

YL·YS|81F – 82F|31C – 21;
YS|31D – 60;
61 – 81F|82F – 100.

origin: X ray induced.

references: The Seattle-La Jolla Drosophila Laboratories, 1971, DIS 47, Suppl.

Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84.

genetics: Male fertile over normal X.

T(Y;2;3)H: Translocation (Y;2;3) Hilliker

origin: γ ray induced.

references: Hilliker and Trusis-Coulte, 1987, Genetics 117: 233-44.

genetics: Viability and fertility of homozygotes not determined.

translocation ^α	cytology	new order
T(Y;2;3)H69	43B;98C	Y ^D 43B-21; Y ^P 98C-100; 60-43B 98C-61.
T(Y;2;3)H121	40-41;86B2-C1	not known
T(Y;2;3)H159	37B;43D-E;64E;98C;het	YL·YS ^P ?; YS ^D 98C-64E 37B-21; het 43D-37B 98C-100; het 43D-60; 61-64E het.

^α Synonym: *T(Y;2;3)69 - T(Y;2;3)159.*

T(Y;2;3)iab7^{MX1}: Translocation (Y;2;3) infraabdominal 7

cytology: *T(Y;2;3)* (cytological breakpoints not given).

origin: X ray induced.

discoverer: Casanova.

synonym: *T(Y;2;3)Abd-B^{MX1}; T(Y;2;3)iab6^{MX1}.*

references: Sánchez-Herrero, Vernós, Marco, and Morata, 1985, Nature 313: 108-13.

Karch, Weiffenbach, Peifer, Bender, Duncan, Celnicker, Crosby, and Lewis, 1985, Cell 43: 81-96.

genetics: Viable over *Df(3R)P9*, showing strong transformation of the sixth and seventh abdominal segments to the fifth.

molecular biology: DNA breakpoint at +126 to +129 kb, 0 being the 89E1-2 breakpoint of *In(3R)Cbx^{rv1}*.

***T(Y;2;3)I**

cytology: *T(Y;2)47A2-3 + T(Y;3)91E2-4 + In(3LR)69C2-3;84E2-3 + In(3LR)74A-B1;99C* (Whittinghill, 1938, DIS 8: 82-84).

new order:

Y^D|47A2 – 21;
Y^D|91E4 – 99C|74B1 – 84E2|69C2 – 61;
60 – 47A3|Y^P|91E2 – 84E3|69C3 – 74A|99C – 100.

origin: X ray induced.

discoverer: Dobzhansky, 1929.

references: 1930, Biol. Zentr. 50: 671-85.

1932, Z. Indukt. Abstamm. Vererbungslehre. 69: 235-86.

T(Y;2;3)SD-L12

cytology: *T(Y;2)41A-B;62F5-6.*

origin: X ray induced in *SD-72* males.

synonym: *T(Y;2;3)SD, T-12.*

references: Lyttle, 1977, Genetics 86: 413-45.

genetics: *K* value 1.00.

T(Y;2;3)Ubx^{2P}: Translocation (Y;2;3) Ultrabithorax

cytology: *T(Y;2;3)39;89E1-2;91F.*

origin: X ray induced.

synonym: *Ubx^{19849.2P}.*

references: E.B. Lewis.

genetics: Associated with *Ubx*.

T(Y;2;4)A96

cytology: *T(Y;2;4)YL;59D;102.*

new order:

YL|101;
YS·YL|59D – 60;
21 – 59D|102.

references: Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics 71: 157-84.

genetics: Male fertile with normal X.

T(Y;2;4)ci12: Translocation (Y;2;4) cubitus interruptus

cytology: *T(2;4)46;102F + Tp(2;Y)54B;57B.* Complex.

new order:

Y|(54B – 57B)|Y;
21 – 46|102F;
101 – 102F|46 – 54B|57B – 60.

origin: X ray induced.

synonym: *R¹²(ci).*

references: Stern and Kodani, 1955, Genetics 40: 343-73.

genetics: *T(Y;2;4)ci12/ci* males show less extreme L4 interruptions than *ci/ci* males. Usually no translocation females produced because of *Y* linkage in *T(Y;2;4)ci12*.

T(Y;2;4)H161

cytology: *T(Y;2;4)YS;35A-B;102.*

new order:

YL·YS|102;
YS|35B – 60;
21 – 35A|101.

references: Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics: 71: 157-84.

T(Y;2;4)J96

cytology: *T(Y;2;4)h16;25A2-3;101-102 + Df(YL)B^SXh;h8* (*Y* breaks from Gatti and Pimpinelli, 1983, Chromosoma 88: 349-73).

new order:

B^SXh|h8 – h16|25A3 – 60;
Xhy⁺ – h16|102;
21 – 25A2|102 – 101.

references: The Seattle-La Jolla Drosophila Laboratories, 1971, DIS 47: Suppl.

Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, Genetics: 71: 157-84.

Ashburner and Velissariou, 1980, DIS 55: 196.

Velissariou and Ashburner, 1980, Chromosoma 77: 13-27.

genetics: Male sterile. Shows variegation for *dp^{ov}* (Kotar-

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ski, Pickert, and MacIntyre, 1983, Genetics 105: 371-86.

T(Y;2;4)SD-L6

cytology: T(Y;2;4)40-41;101-102(?). (Presence of 4 based on its association with SD chromosome centromere).

origin: X ray induced in SD-72 males.

synonym: T(Y;2;4)SD, T-6.

references: Lyttle, 1977, Genetics 86: 413-45.

genetics: K value 1.00.

T(Y;3)

origin: X ray induced using B^SYy⁺.

genetics: Male sterile in combination with Df(1)bb158.

translocation	cytology $\alpha\beta$	ref γ	X/T(Y;3) fertility β
T(Y;3)A13	YL;68B	6, 7	+
T(Y;3)A14	h3;63B	3, 6, 7	-
T(Y;3)A23	h14;70B-C	3, 6, 7	-
T(Y;3)A31	YS;70A-C	6, 7	-
T(Y;3)A34 δ	YS;76A-B;99A-B	4	+
T(Y;3)A60	Xhy ⁺ ;71C1-2 + In(3R)88A3-7;89A3-13	3, 6, 7	+
T(Y;3)A63 δ	YL;68D;80-81	6, 7	-
T(Y;3)A78	YL;87B5	1, 3, 6, 7	-
T(Y;3)A82	Xhy ⁺ ;98F	3, 6, 7	+
T(Y;3)A83	YL;61C	6, 7	+
T(Y;3)A88	80-81	6, 7	+
T(Y;3)A89	B ^S Xhj;91B	3, 6, 7	(-)
T(Y;3)A95	80-81	6, 7	+
T(Y;3)A109 ϵ	YL;83E	6, 7	+
T(Y;3)A112	h11;76E	3, 6, 7	+
T(Y;3)A113	Xhy ⁺ ;100A	3, 6, 7	+
T(Y;3)A114	Xhy ⁺ ;61A	3, 6, 7	+
T(Y;3)A117	h24;96A	3, 6, 7	+
T(Y;3)A121	h3;97D-E	3, 6, 7	-
T(Y;3)A142	87D-88A	2	+
T(Y;3)A148	80-81	6, 7	+
T(Y;3)A150	YS;70D-E	6, 7	+
T(Y;3)A154	B ^S Xhj;82C	3, 6, 7	+
T(Y;3)A155	YS;92A	6, 7	+
T(Y;3)A158	YL;63A	6, 7	-
T(Y;3)A173	h21;88B	3, 6, 7	-
T(Y;3)A176	YL;76C	6, 7	-
T(Y;3)A200 δ	YL;64F;99D-F	6, 7	-
T(Y;3)B12	80-81	6, 7	+
T(Y;3)B20 δ	65F;80-81	6, 7	-
T(Y;3)B21	YS;62C3-4	6, 7	-
T(Y;3)B27	B ^S Xh;94E	3, 6, 7	+
T(Y;3)B49	YL;67D	6, 7	-
T(Y;3)B64 δ	61A;72;85A	2	-
T(Y;3)B68 δ	h3;80-81;87A	3, 6, 7	-
T(Y;3)B71	YS;61B-C	6, 7	+
T(Y;3)B77	YL;61F	6, 7	-
T(Y;3)B81	YL;99C-D + In(3)80-81;87-88	6, 7	+
T(Y;3)B82	80-81	6, 7	-
T(Y;3)B93	h21;93F-94A	3, 6, 7	-
T(Y;3)B96	YL;73A-B	6, 7	+
T(Y;3)B99	YL;71F3-5	6, 7, 8	-
T(Y;3)B108	Xhy ⁺ ;77E-F	3, 6, 7	+
T(Y;3)B115	h15;76D	3, 6, 7	+
T(Y;3)B116	B ^S Xhj;90E	3, 6, 7	+
T(Y;3)B130 δ	h1-2;61D-E;3R	3, 6, 7	-
T(Y;3)B132 δ	h23;76A	3, 6, 7	-
T(Y;3)B141 δ	YS;64E;68A	6, 7	-
T(Y;3)B152	h3;98F	3, 6, 7	-
T(Y;3)B154 δ	64C-D;80-81	6, 7	-
T(Y;3)B155	YL;82C	6, 7	-
T(Y;3)B158	B ^S Xh;97B	3, 6, 7	+
T(Y;3)B165	80-81	6, 7	-
T(Y;3)B170	70D	6, 7	+
T(Y;3)B172	YL;95A + In(3R)93B-C;99A	6, 7	+
T(Y;3)B186	Xhy ⁺ ;65E	3, 6, 7	(-)
T(Y;3)B189	B ^S Xhj;92D	3, 6, 7	+

translocation	cytology $\alpha\beta$	ref γ	X/T(Y;3) fertility β
T(Y;3)B197	h3;96B1-10	3, 6, 7	-
T(Y;3)B207	h25D;72D11-E1	3, 6, 7, 8	+
T(Y;3)B217	h3;96A1-5	3, 6, 7	-
T(Y;3)B222	80-81	6, 7	-
T(Y;3)B223	h13;72A-B	3, 6, 7	+
T(Y;3)B225	YL;73D	6, 7	+
T(Y;3)B226	B ^S Xh;98B	3, 6, 7	+
T(Y;3)B229	h13;64E + In(3R)94E-F;97C-D	3, 6, 7	+
T(Y;3)B234	B ^S Xhj;65B	3, 6, 7	+
T(Y;3)B240	Xhy ⁺ ;94B	3, 6, 7	+
T(Y;3)D8	B ^S Xhj;62A10-B1	6, 7	+
T(Y;3)D85 ϵ	YS;84D7-9	6, 7	-
T(Y;3)D100	h3;94A	3, 6, 7	-
T(Y;3)D107	h7;82E	3, 6, 7	(kl3 ⁻ - kl5 ⁻)
T(Y;3)D210	71F	6, 7	-
T(Y;3)D221	YS;96A	6, 7	-
T(Y;3)D224	h21;64C-D	3, 6, 7	-
T(Y;3)D226	B ^S Xh;87E5-F2	1, 3, 6, 7	+
T(Y;3)D227		5	+
T(Y;3)D228	YL;74A3	1, 6, 7, 9	+
T(Y;3)G7	YL;75C	6, 7	-
T(Y;3)G8	YS;85F9-13	1, 6, 7	-
T(Y;3)G11	h3;62E	3, 6, 7	-
T(Y;3)G24 δ	Xhy ⁺ ;80-81;85D-E	3, 7	+
T(Y;3)G42	h3;85E3	3, 6, 7	-
T(Y;3)G43 ζ	Xhy ⁺ ;63D2-E1	3, 6, 7	+
T(Y;3)G45	h23;61E	3, 6, 7	-
T(Y;3)G48	YS;88C	6, 7	+
T(Y;3)G64	80-81	6, 7	-
T(Y;3)G71	h21;67C	3, 6, 7	-
T(Y;3)G72	80-81F	6, 7	-
T(Y;3)G73	YL;96A1-5	6, 7	+
T(Y;3)G75	YS;97B	6, 7	-
T(Y;3)G101	80-81F	6, 7	(-)
T(Y;3)G110	YL;91E-F	6, 7	-
T(Y;3)G114	80-81	6, 7	(+)
T(Y;3)G116	h3;99F (complex, one break in 64C)	3, 6, 7	-
T(Y;3)G122	h3;67C	3, 6, 7	-
T(Y;3)G130 δ	h23;61E;66B-C	3, 6, 7	+
T(Y;3)G144	h3;83C	3, 6, 7	(+)
T(Y;3)H61	80-81	6, 7	-
T(Y;3)H133	80-81	6, 7	-
T(Y;3)H135	h21;96C5-9	3, 6, 7	-
T(Y;3)H138 δ	YS;66B;66F	6, 7	+
T(Y;3)H140 δ	66F;80-81	6, 7	-
T(Y;3)H147	h3;77B	3, 6, 7	-
T(Y;3)H153	80-81	6, 7	-
T(Y;3)H156	YS;70C	6, 7	(+)
T(Y;3)H159	80-81	6, 7	+
T(Y;3)H163	YL;98B	6, 7	(+)
T(Y;3)H167	66F-67A	2	-
T(Y;3)H172	B ^S Xh;87F	3, 6, 7	+
T(Y;3)H173	Xhy ⁺ ;95E6-8	3, 6, 7	+
T(Y;3)H175 δ	64E-F;81F	2	-
T(Y;3)J1 δ	75C;81F	3, 6	-
T(Y;3)J17	Xhy ⁺ ;82A	3, 6, 7	+
T(Y;3)J23	h3;90D	3, 6, 7	-
T(Y;3)J44	78C-D	3, 6	-
T(Y;3)J82 η	96A;97F	2	+
T(Y;3)J94	h1-2;66B	3, 6, 7	-
T(Y;3)J95	h21;78C	3, 6, 7	-
T(Y;3)J100	h21;75C	3, 6, 7	-
T(Y;3)J112	YS;71B3-8	6, 7, 8	+
T(Y;3)J116	B ^S Xh;97E-F	3, 6, 7	+
T(Y;3)J121 δ	YL;61-65;95-96	6, 7	+
T(Y;3)J128	h21;65F	3, 6, 7	-
T(Y;3)J132	Xhy ⁺ ;70C	3, 6, 7	+
T(Y;3)J139 θ	YL;80	6, 7	+
T(Y;3)J141	Xhy ⁺ ;87A9-B3	1, 3, 6, 7	+
T(Y;3)J142	Xhy ⁺ ;63E	3, 6, 7	+
T(Y;3)J145	80-81	6, 7	+
T(Y;3)J147	YS;76F-77A	6, 7	+

translocation	cytology ^{αβ}	ref ^γ	X/T(Y;3) fertility ^β
T(Y;3)J150	YS;67D	6, 7	-
T(Y;3)J151	YS;98E-F	6, 7	-
T(Y;3)J154	YS;63A	6, 7	-
T(Y;3)J162^δ	h23;79E5-6;99E	3, 6, 7	+
T(Y;3)L14	h3;76B5-10	3, 6, 7, 9	-
T(Y;3)L17	h16;86A6 + In(3L)68B;73F	1, 3, 6, 7	+
T(Y;3)L18	Xhy ⁺ ;64C13-15	3, 6, 7	-
T(Y;3)L20^δ	YS;64C-D;66E;79	2	-
*T(Y;3)L60	YS;67D-E	6, 7	-
T(Y;3)L61	80-81	6, 7	(+)
T(Y;3)L65	80-81	6, 7	-
T(Y;3)L68	80-81	6, 7	-
T(Y;3)L111	h25D;92D-E	3, 6, 7	+
T(Y;3)L113	h25D;71B-C	3, 6, 7	+
T(Y;3)L125	Xhy ⁺ ;94A	3, 6, 7	+
T(Y;3)L129	h25D;100B-C	3, 6, 7	+
T(Y;3)L130^δ	70D-E;87A	6, 7	-
T(Y;3)L131^l	Xhy ⁺ ;75D1	1, 3, 6, 7, 9	+
T(Y;3)L132	YS;83C-D	6, 7	+
T(Y;3)L136	B ^S Xh;83E-F	3, 6, 7	+
T(Y;3)L142	h3;89C	3, 6, 7	-
T(Y;3)P31	80-81	6, 7	-
T(Y;3)P40		5	+
T(Y;3)P50	YL;65D	6	+
T(Y;3)P60	YL;99F	6, 7	-
*T(Y;3)R5	99A-B	7	-
T(Y;3)R6	h3;92B	3, 6, 7	-
T(Y;3)R7	B ^S Xh;69F	3, 6, 7	+
T(Y;3)R13	Xhy ⁺ ;94E	6, 7	+
T(Y;3)R24^δ	80-81 + Tp(3;3)91B-C;94C-E;98E	6, 7	-
T(Y;3)R36	YL;86B	6, 7	+
T(Y;3)R59	78F-79A	6, 7	-
T(Y;3)R71	h1-2;97B	3, 6, 7	-
T(Y;3)R78	h23;98E	3, 6, 7	-
T(Y;3)R83	70B-C	5	+
T(Y;3)R86	h25D;66A	3, 6, 7	-
T(Y;3)R87	YS;97A	6, 7	-
T(Y;3)R91	B ^S Xh;70C-D	3, 6, 7	+
T(Y;3)R92	Xhy ⁺ ;76C-D	5	+
T(Y;3)R98	65D-E	3, 6, 7	-
T(Y;3)R100	80-81	6, 7	-
T(Y;3)R106	B ^S Xh;65D	3, 6, 7	+
T(Y;3)R108	61B-C	5	+
T(Y;3)R114	80-81	5	+
T(Y;3)R117	h3;64E	3, 6, 7	-
T(Y;3)R119	h23;66A	3, 6, 7	-
T(Y;3)R122	Xhy ⁺ ;69F	3, 6, 7	+
T(Y;3)R128	97F	5	+
T(Y;3)R130	h21;100A	3, 6, 7	-
T(Y;3)R132	61F	6, 7	+
T(Y;3)R133	B ^S Xh;99E	3, 6, 7	+
T(Y;3)R135	h25D;91B	3, 6	+
T(Y;3)R142	64D	5	-
T(Y;3)R150^δ	YL;64D;71B	6, 7	-
T(Y;3)R153	YL;78A	6, 7	-
T(Y;3)S50	B ^S Xhj;61A	3, 6, 7	+

^α Y breakpoints given in the terminology of Gatti and Pimpinelli, 1983, whenever available.

^β Parentheses used for data from Lindsley *et al.*, 1972, that differ from data from The Seattle-La Jolla Drosophila Laboratories, 1971, or Gatti and Pimpinelli, 1983.

^γ 1 = Ashburner and Velissariou, 1980, DIS 55: 196; 2 = Baker, 1980, DIS 55: 197; 3 = Gatti and Pimpinelli, 1983, Chromosoma 88: 349-73; 4 = Hazelrigg, Fomili, and Kaufman, 1982, Chromosoma 83: 535-59; 5 = La Jolla Lab data (Rokop); 6 = Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, and Gould-Somero, 1972, Genetics 71: 157-84; 7 = The Seattle-La Jolla Drosophila Laboratories, 1971, DIS 47, Suppl.; 8 = Velissariou and Ashburner, 1981, Chromosoma 84: 173-85; 9 = Walker and Ashburner, 1981, Cell 26: 269-77.

^δ New order:
 $T(Y;3)A34 = YL \cdot YS^P | 99B - 100;$
 $YS^D | 76B - 99A | 76A - 61.$

$T(Y;3)A63 = YL^D | 80 - 100;$
 $YS \cdot YL^P | 68D - 80 | 68C - 61.$
 $T(Y;3)A200 = YL^D | 99E - 64F | 99D - 100;$
 $YS \cdot YL^P | 64 - 61.$
 $T(Y;3)B20 = Y | 65F - 80 | 65F - 61;$
 $Y | 81 - 100.$
 $T(Y;3)B64 = Y^D | 85A - 72 | 61A;$
 $Y^P | 72 - 61A | 85A - 100.$
 $T(Y;3)B68 = B^S Xh - h3 | 81 - 87A | 80 - 61;$
 $Xhy^+ - h3 | 87A - 100.$
 $T(Y;3)B130 = B^S Xh - h1 | 3R^P - 61E | 3R^D - 100;$
 $Xhy^+ - h2 | 61D - 61A.$
 $T(Y;3)B141 = B^S Xh - YS^P | 64E - 61;$
 $Xhy^+ - YS^D | 68A - 64E | 68A - 100.$
 $T(Y;3)B154 = Y | 81 - 100;$
 $Y | 64 - 80 | 64 - 61.$
 $T(Y;3)G24 = B^S Xh - Xhy^+ | 85E - 100;$
 $Xhy^+ | 81 - 85D | 80 - 61.$
 $T(Y;3)G130 = B^S Xh - h23 | 61E - 66B | 61E - 61A;$
 $Xhy^+ - h23 | 66C - 100.$
 $T(Y;3)H138 = YL \cdot YS^P | 66B - 61;$
 $YS^D | 66F - 66B | 66F - 100.$
 $T(Y;3)H140 = Y | 66F - 80 | 66F - 81;$
 $Y | 81 - 100.$
 $T(Y;3)H175 = Y | 64F - 81F | 64E - 61;$
 $Y | 81F - 100.$
 $T(Y;3)J1 = Y^P | 81F - 100;$
 $Y^D | 75C - 81F | 75C - 61.$
 $T(Y;3)J121 = YL^D | 65 - 95 | 61;$
 $YS \cdot YL^P | 96 - 100.$
 $T(Y;3)J162 = B^S Xh - h23 | 79D - 61;$
 $Xhy^+ - h23 | 99E - 79D | 99E - 100.$
 $T(Y;3)L20 = YL \cdot YS^P | 64C - 61;$
 $YS^D | 66E - 64D | 79 - 66E | 79 - 100$ (tentative).
 $T(Y;3)L130 = Y^D | 70E - 87A | 70D - 61;$
 $Y^P | 87A - 100.$
 $T(Y;3)R24 = Y | 80 - 61;$
 $Y | 81 - 91B | 94E - 98E | 94E - 91C | 98E - 100.$
 $T(Y;3)R150 = YL^D | 71B - 64F | 71B - 100;$
 $YS \cdot YL^P | 64F - 61.$

^e Immediately proximal [T(Y;3)A109] or distal [T(Y;3)D85] to the *Antp* complex (Kaufman, Lewis, and Wakimoto, 1980, Genetics 94: 115-33).

^ζ Severe *bb* phenotype in combination with *Df(1)bb158* (Golic).

^η The pieces 96A-97F and 97F-100 not visibly connected in chromocenter.

^θ Breakpoints inferred from Bar-variegated phenotype (Craymer, 1984, DIS 60: 217).

^l Carries *Cat*⁺.

T(Y;3)12-4B

cytology: T(Y;3)78F.

origin: X ray induced on y⁺Y.

references: Novitski and Ehrlich, 1970, DIS 45: 102.

T(Y;3)12-26M

cytology: T(Y;3)83D.

origin: X ray induced on y⁺Y.

references: Novitski and Ehrlich, 1970, DIS 45: 102.

T(Y;3)15

origin: X ray induced.

cytology: T(Y;3)87B5-7.

references: Reuter, Dorn, Wustmann, Friede, and Rauh, 1986, Mol. Gen. Genet. 202: 481-87.

Reuter, Gausz, Gyurkovics, Friede, Bang, Spierer, Hall, and Spierer, 1987, Mol. Gen. Genet. 210: 429-36.

genetics: Associated with *Su(var)3-6*.

*T(Y;3)42I

cytology: Break in middle of one arm of chromosome 3.

origin: X ray induced.

discoverer: Poulson.

references: 1943, DIS 17: 51.

T(Y;3)67-325

cytology: *T(Y;3)87B1-2*. Complex rearrangement.
origin: X ray induced.
discoverer: Brosseau.
references: Ellgaard and Brosseau, 1969, *Genetics* 62: 337-41.

T(Y;3)409

cytology: *T(Y;3)87E1-2*.
origin: X ray induced.
references: Reuter, Dorn, Wustmann, Friede, and Rauh, 1986, *Mol. Gen. Genet.* 202: 481-87.
genetics: Associated with *Su(var)3-7*.

T(Y;3)Antp^{rv}: Translocation (Y;3) Antennapedia-revertant

origin: X ray induced.
genetics: Revertant of *Antp^{Ns}* [partial in *T(Y;3)Antp^{rv18}*].

translocation	cytology	synonym	ref ^α	homoz. viable?
<i>T(Y;3)Antp^{rv3}</i>	84A4-B2	<i>T(Y;3)Antp^{Ns+R3}</i>	4, 5, 6	-
<i>T(Y;3)Antp^{rv18}</i>	84A4-B2	<i>T(Y;3)Antp^{Ns+R18}</i>	4, 5	+
<i>T(Y;3)Antp^{rv19}</i>	84B1-3	<i>T(Y;3)Antp^{Ns+R19}</i>	4, 5, 6	-
<i>T(Y;3)Antp^{rv96}</i>	84B1-2;94C ^β	<i>T(Y;3)Antp^{Ns+R96}</i>	1, 2, 3	-

^α 1 = Denell, 1972, *DIS* 48: 45; 2 = Denell, 1972, *Mutat. Res.* 15: 221-23; 3 = Denell, 1973, *Genetics* 75: 279-97; 4 = Duncan and Kaufman, 1975, *Genetics* 80: 733-52; 5 = Kaufman, Lewis, and Wakimoto, 1980, *Genetics* 94: 115-33; 6 = Lewis, Kaufman, Denell, and Tallericco, 1980, *Genetics* 95: 367-81.

^β New order: Y^P|94C - 84B2|94C-100; Y^D|84B1 - 61.

T(Y;3)B^{SV14}

cytology: *T(Y;2)YL;80*.
discoverer: Craymer.

T(Y;3)bx^{DB7}

cytology: *T(Y;3)89E*.
origin: X ray induced.
discoverer: D. Baker.
genetics: Mutant for *bx^d*.

T(Y;3)CA12

cytology: *T(Y;3)64C*.
origin: X ray induced simultaneously with *Df(2L)el77 = Df(2L)35A1;35B3*.
synonym: *T(Y;3)el⁷⁷*.
references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, *DIS* 55: 193-95.

T(Y;3)CA16

cytology: *T(Y;3)90B-C*.
references: Ashburner.
genetics: Associated with *Df(3L)DTS5-3* (Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, *DIS* 56: 186-91).

T(Y;3)CA49

cytology: *T(Y;3)85E1-2*.
references: Ashburner.

T(Y;3)f4: Translocation (Y;3) free arms 4

cytology: *T(Y;3)66C8-11;80*.
new order:
 61 - 66C8|80 - 100;
 B^SYL|80|66C11 - 82A|YSy⁺.

origin: X ray induced in 3^PY^D elements of *T(Y;3)J17* and *T(Y;3)J139*.

references: Craymer, 1984, *DIS* 60: 217-18, 234-36.
genetics: Phenotypically B^S.

***T(Y;3)H^{58b}: Translocation (Y;3) Hairless**

origin: γ ray induced.
discoverer: Ives, 58b25.
references: 1959, *DIS* 33: 95.
genetics: Mutant for *H*.

***T(Y;3)I**

cytology: *T(Y;3)YS;63C;72E*.
new order:
 YL·YS^P|63C - 72E|63C - 61;
 YS^D|72E - 100.

origin: X ray induced.
discoverer: Muller.
references: Painter and Muller, 1929, *J. Heredity* 20: 287-98.

Muller, 1930, *J. Genet.* 22: 299-334.
 Mohr and Mossige, 1940, *Hereditas* 26: 202-8 (fig.).
genetics: Right break in 3L between *th* and *st*. The Y^P3L^D element recoverable in hyperploid and duplicated for loci from *ru* through *th*.

T(Y;3)j3

cytology: *T(Y;3)91A*.
origin: X ray induced in B^SYy⁺.
references: Novitski and Ehrlich, 1970, *DIS* 45: 102.

T(Y;3)j6

cytology: *T(Y;3)91C*.
origin: X ray induced in B^SYy⁺.
references: Novitski and Ehrlich, 1970, *DIS* 45: 102.

***T(Y;3)K4: Translocation (Y;3) of Krivshenko**

cytology: *T(Y;3)YS;81*; inferred from metaphase cytology. Ganglion metaphases show break in YS distal to *bb* and break in 3R near centromere.
origin: X ray induced.
discoverer: Krivshenko, 59b7.
references: 1959, *DIS* 33: 96.
genetics: Occasional homozygous males apparently sterile.

T(Y;3)Kpn⁺

cytology: Autosomal break in 3R tip.
origin: X ray induced.
references: Lifschytz and Falk, 1969, *Genetics* 62: 353-58.
genetics: Recessive lethal. Revertant of *awd^K*.

T(Y;3)MA9

cytology: *T(Y;3)84B1-2*.
references: Lehmann and Nüsslein-Volhard, 1987, *Dev. Biol.* 119: 402-17.

T(Y;3)P: Translocation (Y;3) Pasadena

origin: X ray induced.

translocation	cytology	discoverer	ref ^α
<i>T(Y;3)P3</i>	61E-F	Rosenthal	3
<i>T(Y;3)P5</i>	62C-D	Rosenthal	3
<i>T(Y;3)P6^β</i>	62A-B	Heinemann	3, 4, 5
<i>T(Y;3)P26</i>	87E-F	E.B. Lewis	2
<i>T(Y;3)P80</i>	YL;88C-F	E.B. Lewis	1, 6
<i>T(Y;3)P102</i>	87B1-2	E.B. Lewis	2

- ^α 1 = CP627; 2 = Ellgaard and Brosseau, 1969, Genetics 62: 337-41; 3 = E.B. Lewis, 1969, DIS 44: 188; 4 = Ripoll and García-Bellido, 1973, DIS 50: 177; 5 = Ripoll and García-Bellido, 1978, Genetics 90: 93-104; 6 = Zuffardi, Tiepolo, Dolfini, Barigozzi, and Fraccaro, 1971, Chromosoma 34: 274-80.
- ^β *Dp(Y;3)P6* carries all the fertility factors on the Y plus *mwh*⁺ and *ve*⁺ (Ripoll and García-Bellido, 1973). Duplication product of *T(Y;3)P5* but not *T(Y;3)P3* also covers *mwh* (Lewis, 1969).

***T(Y;3)pb*²: Translocation (Y;3) proboscipedia**

cytology: *T(Y;3)75C*.
origin: γ ray induced simultaneously with, but independently of, *pb*².
references: Kaufman, 1978, Genetics 90: 579-96.

T(Y;3)PFA34

cytology: *T(Y;3)YS;99B*.
origin: X ray induced.
references: Hazelrigg, Fornili, and Kaufman, 1982, Chromosoma 87: 535-59.

***T(Y;3)ru*: Translocation (Y;3) roughoid**

origin: X ray induced.
references: Hannah-Alava, 1968, Genetica 39: 94-132.
genetics: Associated with *ru*.

***T(Y;3)S*: Translocation (Y;3) Schwartz**

origin: γ ray induced.
discoverer: Schwartz.
references: Gatti and Pimpinelli, 1983, Chromosoma 83: 349-73.

translocation	cytology ^α	genetics
<i>T(Y;3)S20</i> ^β	<i>y</i> ⁺ <i>Xh</i> ;{3R}	<i>kl-3</i> ⁻
<i>T(Y;3)S21</i> ^γ	<i>h11</i> ;{3R} + <i>Df(YL)S21</i>	<i>kl-5</i> ⁻
<i>T(Y;3)S22</i>	<i>h22</i> ;{chrom 3}	<i>ks-1</i>

- ^α Break in chromosome 3 not determined.
^β Male sterile as homozygote and as heterozygote with *T(Y;3)S21*.
^γ Male sterile as homozygote and as heterozygote with *T(Y;3)S20*.

***T(Y;3)Scr*^{E4}: Translocation (Y;3) Sex combs reduced**

origin: Induced by ethyl methanesulfonate.
discoverer: Kennison, 1984.
genetics: Associated with *Scr*.

***T(Y;3)se*^V: Translocation (Y;3) sepia-variegated**

cytology: *T(Y;3)66D*.
origin: X ray induced.
references: Jeffery, Stephans, and Giddings, 1974, Genetics 77: s32.
genetics: Variegates for *se*.

****T(Y;3)sr*^{100.23}: Translocation (Y;3) stripe**

cytology: *T(Y;3)90E2-3*.
origin: X ray induced.
discoverer: Alexander.
references: Ward and Alexander, 1957, Genetics 42: 42-54.
genetics: Mutant for *sr*.

****T(Y;3)st*^{100.126}: Translocation (Y;3) scarlet**

cytology: *T(Y;3)73A2-3*.
origin: X ray induced.
discoverer: Alexander.
references: Ward and Alexander, 1957, Genetics 42: 42-54.

genetics: Mutant for *st*.

T(Y;3)ST1

cytology: *T(Y;3)YL;69F-70A2 + In(3L)62B;74A* (Kennison).

new order:

YL^P|69F - 62B|74A - 100.

YS.YL^P|70A2 - 74A|62B - 61.

origin: γ ray induced in a *mwh iab9^{uh3}* stock.

discoverer: Tiong.

synonym: *T(Y;3)I2-10-1*.

genetics: Duplication segregant *Dp(3;Y)ST1* includes *brm*⁺ *th*⁺ *st*⁺ *tra*⁺ *KL*⁺ *KS*⁺.

***T(Y;3)Tab*^{rv89}: Translocation (Y;3) Tab-reverted**

cytology: *T(Y;3)89E + In(3R)89E;90D*.

origin: X ray induced in *In(3R)Tab*.

references: Celniker and Lewis, 1987, Genes and Development 1: 111-23.

genetics: Revertant of the dominant *Tab* phenotype. When hemizygous, shows stronger transformation of the eighth ventral setal band toward that of the seventh than does *Tab/Df(3R)P9*; also, A7 transformed toward A5 or A6, no posterior spiracles and no ninth ventral setal band.

***T(Y;3)Ubx*^{21R}**

cytology: *T(Y;3)89E*.

origin: X ray induced.

synonym: *Ubx*^{19649.21R}.

references: E.B. Lewis.

genetics: Associated with *Ubx*^{21R}.

***T(Y;3)Ubx*¹³⁴³**

cytology: *T(Y;3)89E*.

origin: X ray induced.

synonym: *Ubx*^{31616.1343}.

references: E.B. Lewis.

genetics: Associated with *Ubx*¹³⁴³.

***T(Y;3)vin*¹⁰¹: Translocation (Y;3) vin**

cytology: *T(Y;3)87F12-14*.

origin: γ ray induced simultaneously with, but independently of, *vin*¹⁰¹.

references: Akam, Roberts, Richards, and Ashburner, 1978, Cell 13: 215-25.

genetics: Homozygous lethal but viable with all *vin* deficiencies.

***T(Y;3;4)fkh*: Translocation (Y;3;4) fork head**

cytology: *T(Y;3;4)98D2-3;99F;101*.

origin: X ray induced.

references: Jürgens and Weigel, 1988, Roux's Arch. Dev. Biol. 197: 345-54.

genetics: Weak *fkh* phenotype.

T(Y;4): see 4Y

Described in section on Y derivatives.

T(Y;4)B15

cytology: *T(Y;4)YL;102F*.

new order:

B^SYL|102F - 101A;

102F|YL·YSy⁺.

origin: X ray induced using B^SYy⁺.

references: Hazelrigg, Fornili, and Kaufman, 1982, Chro-

mosoma 83: 535-59.

genetics: Male fertile as *X/T(Y;4)*.

T(Y;4)ci1: Translocation (Y;4) cubitus interruptus

origin: X ray induced.

synonym: *R¹(ci)*.

references: Stern and Kodani, 1955, *Genetics* 40: 343-73.

other information: Breakpoints and position effect information not given.

T(2;?)odd^{5.1}: see *Tp(2;?)5.1*

T(2;3)3.29

cytology: *T(2;3)25C;100F*.

origin: X ray induced.

discoverer: Nüsslein-Volhard.

synonym: *T(2;3)odd^{3.29}*.

references: Nüsslein-Volhard, Kluding, and Jürgens, 1985, *Cold Spring Harbor Symp. Quant. Biol.* 50: 145-54.

genetics: Second chromosome breakpoint between *slp* and *mid*; neither gene mutant.

T(2;3)63

origin: γ ray induced.

discoverer: Hinton, 63b.

references: 1964, *DIS* 39: 61 (except for *T(2;3)63-5*). 1965, *Genetics* 51: 971-82.

translocation	cytology	genetics
<i>T(2;3)63-1</i>	49D-E;79B-C	homozygous viable, fertile; eyes slightly rough
<i>T(2;3)63-2</i>	27B-C;75C	homozygous lethal
<i>T(2;3)63-3^α</i>	40-41;80-81	homozygous lethal
<i>T(2;3)63-5</i>	40C;89E-F + <i>In(3L)69-70;79-80</i>	homozygous viable, fertile; short bristles; wings obliquely creased, ovate, often asymmetrical
<i>T(2;3)63-6</i>	59E-F;89E-F	homozygous viable, fertile; short bristles; wings obliquely creased, ovate, often asymmetrical
<i>T(2;3)63-7</i>	41C;92D-E	homozygous lethal
<i>T(2;3)63-8</i>	36E;86B	homozygous lethal
<i>T(2;3)63-9</i>	34A-B;75C	homozygous lethal
<i>*T(2;3)63-10</i>	33-34;76D-E	homozygous viable but sterile; abdominal tergites more pigmented than in wild type
<i>T(2;3)63-13</i>	24-25;94D-E	homozygous viable, fertile; variegated eye color
<i>T(2;3)63-14</i>	38A-B;69A-B	homozygous lethal
<i>T(2;3)63-15</i>	41D;64A	homozygous lethal; variegated eye color in heterozygote
<i>T(2;3)63-16</i>	41C-D;93A-B	homozygous viable, fertile; eyes slightly rough
<i>T(2;3)63-17</i>	40C;96A-B	homozygous lethal; eye color in heterozygote variegated over <i>SM5</i> but normal over <i>bw</i> or +
<i>T(2;3)63-18</i>	39B-C;80C	homozygous viable, fertile; trough-like wing posture in 90% of flies
<i>T(2;3)63-19</i>	24D-E;80C	homozygous lethal
<i>T(2;3)63-21</i>	32E;89C-E + <i>In(3LR)65B;84B</i>	homozygous lethal
<i>T(2;3)63-22</i>	40B;84D	homozygous lethal
<i>T(2;3)63-23^α</i>	40-41;80-81	homozygous lethal

^α Inferred from genetic data because salivary chromosomes appear normal.

T(2;3)64

origin: γ ray induced.

discoverer: Hinton, 63b.

references: 1965, *Genetics* 51: 971-82.

translocation	cytology
<i>T(2;3)64-31</i>	36D-E;96B-C + <i>In(2R)41E-F;55F</i>

translocation	cytology
<i>T(2;3)64-32</i>	35D-E;70C-D
<i>T(2;3)64-33^α</i>	40-41;80-81
<i>T(2;3)64-34</i>	25D;86C
<i>T(2;3)64-35</i>	40B;92C
<i>T(2;3)64-36</i>	40D;85E
<i>T(2;3)64-37</i>	60E;82F

^α Inferred from genetic data because salivary chromosomes appear normal.

**T(2;3)100r20*: see *T(1;2;3)100r20*

T(2;3)101

cytology: *T(2;3)44B;83E-F* (Lewis, 1956, *DIS* 30: 130).

discoverer: Sturtevant.

genetics: Homozygous viable; male fertile and female sterile. Crossing over about normal in chromosome 2 of heterozygous female.

****T(2;3)103***

discoverer: Sturtevant.

genetics: Homozygous lethal. Reciprocal translocation with breaks in 2L and 3L. Crossing over in heterozygous female low in 2L, normal in 2R.

T(2;3)108

cytology: *T(2;3)37-38;52D-F;80;81* + *T(2;3)42A2-3;58A4-B1* inferred from a combination of cytological [52D-F (Lewis, 1951, *DIS* 25: 108-9)] and genetic observations.

new order:

21 - 37|(80 - 81)|52D - 42A3|58B1 - 60;
61 - 80|(37 - 42A2|58A4 - 52F)|81 - 100.

origin: Arose in *In(2R)Cy = In(2R)42A2-3;58A4-B1*.

discoverer: Sturtevant.

references: Bahn, 1971, *Hereditas* 67: 75-78.

genetics: Mutant for *Rev*. Homozygous semilethal. The segregant that receives a normal chromosome 2 and the transposed element that might be designated *3L^D2L^P3R^D* survives and is fertile. It is duplicated for the loci of *pr*, *lt*, *rl*, *tk*, and according to Lewis, for *M(2)53*, *sm*, and *hy*; not deficient for chromosome 3 genes.

T(2;3)109

cytology: *T(2;3)22F-23B;55F-56A;80* (Lewis, 1951, *DIS* 25: 108-9).

new order:

21 - 22F|55F - 23B|80 - 61;
60 - 56A|80 - 100.

discoverer: Sturtevant.

genetics: Homozygous viable and wild type. Originated in *In(3R)P* but is separable from it.

****T(2;3)110***

origin: X ray induced.

discoverer: Sturtevant.

genetics: Homozygous lethal. Wings short, extended, and coiled downward in spiral. L4 and marginal veins thickened, L4 sometimes not reaching margin; posterior wing cell reduced. Posterior crossvein absent; L5 reduced and irregularly plexate. Break in 2R near *vg* and one in 3R, which carries *In(3R)P*. New order is 2L + 3L and 2R + 3R.

***T(2;3)135**

cytology: *T(2;3)37;85A*.
origin: X ray induced simultaneously with *T(1;2)l-v135*.
references: Lindsley, Edington, and Von Halle, 1960, Genetics 45: 1663.

T(2;3)203

cytology: *T(2;3)57A5-10;84C8-D1* interband (B.S. Baker, 1985).
discoverer: Ou.
genetics: Transvects *Cbx Ubx/+*.

T(2;3)205

cytology: *T(2;3)41;89D* (E.B. Lewis).
origin: X ray induced in *Canton-S*.
discoverer: Ou.

T(2;3)221

cytology: *T(2;3)22B;62F* (E.B. Lewis).
origin: X ray induced in *Canton-S*.
discoverer: Ou.
references: Craymer, 1984, DIS 60: 234-36.
genetics: Discovered in association with *In(3R)221*; does not transvect *Cbx Ubx/+*.

T(2;3)429 - T(2;3)445

origin: X ray induced.
discoverer: Gelbart.

translocation	cytology
T(2;3)429.3	23A1-2;83C1-2
T(2;3)429.9	38D-E;85C + Df(2L)22D5-6;23B1-2
T(2;3)429.25	40;61F
T(2;3)429.33	40;65E + Tp(3;2)70C;71B
T(2;3)429.80	22F3-23A1;31C;35C;70A;81A;92C
T(2;3)432.4	23C;41A;70B3-C1
T(2;3)432.12	23D;70D
T(2;3)432.30	26C;71E-F;81F
T(2;3)432.45	23D1-2;80F
T(2;3)434.5	25A1-2;81F
T(2;3)434.9	24A1-2;80F
T(2;3)434.12	21F3-4;84F13-14 (B.S. Baker) + 49E-F;90C
T(2;3)434.17	22C;25A1-2;55A1-2;85A1-2;98F
T(2;3)434.19	22C;40;68E;80F;81F;96D
T(2;3)434.25	28E-F;81F(?)
T(2;3)434.31	see T(2;3)DTD36
T(2;3)434.32	49E-F;97A-B
T(2;3)434.35	36C;64D-F + 44E;78E
T(2;3)434.37	40;68B-D + <i>In(2LR)21E;59C</i>
T(2;3)434.54	21B;90A1-2
T(2;3)434.60	57E-F;84A-B + <i>In(2LR)34D;41A</i>
T(2;3)434.61	31E-F;90C-D
T(2;3)434.64	21F-22A1;27E;75C
T(2;3)434.71	see T(2;3)DTD46
T(2;3)434.74	22F3-23A1;78A1-2
T(2;3)434.102	21F;34A1-2;41A;51F;96C
T(2;3)434.121	22B;32B;42A;43A;74E-F;81F;85A
T(2;3)434.122	24F;76D;81F;89E1-3
T(2;3)434.125	35F;84E9 (B.S. Baker) + <i>In(2LR)22A;41A-C</i>
T(2;3)434.128	24A1-2;68F1-2
T(2;3)434.131	25D3-4;97A2-4
T(2;3)434.32	49E-F;97A-B
T(2;3)434.9	24A1-2;80F
T(2;3)445.1-2	41A;98A10-15
T(2;3)445.2-1	27C3-8;82D
T(2;3)445.2-2	33D;82F
T(2;3)445.2-3	40-41;80-81(?)
T(2;3)445.2-4	52F;67E4-F1
T(2;3)445.2-5	40B;69A4-B1

translocation	cytology
T(2;3)445.2-7	35E;89A;92A
T(2;3)445.2-9	40-41;80-81(?)
T(2;3)445.2-10	58C3-D1;93F
T(2;3)445.3-1	57B;77B
T(2;3)445.3-2	42A16-19;86E6-F1
T(2;3)445.3-3	21C1-2;67F
T(2;3)445.3-4	40;95B

T(2;3)A

cytology: *T(2;3)39B-C;83B* (Lewis, 1951, DIS 25: 108-9).
origin: X ray induced in *Bl*.
discoverer: Dobzhansky, 28h.
references: 1929, Biol. Zentr. 49: 408-19.
 1933, Z. Indukt. Abstamm. Vererbungs. 64: 269-309.
 Dobzhansky and Sturtevant, 1931, Carnegie Inst. Washington Publ. No. 421: 29-59.
genetics: Homozygous lethal.

***T(2;3)A1: Translocation (2;3) from Austin**

origin: X ray induced.
references: Patterson, Stone, Bedichek, and Suche, 1934, Am. Nat. 68: 359-69.
 Pipkin, 1940, Texas Univ. Publ. 4032: 73-125.
genetics: Homozygous viable and fertile. Chromosomes 2 and 3 broken at chromocenter. 2L attached to 3R and 3L to 2R.

T(2;3)A1-W: Translocation (2;3) A1 of Wallace

cytology: *T(2;3)22;68* superimposed on *In(2L)22D1-2;33F5-34A1 + In(2R)42A2-3;58A4-B1 + In(3LR)61A-C;74;89D-E;93B;96A*.
origin: Spontaneous in *In(2L)Cy In(2R)Cy; TM2*.
references: Wallace, Zouros, and Krimbas, 1966, Amer. Nat. 100: 245-51.
 Wallace, 1966, Amer. Nat. 100: 565-83.
 Thompson, 1977, Genetics 85: 125-40.
 1983, DIS 59: 129-30.
genetics: Homozygous lethal because of the dominant markers in the component balancers *In(2L)Cy In(2R)Cy, Cy L* and *In(3LR)Ubx¹³⁰, Ubx¹³⁰ e^s (TM2)*. Segregates as a unit and suppresses recombination in chromosomes 2 and 3.

T(2;3)A2

cytology: *T(2;3)46B;47F;78A*.
new order:
 21 - 46B|47F - 46B|78A - 61;
 60 - 47F|78A - 100.
origin: X ray induced.
references: Ashburner, 1972, DIS 49: 34.
genetics: Homozygous lethal.

***T(2;3)A26**

origin: X ray induced.
discoverer: Muller.
references: Painter and Muller, 1929, J. Heredity 20: 287-98.
 Muller, 1930, J. Genet. 22: 299-334.
genetics: Break in 3R between *sr* and *e*.

T(2;3)A147: see T(Y;2;3)A147

T(2;3)abd-A: Translocation (2;3) abdominal-A

origin: X ray induced.
genetics: Mutant for *abd-A*.

translocation	cytology	discoverer	ref ^α	molecular biology ^β
T(2;3)abd-A⁵¹⁷γ	34-5;41;78;83-84;89E	Baker		40-45 kb
T(2;3)abd-A^{MX1}	40;89E1-2	Morata	1, 2	54-56 kb
T(2;3)abd-A^{S2}	37C;69D;89E;complex	Tiong	1, 3	50-55 kb

^α 1 = Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96; 2 = Sánchez-Herrero, Vernós, Marco, and Morata, 1985, Nature 313: 108-13; 3 = Tiong, Bone, and Whittle, 1985, Mol. Gen. Genet. 200: 335-42.
^β Coordinates of 89E breakpoints in kilobases to the right of the right breakpoint of *In(3R)Cbx^{rv1}*.
^γ Synonym: *iab2^{31616.517}*.

T(2;3)Adh1: Translocation (2;3) Alcohol dehydrogenase

cytology: *T(2;3)33E9-34A1;89A3-7* (Ashburner); originally described as *T(2;3)35A;35D;89A* (Nash, 1970).
origin: X ray induced as a "double-crossover" in a *b⁺rk^{EMS}el⁺Adh^D/b rk⁺el Adh^F* female.
discoverer: E.H. Grell.
synonym: *T(2;3)DpAdh1*.
references: Grell, 1969, Genetics 61: s23. Nash, 1970, Genetics 64: 471-99. Ashburner, 1982, Genetics 101: 447-59.
genetics: Originally described as duplicated for *Adh* (Nash, 1970), but presumably has undergone spontaneous exchange and no longer carries the *Adh* duplication nor enhances *H* (Ashburner).

T(2;3)Antp^{Ctx}: Translocation (2;3) Antennapedia-Cephalothorax

cytology: *T(2;3)35B;84B1-2 + In(2R)* (Ashburner).
origin: X ray induced.
discoverer: Duncan and E.B. Lewis.
synonym: *T(2;3)Ctx*.
references: Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, Cell 35: 763-76. Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 283-95.
genetics: Recessive lethal. Heterozygotes show transformation of dorsal head structures into dorsal thoracic structures and, occasionally, transformation of antennae into legs.
molecular biology: Third-chromosome breakpoint 165-166 kb distal to proximal breakpoint of *Tp(3;3)Dfd^{rv16}*.

T(2;3)Antp^{rk4}: Translocation (2;3) Antennapedia-recessive

cytology: *T(2;3)36C-D;84B1-2 + In(3LR)62B;98F*.
origin: X ray induced.
synonym: *T(2;3)antp^{Xbk4}*.
references: Lewis, Kaufman, Denell, and Talerico, 1980, Genetics 95: 367-81. Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, Cell 35: 763-76. Abbott and Kaufman, 1986, Genetics 114: 919-42.
genetics: Recessive lethal without dominant phenotype.
molecular biology: Third-chromosome breakpoint 120 kb distal to the proximal breakpoint of *Tp(3;3)Dfd^{rv16}*.

T(2;3)Antp^{rL1}

cytology: *T(2;3)25F;84B2*.
origin: X ray induced.
synonym: *T(2;3)antp^{L1}*.
references: Abbott and Kaufman, 1986, Genetics 114: 919-42.
genetics: Recessive lethal without dominant phenotype.

T(2;3)Antp^{rv}

genetics: Revertant of *Antp*. Homozygous lethal.

translocation	cytology	origin ^α	synonym	ref ^β
T(2;3)Antp^{rv5}	60B3;84B1-2;97B3	2	<i>T(2;3)Antp^{73b-RX5}</i>	2
T(2;3)Antp^{rv6}	57B6-8;84B1-2	2	<i>T(2;3)Antp^{73b+RX6}</i>	2
T(2;3)Antp^{rv7}	40;84B1-2	2	<i>T(2;3)Antp^{73b+RX7}</i>	2
T(2;3)Antp^{rv8}γ	41;84B1-2	1	<i>T(2;3)Antp^{Ns+RC8}</i>	4, 5
T(2;3)Antp^{rv13}δ	40-41;84A4-B2	3	<i>T(2;3)Antp^{Ns+R13}</i>	1, 3

^α 1 = EMS induced (Struhl, 1981); 2 = X ray induced in *In(3R)Antp^{73b} = In(3R)84B1-2;84C5-6*; 3 = X ray induced in *Antp^{Ns}*.
^β 1 = Duncan and Kaufman, 1975, Genetics 80: 733-52; 2 = Hazelrigg and Kaufman, 1983, Genetics 105: 581-600; 3 = Lewis, Kaufman, Denell, and Talerico, 1980, Genetics 95: 367-81; 4 = Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, Cell 35: 763-76; 5 = Struhl, 1981, Nature 292: 635-38.
^γ Lethal with *Antp^B*.
^δ Molecular biology: Third-chromosome breakpoint 144-147 kb distal to the proximal breakpoint of *Tp(3;3)Dfd^{rv16}* (Scott *et al.*, 1983).

T(2;3)Antp^{Scx}: see *T(2;3)Scx*

T(2;3)Antp^{Yu}: Translocation (2;3) Antennapedia of Yu

cytology: *T(2;3)22A4-5;84B2-4 + T(2;3)37B1-2;98B1-2* (Kaufman *et al.*, 1980).
new order:
21 - 22A4|84B4 - 98B1|37B1 - 22A5|84B2 - 61;
60 - 37B2|98B2 - 100.
origin: X ray induced.
discoverer: Yu, 1948.
references: 1949, Ph.D. Thesis, Calif. Inst. Tech. Lewis, 1956, DIS 30: 76. Kaufman, Lewis, and Wakimoto, 1980, Genetics 94: 115-33.
genetics: Mutant for *Antp*; associated with 84B2-4 breakpoint. Homozygous lethal.

T(2;3)ap^{Xa}: Translocation (2;3) apterous-Xasta

cytology: *T(2;3)41F;89E8-F1* superimposed on *In(2R)42A2-3;58A4-B1 + In(3R)89C2-3;96A18-19* (Bridges in Morgan, Bridges, and Schultz, 1936, Year Book - Carnegie Inst. Washington 35: 294; correction by Lewis, 1951, DIS 25: 108-9).
new order:
21 - 41F|89E8 - 89C3|96A19 - 100;
60 - 58B1|42A3 - 58A4|42A2 - 41F|89F1 - 96A18|89C2 - 61.
origin: X ray induced in *In(2R)Cy; In(3R)P*.
discoverer: Serebrovsky, 28a.
synonym: *T(2;3)Xa: Translocation (2;3) Xasta*.
references: Serebrovsky and Dubinin, 1930, J. Hered. 21: 259-65. Sturtevant, 1934, DIS 2: 19.
genetics: Dominant mutant for *ap*. Homozygote virtually lethal.
other information: The first X-ray-induced mutation recovered in the USSR. Useful as a balancer of 2R and 3R.

***T(2;3)ast^{rv1}: Translocation (2;3) asteroid-reverted**

cytology: T(2;3)21E2-3;68C2-3;88D8-9.
new order:
 21A – 21E2|88D8 – 68C3|88D9 – 100;
 61 – 68C2|21E3 – 60.
origin: X ray induced in *al ast ho*.
discoverer: E.B. Lewis, 1942.
references: 1945, Genetics 30: 158.
genetics: Associated with a reversion of *ast*. Homozygous lethal.

***T(2;3)ast^{rv3}**

cytology: T(2;3)21E2-3;61C2-3.
origin: X ray induced in *net ast dp cl*.
discoverer: E.B. Lewis, 1942.
references: 1945, Genetics 30: 158.
genetics: Associated with reversion of *ast*. Lethal homozygous and heterozygous with *Df(2L)S4 = Df(2L)21C3-4;22B2-3*.

T(2;3)Asx^{T1}: Translocation (2;3) Additional sex combs

cytology: T(2;3)41-42;84E-F;88B.
new order:
 21 – 41|88B – 84F|88B – 100;
 61 – 84E|42 – 60.
origin: γ ray induced in Oregon-R.
discoverer: Tiong.
synonym: T(2;3)OR-6-6-1.
genetics: Associated with *Asx^{T1}* but should be separable.

***T(2;3)Ata: Translocation (2;3) Arista**

cytology: T(2;3)40;66F-67A + T(2;3)47;81.
new order:
 21 – 40|67A – 81|47 – 60;
 61 – 66F|40 – 47|81 – 100.
origin: X ray induced.
discoverer: Krivshenko, 1949.
synonym: T(2;3)At (symbol preoccupied).
references: 1954, DIS 28: 74-75.
 1955, DIS 29: 73.
genetics: Associated with *Ata*. Homozygous lethal.

T(2;3)AWT1

cytology: T(2;3)29F;94B.
origin: Spontaneous in AW chromosome line during successive backcross programs.
references: Yamaguchi and Mukai, 1974, Genetics 78: 1209-21.

T(2;3)b: Translocation (2;3) black

references: Alexandrov and Alexandrova, 1986, DIS 63: 159-61.
genetics: Mutant or deficient for *b*.

translocation	cytology	origin	comments
T(2;3)b ^{79b6}	33E10;34D6;79D3;80		
T(2;3)b ^{79d6}	34A2-3;34D8-E2;79B;80C	neutrons + γ rays	homozygous lethal
*T(2;3)b ⁸³¹²	34B12-C1;35A2-A23;83A3-7;83C + Df(2L)34C2;34E4-5	neutrons	homozygous lethal
T(2;3)b ^{85c2}	34C7-D1;34E1-2;94C4-D1	neutrons	viable homozygous sterile

T(2;3)B

cytology: T(2;3)33;81F (Lewis, 1951, DIS 25: 108-9; 1954, Am. Nat. 88: 225-38).
origin: X ray induced.
discoverer: Dobzhansky, 28h.
references: 1929, Biol. Zentr. 49: 408-19.
 Dobzhansky and Sturtevant, 1931, Carnegie Inst. Washington Publ. No. 421: 29-59.
genetics: Homozygous lethal. Crossing over reduced in 2L.

T(2;3)Ba¹⁵

cytology: T(2;3)60E5-6;64B12-C12.
synonym: *Dll^J;Ba^J*.
references: Cohen, Brönnner, Küttner, Jürgens, and Jäckle, 1989, Nature, 338: 432-34.
genetics: Mutant for *Ba*.
molecular biology: 60E breakpoint located on molecular map (Cohen *et al.*, 1989).

T(2;3)BTD: Translocation (2;3) Bithorax Transvection Disrupter

origin: γ ray induced.
references: Smolik-Utlaut and Gelbart, 1987, Genetics 116: 285-98.

translocation	cytology
T(2;3)BTD3	40;87F + In(3R)81;85D
T(2;3)BTD5	25C;64C;89C
T(2;3)BTD6	27E;81
T(2;3)BTD10	50C;81

T(2;3)BTD73

cytology: T(2;3)58A1;81F.
references: Schejter and Shilo, 1989, Cell 56: 1093-1104.

T(2;3)bw^D Translocation (2;3) brown-Dominant

origin: γ ray induced.
references: P. Simpson.

translocation	cytology
T(2;3)BTD ^{Drv12}	59D4-E1;66D1
T(2;3)BTD ^{Drv18}	59D4-D10;98F13-14

T(2;3)bw^R: Translocation (2;3) brown-Rearranged

origin: X ray induced.
discoverer: Slatis.
references: 1955, Genetics 40: 5-23.
genetics: Associated with corresponding *bw^R*.

translocation	cytology
*T(2;3)bw ^{R4}	59E2-3;80-81
*T(2;3)bw ^{R12}	59D;80C
*T(2;3)bw ^{R14}	59E2-3;80
*T(2;3)bw ^{R15}	59D;80C

T(2;3)bw^V: Translocation (2;3) brown-Variigated

origin: X ray induced.
genetics: Variigated for *bw*.

translocation	cytology	discoverer α	ref β
*T(2;3)bw ^{V3} γ	2R break near <i>bw</i> ; 3L break just left of centromere	3	1, 2

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translocation	cytology	discoverer ^α	ref ^β
<i>T(2;3)bw</i> ^{V4} γ	2R break near <i>bw</i> ; 3L break near centromere	4	1, 2, 3
<i>T(2;3)bw</i> ^{V5} γ	2R break near <i>bw</i> ; 3L break near centromere	4	1, 2
* <i>T(2;3)bw</i> ^{V6} δ	probably breaks in 2L, 2R, and 3R	2	1, 2
* <i>T(2;3)bw</i> ^{V8}	2R break at <i>bw</i> ; 3R break near <i>p</i>	1	

^α 1 = Levy, 1932; 2 = Moore, 1929; 3 = Muller; 4 = Patterson.
^β 1 = Glass, 1933, J. Genet. 28: 69-112; 2 = Glass, 1934, Am. Nat. 68: 107-14; 3 = McKee, 1984, Genetics 106: 403-22.
^γ Translocation parts interchangeable in *T(2;3)bw*^{V3}, *T(2;3)bw*^{V4}, *T(2;3)bw*^{V5} without altering phenotype.
^δ Crossing over reduced in 2L, 2R, and base of 3R; eye color reverted to wild type but translocation remained.

**T(2;3)bw*^{V30k12}
origin: X ray induced.
discoverer: Van Atta, 30k12.
references: 1932, Genetics 17: 637-59.
genetics: Variegated for *bw*. Complex rearrangement with breaks in 2L near centromere, 2R near *bw*, and 3L near centromere; appears to carry an inversion in 3R.

**T(2;3)bw*^{V30k13}
origin: X ray induced.
discoverer: Van Atta, 30k13.
references: 1932, Genetics 17: 637-59.
genetics: Variegated for *bw*. Breaks in 2R near *c* and *bw* and in 3R near *cu*.

T(2;3)bw*^{VD}: **Translocation (2;3) brown-Variegated Dichaete linked
origin: X ray induced.
discoverer: Oliver, 29k24.
references: 1932, Z. Indukt. Abstamm. Vererbungsl. 61: 447-88.
genetics: Variegated for *bw*. Homozygous lethal.

T(2;3)bw^{VDe3}: **Translocation (2;3) brown-Variegated of Demerec**
cytology: *T(2;3)59D;81F*. Also an inversion in 2R.
origin: X ray induced.
discoverer: Demerec, 33j14.
genetics: Variegates for *bw* and *mi* but not *abb*. Mutant for *Dfd*. Homozygous lethal. Gives transvection effects with certain pairs of bithorax pseudoalleles (Lewis, 1955, Am. Nat. 89: 73-89).

T(2;3)bw^{VDe4}
cytology: *T(2;3)59D2-4;80* (Schultz).
origin: X ray induced.
discoverer: Demerec, 33k22.
genetics: Variegates for *bw* and *mi*. Homozygous lethal.

T(2;3)bx^d: **Translocation (2;3) bithoraxoid**
origin: X ray induced.
genetics: Mutant for *bx*^d.

translocation	cytology	discoverer
<i>T(2;3)bx</i> ^{d68}	41;89E	Baker
<i>T(2;3)bx</i> ^{d123}	41;89E	Crosby
<i>T(2;3)bx</i> ^{d127}	59C;89E + <i>In(3R)88C-D;92</i>	E.B. Lewis
<i>T(2;3)bx</i> ^{d266}	40;89E	Tung
<i>T(2;3)bx</i> ^{DB1}	41;89E	D. Baker
<i>T(2;3)bx</i> ^{DB3}	48;89E	D. Baker
<i>T(2;3)bx</i> ^{DB4}	32;89E	D. Baker

translocation	cytology	discoverer
<i>T(2;3)bx</i> ^{dDB5}	41;89E	D. Baker
<i>T(2;3)bx</i> ^{dDB6}	22A;43A-C;80D;84;89E;92F	D. Baker
<i>T(2;3)bx</i> ^{dX}	42B-C;89E1-2	E.B. Lewis

T(2;3)C

origin: X ray induced.
discoverer: Dobzhansky, 28h.
references: 1929, Biol. Zentr. 49: 408-19.
 Dobzhansky and Sturtevant, 1931, Carnegie Inst. Washington Publ. No. 421: 29-59.
genetics: Break near centromere in chromosomes 2 and 3. New order is 2L + 3L; 2R + 3R. Homozygous lethal.

T(2;3)C4 – T(2;3)C591: Translocation (2;3) Crossover suppressor

origin: X ray induced; selected on the basis of reduced recombination in heterozygous females.
references: Thomas and Roberts, 1966, Genetics 53: 855-62.
 Roberts, 1970, Genetics 65: 429-48.

translocation	cytology	new order ^α	homozygous viable?	Xover reduced in
<i>T(2;3)C4</i>	40-41;94A		-	3R
<i>T(2;3)C11</i>	40-41;64D;77A	1	+	2L
<i>T(2;3)C17</i>	56F;67E		-	2R, 3L
<i>T(2;3)C24</i>	53B;80-81		+	2R
<i>T(2;3)C29</i>	43F;92D		+	3R
<i>T(2;3)C49</i>	22C-D;86E		+	2L
<i>T(2;3)C58</i>	40-41;96F		-	3R
<i>T(2;3)C65</i> ^β	40-41;75A;80-81 + <i>In(3L)64C;77A</i>	2	-	3L
<i>T(2;3)C101</i>	29B;80-81		±	2L
<i>T(2;3)C111</i>	40-41;70F + <i>In(3L)62B;79D-E</i>		-	3L
<i>T(2;3)C122</i>	60B;80-81		+	2R
<i>T(2;3)C124</i>	34D;75F		-	2L
<i>T(2;3)C132</i>	55E;80-81		-	2R
<i>T(2;3)C149</i>	52A;93B		-	2R, 3R
<i>T(2;3)C157</i>	41;96D-E + <i>In(2LR)24F;54F</i>		+	2L, 3R
<i>T(2;3)C164</i>	32F;64B		-	2L, 3L
<i>T(2;3)C177</i>	40-41;62F + 56F;79B		-	2R, 3L
<i>T(2;3)C199</i>	41;93E		-	3R
<i>T(2;3)C202</i>	56D;89D		+	2R
<i>T(2;3)C211</i>	40-41;70C		-	3L
<i>T(2;3)C218</i>	40-41;70F		+	3L
<i>T(2;3)C230</i>	35D;61A		-	3L
<i>T(2;3)C231</i>	50D;62B + <i>In(2LR)35C-D;52A-B</i>		-	2R, 3L
<i>T(2;3)C248</i>	52C;94D;96B	3	-	2R, 3R
<i>T(2;3)C257</i>	50F;80		-	2R
<i>T(2;3)C267</i>	21D;63F;64E + <i>In(3LR)74F;88D</i>		-	3L
<i>T(2;3)C287</i>	56D;89F (Craymer, 1984, Genetics 108: 573-87)		-	2R
<i>T(2;3)C293</i>	43A;67A;80-81	4	-	3L
<i>T(2;3)C304</i>	48A;83C;100B	5	-	3R
<i>T(2;3)C308</i> ^γ	40-41;84B;94D;99B	6	-	3R
<i>T(2;3)C309</i>	58D;68F		-	2R, 3L
<i>T(2;3)C311</i>	54C;64C		-	2R, 3L
<i>T(2;3)C313</i>	27B;80-81		-	2L
<i>T(2;3)C316</i>	25F;80-81		-	2L
<i>T(2;3)C317</i>	24D;97D		-	2L, 3R
<i>T(2;3)C356</i>	29F;80-81		+	2L
<i>T(2;3)C591</i>	28D;69D		-	2L

^α 1 = 21 - 40|77A - 64D|77A - 100;
 60 - 40|64D - 61.
 2 = 21 - 40|80 - 100;
 60 - 40|75A - 64C|77A - 80|75A - 77A|64C - 61 (tentative).

- 3 = 60-52C|94D-61;
21-52C|96B-94D|96B-100.
- 4 = 21-43A|67A-61;
60-43A|80-67A|81-100.
- 5 = 21-48A|100B-100F;
60-48A|83C-100B|83C-61.
- 6 = 21-40|94D-84D|94D-99B|84B-61;
60-40|99B-100.

β Involvement of chromosome 2 inferred from genetic data.
 γ Variegates for *ca* but not for *AcpH-1* (Morrison and MacIntyre, 1978, Genetics 83: 487-97).

***T(2;3)C-K: Translocation (2;3)
Curved of Krivshenko**

cytology: T(2;3)52;76;81;86.
new order:
 21-52|86-100;
 60-52|81-76|81-86|76-61.
origin: X ray induced.
discoverer: Krivshenko, 5513.
references: 1956, DIS 30: 74.
genetics: Associated with C-K. Homozygous lethal.

T(2;3)ca^{nd3} Translocation (2;3) claret

cytology: T(2;3)44A-B;100B.
origin: γ ray induced.
references: Sequeira, Nelson, and Szauter, 1989, Genetics 123: 511-214.
genetics: Distal break near but distinct from *Ca^{nd3}*.

T(2;3)ca^v

cytology: Df(3R)81F;99C-E + T(2;3)59D;94.
new order:
 21-59D|94-99C|81F-94|59D-60;
 61-81F|99E-100.
references: Craymer, 1980, DIS 55: 199.
genetics: Associated with variegation for *ca*. Possible to recover the T(2;3) without the deficiency as a consequence of double crossover. The reciprocal product not recoverable; the deficiency is too large.

T(2;3)CA: Translocation (2;3) Cambridge

translocation	cytology	origin	discov. or ref ^{α}	associated with
T(2;3)CA1	45D1-2;96D10-11	X ray	2	Df(2L)A63
T(2;3)CA2	42D6;67A3-4	X ray	2	Df(2L)A63
T(2;3)CA3	41F;90A-B	X ray	2	Df(2L)A178
T(2;3)CA7	22B4-5;100B5	γ ray	1	Df(2L)osp141
T(2;3)CA8	36B8;67F1-2	γ ray	1	Df(2L)osp141
T(2;3)CA9	25A3-8;67C3	γ ray	3	Df(2L)TE35A-3
T(2;3)CA15	42F;100F	γ ray	1	In(3L)jz ⁴
T(2;3)CA34	21A4;64E3-13	γ ray	1	
T(2;3)CA43	57C;64E-F	γ ray	1	
T(2;3)CA46	54C;64A	γ ray	1	Df(2L)TE35A-8
T(2;3)CA47	50A-B;84A-B	γ ray	1	In(2LR)TE35A-205
T(2;3)CA48	45E-F;75C	γ ray	1	In(2LR)TE35A-205
T(2;3)CA54	50B;81	γ ray	1	T(2;3)GT8
T(2;3)CA56	40;70D	γ ray	1	
T(2;3)CA57	43A3;43F6	γ ray	1	

^{α} 1 = Ashburner; 2 = Ashburner, Aaron, and Tsubota, 1982, Genetics 102: 421-35; 3 = Velissariou.

T(2;3)cb25-17

origin: γ ray induced.
discoverer: Lyttle.
cytology: T(2;3)52B;99C.

T(2;3)Ctx: see T(2;3)Antp^{Ctx}

**T(2;3)Cy^{rvC3}: Translocation (2;3)
Curly-revertant**

cytology: T(2;3)23B3-8;72F3-4 + In(2L)22D1-2;33F5-34A1.
new order:
 21-22B3|72F4-100;
 61-72F3|22B8-22D1-2|33F5-22D2|34A1-60.
origin: X ray induced in In(2L)Cy.
discoverer: Littlewood.
references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.
genetics: Revertant of Cy.

***T(2;3)D**

origin: X ray induced.
discoverer: Dobzhansky, 28h.
references: 1929, Biol. Zentr. 49: 408-19.
 Dobzhansky and Sturtevant, 1931, Carnegie Inst. Washington Publ. No. 421: 29-59.
genetics: Heterozygote short lived and frequently sterile, especially in female. Wings misshapen and legs short.

**T(2;3)D⁴: Translocation (2;3) with
Dichaete-4**

cytology: T(2;3)21D;70-71 (Lewis).
origin: X ray induced.
discoverer: Sigmund, 1978.
references: Craymer, 1980, DIS 55: 197-200.
genetics: Mutant for *D* (missing alulae) and deficient for *ds* (shortened wings with close crossveins and widely spaced posterior scutellar bristles); also body small. Homozygous lethal and lethal over *D*: T(2;3)D⁴/ds^W shows mutant characteristics of ds^W/ds^W.

T(2;3)dp⁵

cytology: T(2;3)25A2-3;95B3-5.
origin: X ray induced.
discoverer: Velissariou.
references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.
 Velissariou and Ashburner, 1980, Chromosoma 77: 13-27.
genetics: Associated with *dp*.

T(2;3)dp⁶

cytology: T(2;3)25A2-3;81F.
origin: X ray induced.
discoverer: Velissariou.
references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.
 Velissariou and Ashburner, 1980, Chromosoma 77: 13-27.
genetics: Mutant for *dp*.

**T(2;3)dp^D: Translocation (2;3)
dumpy-Dominant**

cytology: T(2;3)25A;95B-D (Lewis).
origin: X ray induced.
discoverer: E.B. Lewis, 1962.
references: Del Campo, 1963, DIS 38: 32.
genetics: Mutant for *dp*. Homozygous lethal.

***T(2;3)dp^{w1}*: Translocation (2;3) dumpy-warped**

origin: X ray induced.
discoverer: Schalet, 1955.
references: Carlson and Schalet, 1955, DIS 29: 71-72.
 Carlson, 1958, DIS 32: 117-18.
genetics: Apparently variegated for *dp*. Homozygous lethal.

***T(2;3)Dp-S*: Translocation (2;3) with Duplication Star**

cytology: *T(2;3)21D4-E1;81F* superimposed on *Dp(2;2)21D2-3;21E2-3*.
new order:
 21A – 21E2|21D3 – 21D4|81F – 61;
 60 – 21E1|81F – 100.
origin: X ray induced in *Dp(2;2)S*, *ast ast*.
discoverer: E.B. Lewis.
references: 1945, Genetics 30: 137-66.
genetics: *Y*-suppressible expression of *ast*.

T(2;3)DpAdhl: see *T(2;3)Adhl*

***T(2;3)dpp*: Translocation (2;3) decapentaplegic (W.M. Gelbart)**

references: Spencer, Hoffmann, and Gelbart, 1982, Cell 28: 451-61.
 Segal and Gelbart, 1985, Genetics 109: 119-43.
 Irish and Gelbart, 1987, Genes Dev. 1: 868-79.
 St. Johnston, Hoffmann, Blackman, Segal, Grimaila, Padgett, Irick, and Gelbart, 1990, Genes Dev. 4: 1114-27.

alleles:

allele	class	origin	cytology	discov
* <i>T(2;3)dpp^{3α}</i>	<i>d-III</i>	X ray	<i>T(2;3)22E3-F1;85B-D</i>	Ashburner
<i>T(2;3)dpp⁷</i>	<i>d-III</i>	X ray	<i>T(2;3)22F1-2;80F</i>	Spenser
<i>T(2;3)dpp¹⁸</i>	<i>d-III</i>	X ray	<i>In(2L)22F1-2;36C4-6 + T(2;3)28A1-2;75A1-2</i>	Spenser
<i>T(2;3)dpp²⁰</i>	<i>d-III</i>	X ray	<i>T(2;3)22F1-3;64D</i>	Spenser
<i>T(2;3)dpp²²</i>	<i>d-III</i>	X ray	<i>T(2;3)22F1-3;67E</i>	Spenser
<i>T(2;3)dpp^{25β}</i>	<i>d-V</i>	X ray	<i>T(2;3)21F;22F1-3;72A-B;80F</i>	Spenser
<i>T(2;3)dpp²⁸</i>	<i>d-II</i>	EMS	<i>T(2;3)22F2-3;86E15-18</i>	Hoffmann
<i>T(2;3)dpp²⁹</i>	<i>d-V</i>	X ray	<i>T(2;3)22F2-3;87D1-2</i>	Gelbart
<i>T(2;3)dpp⁴⁴</i>	<i>d-V</i>	γ ray	<i>T(2;3)22F1-2;80C</i>	Irish
<i>T(2;3)dpp^{52γ}</i>	<i>d-V</i>	γ ray	<i>T(2;3)22F1-2;86A-B</i>	Irish
<i>T(2;3)dpp⁷¹</i>	<i>d-III</i>	γ ray	<i>T(2;3)22F1-2;101</i>	
<i>T(2;3)dpp⁷⁷</i>	<i>d-V</i>	γ ray	<i>T(2;3)22F1-2;80F</i>	Segal
<i>T(2;3)dpp⁷⁸</i>	<i>d-V</i>	γ ray	<i>T(2;3)22F1-2;95A1-2</i>	Segal

^α Recovered by Faithfull as *T(2;3)ho5 + In(2LR)22F1-2;58B* (Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95).

^β New order: 21A – 21F|80F – 72B|22F3 – 60; 61A – 72A|21F – 22F1|80F – 100.

^γ Synonym: *T(2;3)dpp^{d52}* (Irish and Gelbart, 1987, Genes and Development 1: 868-79).

***T(2;3)Dr^L*: Translocation (2;3) Drop of Lewis**

cytology: *T(2;3)44;89F-90A + In(3R)89C;95D-96B1*.

new order:
 21 – 44|89F – 89C|96D1 – 100;
 60 – 44|90A – 95B1|89C – 61.

origin: X ray induced.
discoverer: E.B. Lewis.
genetics: Mutant for *Dr*, which is probably independent of rearrangement.

***T(2;3)ds¹⁴*: Translocation (2;3) dachsous**

cytology: *T(2;3)21D3;50A-B1;87B*.
new order:
 21A – 21D|87B – 61;
 60 – 50B1|21D3 – 50A|87B – 100.
origin: X ray induced.
references: Korochkina and Golubovsky, 1978, DIS 53: 197-200.
genetics: Homozygous lethal. Heterozygotes show *ds* and weak *al*.

***T(2;3)dsx* Translocation (2;3) doublesex**

origin: X ray induced in *dsx^M*.
references: Baker, Hoff, Kaufman, Wolfner, and Hazelrigg, 1991, Genetics 127: 125-38.
genetics: Recessive *dsx*. Revertant of *dsx^M*; *dsx* mutants..

translocation	cytology	synonym
<i>T(2;3)dsx³⁰</i>	28F;32F;84A1-2;	<i>T(2;3)dsx^{M+R1}</i>
<i>T(2;3)dsx³²</i>	84D11-E2;88B1-2	<i>T(2;3)dsx^{M+R5}</i>
	59D1-2;84E1-2	
<i>T(2;3)dsx⁴⁵</i>	58A1-2;84E1-2	<i>T(2;3)dsx^{M+47}</i>

***T(2;3)DTD*: Translocation (2;3) Disrupter of Transvection at Decapentaplegic**

origin: X ray induced. 3 and 85-133 induced in *Dp(1;2)w⁺70h, dpp^{ho2}*, 36 and 46 in *dpp⁴* and 85-133 in *dpp^{ho2} TE23C-D[w⁺]*.
genetics: Disrupt transvection at *dpp*.

translocation	cytology	ref ^α
<i>T(2;3)DTD3</i>	25F1-2;37B1-2;80	3
<i>T(2;3)DTD14^β</i>	22D-E;70A + 31C-D;81F + 35C;92C	1
<i>T(2;3)DTD36</i>	23D1-2;81F	2
<i>T(2;3)DTD43</i>	57E-F;84A-B + <i>In(3LR)35B1-2;41A</i>	2
<i>T(2;3)DTD46</i>	26F;81F	2
<i>T(2;3)DTD66^γ</i>	21E;80F + 34D;61A	1
<i>T(2;3)DTD85</i>	34C;78E	
<i>T(2;3)DTD95</i>	23C;62B	
<i>T(2;3)DTD97</i>	40A;53F	
<i>T(2;3)DTD100</i>	24E;60A;81F	
<i>T(2;3)DTD101</i>	32E-33A;80	
<i>T(2;3)DTD102</i>	30B;100D-E	
<i>T(2;3)DTD104</i>	25C;64A	
<i>T(2;3)DTD105</i>	22;85B + 39F-40A;95A	
<i>T(2;3)DTD106</i>	26A;75A + <i>Dp(3;3)81F;82F-83A;95B</i>	
<i>T(2;3)DTD108</i>	35E-F;81F;82F	
<i>T(2;3)DTD110</i>	29C;85C + 40C;90C	
<i>T(2;3)DTD113</i>	24A1-2;68E1-2	
<i>T(2;3)DTD115</i>	40;89F-90A	
<i>T(2;3)DTD118</i>	29E1-2;32B-C;83D	
<i>T(2;3)DTD120</i>	24E;65F	
<i>T(2;3)DTD122</i>	32E;89B	
<i>T(2;3)DTD123</i>	39E-F;89E	
<i>T(2;3)DTD127</i>	40;88E1-2	
<i>T(2;3)DTD130</i>	22E1-2;89A	
<i>T(2;3)DTD131</i>	26A;46E;100B	
<i>T(2;3)DTD132</i>	28D;97E	
<i>T(2;3)DTD133</i>	50A;68A + <i>In(3R)81F;85D</i>	

^α 1 = Gelbart; 2 = Gelbart, 1982, Proc. Nat. Acad. Sci. USA 79: 2636-40; 3 = Smolnik-Utlaut and Gelbart, 1987, Genetics 116: 285-98.

^β New order: 21 – 22D|70A – 81F|31C – 22E|70A – 61A; 60F – 35C|92C – 81F|31D – 35C|92C – 100.

^γ New order: 21A – 21E|80F – 61A|34D – 21E|80F – 100; 60 – 34D|61A.

***T(2;3)e^{D8}*: Translocation (2;3) ebony**

cytology: *T(2;3)40-41;93D1-6*.
origin: X ray induced.
references: D'Alessandro, Ritossa, and Scalenghe, 1977, DIS 52: 46.
genetics: Homozygous lethal. Associated with *e*.

***T(2;3)e^{H1}*: Translocation (2;3) ebony of Henikoff**

cytology: *T(2;3)40-41;93D*.
origin: X ray induced.
references: Henikoff, 1980, DIS 55: 61-62.
genetics: Mutant for *e* (no variegation even in presence of extra *Y*).

****T(2;3)E***

cytology: *T(2;3)30B;67E* (Schultz).
origin: Spontaneous.
discoverer: Sturtevant, 1929.
references: Dobzhansky and Sturtevant, 1931, Carnegie Inst. Washington Publ. No. 421: 29-59.
genetics: Homozygous lethal.

***T(2;3)E^{HH}*: Translocation (2;3) E of Hilliker and Holm**

cytology: *T(2;3)40;92E-F*.
new order:
 21 – 40F|92E – 61;
 60 – 40F|92F – 100;
origin: γ -ray-induced detachment of *C(2L)RM-SH3* *C(2R)RM-SH3* females.
references: Hilliker and Holm, 1975, Genetics 81: 705-21.
genetics: Lethal with *Df(2L)C*, *Df(2L)C'*, and *In(2LR)bw^{VI}*; viable with *Df(2L)D* and *Df(2L)D'*.
 Variegates for *lt*.

***T(2;3)E(da)*: Translocation (2;3) Enhancer of daughterless**

cytology: *T(2;3)41;66C*.
origin: X ray induced.
references: Mange and Sandler, 1973, Genetics 73: 73-86.
genetics: Associated with *E(da)*.

***T(2;3)E(SD)1*: Translocation (2;3) Enhancer of Segregation Distorter**

cytology: *T(2;3)41;100 + Df(2L)E(SD)1 = Df(2L)40A-B;40E*.
origin: γ ray induced.
references: Ganetzky, 1977, Genetics 86: 321-55.
genetics: Associated with *E(SD)*.

***T(2;3)eg^{spy}*: Translocation (2;3) eagle-spready**

cytology: *T(2;3)33D4-E3;79A4-B1*.
origin: X ray induced.
discoverer: Puro, 1961.
synonym: *T(2;3)spy*.
references: 1968, DIS 43: 59.
 Puro and Arajarvi, 1968, DIS 43: 89-90.
 1969, Hereditas 62: 414-18.
 Puro, 1982, DIS 58: 205-08.
genetics: Homozygous viable and fertile. Mutant for *eg*.

***T(2;3)el24*: Translocation (2;3) elbow**

cytology: *T(2;3)35B1-3;93C3-7*.
origin: Induced by ethyl methanesulfonate.
genetics: Mutant for *el*.

***T(2;3)en*: Translocation (2;3) engrailed**
genetics: Associated with *en*.

translocation	cytology	origin	ref ^{α}	<i>T/T</i> viable?	DNA breakpoints ^{β}
<i>T(2;3)en^{Es} γ</i>	48A3-4;84E3-4 (B.S. Baker)	X ray	1, 2, 4, 5, 6	+	+32 to +33
<i>T(2;3)en^{LA3}</i>	48A;96C	EMS	3, 4	+	
<i>T(2;3)en^{SF24}</i>	48A;90C	EMS	2, 3, 4	-	+8
<i>T(2;3)en^{SF37} δ</i>	46C;48A;80	X ray	2, 3, 4, 5	-	-31, +5
<i>T(2;3)en^{SF42}</i>	48A;65F	X ray	2, 4	-	-13
<i>T(2;3)en^{SF50}</i>	48A;57A;81A	X ray	2, 4	-	-3
<i>T(2;3)en^{SF52}</i>	48A;57B;88F	X ray	2, 4	-	-32
<i>T(2;3)en^{SF61}</i>	48A;89A3;96B	X ray	2, 4	-	+18
<i>T(2;3)en^{SF62}</i>	48A;84D	X ray	2, 4	+	-10

^{α} 1 = Epper and Sánchez, 1983, Dev. Biol. 100: 387-98; 2 = Kornberg; 3 = Kornberg, 1981, Proc. Nat. Acad. Sci. USA 78: 1095-99; 4 = Kuner, Nakanishi, Ali, Drees, Gustavson, Theis, Kauvar, Kornberg, and O'Farrell, 1985, Cell 42: 309-16; 5 = Sato, Russell, and Denell, 1983, Genetics 105: 357-70; 6 = The Seattle-La Jolla Drosophila Laboratories, 1971, DIS 47, Suppl.

^{β} 0 coordinate at the point of the insertion associated with *en^I*. Minus values to the left; plus values to the right.

^{γ} Dominant phenotype: Partial absence of abdominal markings ("erased").

^{δ} Homozygous lethal. Enhances extra sex comb phenotype of *Pc* (Sato *et al.*, 1983).

T(2;3)Es: see *T(2;3)en^{Es}*

T(2;3)eve^{1.18}: see *Tp(2;3)eve^{1.18}*

T(2;3)FC9

cytology: *T(2;3)84D-86E-F;25F7-26A*.
references: Lehmann and Nüsslein-Volhard, 1987, Dev. Biol. 119: 402-17.

T(2;3)FC10

cytology: *T(2;3)35;84A-B;93D*.
references: Lehmann and Nüsslein-Volhard, 1987, Dev. Biol. 119: 402-17.

***T(2;3)fkh*: Translocation (2;3) fork head**

cytology: *T(2;3)38;98D2-3*.
origin: X ray induced.
references: Jürgens and Weigel, 1988, Roux's Arch. Dev. Biol. 197: 345-54.
genetics: Weak *fkh* phenotype.

T(2;3)FM27: see *Tp(3;2)N2-27*

T(2;3)FM29: see *T(2;3)N2-29*

T(2;3)FM46: see *T(2;3)N2-46*

***T(2;3)ftz^{Rpl}*: Translocation (2;3) fushi-tarazu-regulator of postbithorax-like**

cytology: *T(2;3)40-41;84B1-2*.
origin: X ray induced.
discoverer: Duncan.
synonym: *T(2;3)Rpl*.
references: Weiner, Scott, and Kaufman, 1984, Cell 37: 843-51.
 Laughon and Scott, 1984, Nature 310: 25-31.
 Duncan, 1986, Cell 47: 297-304.

genetics: Heterozygotes show transformations of posterior haltere to posterior wing.

molecular biology: Encodes truncated protein in which the C-terminal amino acids are replaced by 10 novel amino acids (Laughon and Scott, 1984). Breakpoint on 3 lies within the homeobox of the *ftz* coding sequence.

T(2;3)G16: Translocation (2;3) Gelbart

cytology: T(2;3)35D5-7;85F6-8;87F (Ashburner).

new order:

21 – 35D5|85F6 – 61;
60 – 35D7|87F – 85F8|87F – 100.

origin: X ray induced.

discoverer: Gelbart.

references: Ashburner, Tsubota, and Woodruff, 1982, Genetics 102: 401-20.

genetics: 2L break associated with l(2)35Dd.

T(2;3)G24

cytology: T(2;3)35E1-2;89A1-2;92A1-2.

new order:

21 – 35E1|89A2 – 92A1|89A1 – 61;
100 – 92A2|35E2 – 60.

origin: X ray induced.

discoverer: Gelbart.

T(2;3)G40

cytology: T(2;3)34F4-5;91E5-6.

discoverer: Gelbart.

genetics: Broken proximal to *noc*.

T(2;3)gl^{63d}: Translocation (2;3) glass

origin: γ ray induced.

discoverer: Ives, 63d29.

references: 1965, DIS 40: 35.

genetics: Mutant for *gl*.

T(2;3)Gla²: Translocation (2;3) Glazed-2

cytology: T(2;3)27-28;87-88.

origin: X ray induced.

references: The Seattle-La Jolla Drosophila Laboratories, 1971, DIS 47, Suppl.

genetics: Associated with *Gla*².

T(2;3)Gld^{d5}: Translocation (2;3) Glucose dehydrogenase

cytology: T(2;3)?;84C8-D1.

references: Cavener, Otteson, and Kaufman, 1986, Genetics 14: 111-23.

Cavener, Corbett, Cox, and Whitten, 1986, EMBO J. 5: 2939-48.

genetics: Mutant for *Gld*.

molecular biology: Distal breakpoint at about +134 kb on the molecular map of Cavener, Corbett, Cox, and Whitten.

T(2;3)gls: Translocation (2;3) glassy

cytology: T(2;3)47B;91A.

synonym: T(2;3)T2.

references: Robinson and Curtis, 1972, Can. J. Genet. Cytol. 14: 129-37.

genetics: Homozygotes viable and fertile, with abnormal eye. In heterozygotes T(2;3)gls/T(2;3)smg, females are more fertile than males.

T(2;3)Gr: see T(2;3)Pu^{Gr}

T(2;3)GT

origin: γ ray induced.

discoverer: Durrant.

translocation	cytology	genetics
T(2;3)GT3 ^α	28B-C;35B1-2;41;42F;98D	
T(2;3)GT7 ^β	35B3;81;92F1-2	<i>noclosp</i>
T(2;3)GT8	35A1-4;62F3-6 + T(2;3)50B;81	l(2)35Ba <i>noc</i>

translocation	cytology	genetics
T(2;3)GT10	34A1-4;76A5-7	l(2)34Fcl(2)34Fd

^α New order: 21 – 22C|41 – 35B1-2|28B-C – 22C|42F – 60;
61 – 98D|35B1-2 – 28B-C|42F – 41|98D – 100.

^β New order: 21 – 35B3|81 – 61;
60 – 35B3|92F1-2 – 81|92F1-2 – 100.

T(2;3)h: Translocation (2;3) hairy

origin: X ray induced.

genetics: Homozygous lethal.

translocation	cytology	synonym	ref ^α
T(2;3)h ^{m1}	66D9-11;41A		1
T(2;3)h ^{R40} βγ	30B7-C1;66D6-10;73B-C	<i>h</i> ⁺⁴⁰	1, 2, 3
T(2;3)h ^{R47} γδ	23A2-B1;66D6-10 + 34C2-4;98F12-99A1	<i>h</i> ⁺⁴⁷	1, 2, 3

^α 1 = Ingham, Pinchin, Howard, and Ish-Horowitz, 1985, Genetics 111: 463-86; 2 = Jeffery, 1971, DIS 47: 37; 3 = Jeffery, 1979, Genetics 91: 105-25.

^β New order: 21 – 30B7|73B – 66D10|73C – 100;
61 – 66D6|30C1 – 60.

^γ Heterozygotes over *h* variegate for *h* (Jeffery, 1971).

^δ Molecular biology: 66D6 break located at -19.8 to -14.9 on the DNA map of *h* (Howard, Ingham, and Rushlow, 1988, Genes Dev. 2: 1037-46); "+" values to right, "-" values to left. The coordinate system has 0 at the start of transcription (Rushlow).

***T(2;3)h^{100.271}**

cytology: T(2;3)41;66D14-E1.

origin: X ray induced.

discoverer: Alexander.

references: Ward and Alexander, 1957, Genetics 42: 42-54.

genetics: Mutant for *h*.

T(2;3)H: Translocation (2;3) Hilliker

origin: α ray induced.

references: Hilliker and Trusis-Coulter, 1987, Genetics 117: 233-44. Table I includes translocations available for complementation studies; Table II includes translocations that were lost and not available for complementation studies.

Table I

translocation ^α	cytology	genetics (homozygotes)
T(2;3)H1	43F1-2;87D4-13	viable; fertile
T(2;3)H2	38D-E;78B	lethal
T(2;3)H3 ^β	42A6-19;83D5-E1 + ln(3R)84F12-16;98C3-D1	lethal
T(2;3)H4	25B-C;87B4-5	lethal
T(2;3)H5	42B1-4;82C2-D1	lethal
T(2;3)H6	21B2-8;82F8-83A1	lethal
T(2;3)H7	24D2-E;78C	lethal
T(2;3)H8	40-41;98D1-2	viable; fertile
T(2;3)H9	24D1-2;80-81	lethal
T(2;3)H10	44C5-D1;84D3-8	viable; fertile
T(2;3)H11	40-41;80-81	lethal
T(2;3)H12	21E2-F1;83C2-D1	lethal
T(2;3)H13	40-41;83E	lethal
T(2;3)H14	53D3-E1;79E2-5	viable; fertile
T(2;3)H15	47B;92D3-9	viable; fertile
T(2;3)H16	59C5-D1;80	viable; fertile
T(2;3)H17	41;62D6-E2	lethal
T(2;3)H18	51D2-7;96E5-9	semilethal; male sterile; female fertile
T(2;3)H19	43B1-C1;87D3-E1	lethal
T(2;3)H20	21B;74C	viable
T(2;3)H21 ^β	46F5-47A1;87B +	male sterile;

CHROMOSOMES - TRANSLOCATIONS

translocation α	cytology	genetics (homozygotes)	translocation α	cytology	genetics (homozygotes)
T(2;3)H22	<i>In(2LR)34A;53D</i>	female sterile	T(2;3)H88	<i>In(3R)85C;87B</i>	fertile
T(2;3)H23^B	40-41;80;81	viable; fertile	T(2;3)H89^B	40-41;98C2-D2	lethal
T(2;3)H24	25A2-4;71F2-72A2;98D1-2	lethal	T(2;3)H91	30A;35A;81	lethal
	60-25A 71F-61		T(2;3)H93	50C2-4;79D2-4	viable; fertile
	49C-D;81	semilethal;	T(2;3)H94	57B14-C1;81	lethal
		male sterile;	T(2;3)H95	38B;71E	lethal
T(2;3)H25	57A3-B1;87D1-2	female fertile	T(2;3)H96	36C;84B	lethal
T(2;3)H26	42B1-4;66A	viable; fertile	T(2;3)H97	25E3-F1;80	lethal
T(2;3)H27	56F2-5;68C	lethal	T(2;3)H98	49A-B;88F	viable; fertile
T(2;3)H28	40;98F1-2	lethal	T(2;3)H99	30E-F;81	lethal
T(2;3)H29	30C;80-81	lethal	T(2;3)H100	41;99A	lethal
T(2;3)H30	21A;83D2-4	lethal	T(2;3)H102^B	33B;86B	lethal
T(2;3)H31	32D3-5;79F3-6	lethal	T(2;3)H103	21F;2F;62F;63E	lethal
T(2;3)H32^B	44E;72F3-5;	lethal	T(2;3)H104	43F2-44A2;94D4-E7	lethal
	85F-86A;het		T(2;3)H105^B	52D;65B	lethal
T(2;3)H33	41;91F	lethal		34C;71F +	semilethal;
T(2;3)H34	22F2-23A1;81	semilethal;	T(2;3)H106	35B-70C	
		fertile	T(2;3)H110	25E2-F2;80-81	semilethal; fertile
T(2;3)H35	40-41;80-81	viable;	T(2;3)H111	54C;95D	lethal
		male sterile;	T(2;3)H112^B	30A2-7;65C1-2	viable; fertile
		female fertile		29F;83E + 53C;66A +	lethal
T(2;3)H36	43B;83A	lethal	T(2;3)H113	<i>Df(3R)81;96A</i>	
T(2;3)H37	41;98B	semilethal; fertile	T(2;3)H114^B	30A-80	lethal
T(2;3)H38	55F3-56A1;64F	lethal	T(2;3)H116	41F;57B + 57F;het	lethal
T(2;3)H39	40-41;80-81	viable; fertile	T(2;3)H117	42A2-B1;96C2-D1	lethal
T(2;3)H40	40;95F	lethal		23D;93E4-F2	viable;
T(2;3)H41	40-41;80-81	viable; fertile	T(2;3)H118	34C;80	male sterile;
T(2;3)H42	28A;81	lethal	T(2;3)H119	32F2-33A1;82D	female fertile
T(2;3)H43	42A;82F	viable; fertile	T(2;3)H120	40-41;80-81 +	semilethal; fertile
T(2;3)H44	28A;63E	lethal		<i>In(3R)82C-D;90A2-B1</i>	
T(2;3)H45	38B;80-81	semilethal; fertile	T(2;3)H122	36F;80-81	lethal
T(2;3)H46	40-41;67E	lethal	T(2;3)H123	55C4-D1;80	lethal
T(2;3)H47	35A-B;65F	viable; fertile	T(2;3)H124	48C;74B-C	viable; fertile
T(2;3)H48	40-41;80-81	semilethal; fertile	T(2;3)H125	41;75D1-2	lethal
T(2;3)H49	47D4-8;88B	lethal	T(2;3)H126	48A2-B1;99F	lethal
T(2;3)H50	35D;80-81	semilethal; fertile	T(2;3)H127	41;94D1-2	viable; fertile
T(2;3)H51	38A1-2;78A1-2	semilethal; male	T(2;3)H128	53D;88D	lethal
		sterile; female fertile	T(2;3)H129	47A1-2;71C1-2	lethal
T(2;3)H52	40-41;85E5-F1	lethal	T(2;3)H130	42B2-C1;80-81	viable; fertile
T(2;3)H53	36B2-C1;62B8-10	lethal	T(2;3)H131	47A2-B1;84D4-9	lethal
T(2;3)H54	56C;70B	viable; fertile	T(2;3)H132	41;97B6-C2	lethal
T(2;3)H55	59C3-D3;81	lethal	T(2;3)H133	44D5-E1;64E	lethal
T(2;3)H56	45E;87E	lethal	T(2;3)H136	57D;80-81	semilethal;
T(2;3)H58	41;70A	viable; male sterile;			male sterile;
		female fertile	T(2;3)H137	40;84F12-85A1	female fertile
T(2;3)H61	22D;64C	viable; fertile	T(2;3)H139	25E1-2;75C1-2	lethal
T(2;3)H62^B	22F3-23A2;55E;66A8-20	lethal	T(2;3)H140	33B;82C	lethal
T(2;3)H63	58D;72D	lethal	T(2;3)H141	40-41;66B	lethal
T(2;3)H64	40-41;80-81	viable; fertile	T(2;3)H144	23A1-2;99A7-B1	lethal
T(2;3)H65^B	45B;50F;87C	lethal	T(2;3)H147	34C3-6;80	viable; fertile
T(2;3)H66^B	24D3-5;85C;88B	lethal	T(2;3)H148	22D1-2;87D	viable; fertile
T(2;3)H67	35B9-C2;80	lethal	T(2;3)H151	48F;81	viable; fertile
T(2;3)H68	35B-84B	lethal	T(2;3)H152^B	30A2-7;34D;99B5-C1	semilethal;
T(2;3)H70	40-41;92E-F	viable; male fertile;			male sterile;
		female low fertility	T(2;3)H153	37C2-D1;75A5-B2	female fertile
T(2;3)H71^B	41;87A +	semilethal;	T(2;3)H154^B	22A5-B1;25A;78E2-5	lethal
	<i>In(2R)54D;59D</i>	male sterile;	T(2;3)H155^B	60D1-2;93A1-2;98C1-2;	lethal
		female fertile		100D2-E1	
T(2;3)H72	41;65D	viable; fertile			
T(2;3)H73	41;65F	viable; fertile	α	Synonym: <i>T(2;3)1 - T(2;3)155</i> .	
T(2;3)H74	41;93C	lethal	β	New order:	
T(2;3)H75	58F;80-81	lethal		<i>T(2;3)H3 = 21-42A 83D-84F 98C-84F 98C-100;</i>	
T(2;3)H76	414;64E	viable; fertile		<i>60 76A-83D 42A-60C 76A-61.</i>	
T(2;3)H77	33F3-5;82D	viable; fertile		<i>T(2;3)H21 = 21-25A 98D-71F 98D-100;</i>	
T(2;3)H78	48C1-2;61C	viable; sterile		<i>60-25A 71F-61.</i>	
T(2;3)H80	41;87E	lethal		<i>T(2;3)H23 = 21-34A 53D-47A 87B-61;</i>	
T(2;3)H82	45A2-C1;87D3-E1	lethal		<i>60-53D 34A-46F 87B-100.</i>	
T(2;3)H83	27E-F;81	lethal		<i>T(2;3)H32 = 21-44E 72-61;</i>	
T(2;3)H84	36C;67D1-9	viable; fertile		<i>het? 72F-85F 44E-60;</i>	
T(2;3)H85	48F;85C	lethal		<i>het? 85F-100.</i>	
T(2;3)H86	33B;62A	low viability;		<i>T(2;3)H62 = 21-22F 66A-100;</i>	
		male sterile;			
		female fertile			
T(2;3)H87^B	23D;85C;87B	semilethal;			

60-55E|23A-55E|66A-61.
 T(2;3)H65 = 21-45B|87C-100;
 61-87C|50C-45B|50F-60.
 T(2;3)H66 = 21-24D|85C-88B|85C-61;
 60-24D|88B-100.
 T(2;3)H71 = 21-41;
 87A-100;
 60-54D|59D-54D|59D-41|87A-61.
 T(2;3)H87 = 21-23D|61-85C;
 60-23D|87B-85C|87B-100.
 T(2;3)H89 = 21-30A|35A-30|81-61;
 60-35A|81-100.
 T(2;3)H102 = 60-22F|62F-61;
 21A-21F|63E-62F|21F-22F|63-100.
 T(2;3)H105 = 21C-34C|71F-100;
 60-35B|70C-71F|34C-35B|70C-61.
 T(2;3)H112 = 21-29F|83E-81|96A-83E|
 29F-53C|66A-61;
 60-53C|60-53C|66A-81|96A-100.
 T(2;3)H114 = 21-41F|67B-61;
 het|57F-41F|67B-100;
 60-57F|het.
 T(2;3)H152 = 21-30A|99B-61;
 60-34D|30A-34D|99B-100.
 T(2;3)H154 = 21-22A|78E-100;
 60-25A|22B-25A|78E-61.
 T(2;3)H155 = 21-60D|100D-100F;
 60F-60D|93A-98C|100D-98C|83A-61.

Table II

translocation ^α	cytology	genetics (homozygotes)
*T(2;3)H2a	41;100F2-5	lethal
*T(2;3)H4a	50A10-15;90C8-D1	viable
*T(2;3)H5a	41;63E3-8	lethal
*T(2;3)H6a	60D6-9;94A1-3 + In(3R)87D3-10;96F9-11	lethal
*T(2;3)H7a	40-41;57F;70C2-12;90C	lethal
*T(2;3)H8a	36C-D;83C2-4	not determined
*T(2;3)H11a	36C2-E1;80-81	viable
*T(2;3)H16a	56D2-E1;67C2-4	viable
*T(2;3)H18a	31F2-32A1;81	not determined
*T(2;3)H22a	40-41;80-81	lethal
*T(2;3)H23a	40-41;72E2-F1	viable
*T(2;3)H26a	57A2-4;65F2-66A1	lethal
*T(2;3)H28a	41;88C10-E1	viable
*T(2;3)H31a	38D;69F2-70A1	viable
*T(2;3)H33a	40-41;82E2-7	not determined
*T(2;3)H36a	33A1-B12;71F2-72A1	viable
*T(2;3)H38a	50C11-20;80	viable
*T(2;3)H40a	60F1-2;73A;het(?)	lethal
*T(2;3)H42a	38A2-C1;89A1-3;80	viable
*T(2;3)H48a	22A1-2;81	lethal
*T(2;3)H49a	35B2-9;80-81	viable
*T(2;3)H50a	59E2;80-81	not determined
*T(2;3)H51a	56C3-D1;82E7-8	lethal
*T(2;3)H52a	40;99C3-D1	lethal
*T(2;3)H54a	36C;80-81	viable
*T(2;3)H55a	23A;98B-C	lethal
*T(2;3)H57a	51F;61C7-9	lethal
*T(2;3)H61a	23E1-2;64E1-2;het	lethal
*T(2;3)H63a	42A;43F;99D2-4;het	lethal
*T(2;3)H65a	59D;64A12-B2	lethal
*T(2;3)H66a	21D2-E1;80	viable
*T(2;3)H67a	41;99A8-B1	lethal

^α Synonym: T(2;3)LO2 - T(2;3)LO67 ("LD" = lost).

T(2;3)H²⁴: Translocation (2;3) Hairless

cytology: T(2;3)41;92F1-2.
 references: Bang, Hartenstein and Posakony, 1991,
 Development 111: 89-104.
 genetics: Mutant for *H*.

***T(2;3)HK: Translocation (2;3) Half of Krivshenko**

cytology: T(2;3)22A;61A.
 origin: X ray induced in female.
 discoverer: Krivshenko, 56114.
 references: 1959, DIS 33: 95.
 genetics: Only the 3^P2L^D element recovered from the treated oocyte.

T(2;3)Hm: Translocation (2;3) Haltere mimic

cytology: T(2;3)29;32;88F;89E3-4.
 new order:
 21 - 29|88F - 61;
 60 - 32|88F - 89E3|29 - 32|89E4 - 100.
 origin: X ray induced.
 discoverer: Slatis, 49b5.
 references: Lewis, 1982, Embryonic Development: Genetic Aspects (Burger and Weber, eds.). Alan Liss, Inc., New York. Progress in Clin. Biol. Res. 85A: 269-89.
 genetics: Associated with *Cbx^{Hm}*.

T(2;3)Hn: Translocation (2;3) Henna

cytology: T(2;3)53E-54A;77A;94F;96A (Lewis).
 new order:
 21 - 53E|77A - 61;
 60 - 54A|94F - 96A|77A - 94F|96A - 100.
 Tentative.
 origin: X ray induced.
 discoverer: Van Atta, 30k.
 references: 1932, Am. Nat. 66: 93-95.
 1932, Genetics 17: 637-59.
 genetics: Separable from *Hn*, which is associated with *Df(3L)Hn = Df(3L)66A;66B*.

T(2;3)ho5: see T(2;3)dpp³

T(2;3)HR

origin: X ray induced.
 genetics: Crossover suppressor between *h* and *ri*.

translocation	cytology	ref ^α	homozygous viability
T(2;3)HR17	26A;69F	1	-
T(2;3)HR26	28A;64D	1	-
T(2;3)HR28	25A;91D + 42A;71A-B	1	-
T(2;3)HR30^β	34E1-3;70C1-2	1, 2, 3	+

^α 1 = Ashburner, 1972, DIS 49: 34; 2 = Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91; 3 = Craymer, 1981, Genetics 99: 75-97.
^β Also fertile and wild type.

T(2;3)I: Translocation (2;3) Ising

discoverer: Ising.
 references: Ashburner.

translocation	cytology
T(2;3)I805	40F;75AB + In(3R)89;94
T(2;3)I806	55E;66A
T(2;3)I810	53F;95E-F

T(2;3)iab4⁴⁵: Translocation (2;3) infraabdominal

cytology: T(2;3)32;41;89E. Complex.
 origin: X ray induced.
 discoverer: R.H. Baker.
 genetics: Mutant for *iab4*.

molecular biology: 89E breakpoint 78.5-82 kb to the right of right breakpoint of *In(3R)Cbx^{rv1}*.

T(2;3)iab6

origin: X ray induced.
genetics: Mutant for *iab6*.

translocation	cytology	discoverer	molecular biology ^α
T(2;3)iab6⁷⁵	30A-B;31D-E; 59F;89E;89F	Baker	103--108 kb
T(2;3)iab6¹⁰⁵	60C;89E	Lewis	108-111 kb
T(2;3)iab6^{C1}	60B;81;89E;	Crosby	108-111 kb

^α Coordinates of 89E breakpoints in kilobases to the right of the right breakpoint of *In(3R)Cbx^{rv1}*.

T(2;3)iab8^{rv}

genetics: Revertants of *iab8* in the *BXC*.

translocation	cytology
T(2;3)iab8^{rv100}	37E-38A;89E
T(2;3)iab8^{rv185}	50D;89E

T(2;3)iab9⁶⁵

cytology: T(2;3)41;89E.
origin: X ray induced.
discoverer: R.H. Baker.
references: Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.
Sánchez-Herrero, Vernós, Marco, and Morata, 1985, Nature 313: 108-13.
Tiong, Bone, and Whittle, 1985, Mol. Gen. Genet. 200: 335-42.

genetics: Mutant for *iab9*.
molecular biology: 89E breakpoint 163-166.5 kb to the right of the right breakpoint of *In(3R)Cbx^{rv1}*.

T(2;3)IC67-98

cytology: T(2;3)24E-F;91D.
origin: X ray induced.
references: Brosseau, 1969, DIS 44: 45.
genetics: Homozygous lethal.

T(2;3)in⁶⁰ⁱ²: Translocation (2;3) inturned

cytology: T(2;3)62D-F;77B;80C. Complex translocation with inversion(s) in 3L.
references: Arajarvi and Hannah-Alava, 1969, DIS 44: 73-74.
genetics: Associated with *in⁶⁰ⁱ²*.

T(2;3)Ir: Translocation (2;3) Irish

cytology: T(2;3)55D-E;98F.
origin: γ ray induced.
discoverer: Irish.
other information: Induced along with *Df(2L)dpp43*.

T(2;3)JHT1

cytology: T(2;3)34C;96F.
origin: Spontaneous in *JH* chromosome line during successive backcross programs.
references: Yamaguchi and Mukai, 1974, Genetics 78: 1209-21.

T(2;3)JHT2

cytology: T(2;3)56F;97B.
origin: Spontaneous in *JH* chromosome line during successive backcross programs.

references: Yamaguchi and Mukai, 1974, Genetics 78: 1209-21.

T(2;3)L141

origin: γ ray induced in *T(Y;2)L141/+* males.
discoverer: Lyttle.

translocation	cytology
T(2;3)L141-2	36B;61B
T(2;3)L141-3	40-41;80-81
T(2;3)L141-4	35D;79F
T(2;3)L141-5	40-41;84F-85A
T(2;3)L141-6	40-41;80-81
T(2;3)L141-7	42A;68D-E
T(2;3)L141-8	40-41;80-81
T(2;3)L141-9	33C-D;83D-E
T(2;3)L141-10	40-41;80-81
T(2;3)L141-11	40-41;80-81
T(2;3)L141-12	53C;64B
T(2;3)L141-13	52D;87F
T(2;3)L141-14	27E;86C
T(2;3)L141-15	35B;80-81
T(2;3)L141-17	23C;70F-71A
T(2;3)L141-18	51A;80C
T(2;3)L141-19	40-41;85D
T(2;3)L141-21	57E;94E
T(2;3)L141-22	50C5-10;69E
T(2;3)L141-23	35B;80-81
T(2;3)L141-24	22B3-6;98E
T(2;3)L141-25	40-41;62F
T(2;3)L141-26	34D;87D-E
T(2;3)L141-28	43D5;75C4
T(2;3)L141-29	58E;64B-C
T(2;3)L141-30	47E-F;80-81
T(2;3)L141-31	57A;87C
T(2;3)L141-32	58D;61E
T(2;3)L141-33	40-41;97D
T(2;3)L141-34	55B;76B
T(2;3)L141-36	37B;80-81
T(2;3)L141-37	40-41;80-81
T(2;3)L141-38	45A;65E-F
T(2;3)L141-39	26B-C;98F
T(2;3)L141-40	53E-F;82C-D
T(2;3)L141-41	41;80
T(2;3)L141-43	40-41;80-81
T(2;3)L141-44	40;61E
T(2;3)L141-45	57D2-4;78A
T(2;3)L141-46	40-41;86E
T(2;3)L141-47	60C-D;64E
T(2;3)L141-48	38D;80-81
T(2;3)L141-49	24F;96E
T(2;3)L141-50	41;98F
T(2;3)L141-51	21C5;97D3
T(2;3)L141-52	40-41;93A
T(2;3)L141-53	24F;61E-F

T(2;3)LD28

cytology: T(2;3)52A-C;81F.
discoverer: De Jongh.

T(2;3)lt^m Translocation

origin: X ray induced.
discoverer: Hessler, 1957.
references: 1958, Genetics 43: 395-403.
genetics: Variegated for *lt*.

translocation	cytology
*T(2;3)lt^{m1}	40B-F;63E-F
*T(2;3)lt^{m4}	40B-F;67E
*T(2;3)lt^{m5}	40B-F;98C
*T(2;3)lt^{m6} α	26E-F;40B-F;96E
T(2;3)lt^{m7}	40B-F;100F
*T(2;3)lt^{m8}	40B-F;92B
*T(2;3)lt^{m10}	40B-F;64E

translocation	cytology
*T(2;3)lt m11	40B-F;96F
*T(2;3)lt m13	40B-F;64F
*T(2;3)lt m14	40B-F;95F
*T(2;3)lt m15	40B-F;92E
*T(2;3)lt m17	40B-F;95C-D
*T(2;3)lt m18	40B-F;98A
*T(2;3)lt m19	40B-F;94B
*T(2;3)lt m21	40B-F;93D
*T(2;3)lt m23	40B-F;62F
*T(2;3)lt m24 β	40B-F;59F;75C
*T(2;3)lt m27	40B-F;88E-F
*T(2;3)lt m28	40B-F;97E
T(2;3)lt m29	40B-F;99F
*T(2;3)lt m30	40B-F;99C
*T(2;3)lt m32	40B-F;97A
*T(2;3)lt m34	40B-F;61B
*T(2;3)lt m35	40B-F;64C

α New order: 21 – 26E|40B – 26F|96E – 61;
60 – 40F|96E – 100.
β New order: 21 – 40B|75C – 100;
60 – 59F|40F – 59F|75C – 61.

T(2;3)lt^{m100}

cytology: T(2;3)40;97F.
origin: X ray induced.
references: Baker and Rein, 1962, Genetics 47: 1399-1407.
genetics: Variegated for *lt*. Homozygous lethal.

T(2;3)lt^{x13}

cytology: T(2;3)40F;97D2.
discoverer: Wakimoto.
references: James, Eissenberg, Craig, Dietrich, Hobson, and Elgin, 1989, Eur. J. Cell Biol. 50: 170-80.

T(2;3)M^{V54d}: see T(2;3)MV

***T(2;3)Me²**

origin: X ray induced.
discoverer: Moore, 1929.
references: Glass, 1933, J. Genet. 28: 69-112.
genetics: Break in 2L near centromere. Mutant for *Me*.

***T(2;3)Me^{So}: Translocation (2;3) Moiré of Sytko**

discoverer: Sytko.
references: Agol, 1936, DIS 5: 7.
genetics: Breaks reportedly in 2R and 3R, yet mutant for *Me* in 3L.

T(2;3)Met: Translocation (2;3) Metatarsi irregular

origin: X ray induced.
discoverer: Jonsson, 56a10.
references: 1956, DIS 30: 73.
genetics: Associated with *Met*.

T(2;3)Mg: Translocation (2;3) Mglinetz

translocation	cytology	origin ^α	ref ^β
T(2;3)Mg15	31B;64A	1	2
T(2;3)Mg22	40;61F	1	3
T(2;3)Mg23	40;96A	1	3
T(2;3)Mg24	40;73F	1	3
T(2;3)Mg51	47D;93B	1	3
T(2;3)Mg52	25A;81F	1	3
T(2;3)Mg53	51F;94E	1	3
T(2;3)Mg54	60A;81F	1	3
T(2;3)Mg64	59D;68A	1	1
T(2;3)Mg166	23C;75C	1	1

translocation	cytology	origin ^α	ref ^β
T(2;3)Mg167	44A;87A	1	1
T(2;3)Mg168	55F;76E	1	1
T(2;3)Mg169	56E;91F	1	1
T(2;3)Mg170	48B;67C	1	1
T(2;3)Mg171	24F;74B	1	1
T(2;3)Mg172	32C;77B	1	1
T(2;3)Mg173	34A;82F	1	1
T(2;3)Mg174	53D;97B	1	1
T(2;3)Mg175	44F;99B	1	1
T(2;3)Mg176	31B;94D	1	1
T(2;3)Mg177	26B;71C	1	1
T(2;3)Mg178	50C;82A	1	1
T(2;3)Mg179	34A;79B	1	1
T(2;3)Mg180	28C;95F	1	1
T(2;3)Mg181	56E;78F	1	1
T(2;3)Mg182	45F;74B	1	1
T(2;3)Mg183	27C;62A	2	1
T(2;3)Mg184	28F;94A	2	1
T(2;3)Mg185	31B;87F	2	1
T(2;3)Mg186	43A;85B	2	1
T(2;3)Mg187	49E;92E	2	1
T(2;3)Mg188	35A;71C	2	1
T(2;3)Mg189	53C;88B	2	1
T(2;3)Mg190	39F;89E	2	1
T(2;3)Mg191	57D;99B	2	1
T(2;3)Mg192	26B;98F	2	1
T(2;3)Mg193	37F;84A	2	1
T(2;3)Mg194	55C;70C	2	1
T(2;3)Mg195	59D;83D	2	1
T(2;3)Mg196	50E;72C	2	1

α 1 = γ ray induced; 2 = ³²P feeding.
β 1 = Mglinetz, 1968, Genetika (Moscow) 4(8): 81-86; 2 = Mglinetz, 1971, Genetika (Moscow) 7(8): 108-14; 3 = Mglinetz, 1972, Genetika (Moscow) 8(2): 82-91.

T(2;3)ML

origin: X ray induced.
references: Mukhina and Zhimulev, 1980, DIS 55: 209.

translocation	cytology	homozygous viability
T(2;3)ML2	53A1;66C1	–
T(2;3)ML3	34C6-7;70A4-5	–
T(2;3)ML4	40-41;80-81	–
T(2;3)ML5	41A;62B	–
T(2;3)ML8	40-41;80-81	–
T(2;3)ML9	44C-D;64C	–
T(2;3)ML14	40-41;80-81	–
T(2;3)ML76	32E;70A	–
T(2;3)ML122	21F;82E	–
T(2;3)ML192 ^α	41;97A + 52B;86D	–
T(2;3)ML193	52E1;80-81 + In(3R)93E;100F	–
T(2;3)ML225	53C;96C	+
T(2;3)ML270	40-41;80-81	–
T(2;3)ML307 ^β	50D1;79A	–
T(2;3)ML348	35C;70A	–
T(2;3)ML393	40B;63A	–
T(2;3)ML405	57A;92E	–
T(2;3)ML436	40-41;80-81	–
T(2;3)ML443 ^γ	24D;87C1	–
T(2;3)ML457	40-41;80-81 + Df(3R)87A;87C	–
T(2;3)ML460	39E;75C	+
T(2;3)ML464	39E;61D	–
T(2;3)ML466	40-41;80-81	–
T(2;3)ML472	32E;96F	–
T(2;3)ML474	35B3-5;94D5-13	–
T(2;3)ML478	40-41;80-81	–
T(2;3)ML484	40-41;80-81	–
T(2;3)ML486	39E;90D	+
T(2;3)ML488	40-41;80-81	–
T(2;3)ML490	27E;92B1	–
T(2;3)ML491	40-41;80-81	+

translocation	cytology	homozygous viability
<i>T(2;3)ML494</i>	60E6;99C1	-
<i>T(2;3)ML495</i>	22B1;81F	-
<i>T(2;3)ML498</i>	35A;87F	-
<i>T(2;3)ML499</i>	40-41;80-81	-
<i>T(2;3)ML501</i>	50A;64E	+
<i>T(2;3)ML502</i>	40-41;80-81	-
<i>T(2;3)ML505</i>	21B;97C	-
<i>T(2;3)ML506</i> ^δ	55C;85C	-
<i>T(2;3)ML507</i>	40-41;80-81	-
<i>T(2;3)ML508</i>	40-41;80-81	-
<i>T(2;3)ML509</i>	40-41;80-81	-
<i>T(2;3)ML510</i>	40-41;80-81	-
<i>T(2;3)ML511</i>	40-41;80-81	-
<i>T(2;3)ML512</i>	40-41;80-81	-
<i>T(2;3)ML514</i>	40-41;80-81	-

α New order: 21 - 41|97A - 86D|52B - 60;
61 - 86D|52B - 41|97A - 100.
β L4 interrupted.
γ Extra veins.
δ L2 and L4 veins thin.

****T(2;3)MO***

origin: Spontaneous.
discoverer: Imaizumi, 59a.
references: 1962, *Cytologia* 27: 212-28.
genetics: Breaks between *cn* and *vg* in 2R and between *st* and *ss* in 3R.

T(2;3)Mot-K: Translocation (2;3) Mottled of Krivshenko

cytology: *T(2;3)41;60D;80-81*; breakpoint in chromosome 3 with respect to centromere not determined; association of arms therefore ambiguous.
new order:
21 - 41|80 - 61;
60F - 60D|41 - 60D|80 - 100.
Tentative.
origin: X ray induced.
discoverer: Krivshenko, 54c25.
references: 1954, *DIS* 28: 75.
1955, *DIS* 29: 76.
genetics: Associated with *Mot-K*. Homozygous lethal.

T(2;3)MP: Translocation (2;3) Mamon Petrukhina

origin: X ray induced.
references: Mamon, Petrukhina, Rasheva, and Vatti, 1977, *Genetika (Moscow)* 13(8): 1378-86.

translocation	cytology
<i>T(2;3)MP3</i>	56C;89A
<i>T(2;3)MP4</i>	56-57;62E
<i>T(2;3)MP5</i>	28B-C;98B-C
<i>T(2;3)MP6</i>	38B;77D-E
<i>T(2;3)MP9</i>	40-41;80-81
<i>T(2;3)MP10</i>	26D-E;66A-B
<i>T(2;3)MP15</i>	40-41;80-81
<i>T(2;3)MP19</i>	43B-C;96C
<i>T(2;3)MP19a</i>	31E-F;69D-E
<i>T(2;3)MP22</i>	33F;65C
<i>T(2;3)MP26</i>	49B-C;92D-E
<i>T(2;3)MP28</i>	50A;85D-F
<i>T(2;3)MP35</i>	38A;84C-D
<i>T(2;3)MP37</i>	50B-C;66D-E
<i>T(2;3)MP41</i>	40-41;80-81
<i>T(2;3)MP42</i>	40-41;80-81
<i>T(2;3)MP44</i>	52F;96B
<i>T(2;3)MP46</i>	28A-B;78D
<i>T(2;3)MP47</i>	40-41;80-81

translocation	cytology
<i>T(2;3)MP48</i>	48C-D;80-81
<i>T(2;3)MP55</i>	21B;61A
<i>T(2;3)MP63</i>	52D-E;95-96
<i>T(2;3)MP64</i>	40-41;70A
<i>T(2;3)MP65</i>	30-31;79A
<i>T(2;3)MP69</i>	23D;87A-B
<i>T(2;3)MP72</i>	33A-B;80-81
<i>T(2;3)MP74</i>	41-42;98
<i>T(2;3)MP75</i>	50A-B;100C
<i>T(2;3)MP78</i>	40-41;80-81
<i>T(2;3)MP80</i>	53B;97C-D
<i>T(2;3)MP81</i>	40-41;80-81
<i>T(2;3)MP84</i>	50-51;97-98
<i>T(2;3)MP85</i>	52;86-87
<i>T(2;3)MP87</i>	27A-B;96C-D
<i>T(2;3)MP100</i>	39C;84A-B
<i>T(2;3)MP101</i>	40-41;80-81
<i>T(2;3)MP104</i>	40-41;80-81
<i>T(2;3)MP108</i>	40-41;80-81
<i>T(2;3)MP109</i>	50A;81-82
<i>T(2;3)MP110</i>	35D-E;70-71
<i>T(2;3)MP111</i>	40-41;80-81
<i>T(2;3)MP112</i>	40C-F;98D-F
<i>T(2;3)MP113</i>	40-41;80-81
<i>T(2;3)MP114</i>	49B;65-66
<i>T(2;3)MP115</i>	40-41;80-81

T(2;3)Mpe: Translocation (2;3) Monoplane

cytology: *T(2;3)35B2-3;86C1-2* (Ashburner).
origin: X ray induced.
discoverer: Shelton.
references: Hughes and Shelton, 1980, *DIS* 55: 204-05.
Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, *DIS* 56: 186-91.
Chia, Karp, McGill, and Ashburner, 1985, *J. Mol. Biol.* 186: 689-706.
McGill, Chia, Karp, and Ashburner, 1988, *Genetics* 119: 647-61.
genetics: Associated with dominant *Mpe* phenotype and recessive *osp* allele. Also shows a dominant leg phenotype (thickening of the tibial-tarsal joint of the metathoracic leg). Phenotypes of leg and outstretched-wing associated with 2^P3^D element of the translocation. Lethal homozygous or with *Df(3R)cu40* and *Df(3R)by77*. Heterozygotes show reduced viability and partial sterility (Hughes and Shelton, 1980). *T(2;3)Mpe* heterozygous with a deletion of the entire *noc* region is *noc*⁺ (McGill *et al.*, 1988).

molecular biology: 35B2-3 breakpoint at -103 to -106.5 bp in the molecular map of the wild-type *noc-Adh* region (McGill *et al.*, 1988) (coordinate 0 on *EcoR1* restriction site 1321 bp to the left of the start of transcription of the larval *Adh* transcript, "+" values to the right, "-" values to the left).

T(2;3)Msc^{T3}: Translocation (2;3) Multiple sex comb

origin: γ ray induced.
discoverer: Tiong.
references: Kennison and Russell, 1987, *Genetics* 116: 75-86.
genetics: Mutant for *Msc*.

***T(2;3)MV: Translocation (2;3) Variegated of Mickey**

cytology: T(2;3)43E;75C.
origin: γ ray induced.
discoverer: Mickey, 54d.
synonym: T(2;3)M^{V54d}.
references: 1963, DIS 38: 30.
genetics: Eye color variegated, more prominent in male.

T(2;3)N

origin: X ray induced.

translocation	cytology	ref ^α	genetics of homozygotes
T(2;3)N1-14	32C2-D1;99F6-100A1	2	viable, female sterile
T(2;3)N1-15	56A2-B1;70A-B	2	viable
T(2;3)N1-17	28D4-E1;72E2-73A1	2	poorly viable
T(2;3)N1-18	38B;77A2-B1	2	lethal
*T(2;3)N1-20	23E;86E2-7	2	poorly viable
T(2;3)N1-23	40-41;80-81	2	viable, sterile
T(2;3)N1-25	36B-D;84D	2	lethal
*T(2;3)N2-28	36E-F;80B-C	2	lethal
T(2;3)N2-29 ^β	40-41;80-81	1, 2, 3, 4	viable, sterile
T(2;3)N2-32 ^γ	48B;71C1-2;89C	2	lethal
T(2;3)N2-33	23C-D;90C-D	2	lethal
*T(2;3)N2-34 ^γ	37D-E;65;70 + Dp(3L)65;70	2	dominant female sterile
T(2;3)N2-41 ^γ	23A2-B3;24F-25A1;96A23-B1	2	poorly viable
T(2;3)N2-46 ^δ	40-41;80-81	1, 2, 3, 4	viable, fertile
T(2;3)N2-48	57E2-F1;98C	2	lethal

^α 1 = Puro, 1973, Hereditas 75: 140-43; 2 = Puro, 1982, DIS 58: 205-08; 3 = Puro, 1985, Genet. Res. 46: 287-307; 4 = Puro and Kiiskilä, 1982, DIS 58: 125-26.

^β Synonym: T(2;3)FM29.

^γ New order:

T(2;3)N2-32 = 21 - 48B|71C1 - 61;
 60 - 48C|89C2 - 71C2|89D - 100.
 T(2;3)N2-34 = 21 - 37D|65 - 100;
 61 - 69|37E - 60.
 T(2;3)N2-41 = 21 - 23A2|96A23 - 61;
 60 - 25A1|23B3 - 24F|96B1 - 100.

^δ Synonym: T(2;3)FM46.

T(2;3)net¹⁸: Translocation (2;3) net

cytology: T(2;3)27E-F;65A + In(2LR)net¹⁸ = In(2LR)21B3-4;42C-D1; small deficiency at distal end of 2L.

origin: X ray induced.

references: Golubovsky, Kulakov and Korochkina, 1978, Genetika 14(2): 294-305.

Korochkina and Golubovsky, 1978, DIS 53: 197-200.

genetics: Deficient for *net* and *Igl* but not *al*. Homozygous lethal and Minute.

***T(2;3)Nu: Translocation (2;3) Nude**

cytology: T(2;3)24;36-37;39-40;73-74;75-76;77-78;81-82;85-86;89-90.

origin: X ray induced.

discoverer: Sutton, 41a27.

references: CP627.

genetics: Associated with *Nu*. Homozygous lethal.

T(2;3)odd^{3,29}: see Tp(2;3)odd^{3,29}

T(2;3)osp⁹⁰: Translocation (2;3) outspread

cytology: T(2;3)35B3-4;89B9-11.

origin: γ ray induced.

discoverer: Detwiler.

references: Chia, Karp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706.

genetics: Associated with *osp*⁹⁰.

molecular biology: Breakpoint on 2 between 14 and 18 kilobases to the right of the *EcoRI* restriction site immediately distal to *Adh* (Chia *et al.*).

T(2;3)p⁴: Translocation (2;3) pink

cytology: T(2;3)34D;84D6-10;85A1-2. Deficient for 84D6-10 to 85A1-2.

origin: X ray induced.

references: Kempthues, Raff, Raff, and Kaufman, 1980, Cell 21: 445-51.

Kempthues, Raff, and Kaufman, 1983, Genetics 105: 345-56.

genetics: Deficient for *p*.

T(2;3)p^{XT126}

cytology: T(2;3)44F;85A.

origin: X ray induced.

references: Lehmann and Nüsslein-Volhard, 1987, Wilhelm Roux's Arch. Dev. Biol. 119: 402-07.

genetics: Mutant for *p*.

T(2;3)P: Translocation (2;3) Pasadena

origin: X ray induced.

discoverer: E.B. Lewis.

translocation	cytology	ref ^α
T(2;3)P3	40;88B	4
T(2;3)P8 ^β	48C1-2;84D	2
T(2;3)P15	60E;86C-D	1
T(2;3)P23 ^γ	56F;81F	5
T(2;3)P49	57D;81F	4
T(2;3)P58	41;89E	4
T(2;3)P71	60E;81F	2
T(2;3)P89	36-39;86E11	3

^α 1 = Craymer; 2 = Craymer, 1984, Genetics 108: 573-87; 3 = Ellgaard and Brosseau, 1969, Genetics 62: 337-41; 4 = Holm, 1976, The Genetics and Biology of *Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1b, pp. 529-61; 5 = Lewis, 1963, Am. Zool. 3: 33-56.

^β Induced with In(3L)63A-C;78F (E.B. Lewis).

^γ T(2;3)P23, *Ubx/bx*^{34e} heterozygote shows transvection effect.

T(2;3)pb³: Translocation (2;3) proboscipedia

cytology: T(2;3)35B2-3;83E2-8;89A9-10 + T(2;3)50C14;80 (Ashburner).

new order:

21 - 35B2|83E8 - 89A9|83E2 - 80|50C14 - 60;
 61 - 80|50C14 - 35B3|89A10 - 100.

origin: X ray induced.

references: Kaufman, 1978, Genetics 90: 579-96.

Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

Chia, Karp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706.

genetics: Homozygous lethal. Associated with *osp*^{pb3}, a viable recessive *osp* allele at 35B (Ashburner *et al.*, 1981).

molecular biology: Breakpoint proximal to *Adh* by molecular criteria (McGill).

T(2;3)Pcl Translocation (2;3) Polycomblike

cytology: T(2;3)55A;80 (Kennison).

origin: γ ray induced in a *mwh iab9^{uh3}* stock.

discoverer: Tiong.

genetics: Associated with *Pcl*. Breakpoint in 3L proximal to *ri*⁺.

T(2;3)prd^{2,27}: see *Tp(2;3)prd*^{2,27}

***T(2;3)Pu: Translocation (2;3) Punch**

cytology: *T(2;3)41A;70D-E + T(2;3)57B5-C1;79F.*

new order:

21 – 41A|70E – 79F|57C1 – 60;

61 – 70D|41A – 57B5|79F – 100.

Tentative.

origin: X ray induced.

discoverer: Oliver, 28k4.

references: Muller, 1930, J. Genet. 22: 326.

Oliver, 1932, Z. Indukt. Abstamm. Vererbungsl. 61: 484.

genetics: Associated with *Pu*. Homozygous lethal.

T(2;3)Pu^{Gr}: Translocation (2;3) Punch-Grape

cytology: *T(2;3)57C;81F* (Lewis, 1956, DIS 30: 130).

origin: X ray induced.

discoverer: Muller, 291.

synonym: *T(2;3)Gr: Translocation (2;3) Grape;*

T(2;3)p^{Gr}: Translocation (2;3) pink-Grape.

references: Glass, 1933, J. Genet. 28: 69-112.

1934, Am. Nat. 68: 107-14.

Mackay and O'Donnell, 1983, Genetics 105: 35-53.

genetics: Mutant for *Pu*, showing dominant variegated eye color. Homozygous lethal.

T(2;3)Pu^{H16}

cytology: *T(2;3)57C3-9;63B.*

origin: γ ray induced.

references: O'Donnell, Boswell, Reynolds, and Mackay, 1989, Genetics 121: 273-80.

genetics: Mutant for *Pu*.

***T(2;3)Pu^{rv}: Translocation (2;3) Punch-reverted**

cytology: *T(2;3)40F-41A;70D-E + T(2;3)57B5-C1;79F.*

new order:

21 – 40F|70E – 79F|57B5 – 41A1|70D – 61;

60 – 57C1|79F – 100.

Tentative.

origin: X-ray-induced derivative of *T(2;3)Pu* = *T(2;3)40F-41A;70D-E + T(2;3)57B5-C1;79F.*

discoverer: Oliver, 32127.

references: 1939, Genetics 24: 82.

1941, Proc. Intern. Congr. Genet., 7th., p. 228.

genetics: Partial reversal of *Pu*. Homozygous lethal.

T(2;3)Pu^W: Translocation (2;3) Punch-Wine

cytology: *T(2;3)57C4-6;80.*

origin: X ray induced.

discoverer: E.B. Lewis, 55h.

references: Mackay and O'Donnell, 1983, Genetics 105: 35-53.

genetics: Mutant for *Pu*, showing dominant variegated eye color. Homozygous lethal.

T(2;3)RBR37

cytology: *T(2;3)26A;88F.*

origin: Spontaneous [accumulated on second chromosome lines during repeated backcrossing of *In(2LR)bw^{VI}/+* males to *In(2LR)bw^{VI}/SM1* females].

references: Yamaguchi, Cardellino, and Mukai, 1976, Genetics 83: 409-22.

T(2;3)RBR77

cytology: *T(2;3)33B;99F.*

origin: Spontaneous [accumulated on second chromosome lines during repeated backcrossing of *In(2LR)bw^{VI}/+* males to *In(2LR)bw^{VI}/SM1* females].

references: Yamaguchi, Cardellino, and Mukai, 1976, Genetics 83: 409-22.

T(2;3)rg35

cytology: *T(2;3)27E-F;62C2-D1.*

origin: γ ray induced. Sliter, Henrioh, Tucker, and Gilbert, 1989, Genetics 123: 327-36.

T(2;3)ri^{60b2}: Translocation (2;3) radius incompletus

cytology: *T(2;3)?;77E3-78A1.*

origin: X ray induced.

references: Arajarvi and Hannah-Alava, 1969, DIS 44: 73-74.

genetics: Mutant for *ri*.

T(2;3)rn: Translocation (2;3) rotund

cytology: *T(2;3)40-41;80-81;84D8-9* (Duncan and Kaufman, 1978; B.S. Baker). Configuration is *2L-3L;2R-3R* (Puro and Kiiskilä, 1982).

new order:

21 – 40|80 – 61;

60 – 41|84D8 – 81F|84D9 – 100.

Tentative.

origin: Probably X ray induced.

discoverer: Glass, 1929.

references: 1934, DIS 2: 8.

Muller, 1953, DIS 27: 106-7.

Carlson, 1956, DIS 30: 109.

Duncan and Kaufman, 1975, Genetics 80: 733-52.

Puro and Kiiskilä, 1982, DIS 80: 125-26.

genetics: Mutant for *rn*. Homozygous viable and sterile in both sexes. About 10% of the progeny of parents heterozygous for *T(2;3)rn* and chromosome 2 inversions are nondisjunctional for chromosome 2 (Muller, 1953).

T(2;3)Rpl: see *T(2;3)ftz^{Rpl}*

T(2;3)RY2

origin: X ray induced.

discoverer: Simpson.

genetics: Deficient for *sna*.

***T(2;3)S^L: Translocation (2;3) Star of Lewis**

cytology: *T(2;3)21D;81;88D* (Craymer, 1984).

new order:

21A – 21D|81F – 88D|81F – 61;

60 – 21D|88D – 100.

Tentative.

origin: X ray induced.

discoverer: E.B. Lewis, 1940.

references: 1945, Genetics 30: 137-66.

Craymer, 1984, Genetics 108: 573-87.

genetics: Mutant for *S*.

T(2;3)S^M: Translocation (2;3) Star of Muller

cytology: *T(2;3)21E2-3;79D2-E1* superimposed on *In(2L)22D1-2;33F5-34A1 + In(2R)42A2-3;58A4-B1.*

origin: X ray induced in *In(2L)Cy + In(2R)Cy*.

discoverer: Muller, 1928.

references: Painter and Muller, 1929, J. Heredity

20: 287-98.

Muller, 1930, *J. Genet.* 22: 335-57.

Morgan, Bridges, and Schultz, 1936, *Year Book - Carnegie Inst. Washington* 35: 293.

genetics: Mutant for *S*; also carries *Cy*.

T(2;3)S9

cytology: *T(2;3)40-41;80-81*.

origin: X ray induced.

references: Sandler, Lindsley, Nicoletti, and Trippa, 1968, *Genetics* 60: 525-58.

genetics: Homozygous lethal.

****T(2;3)Sa: Translocation (2;3) Salmon***

origin: X ray induced.

discoverer: Van Atta, 30k1.

references: 1932, *Am. Nat.* 66: 93-95.

1932, *Genetics* 17: 637-59.

genetics: Associated with *Sa*. Homozygous lethal. Break in *2L* between *pr* and centromere and in *3L* near centromere.

T(2;3)Sb^V: Translocation (2;3) Stubble-Variigated

cytology: *T(2;3)41A-C;88;89B* superimposed on *In(3R)93D7-E1;98F2-6*. *In(3LR)P35 = In(3LR)65;83D-E* induced simultaneously but was separated from it by recombination.

new order:

21 – 41A|89B – 93D7|98F2 – 93E1|98F6 – 100;

61A – 88|89B – 88|41C – 60.

origin: X ray induced in *In(3R)Mo*, *Sb sr*.

discoverer: E.B. Lewis, 1948.

references: 1956, *DIS* 30: 76-77.

genetics: Variates for phenotype of deficiency for *Sb*, which is normal.

T(2;3)sbd⁴⁷: Translocation (2;3) stubbloid

cytology: *T(2;3)41;89B10-12*.

origin: X ray induced.

references: Spillman and Nöthinger, 1978, *DIS* 53: 164-65.

genetics: Associated with *sbd⁴⁷*. Homozygous lethal.

T(2;3)sbd¹⁰⁶

cytology: *T(2;3)22E;89B*.

origin: X ray induced.

discoverer: E.B. Lewis.

****T(2;3)Scar: Translocation (2;3) Scarred***

cytology: *T(2;3)27E;95A + In(3R)91F;96A*.

origin: X ray induced.

discoverer: Yu, 48h.

references: 1949, *DIS* 23: 65.

genetics: Associated with *Scar*.

T(2;3)Sco^{rv7}: Translocation (2;3) scutoid-revertant

cytology: *T(2;3)35D1-2;93F11-94A4* superimposed on *Tp(2;2)Sco*.

new order:

21 – 35A4|(35C2 – 35C5)|35B4 – 35C1|

35B3 – 35B1|93F11 – 61;

60 – 35D2|93A4 – 100.

origin: X ray induced.

discoverer: Ashburner.

references: Woodruff and Ashburner, 1979, *Genetics*

92: 117-32.

Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, *DIS* 55: 193-95.

Ashburner, Detwiler, Tsubota, and Woodruff, 1983, *Genetics* 104: 405-31.

Ashburner and Harrington, 1984, *Chromosoma* 89: 329-37.

genetics: Homozygous lethal. Revertant for *Sco*.

T(2;3)Sco^{rv13}

cytology: *T(2;3)35D1-2;71B1-2;81*.

new order:

21 – 35A4|(35C2 – 35C5)|35B4 – 35C1|35B3 – 35B1|

71B2 – 81|71B1 – 61;

60 – 35D2|81 – 100.

origin: X ray induced.

discoverer: Ashburner.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, *DIS* 55: 193-95.

Ashburner, Detwiler, Tsubota, and Woodruff, 1983, *Genetics* 104: 405-31.

Ashburner and Harrington, 1984, *Chromosoma* 89: 329-37.

genetics: Homozygous lethal. *2L* breakpoint maps between transposed *noc* allele and *sna*.

molecular biology: 35D1--2 breakpoint mapped to the DNA (McGill, Chia, Karp, and Ashburner, 1988, *Genetics* 119: 647-61).

T(2;3)Scr^{T3}: Translocation (2;3) Sex combs reduced

cytology: Complex with breakpoints in at least *25C-D;84B1-2;92A* (Kennison).

origin: γ ray induced in a *mwh iab9^{uh3}* stock.

discoverer: Tiong.

genetics: *Dfd⁺ Scr⁻ ftz⁺ Antp⁺*; associated with *Scr^{T3}*.

T(2;3)Scx^{Wrv1}: Translocation (2;3) sex combs extra revertant

cytology: *T(2;3)58F1-2;84B1-2*.

origin: X ray induced.

references: Hazelrigg and Kaufman, 1983, *Genetics* 105: 581-600.

Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, *Cell* 35: 763-76.

genetics: Homozygous lethal; heterozygotes show reduced sex comb phenotype. Fails to complement *Antp* and certain *Scr* alleles for lethality; viable or semilethal with other *Scr* alleles.

molecular biology: Third-chromosome breakpoint 79 kilobases to the right of the proximal breakpoint of *Tp(3;3)Dfd^{rv16}* (Scott *et al.*, 1983).

T(2;3)Scx^{Wrv6}

cytology: *T(2;3)22D1;63A1-2 + T(2;3)54A1-2;80-81*. Pairing of polytene chromosomes in proximal *3R* usually disrupted.

synonym: *T(2;3)Scx^{W+RX6}*.

origin: X ray induced.

references: Hazelrigg and Kaufman, 1983, *Genetics* 105: 581-600.

Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, *Cell* 35: 763-76.

genetics: Homozygous lethal; heterozygotes show reduced sex comb phenotype. Fails to complement *Scr* and *Antp*

for lethality.

**T(2;3)SD: Translocation (2;3)
Segregation Distorter**

origin: X ray induced in SD-72 males.
references: Yamazaki and Thompson, 1973, Jpn. J. Genet. 48: 217-29.
genetics: Associated with SD. K values tested.

translocation	cytology	synonym
T(2;3)SD-1	25D;61E	T(2;3)SD72-9
T(2;3)SD-2	56;94	T(2;3)SD-182
T(2;3)SD-3	40-41;97	T(2;3)SD-392

**T(2;3)shv^S: Translocation (2;3)
shortvein of Segal**

references: Segal and Gelbart, 1985, Genetics 109: 119-43.
genetics: Associated with shv alleles. Lethal over shv and other dpp lethals.

translocation	cytology	origin
T(2;3)shv S1 α	22F1-2;64E1-2	X ray
T(2;3)shv S13	22F1-2;93B8-10	γ ray
T(2;3)shv S18	22F1-2;88E1-4	γ ray
T(2;3)shv S19 β	22F1-2;35A1-4;97A	γ ray
T(2;3)shv S24 γ	22F1-2;59D;80F;81F;87C;88D;94D + In(3LR)63B;85D	γ ray
T(2;3)shv S25	22F1-2;88E1-4	γ ray

α Induced on dpp^{d-ho} chromosome.
β New order: 21 - 22F|35A - 22F|97A - 61A;
100F - 97A|35A - 60F.
γ New order:
21 - 22F|(80F - 81F)|87C - 85D|63B - 80F|81F - 85D|63B - 61A;
60F - 59D|88D - 87C|88D - 94D|22F - 59D|94D - 100F.
(tentative chromocentral arrangement)

***T(2;3)SM2: Translocation (2;3)
Second Multiple**

cytology: T(2;3)21A;40F;80-81 superimposed on In(2L)22D1-2;33F5-34A1 + In(2LR)22A3-B1;60B-C + In(2R)42A2-3;58A4-B1; position of breaks in proximal heterochromatin with respect to centromeres not determined.

origin: X ray induced in In(2LR)SM1.
discoverer: R. F. Grell, 1953.
references: Lewis and Mislove, 1953, DIS 27: 58. Mislove and Lewis, 1954, DIS 28: 77.
genetics: Variegated for lt.

other information: The translocation impairs its general usefulness as a chromosome 2 balancer; described as SM2 (see Balancers in Special Chromosomes section).

T(2;3)smg: Translocation (2;3) smudge

cytology: T(2;3)24D;92A.
synonym: T(2;3)T1.
references: Robinson and Curtis, 1972, Can. J. Genet. Cytol. 14: 129-37. 1973, Genetica 44: 591-601.
genetics: Homozygotes viable and fertile, with abnormal eye ("smudge" effect). Heterozygotes wild type and semisterile (Robinson and Curtis, 1973). In double heterozygotes T(2;3)gls/T(2;3)smg, females are more fertile than males (Robinson and Curtis, 1972).

**T(2;3)SMG: Translocation (2;3)
Semenova Mglinetz Glotoff**

origin: γ ray induced.
references: Semenova, Mglinetz, and Glotoff, 1970, Genetika (Moscow) 6: 165-69.

translocation	cytology
T(2;3)SMG41	27B;92C
T(2;3)SMG42	27F;66B
T(2;3)SMG43	26E;54D;76C;84F

T(2;3)sna: Translocation (2;3) snail

cytology: T(2;3)24B-C;35C-D;41;81. 24C-D to 35B-C inverted.

discoverer: Simpson.
synonym: RY2.
genetics: Mutant for sna. Homozygous lethal.

**T(2;3)so^{Drv}: Translocation (2;3)
sine-oculis**

references: Ashburner.
genetics: so^D revertant.

translocation	cytology
T(2;3)so ^{Drv1}	43B1-2;86E14-20
T(2;3)so ^{Drv2}	43B3-8;91A3-8

***T(2;3)sr^{4.2}: Translocation (2;3) stripe**

cytology: T(2;3)30C;90C-96.
origin: X ray induced.
discoverer: Alexander.
references: 1960, Genetics 45: 1019-22.
genetics: Mutant for sr. Homozygous lethal.

***T(2;3)sr^{100.312}**

cytology: T(2;3)40-41;90D2-E1.
origin: X ray induced.
discoverer: Alexander.
references: Ward and Alexander, 1957, Genetics 42: 42-54.
genetics: Mutant for sr. Homozygous lethal.

T(2;3)ss^{a75}

cytology: T(2;3)36D;89C.
origin: γ ray induced.
references: Kaufman, 1978, Genetics 90: 579-96.
genetics: Mutant for ss^a.

***T(2;3)st^{100.359}: Translocation (2;3) scarlet**

cytology: T(2;3)21C3-5;73A2-3;98F2-4.
new order:
21A - 21C3|73A3 - 98F2|73A2 - 61;
60 - 21C5|98F4 - 100.
origin: X ray induced.
discoverer: Alexander.
references: Ward and Alexander, 1957, Genetics 42: 42-54.
genetics: Mutant for st. Homozygous lethal.

**T(2;3)Su(var)3-5: Translocation (2;3)
Suppressor of
variegation 3-5**

cytology: T(2;3)58B;86B.
origin: X ray induced.
references: Reuter, Dorn, Wustmann, Friede, and Rauh, 1986, Mol. Gen. Genet. 202: 481-87.

genetics: Associated with *Su(var)3-5*. Recessive semilethal and sterile in both males and females.

T(2;3)Su(z)41: Translocation (2;3) Suppressor of zeste

cytology: *T(2;3)40-41;80-81*.

references: Gelbart, 1971, Ph.D. Thesis, Univ. of Wisconsin.

genetics: Associated with *Su(z)*.

T(2;3)Su(z)56

cytology: *T(2;3)40-41;97F-98B*.

references: Gelbart, 1971, Ph.D. Thesis, Univ. of Wisconsin.

genetics: Associated with *Su(z)*.

T(2;3)scx²: Translocation (2;3) super sex combs

cytology: *T(2;3)41C1-2;98C-D;99A-B*.

new order:

21A – 41C1|99B – 100;

61 – 98C|99A – 98D|41C2 – 60.

origin: X ray induced.

references: Ingham, 1984, Cell 37: 815-23.

genetics: Mutant for *scx*. Homozygous lethal and lethal over *Df(2R)M41A4*.

T(2;3)T

origin: X ray induced in stage 7 or stage 14 oocytes of *T(2;3)eg^{SPY}/+* females or *T(2;3)ap^{Xa}/+* females.

references: Puro, 1982, DIS 58: 205-08.

translocation	cytology	new order α	genetics of homozygotes
<i>T(2;3)T1-10</i>	<i>21A-B;34B-C;98F</i>	1	lethal
<i>T(2;3)T1-11</i>	<i>39D-F;43E-F;87B</i>	2	viable
<i>*T(2;3)T1-12</i>	<i>35B-F;39-41;75C</i>	3	lethal
<i>T(2;3)T1-13</i>	<i>30D2-F1;83A2-C1</i>		lethal
<i>*T(2;3)T1-16</i>	<i>31F1-32A1;71A1-2</i>		poorly viable
<i>*T(2;3)T1-19</i>	<i>22E;89D</i>		low viability and fertility over <i>Ubx</i> ¹³⁰
<i>T(2;3)T1-21</i>	<i>41C-42A;45D2-46A1;98F;</i> deficient for 41F-42A?	4	lethal
<i>T(2;3)T1-53^B</i>	<i>33D-E;79B;80A;</i> duplicated for 79B-80A	5	poorly viable
<i>T(2;3)T1-55</i>	<i>49A2-B1;75B</i>		lethal
<i>T(2;3)T1-56</i>	<i>32E2-F1;96C2-D1</i>		lethal
<i>T(2;3)T1-57</i>	<i>40-41;80-81</i>		lethal
<i>T(2;3)T1-58</i>	<i>60F5;64B-C</i>	6	Dp segregant viable and fertile; Df segregant lethal
<i>T(2;3)T2-30</i>	<i>41;89A-C</i>		

- α
- 1 = 21A|34B – 21B|98F – 61;
60 – 34C|99A – 100.
 - 2 = 21 – 39D|87B – 61;
60 – 43F|39F – 43E|87B – 100.
 - 3 = 21 – 35B|75C – 100;
61 – 75C|(35F – 39)|41 – 60.
 - 4 = 21 – 41C|45D2 – 42A|98F – 100;
61 – 98F|46A1 – 60.
 - 5 = 21 – 33D|79C – 100;
60 – 33E|80A2 – 61.
 - 6 = 21 – 60F5|64B – 61;
?|64C – 100

(terminal attachment of 3L segment to 2R distal to 60F5).

β Break in 2 and left break in 3 identical to those in *T(2;3)eg^{SPY}*; *T(2;3)T1-53* derived from *T(2;3)eg^{SPY}* by replacement of 61-79A4 by 61-80A1.

T(2;3)Ta¹: Translocation (2;3) thickened arista

cytology: *T(2;3)51E1-2;84B1-2* (Kaufman *et al.*, 1980) or *.84C1-2* (Cavener *et al.*, 1986).

synonym: *T(2;3)Ta^L*.

origin: γ ray induced.

references: Kaufman, 1978, Genetics 90: 579-96.

Kaufman, Lewis, and Wakimoto, 1980, Genetics 94: 115-33.

Cavener, Corbett, Cox, and Whetten, 1986, EMBO J. 5: 2939-48.

Cavener, Otteson, and Kaufman, 1986, Genetics 114: 111-23.

genetics: Associated with *Ta¹*. Homozygous lethal. When heterozygous with *Ta²*, the translocation is viable and shows a transformation of the whole arista and antennal segments four and five into tarsus and, in males, an extreme reduction in number of sex-comb teeth on the first leg (Kaufman *et al.*, 1980). Fails to complement both *hat* and *stk* and gives rise to the same lethal "partial eclosion" phenotype (Cavener *et al.*, 1986).

molecular biology: Distal (84C1-2) breakpoint at +34 kb; in the 15 kb overlap of *Df(3R)Scx2* and *Df(3R)dsx2M* (Baker and Wolfner, 1988, Genes Dev. 2: 477-89; 0 point = *HindIII* site in α *Tub84D*; "+" values to the right, "-" values to the left).

T(2;3)Tab^{rv100}: Translocation (2;3) Transabdominal-reverted

cytology: *T(2;3)37F-38A;89E + In(3R)89E;90D*.

origin: X ray induced in *In(3R)Tab*.

references: Celniker and Lewis, 1987, Genes and Development 1: 111-23.

genetics: Revertant of dominant *Tab* phenotype. When hemizygous, shows stronger transformation of the eighth ventral setal band toward that of the seventh than does *Tab/Df(3R)P9*; also abnormal posterior spiracles and a rudimentary ninth ventral setal band.

molecular biology: Lesion associated with the 89E breakpoint lies between 183 and 184 kb on the composite restriction map of the BXC (Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, Science 21: 23-29).

T(2;3)Tab^{rv185}

cytology: *T(2;3)50D;89E + In(3R)89E;90D*.

origin: X ray induced in *In(3R)Tab*.

references: Celnicker and Lewis, 1987, Genes and Development 1: 111-23.

genetics: Revertant of dominant *Tab* phenotype. When hemizygous, shows stronger transformation of the eighth ventral setal band toward that of the seventh than does *Tab/Df(3R)P9*; also abnormal posterior spiracles and a rudimentary ninth ventral setal band.

T(2;3)TE34Cc: Translocation (2;3) Transposing Element

cytology: *T(2;3)34D1-2;80-81*.

origin: γ ray induced in *TE34Cc*.

discoverer: Gubb.

synonym: *T(2;3)TE94^{V1}*.

references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

genetics: Variegates for *w*. Lethal with *l(2)34Da*

deficiencies.

T(2;3)TE35A

origin: γ ray induced in TE35A; selected as reversion of z.
synonym: T(2;3)TE146Z.

translocation	cytology	discov ^{α}	genetics
T(2;3)TE35A-2a	35B1-2;80	1	w ^v
T(2;3)TE35A-3	35B1-2;86F12-87A2	4	
T(2;3)TE35A-3a	35B1-2;80-81(?) + T(2;3)25A3-8;67C9	2	w ^v
T(2;3)TE35A-4	35B1-2;81	3	w ^v
T(2;3)TE35A-5	35B1-2;80-81	3	w ^v ; noc - vasa
T(2;3)TE35A-7	35B1-2;80-81	2	w ^v
T(2;3)TE35A-8	32B2-3;81 + 55;65 + In(2L)29D2-3;34D4 ^{β}	4	w ^v
T(2;3)TE35A-25	35B1-2;70B1-2	4	
T(2;3)TE35A-26	35B1-2;69F6-7;81;100F ^{γ}	4	
T(2;3)TE35A-28	35B1-2;90C3-6	4	
T(2;3)TE35A-58	35B1-2;94A4-5	1	
T(2;3)TE35A-200	35B;94A1-2	5	
T(2;3)TE35A-202	35B;81	5	
T(2;3)TE35A-207	35B;80 + T(2;3)CA47	5	
T(2;3)TE35A-208	35B;80-81	5	
T(2;3)TE35A-209	35B;80	5	
T(2;3)TE35A-212	35B;67A7-15;80-81 ^{δ}	5	
T(2;3)TE35A-215	35B;82B	5	
T(2;3)TE35A-216	35B;80-81	5	
T(2;3)TE35A-218	35B;86E12-13	5	
T(2;3)TE35A-221	35B;80-81	5	
T(2;3)TE35A-224	35B;67F2-68A1	5	
T(2;3)TE35A-227	40;72B-C + In(2L)35B1-2;40 + In(3R)81;88B		
T(2;3)TE35A-229	35B;80-81	5	
T(2;3)TE35A-230	21A4;26C4-D1;68B4-C1 + 35B;62A2-3 ^{ϵ}	5	

α 1 = Ashburner; 2 = Durrant; 3 = Gubb; 4 = Roote; 5 = Samkange.
 β New order: 21 - 29D2|34D4 - 29D3|34D4 - 35B2|81 - 65|55 - 60F;
61 - 65|55 - 35B3|81 - 100.
 γ New order: 21 - 35B|69F - 81|69F - 61A;
60 - 35B|81 - 100.
 δ New order: 21 - 35B|67A - 100;
60 - 35B|67A - 61.
 ϵ New order: 21 - 21A4|26D1 - 35B|62A3 - 68B4|21A4 - 26C4|68C1 - 100;
60 - 35B|62A2 - 61.

T(2;3)TE35BC

origin: γ ray induced in TE35BC; selected as w^v derivatives.

synonym: T(2;3)TE36R.

genetics: Variegates for w⁺ of TE35BC.

translocation	cytology	discoverer
T(2;3)TE35BC-1 ^{α}	35C1-3;80	Gubb
T(2;3)TE35BC-2	see Tp(2;3)TE35BC-2	
T(2;3)TE35BC-3 ^{β}	35B5-10;81	Gubb
T(2;3)TE35BC-6	35B;80(?)	Shelton
T(2;3)TE35BC-8 ^{γ}	35;80 + In(3L)65;79	Shelton
T(2;3)TE35BC-9 ^{δ}	33B;35B;81	
T(2;3)TE35BC-11	33B;80-81	

α Lethal with ck alleles, but not otherwise mutant in region 35. 3L break determined genetically.
 β w^v on 3⁺2^D element. Genetically broken between Adh and l(2)35Bb. Lethal with lethal ck alleles but breakpoint not lethal. 3R break determined genetically.
 γ New order: 21 - 35|80 - 100;
61 - 65|79 - 65|79 - 80|35 - 60.
Tentative.
 δ New order: 21 - 35B|81|(33B - 35B)|80 - 61;
60 - 35B|81 - 100.

T(2;3)TE36: see T(2;3)35BC

T(2;3)TE94R: see T(2;3)TE34Cc

T(2;3)TE146Z: see T(2;3)35A

T(2;3)TI^{rv3}: Translocation (2;3) Toll-revertant

cytology: T(2;3)40-41;97D1-2.

origin: X ray induced.

synonym: T(2;3)TI^{RXE}.

references: Anderson, Jürgens, and Nüsslein-Volhard, 1985, Cell 42: 779-89.

genetics: Revertant of Tl. Heterozygous females viable, fertile, and produce dorsalized lethal embryos as maternal effect.

T(2;3)Ubx: Translocation (2;3) Ultrabithorax

genetics: Mutant for Ubx.

translocation	cytology	origin	discov.	ref ^{α}
T(2;3)Ubx 1A	41;89E	X ray		see BXC
T(2;3)Ubx 1U	31;89E			see BXC
T(2;3)Ubx 4-30	34;89E1-2	X ray		2
T(2;3)Ubx 6-26	54E;75C;89E1-2	X ray		2
T(2;3)Ubx 105	53C;89E1-2		E.B. Lewis	
T(2;3)Ubx 175	22B1-2;89E			1
T(2;3)Ubx 1069	41A;89E	X ray	R.H. Baker	
T(2;3)Ubx B18	21D1-2;89E	EMS	Bacher, 65i	
T(2;3)Ubx B36	41F;89E	EMS	Bacher, 65i	
T(2;3)Ubx D1	41;89E1-2			
T(2;3)Ubx KM26	59E;75C;89E1-2	X ray		2
T(2;3)Ubx KM30	34;89E1-2	X ray		2
T(2;3)Ubx R10 ^{β}	41;89D-E	X ray	Ramey	
T(2;3)Ubx R17 ^{β}	22B1-2;89E1-2	X ray	Ramey	
T(2;3)Ubx R22 ^{β}	41;89E	X ray	Ramey	
T(2;3)Ubx R34 ^{β}	41;89E	X ray	Ramey	
T(2;3)Ubx X-type	52A-C;89E		E.B. Lewis	

α 1 = Craymer; 2 = Kerridge and Morata, 1982, J. Embryol. Exp. Morphol. 68: 211-34.
 β Synonym: Cbx^{rev.R17}.

T(2;3)V: Translocation (2;3) Valencia

origin: X ray induced.

references: Valencia, 1970, DIS 45: 37-38.

translocation	cytology	genetics of homozygotes
T(2;3)V3-2	27C-D;74C-D	arched wings; semilethal
T(2;3)V4-13	58B-C;78B	lethal
T(2;3)V8-2	30B;97F-98A	lethal
T(2;3)V9-1	40-41;62F	lethal
T(2;3)V9-10	35A;90F	lethal
T(2;3)V10-7b	56D-E;89D	lethal
T(2;3)V11-1	49C-D;65F	viable, fertile
T(2;3)V11-3-3	54C;62A1	lethal
T(2;3)V11-3e	46D;63C	lethal
T(2;3)V11-3g ^{α}	24A-B;53B;81F	lethal
T(2;3)V12-1-6	55F;62E + In(2L)21F;29B-C	lethal
T(2;3)V12-1-10	42F-43A;81-82	lethal
T(2;3)V12-1-32	59F;79F	lethal
T(2;3)V12-1-41	22E;91F	lethal
T(2;3)V12-1b	31D;85D	lethal
T(2;3)V12-2d ^{β}	31F-32A;41;53B;78F;94C	
T(2;3)V13-1	35B;96E + In(3L)70B;71E-F	lethal
T(2;3)V13-1b	33B;79F	lethal
T(2;3)V13-2b	60D;96F + Df(2R)49D-E;50B	lethal
T(2;3)V13-3	40-41;85F	lethal
T(2;3)V14	40;62C	semilethal
T(2;3)V16	60B-C;84A + In(3R)93F;99C	partially viable
T(2;3)V103	58F;67B4 + In(3R)85A;96E	lethal
T(2;3)V116	41C;75B	lethal

- α New order: 21 – 24A|81F – 61;
60F – 53B|24B – 53B|81F – 100.
 β New order: 21 – 31F|78F – 94C|41 – 53B|94C – 100;
61 – 78F|41 – 32A|53B – 60.

T(2;3)vg: Translocation (2;3) vestigial

references: Alexandrov and Alexandrova, 1987, DIS 66: 185-87.

translocation	cytology	origin	phen under 25°C
<i>T(2;3)vg 67d1</i>	49C2-D2;93F-F1	γ ray	lethal
<i>T(2;3)vg 76l1</i>	49D2-E1;84E2-3	actinomycin-D + γ ray	lethal
<i>T(2;3)vg 78b4</i>	49D2-3;49E7-F1;80C	NaF + γ ray	lethal
<i>T(2;3)vg 79b3</i>	49C2-3;94A2-3 + Df(2R)49C4;49E2	γ ray	lethal
<i>T(2;3)vg 81a</i>	49D2-F1;64B2-12	γ ray	lethal
<i>T(2;3)vg 83c</i>	49D2-E;65F6-66A	γ ray	vg ^{RP}
<i>T(2;3)vg 85d2</i>	49D2-E;84F4-6	0.85 MeV	vg

T(2;3)VV1: Translocation (2;3) V. Velissariou

cytology: *T(2;3)27A1-2;85A2-6*.

origin: X ray induced.

discoverer: Velissariou.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.

T(2;3)VV6

cytology: *T(2;3)57D;65D*.

origin: X ray induced.

discoverer: Velissariou.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.

T(2;3)VW2: Translocation (2;3) V. Walker

cytology: *T(2;3)49E-50A4;80C*.

origin: γ ray induced.

discoverer: Walker.

references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

genetics: Minute in phenotype.

T(2;3)Xa: see *T(2;3)ap^{Xa}*

T(2;3)Y: Translocation (2;3) Ytterborn

origin: X ray induced.

references: Ytterborn, 1968, Hereditas 59: 49-62.

translocation	cytology
<i>T(2;3)Y1</i>	30;96
<i>T(2;3)Y2</i>	54;98
<i>T(2;3)Y3</i>	57;97

T(2;3;4)429.18

cytology: *T(2;3)49A1-2;50B;66B + T(2;4)29F1-2;101 + In(3R)86B-C;87F1-2 + In(3R)89D6-E2;96A*.

new order:

21 – 29F1|101;

60 – 50B|49A1 – 29F2|101 – 102;

61A – 66B|50B – 49A2|66B – 86B|87F1 – 86C

|87F2 – 89D6|96A – 89E2|96A – 100.

origin: X ray induced.

discoverer: Gelbart.

T(2;3;4)429.29

cytology: *T(2;3;4)28D1-2;31D-E;80F;102E-F*.

new order:

21 – 28D1|31E – 60;

61 – 80F|28D2 – 31D|102E – 101A;

100 – 80F|102F.

origin: X ray induced.

discoverer: Gelbart.

T(2;3;4)429.36

cytology: *T(2;3)40;65A1-2 + T(3;4)71F-72A;101F*.

origin: X ray induced.

discoverer: Gelbart.

****T(2;3;4)bw^{R58}: Translocation (2;3;4) brown-Rearranged***

cytology: *T(2;3;4)59D;65;101C*.

new order:

21 – 59D|65 – 61;

60 – 59D|101C – 102;

?|65 – 100.

101A to C lost.

origin: X ray induced.

discoverer: Slatis.

references: 1955, Genetics 40: 5-23.

genetics: Associated with *bw^{R58}*.

T(2;3;4)bw^{V30k18}: Translocation (2;3;4) brown-Variegated

origin: X ray induced.

discoverer: Van Atta, 30k13.

references: 1932, Genetics 17: 637-59.

genetics: Variegated for *bw*. Produces aneuploids that have Minute bristles.

T(2;3;4)C: Translocation (2;3;4) Crossover suppressor

origin: X ray induced.

discoverer: Roberts.

synonym: *T(2;3;4)16 – T(2;3;4)36*.

references: 1972, Genetics 71: 401-15.

translocation	cytology	recombination reduced in
<i>*T(2;3;4)C16</i>	<i>T(2;3)35B;97F + T(2;4)48E;101</i>	2L + 2R
<i>*T(2;3;4)C20</i>	<i>T(2;3)40-41;80 + T(3;4)89E;101</i>	3R
<i>*T(2;3;4)C21</i>	<i>T(2;3)57C;98A + T(2;4)28C;101</i>	2R + 2L
<i>*T(2;3;4)C26</i>	<i>T(2;3)21A;68F + T(3;4)89A;101</i>	2L + 3R
<i>*T(2;3;4)C36</i>	<i>T(2;3)27B;88A + T(3;4)64F;101</i>	2L + 3L

T(2;3;4)CA4

cytology: *T(2;3;4)50E-F;81;89A;94F;101*.

new order:

21 – 50E|94F – 89A|50F – 60;

61 – 81|89A – 81|101 – 102;

100 – 94F|101 (tentative).

origin: γ ray induced.

discoverer: Ashburner.

references: Ashburner, Aaron, and Tsubota, 1982, Genetics 102: 421-35.

genetics: Induced with *Df(2L)A400*.

T(2;3;4)cl7: Translocation (2;3;4) cubitus interruptus

cytology: *T(2;3;4)24A1;98A1;101D-F + In(2L)27B;29B*.

new order:

21 – 24A1|98A1 – 61;

60 – 29B|27B – 29B|27B – 24A1|101F – 102;
100 – 98A1|101D – 101A.

origin: X ray induced.

synonym: $R^7(ci)$.

references: Stern and Kodani, 1955, Genetics 40: 343-73.

genetics: $T(2;3;4)ci7/ci$ flies show more extreme L4 vein interruptions than ci/ci flies.

$T(2;3;4)ci10$

cytology: $T(2;3;4)48B1;49F-50A1;81F;85F;97F1;101D-F;102C1$. Complex.

new order:

21 – 48B1|85F – 97F1|102C1 – 102F;
61 – 81F|50A1 – 60;
101A – 101D|81F – 85F|101F – 102C1|48B1 – 49F|97F1 – 100.

origin: X ray induced.

synonym: $R^{10}(ci)$.

references: Stern and Kodani, 1955, Genetics 40: 343-73.

genetics: $T(2;3;4)ci10/ci$ flies show less extreme L4 vein interruptions than ci/ci flies.

$T(2;3;4)ci^+3$: Translocation (2;3;4) cubitus interruptus-wild type

cytology: $T(2;3)21D1;75A1 + T(3;4)67C2;101F1 + Tp(3;4)95D-E;97C2;101F1;102F$.

new order:

102 – 101F1|67C2 – 75A1|21D1 – 60;
21A – 21D1|75A1 – 95D|97C2 – 100;
101A – 101F1|67C2 – 61;
101A – 101F1|95E – 97C2|102F (tentative). Chromosome 4 believed to be split into two chromatids before breaks induced, with one chromatid attached to 3L at 67C2 and the other carrying an insertion of 95E-97C2 and a deletion of most of 4R (Stern *et al.*, 1946).

origin: X ray induced.

synonym: $R^3(+)$; $T(2;3;4)+3$.

references: Stern, MacKnight, and Kodani, 1946, Genetics 31: 598-619.

Stern and Kodani, 1955, Genetics 40: 343-73.

genetics: $T(2;3;4)ci^+3/ci$ flies show L4 vein interruptions, whereas ci^+3/ci flies are wild type.

$T(2;3;4)ci^+14$

cytology: $T(2;3;4)60B;99B1;101F$. Complex.

new order:

21 – 60B|99B1 – 100;
61 – 99B1|101F – 102;
101A – 101F|60B – 60F.

origin: X ray induced.

synonym: $R^{14}(+)$.

references: Stern and Kodani, 1955, Genetics 40: 343-73.

genetics: $T(2;3;4)ci^+14/ci$ flies show L4 vein interruptions, whereas ci^+14/ci flies are wild type.

$T(2;3;4)dpp^9$: Translocation (2;3;4) decapentaplegic

cytology: $T(2;3)22F2-23A1;41A;64F + T(2;3;4)57E-F;80B;101F$.

new order:

21A – 22F2|41A – 57E|80B – 100;
102F – 101F|80B – 64F|41A – 23A1|64F – 61A;
60F – 57F|101F – 101A (tentative).

origin: X ray induced.

discoverer: Spenser.

references: Gelbart.

genetics: $d-III$ mutant.

$T(2;3;4)dpp^{81}$

cytology: $T(2;3)22F1-2;80 + T(2;4)30C;101$.

origin: γ ray induced.

discoverer: Segal.

references: Gelbart.

genetics: $d-V$ mutant.

$T(2;3;4)iab4^{302}$: Translocation (2;3;4) infraabdominal4

cytology: $T(2;3;4)60D;81;89E;100F;101$. Complex.

origin: X ray induced.

discoverer: R.H. Baker.

synonym: $iab4^{31616.302}$.

genetics: Mutant for $iab4$.

molecular biology: 89E breakpoint 83-86.5 kb distal to the right breakpoint of $In(3R)Cbx^{rv1}$.

$T(2;4)429 - T(2;4)434$

origin: X ray induced.

discoverer: Gelbart.

translocation	cytology
$T(2;4)429.73$	see $T(2;4)DTD13$
$T(2;4)429.90$	see $T(2;4)DTD15$
$T(2;4)432.37$	28A;101
$T(2;4)432.64$	35E;101
$T(2;4)434.06$	26A1-2;101A
$T(2;4)434.33$	see $T(2;4)DTD37$
$T(2;4)434.36$	see $T(2;4)DTD38$
$T(2;4)434.42$	see $T(2;4)DTD39$
$T(2;4)434.53$	see $T(2;4)DTD40$
$T(2;4)434.119$	32A1-2;102E-F

$T(2;4)a$

cytology: $T(2;4)50B2-3;102E$ (Lewis).

origin: X ray induced.

discoverer: Dobzhansky, 1929.

references: 1930, Biol. Zentr. 50: 671-85.

1931, Genetics 16: 629-58.

genetics: Homozygous lethal. Fly hyperploid for 4^P2R^D element rarely survives and is sterile.

$T(2;4)A$: Translocation (2;4) from Austin

origin: X ray induced.

translocation	cytology α	ref β	genetics of homozygote
* $T(2;4)A6^{\gamma}$	57F2-3	2, 3	viable, sterile
* $T(2;4)A8^{\delta}$	26F4-27A1	2, 3	viable, fertile
* $T(2;4)A23$	58F	3	viable, sterile
* $T(2;4)A27$	40D1-F1	1, 2, 3	lethal
* $T(2;4)A29$	47A4-5	2, 3	lethal
* $T(2;4)A30$	53B2-C1	1, 2, 3	viable, fertile
* $T(2;4)A34^{\epsilon}$	56A6-7	1, 2, 3	viable
* $T(2;4)A35$	26E	3	viable
* $T(2;4)A40$	49F3-50A1	1, 2, 3	viable, fertile
* $T(2;4)A43^{\zeta}$	22C	2, 3	viable, fertile
* $T(2;4)A45$	36D	3	lethal
			viable, fertile
* $T(2;4)A52$	36B	3	viable, fertile
$T(2;4)A53$	36E1-3	1, 2, 3	viable, fertile

α Breakpoint in chromosome 4 not determined.

β 1 = Burdette, 1940, Texas Univ. Publ. 4032: 157-63; 2 = Patterson, Brown, and Stone, 1940, Texas Univ. Publ. 4032: 167-89; 3 = Patterson, Stone, Bedicheck, and Suche, 1934, Am. Nat. 68: 359-69.

γ Fly hyperploid for the 4^P2R^D element viable and fertile.

δ Fly hyperploid for the 4^P2L^D element viable and fertile.

^ε Either acts as or carries a dominant suppressor of *Pu* (Oliver, 1943, Anat. Rec. 87: 461).
^ζ $4^P 2L^D$ element not recoverable in hyperploid; therefore translocation probably more complex than given.

T(2;4)ast^V: Translocation (2;4) asteroid-variegated

cytology: *T(2;4)21E2-3;101*.
origin: X ray induced.
discoverer: E.B. Lewis, 1940.
references: 1945, Genetics 30: 137-166.
genetics: Variegates for *S*, *ast*, and *ci*. Homozygous lethal. Fly with $2^P 4^D$ element in place of one chromosome 2 survives and has extremely rough eyes. $2^P 4^D$ is deficient for *l(2)gl* and *net* and presumably for *al*, *ex*, and *ds*. Fly hyperploid for complementary $4^P 2L^D$ also survives.

T(2;4)b

cytology: *T(2;4)25E;102C15-D1* (Schultz and Lewis). Metaphase chromosome 4 twice normal size.
origin: X ray induced.
discoverer: Dobzhansky, 1929.
references: 1930, Biol. Zentr. 50: 671-85. 1931, Genetics 16: 629-58.
genetics: *ci* not affected. Homozygous viable and fertile. Fly hyperploid for $4^P 2L^D$ element survives; short and thick with flattened abdomen, bulging eyes, and curved wings; both sexes sterile. Duplicated for *M(2)25A* and *dp* but not *cl*, *ey*, or *sv* (Morgan, 1946, DIS 20: 88).

T(2;4)BIII17

cytology: *T(2;4)40-41;101F*.
references: Yamamoto, 1987, DIS 66: 192-93.

****T(2;4)bw^{R25}: Translocation (2;4) brown-Rearranged***

cytology: *T(2;4)59D;101E*.
origin: X-ray-induced derivative of *bw*.
discoverer: Slatis.
references: 1955, Genetics 40: 5-23.
genetics: Associated with *bw^{R25}*.

****T(2;4)c***

cytology: Metaphase chromosome 4 about twice normal size.
origin: X ray induced.
discoverer: Dobzhansky, 1929.
references: 1930, Biol. Zentr. 50: 671-85. 1961, Genetics 16: 629-58.
genetics: Homozygote nearly lethal; wings do not expand; fly dies early. Break in 2L between *dp* and *b*, close to *dp*. Male hyperploid for $4^P 2L^D$ element poorly viable and sterile. No variegation for *ci⁺* (Stern).

T(2;4)C: Translocation (2;4) Crossover suppressor

origin: X ray induced.
discoverer: Roberts.
synonym: *T(2;4)2 - T(2;4)90*.
references: 1972, Genetics 71: 401-15.

translocation	cytology	Xover reduced in
* <i>T(2;4)C2</i>	60A;101	2R
* <i>T(2;4)C4</i>	53E;101	2R
* <i>T(2;4)C7</i>	57E;101	2R
* <i>T(2;4)C8</i>	48C;101	2R
* <i>T(2;4)C9</i>	33C;101	2L

translocation	cytology	Xover reduced in
* <i>T(2;4)C13</i>	53E;101	2R
* <i>T(2;4)C14</i>	55F;101	2R
* <i>T(2;4)C15</i>	29B;101	2L
* <i>T(2;4)C18</i>	34B;101	2L
* <i>T(2;4)C19</i>	26C;101	2L
* <i>T(2;4)C22</i>	33A;101	2L
* <i>T(2;4)C24</i>	35B;101	2L
* <i>T(2;4)C25</i>	30B;101	2L
* <i>T(2;4)C29</i>	34E;101	2L
* <i>T(2;4)C31</i>	30E;101	2L
* <i>T(2;4)C32</i>	22A;101	2L
* <i>T(2;4)C33</i>	30B;101	2L
* <i>T(2;4)C37</i>	35F;101	2L
* <i>T(2;4)C39</i>	60A;101	2R
* <i>T(2;4)C58</i>	49A;101	2R
* <i>T(2;4)C81</i>	60E;101	2R
* <i>T(2;4)C84</i>	24F;101	2L
* <i>T(2;4)C90</i>	32A;101	2L

T(2;4)CA36

cytology: *T(2;4)48C1-2;102A-B*.
origin: γ ray induced.
discoverer: Ashburner.
references: Gubb, Shelton, Roote, McGill, and Ashburner, 1984, Chromosoma 91: 54-64.
other information: Associated with *Df(2L)TE35BC-29*.

T(2;4)ci: Translocation (2;4) cubitus interruptus

origin: X ray induced.
synonym: *R^X(ci)*.

translocation	cytology	ref ^α	genetics ^β
<i>T(2;4)cl2</i>	44C3;102C3	2	1 (females); 2 (males)
<i>T(2;4)cl3</i>	52E1;101	2	1
<i>T(2;4)cl6</i>	47D3;102D3	2	2
<i>T(2;4)ci7</i>	see <i>T(2;3;4)cl7</i>		
<i>T(2;4)cl9</i>	38B2;101	2	1
<i>T(2;4)cl16</i>	45E1;102D3	2	2
<i>T(2;4)cl30</i>	40-41;101	2	1
<i>T(2;4)cl33</i>	48C-D;101F	2	1
<i>T(2;4)cl36</i>	47A1;102C1	1, 2	2
<i>T(2;4)cl42</i>	40-41;101	2	1
<i>T(2;4)cl44</i>	40-41;101	2	1
<i>T(2;4)cl45</i>	58D;102D-E	1, 2	2
<i>T(2;4)cl53</i>	47B5;101	2	1

^α 1 = Altorfer, 1967, Genetics 55: 755-67; 2 = Stern and Kodani, 1955, Genetics 40: 343-73.
^β 1 = *T(2;4)ci/ci* flies show more extreme L4 vein interruptions than *ci/ci* flies; 2 = *T(2;4)ci/ci* flies show less extreme L4 vein interruptions than *ci/ci* flies.

T(2;4)ci⁺: Translocation (2;4) cubitus interruptus-wild type

origin: X ray induced.
synonym: *R^X(+)*.
references: Stern and Kodani, 1955, Genetics 40: 343-73.
genetics: *T(2;4)ci⁺/ci* flies show L4 vein interruptions, whereas *ci⁺/ci* flies are wild type.

translocation	cytology
<i>T(2;4)ci⁺ 6</i>	59D;101
<i>T(2;4)ci⁺ 10</i>	31F1;102B1 (complex)
<i>T(2;4)ci⁺ 11</i>	47C;102B1
<i>T(2;4)ci⁺ 18</i>	57F1;101F
<i>T(2;4)ci⁺ 20</i>	55A;101
<i>T(2;4)ci⁺ 21</i>	60E1;101F

T(2;4)d

cytology: *T(2;4)55E-F* (Lewis, 1956, DIS 30: 130); break-point in chromosome 4 not determined.
origin: X ray induced.
discoverer: Dobzhansky, 1929.
references: 1930, Biol. Zentr. 50: 671-85. 1931, Genetics 16: 629-58.
genetics: Homozygote nearly lethal; fly is short lived and has inflated wings. No viable aneuploid product; therefore, probably more complex than indicated.

T(2;4)dpp⁷¹: Translocation (2;4) decapentaplegic

cytology: *T(2;4)22F1-2;101*.
origin: γ ray induced.
discoverer: Segal.
references: Gelbart.
genetics: *d-III* mutant.

T(2;4)dpp⁷³

cytology: *T(2;4)22F1-2;57A;101F*.
new order:
 21A – 22F1|101F – 101A;
 60F – 57A|22F2 – 57A|101F – 102F.
origin: γ ray induced.
discoverer: Segal.
references: Gelbart.
genetics: *d-III* mutant.

T(2;4)DTD: Translocation (2;4) Disrupter of Transvection at Decapentaplegic

origin: X ray induced; 15 induced in *dpp*⁴, 22 in *Dp(1;2)w⁺70h*, *dpp*^{ho2}, 37-40 in *dpp*^{ho2}, and 103 and 112 in *dpp*^{ho2} *TE23C-D[w⁺]*.
genetics: Disrupts transvection at *dpp*.

translocation	cytology	ref ^α
<i>T(2;4)DTD13</i>	<i>24A1-2;101A-D</i>	2
<i>T(2;4)DTD15</i>	<i>22F3-4;102F</i>	2
<i>T(2;4)DTD22</i>	<i>35E1-2;101</i>	3
<i>T(2;4)DTD22.12</i>	<i>25D-E;102D-E</i>	1
<i>T(2;4)DTD37</i>	<i>28D;101A</i>	2
<i>T(2;4)DTD38</i>	<i>29B;32A;101A</i>	2
<i>T(2;4)DTD39</i>	<i>24C;102B</i>	2
<i>T(2;4)DTD40</i>	<i>25C;102B</i>	2
<i>T(2;4)DTD103</i>	<i>35D-E;102D-E</i>	3
<i>T(2;4)DTD112</i>	<i>23C-D;102E-F</i>	3

^α 1 = Gelbart. 2 = Gelbart, 1982, Proc. Nat. Acad. Sci. USA 79: 2636-40; 3 = Smolik-Utlaut and Gelbart, 1987, Genetics 116: 285-98.

T(2;4)GT6

cytology: *T(2;4)34F3;101-102*.
origin: γ rays.
discoverer: Durrant.
genetics: Mutant for *wb-l(2)34Fc*.

T(2;4)K

references: Yamamoto, 1987, DIS 66: 192-93.

translocation	cytology	comments
<i>T(2;4)K10</i>	<i>57F3-6;101F</i>	
<i>T(2;4)K12</i>	<i>49B;101F</i>	
* <i>T(2;4)K13</i>	<i>25A3-4;101F</i>	
<i>T(2;4)K22</i>	<i>56F12-15;101F</i>	eye color brownish
<i>T(2;4)K44</i>		
<i>T(2;4)K53</i>	<i>53C;101F</i>	
<i>T(2;4)K55</i>	<i>27E;101F</i>	
<i>T(2;4)K86</i>	<i>42A;101F</i>	homozygous viable

translocation	cytology	comments
<i>T(2;4)K89</i>	<i>52F7-9;101F</i>	homozygous viable
<i>T(2;4)K92</i>	<i>47E;101F</i>	
<i>T(2;4)K109</i>	<i>50C1-2;101F</i>	
<i>T(2;4)K419</i>	<i>35F;101F</i>	

T(2;4)lt^{m3}: Translocation (2;4) light-mottled 3

cytology: *T(2;3)40;60D;102F* (Craymer).
synonym: *ln(2LR)lt^{m3}*.
references: Hessler, 1957, Genetics 43: 495-503.
genetics: Variegated for *lt*.

T(2;4)shv^{S9}: Translocation (2;4) shortvein of Segal

cytology: *T(2;4)22F1-2;101*.
origin: γ ray induced.
references: Segal and Gelbart, 1985, Genetics 109: 119-43.
genetics: Associated with *shv*. Lethal over *shv* and other lethal *dpp* alleles.

T(2;4)shv^{S14}

cytology: *T(2;4)22F1-2;101-102*.
origin: γ ray induced.
references: Segal and Gelbart, 1985, Genetics 109: 119-43.
genetics: Associated with *shv*. Lethal over *shv* and other lethal *dpp* alleles.

T(2;4)TE35A-11

cytology: *T(2;4)35B;101F*.
origin: γ ray induced in *TE35A*.
discoverer: Durrant.
synonym: *T(2;4)TE146Z*.
genetics: Variegates for *w⁺*.

T(2;4)TE35A-50

cytology: *T(2;4)35B1-2;101-102*.
origin: γ ray induced in *TE35A*.
discoverer: Roote.
synonym: *T(2;4)TE146Z*.
genetics: Red-eyed on *z w* background.

T(2;4)TE35A-101

cytology: *T(2;4)35B;101-102*.
origin: γ ray induced.
synonym: *T(2;4)TE146Z*.

T(2;4)TE35A-214

cytology: *T(2;4)35A-B;102C*.
origin: γ ray induced in *TE35A*.
discoverer: Samkange.
synonym: *T(2;4)TE146Z*.

T(2;4)V24: Translocation (2;4) Valencia

cytology: *T(2;4)60A-B;102D-E*.
origin: X ray induced.
references: Valencia, 1970, DIS 45: 37-38.
genetics: Homozygous lethal.

T(2;4)Y

origin: X ray induced.
references: Yamamoto, 1987, DIS 66: 192-93.

translocation	cytology	comments
<i>T(2;4)Y2</i>	<i>35D3-4;101F</i>	
* <i>T(2;4)Y7</i>	<i>50C14-D1;102C-D</i>	wing margin depleted

translocation	cytology	comments
*T(2;4)Y22	41;101F	
T(2;4)Y34	36C-D;101F	
T(2;4)Y40	56B-C;101F	
T(2;4)Y47	31D-E;101F	
*T(2;4)Y64	35C;101F	
T(2;4)Y76	40-41;101F	homozygous viable
T(2;4)Y86	21E1-2;101F	
T(2;4)Y92	57B4-6;101F	
*T(2;4)Y100	21D;101F	
T(2;4)Y106	55A;101F	homozygous viable
T(2;4)Y109	40-41;59B;101F	
*T(2;4)Y141	40-41;101F	
T(2;4)Y142	52E;102C	homozygous viable
T(2;4)Y164	33A;101F	
T(2;4)Y185	52D;101F	
T(2;4)Y209	36C-D;101F	homozygous viable
T(2;4)Y220	56F6-8;101F	
T(2;4)Y231	52F;102D	
T(2;4)Y241	54D-E;101F	
T(2;4)Y296		
T(2;4)Y308	49F10-15;101F	
*T(2;4)Y316		
*T(2;4)Y318	40-41;101F	
T(2;4)Y325	34E-F;101F	homozygous viable
T(2;4)Y344	59F;101F	
T(2;4)Y375	60F5;101F	
T(2;4)Y376	33C;101F	
T(2;4)Y423-A	21B3-5;101F	
T(2;4)Y465	52D9;101F	
T(2;4)Y476	29D;101F	
T(2;4)Y492	30A7-9;56F8-9;101F	
T(2;4)Y496	22A1-2;101F	
T(2;4)Y517	54F;101F	

T(3;4)85C

cytology: T(3;4)85C; breakpoint in chromosome 4 not determined.

discoverer: E.B. Lewis.

references: Pipkin, 1959, Texas Univ. Publ. 5914: 69-88.

T(3;4)86D

cytology: T(3;4)86D2-3;101F.
origin: Neutron induced in $bx^{34e}e^4$.

discoverer: E.B. Lewis.

references: Grell, 1959, Genetics 44: 421-35.
1959, Genetics 44: 911-22.

Grell and Day, 1970, Chromosoma 31: 434.

genetics: Homozygous viable and fertile. T(3;4)86D/ci has ci effect, is enhanced by low temperature, and tends to be suppressed by extra Y chromosome. Venation of homozygote and haplo-4 is ci^+ .

T(3;4)88B

cytology: T(3;4)88B; breakpoint in 4 not determined.

origin: X ray induced in *Ubx*.

discoverer: E.B. Lewis.

references: Grell, 1959, Genetics 44: 421-35.

genetics: Homozygous lethal. Has no position effect on *ci*.

T(3;4)89E

cytology: T(3;4)89E2-3;101F.

origin: X ray induced in $ss\ bx\ Su(ss)^2$.

discoverer: E.B. Lewis.

references: Grell, 1959, Genetics 44: 911-22.

genetics: Associated with bxd^{101} . Homozygous lethal. T(3;4)89E/ci has a ci effect, is enhanced by low temperature, and tends to be suppressed by extra Y chromosome.

T(3;4)104: see Tp(4;3)f

T(3;4)429.10

cytology: T(3;4)99B1-2;102D + In(3R)81F;86B-C.

origin: X ray induced.

discoverer: Gelbart.

***T(3;4)684**

cytology: T(3;4)67;101; breakpoints roughly estimated from figure of Dubinin and Sidorov (1935).

origin: X ray induced.

discoverer: Dubinin and Sidorov.

references: 1934, Biol. Zh. (Moscow) 3: 307-31.

1935, Biol. Zh. (Moscow) 4: 555-68 (fig.).

genetics: Position effects on both *h* and *ci*.

T(3;4)a

cytology: Chromosome 4 increased to about one-half length of 3L in metaphase figures.

origin: X ray induced.

discoverer: Dobzhansky, 29h.

references: 1929, Biol. Zentr. 49: 408-19.

1929, Proc. Nat. Acad. Sci. USA 15: 633-38.

1930, Genetics 15: 347-99.

genetics: Homozygous lethal. Break in 3L between *D* and *th*.

T(3;4)A: Translocation (3;4) from Austin

origin: X ray induced.

translocation	cytology	ref ^α	genetics of homozygote
*T(3;4)A1	89A6-B1;102B	3, 5	viable and fertile (Patterson <i>et al.</i> , 1934); lethal (CP552)
T(3;4)A2 ^β	94A3-4;101F (Brown)	1, 3, 5	viable and fertile
*T(3;4)A3		3, 5	lethal; 3R break between <i>e</i> and <i>ca</i>
*T(3;4)A4	80-81;101	3, 5	lethal
*T(3;4)A5	92A5-6	2, 5	lethal
*T(3;4)A8	75B4-5;102D1-3 (Brown)	1, 2, 3, 5	viable and fertile
*T(3;4)A9	87E3-F1;102F	1, 3, 5	viable and fertile
T(3;4)A12 ^γ	73C1-2;102C	2, 3, 5, 6	poorly viable and fertile
T(3;4)A13 ^γ	67E3-4;102D-E	3, 4, 5, 6	lethal
*T(3;4)A14	80;101	3, 5	lethal
*T(3;4)A20	89A;101F	3, 5	lethal
*T(3;4)A22	61E-F;102B-C	3, 5	lethal; 3L broken to left of <i>ru</i>
*T(3;4)A23	66D5-E1;101F	3, 5	lethal in male; viable in female
*T(3;4)A24	99;102B-C	3, 5	viable and fertile
*T(3;4)A27	82B3-C1;101A-D	1, 2, 3, 5	viable and fertile
T(3;4)A28	94D3-4;102 (E.B. Lewis)	3, 5, 6	viable and fertile
*T(3;4)A30 ^β	96E5-F1;102B-C	3, 5, 6	lethal
*T(3;4)A31	80;101	1, 2, 3, 5	viable and fertile
*T(3;4)A34	61F;101F	3, 5	lethal; 3L broken to left of <i>ru</i>
*T(3;4)A36	80B3-C1;102E (Brown)	1, 2, 3, 5	viable and fertile
*T(3;4)A37	86E5-6;101F	3, 5	lethal
*T(3;4)A39	94B4-C1;101F	2, 3, 5	lethal
*T(3;4)A43		3, 5	lethal; 3R broken near <i>sr</i>
*T(3;4)A44 ^δ	76;99;102D-F	3, 5	lethal
*T(3;4)A45	80;101	3, 5	viable but sterile
*T(3;4)A52	65D3-F2	3, 5	viable and fertile
*T(3;4)A56	76E2-F3;101F	3, 5	lethal
*T(3;4)A60	see *T(3;4)A3		

α 1 = Brown, 1940, Texas Univ. Publ. 4032: 11-64; 2 = Burdette, 1940, Texas Univ. Publ. 4032: 157-63; 3 = Painter, 1935, Genetics 10: 301-26; 4 = Patterson, Brown, and Stone, 1940, Texas Univ. Publ. 4032: 167-89; 5 = Patterson, Stone, Bedichek, and Suche, 1934, Am. Nat. 68: 359-69. 6 = Pipkin, 1959, Texas Univ. Publ. 5914: 60-88.
 β Fly hyperploid for 4^P 3R^D element survives.
 γ Fly hyperploid for 4^P 3L^D element survives.
 δ New order: 61 - 76|102D - 101; 100 - 99|76 - 99|102F.

T(3;4)Antp^{Ns}: Translocation (3;4) Antennapedia-revertant

cytology: T(3;4)84B1-3;102F.
origin: X ray induced in Antp^{Ns}.
synonym: T(3;4)Antp^{Ns+R2}.
references: Duncan and Kaufman, 1975, Genetics 80: 733-52.
 Lewis, Kaufman, Denell, and Talerico, 1980, Genetics 95: 367-81.
genetics: Revertant of Antp^{Ns}. Lethal homozygous and when heterozygous with ANTC mutants.

***T(3;4)b**

cytology: Chromosome 4 increased to one-half the length of 3L in metaphase figures.
origin: X ray induced.
discoverer: Dobzhansky, 28h.
references: 1929, Biol. Zentr. 49: 408-19.
 1929, Proc. Nat. Acad. Sci. USA 15: 633-38.
 1930, Genetics 15: 347-99.
genetics: Breakpoint in 3L near th. Crossing over markedly lowered near th and somewhat so at 3L tip.

T(3;4)BII8

cytology: T(3;4)77A;101F.
references: Yamamoto, 1987, DIS 66: 192-93.

T(3;4)BII13

cytology: T(3;4)93E;101F.
references: Yamamoto, 1987, DIS 66: 192-93.

T(3;4)BTD1: Translocation (3;4) Bithorax Tranvection Disrupter

cytology: T(3;4)87A;102D
origin: γ ray induced.
references: Smolik-Utlaut and Gelbart, Genetics 116: 285-98.

T(3;4)bx^d101: Translocation (3;4) bithoraxoid

cytology: T(3;4)89E;101F.
origin: X ray induced.
discoverer: E.B. Lewis.
references: 1981, Developmental Biology Using Purified Genes (Brown and Fox, eds.). Academic Press, New York, Vol. 23, pp. 189-208.
genetics: Mutant for bx^d.

T(3;4)c

cytology: T(3;4)86B-C;101F (Lewis, 1951, DIS 25: 108-9).
origin: X ray induced.
discoverer: Dobzhansky, 28h.
references: 1929, Biol. Zentr. 49: 408-19.
 1929, Proc. Nat. Acad. Sci. USA 15: 633-38.
 1930, Genetics 15: 347-99.
genetics: Homozygous viable and fertile. ci not affected. Crossing over much reduced near breakpoint in heterozygote and even more reduced in homozygote in some

regions (Beadle, 1932, Proc. Nat. Acad. Sci. USA 18: 160-65).

T(3;4)C: Translocation (3;4) Crossover suppressor

origin: X ray induced.
discoverer: Roberts.
synonym: T(3;4)I - T(3;4)90.

translocation	cytology	ref ^{α}	Xover reduced in
*T(3;4)C1	92A;101	2	3R
*T(3;4)C3	94C;101	2	3R
*T(3;4)C5	69B;101	2	3L
*T(3;4)C6	92B;101	2	3R
*T(3;4)C10 ^{β}	100B;101	1, 2, 3	3R
*T(3;4)C11	89A;101	2	3R
*T(3;4)C12	66B;101	2	3L
*T(3;4)C17	98A;101	2	3R
*T(3;4)C23	86E;101	2	3R
*T(3;4)C27	61A;101	2	3L
*T(3;4)C28	93D;101	2	3R
*T(3;4)C40	61F;101	2	3L
*T(3;4)C44	96F;101	2	3R
*T(3;4)C46	67F;101	2	3L
*T(3;4)C49	100B;101	2	3R
*T(3;4)C51	92D;102A	2	3R
*T(3;4)C61	62E;101	2	3L
*T(3;4)C80	66A;101	2	3L
*T(3;4)C82	94A;101	2	3R
*T(3;4)C83	97D;101C	2, 3	3R
*T(3;4)C85	61F;101	2	3L
*T(3;4)C87	91C;101	2	3R

α 1 = Roberts, 1972, DIS 48: 92; 2 = Roberts, 1972, Genetics 71: 401-15; 3 = Roberts, 1972, Genetics 72: 607-14.
 β Homozygous lethal.

T(3;4)CA28

cytology: T(3;4)83D;102C.
origin: γ ray induced with T(2;3)TE35BC-6.
discoverer: Ashburner.

T(3;4)ci: Translocation (3;4) cubitus interruptus

origin: X ray induced.
synonym: R^X(ci).
genetics: T(3;4)ci/ci flies show more extreme vein interruption than ci/ci flies in all cases except in females of T(3;4)ci4 and T(3;4)ci17 and males of T(3;4)ci35 in which the situation is reversed.

translocation	cytology	ref ^{α}
T(3;4)ci4	80-81;101	1, 2
T(3;4)ci5	69E2;102B1	1, 2
T(3;4)ci8	69F2;101	1, 2
T(3;4)ci13	80-81;102E1	2
T(3;4)ci15	100A2;102C1	2
T(3;4)ci17	67E1;102C1	1, 2
T(3;4)ci18	86C8;102B1	2
T(3;4)ci19	97D9;101	2
T(3;4)ci22	8C1;102B1	1, 2
T(3;4)ci23	92E2;101F	1, 2
T(3;4)ci25	81F;101	1, 2
T(3;4)ci26	90B1;102D1	2
T(3;4)ci27	84B3-4;102F1	2
T(3;4)ci28	80-81;101	1, 2
T(3;4)ci29	94B2;101	1, 2
T(3;4)ci31	75A1;101	1, 2
T(3;4)ci32	2E2;102A	1, 2
T(3;4)ci34	80;101	2
T(3;4)ci35	79F;101	1, 2
T(3;4)ci37	80-81;101	2
T(3;4)ci39	80-81;102D1	2

translocation	cytology	ref ^α
T(3;4)ci40^β	94A1;102F2	2
T(3;4)ci41	98C-D;102C1	1, 2
T(3;4)ci43	86A1;102F	2
T(3;4)ci48	80-81;101	1, 2
T(3;4)ci52^γ	76A1;101F	2
T(3;4)ci54	87F;101F	2
T(3;4)ci55	65F2;101F	2

^α 1 = Altorfer, 1967, Genetics 55: 755-67; 2 = Stern and Kodani, 1955, Genetics 40: 343-73.
^β Originally a T(Y;3;4) but Y component lost before testing for ci phenotype (Stern and Kodani, 1955).
^γ Altorfer (1967) claims linkage to chromosome 3 lost later.

T(3;4)ci⁺: Translocation (3;4) cubitus interruptus-wild type

origin: X ray induced.
synonym: R^X(+).
references: Stern and Kodani, 1955, Genetics 40: 343-73.
genetics: T(3;4)ci⁺/ci flies show L4 vein interruptions, whereas ci⁺/ci flies are wild type.

translocation	cytology
T(3;4)ci⁺ 2^α	82D;101A-D
T(3;4)ci⁺ 5	96E1;101F
T(3;4)ci⁺ 8	96F6;102A
T(3;4)ci⁺ 9	97C1;101F
T(3;4)ci⁺ 12^α	98F1;101
T(3;4)ci⁺ 13	65D1;102A
T(3;4)ci⁺ 16	90E1;101F
T(3;4)ci⁺ 19	62D3;102B1

^α Involves break between ci and the centromere (Stern, MacKnight, and Kodani, 1946, Genetics 31: 598-619).

***T(3;4)d**

cytology: Metaphase figures show barely detectable increase in size of chromosome 4.
origin: X ray induced.
discoverer: Dobzhansky, 28h.
references: 1929, Biol. Zentr. 49: 408-19.
 1929, Proc. Nat. Acad. Sci. USA 15: 633-38.
 1930, Genetics 15: 347-99.
genetics: Homozygous lethal. ci not affected (Stern). Breakpoint in 3R between ca and M(3)99E and in 4 to the left of M(4) and ey. Apparently, 3^P4^D element can substitute for a normal 3, producing Minute flies. Hyperploids for 4^P3^D element probably also survive.

T(3;4)DI^{7P}: Translocation (3;4) Delta-7 of Panshin

origin: X ray induced.
discoverer: Panshin.
references: 1935, Dolk. Akad. Nauk SSSR 4: 85-88.
genetics: Chromosome 3 broken to the right of cu. Mutant for Dl; position effect that weakens dominance of cu⁺.

T(3;4)e

cytology: T(3;4)79E;102F (Lewis, 1956, DIS 30: 130).
origin: X ray induced.
discoverer: Dobzhansky, 28h.
references: 1929, Biol. Zentr. 49: 408-19.
 1929, Proc. Nat. Acad. Sci. USA 15: 633-38.
 1930, Genetics 15: 347-99.
genetics: Homozygous semilethal and female sterile. ci not affected (Stern). Crossing over normal in heterozygote except near p.

T(3;4)E(spl)^{IV}: Translocation (3;4) Enhancer of split-reverted

origin: X ray induced.
genetics: Loss of function mutants. Lethal as homo- and hemizygotes. Variable neural hypoplasia in deficiency heterozygotes.

translocation	cytology	discoverer	ref ^α	molecular biology ^β
T(3;4)E(spl)^{IV} 4^γ	96F8-9;100F5;100B-D	Bremer	1, 2, 3	proximal to -15 kb
T(3;4)E(spl)^{IV} 6^δ	96F12-14;102E-F	Knust, Ziemer	1, 2, 3	-5 kb
T(3;4)E(spl)^{IV} 7	96F7-11;102B-C	Tietz	2, 3	+2 kb to +5 kb

^α 1 = Knust, Bremer, Vässin, Ziemer, Tepass, and Campos-Ortega, 1987, Dev. Biol. 122: 262-73; 2 = Knust, Tietze, and Campos-Ortega, 1987, EMBO J. 6: 4113-23; 3 = Ziemer, Andreas, Tietze, Knust, and Campos-Ortega, 1988, Genetics 119: 63-74.
^β Position of 3R breakpoint. Origin (coordinate 0) is the first EcoRI site to right of the genomic fragment homologous to the cDNA clone 126 D12 used to start the E(spl) chromosomal walk, "+" values to the right, "-" values to the left (Knust et al., 1987b).
^γ 70% lethality with E(spl). 40-50% spl wing in heterozygotes over wild-type.
^δ 60% lethality with E(spl). No spl wings in heterozygotes over wild type.

***T(3;4)H: Translocation (3;4) from Howard University**

origin: X ray induced.
discoverer: Pipkin.
references: 1959, Texas Univ. Publ. 5914: 69-88.

translocation	cytology of 3
*T(3;4)H1	80-81
*T(3;4)H3	80-81
*T(3;4)H5^α	96E
*T(3;4)H6^α	98A
*T(3;4)H7	66C

^α Fly hyperploid for 4^P3R^D survives.

T(3;4)iab4¹²⁵: Translocation (3;4) infraabdominal4

cytology: T(3;4)89E;101D.
origin: X ray induced in Mcp.
discoverer: R.H. Baker.
references: Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.
genetics: Associated with iab4. Partial revertant of Mcp.
molecular biology: 89E breakpoint 81-83 kb distal to the right breakpoint of In(3R)Cbx^{rv1}.

T(3;4)K

references: Yamamoto, 1987, DIS 66: 192-93.

translocation	cytology	comments
T(3;4)K5		
T(3;4)K11		homozygous viable
*T(3;4)K19		
T(3;4)K19	94D;101F	
*T(3;4)K30		
T(3;4)K30		
T(3;4)K31	79F;101F	
T(3;4)K85	64C;101F	
T(3;4)K87	87D;101F	
T(3;4)K95		
T(3;4)K104		homozygous viable
T(3;4)K108		

translocation	cytology	comments
<i>T(3;4)K116</i>		homozygous viable
<i>T(3;4)K117</i>		homozygous viable

****T(3;4)K*: Translocation (3;4) of Kirssanov**

origin: X ray induced.
discoverer: Kirssanov.
references: 1933, Biol. Zh. (Moscow) 2: 447-50.

***T(3;4)l-18*: Translocation (3;4) lethal**

origin: X ray induced.
discoverer: Gloor and Green, 1957.
genetics: Variegates for *ci*. Mutant for *l(4)l02ABe*.

***T(3;4)Mg198* Translocation (3;4) Mglinetz**

cytology: *T(3;4)93E;101-102*.
origin: ³²P feeding.
references: Mglinetz, 1968, Genetika (Moscow) 4(8): 81-86.

***T(3;4)p⁴²*: Translocation (3;4) pink**

cytology: *T(3;4)85A;101D*.
origin: X ray induced.
references: Kempfues, Raff, and Kaufman, 1983, Genetics 105: 345-56.
genetics: Mutant for *p*.

***T(3;4)P86*: Translocation (3;4) from Pasadena**

cytology: *T(3;4)88B-C;101* (Lewis).
origin: X ray induced.
discoverer: E.B. Lewis.

***T(3;4)ry^{ps1149}*: Translocation (3;4) rosy-purine-sensitive**

cytology: *T(3;4)87B;101F*. 87B-D thought to be inverted [*In(3R)81;87B-D*] so that 87D placed next to heterochromatin (Rushlow and Chovnick, 1984).
origin: γ ray induced in *ry⁺¹¹*-bearing third chromosome.
references: Rushlow and Chovnick, 1984, Genetics 108: 589-602.
 Rushlow, Bender, and Chovnick, 1984, Genetics 108: 603-15.
 Clark and Chovnick, 1986, Genetics 114: 819-40.
genetics: Homozygous lethal. Variegates for *ry* with respect to XDH content of Malpighian tubules (Rushlow *et al.*, 1984); also variegates for *snk* (Clark and Chovnick, 1986). Purine-sensitive in heterozygotes with *ry²* or *ry⁴¹* (Rushlow and Chovnick, 1984). Has break in *pic* locus and is associated with *pic⁻* phenotype (Clark and Chovnick, 1986).
molecular biology: 3R breakpoint between 160 and 150 kb proximal to the origin of the walk, which is 6.5 kb distal to the proximal breakpoint of *In(3R)Cbx^{rv1}*; approximately 15 kb distal to *ry* (Rushlow *et al.*, 1984).

T(3;4)SS406

cytology: *T(3;4)65B;101*.
origin: X ray induced.
discoverer: Smith.

references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.

***T(3;4)Ubx^A* Translocation (3;4) Ultrabithorax**

cytology: *T(3;4)67;69;70;84;85;87F;88F;89E;100B;102E-F*. Extremely complex; breakpoints identified tentatively (E.B. Lewis).

origin: X ray induced.
discoverer: Schalet, 1959.
synonym: *In(3LR)Ubx^A*.
references: 1960, DIS 34: 53, 55.
 Craymer, 1980, DIS 55: 197-200.
genetics: Associated with *Ubx^A*. Homozygous lethal.

T(3;4)Ubx^{R40}

cytology: *T(3;4)89E;101*.
origin: X ray induced.
discoverer: Ramey.
synonym: *Cbx^{revR17.40R}*.
genetics: Associated with *Ubx^{R40}*.

T(3;4)Y

references: Yamamoto, 1987, DIS 66: 192-93.

translocation	cytology	comments
<i>T(3;4)Y9</i>	<i>65B4;101F</i>	
<i>T(3;4)Y13</i>	<i>80-81;101F</i>	
<i>T(3;4)Y25 + In(3R)</i>	<i>90C;98C;101F</i>	
<i>T(3;4)Y37</i>	<i>80-81;101</i>	
<i>T(3;4)Y57</i>	<i>75F;102C</i>	
* <i>T(3;4)Y61</i>	<i>92A;101F</i>	
<i>T(3;4)Y63</i>	<i>80-81;101F</i>	homozygous viable
<i>T(3;4)Y71</i>	<i>81F;101F</i>	
<i>T(3;4)Y78</i>	<i>100F;102C14</i>	
<i>T(3;4)Y81</i>	<i>64E;101F</i>	eye deformed
<i>T(3;4)Y104</i>	<i>96A1-4;101F</i>	
<i>T(3;4)Y121</i>	<i>80-81;101</i>	
* <i>T(3;4)Y140</i>	<i>96A20-25;101F</i>	
<i>T(3;4)Y144</i>	<i>88C;101F</i>	
<i>T(3;4)Y151</i>	<i>78B;101F</i>	
<i>T(3;4)Y154</i>	<i>72C;101F</i>	
<i>T(3;4)Y161</i>	<i>99E;101F</i>	
<i>T(3;4)Y168</i>	<i>76B1;102D</i>	
<i>T(3;4)Y175</i>	<i>91D;101F</i>	
<i>T(3;4)Y177</i>	<i>77B;101F</i>	
* <i>T(3;4)Y183</i>	<i>68D;101F</i>	
<i>T(3;4)Y210</i>	<i>67E;101F</i>	homozygous viable
<i>T(3;4)Y226</i>	<i>63D;101F</i>	homozygous viable
<i>T(3;4)Y249</i>	<i>80-81;101F</i>	
<i>T(3;4)Y252</i>	<i>80-81;101F</i>	homozygous viable
<i>T(3;4)Y255</i>	<i>94B;101F</i>	
<i>T(3;4)Y262</i>	<i>71F;101F</i>	homozygous viable
<i>T(3;4)Y285</i>	<i>96D-E;101F</i>	homozygous viable
<i>T(3;4)Y291</i>	<i>80-81;101F</i>	homozygous viable
<i>T(3;4)Y320</i>	<i>88D;101F</i>	homozygous viable
<i>T(3;4)Y391</i>	<i>98F11-12;101F</i>	
<i>T(3;4)Y403</i>	<i>67E3-4;101F</i>	
<i>T(3;4)Y425</i>	<i>83A;101F</i>	homozygous viable
<i>T(3;4)Y434</i>	<i>80-81;101F</i>	
<i>T(3;4)Y446</i>	<i>70D;101F</i>	
<i>T(3;4)Y449</i>	<i>88E4-6;101F</i>	
<i>T(3;4)Y477</i>	<i>75C;101F</i>	
<i>T(3;4)Y494</i>	<i>98B;101F</i>	
<i>T(3;4)Y495</i>	<i>70C;101F</i>	homozygous viable
<i>T(3;4)Y512 + In(3R)</i>	<i>94B-C;96E;101F</i>	

TRANSPOSITIONS

Tp(1;1)7B-7E

cytology: *Tp(1;1)7B5-C1;7E1-2;9C2-D1*.

origin: Spontaneous in an outcross of the L strain of Lim, 1979. The L strain has numerous gypsy elements which are mobilized in certain outcrosses (Jack and Judd).

references: Lim, 1979, *Genetics* 93: 681-701.

Tp(1;1)303-1: see *In(1)303-1*

Tp(1;1)A101

cytology: *Tp(1;1)11A1-2;12E8-9;18B*.

new order:

1 – 11A1|18B – 12E9|11A2 – 12E8|18B – 20.

discoverer: Lefevre.

genetics: Lethal at 11A1-2.

Tp(1;1)At: see *In(1)At*

Tp(1;1)B²⁶³⁻²⁴: see *In(1)B²⁶³⁻²⁴*

****Tp(1;1)B²⁶³⁻⁴⁸: Transposition (1;1) Bar***

cytology: *Tp(1;1)3E2-3;15F9-16A1;20A2-3*.

new order:

1 – 3E2|16A1 – 20A2|3E3 – 15F9|20A3 – 20F.

origin: X ray induced.

discoverer: Bishop, 1939.

references: Sutton, 1943, *Genetics* 28: 99.

genetics: Male and homozygous female viable. Crossing over in *Tp(1;1)B²⁶³⁻⁴⁸/+* heterozygote yields *Dp(1;1)B²⁶³⁻⁴⁸* (new order: 1 – 3E2|16A1 – 20A2|3E3 – 20F), which is heterozygous viable and Bar. The complementary deficiency (new order: 1 – 15F9|20A3 – 20F) is heterozygous lethal.

Tp(1;1)bi^{D1}: Transposition (1;1) bifid

cytology: Break in 4C5-6; insertion of extra band.

origin: X ray induced.

references: Banga, Bloomquist, Brodberg, Pye, Larrivee, Mason, Boyd, and Pak, 1986, *Chromosoma* 93: 341-46.

genetics: Mutant for *bi*. Male lethal.

****Tp(1;1)ct^{6a1}: Transposition (1;1) cut***

cytology: *Tp(1;1)7B2-C1;19;20F*.

new order:

|1 – 7B2|(19 – 20)|7C1 – 19|20F|.

Nucleolus organizer included in transposed piece.

origin: X ray induced in *R(1)2*.

references: Hannah, 1949, *Proc. Intern. Congr. Genet.*, 8th., pp. 588-89.

Hannah-Alava, 1971, *Mol. Gen. Genet.* 113: 191-203.

genetics: Variegated for *ct*. Male lethal.

Tp(1;1)f^{67a}: see *In(1)f^{67a}*

Tp(1;1)FN201

cytology: *Tp(1;1)3A3-4;5D;8F*.

discoverer: Lefevre.

references: Young and Judd, 1978, *Genetics* 88: 723-42.

genetics: Mutant for *z* (*z^a*-like).

Tp(1;1)hill: see *In(1)hill*

Tp(1;1)L2

cytology: *Tp(1;1)3A2-3;8D;10B1-2*.

new order:

1 – 3A2|8D – 10B1|3A3 – 8D|10B2 – 20.

origin: X ray induced.

discoverer: Lefevre.

genetics: Homozygous lethal. Mutant for *gt* and *lz*.

Tp(1;1)l272-13: see *In(1)272-13*

Tp(1;1)lz¹⁴⁴: Transposition (1;1) lozenge

cytology: *Tp(1;1)8E;20F*. Transposition of *NO* to 8E.

discoverer: Green and Green, 1956.

synonym: *lz^{144A}* of Ives (Green).

references: Hannah-Alava, 1971, *Mol. Gen. Genet.* 113: 191-203.

genetics: *lz^s* phenotype.

Tp(1;1)lz⁴⁹¹

cytology: *Tp(1;1)8D;20F*. Transposition of *NO* to 8D.

discoverer: Green and Green, 1956.

references: Hannah-Alava, 1971, *Mol. Gen. Genet.* 113: 191-203.

genetics: *lz^s* phenotype. Recombination reduced in *sn-lz* and *lz-v* regions (Green).

****Tp(1;1)N²⁶⁴⁻⁶³: Transposition (1;1) Notch***

cytology: *Tp(1;1)3C7-9;13C7-8;19F* (Hoover).

origin: X ray induced.

discoverer: Demerec, 38e.

genetics: Mutant for *N* but not for *w*, *rst*, or *dm*.

Tp(1;1)sc²⁶⁰⁻²⁵: see *In(1LR)sc²⁶⁰⁻²⁵*

Tp(1;1)Si1: Transposition (1;1) Simmons

cytology: *Tp(1;1)11A8-9;14D1-2;19C*.

new order:

1A – 11A8-9|(14D1-2 – 19C)|11A8-9 – 14D1-2|19C – 20F.

origin: Induced by hybrid dysgenesis.

references: Simmons, Raymond, Culbert, and Laverty, 1984, *Genetics* 107: 49-63.

genetics: Chromosome has lethal at *r* locus.

Tp(1;1)Si2

cytology: *Tp(1;1)1A1-8;14D2-E1;18F*.

new order:

1A1 – 8|14D2-E1 – 18F|1A1-8 – 14D2-E1|18F – 20F.

origin: Induced by hybrid dysgenesis.

references: Simmons, Raymond, Culbert, and Laverty, 1984, *Genetics* 107: 49-63.

genetics: Chromosome has lethal at *r* locus.

Tp(1;1)sn^{S93}: Transposition (1;1) singed

cytology: *Tp(1;1)7B5-6;7D1;8D*.

new order:

1 – 7B5|7D2 – 8D|7B6 – 7D1|8D – 20.

origin: X ray induced.

references: Lefevre and Johnson, 1973, *Genetics* 74: 633-45.

genetics: Shows *sn* phenotype in combination with *sn³*. Male lethal. Female heterozygote viable. When the components of the transposition are separated by crossing over in the 7D2-8D region, the deficiency alone (new order: 1 – 7B5|7D2 – 20) behaves as a dominant lethal, whereas the duplication (new order: 1 – 8D|7B6 – 7D1|8D – 20) behaves as a recessive lethal.

***Tp(1;1)v^{N48}*: Transposition (1;1) vermilion**

cytology: *Tp(1;1)9F;10C3-5;20*.

origin: Neutron induced.

discoverer: Schalet.

references: Voelker, Wisely, Huang, and Gyurkovics, 1985, *Mol. Gen. Genet.* 201: 437-45.

genetics: Mutant for *v*. Males viable, fertile and show additional mutant phenotypes such as rough eyes, missing bristles, and abnormal wings (Schalet). Females heterozygous for *Df(1)v64f*, *Df(1)v-L1*, and *Df(1)v-L2* are viable and show the additional mutant phenotypes described for males, but females heterozygous for *Df(1)v-L15*, *Df(1)N71*, or *Df(1)m259-4* are lethal.

Tp(1;1)z*52

cytology: *Tp(1;1)3C;10A;11A*.

references: Gelbart, 1971, Ph.D. Thesis, University of Wisconsin.

Tp(1;1)z*55

cytology: *Tp(1;1)3B-C;17F;20A*.

references: Gelbart, 1971, Ph.D. Thesis, University of Wisconsin.

Tp(1;Y)

origin: X ray induced.

references: Stewart and Merriam, 1973, *DIS* 50: 167-69.

transposition	cytology	genetics
<i>Tp(1;Y)B3</i>	3C2-3	viable, fertile
<i>Tp(1;Y)B4</i>	1F;3C	viable, fertile
<i>Tp(1;Y)B11</i>	1B;4A	viable, fertile
<i>Tp(1;Y)B27</i>	2B	viable, fertile
<i>Tp(1;Y)B33^α</i>	3C2;12E7-8;20	viable, fertile
<i>Tp(1;Y)L3</i>	9C;9D	viable, fertile
<i>Tp(1;Y)P11</i>	10C	viable, fertile
<i>Tp(1;Y)P11a</i>	10C	lethal

^α New order: 1 - 3C2|12E7-8 - 3C2|20; YL|12E7-8 - 20|YS.
3C break between *w* and *rst* (Young and Judd, 1978, *Genetics* 88: 723-42).

Tp(1;2)25

cytology: Euchromatic portion of left arm of X transposed into heterochromatic region of 2.

discoverer: Lindsley.

references: Shoup, 1967, *J. Cell Biol.* 32: 663-75.

genetics: Males sterile; sperm heads fail to differentiate and do not convert lysine-rich to arginine-rich histone.

Tp(1;2)26

origin: X ray induced in *R(1)2*.

discoverer: Pontecorvo, 1941.

synonym: *T(X^{c2};2)26*.

references: 1942, *DIS* 16: 65.

genetics: Section of X including *car* and *bb* inserted into base of 2L. Homozygous lethal.

Tp(1;2)51b

cytology: *Tp(1;2)3C1-2;3D6-E1;20A;52E*.

new order:

1 - 3C1|20A - 3E1|20A - 20F;
21 - 52E|(3C2 - 3D6)|52E - 60.

origin: X ray induced in *In(1)w^{m4}* = *In(1)3C1-2;20A*.

discoverer: Lefevre, 51b7.

synonym: *T(1;2)w^{+51b7}*.

references: 1951, *DIS* 25: 71.

1952, *DIS* 26: 66.

Ratty, 1954, *Genetics* 39: 513-28.

Lefevre, 1970, *DIS* 45: 39.

genetics: Segregant *Dp(1;2)51b* = *Dp(1;2)3C1-2;3D6;52E* survives; duplicated for loci of *w*, *rst*, *spl*, *fa*, and *dm*. Duplication used to cover lethality of *N* in studies of pseudoallelism at the *N* locus (Welshons and Von Halle, 1962, *Genetics* 47: 743-59).

****Tp(1;2)A50b*: Translocation (1;2) from Austin**

cytology: *Tp(1;2)2B;15F;41*; inferred from fig. 17H of Mackensen (1935).

new order:

1 - 2B|15F - 20;
21 - 41|15F - 2B|41 - 60.

references: Mackensen, 1935, *J. Heredity* 26: 163-74 (fig.).

genetics: Left break in X between *br* and *pn*; right break between *r* and *f*. Mutant for *f*.

****Tp(1;2)A106***

cytology: *Tp(1;2)6-7;12;17*; rough estimates of break-points in X from fig. 17I of Mackensen (1935); chromosome 2 broken in euchromatin of left arm.

new order:

1 - 6|17 - 20;
21 - ?|12 - 17|7 - 12|? - 60.

references: Mackensen, 1935, *J. Heredity* 26: 163-74 (fig.).

genetics: Mutant for *Bx*.

****Tp(1;2)A124***

cytology: *Tp(1;2)10A;13A1-2;59*.

new order:

1 - 10A|13A2 - 20;
21 - 59|(10A - 13A1)|59 - 60.

origin: X ray induced.

references: Mackensen, 1935, *J. Heredity* 26: 163-74 (fig.).

Patterson, Stone, and Bedichek, 1935, *Genetics* 20: 259-79 (fig.).

1937, *Genetics* 22: 407-26.

Pipkin, 1940, *Texas Univ. Publ.* 4032: 126-56.

genetics: Left break between *ras* and *v*; right break between *g* and *pl*. Male fertile. The segregant *Dp(1;2)A124* = *Dp(1;2)10A;13A1-2;59*, which is duplicated for *v⁺* through *g⁺*, survives as a fairly viable and fertile female, but male carrying *Dp(1;2)A124* dies as embryo. The complementary *Df(1)A124* = *Df(1)10A;13A1-2* survives as a fertile *X/X/Df* triploid female and as an *X/X/Df* diploid metafemale but not as an *X/Df* diploid.

****Tp(1;2)B^{DG}*: Transposition (1;2) Bar of Dubinin and Goldat**

cytology: *Tp(1;2)4;15F-16A;20;40-41*; inferred from figure of Dubinin and Goldat.

new order:

1 - 4|15F - 4|20;
21 - 40|(16A - 20)|41 - 60.

origin: X ray induced.

references: Dubinin and Goldat, 1936, *Biol. Zh.* 5: 881-84 (fig.).

genetics: Position effect for *B*. Male lethal.

Tp(1;2)C84

cytology: *Tp(1;2)3F;17E-F;30A*.

new order:

1 – 3F|17F – 20;

21 – 30A|3F – 17E|30A – 60.

origin: X ray induced.

references: Roberts, 1970, *Genetics* 65: 429-48.

genetics: Male viable and sterile. Recombination reduced in X and 2L.

****Tp(1;2)ct^{7c1}: Transposition (1;2) cut***

cytology: *Tp(1;2)7B2-3;8E2-3;25C* superimposed on *R(1)1A3-4;19F-20A1*.

new order:

|1A4 – 7B2|8E2 – 20-20F – 20A1|;

21 – 25C|(7B3 – 8E2)|25C – 60.

origin: X ray induced in *R(1)2*.

discoverer: Hannah, 1947.

genetics: Mutant for *ct* but not *cm* or *sn*; male lethal.

Tp(1;2)ct^{7c1}/Dp(1;3)sn^{13al} male survives and is fertile, suggesting that lethality is associated with 7B2-3 breakpoint. The segregant *Dp(1;2)ct^{7c1} = Dp(1;2)7B2-3;8E2-3;25C* survives; duplicated for *sn* but not *cm*; male and female have darker, roof-like wings, enlarged abdomens, and are sterile.

****Tp(1;2)ct^{14a2}***

cytology: *Tp(1;2)7B2-4;19-20;41E1-2* superimposed on *R(1)1A3-4;19F-20A1*.

new order:

|1A4 – 7B2|20-20F – 20A1|;

21 – 41E1|7B4 – 19|41E2 – 60.

origin: X ray induced in *R(1)2*.

discoverer: Hannah, 1947.

genetics: Mutant for *ct* but not *cm*, *sn*, or *oc*. Male lethal.

Tp(1;2)ct^{14a2}/Dp(1;3)sn^{13al} male rarely survives, probably sterile.

****Tp(1;2)f²⁵⁷⁻¹⁵: Transposition (1;2) forked***

cytology: *Tp(1;2)13E9-10;15E2-3;24F* (Sutton).

origin: X ray induced.

discoverer: Demerec, 35a.

genetics: Mutant for *f* but not *M(1)15D* or *B*. Male lethal.

Tp(1;2)GF325

cytology: *Tp(1;2)2B7;7D;36C*.

origin: X ray induced.

discoverer: Lefevre.

genetics: Homozygous lethal. Mutant for *br*.

****Tp(1;2)l-v47: Transposition (1;2) lethal-variegated***

cytology: *Tp(1;2)8F-9B*; heterochromatic material inserted in X; genetic results suggest linkage between X and 2.

origin: X ray induced.

references: Lindsley, Edington, and Von Halle, 1960, *Genetics* 45: 1649-70.

genetics: Variegated for a lethal; *gg*-like phenotype.

****Tp(1;2)lt^{m16}: Transposition (1;2) light-mottled***

cytology: *Tp(1;2)11A;12F;22D;40B-F*.

new order:

1 – 11A|12F – 20;

21 – 22D|11A – 12F|40B – 22D|40F – 60.

origin: X ray induced.

references: Hessler, 1958, *Genetics* 43: 395-403.

genetics: Variegated for *lt*.

Tp(1;2)m^v: Transposition (1;2) miniature-variegated

cytology: *Tp(1;2)7C;10F;40-41*.

origin: X ray induced.

references: Lefevre, 1969, *Genetics* 63: 589-600.

genetics: Deficiency segregant lethal in heterozygous female. Duplication segregant *Dp(1;2)m^v* variegates for *m*; does not carry *fw*.

****Tp(1;2)N²⁶⁴⁻⁵⁰: Transposition (1;2) Notch***

cytology: *Tp(1;2)3C7-9;20C1-F;22A2-3* (Hoover).

new order:

1 – 3C7|20F;

21 – 22A2|3C9 – 20C1|22A3 – 60.

origin: X ray induced.

discoverer: Demerec, 37k.

references: 1941, *Proc. Intern. Congr. Genet.*, 7th., pp. 99-103.

genetics: Variegates for *fa* but not *w*, *rst*, or *dm*.

Tp(1;2)OR9: Transposition (1;2) from Oak Ridge

cytology: *Tp(1;2)3A;14F;41*.

new order:

1 – 3A|14F – 20;

21 – 41|14F – 3A|41 – 60.

origin: X ray induced in *y*.

discoverer: Wartens, 1959.

genetics: Male lethal; lethality not covered by *B^{S_w}+Y*; therefore, not associated with break at 3A.

Tp(1;2)pn1: Transposition (1;2) prune

cytology: *Tp(1;2)2E1-2;20A1-2;20D;43F* (Slobodyanyuk and Serov, 1983).

new order:

1 – 2E1|20D – 20F;

21A – 43F|20A2 – 20D|2E1 – 20A1|43F – 60F.

references: Ilyina, Sorokin, Belyaeva, and Zhimulev, 1980, *DIS* 55: 205.

Slobodyanyuk and Serov, 1983, *Mol. Gen. Genet.* 192: 372-77.

genetics: Homozygous lethal. Heterozygotes with *pn* are *pn*.

Tp(1;2)r⁺75c: Transposition (1;2) rudimentary-wild type

cytology: *Tp(1;2)14B13;15A9;35D-E*.

new order:

1 – 14B13|15A9 – 20;

21 – 35D|14B13 – 15A9|35E – 60.

origin: X ray induced.

discoverer: Lefevre.

references: Craymer and Roy, 1980, *DIS* 55: 200-04.

Ganetzky and Wu, 1982, *Genetics* 100: 597-614.

genetics: "Pebbly" eyes (Craymer and Roy, 1980) is the result of a *sl* mutation associated with the distal X breakpoint at 14B13 (Schalet, 1986, *Mutat. Res.* 163: 115-44). *Dp(1;2)* segregant covers *l(1)14Cc* (Jones and Rubin, 1990, *Neuron* 4: 711-23), probably through lethals in 15A.

Tp(1;2)rb⁺71g: Transposition (1;2) ruby-wild type

cytology: *Tp(1;2)3F3;5E8;23A15.*

new order:

1 – 3F3|5E8 – 20.

21 – 23A15|3F3 – 5E8|23A15 – 60.

origin: X ray induced.

discoverer: Lefevre.

synonym: *T(1;2)A1125.*

references: Craymer and Roy, 1980, DIS 55: 200-04.

Busson, Gans, Komitopoulou and Masson, 1983, Genetics 105: 309-25.

genetics: Females heterozygous for the deficiency segregant and males carrying the duplication segregant are lethal (Busson *et al.*, 1983). Duplication carries wild-type alleles of *vs* (Craymer and Roy, 1980), *l(1)5CDa* (Voelker and Wisely, 1982, DIS 58: 150-51), and *ovo* (Oliver, Perrimon, and Mahowald, 1987, Genes Dev. 1: 913-23).

Tp(1;2)sc¹⁹: Transposition (1;2) scute

cytology: *Tp(1;2)1B1-2;1B4-5;25A3-7* (Lefevre; García-Bellido, 1979).

new order:

1A – 1B1|1B5 – 20;

21 – 25A|(1B2 – 1B4)|25A – 60.

origin: X ray induced.

discoverer: League.

references: Muller, 1935, Genetica 17: 237-52.

García-Bellido and Santamaria, 1978, Genetics 88: 469-86.

García-Bellido, 1979, Genetics 91: 491-520.

genetics: Mutant for *sc*, showing slight suppression of scutellar chaetae (García-Bellido and Santamaria, 1978). A small subterminal piece of *X* is inserted into 2*L* one or two units to the right of *dp*. The two halves of the transposition can be recovered independently as *Df(1)sc19* and *Dp(1;2)sc¹⁹*. Homozygous clones in wing survive but fail to differentiate sensilla (Schubiger and Palka, 1985, Dev. Biol. 108: 399-410). *Df(1)sc19* is deficient for *y*, *ac*, *sc*, and *sc¹* but not *l(1)1Ac*, *om*, or *M(1)1B*; it is male lethal but survives in the heterozygous female. *Dp(1;2)sc¹⁹* carries, in addition to *sc¹⁹*, normal alleles of *y*, *ac*, and *sc¹*; it is viable homozygous and does not affect crossing over in 2*L*. X-ray-induced losses of *y⁺* from the transposed fragment are *M* and male and female sterile (Velissariou and Ashburner, 1980, Chromosoma 77: 13-27).

molecular biology: 1B4-5 breakpoint at coordinate -13.1 (Carramolino, Ruiz-Gómez, Guerrero, Campuzano, and Modolell, 1982, EMBO J. 1: 1185-91).

****Tp(1;2)sc²⁶⁰⁻²⁷***

cytology: *Df(1)1A8-B1;19F + T(1;2)15E;33-34;57B-C.*

new order:

1A1 – 1A8|19F – 20;

21 – 33|15E – 19F|1B1 – 15E|57B – 34|57C – 60.

origin: X ray induced.

references: Sutton, 1943, Genetics 28: 210-17.

genetics: The segregant *Df(1)sc260-27* separates as a free duplication from the segregant *Dp(1;2)sc²⁶⁰⁻²⁷*, which carries wild-type alleles of *y* and *ac*. The transposition as a whole is mutant for *sc* and male sterile.

Tp(1;2)sn⁺72d: Transposition (1;2) singed-wild type

cytology: *Tp(1;2)7A8;8A5;32C;58E.*

new order:

1 – 7A7|8A6 – 20;

21 – 32C|58E – 32C|(7A8 – 8A5)|58E – 60 (Helfand).

origin: X ray induced.

discoverer: Lefevre.

synonym: *T(1;2)FN107.*

references: Johnson and Judd, 1979, Genetics 92: 485-92.

Craymer and Roy, 1980, DIS 55: 200-04.

Lefevre, 1981, Genetics 99: 461-80.

genetics: Piece of the *X* carrying *ct⁺* - *ptg⁺* inserted into a pericentric inversion of 2. Males carrying the duplication segregant and a normal *X* are viable and fertile. Females with the deficiency segregant and a normal *X* are lethal.

Tp(1;2)v⁺65b: Transposition (1;2) vermilion

cytology: *Tp(1;2)10A1;11A7-8;40-41.*

new order:

1 – 10A1|11A8 – 20;

21 – 40|(10A1 – 11A7)|41 – 60.

origin: X ray induced.

synonym: *T(1;2)65b.*

references: Lefevre, 1969, Genetics, 63: 589-600.

Craymer and Roy, 1980, DIS 55: 200-04.

Lefevre, 1981, Genetics 99: 461-80.

genetics: Male viable and fertile with *v*-like eye color and outstretched wings (Lefevre, 1969).

**Tp(1;2)v⁺63i: see Dp(1;2)v⁺63i*

Tp(1;2)v⁺75d

cytology: *Tp(1;2)9A2;10C2;40-41.*

new order:

1 – 9A1|10C3 – 20;

21 – 40|(9A2 – 10C2)|41 – 60.

origin: X ray induced.

discoverer: Lefevre.

references: Craymer and Roy, 1980, DIS 55: 200-04.

genetics: Covers *v*.

Tp(1;2)w⁺: Transposition (1;2) white - wild type

origin: *y w⁺ f* female progeny from cross of *Dp(1;1)w⁺1* or *Dp(1;1)w⁺2* males marked with *sc z ec* to *C(1)DX*, *y w f* females.

references: Green, 1984, Mol. Gen. Genet. 194: 275-78.

genetics: *w⁺* transposed to 2 (*crm⁺* and *rst⁺* not affected).

transposition	genetic map position
<i>Tp(1;2)w⁺1</i>	2-24
<i>Tp(1;2)w⁺4</i>	2-76
<i>Tp(1;2)w⁺5</i>	2-80
<i>Tp(1;2)w⁺6</i>	2-31

****Tp(1;2)w⁺62g26***

cytology: *Tp(1;2)2D6-E1;4A1-2;40-41.*

new order:

1 – 2D6|4A2 – 20;

21 – 40|(2E1 – 4A1)|41 – 60.

origin: X ray induced.

discoverer: Robins, 62g26.

synonym: *T(1;A)pn-ec; T(1;2)w⁺62g26.*

references: Lefevre, 1963, DIS 37: 50.
1970, DIS 45: 39.

genetics: No variegation for w^+ . Male lethal. Deficiency segregant uncovers *pn* and *ec*; females carrying the deficiency over a normal X survive, though poorly viable and fertile. Duplication segregant does not cover *pn* (Lefevre, 1970); males carrying the duplication over a normal X are viable but sterile.

Tp(1;2)w⁺64b

cytology: *Df(1)5A1-2;20F* [superimposed on *In(1)w^{m4}* = *In(1)3C1-2;20F + T(1;2)4E3;26D7*, the latter involving the acentric ring concomitant of the *Df(1)*]. Fragment 3C2 through 5A1-2 broken into two pieces at 4E3 and the pieces, separated by an appreciable amount of proximal heterochromatin, inserted into 2L at 26D7.

new order:

1 – 3C1|20F – 5A2|20F;
21 – 26D7|4E3 – 3C2|20F|5A1 – 4E3|26D7 – 60.

origin: X ray induced in *In(1)w^{m4}*.

discoverer: Lefevre, 64b13.

references: 1968, DIS 43: 62-63.

Craymer and Roy, 1980, DIS 55: 200-04.

genetics: Males carrying the transposition are viable, fertile and wild type. X0 transposition males are viable but show *w* variegation. Females heterozygous for the deficiency are poorly viable, *N*, and almost sterile. Males carrying the duplication are lethal, but hyperploid females are viable and fertile (Lefevre, 1968).

****Tp(1;2)w^{m52b}: Transposition (1;2) white-mottled***

cytology: *Tp(1;2)1E5-F1;3C3-5;20F;40-41*.

new order:

1A – 1E5|20F – 3C5|20F;
21 – 40|(1F1 – 3C3)|41 – 60.

origin: X ray induced in *In(1)rst³* = *In(1)3C3-5;20F*.

discoverer: Ratty, 52b12.

references: Lefevre, 1953, DIS 27: 57.

genetics: Variegated for *w*. *Dp(1;2)w^{m52b}* survives.

****Tp(1;2)w^{m53a}***

cytology: *Tp(1;2)3B2-C1;3C9-D1;40-41*.

new order:

1 – 3B2|3D1 – 20;
21 – 40|(3C1 – 3C9)|41 – 60.

origin: X ray induced.

discoverer: Farnsworth, 53a4.

references: Lefevre, 1953, DIS 27: 57.

genetics: Variegated for *w*. The segregant *Dp(1;2)w^{m53a}* survives and is duplicated for the loci of *w*, *rst*, and *N*. *Df(1)w-m53a* survives as Notch female; deficient for *w*, *rst*, and *N*.

Tp(1;2)w^{m258-36}

cytology: *Tp(1;2)3C6-7;4C2-3;41A-B;41F5-6* (Demerec and Hoover).

new order:

1 – 3C6|(41B – 41F5)|4C3 – 20;
21 – 41A|(3C5 – 4C2)|41F6 – 60.

Insertions said to be in inverted order but not specified with respect to centromere or numerical order.

origin: X ray induced.

discoverer: Demerec, 38b.

references: Sutton, 1940, Genetics 25: 534-40 (fig.).

genetics: Variegated for *w* and *rst* but not *pn*, *fa*, or *dm*.

Male viable. Cytology predicts that each element of the translocation should survive as aneuploid but not so recorded.

Tp(1;2)w-ec: Transposition (1;2) white-echinus

cytology: *Tp(1;2)3C1-2;3E7-8;37D*.

new order:

1 – 3C1|3E8 – 20;
21 – 37D|(3C2 – 3E7)|37D – 60.

origin: X ray induced.

discoverer: Stafford, 64d20.

synonym: *T(1;2)(w-ec)^{64d}*; *T(1;2)NSt*; *Stafford-Notch*; *T(1;2)w^{64d}*; *T(1;2)w-ec*.

references: Lefevre and Wilkins, 1966, Genetics 53: 175-87.

Lefevre, 1968, DIS 43: 63.

Lefevre and Moore, 1968, Genetics 58: 557-71.

Craymer and Roy, 1980, DIS 55: 200-04.

genetics: Males carrying the transposition are viable, *w*, *ec*, and *N⁺* (Lefevre and Wilkins, 1966). Females heterozygous for the deficiency show *N*. Neither the duplication nor the deficiency complements *w^{sp}* (Lefevre, 1968) nor are *w ec* males or females with the duplication wild type.

Tp(1;2)y^{R3}: Transposition (1;2) yellow of Roberts

cytology: *Tp(1;2)1B2-3;1E2-3;35F*.

origin: X ray induced.

references: Roberts, 1974, Mutat. Res. 22: 139-44.

genetics: Mutant for *y*. Male viable and fertile.

Tp(1;2;3)pn³: Transposition (1;2;3) prune

cytology: *Tp(1;2)12E1-2;20A;41A + Tp(1;3)2E1-2;12E1-2;80C*.

new order:

1A – 2E1|20A2 – 20;
21A – 41A|12E2 – 20A1|41A – 60;
61A – 80C|2E2 – 12E1|80D – 100.

origin: X ray induced.

references: Ilyina, Sorokin, Belyaeva, and Zhimulev, 1980, DIS 55: 205.

Slobodyanyuk and Serov, 1983, Mol. Gen. Genet. 191: 372-77.

genetics: Mutant for *pn*. Homozygous lethal.

Tp(1;3)20A11

cytology: *Tp(1;3)5B;17D;81*.

new order:

1 – 5B|17D – 20;
61 – 81|5B – 17D|81 – 100.

origin: Induced by ethyleneimine.

references: Lim and Snyder, 1968, Mutat. Res. 6: 129-37.

genetics: Male lethal.

****Tp(1;3)A1: Transposition (1;3) from Austin***

origin: X ray induced.

discoverer: Muller, 1926.

references: Painter and Muller, 1929, J. Heredity 20: 287-98.

genetics: Breakpoints in X chromosome between *dm* and *ec* and between *car* and *bb*. Midsection of X transposed to 3R. Male hyperploid for *Dp(1;3)A1* survives; duplicated for loci of *y* through *dm* as well as *bb* (Schultz).

***Tp(1;3)B^{S3i}*: Transposition (1;3) Bar-Super inserted in chromosome 3**

cytology: *Tp(1;3)15F9-16A1;16A7-B1;20F;Y;66B13-C1* (Muller; Lindsley); translocation between *Dp(1;1)B = Dp(1;1)15F9-16A1;16A7-B1* and chromosome 3. X break can be shown genetically to separate *f⁺* from *B* and is assumed to separate the two halves of the Bar duplication.

new order:

1 – 16A7|20F·Y^S;
61 – 66B13|(16A1 – 20F)|66C1 – 100.
Tentative.

origin: Neutron induced in *X·Y^S, sc w B*.

discoverer: Norby.

references: Muller and Norby, 1949, DIS 23: 61.

genetics: Associated with *B^{S3i}*. Male viable. Homozygous female lethal. Chromosome 3 containing inserted X material survives as duplication in presence of normal X chromosomes; male sterile; female fertile. Duplication segregant *Dp(1;3)B^{S3i}* carries *bb⁺* (N. Scott) and has extreme *B* phenotype.

Tp(1;3)C92

cytology: *Tp(1;3)6E1-2;11D9-10;chrom3* (insertion of *6E1-2;11D9-10* into 3).

origin: X ray induced.

references: Lefevre and Peterson, 1972, DIS 48: 126-27.

genetics: Mutant for *sno*.

****Tp(1;3)ct^{11a}*: Transposition (1;3) cut**

cytology: *Tp(1;3)1B;7B2-3;8E-F;84B* superimposed on *R(1)1A3-4;19F-20A1*.

new order:

|1A4 – 1B|8E – 7B3|8F – 20·20F – 20A1|;
61 – 84B|(1B – 7B2)|84B – 100.

origin: X ray induced in *R(1)2*.

discoverer: Hannah, 1947.

genetics: Mutant for *ct* but not *y*, *ac*, *sc*, *cm*, *sn*, or *oc*. Male lethal. Female carrying *Dp(1;3)ct^{11a}* survives and has small eyes and arc-like wings with delta-like venation; duplicated for *cm*.

****Tp(1;3)ct^{12c1}***

cytology: *Tp(1;3)7B2-3;7D2-6;85* superimposed on *R(1)1A3-4;19F-20A1*.

new order:

|1A4 – 7B2|7D6 – 20·20F – 20A1|;
61 – 85|(7B3 – 7D2)|85 – 100.

origin: X ray induced in *R(1)2*.

discoverer: Hannah, 1947.

genetics: Mutant for *ct* but not *cm* or *sn*. Male lethal. The derived *Dp(1;3)ct^{12c1}* survives as female and as sterile male; duplicated for *sn*.

****Tp(1;3)ct²⁶⁸⁻³¹***

cytology: *Tp(1;3)3D2-3;7B2-5;84D4-5;86B4-C1;88F* (Hoover).

new order:

61 – 84D4|(3D3 – 7B2)|88F – 100;
remainder not described.

origin: X ray induced.

discoverer: Demerec, 38d.

genetics: Mutant for *ct* and *dm* but not *scp*, *sn*, or *fa*. Male lethal.

Tp(1;3)ct²⁶⁸⁻³⁷

cytology: *Tp(1;3)5D2-3;7B2-3;80C-F*.

new order:

1 – 5D2|7B3 – 20;
61 – 80C|7B2 – 5D3|80F – 100.

origin: X ray induced.

discoverer: Demerec, 39k.

references: Sutton, 1940, Genetics 25: 534-40 (fig.).

Demerec, 1940, Genetics 25: 618-27.

genetics: Mutant for *ct*; variegated for *rux* and *vs*; *shf*, *cm*, and *sn* not affected. The segregant *Dp(1;3)ct²⁶⁸⁻³⁷* viable and fertile in both male and female. Its complement, *Df(1)ct268-37 = Df(1)5D2-3;7B2-3*, survives as a Minute female; deficient for *M(1)5D6A*, *rux*, *vs*, *shf*, and *cm* but not *rg*, *cx*, *cv*, or *sn*; mutant for *ct*.

Tp(1;3)ct^{4B}

cytology: *Tp(1;3)4D1;7B3-4;92A*.

origin: X ray induced.

references: Lefevre and Johnson, 1973, Genetics 74: 633-45.

genetics: Male lethal.

***Tp(1;3)f⁺71b*: Transposition (1;3) forked-wild type**

cytology: *Tp(1;3)15A4;16C2-3;80-81*.

new order:

1 – 15A4|16C3 – 20;
61 – 80|15A4 – 16C2|81 – 100.

origin: X ray induced.

discoverer: Lefevre.

synonym: *T(1;3)A59; Tp(1;3)71b*.

references: Craymer and Roy, 1980, DIS 50: 200-04.

Ganetzky and Wu, 1982, Genetics 100: 597-614.

genetics: The segregant *Dp(1;3)f⁺71b* is duplicated for *M(1)15D⁺ - f⁺*.

Tp(1;3)HF308

cytology: *Tp(1;3)1E4;12A;81*.

origin: X ray induced.

discoverer: Lefevre.

genetics: Male lethal.

Tp(1;3)JC153

cytology: *Tp(1;3)16E2-4;17A-B;99D*.

origin: X ray induced.

references: Tanouye, Ferrus, and Fujita, 1981, Proc. Nat.

Acad. Sci. USA 78: 6548-52.

Baumann, Krah-Jentgens, Müller, Müller-Haltkamp, Seidel, Kecskemethy, Casal, Ferrus, and Pongs, 1987, EMBO J. 6: 3419-29.

molecular biology: Maps between 76 and 73 kb distal to the start site of the *Sh* chromosomal walk (Baumann et al., 1987).

Tp(1;3)m⁺82f*: see *Dp(1;3)m⁺82f

***Tp(1;3)N^{50k}*: Transposition (1;3) Notch**

cytology: *Tp(1;3)1E3-4;3C6-7;3C8-9;89A*.

new order:

1A1 – 1E3|3C9 – 20;
61 – 89A|(1E4 – 3C6)|89A – 100.
3C7-8 missing (Lefevre).

origin: X ray induced.

references: Lefevre, 1951, DIS 25: 71.
1952, DIS 26: 66.

Ratty, 1954, *Genetics* 39: 513-28.

genetics: Mutant for *N*. The segregant *Dp(1;3)N^{50k}* is viable and carries normal alleles of *pn*, *w*, and *rst*.

Tp(1;3)N²⁶⁴⁻⁵⁸

cytology: *Tp(1;3)3B2-3;3D6-7;80D-F* (Sutton).

new order:

1 – 3B2|3D7 – 20;

61 – 80D|3D6 – 3B3|80F – 100.

origin: X ray induced.

discoverer: Demerec, 38d.

synonym: *T(1;3)w^{m264-58}*.

references: 1940, *Genetics* 25: 618-27.

Sutton, 1940, *Genetics* 25: 534-40 (fig.).

Spofford, 1973, *DIS* 50: 98.

genetics: Variegates for *w*, *rst*, *N*, and its pseudoalleles (Cohen, 1962, *Genetics* 47: 647-59). Spofford, 1973, thinks *dm* phenotype result of extreme variegation; *dm/Dp(1;3)N²⁶⁴⁻⁵⁸* males in a Suppressor-of-variegation background are *dm⁺*. The segregant *Df(1)N²⁶⁴⁻⁵⁸* survives in heterozygous female and is deficient for *w*, *rst*, *fa*, and *dm* but not *pn* or *ec*. *Dp(1;3)N²⁶⁴⁻⁵⁸* survives as both male and female.

****Tp(1;3)N²⁶⁴⁻¹⁰⁰***

cytology: *Tp(1;3)3B4-C1;4B4-5;80* [Sutton, 1940, *Genetics* 25: 534-40 (fig.); Gersh, 1959, *Genetics* 44: 163-72].

new order:

1 – 3B4|4B5 – 20;

61 – 80|4B4 – 3C1|80 – 100.

origin: X ray induced.

references: Demerec, 1940, *Genetics* 25: 618-27.

genetics: Variegates for *w*, *rst*, *fa*, *dm*, and *ec* but not *pn* or *bi*. The segregant *Df(1)N²⁶⁴⁻¹⁰⁰*, which is deficient for *N*, survives in heterozygous female. *Dp(1;3)N²⁶⁴⁻¹⁰⁰* originally survived in female but not male; more recently, male carrying duplication found to survive (Gersh, 1959).

Tp(1;3)O4: Transposition (1;3) of Oliver

origin: X ray induced.

discoverer: Oliver, 29k24.

references: Dobzhansky and Schultz, 1934, *J. Genet.* 280: 373-77.

Oliver, 1937, *DIS* 7: 19.

genetics: X chromosome broken between *m* and *g* and between *f* and *car*. Center section of X then inserted into 3L. The segregant *Df(1)O4* is inviable when added to a normal male genotype, poorly viable when added to a normal female genotype, and survives well when added to a triploid intersex (2X:3A) genotype where it confers a low degree of fertility. The reciprocal segregant, *Dp(1;3)O4*, is lethal in the male, survives well in the female, and poorly in the intersexes.

Tp(1;3)pn²⁵: Transposition (1;3) prune

origin: X ray induced.

references: Ilyina, Sorokin, Belyaeva, and Zhimulev, 1980, *DIS* 55: 205.

genetics: Homozygous lethal. Heterozygotes with *pn* are *pn*.

transposition	cytology	orientation of inserted segment
<i>Tp(1;3)pn^{25α}</i>	2E1-2;20A1-2;70A5-6	eucentric
<i>Tp(1;3)pn²⁶</i>	2E1-2;20A1-2;70C1-2	eucentric

^α See also: Slobodyanyuk and Serov, 1983, *Mol. Gen. Genet.* 191: 372-77.

Tp(1;3)ras^v: Transposition (1;3) raspberry-variegated

cytology: *Tp(1;3)9E;13C;81F* (Lewis). Segment of X with *v⁺* inserted in centric heterochromatin of 3.

origin: Fast neutron induced.

discoverer: E.B. Lewis.

references: Brokaw, 1954, *DIS* 28: 73.

Barr, Valencia, and Plaut, 1968, *J. Cell Biol.* 39: 8a.

Tobler, Bowman, and Simmons, 1971, *Biochem. Genet.* 5: 111-117

1971, *Genetics* 68: s67.

genetics: Shows recessive variegation for *ras* and a rough eye and dominant variegation for a wing effect resembling *Bg/+*. No variegation for *m*, *vb*, *sd*, *sl*, or *un*. Is probably an enhancer of *B*; a few *ras^v/ras^v* females somewhat resemble *B/+*. *Dp(1;3)ras^v* male dies but female survives; duplicated for *ras*, *v*, *m*, *dy*, and *g* but not *un* or *r*. Tryptophane pyrrolase activity less when *v⁺* in translocated position (Tobler *et al.*, 1971). *Df(1)ras-v* is lethal in both sexes.

****Tp(1;3)SFV-70k***

cytology: *Tp(1;3)1D1-2;5C5-6;100D4-E1*. Segment of X inserted near 3R tip.

new order:

1A – 1D1|5C6 – 20;

61 – 100D4|5C5 – 1D2|100E1 – 100F.

origin: Induced by ethyl methanesulfonate.

references: Lefevre, 1971, *DIS* 47: 70.

genetics: Deficiency segregant dominant lethal. Duplication segregant female viable and fertile but male lethal; duplication carries *su(w^a)⁺* through *cv⁺*.

Tp(1;3)sn^{13a1}: Transposition (1;3) singed

cytology: *Tp(1;3)6C;7C9-D1;79D2-E1*; material inserted into chromosome 3.

new order:

|1A4 – 6C|7D1 – 20-20F – 20A1|;

61 – 79D2|(6C – 7C9)|79E1 – 100.

origin: X ray induced in *R(1)2*.

discoverer: Hannah, 1947.

references: Valencia, 1966, *DIS* 41: 58.

Hall and Kankel, 1976, *Genetics* 83: 517-35.

Lefevre, 1981, *Genetics* 99: 461-80.

genetics: Mutant for *sn*. The segregant *Dp(1;3)sn^{13a1}* survives and is duplicated for *cm* and *ct*.

Tp(1;3)sn^{19Bb5}

cytology: *Tp(1;3)3C1-2;7C9-10;72A-B* superimposed on *In(1)1B3-4;20F^L1B2-3;20F^R + In(1)4D7-E1;11F2-4*.

new order:

1A – 1B3|20F – 11F4|4E1 – 7C9|3C1 – 1B3|20F;

61 – 72A|(3C2 – 4D7)|11F2 – 7C10|72B – 100.

origin: X ray induced in *In(1)sc^{SILsc}8R + In(1)dI49*.

references: Valencia, 1966, *DIS* 41: 58.

genetics: Associated with *sn^{19Bb5}*; *w* not affected.

Tp(1;3)sta: Transposition (1;3) stubarista

cytology: *Tp(1;3)1D3-E1;2A;89B21-C4* (Lewis);

Tp(1;3)1E1-2;2B3-4;89B21-C4 (Belyaeva *et al.*, 1980).

new order:

1A – 1D3|2A – 20;

61 – 89B21|1E1 – 2A|89C4 – 100.

origin: X ray induced.

discoverer: Oliver, 32122.

references: 1935, DIS 4: 15.

Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelova, and Zhimulev, 1980, Chromosoma 81: 281-306.

genetics: Mutant for *sta* and *ss^a*. Male viable and fertile; homozygous female lethal. The segregant *Dp(1;3)sta* is viable. The complementary *Df(1)sta* is viable in heterozygous female either as *Df(1)sta/+*; *+/+* or as *Df(1)sta/Df(1)sta*; *Dp(1;3)sta/+* but the second type is sterile. *Df(1)sta* is deficient for *su(w^a)*; *Dp(1;3)sta* carries the *su(w^a)* locus (Rayle and Hoar, 1969, DIS 44: 94).

molecular biology: 2B3-4 breakpoint between coordinates 78 and 106.4 (Chao and Guild, 1986, EMBO J. 5: 143-50).

Tp(1;3)v⁺74c

cytology: *Tp(1;3)9E3-4;11B12;80-81*.

new order:

1 - 9E3|11B12 - 20;
61 - 80|(9E4 - 11B12)|81 - 100.

origin: X ray induced.

discoverer: Lefevre.

references: Craymer and Roy, 1980, DIS 55: 200-04.

Martin and Lefevre, 1981, Chromosoma 82: 237-47.

genetics: Duplication segregant *Dp(1;3)v⁺74c* includes *v⁺* and *Rp11215⁺*.

Tp(1;3)w^{68a}

cytology: *Tp(1;3)2F3-4;3C1-2;12A10-B1;12B8-9;77C*.

Heterochromatin inserted adjacent to 3C1.

new order:

1 - 2F3|12A10 - 3C2|12B9 - 20.
61 - 77C|het|3C1 - 2F4|77C - 100. Lacks 12B1-8.

references: Sorsa, Green, and Beerman, 1973, Nature (London) New Biology 245: 34-37.

genetics: Male lethal *w* variant, viable in compound with *Df(1)w258-45 = Df(1)3B2-3;3C2-3*; deficient for *crm* and *g*. Deficiency segregant lacks *z - w* and the *g* region, while the duplication segregant carries the wild-type allele of *z* but lacks *g⁺*.

Tp(1;3)w⁺

origin: *y w⁺ f* female progeny from cross of *Dp(1;1)w⁺1* or *Dp(1;1)w⁺2* males marked with *sc z ec* to *C(1)DX*, *y w f* females.

references: Green, 1984, Mol. Gen. Genet. 194: 275-78.

genetics: *w⁺* transposed to 3 (*crm⁺* and *rst⁺* not affected).

transposition	genetic map	
	position	
<i>Tp(1;3)w⁺2</i>	3-10	
<i>Tp(1;3)w⁺3</i>	3-28	
<i>Tp(1;3)w⁺7</i>	3-72	

****Tp(1;3)w⁺54a4***

origin: X ray induced.

synonym: *T(1;3)54a; Dp(1;3)w⁺54a4*.

references: Lefevre, 1955, DIS 29: 73.

genetics: Section of X including *w⁺* inserted into 3.

****Tp(1;3)w⁺54c10***

origin: X ray induced.

synonym: *T(1;3)54c; Dp(1;3)w⁺54c10*.

references: Lefevre, 1955, DIS 29: 73.

genetics: Section of X including *w⁺* inserted into 3.

Tp(1;3)w⁺67k

cytology: *Tp(1;3)3A4-5;3E8-F1;8E5;87E17*.

new order:

1A - 3A4|8E5 - 3F1|8E5 - 20.
60 - 87E17|3A5 - 3E8|87E17 - 100.

discoverer: Lefevre, 67k27.

synonym: *T(1;3)w^{67k27}*; duplication segregant: *G spot; Tp(1;3)67k*.

references: Lefevre, 1970, DIS 45: 32.

Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56.

Craymer and Roy, 1980, DIS 55: 200-04.

Gelbart and Wu, 1982, Genetics 102: 179-89.

genetics: The duplication segregant carries wild-type alleles of *l(1)3Ad* through *w* and *N* but not *ec*. It only partially suppresses *z*, leading to a brownish eye color in *z w⁺* flies (Gelbart and Wu, 1982).

Tp(1;3)w^c: Transposition (1;3) white-crimson

origin: Believed to be effect of transposing element, the distal part of the *w* locus being moved to chromosome 3.

references: Green, 1969, Genetics 61: 429-41.

Paro, Goldberg, and Gehring, 1983, EMBO J. 2: 853-60.

genetics: Male with *w* or *Df(1)w* is *w^c*. Transposition recessive to *w⁺*, complements *w^{sp}*, and is mutable (Green, 1969).

transposition	genetic location of <i>w^c</i>	chromosome of origin	viability and fertility
<i>Tp(1;3)w^c-1^α</i>	3-3.8	<i>w^c w^{sp}</i>	homozygous lethal
<i>Tp(1;3)w^c-2</i>	3-10.2	<i>w^c w^{sp}</i>	homozygous viable
<i>Tp(1;3)w^c-3</i>	3-46 (left of <i>ri</i>)	<i>w^c w^{sp}</i>	
<i>Tp(1;3)w^c-4</i>	3-92	<i>w^c w^{-67c}</i>	homozygous viable but male sterile

^α *In situ* hybridization of single copy *w* DNA to *Tp(1;3)w^c-1* locates the transposon at 62E; transposon associated with FB elements (Paro et al., 1983).

Tp(1;3)w^{m49a}: Transposition (1;3) white-mottled

cytology: *Tp(1;3)3A10-B1;3E2-3;80-81*.

new order:

1 - 3A10|3E3 - 20;
61 - 80|(3B1 - 3E2)|81 - 100.

origin: X ray induced.

discoverer: Lefevre, 49a7.

synonym: *T(1;3)w^{mSp}: Translocation (1;3) white-mottled Spotted*.

references: 1949, DIS 23: 59.

1951, DIS 25: 71.

Ratty, 1954, Genetics 39: 513-28.

genetics: Variegated for *w*, *rst*, and *spl*. The two elements of the translocation can be separated; *Df(1)w-m49a* survives in heterozygous female and is *N*; *Dp(1;3)w^{m49a}* survives in both male and female and carries the loci of *w*, *rst*, *N*, and (from the cytology) presumably *dm*.

***Tp(1;3)w^{vco}*: Transposition (1;3)
white-variegated cobbled**

cytology: *Tp(1;3)2B17-C1;3C5-6;77D3-5;81* (2C1-2 may not be included in transposed region according to Lefevre).

new order:

1A – 2B17|3C6 – 20;
61 – 77D3|2C1 – 3C5|81 – 77D5|81 – 100.

discoverer: Clausen.

references: Seecof, Kaplan, and Futch, 1971, Proc. Nat. Acad. Sci. USA 62: 528-35.

Bowman and Simmons, 1973, Biochem. Genet. 10: 319-31.

Lucchesi and Rawls, 1973, Genetics 73: 459-64.

Lucchesi, Rawls, and Maroni, 1974, Nature 248: 564-67.

Lefevre, 1981, Genetics 99: 461-80.

genetics: Insertion of small segment of X with *Pgd*⁺, *w*⁺, and *rst*⁺ into 3 (Lucchesi and Rawls, 1973). Variegated for *w* and *rst* and apparently mutant for *in* but *eg* not affected; *in* effect probably associated with 77D3-5 break (Arajarvi and Hannah-Alava, 1969, DIS 44: 73). Each element of the translocation survives as an aneuploid. *Df(1)w-vco* is deficient for recessives from *Pgd* through *w* and *rst*. *Df(1)w-vco/Tp(1;3)w^{vco}* is *w*, extreme *rst*, and highly infertile. *Dp(1;3)w^{vco}* covers *Pgd*, *w*, and *rst*.

***Tp(1;3)w^{zh}*: Transposition (1;3)
white-zeste halo**

cytology: *Tp(1;3)3C2-3;3C6;61D*.

origin: Spontaneous derivative of *w^{zm}* (Judd, 1975).

references: Judd, 1975, The Eukaryotic Chromosome (Peacock and Brock, eds.). Australian National University Press, Canberra, pp. 169-84.

Jack and Judd, 1979, Proc. Nat. Acad. Sci. USA 76: 1368-72.

Levis, Bingham, and Rubin, 1982, Proc. Nat. Acad. Sci. USA 79: 564-68.

genetics: Duplication segregant carries *w*⁺, *rst*⁺, and *vt*⁺. Deficiency segregant male lethal in absence of the duplication. *z* males and females when homozygous for *Tp(1;3)w^{zh}* have "halo" eyes (*z* in center, *w*⁺ on periphery) as do *z* females that are homozygous for *Dp(1;3)w^{zh}* (Jack and Judd, 1979). *z* males homozygous for *Dp(1;3)w^{zh}*, however, have *w*⁺ eyes as do *z* males and females heterozygous for *Dp(1;3)w^{zh}*. The transposition mutates frequently (Judd, 1975). Some mutations from *w^{zh}* show changes in the amount and distribution of eye pigment; other mutations involve lethality.

molecular biology: According to molecular data (Levis *et al.*, 1982), the white-associated breakpoints of *Df(1)w-zh* and *Dp(1;3)w^{zh}* are not identical. *Dp(1;3)w^{zh}* carries sequenced beginning between the coordinates -3.2 and -1.4 and extending rightward, with 0 the site of the copia insertion in *w^a*.

Tp(1;3)ZWD10

cytology: *In(1)4A;20 + Tp(1;3)3C6-7;92B*.

origin: γ ray induced.

references: Smolik-Utlaut and Gelbart, 1987, Genetics 116: 285-98.

genetics: Male lethal.

Tp(1;3)ZWD13

cytology: *Tp(1;3)3C1-3;9B;77E + Tp(1;3)16C;20;94B*.

origin: γ ray induced.

references: Smolik-Utlaut and Gelbart, 1987, Genetics 116: 285-98.

genetics: Male lethal.

****Tp(1;4)A1*: Transposition (1;4) from Austin**

cytology: *Tp(1;4)9B;20;101-102*.

new order:

1 – 9B|20;
101|9B – 20|102.

origin: X ray induced.

discoverer: Muller, 1928.

synonym: *CRB*; *W13*; *T(1;4)w¹³*; *T(1;4)I*.

references: Muller and Stone, 1930, Anat. Record 47: 393-94.

Muller and Painter, 1932, Z. Indukt. Abstamm. Vererbungsl. 62: 316-65.

Painter, 1934, Genetics 19: 448-69.

genetics: X chromosome broken between *lz* and *ras* and between *bb* and the centromere. The *Df(1)A1 = Df(1)9B;20* segregant dies in the egg stage, showing incomplete blastoderm formation, while the *Dp(1;4)A1 = Dp(1;4)9B;20;101-102* segregant dies later in the egg stage without separation of the germ layers (Poulson, 1940, J. Exp. Zool. 83: 271-325). Males with one entire X chromosome plus either segregant die, but females with two entire X's plus either segregant survive with low fertility (Muller and Stone, 1930). Triploid intersexes with either segregant also show very low fertility (Pipkin, 1940, Texas Univ. Publ. 4032: 126-56).

****Tp(1;4)ct^{13b1}*: Transposition (1;4) cut**

cytology: *Tp(1;4)7B2-3;20;101A-D* superimposed on *R(1)1A3-4;19F-20A1*.

new order:

|1A4 – 7B2|20:20F – 20A1|;
101A|7B3 – 20|101D – 102;

Tentative.

origin: X ray induced in *R(1)2*.

discoverer: Hannah, 1947.

genetics: Mutant for *ct* but not *y*, *ac*, *sc*, *cm*, *sn*, or *oc*. Male lethal.

****Tp(1;4)N²⁶⁴⁻⁸⁶***

cytology: *Tp(1;4)3C6-7;3C7-8;3E5-6;101F*.

new order:

1 – 3C7|3E6 – 20;
101A – 101F|3C7 – 3E5|101F – 102;

band 3C7 present twice and postulated to have been derived from each of two chromatids in the sperm (Demerec and Sutton, 1940, Proc. Nat. Acad. Sci. USA 26: 532-36).

origin: X ray induced.

discoverer: Demerec, 39i.

references: 1940, Genetics 25: 618-27.

Sutton, 1940, Genetics 25: 534-40 (fig.).

genetics: Supposedly carries two *N* loci, one mutant and one variegated. Also carries a mutant allele of *rst* (*rst²⁶⁴⁻⁸⁶*) and variegates for *dm* but not *w* or *ec*. Carries normal allele of *ci*⁺ (Stern). *Dp(1;4)N²⁶⁴⁻⁸⁶ = Dp(1;4)3C6-7;3E5-6;101F-102* viable and fertile in both sexes; *Df(1)N264-86 = Df(1)3C7-8;3E5-6* viable in heterozygote.

Tp(1;4)w^{+IV}

cytology: *Tp(1;4)3C2;102F*.
origin: Activity of transposing element.
references: Rasmuson, Montell, Svahlin, and Westerberg, 1980, *Mol. Gen. Genet.* 177: 567-70.
 Paro, Goldberg, and Gehring, 1983, *EMBO J.* 2: 853-60.
genetics: Transposition of *w^{spw17g}* to 4; shows *w^m* phenotype (Rasmuson *et al.*, 1980).
molecular biology: Seems to be associated with FB sequences. Located on 4 by *in situ* hybridization of single copy *w* DNA close to telomere of 4 at 102F.

Tp(1;4)w^{m51c}: Transposition (1;4) white-mottled

cytology: *Tp(1;4)3C1-2;3C4-7;20E-F;101*.
new order:
 1 – 3C1|20F – 3C7|20F;
 101|(3C2 – 3C4)|101 – 102.
origin: X ray induced in *In(1)w^{m4} = In(1)3C1-2;20F*.
discoverer: Lefevre, 51c20.
references: 1951, *DIS* 25: 71.
 1952, *DIS* 26: 66.
 Ratty, 1954, *Genetics* 39: 513-28.
genetics: Variegated for *w* and *rst*. Male lethal. *Dp(1;4)w^{m51c} = Dp(1;4)3C2-3;3C4-7;101* viable and fertile; carries loci of *w* and *rst* but not *spl*.

****Tp(1;4)w^{m52b13}***

cytology: *Tp(1;4)2A2-3;3C3-4;20F;101*.
new order:
 1 – 2A2|20F – 3C4|20F;
 101|(2A3 – 3C3)|101 – 102.
origin: X ray induced in *In(1)rst³ = In(1)3C3-4;20F*.
discoverer: Ratty, 52b13.
references: Lefevre, 1953, *DIS* 27: 57.
genetics: Variegated for *w*.

****Tp(2;1)260-31***

cytology: *Tp(2;1)9A;24;29*.
new order:
 1 – 9A|(24 – 29)|9A – 20;
 21 – 24|29 – 60.
origin: X ray induced simultaneously with *y²⁶⁰⁻³¹*.
discoverer: Fano, 1941.
references: Sutton, 1943, *Genetics* 28: 210-17.
genetics: Male lethal; lethality attributable to the independent mutation to *y²⁶⁰⁻³¹* since *Tp(2;1)260-31, y²⁶⁰⁻³¹/Df(1)sc260-25* is lethal.

Tp(2;1)429.65

cytology: *Tp(2;1)20F;21D1-2;25E3-4* (tentative).
new order:
 1A – 20F|(21D2 – 25E3)|20F;
 21A – 21D1|25E4 – 60.
discoverer: Gelbart.

Tp(2;1)B19

cytology: *Tp(2;1)9B14-C1;24D2-5;25F1-2*.
new order:
 1 – 9B14|25F1 – 24D5|9C1 – 20;
 21 – 24D2|25F2 – 60.
origin: X ray induced.
references: Reuter and Szidonya, 1983, *Chromosoma* 88: 277-85.
 Semeshin and Szidonya, 1985, *DIS* 61: 148-54.
 Szidonya and Reuter, 1988, *Genet. Res.* 51: 187-208.

genetics: The region transposed carried *ed-mid*.

Tp(2;1)C121: Transposition (2;1) Crossover suppressor

cytology: *Tp(2;1)20;35F;40*.
new order:
 1 – 20|(35F – 40)|20;
 21 – 35F|40 – 60.
origin: X ray induced.
discoverer: Roberts, 1965.
references: 1970, *Genetics* 65: 429-48.
genetics: Male viable and fertile. Recombination reduced in 2*L*.

Tp(2;1)C239

cytology: *Tp(2;1)7A-B;36C;39E*.
new order:
 1 – 7A|36C – 39E|7B – 20;
 21 – 36C|39E – 60.
origin: X ray induced.
discoverer: Roberts, 1965.
references: 1970, *Genetics*, 65: 429-48.
genetics: Male lethal. Recombination reduced in X chromosome. The segregant *Dp(2;1)C239 = Dp(2;1)7A-B;36C;39E* survives; carries *His* (Moore, Sinclair, and Grigliatti, 1981, *Genetics* 97: 75-76).

Tp(2;1)DTD2: Transposition (2;1) Disrupter of Transvection at Decapentaplegic

cytology: *Tp(2;1)20;22D4-5;23B1-2*.
new order:
 1 – 20|(22D5 – 23B1)|20;
 21 – 22D4|23B2 – 60.
origin: X ray induced.
references: Spencer, Hoffmann, and Gelbart, 1982, *Cell* 28: 451-61.
 Segal and Gelbart, 1985, *Genetics* 109: 119-43.
genetics: Disrupts transvection at *dpp*. *Df(2L)DTD2* lacks and *Dp(2;1)DTD2* carries the entire *dpp* locus.

Tp(2;1)I-v223

cytology: *Tp(2;1)14F;41;50E*.
new order:
 1A – 14F|(41 – 50E)|14F – 20;
 21A – 41|50E – 60.
origin: X ray induced.
discoverer: Lindsley, Edington, and Von Halle.
references: 1960, *Genetics* 45: 1640-70.
genetics: Variegated for a lethal and has defective external male genitalia; male sterile.

****Tp(2;1)It^{m31}***

cytology: *Tp(2;1)8F;28D;40B-F*.
new order:
 1 – 8F|28D – 40B|8F – 20;
 21 – 28D|40F – 60.
origin: X ray induced.
discoverer: Hessler, 1957.
references: 1958, *Genetics* 43: 395-403.
genetics: Variegated for *It*.

****Tp(2;1)N²⁶⁴⁻⁸⁰***

cytology: *Tp(2;1)3C6-7;36;40 + In(1)11;20* (Sutton).
new order:
 1 – 3C6|(36 – 40)|3C7 – 11|20 – 11|20;

21 – 36|40 – 60.

origin: X ray induced.

discoverer: Demerec, 39d.

references: 1941, Proc. Intern. Congr. Genet., 7th., pp. 99-103.

genetics: Contains mutant allele of *N* but normal alleles of *w*, *rst*, *dm*, and *ec*.

***Tp(2;1)N²⁶⁴⁻¹⁰²**

cytology: *Tp(2;1)3C6-7;50E;56C* (Sutton).

new order:

1 – 3C6|(50E – 56C)|3C7 – 20;

21 – 50E|56C – 60.

discoverer: Demerec, 39l.

genetics: Carries mutant allele of *N* and normal alleles of *w*, *rst*, and *dm*.

Tp(2;1)odd^{1.10}: Transposition (2;1) odd-skipped

cytology: *Tp(2;1)4A3-4;21A;24B*.

new order:

1 – 4A3|(21A – 24B)|4A4 – 20;

21A|24B – 60.

discoverer: Nüsslein-Volhard.

synonym: *T(1;2)odd^{1.10}*.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

genetics: Deficiency segregant uncovers *odd*.

Tp(2;1)OR19

cytology: *Tp(1;2)20;51F + In(2R)42B;48E;57C*.

new order:

1 – 20|51F – 48E|57C – 51F|20;

21 – 42B|48E – 42B|57C – 60E.

Three break rearrangement in chromosome 2 deletes 48E-57C and inverts 42B-48E; break between 20 on X and 51F of deleted acrocentric ring.

origin: X ray induced.

discoverer: Warters, 1961.

genetics: Male viable and weakly fertile. Homozygous female viable. *Dp(2;1)OR19 = Dp(2;1)20;48E;57C* survives in both male and female.

***Tp(2;1)sc²⁶⁰⁻²⁶**

cytology: *Tp(2;1)1B4-5;41F2-3;58B2-3 + In(2LR)27D2-3;41A*.

new order:

1A – 1B4|41F3 – 58B2|1B5 – 20;

21 – 27D2|41A – 27D3|41A – 41F2|58B3 – 60.

origin: X ray induced.

discoverer: Sutton, 39l.

references: 1943, Genetics 28: 210-17.

genetics: Mutant for *sc* but not *y*, *ac*, or *svr*.

Tp(2;1)Sco^{rv23}: Transposition (2;1) Scutoid

cytology: *Tp(2;1)h29;34F1-2;35B1-D1* superimposed on *Tp(2;2)Sco*. *Dp(2;1)Sco^{rv23}* visible in salivaries "floating" in the nucleolus (Ashburner *et al.*, 1981).

new order:

1 – h29|(34F2 – 35A4|(35C2 – 35C5)|34B4 – 35C1|35B3 – 35B1)|h29 – h34;

21 – 34F1|35D2 – 60.

origin: X ray induced in *Tp(2;2)Sco*.

discoverer: Littlewood.

synonym: *Tp(2;1)Sco^{R+23}*.

references: Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

Ashburner, 1982, Genetics 101: 447-59.

Ashburner, Detwiler, Tsubota, and Woodruff, 1983, Genetics 104: 405-31.

Ashburner and Harrington, 1984, Chromosoma 89: 329-37.

genetics: *Sco* revertant; homozygous lethal as transposition. *Dp(2;1)Sco^{rv23}* homozygotes and hemizygotes are viable and fertile; the duplication covers *l(2)34Fa – l(2)35Da* but not *sna* or *lace*, which are exposed by *Df(2L)Sco23*. The duplication is mutant for *noc* and is a strong enhancer of *H*. *Df(2L)Sco23* is deficient for *l(2)34Fa – lace* and is a dominant suppressor of *H* (Ashburner, 1982).

Tp(2;1)ZWD11

cytology: *Tp(2;1)3C7;41;56F*.

origin: γ ray induced.

references: Smolik-Utlaut and Gelbart, 1987, Genetics 116: 285-98.

genetics: Males *w*⁺ and sterile.

Tp(2;?)odd^{5.1}

cytology: *Tp(2;Y)23E;24E-F*; insertion position not known.

origin: X ray induced.

discoverer: Nüsslein-Volhard.

references: Nüsslein-Volhard, Kluding, and Jürgens, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 145-54.

genetics: Transposed element carries normal alleles of *odd* and *slp*.

Tp(2;?)shv^{S28}: Transposition (2;?) short vein

cytology: *Tp(2;?)22F1-2;24A1-2* inserted into chromosome (chromosome arm not known).

origin: γ ray induced.

references: Segal and Gelbart, 1985, Genetics 109: 119-43.

genetics: Associated with *shv^{S28}*. Lethal over *shv* and lethal *dpp* alleles.

Tp(2;Y)

Table I: *Tp(2;Y)b10-4-Tp(2;Y)t223*; γ ray induced recombinations of *Y* and chromosome 2 from *T(Y;2)*; recovered as fertile males in combination with *Df(1)bb158*.

CHROMOSOMES - TRANSPOSITIONS

transposition	cytology	new order α	source	ref β	comments
Tp(2;Y)b10-4	YL;36D;40A-F	$y^+YS-YL^P 40A - 36D YL^D B^S$; 21A - 36D 40F - 60F.	T(Y;2)b10	1, 2, 3	Sd E(SD) in YL of $B^S.y^+$
T(2;Y)b10-6	YL;35F;36D	$y^+YS-YL^P 36D - 35F YL^D B^S$; 21A - 35F 36D - 60F.	T(Y;2)b10	1, 3	
T(2;Y)b10-8γ	YL;35F;36D	$y^+YS-YL^P 36D - 35F YL^D$; 21A - 35F (B ^S) 36D - 60F.	T(Y;2)b10	1, 3	
Tp(2;Y)b10-9	YL;36D;40A-F	$y^+YS-YL^P 40A - 36D YL^D B^S$; 21A - 36D 40F - 60F.	T(Y;2)b10	1, 3	Sd E(SD) in YL of $B^S.y^+$
Tp(2;Y)b10-10	YL;36D;40A-F	$y^+YS-YL^P 40A - 36D YL^D B^S$; 21A - 36D 40F - 60F.	T(Y;2)b10	1, 3	Sd E(SD) in YL of $B^S.y^+$
Tp(2;Y)b10-11	YL;33A;38E-F	$y^+YS-YL^P 36D - 33A 38E - 36D YL^D B^S$; 21A - 33A 38F - 60F.	T(Y;2)b10	1, 3	Sd in YL of $B^S.y^+$
Tp(2;Y)b10-12	YL;34D-E;37D3-E1	$y^+YS-YL^P 36D - 34E 37D3 - 36D YL^D B^S$; 21A - 34D 37E1 - 60F.	T(Y;2)b10	1, 3	
Tp(2;Y)b10-20	YL;34A;40A-E	$y^+YS-YL^P 36D - 34A 40A - 36D YL^D B^S$; 21A - 34A 40F - 60F.	T(Y;2)b10	1, 3	Sd E(SD) in YL of $B^S.y^+$
Tp(2;Y)b10-21	YL;36D;40A-F	$y^+YS-YL^P 40A - 36D YL^D B^S$; 21A - 36D 40F - 60F.	T(Y;2)b10	1, 3	Sd E(SD) in YL of $B^S.y^+$
Tp(2;Y)b10-22	YL;34A;38B	$y^+YS-YL^P 36D - 34A 38B - 36D YL^D B^S$; 21A - 34A 38B - 60F.	T(Y;2)b10	1, 3	Sd in YL of $B^S.y^+$
Tp(2;Y)b10-23	YL;36D;40A-F	$y^+YS-YL^P 40A - 36D YL^D B^S$; 21A - 36D 40F - 60F.	T(Y;2)b10	1, 3	Sd E(SD) in YL of $B^S.y^+$
Tp(2;Y)b10-24	YL;35F;40A-F	$y^+YS-YL^P 36D - 35F 40A - 36D YL^D B^S$; 21A - 35F 40F - 60F.	T(Y;2)b10	1, 3	Sd E(SD) in YL of $B^S.y^+$
Tp(2;Y)b10-25	YL;35F;40A-F	$y^+YS-YL^P 36D - 35F 40A - 36D YL^D B^S$; 21A - 35F 40F - 60F.	T(Y;2)b10	1, 3	Sd E(SD) in YL of $B^S.y^+$
Tp(2;Y)b10-26	YL;30B;40F	$y^+YS-YL^P 36D - 30B 40F - 36D YL^D B^S$; 21A - 30B 40F - 60F.	T(Y;2)b10	1, 3	Sd E(SD) in YL of $B^S.y^+$
Tp(2;Y)b10-27	YL;36D;40A-F	$y^+YS-YL^P 40A - 36D YL^D B^S$; 21A - 36D 40F - 60F.	T(Y;2)b10	1, 3	Sd E(SD) in YL of $B^S.y^+$
Tp(2;Y)b10-29	YL;36D;38B	$y^+YS-YL^P 38B - 36D YL^D B^S$; 21A - 36D 38B - 60F.	T(Y;2)b10	1, 3	Sd in YL of $B^S.y^+$
Tp(2;Y)c53	YL;38A3;38E-F	$y^+YS-YL^P 38E - 38A3 YL^D B^S$; 21A - 38A3 38F - 60F.		3	Sd in YL of $B^S.y^+$
Tp(2;Y)cb50	YL;30C;33E			3	
Tp(2;Y)j13	YS;56C;56F	YL-YS ^P 56C - 56F YS ^D y ⁺ ; 21A - 56C 56F - 60F.	T(Y;2)L141	1, 3	
Tp(2;Y)j115	YS;55A;57C	YL-YS ^P 56F - 57C 55A - 56F YS ^D y ⁺ ; 21A - 55A 57C - 60F.	T(Y;2)L141	1, 3	
Tp(2;Y)j121	YS;54A;57B	YL-YS ^P 56F - 57B 54A - 56F YS ^D y ⁺ ; 21A - 54A 57B - 60F.	T(Y;2)L141	3	
Tp(2;Y)j129	YS;54A;56F	YL-YS ^P 54A - 56F YS ^D y ⁺ ; 21A - 54A 56F - 60F.	T(Y;2)L141	3	
Tp(2;Y)j134	YS;53D;56F	YL-YS ^P 53D - 56F YS ^D y ⁺ ; 21A - 53D 56F - 60F.	T(Y;2)L141	3	
Tp(2;Y)j136	YS;53D;57C	YL-YS ^P 56F - 57C 53D - 56F YS ^D y ⁺ ; 21A - 53D 57C - 60F.	T(Y;2)L141	3	
Tp(2;Y)ra38	YL;36B-E;40A-F			3	Sd E(SD) into $B^S.YL$
Tp(2;Y)j221δ	YL;36E;37E-F	YS-YL ^P 37E - 36E YL ^D ; 21A - 36E (kl?) 37F - 60F.	T(Y;2)j22	1, 3	Sd in YL
Tp(2;Y)j223δ	YL;33B;36E	YS-YL ^P 36E - 33B YL ^D ; 21A - 33B (kl?) 36E - 60F.	T(Y;2)j22	1, 3	Sd in YL

α Some material from the Y may be retained within the reconstituted second chromosome in those new orders with but two breakpoints indicated.
 β 1 = Lyttle, 1984, Genetics 106: 423-34; 2 = Lyttle, 1986, Genetics 114: 203-16; 3 = Lyttle, 1988.
 γ B^S inserted into resealed autosome (Lyttle, 1984).
 δ Fertility factor(s) inserted into resealed autosome (Lyttle, 1984).

Table II: *Tp(2;Y)A151-Tp(2;Y)R70*; X ray induced in $B^S Y y^+$.

transposition	cytology	ref α	X/T(Y;2) fertility
Tp(2;Y)A151	41;42E-F	1	-
Tp(2;Y)B127	49C;49F	2	+
Tp(2;Y)B231	27D;31E	2	+
Tp(2;Y)D203	47A-B;59	1	-
Tp(2;Y)D208	21F;36F	2	+
Tp(2;Y)G44	44C;50B	2	-
Tp(2;Y)H64	41;57D	2	-
Tp(2;Y)J54	35A;40	2	+
Tp(2;Y)J64	56F;57A	2	-
Tp(2;Y)L12	41A;43E	2	+
Tp(2;Y)R31β	56F;57D	2	-
Tp(2;Y)R70	21C3;21C5	1,3	-

α 1 = Baker, 1980, DIS 55: 197; 2 = The Seattle-La Jolla Drosophila Laboratories, 1971, DIS 47, Suppl.
 β *Df(2;Y)R31* carries Pu^+ .

***Tp(2;Y)bw⁺*: Transposition (Y;2) brown-wild type**

cytology: *Tp(2;YL);58F1-59A2;60E3-F1* (Gersh, 1956, DIS 30: 115; Nicoletti).

new order:
 $YL^D | (59A2 - 60E3) | YL^P - YS$;
 21 - 58F1 | 60F1 - 60F5.

origin: X ray induced.

discoverer: Dempster.

references: Brosseau, Nicoletti, Grell, and Lindsley, 1961,

Genetics 46: 339-46.

genetics: *Dp(2;Y)bw⁺* carries loci from *bw* through *ba* but not *hv* or *M(2)60E*; it is used as a marked *Y* and referred to as *bw⁺Y*. *Df(2R)bw* lost.

Tp(2;Y)C

cytology: [*Tp(2;Y)41A;43*]. Breaks in *2R* just to left of 41A (Whittinghill, 1937, DIS 8: 82-84) and distal to *cn* (located in 43E by Zacharopoulou, Yannopoulos, and Stamatis, 1981, DIS 56: 166-67).

origin: X ray induced.

discoverer: Dobzansky, 1929.

synonym: *T(Y;2)C*; *cn⁺Y* [= *Dp(2;Y)C*].

references: 1930, Biol. Zentr. 50: 671-85.

1932, Z. Indukt. Abstamm. Vererbungsl. 60: 235-86.

Sullivan, Kitos, and Sullivan, 1973, Genetics 75: 651-61.

genetics: Duplication segregant *Dp(2;Y)C* carries *cn⁺* (Sullivan *et al.*, 1973). Translocation heterozygotes over *cn* viable and fertile, the males with wild-type and the females with *cn* eyes.

***Tp(2;Y)en^{SF32}*: Transposition (2;Y) engrailed**

cytology: *Tp(2;Y)48A*.

references: Kuner, Nakanishi, Ali, Drees, Gustavson, Theis, Kauvar, Kornberg, and O'Farrell, 1985, Cell 42: 309-16.

genetics: Homozygous lethal. Associated with *en^{SF32}*.

molecular biology: DNA breakpoint at about -2 kb, *i.e.*, 2 kb to the left of the *en¹* insertion point.

Tp(2;Y)G

cytology: *Tp(2;Y)36B5-C1;40F*; metaphase chromosomes appear normal (Morgan, Bridges, and Schultz, 1935, Year Book - Carnegie Inst. Washington 34: 287).

new order:

Y|(36C1 - 40F)|Y;

21 - 36B5|40F - 60.

origin: X ray induced.

discoverer: Dobzhansky, 1929.

references: 1930, Biol. Zentr. 50: 671-85.

Rhoades, 1931, Genetics 16: 490-504.

Yim, Grell, and Jacobsen, 1977, Science 198: 1168-70.

Nakamura, Gay, and Contamine, 1986, Biol. Cell. 56: 227-38.

genetics: *Dp(2;Y)G* has normal phenotype and is fertile when hyperploid in either sex; duplicated for the loci of *M(2)36F*, *M(2)39F*, *hk*, *pr*, *Bl*, *lt*, and *bw^{V32g}* but not *rd*; covers the deficiency segregant of *Tp(2;Y)b10-4* and includes *Sd⁺* and *E(SD)⁺* (Lyttle, 1986, Genetics 114: 203-16).

Tp(2;Y)G-H1: see *Dp(2;Y)H1*

Tp(2;Y)G-H2: see *Dp(2;Y)H2*

Tp(2;Y)G-H3: see *Dp(2;Y)H3*

Tp(2;Y)G-M15

cytology: *Tp(2;Y)36B5-C;37E;40F*. Heterochromatin from the *Y* inserted in the transposed segment of 2.

origin: X ray induced in *Tp(2;Y)G*.

new order:

Y|36C1 - 37E|Y|37E - 40F|Y;

21 - 36B5|40F - 60.

references: Nakamura, 1973, Thesis, Paris-Sud.

1978, Mol. Gen. Genet. 159: 285-92.

Nakamura, Gay, and Contamine, 1986, Biol. Cell. 56:

227-38.

genetics: Duplication segregant carries *M(2)36F⁺* - *hk⁺* and *pr⁺* - *M(2)39F⁺* but not *ref(2)P⁺*.

****Tp(2;Y)H***

cytology: *Tp(2;Y)37B1-2;40B2-3*; also an inversion in *2R* from near centromere to left of *px* (Morgan, Bridges, and Schultz, 1935, Year Book - Carnegie Inst. Washington 34: 287).

new order:

Y^D|(37B2 - 40B2)|Y^P;

21 - 37B1|40B3 - | - | - 60.

origin: X ray induced.

discoverer: Dobzhansky, 1929.

references: 1930, Biol. Zentr. 50: 671-85.

Schultz and Bridges, 1932, Am. Nat. 66: 323-34.

genetics: Male fertile. Homozygote viable and male sterile. *Df(2L)H* survives and is deficient for *M(2)39F*, *hk*, and *pr* but not *M(2)36F* or *lt*; somewhat sterile. *Dp(2;Y)H* appears normal; duplicated for the loci for which *Df(2L)H* is deficient.

***Tp(2;Y)odd*: Transposition (2;Y) odd-skipped**

discoverer: Nüsslein-Volhard.

references: Nüsslein-Volhard, Wieschaus and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

Nüsslein-Volhard, Kluding, and Jürgens, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 145-54.

transposition	cytology	associated with
<i>Tp(2;Y)odd 2.31</i> ^α	21A;25C;38B	<i>odd</i> , <i>slp</i>
<i>Tp(2;Y)odd 4.13</i>	22A;25F	<i>odd</i> , <i>mid</i> , <i>slp</i>
<i>Tp(2;Y)odd 4.25</i>	21A;24D	<i>odd</i> , <i>slp</i>

^α New order: Y|(21A - 25C)|Y;
21A|38B - 25C|38B - 60.

***Tp(2;Y)pr^{C5}*: Transposition (2;Y) purple**

origin: Induced by ethyl methanesulfonate.

references: Yim, Grell, and Jacobsen, 1977, Science 198: 1168-70.

Tobler, Yim, Grell, and Jacobsen, 1979, Biochem. Genet. 17: 197-206.

genetics: Variegates for *pr*.

***Tp(2;Y)prd*: Transposition (2;Y) paired**

cytology: *Df(2L)33A1-2;34F1-2 Df(2L)33A1-2;34F1-2 + T(Y;2)33F2-3*.

new order:

Y|33F2 - 34F1|33A2 - 33F1|Y;

21 - 33A1|34F2 - 60.

synonym: *T(Y;2)prd*.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.

genetics: Deficiency segregant [*Df(2L)prd*] uncovers *prd* - *sal*.

****Tp(2;Y)R24***

cytology: *Tp(2;Y)45A;51E*.

new order:

Y^D|(45A - 51E)|Y^P;

21 - 45A|51E - 60.

origin: X ray induced.

discoverer: Slatis.

references: 1955, Genetics 40: 8.

genetics: Induced simultaneously with (but independently

of *bw*²⁴, an isoallele of *bw*. Associated with a rough eye phenotype. *Dp(2;Y)R24*-bearing males viable and sterile.

Tp(2;Y)Rsp: Transposition (2;Y) Responder

cytology: Insertion of proximal heterochromatin of 2R into *B^SYy⁺* between *B^S* and *kI5*.

origin: X-ray-induced reconstitution of intact Y and second chromosomes from *T(Y;2)cb25 = T(Y;2)B^SXh;41A*.

references: Lyttle, 1984, Genetics 106: 423-34.

Lyttle and Ault, 1985, Genetics 110: s23.

Lyttle, 1989, Genetics 121: 751-63.

properties: A series of reconstitutions that result in the insertion of variable amounts of 2R heterochromatin into *YL* and the deletion of that amount from 2R. Both 2R and Y respond independently to *SD* and in proportion to the number of *Rsp* sequences that they carry. Each of the segregants, *Dp(2;Y)Rsp (=B^SRspYy+)* or *Df(2R)Rsp*, survives in the absence of the other and can be studied individually.

chromosome	cytology	genetics	k_Y^α	k_A^α
<i>Tp(2;Y)Rsp1</i>	<i>h38-h40</i>	<i>l(2)41Aa-</i>	0.804	
<i>Tp(2;Y)Rsp4</i>	<i>h38-h44</i>	<i>l(2)41Aa⁺-l(2)41Af⁺</i>	0.956	0.593
<i>Tp(2;Y)Rsp22</i>	<i>h38-h43</i>	<i>l(2)41Aa⁺-r1⁺</i>	0.961	0.514
<i>Tp(2;Y)Rsp24</i>	<i>h38-h39</i>	<i>l(2)41Aa-</i>	0.885	0.479
<i>Tp(2;Y)Rsp29</i>	<i>h38</i>	<i>l(2)41Aa-</i>	0.606	0.998
<i>Tp(2;Y)Rsp31</i>	<i>h38</i>	<i>l(2)41Aa-</i>	0.623	0.979
<i>Tp(2;Y)Rsp42</i>	<i>h38</i>	<i>l(2)41Aa-</i>	0.599	0.973

^α k_Y is the k value indicating the response to *SD* of the Responder sequences inserted into the Y, whereas k_A reflects the response of the Responder sequences remaining in the reconstituted second chromosome.

Tp(2;Y)vg^{76d2}

cytology: *Tp(2;Y)58B + Dp(2R)58B-D*.

origin: Caffeine and γ rays.

references: Alexandrov and Alexandrova, 1987, DIS 66: 185-87.

genetics: Mutant for *vg*.

Tp(2;2)433.56

cytology: *Tp(2;2)28D2-3;29E1-2;40-41*.

new order:

21 - 28D2|29E2 - 40|(28D3 - 29E1)|41 - 60.

discoverer: Gelbart.

Tp(2;2)A446

cytology: *Tp(2;2)35F1-2;36C1-2;49B1-7 + Df(1)35B1-3;35F1-2*.

new order:

21 - 35B1|36C2 - 49B1|35F2 - 36C1|49B3 - 60F.

origin: X ray induced.

discoverer: Aaron.

synonym: *Df(2L)A446*.

references: Ashburner, Aaron, and Tsubota, 1982, Genetics 102: 421-35.

Gubb, Roote, McGill, Shelton, and Ashburner, 1986, Genetics 112: 551-75.

genetics: *Df(2L)A446 = Df(2L)35B1-3;35F1-2* is deficient for *noc - l(2)35Eb*, and heterozygotes are semi-lethal; escapers are sterile and have a wavy-wing and small-fly phenotype. *Dp(2;2)A446* (recombinant between *Tp(2;2)A446* and a wild-type 2) is viable.

Tp(2;2)b: Transposition (2;2) black

origin: γ ray induced.

references: Alexandrov and Alexandrova, 1986, DIS 63: 159-61.

Alexandrova, 1986, DIS 63: 21.

genetics: Mutant for *b* but not for *nub* or *j*. Homozygous lethal.

transposition	cytology
<i>Tp(2;2)b^{71k1}</i>	<i>34D2-4;34D8-E1;43C2-4</i>
<i>Tp(2;2)b^{79h1}</i>	<i>34D2-4;34D8-E1;41</i>
<i>Tp(2;2)b^{81a}</i>	<i>34D2-4;34D8-E1;41D-E1</i>

Tp(2;2)C123: Transposition (2;2)

Crossover suppressor

cytology: *Tp(2;2)23D-E;38C;39A3*.

new order:

21A - 23D|38C - 39A3|23E - 38C|39A3 - 60.

discoverer: Roberts.

synonym: *In(2L)C123*.

references: 1970, Genetics 65: 429-48.

genetics: Crossing over reduced in 2L.

Tp(2;2)CA30

cytology: *Tp(2;2)26C1;27D1;34A4-B1*.

origin: γ ray induced.

discoverer: Ashburner.

other information: Recovered with *Df(2R)pk78k*.

Tp(2;2)dpp: Transposition (2;2)

decapentaplegic (W.M. Gelbart)

references: Spencer, Hoffmann, and Gelbart, 1982, Cell 28: 451-61.

Segal and Gelbart, 1985, Genetics 109: 119-43.

Irish and Gelbart, 1987, Genes Dev. 1: 868-79.

St. Johnston, Hoffmann, Blackman, Segal, Grimaila,

Padgett, Irick, and Gelbart, 1990, Genes Dev. 4: 1114-27.

alleles:

transposition	class	origin	cytology	discov.
<i>Tp(2;2)dpp¹¹</i>	<i>d-III</i>	X-ray	<i>22F1-2;29C;32C-D;39B^α</i>	Spenser
<i>Tp(2;2)dpp¹³</i>	<i>d-V</i>	X-ray	<i>22F1-2;24C1-2;37F;40^β</i>	Spenser
<i>Tp(2;2)dpp²¹</i>	<i>d-V</i>	X-ray	<i>22A2-3;22F1-2;52F</i>	Spenser
<i>Tp(2;2)dpp⁷⁰</i>	<i>d-V</i>	γ-ray	<i>21A;21D;22F^γ</i>	Segal
<i>Tp(2;2)dpp⁷⁴</i>	<i>d-III</i>	γ-ray	<i>22F1-2;26A-B;29D-E^δ</i>	Segal
<i>Tp(2;2)dpp^{s7}</i>	<i>shv-lc</i>	X-ray	<i>22F1-2;24E1-2;25A1-2</i>	Segal

^α 21-22F1|32D-39B|22F2-29C|32C-29C|39B-60.

^β 21-22F1|24C2-37F|22F2-24C1|40-37F|40-60.

^γ 21-21A|22F-21D|21A-21D|22F-60.

^δ 21-22F1|26B-29D|22F2-26A|29E-60.

Tp(2;2)JHIP1

cytology: *Tp(2;2)26A;29B;34D*.

origin: Spontaneous in *JH* chromosome line during successive backcrosses.

references: Yamaguchi and Mukai, 1974, Genetics 78: 1209-21.

Tp(2;2)Mg: Transposition (2;2) Mglinetz

origin: γ ray induced.

references: Mglinetz, 1971, Genetika (Moscow) 7(8): 108-14.

transposition	cytology	new order
<i>Tp(2;2)Mg5</i>	<i>58F;59D;60E</i>	<i>21-58F 59D-60E 58F-59D 60E-60F</i>
<i>Tp(2;2)Mg9</i>	<i>45D;50B;53F</i>	<i>21-45D 50B-53F 45D-50B 53F-60</i>
<i>Tp(2;2)Mg10</i>	<i>33B;35B;40-41</i>	<i>21-33B 35B-40 33B-35B 41-60</i>

transposition cytology new order

***Tp(2;2)MS5: Transposition (2;2)
Mglinetz Semenova*****cytology:** *Tp(2;2)27D;33A;50F*.**new order:**

21A – 27D|33A – 50F|(27D – 33A)|50F – 60.

origin: γ ray induced.**references:** Semenova, Mglinetz, and Glotoff, 1970, *Genetika (Moscow)* 6(4): 165-69.Mglinetz and Semenova, 1970, *Genetika (Moscow)* 6(8): 86-94.***Tp(2;2)Pu^{RP1}: Transposition (2;2) Punch*****cytology:** *Tp(2;2)25C-D;40-41;57C4-5*.**new order:**

21 – 25C|41 – 57C4|40 – 25D|57C5 – 60.

origin: Induced by ethyl methanesulfonate.**references:** Mackay and O'Donnell, 1983, *Genetics* 105: 35-53.Mackay, Reynolds, and O'Donnell, 1985, *Genetics* 111: 885-904.O'Donnell, Boswell, Reynolds, and Mackay, 1989, *Genetics* 121: 273-80.**genetics:** Associated with *Pu*. Recessive eye color. GTP cyclohydrolase activity lost in eye but present elsewhere. Homozygous lethal.***Tp(2;2)RBR*****origin:** Spontaneous [accumulated on second chromosome lines during repeated backcrossing of *In(2LR)bw^{V1/+}* males to *In(2LR)bw^{V1/SMI}* females].**references:** Yamaguchi, Cardellino, and Mukai, 1976, *Genetics* 83: 409-22.

transposition	cytology
<i>Tp(2;2)RBR3</i>	45F;47C;57D
<i>Tp(2;2)RBR33</i>	28C;32D;57A
<i>Tp(2;2)RBR49</i>	26E;34A;57C
<i>Tp(2;2)RBR66</i>	35B;45F;51E
<i>Tp(2;2)RBR117</i>	45A;47F;50F + <i>In(2R)42A;59E</i>
<i>Tp(2;2)RBR141</i>	42B;47A;57C

Tp(2;2)Sco: Transposition (2;2) Scutoid**cytology:** *Tp(2;2)35A4-B1;35B3-4;35C1-2;35C5-D1* or *In(2L)35A4-B1;35C5-D1* + *In(2L)35B3-4;35C1-2*. Inferred from cytogenetic data.**new order:**

21 – 35A4|(35C2 – 35C5)|35B4 – 35C1|35B3 – 35B1|35D2 – 60.

origin: X ray induced.**synonym:** *Sco*; *Tp(2;2)Sco1* *Tp(2;2)Sco2*.**references:** Maroni, 1980, *DIS* 55: 96-98.Ashburner, Tsubota, and Woodruff, 1982, *Genetics* 102: 401-20.Ashburner, Detwiler, Tsubota, and Woodruff, 1983, *Genetics* 104: 405-31.Ashburner and Harrington, 1984, *Chromosoma* 89: 329-37.**genetics:** Homozygous lethal. Mutant for *Sco*, with phenotype enhanced by *noc* mutations and suppressed by *noc*⁺ duplications. *Adh^F* carried on *Tp(2;2)Sco*. Gene sequence as follows: – *noc*|*rd* – *l(2)35Da*|*l(2)35Bb* – *l(2)35Cb*|*Adh* – *noc*|*sna* –.**molecular biology:** *Adh-noc* region cloned (Chia, Karp, McGill, and Ashburner, 1985, *J. Mol. Biol* 186: 689-706) (coordinate 0 an *EcoRI* restriction site 1321 bp to the left of the start of transcription of the larval *Adh* transcript; positive values toward the centromere). The distal breakpoint of the *Sco* rearrangement is within the *noc* gene complex between the *nocA* and *nocB* components, such that *nocB* and *nocC* are brought into juxtaposition with *sna*. The molecular coordinates are complex, in that the distal break point as judged from the uninverted portion of *noc* is between coordinates -103.3 and -102.3; as judged from the *noc-sna* junction, however, it is between coordinates -108.5 and -107.8 kb, very close to the -107.8 position. Both these breakpoints are between *nocA* and *nocB*; the *noc-Adh* transposed segment extends from -107.8 to at least +40 kb (McGill, Chia, Karp, and Ashburner, 1988, *Genetics* 119: 647-61). The proximal-most breakpoint of *Tp(2;2)Sco* is within 16 kb to the left of *sna* (Alberga).***Tp(2;2)Sco^{rv1}-DV1*****cytology:** *Tp(2;2)44C3-4;48A5;49F7* superimposed on the *In(2LR)35D1-2;44C3-5* revertant of *Sco*.**new order:**

21 – 35A4|(35C2 – 35C5)|34B4 – 35C1|35B3 – 35B1|44C1 – 35D2|48A5 – 49F7|44C4 – 48A5|49F7 – 60|.

origin: γ -ray induced reversion of the lethal effect of *In(2LR)Sco^{rv1}* in combination with *noc^{TE35A}*.**references:** Ashburner and Harrington, 1984, *Chromosoma* 89: 329-37.***Tp(2;2)Sco^{rv12}: Transposition (2;2)
Scutoid-revertant*****cytology:** *Tp(2;2)34A4-B1;34F2-4;35E1-2* superimposed on *Tp(2;2)Sco*.**new order:**

21 – 34A8|35E1 – 35D2|35B1 – 35B3|35C1 – 35B4|35C5 – 35C2|35A4 – 35F4|34B1 – 34F2|35E2 – 60.

origin: X ray induced.**discoverer:** Ashburner.**synonym:** *Sco^{R+12}*.**references:** Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, *DIS* 55: 193-95.Ashburner, Detwiler, Tsubota, and Woodruff, 1983, *Genetics* 104: 405-31.Ashburner and Harrington, 1984, *Chromosoma* 89: 329-37.**genetics:** Revertant of *Sco*. Homozygous lethal.***Tp(2;2)shv^{S7} Transposition (2;2) shortvein*****cytology:** *Tp(2;2)22F1-2;24E1-2;25A1-2*.**new order:**

21A – 22F1|25A1 – 24E2|22F2 – 24E1|25A2 – 60.

origin: X ray induced.**references:** Segal and Gelbart, 1985, *Genetics* 109: 119-43.**genetics:** Associated with *shv^{S7}*. Lethal over *shv* and lethal *dpp* alleles.***Tp(2;2)TE35A: Transposition (2;2)
Transposable Element*****cytology:** *Tp(2;2)35A4-B1;35C1;38F*.**new order:**

21 – 35A4|35C1 – 38F|35B1 – 35C1|38F – 60.

origin: γ ray induced in *TE35A*.**discoverer:** Wilkins.**synonym:** *Tp(2;2)TE146Z*.

Tp(2;2)vg: Transposition (2;2) vestigial

references: Alexandrov and Alexandrova, 1987, DIS 66: 185-87.

transposition	cytology	origin	phen. under 25°C
<i>Tp(2;2)vg 76j1</i>	49D;49E;50F	actinomycin-D + γ ray	lethal
<i>Tp(2;2)vg 77d2</i>	49B4;49B5-12;49D2-E	0.35 MeV neutrons	vg ^s
<i>Tp(2;2)vg 78j1</i>	49D2-3;49D7-E1;50C1-6	γ ray	vg ^{nw}
<i>Tp(2;2)vg 79b6</i>	49D2;49E1;50C9-14	0.35 MeV neutrons	lethal
<i>Tp(2;2)vg 79f1</i>	36D;41A;49E;53F;54A	NaF + γ ray	lethal
<i>Tp(2;2)vg 85e2</i>	41B;49E;55F	γ ray	vg ^{nw}

Tp(2;3)17

cytology: *Tp(2;3)31B1;33F1-2;34A1-2;34D3-4;76B2-C1*.

new order:

21 – 31B1|33F2 – 34A1|34D4 – 60;
61 – 76B2|34A2 – 34D3|31B1 – 33F1|76C1 – 100.

discoverer: Ising.

references: Struhl, 1981, Nature 293: 36-41.

genetics: Includes *esc* (Struhl), *l(2)34Ca*, and *l(2)34Db* but not *l(2)34De* – *l(2)34Ea* or more proximal loci (Ashburner).

Tp(2;3)707

discoverer: Ising.

references: Gubb, 1985, Wilhelm Roux's Arch. Dev. Biol. 194: 236-46.

genetics: Duplication segregant carries *en*⁺.

Tp(2;3)Ba^G Transposition (2;3) Brista

cytology: *Tp(2;3)52E;60E;81*.

synonym: *Dll^G*.

references: Cohen, Bröner, Küttner, Jürgens, and Jäckle, 1989, Nature 338: 432-34.

genetics: Mutant for *Ba*.

molecular biology: 60E breakpoint located on the molecular map.

Tp(2;3)C18: Transposition (2;3)

Crossover suppressor

cytology: *Tp(2;3)25B;40;84B*.

new order:

21 – 25B|40 – 60;
61 – 84B|25B – 40|84B – 100.

origin: X ray induced.

discoverer: Roberts, 1964.

references: 1970, Genetics 65: 429-48.

genetics: Homozygous lethal. Recombination reduced in 2L.

Tp(2;3)C328

cytology: *Tp(2;3)55C;58B;80-81*; position of breakpoint in chromosome 3 with respect to centromere not determined.

new order:

21 – 55C|58B – 60;
61 – 80|(55C – 58B)|81 – 100.

origin: X ray induced.

discoverer: Roberts, 1965.

references: 1970, Genetics 65: 429-48.

genetics: Homozygous viable. Recombination reduced in 2R. The segregant *Dp(2;3)C328 = Dp(2;3)55C;58B;80-81* survives but not the complementary deficiency.

Tp(2;3)CA21 + Tp(3;2)CA20: see Tp(3;2)CA20

Tp(2;3)Cha¹⁷: Transposition (2;3)

Choline acetyltransferase

cytology: *Tp(2;3)41;91C*. Complex rearrangement.

origin: γ ray induced.

discoverer: Myers and Gelbart.

Tp(2;3)dp: Transposition (2;3) dumpy

cytology: *Tp(2;2)27E1-2;32E2-3;45A + Tp(2;3)34D7-E1;41;48A1;80-81* (inferred). Breakpoints listed by Bridges (CP627) have been revised by Woodruff and Ashburner, 1979, who describe a stock obtained from the Bowling Green Stock Center. Examination of the stock by standard genetic tests indicates a transposition between chromosome 2 and chromosome 3. Metaphase preparations of the balanced stock show, in addition to two normal-looking metaphase chromosomes, a long metacentric and a short submetacentric, believed to be the transposed chromosome.

new order:

21 – 27E1|32E3 – 34D7|41 – 34E1|48A1 – 60;
61 – 80|(41 – 45A|27E2 – 32E2|45A – 48A1)|81 – 100.
Tentative (Ashburner). Chromosome 3 breaks inferred from genetic data (Muller, 1942; Cooper, Zimmering, and Krivshenko, 1955, Proc. Nat. Acad. Sci. USA 41: 911-14)).

origin: Reportedly spontaneous.

discoverer: Nichols-Skoog, 36e16.

synonym: *In(2LR)dp*.

references: Morgan, Bridges, and Schultz, 1937, Year Book - Carnegie Inst. Washington 36: 301.

Curry, 1939, DIS 12: 46.

Muller, 1942, DIS 16: 64.

Woodruff and Ashburner, 1979, Genetics 92: 133-49.

Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, DIS 56: 186-91.

Ashburner, Aaron, and Tsubota, 1982, Genetics 102: 421-35.

genetics: Homozygous lethal. Deficient for *b* and *l(2)34Dd* – *l(2)34Dg*; probably mutant for *l(2)34Ea*. Mutant for *rl*, *tft* and a lethal uncovered by *Df(2R)Np*. Not necessarily associated with *dp^{36e}*; semilethal with *CyO*. May be associated with a dominant rough eye mutation. The chromosome 3 segregant *Dp(2;3)dp* was said by Muller, 1942, to survive, but to be poorly fertile in males and sterile in females, both sexes having arched wings and low viability.

Tp(2;3)dp^{h27}

cytology: *Tp(2;3)24F4-7;32B2;91D-E*.

origin: X ray induced.

references: Szidonya and Reuter, 1988a, D15 67: 77-79. 1988b, Genet. Res. 51: 197-208.

genetics: Mutant for *dp*.

Tp(2;3)dp⁷²: Transposition (2;3) decapentaplegic

cytology: *Tp(2;3)22F1-2;34B;81F*.

new order:

(Orientation of insertion tentative).
21A – 22F1|34B – 60;
61A – 81F|22F2 – 34B|81F – 100.

origin: γ ray induced.

discoverer: Segal.

references: St. Johnston, Hoffmann, Blackman, Segal, Grimalta, Padgett, Irick, and Gelbart, 1990, *Genes Dev.* 4: 1114-27.

genetics: Associated with *dpp*^{d72}, class *d-V*.

Tp(2;3)DTD33: Transposition (2;3) Disrupter of Transvection at Decapentaplegic

cytology: *Tp(2;3)22C;25A1-2;85A1-2*.

new order:

21 – 22C|25A2 – 60;

61 – 85A1|22C – 25A1|85A2 – 100.

origin: X ray induced.

references: Spencer, Hoffmann, and Gelbart, 1982, *Cell* 28: 451-61.

genetics: Deficiency lacks and duplication carries the entire *dpp* locus.

Tp(2;3)DTD71

cytology: *Tp(2;3)29F;40;89B-C*.

origin: X ray induced.

references: Smolik-Utlaut and Gelbart 1987, *Genetics* 116: 285-98.

Tp(2;3)en⁴³ Transposition (2;3)

cytology: *Tp(2;3)46C;48A;81F*.

new order:

21 – 46C|48A – 60;

61A – 81F|46C – 48A|81F – 100.

origin: X ray induced.

references: Kornberg, 1981, *Proc. Nat. Acad. Sci. USA* 78: 1095-99.

genetics: Deficiency and duplication segregants for *en* obtained.

Tp(2;3)en⁺: Transposition engrailed-wild type

cytology: *Tp(2;3)47B3;47B9-14;48A1-2;48B-C;62C1*.

origin: γ ray induced.

references: Eberlein and Russell, 1983, *Dev. Biol.* 100: 227-37.

Epper and Sánchez, 1983, *Dev. Biol.* 100: 387-98.

genetics: Deficiency segregant lacks 47B3 to 47B9-14 and 48A1-2 to 48B-C1 and carries *In(2R)en²⁸* = *In(2R)47B9-14;48A1-2*; it is homozygous lethal and cell lethal. The duplication segregant covers all *en* point mutants.

Tp(2;3)eve^{1.18}: Transposition (2;3) even-skipped

cytology: *Tp(2;3)44B;46D-E;chrom3*.

discoverer: Nüsslein-Volhard.

synonym: *T(2;3)eve^{1.18}*.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 267-82.

genetics: Deficiency segregant uncovers *tuf – lin*.

Tp(2;3)eve^{2.28}

cytology: *Tp(2;3)44F;47A;chrom3*.

discoverer: Nüsslein-Volhard.

synonym: *T(2;3)eve^{2.28}*.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 267-82.

genetics: Deficiency segregant uncovers *flz – lin* in embryos.

Tp(2;3)I: Transposition (2;3) Ising

discoverer: Ising.

references: Ashburner.

transposition	cytology	new order
<i>Tp(2;3)I704</i>	<i>46D;49A;63A-B</i>	
<i>Tp(2;3)I709</i>	<i>Df(2L)31B2-5;34D6-8;</i> <i>+ T(2;3)34A1-7;76B5-C1 on</i> <i>In(3L)63C;72E1-2</i>	21 – 31B2 34D8 – 60; 61 – 63C 72E1 – 63C 72E2 – 76B5 34A7 – 34D6 31B5 – 34A1 76C1 – 100.

Tp(2;3)Me: Transposition (2;3) Moiré

cytology: *Tp(2;3)48C1-2;59D2-3;60F;80-81* (tentative) +

In(3LR)69E;91C + In(3R)89B;97D superimposed on

In(3L)63C;72E1-2 (Whittinghill, 1937, *DIS* 8: 83);

breakpoint in chromosome 3 with respect to centromere not determined; new order therefore ambiguous.

new order:

21 – 48C1|59D2 – 48C2|60F;

61 – 63C|72E1 – 69E|91C – 97D|89B – 81|59D3 – 60F

|80 – 72E2|63C – 69E|91C – 89B|97D – 100.

Tentative.

origin: X ray induced in *In(3L)P, Me*.

discoverer: Muller, 1930.

references: Glass, 1933, *J. Genet.* 28: 104.

genetics: Mutant for *sbd* (*sbd*¹). *Dp(2;3)Me* = *Dp(2;3)59D2-3;60F;80-81* survives.

Tp(2;3)ML

origin: X ray induced.

references: Mukhina and Zhimulev, 1980, *DIS* 55: 209.

genetics: Homozygous lethal.

transposition	cytology	new order
<i>Tp(2;3)ML328</i>	<i>44D;57E;83A1-2</i>	21 – 44C 57E – 60; 61 – 83A1 57E – 44D 83A2 – 100
<i>Tp(2;3)ML473</i>	<i>43A;52B1;89A</i>	21 – 43A 52B1 – 60; 61 – 89A 43A – 52B1 89A – 100

Tp(2;3)N1-22

cytology: *Tp(2;3)46B2-D1;56F9-57A1;64B*.

new order:

21 – 46B2|57A1 – 60;

61 – 64B|46D1 – 56F9|64B – 100.

origin: X ray induced.

references: Puro, 1982, *DIS* 58: 205-08.

genetics: Homozygous lethal.

Tp(2;3)odd^{3.29}: Transposition (2;3) odd-skipped

cytology: *Tp(2;3)21A;25C;100F*.

synonym: *T(2;3)odd^{3.29}*.

references: Nüsslein-Volhard, Kluding, and Jürgens, 1985, *Cold Spring Harbor Symp. Quant. Biol.* 50: 145-54.

genetics: Deficiency segregant uncovers *odd* and *slp* in embryos.

Tp(2;3)odd^{5.1}

cytology: *Tp(2;3)23E;24E-F;chrom3*.

synonym: *T(2;3)odd^{5.1}*.

references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 267-82.

genetics: Deficiency segregant uncovers *odd* and *slp*.

***Tp(2;3)osp³*: Transposition (2;3) outspread**

cytology: *Tp(2;3)35B3-4;36C11-D1;98E2-3*.
new order:
 21 – 35B3|36D1 – 60;
 61 – 98E2|35B4 – 36C11|98E3 – 100 (Ashburner).
origin: γ ray induced.
discoverer: Ashburner.
references: Chia, Karp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706.
genetics: Associated with *osp³*. Duplication segregant viable and covers *l(2)35Bb* to *l(2)35De*. Deficiency segregant dominant lethal.

Tp(2;3)osp²⁰⁴

cytology: *Tp(2;3)35B3;86D4-5*; deficient for region in 35B.
references: Ashburner.
genetics: Deficiency segregant uncovers *noc-osp*.

***Tp(2;3)P*: Transposition (2;3) Pale**

cytology: *Tp(2;3)58E3-F2;60D14-E2;96B5-C1* (Morgan, Bridges, and Schultz, 1935, Year Book - Carnegie Inst. Washington 34: 286).
new order:
 21 – 58E3|60E2 – 60F;
 61 – 96B5|60D14 – 58F2|96C1 – 100.
origin: Spontaneous.
discoverer: Bridges, 17j16.
references: 1919, Anat. Record 15: 357-58.
 1923, Anat. Record 24: 426-27.
 Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 184-87.
 Li, 1927, Genetics 12: 1-58.
 Kossikov and Muller, 1935, J. Heredity 26: 305-17.
 Bridges, 1937, Cytologia (Tokyo), Fujii Jub., Vol. 2: 745-55.

genetics: Associated with *P*. Homozygote ordinarily lethal but survives in presence of *bw⁺Y = Dp(2;Y)YL;58F1-59A2;60E3-F1*; lethality therefore associated with 60D14-E2 breakpoint (Muller, 1942, DIS 16: 64). *Dp(2;3)P* is viable and fertile; duplicated for loci of *px*, *M(2)58f*, *crs*, *bw*, *mi*, *abb*, *pd*, *ll*, *l(2)NS*, *sp*, *bs*, and *ba* but not *a* or *M(2)60E*. Homozygous *Dp(2;3)P* (i.e., four doses of 59A2-60E3) is lethal unless one chromosome 2 is *Df(2R)P*. *Df(2R)P* with two normal third chromosomes is lethal.

other information: First transposition recorded in *D. melanogaster*.

***Tp(2;3)P32*: Transposition (2;3) Pasadena 32**

cytology: *Df(2R)41A;44C-D + T(2;3)42D-E;89D7-E1*. (Translocation involves the acentric ring produced as a concomitant of the deficiency.)
new order:
 21 – 41A|44D – 60;
 61 – 89D7|42D – 41A|44C – 42E|89E1 – 100.
origin: X ray induced in *bx^{34e}*.
discoverer: E.B. Lewis, 50i.
genetics: Gives transvection effect in *Tp(2;3)P32*, *bx^{34e}/Ubx* heterozygote. The segregant *Dp(2;3)P32* survives and is fertile and virtually wild type; duplicated for *stw*, *ap*, *tuf*, and *cn* but not *ltd*.

***Tp(2;3)prd^{2.27}*: Transposition (2;3) paired**

cytology: *Tp(2;3)31B;33D-E;97C-D*.
discoverer: Nüsslein-Volhard.
synonym: *T(2;3)prd^{2.27}*.
references: Nüsslein-Volhard, Wieschaus, and Kluding, 1984, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82.
 Nüsslein-Volhard, Kluding, and Jürgens, 1985, Cold Spring Harbor Quant. Biol. 50: 145-54.
genetics: Deficiency segregant [*Df(2L)prd2.27*] uncovers *bsk – prd*.

***Tp(2;3)SS301* Transposition (2;3) S. Smith**

cytology: *Tp(2;3)21A;21D1;80F* (Ashburner *et al.*, 1980, corrected).
origin: X ray induced.
discoverer: Smith.
references: Ashburner, Faithfull, Littlewood, Richards, Smith, Velissariou, and Woodruff, 1980, DIS 55: 193-95.

***Tp(2;3)TE35A-203*: Transposition (2;3) Transposable Element**

cytology: *Tp(2;3)34C1-2;35B;80-81* (Ashburner).
origin: γ ray induced in *TE35A*.
discoverer: Samkanje.
synonym: *Tp(2;3)TE146Z*.
genetics: Deficiency segregant dominant lethal.

Tp(2;3)TE35BC-2

cytology: *Df(2L)25A1-2;37A1-2 + Tp(2;3)35C1-3;80-81*.
new order:
 21 – 25A1|37A2 – 60;
 61 – 80|(35C1 – 25A2|37A1 – 35C3)|81 – 100.
origin: γ ray induced in *TE35BC*.
discoverer: Gubb.
synonym: *Tp(2;3)36R*.
genetics: Variegates for *w⁺* of *TE35BC*. Does not cover deficiency for *rst*. Lethal with lethal *ck* alleles.

***Tp(2;3)tkv*: Transposition (2;3) thick veins**

cytology: *Tp(2;3)25A2-3;25D5-E1;69C*.
new order:
 21 – 25A2|25E1 – 60;
 61 – 69C|25A3 – 25D5|69C – 100.
discoverer: Szidonya.
references: Szidonya and Reuter, 1988, Genet. Res. 51: 197-208.
genetics: Deficiency segregant is *M* and female sterile; very weak but fertile with *SM1* or *In(2L)NS*, sterile with *SM5*.

***Tp(2;3)V*: Transposition (2;3) Valencia**

origin: X ray induced.
references: Valencia, 1970, DIS 45: 37-38.

	transposition	cytology	new order α	genetics (homozygotes)
	<i>Tp(2;3)V10-7m</i>	<i>26A3;26F;86E</i>	1	lethal
	<i>Tp(2;3)V12-2-2</i>	<i>44E;50B;80</i>	2	partially viable male sterile
	<i>Tp(2;3)V13-2a</i>	<i>41A;50C-D;100A-B</i>	3	lethal
α	1	<i>21 – 26A3 26F – 60; 61 – 86E 26A3 – 26F 86E – 100.</i>		
	2	<i>21 – 44E 50B – 60; 61 – 80 44E – 50B 80 – 100.</i>		
	3	<i>21 – 41A 50D – 60; 61 – 100A (41A – 50C) 100B – 100F.</i>		

Tp(2;4)X2

cytology: *Tp(2;4)23D-E;24A1-2;102D*.

synonym: *Tp(2;4)ey^D*.

references: Ashburner.

Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.

genetics: Mutant for *Alp²* and *ey^D*.

Tp(3;1)bx^d111: Transposition (3;1) bithoraxoid

cytology: *Tp(3;1)4D;89E;90B2*.

new order:

1 – 4D|89E – 90B2|4D – 20;

61 – 89E|90B2 – 100.

references: Lewis, 1981, *Developmental Biology Using Purified Genes* (Brown and Fox, eds.). Academic Press, London, New York, San Francisco, Vol. 23, pp. 189-208.

genetics: Transposes rightmost part of the *BXC* to 4D. Mutant for *bx^d* and *pbx*. Distinguishable from other *bx^d* rearrangements by larval phenotype.

molecular biology: 89E breakpoint about 5 kb to the left of the right breakpoint of *In(3R)Cbx^{rv1}* (Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, *Science* 221: 23-29).

Tp(3;1)C152

cytology: *Df(3R)88B-C;94A + T(1;3)20;90E..*

new order:

1 – 20|(90E – 88C|94A – 90E)|20;

61 – 88B|94A – 100.

origin: X ray induced.

discoverer: Roberts, 1965.

references: 1970, *Genetics* 65: 429-48.

genetics: Male fertile. Recombination reduced in *3R*.

Tp(3;1)C210

cytology: *Tp(3;1)7F10-8A1;76B;80*.

new order:

1 – 7F10|76B – 80|8A1 – 20;

61 – 76B|80 – 100.

origin: Lefevre.

genetics: Associated with *gg*.

Tp(3;1)DEB8: Transposition (3;1) diepoxybutane

cytology: *Tp(3;1)20F;70E-F;77C*.

new order:

1 – 20F|70F – 77C|20F;

61 – 70E|77C – 100.

origin: Induced by diepoxybutane.

references: Denell, Lim, and Auerbach, 1978, *Mutat. Res.* 49: 219-24.

genetics: Male fertile.

Tp(3;1)FA11

cytology: *Tp(3;1)X;84D-E;87D*.

synonym: *T(1;3)FA11*.

discoverer: Lehman.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95.

genetics: Deficiency segregant uncovers *hb – sad*.

Tp(3;1)HF318

cytology: *Tp(3;1)3A3-4;66D-E;80*.

origin: X ray induced.

discoverer: Lefevre.

genetics: Hemizygous lethal.

Tp(3;1)KA21

cytology: *Tp(3;1)2C9;66B;67E*.

origin: X ray induced.

references: Lefevre, 1981, *Genetics* 99: 461-80.

Oro, McKeown, and Evans, 1990, *Nature* 347: 298-361.

genetics: Hemizygous lethal. Mutant for *usp*.

molecular biology: 2C9 break results in deletion of three nucleotides in *usp²*.

Tp(3;1)kar⁵¹: Transposition (3;1) karmoisin

cytology: *Tp(3;1)20;87C7-D1;88E2-3*.

new order:

1 – 20|87D1 – 88E2|20;

61 – 87C7|88E3 – 100.

discoverer: Gelbart.

references: Hall and Kankel, 1976, *Genetics* 83: 517-35.

Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95.

genetics: Associated with *kar*. Deficiency segregant uncovers *ems – put* (Jürgens *et al.*, 1984). Duplication segregant includes *trx⁺* (Ingham, 1981, *Wilhelm Roux's Arch. Dev. Biol.* 190: 365-69).

Tp(3;1)N²⁶⁴⁻⁶: Transposition (3;1) Notch

cytology: Breakpoints given in CP627 revised by Ashburner and Walker (Ashburner *et al.*, 1981). *X* consists of inverted *X* and inserted third chromosome material; *3* is both inverted and deficient.

new order:

1 – 3C|20 – 3C|80 – 73F4|71B8 – 73A5|20F;

61A – 61F5|71B7 – 61F8|73F1 – 73B2|80 – 100.

This cytology incompatible with reported survival of aneuploid segregants.

origin: X ray induced.

discoverer: Demerec, 33k20.

synonym: *T(1;3)N^{N264-6}* (CP627).

references: CP627.

Ashburner, Angel, Detwiler, Faithfull, Gubb, Harrington, Littlewood, Tsubota, Velissariou, and Walker, 1981, *DIS* 56: 186-91.

genetics: Variegates for *w* and *N*. *X/Y* male lethal; *X/Y/Y* viable and sterile. Duplication segregant viable; deficiency segregant survives and is *M. Tp(1;3)N²⁶⁴⁻⁶* (CP627).

Tp(3;1)O5

cytology: *Tp(1;3)4F2-3;62B-C;88A-C;92C-D* (Lewis, 1951, *DIS* 25: 108-9). *Dp(3;1)O5* similar to standard *X* in size, staining of salivaries, and early completion of replication (Lakhotia, 1970, *Genet. Res.* 15: 301-07).

new order:

1 – 4F2|88C – 92C|4F3 – 20;

61 – 62B|88A – 62B|92D – 100.

origin: X ray induced.

discoverer: Oliver, 29130.

references: 1937, *Am. Nat.* 71: 560-66.

1938, *Genetics* 23: 162.

genetics: Male viable and fertile. Homozygous female viable and sterile. The segregant *Dp(3;1)O5* is viable and fertile in male and female. It is duplicated for loci of *red*, *jvl*, *cv-c*, *su(Hw)*, *sbd*, *ss*, *bx*, *sr*, *gl*, *k*, and *Dl* but not *cu*, *ry*, *kar*, or *e* (Lindsley and Grell, 1958, *DIS* 32: 136; Lewis). Produces roughish eyes, spread and nicked

wings, coarse bristles, and a darkly-pigmented abdomen.

Tp(3;1)P115: Transposition (3;1) Pasadena 115

cytology: *Tp(3;1)20;89B7-8;89E7-8* (Sánchez-Herrero *et al.*, 1985).

discoverer: E.B. Lewis.

references: Morata and García-Bellido, 1976, Wilhelm Roux's Arch. Dev. Biol. 179: 125-43.

Sánchez-Herrero, Vernós, Marco, and Morais, 1985, Nature 313: 108-13.

genetics: The deficiency segregant, *Df(3R)P115*, includes the entire *BXC* and several neighboring genes (Sánchez-Herrero *et al.*, 1985). The duplication segregant, *Dp(3;1)P115*, carries all the wild-type alleles in the *BXC*.

Tp(3;1)pn³⁶ Transposition (3;1) prune

cytology: *Tp(3;1)1A;2E1-2;61A;62C2-3*.

new order:

1A|2E1 – 1A|61A – 62C1|2E2 – 20;
61A|62C4 – 100.

origin: X ray induced.

references: Ilyina, Sorokin; Belyaeva, and Zhimulev, 1980, DIS 55: 205.

Slobodyanyuk and Serov, 1983, Mol. Gen. Genet. 191: 372-77.

genetics: Mutant for *pn*.

Tp(3;1)ry³⁵: Transposition (3;1) rosy

cytology: *Tp(3;1)20;87C-E;91B-C* (Lindsley). Left breakpoint in or near *ry*.

new order:

1 – 20|(87E – 91B)|20;
61 – 87C|91C – 100.

origin: X ray induced in *cu kar* chromosome.

discoverer: Schalet.

references: 1964, DIS 39: 62-64.

Schalet, Kernaghan, and Chovnick, 1964, Genetics 50: 1261-68.

Roehdanz, Kitchens, and Lucchesi, 1977, Genetics 85: 489-96.

genetics: Transposition associated with *cu*, *ry³⁵*, and *kar*. The segregant, *Dp(3;1)ry³⁵* is viable and fertile in male and female; duplicated for loci of *Aldox*, *Sb* and *Ubx*.

Tp(3;1)ss^V: Transposition (3;1) spineless-variegated

cytology: *Tp(3;1)20;89B;100F*.

new order:

1 – 20|89B – 100F|20;
61 – 89B|100F.

origin: X ray induced.

discoverer: E.B. Lewis.

genetics: Variegated for *ss^l* and mutant for *ss^a*. Male viable and sterile.

Tp(3;1)st^{g82}: Transposition (3;1) scarlet

cytology: *Tp(3;1)5B6-7;72D10-11;73B1-2;80F*; deficient for 72D11-73B1.

new order:

1 – 5B6|80F – 73B2|5B7 – 20;
61 – 72D10|80F – 100.

origin: X ray induced.

discoverer: Belote.

references: Belote and McKeown.

genetics: Deficient for *st*.

Tp(3;1)ZWD14

cytology: *Tp(3;1)3C1-2;81;86B-C*.

origin: γ ray induced.

references: Smolik-Utlaut and Gelbart, 1987, Genetics 116: 285-98.

genetics: Males *w⁺*; homozygous females show mottled eye color.

Tp(3;Y)

origin: X ray induced in *B^SY⁺*.

transposition	cytology	ref ^{α}	X/T(Y;3) fertility
<i>Tp(3;Y)A76</i>	65A;66A	2	+
<i>Tp(3;Y)A81</i>	75D;80	2	–
<i>Tp(3;Y)B150</i>	88F;94A	2,3	–
<i>Tp(3;Y)B162</i>	65E;71A	2	–
<i>Tp(3;Y)B204</i>	93B;98B	2,3	+
<i>Tp(3;Y)B216</i>	85F;91C	2	–
<i>Tp(3;Y)B219</i>	94C;100A	2	+
<i>Tp(3;Y)B233</i>	67E;70A	2	–
<i>Tp(3;Y)G63^{β}</i>	70A;77B-C + 83C;85A	1	+
<i>Tp(3;Y)G145</i>	68D;70D	2	+
<i>Tp(3;Y)H141</i>	61B;62D	2	+
<i>Tp(3;Y)J55</i>	98A;100B	2	–
<i>Tp(3;Y)J158</i>	73C;79D	2	–
<i>Tp(3;Y)L58</i>	88D;93D	1	–
<i>Tp(3;Y)L109^{γ}</i>	83D-E;86C;91A-C;95D	2	–
<i>Tp(3;Y)L127^{δ}</i>	99B;99E	2	–
<i>Tp(3;Y)P3</i>	61E1;61F7	2	–
<i>Tp(3;Y)P6</i>	61A;62A-B	2	–

^{α} 1 = Baker, 1980, DIS 55: 197; 2 = The Seattle-La Jolla Drosophila Laboratories, 1971, DIS 47, Suppl. 3 = Voelker, Ohnishi, and Langley, 1979, Biochem. Genet. 17: 769-83.

^{β} Pieces 83C–85A and 70A–77B-C not visibly connected in chromocenter.

^{γ} New order: *Y^D|(86C – 91A|95D – 91C|83E – 86C)|Y^P*;
61 – 83D|95D – 100.

^{δ} Carries *Acp⁺*, *bv⁺*, and *ca⁺* (Morrison and MacIntyre, 1978, Genetics 88: 487-97).

Tp(3;Y)Abd-B: Transposition (3;Y) Abdominal-B

cytology: *Tp(3;Y)89C;89E1-2*.

new order:

Y|89C – 89E1|Y;
61 – 89C|89E2 – 100.

origin: γ ray in *mwh* stock.

references: Tiong, Whittle, and Gribbin, 1987, Development 101: 135-42.

genetics: Mutant for *Abd-B*, *Ubx*, *abd-A*; proximal part of *Abd-B* inserted in *Y*.

molecular biology: Distal breakpoint at +54 kb (Bender) proximal to the *Abd-B* homeobox.

Tp(3;Y)L131-D3

cytology: *Tp(3;Y)72A-B;75C*; *y⁺* inserted in *3L*.

origin: γ ray-induced derivative of *T(Y;3)L131* that is no longer male-sterile in combination with *Df(1)bb158*.

discoverer: Kennison.

genetics: Duplication segregant survives and is *B^S* but not *y⁺*.

Tp(3;Y)P92: Transposition (3;Y) Pasadena 92

cytology: *Tp(3;Y)84D9-10;85A6* on *In(3R)85B;88C*. Extra bands distally and proximally (B.S. Baker).

new order:

Y|84D10 – 85A6|Y;
61 – 84D9|85A6 – 85B|88C – 85B|88C – 100.

origin: X-ray-induced derivative of *In(3R)hb^D*.

discoverer: E.B. Lewis.

references: Duncan and Kaufman, 1975, *Genetics* 80: 733-52.

Bender, Turner, and Kaufman, 1986, *Dev. Biol.* 119: 418-32.

genetics: *hb^D* transposed to *Y*. Duplication segregant complements the recessive lethality of *Dfd* and covers *dsx* and *p^P*, but does not cover the *Dfd* eye phenotype (Duncan and Kaufman, 1975).

Tp(3;2)BTD9

cytology: *Tp(3;2)58A;81;88E*.

new order:

41 – 58A|88E – 81|58A – 60;

61 – 81|88E – 100F.

origin: γ ray induced.

references: Smolik-Utlaut and Gelbart, 1987, *Genetics* 116: 285-98.

Tp(3;2)C16

cytology: *Tp(3;2)50E;66C;70C*.

new order:

21 – 50E|70C – 66C|50E – 60;

61 – 66C|70C – 100.

origin: X ray induced.

discoverer: Roberts, 1964.

references: 1970, *Genetics* 65: 429-48.

genetics: Homozygous lethal. Recombination reduced in 2R and 3L.

Tp(3;2)C259

cytology: *Tp(3;2)40-41;61E;73A*; position of breakpoint in chromosome 2 with respect to centromere not determined.

new order:

21 – 40|61E – 73A|41 – 60;

61A – 61E|73A – 100.

Tentative.

origin: X ray induced.

discoverer: Roberts, 1965.

references: 1970, *Genetics* 65: 429-48.

genetics: Homozygous lethal. Recombination reduced in 3L.

Tp(3;2)ca^v: Transposition (3;2) claret-variegated

cytology: *Df(3R)81F;99C-E + T(2;3)59D;94*. (Translocation involves the acentric ring formed as a concomitant of the deficiency.)

new order:

21 – 59D|94 – 99C|81F – 94|59D – 60;

61 – 81F|99E – 100.

origin: X ray induced.

discoverer: E.B. Lewis.

synonym: *In(3R)ca^v* (CP627).

references: Craymer, 1980, *DIS* 55: 197-200.

genetics: Variegates for *ca*.

Tp(3;2)CA20

cytology: *Df(2L)34D;35C + T(2;3)45A-B;69F-70A;98E-F + T(2;3)48E-F;99E*.

new order:

21 – 34D|35C – 45A|98F – 99E|48F – 60F;

61 – 69F|98E – 70A|45B – 48E|99E – 100.

references: Gubb, Roote, Harrington, McGill, Durrant, Shelton, and Ashburner, 1984, *Chromosoma* 92: 116-23.

genetics: Associated with *Df(2L)TE35A-5*.

Tp(3;2)Ctx: Transposition (3;2) Cephalothorax

cytology: *Tp(3;2)35B3-4;83C9-D1;85E5-9* (E.B. Lewis; Jürgens).

new order:

21 – 35B3|83D1 – 85E5|35B4 – 60;

61 – 83C9|85E9 – 100.

synonym: *T(2;3)Ctx*.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95.

genetics: Mutant for *l(2)35Bd* and *Anp^{Cx}*. Deficiency segregant uncovers *Scr - hb*.

molecular biology: Third chromosome breakpoint 165-166 kb distal to proximal breakpoint of *Tp(3;3)Dfd^{rv16}*.

Tp(3;2)DTD129

cytology: *Dp(3;2)26F;81F;99A*.

origin: γ ray induced.

references: Smolik-Utlaut and Gelbart, 1987, *Genetics* 116: 258-98.

Tp(3;2)FA12

cytology: *Tp(3;2)chrom2;77A-B;80F*.

synonym: *T(2;3)FA12*.

discoverer: Lehman.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95.

genetics: Deficiency segregant uncovers *kni* and *Pc* in heterozygous embryos.

Tp(3;2) fz^{C21}: Transposition (3;2) frizzled

cytology: *Tp(3;2)40;66B-D;70D4-7*.

new order:

21A – 40|66D – 70D4|40 – 60;

61 – 66B|70D7 – 100.

origin: γ ray induced.

discoverer: Adler.

genetics: Moderate *fz* phenotype. Homozygous lethal (occasional homozygotes dying as pharate adults, but most dying earlier). Viable over all other *fz* alleles.

Tp(3;2)I702

cytology: *Tp(3;2)29F;64F;77F* on *In(3L)63C;72E1-2*.

new order:

21 – 29F|77F – 72E2|63C – 64F|29F – 60;

61 – 63C|72E1 – 64F|77F – 100.

discoverer: Ising.

references: Ashburner.

Tp(3;2)iab2^{P10}: Transposition (3;2) infraabdominal 2 - Pasadena 10

cytology: *Tp(3;2)29A-C;89C1-2;89E1-2*.

new order:

21 – 29A|89C2 – 89E1|29C – 60;

61 – 89C1|89E2 – 100.

origin: X ray induced.

discoverer: Shaw, 1974.

synonym: *Tp(3;2)P10*.

references: Lewis, 1978, *Nature* 276: 565-70. 1980, *DIS* 55: 207-08.

Struhl, 1981, *Nature* 293: 36-41.

1984, Nature 308: 354-57.

Morata, Botas, Kerridge, and Struhl, 1983, J. Embryol. Exp. Morphol. 78: 319-41.

Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 45: 81-96.

genetics: Insertion of *ss⁺-pbx⁺* and perhaps part of *iab2⁺* into 2L (Lewis, 1978; Struhl, 1984). Homozygous lethal in late embryo (Lewis, 1980). *Dp(3;2)P10* homozygous viable; *Df(3R)P10* heterozygous viable and *Ubx* in phenotype. It deletes all the thoracic *BXC* genes and seems to damage *iab2* (Morata *et al.*, 1983).

molecular biology: 89E breakpoint 35-36 kb to the right of right breakpoint of *In(3R)Cbx^{rv1}*. (Karch *et al.*, 1985).

Tp(3;2)iab5³⁰¹: Transposition (3;2) infraabdominal 5

cytology: *Tp(3;2)41;86E;89E*.

discoverer: R.H. Baker.

genetics: Mutant for *iab5³⁰¹*.

Tp(3;2)iab5⁸⁴³

cytology: *Tp(3;2)23;81;89E3-4*.

new order:

21 - 23|81 - 89E3|23 - 60;

61 - 81|89E4 - 100.

origin: X ray induced.

discoverer: R.H. Baker.

synonym: *iab5^{31616.843}*.

references: Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.

genetics: Mutant for *iab5*.

molecular biology: 89E3-4 breakpoint 93-99.5 kb to the right of the right breakpoint of *In(3R)Cbx^{rv1}*.

Tp(3;2)iab5⁹⁹⁴

cytology: *Tp(3;2)22;89B;89E;90A*.

origin: X ray induced.

discoverer: R.H. Baker.

synonym: *iab5^{31616.994}*.

genetics: Mutant for *iab5*.

Tp(3;2)MS6: Transposition (3;2) Mglinetz Semenova

cytology: *Tp(3;2)60A;61C;64B*.

new order:

21 - 60A|61C - 64B|60A - 60F;

61A - 61C|64B - 100.

origin: γ ray induced.

references: Semenova, Mglinetz, and Glotoff, 1970, Genetika (Moscow) 6(4): 165-69.

Mglinetz and Semenova, 1970, Genetika (Moscow) 6(8): 86-94.

Tp(3;2)MS7

cytology: *Tp(3;2)21A;61A;63B*.

new order:

21A|61A - 63B|21A - 60;

61A|63B - 100.

origin: γ ray induced.

references: Semenova, Mglinetz, and Glotoff, 1970, Genetika (Moscow) 6(4): 165-69.

Mglinetz and Semenova, 1970, Genetika (Moscow) 6(8): 86-94.

Tp(3;2)N2-27

cytology: *Tp(3;2)21F-22A;35D-F;75A-B;80*.

new order:

21A - 21F|75B - 80|35D - 22A|35F - 60;

61 - 75A|80 - 100.

origin: X ray induced.

synonym: *T(2;3)FM27*.

references: Puro, Nygren, and Nuutila, 1973, DIS 50: 108.

Puro and Nygren, 1975, Hereditas 81: 237-48.

Puro, 1982, DIS 58: 205-08.

genetics: Homozygous lethal. Duplication segregant survives and carries wild-type alleles of *in*, *ri*, and *Pc*. Deficiency segregant is lethal.

Tp(3;2)osp²⁰⁴: Transposition (3;2) outspread

cytology: *Tp(3;2)35B2-3;86C2-D4;89A4-7*.

new order:

21 - 35B2|89A4 - 86D4|35B3 - 60;

61 - 86C2|89A7 - 100.

origin: α ray induced.

discoverer: Detwiler.

genetics: Deficient for *noc-osp* but not *Adh*. Shows stronger *Mpe* leg phenotype than *T(2;3)Mpe*, but does not show dominant *Mpe* wing phenotype.

Tp(3;2)P10: see *Tp(3;2)iab2^{P10}*

Tp(3;2)ry⁺: Transposition (3;2) rosy - wild type

cytology: *Tp(3;2)2L;87C2-3;88C2-3* (Gelbart). Amount of 87C2-3 material duplicated is uncertain (Gyurkovics).

references: Hall and Kankel, 1976, Genetics 83: 517-35.

Gausz, Bencze, Gyurkovics, Ashburner, Ish-Horowicz, and Holden, 1979, Genetics 93: 917-34.

genetics: With *Dp(3;2)ry⁺*, *Df(3R)kar^{3J}* homozygotes or *Df(3R)kar^{3J}/Df(3R)kar^{3I}* heterozygotes are viable but female sterile, whereas *Df(3R)kar^{Sz29}/Df(3R)kar^{3J}* heterozygotes are female fertile (Ish-Horowicz).

Tp(3;2)S485

cytology: *Tp(3;2)21C;89F;96A*. 89F break believed to be outside the *BXC*.

new order:

21A - 21C|89F - 96A|21C - 60;

61 - 89F|96A - 100.

origin: X ray induced.

discoverer: Shaw.

references: Karch, Weiffenbach, Peifer, Bender, Duncan, Crosby, and Lewis, 1985, Cell 45: 81-96.

genetics: *Tp(3;2)S485/Df(3R)P9* flies viable and do not show more segmental transformation than *Df(3R)P9/+* flies.

molecular biology: 89F breakpoint 205-216 kb to the right of right breakpoint of *In(3R)Cbx^{rv1}*.

Tp(3;2)shv^{S3}: Transposition (3;2) shortvein

cytology: *Tp(3;2)22F1-2;40C;82A;92A5-8*.

new order:

21A - 22F1|92A5 - 82A|40C - 22F2|40C - 60F (order of reattachment of chromocentral breaks uncertain);

61A - 82A|92A8 - 100F.

origin: X ray induced.

discoverer: Segal.

references: Segal and Gelbart, 1985, Genetics 109: 119-43.

genetics: Associated with *shv^{S3}*. Lethal over *shv* and

lethal *dpp* alleles.

Tp(3;2)SNS

cytology: *Tp(3;2)33A;90C;92D*.

origin: γ ray induced.

references: Sequeira, Nelson, and Szauter, 1989, *Genetics* 123: 511-24.

other information: Induced at same time as *ca*⁵.

Tp(3;2)Ubx*⁸⁹⁵: *Transposition (3;2) Ultrabithorax

cytology: *Tp(3;2)2R;89E;90A*.

origin: X ray induced.

discoverer: R.H. Baker, 84k.

genetics: Associated with *Ubx*⁸⁹⁵.

Tp(3;2)V4-8: Transposition (3;2) Valencia

cytology: *Tp(3;2)43E;65F;79E-F*.

new order:

21 – 43E|65F – 79E|43E – 60;

61 – 65F|79F – 100.

origin: X ray induced.

references: Valencia, 1970, *DIS* 45: 37-38.

genetics: Homozygous lethal.

Tp(3;2)V12-1a

cytology: *Tp(3;2)55B;88B;91B*.

new order:

21 – 55B|88B – 91B|55B – 60;

61 – 88B|91B – 100.

origin: X ray induced.

references: Valencia, 1970, *DIS* 45: 37-38.

genetics: Homozygous lethal.

Tp(3;2)XM54

cytology: *Tp(3;2)chrom2;88C2-3;96B11-C1*.

synonym: *T(2;3)XM54*.

references: Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, *Wilhelm Roux's Arch. Dev. Biol.* 193: 283-95.

genetics: Deficiency segregant uncovers *put* – *tld* in heterozygous embryos.

Tp(3;3)bx¹⁰⁰*: *Transposition (3;3) bithoraxoid

cytology: *Tp(3;3)66;89B5-6;89E2-3* (89E breakpoint between *Ubx* and *bx¹⁰⁰*).

new order:

61 – 66C|(89B6 – 89E2)|66C – 89B5|89E3 – 100.

origin: X ray induced.

discoverer: E.B. Lewis.

references: 1951, *Cold Spring Harbor Symp. Quant. Biol.* 16: 159-74.

Lewis, 1978, *Nature* 276: 565-70.

Morata and Kerridge, 1981, *Nature* 290: 778-81.

Struhl, 1981, *Nature* 293: 36-41.

Gailey, Jackson, and Siegel, 1982, *Genetics* 102: 771-82.

Struhl, 1984, *Nature* 308: 454-57.

genetics: Transposes small, proximal part of the *BXC* to 3*L*. Mutant for *bx¹⁰⁰* and *pbx* but not *bx*, *Cbx*, or *Ubx*. *Dp(3;3)bx¹⁰⁰* survives, and is duplicated for *ss* – *Ubx* but not for *bx¹⁰⁰*. *Df(3R)bx¹⁰⁰* survives in the heterozygote where it shows the *Ubx* phenotype.

molecular biology: 89E2-3 breakpoint between 15 and 20 kb proximal to the distal breakpoint of *In(3R)Cbx^{rv1}* (Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis,

and Hogness, 1983, *Science* 221: 23-29).

Tp(3;3)bx¹¹⁰

cytology: *Tp(3;3)89E2-3;91D1-2;92A2-3*.

new order:

61 – 89E2|(91D2 – 92A2)|89E3 – 91D1|92A3 – 100.

origin: X ray induced.

discoverer: E.B. Lewis.

genetics: Transposes the *Dl* region into the *BXC*. Mutant for *bx¹¹⁰* but not *bx* or *Dl*. *Df(3R)bx¹¹⁰* = *Df(3R)91D1-2;92A2-3*, formed by recombination with a normal chromosome in region 89E3-91D1, survives in the heterozygote where it shows the *Dl* phenotype. The complementary *Dp(3;3)bx¹¹⁰* is duplicated for *Dl*⁺ and acts as a suppressor of *Dl* in *Dp(3;3)bx¹¹⁰/Dl*⁷ heterozygote (Lewis).

molecular biology: 89E2-3 breakpoint between 20 and 30 kb proximal to the right breakpoint of *In(3R)Cbx^{rv1}* (Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, *Science* 221: 23-29).

Tp(3;3)bx^{DB9}

cytology: *Tp(3;3)80;89E*.

origin: X ray induced.

discoverer: D. Baker.

genetics: Mutant for *bx¹¹⁰*.

Tp(3;3)C285: Transposition (3;3) Crossover suppressor

cytology: *Tp(3;3)88F;98B;99B*.

new order:

61 – 88F|98B – 99B|88F – 98B|99B – 100.

origin: X ray induced.

discoverer: Roberts, 1965.

references: 1970, *Genetics* 65: 429-48.

genetics: Homozygous lethal. Recombination between *st* and *ca* sharply reduced.

Tp(3;3)C341

cytology: *Tp(3;3)63C;71E;80-81*; position of right breakpoint with respect to centromere not determined.

new order:

61 – 63C|71E – 80|(63C – 71E)|81 – 100.

origin: X ray induced.

discoverer: Roberts, 1965.

references: 1970, *Genetics* 65: 429-48.

genetics: Homozygous viable. Recombination reduced in 3*L*.

Tp(3;3)Dfd^{rv1}: Transposition (3;3) Deformed-revertant

cytology: *Tp(3;3)83D4-5;84A4-5;98E6-F2*.

new order:

61 – 83D4|84A5 – 98E6|83D5 – 84A4|98F2 – 100.

origin: X ray induced.

references: Hazelrigg and Kaufman, 1983, *Genetics* 105: 581-600.

genetics: Revertant of *Dfd*.

Tp(3;3)Dfd^{rv16}

cytology: *Tp(3;3)84A4-5;86F11;87D13-14*.

new order:

61 – 84A4|86F11 – 87D13|84A5 – 86F11|87D14 – 100.

origin: X ray induced.

references: Hazelrigg and Kaufman, 1983, *Genetics* 105: 581-600.

Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman, 1983, Cell 35: 763-76.

genetics: Revertant of *Dfd*. Shows a *pic⁻* phenotype (Clark and Chovnick, 1986, Genetics 114: 819-40).

molecular biology: DNA breakpoint at 84A4-5 is coordinate 0 in the walk of Scott *et al.*

***Tp(3;3)DI^{III3}*: Transposition (3;3) Delta**

cytology: *Tp(3;3)88F5-9;91A3-8;92A2*.

new order:

61 – 88F5|91A8 – 92A2|88F9 – 91A3|92A2 – 100.

origin: X ray induced.

discoverer: Shrons.

references: Vässin and Campos-Ortega, 1987, Genetics 116: 433-45.

genetics: Associated with *DI^{III3}*.

***Tp(3;3)h^{m3}*: Transposition (3;3) hairy**

cytology: *Tp(3;3)61E;66D2-3;66D9-10*.

new order:

61A – 61E|66D3 – 66D9|61E – 66D2|66D10 – 100.

origin: X ray induced.

references: Ingham, Pinchin, Howard, and Ish-Horowicz, 1985, Genetics 111: 463-86.

genetics: Homozygous lethal. Associated with *se* and *h*.

molecular biology: 66D break located at -10.8 to -9.35 kb on the DNA map of *h* (Howard, Ingham, and Rushlow, 1988, Genes Dev. 2: 1037-46); "+" values to the right, "-" values to the left. The coordinate system has 0 at the start of transcription (Rushlow).

***Tp(3;3)H⁵⁷*: Transposition (3;3) Hairless**

cytology: *In(3R)86F7-11;97D1-2 + In(3R)95C1-2;98C5*. (Ashburner).

new order:

61 – 86F7|97D1 – 95C2|98C5 – 97D2|86F11 – 95C1|98C5 – 100.

discoverer: Gloor.

references: van Breugel, Ray, and Gloor, 1968, Genetika 39: 165-92.

Ashburner, 1982, Genetics 101: 447-59.

genetics: Associated with *H⁵⁷* (van Breugel *et al.* 1968), but, presumably, *H⁵⁷* and the transposition arose by independent mutational events (Ashburner, 1982) since none of the breakpoints is between 92D and 94A, the location of *H*.

***Tp(3;3)iab3²⁷⁷* Transposition (3;3) infraabdominal**

cytology: *Tp(3;3)89E;94A;96F*.

new order:

61 – 89E|94A – 96F|89E – 94A|96F – 100.

origin: X ray induced in *Mcp*.

discoverer: D. Baker.

synonym: *Mcp^{revB277}*, *iab3^{B277}*, *iab3^{DB}*.

references: Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.

genetics: Mutant for *iab3*. Partial revertant of *Mcp*.

molecular biology: 89E breakpoint 63-64.5 kb to the right of the right breakpoint of *In(3R)Cbx^{rv1}*.

Tp(3;3)iab6^{Vno}

cytology: *Tp(3;3)89E;94A;96F*.

new order:

61 – 89E|94A – 96F|89E – 94A|96F – 100.

origin: X ray induced.

discoverer: E.H. Grell, 56c.

synonym: *Tp(3;3)Vno*.

references: 1959, DIS 33: 94.

Karch, Weiffenbach, Peifer, Bender, Duncan, Celniker, Crosby, and Lewis, 1985, Cell 43: 81-96.

genetics: Associated with *Vno* and *iab6*. Insertion at 89E separable by recombination from the deletion at 94A-96F, the *Vno* wing phenotype segregating with the deletion (Karch *et al.*, 1985). Homozygous lethal.

molecular biology: 89E breakpoint 108-111 kb to the right of the distal breakpoint of *In(3R)Cbx^{rv1}*.

Tp(3;3)iab8^{rv}

transposition	cytology
<i>Tp(3;3)iab8^{rv2}</i>	64E-F;65A;89E
<i>Tp(3;3)iab8^{rv6}</i>	64E-F;65A;89E + <i>Df(3R)80;89E</i>

Tp(3;3)Mg: Transposition (3;3) Mglinetz

origin: γ ray induced.

transposition	cytology	new order	ref ^{α}
<i>Tp(3;3)Mg16</i>	64A;89B;90F	61-64A 89B-90F 64A-89B 90F-100	2
<i>Tp(3;3)Mg25</i>	83C;87A;98A	61-83C 87A-98A 83C-87A 98A-100	3
<i>Tp(3;3)Mg79</i>	72A;90A;93F	61-72A 90A-93F 72A-90A 93F-100	4
<i>Tp(3;3)Mg80</i>	86A;90E;99D	61-86A 90E-99D 86A-90E 99D-100	4
<i>Tp(3;3)Mg197</i>	75A;84A;93F	61-75A 84A-93F 75A-84A 93F-100	1

^{α} 1 = Mglinetz, 1968, Genetika (Moscow) 4(8): 81-86; 2 = Mglinetz, 1971, Genetika (Moscow) 7(8): 108-14; 3 = Mglinetz, 1972, Genetika (Moscow) 8(2): 82-91; 4 = Mglinetz, 1973, Genetika (Moscow) 9(3): 69-74.

Tp(3;3)MKRS

cytology: *In(3LR)71B2-C2;92E + Df(3R)87E8-F1;93C*; the right inversion breakpoint involves the acentric ring generated by the deficiency.

new order:

61 – 71B2|92E – 93C|87F1 – 92E|71C2 – 87E8|93C – 100.

(Hilliker *et al.*, 1980).

origin: Insertion of *kar* into *Tp(3;3)MRS*.

synonym: *In(3LR)MKRS*.

references: Chovnick, Schalet, Kernaghan, and Talsma, 1962, Am. Nat. 96: 281-96.

Chovnick, Ballantine, Baillie, and Holm, 1970, Genetics 66: 315-29.

Chovnick, 1973, Genetics 75: 123-31.

McCarron, Gelbart, and Chovnick, 1974, Genetics 76: 289-99.

Hilliker, Clark, Chovnick, and Gelbart, 1980, Genetics 95: 95-110.

genetics: Carries *M(3)76A*, *kar*, *ry²*, and *Sb*.

other information: Used as balancer for proximal region of chromosome 3; described as MKRS in the section on balancers. Also exists with *kar⁺* rather than *kar*, abbreviated *MRS*.

Tp(3;3)MRS*: see *Tp(3;3)MKRS

Tp(3;3)MS8: Transposition (3;3) Mglinetz Semenova

cytology: *Tp(3;3)66C;71E;95C*.

new order:

61 – 66C|71E – 95C|66C – 71E|95C – 100.

origin: γ ray induced.

references: Semenova, Mglinetz, and Glotoff, 1970,

Genetika (Moscow) 6(4): 165-69.

Mglinetz and Semenova, 1970, Genetika (Moscow) 6(8): 86-94.

Tp(3;3)P6: see *Tp(3;3)P26*

Tp(3;3)P20: see *Tp(3;3)Ubx^{P20}*

Tp(3;3)P26: Transposition (3;3) Pasadena

cytology: *Tp(3;3)81F;88B;91C-F(?)*.

synonym: *Tp(3;3)P6*.

references: Craymer.

Tp(3;3)P47

cytology: *Tp(3;3)66B;89D;92A*.

new order:

61 – 66B|(89D – 92A)|66B – 89D|92A – 100.

origin: X ray induced.

references: Craymer.

genetics: Transposition of segment carrying *Ubx⁺-D1⁺*.

Described by Craymer (1984, DIS 60: 80-82).

Tp(3;3)P146: see *Tp(3;3)S462*

Tp(3;3)S462: Transposition (3;3) of Shaw

cytology: *Tp(3;3)64C-E;89D1-2;90D1*.

new order:

61 – 64C|89D2 – 90D1|64E – 89D1|90D1 – 100.

origin: X ray induced.

discoverer: Shaw, 1973.

synonym: *Tp(3;3)P146*.

references: Lewis, 1980, DIS 55: 207-08.

Morata, Botas, Kerridge, and Struhl, 1983, J. Embryol. Exp. Morphol. 78: 319-41.

genetics: Entire *BXC* plus the normal allele of *l(3)89Ej* (Ivy) transposed to *3L*. Transposition is homozygous lethal, but the duplication recombinant *Dp(3;3)S462* is homozygous viable. The deficiency segregant *Df(3R)S462* survives as a heterozygote but ecloses one or two days later than wild type and is sterile in both sexes (Lewis, 1980).

Tp(3;3)sbd¹⁰⁴: Transposition (3;3) stubbloid

cytology: *Tp(3;3)89B5;89C;91B*.

new order:

61 – 89B5|89C – 91B|89B5 – 89C|91B – 100.

origin: X ray induced.

discoverer: E.B. Lewis, 1947.

references: 1980, DIS 55: 207-08.

Gailey, Jackson, and Siegel, 1982, Genetics 102: 771-82. Struhl, 1984, Nature 308: 454-57.

genetics: Associated with *sbd¹⁰⁴*. Homozygous lethal and lethal over *Sb*. *Dp(3;3)sbd¹⁰⁴* and *Df(3R)sbd104* derivable from *Tp(3;3)sbd¹⁰⁴/+* by recombination. *Df(3R)sbd104* is deficient for *sbd-ss* but not for the *BXC* (Lewis, 1980; Struhl, 1984).

Tp(3;3)SMG40

cytology: *Tp(3;3)87B;92A;99A*.

new order:

61 – 87B|92A – 99A|87B – 92A|99A – 100.

origin: γ ray induced.

references: Semenova, Mglinetz, and Glotoff, 1970, Genetika (Moscow) 6(4): 165-69.

****Tp(3;3)sr^{300.240}: Transposition (3;3) stripe***

cytology: *Tp(3;3)75C;89E;92A*.

new order:

61 – 75C|(89E – 92A)|75C – 89E|92A – 100.

Inserted piece said to be in inverted order but not specified whether with respect to numerical sequence or centromere.

origin: X ray induced.

discoverer: Alexander.

references: Ward and Alexander, 1957, Genetics 42: 42-54.

genetics: Mutant for *sr*. Homozygous lethal.

Tp(3;3)Tab^{rv82}: Translocation (3;3)

Transabdominal-reverted

cytology: *Tp(3;3)64F-65A;89E + In(3R)89E;90D*.

origin: X ray induced in *In(3R)Tab*.

references: Celniker and Lewis, 1987, Genes and Development 1: 111-23.

genetics: Revertant of the dominant *Tab* phenotype. When hemizygous, shows stronger transformation of the eighth ventral setal band toward that of the seventh than does *Tab/Df(3R)P9*; also A7 transformed toward A5 or A6, no posterior spiracles, and no ninth ventral setal band.

Tp(3;3)Tab^{rv96}

cytology: *Tp(3;3)80;89E + In(3R)89E;90D*.

origin: X ray induced in *In(3R)Tab*.

references: Celniker and Lewis, 1987, Genes and Development 1: 111-23.

genetics: Revertant of the dominant *Tab* phenotype. When hemizygous, shows stronger transformation of the eighth ventral setal band toward that of the seventh than does *Tab/Df(3R)P9*; also, abnormal posterior spiracles, and a rudimentary ninth ventral setal band.

molecular biology: Lesion associated with the 89E breakpoint between 170 and 173 kb to the right of the right breakpoint of *In(3R)Cbx^{rv1}*.

Tp(3;3)th^{100.293}: see *In(3L)th^{100.293}*

Tp(3;3)Ubx^{P20}: Transposition (3;3)

Ultrabithorax-Pasadena 20

cytology: *Tp(3;3)68A;68E;89E* (corrected by E.B. Lewis).

new order:

61 – 68A|68E – 89E|68A – 68E|89E – 100.

origin: X ray induced.

synonym: *Tp(3;3)P20*.

references: E.B. Lewis, 1980, DIS 55: 207-08.

genetics: Associated with *Ubx^{P20}*. Heterozygotes show slight *M* and seem to have reduced female fertility. Both duplication and deficiency derivable from the transposition.

Tp(3;3)V13: Transposition (3;3) Valencia

cytology: *Tp(3;3)61F;62A;64C* (61F deleted).

origin: X ray induced.

references: Valencia, 1970, DIS 45: 37-38.

genetics: Semi-lethal and sterile.

Tp(3;3)Vno: see *Tp(3;3)iab6^{Vno}*

Tp(3;4)h⁺44: Transposition (3;4)

hairy - wild type

cytology: *Tp(3;4)63A3-B1;67A1-2;101F* (Jeffery, 1979; Ingham *et al.*, 1981).

new order:

61 – 63A3|67A2 – 100;
101A – 101F|63B1 – 67A1|102.

origin: X ray induced.

references: Jeffery, 1971, DIS 47: 37.

1979, Genetics 91: 105-25.

Ingham, Pinchin, Howard, and Ish-Horowicz, 1985, Genetics 111: 463-86.

genetics: Homozygous lethal. Heterozygotes over *h* variegate for *h* (Jeffery, 1971). *Dp(3;4)h⁺44* carries *h⁺* and is dominant over two doses of *h* (Ingham *et al.*, 1985).

Tp(4;2)ci⁺15

cytology: *Tp(4;2)30A;101F1;102C4*.

new order:

21 – 30A|(101F1 – 102C4)|30A – 60F;

101A – 101F1|102C4 – 102F.

origin: X ray induced.

synonym: *R¹⁵(+)*.

references: Stern, MacKnight, and Kodani, 1946, Genetics 31: 598-619.

Stern and Kodani, 1955, Genetics 40: 343-73.

genetics: *Tp(4;2)ci⁺15/ci* flies show L4 vein interruptions, whereas *ci⁺/ci* flies are wild type.

Tp(4;3)ey^D: Transposition (4;3) eyeless-Dominant

cytology: *Tp(4;3)70C;101-102*.

new order:

61 – 70C|101 – 102|70C – 100;

101|102.

Most of the right arm of chromosome 4 inserted into 3L.

origin: X-ray induced derivative of *ey^D*.

references: Stern and Tokunaga, 1967, Proc. Nat. Acad. Sci. USA 57: 658-64.

genetics: Used in generation of *ey⁺* clones in *ey^D* background by mitotic exchange. + tissue develops non-autonomously with *ey^D* phenotype in basitarsus.

Tp(4;3)f

cytology: *Tp(4;3)65D;chrom4*; at least seven bands of chromosome 4 inserted into 3L (Lewis, 1956, DIS 30: 130).

origin: X ray induced.

discoverer: Sturtevant, 1930.

synonym: *T(3;4)104;T(3;4)f*.

references: Beadle, 1933, Z. Indukt. Abstamm. Vererbungslehre 65: 111-28.

James, Eissenberg, Craig, Dietrich, Hobson, and Elgin, 1989, Eur. J. Cell Biol. 50: 170-80.

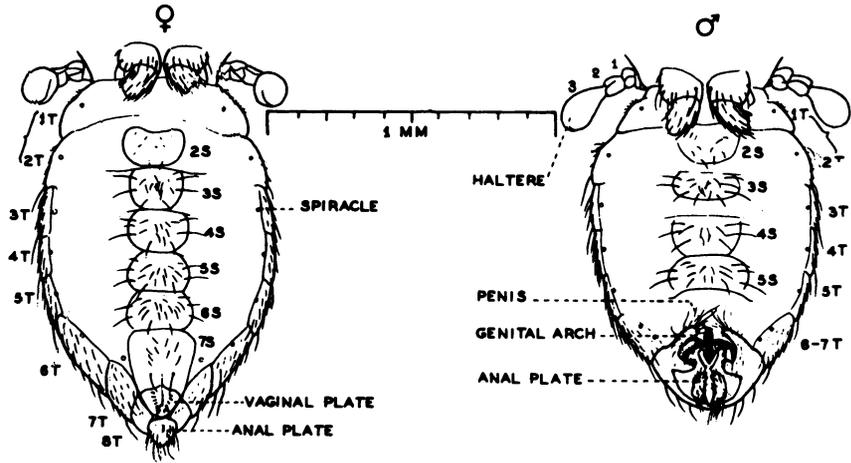
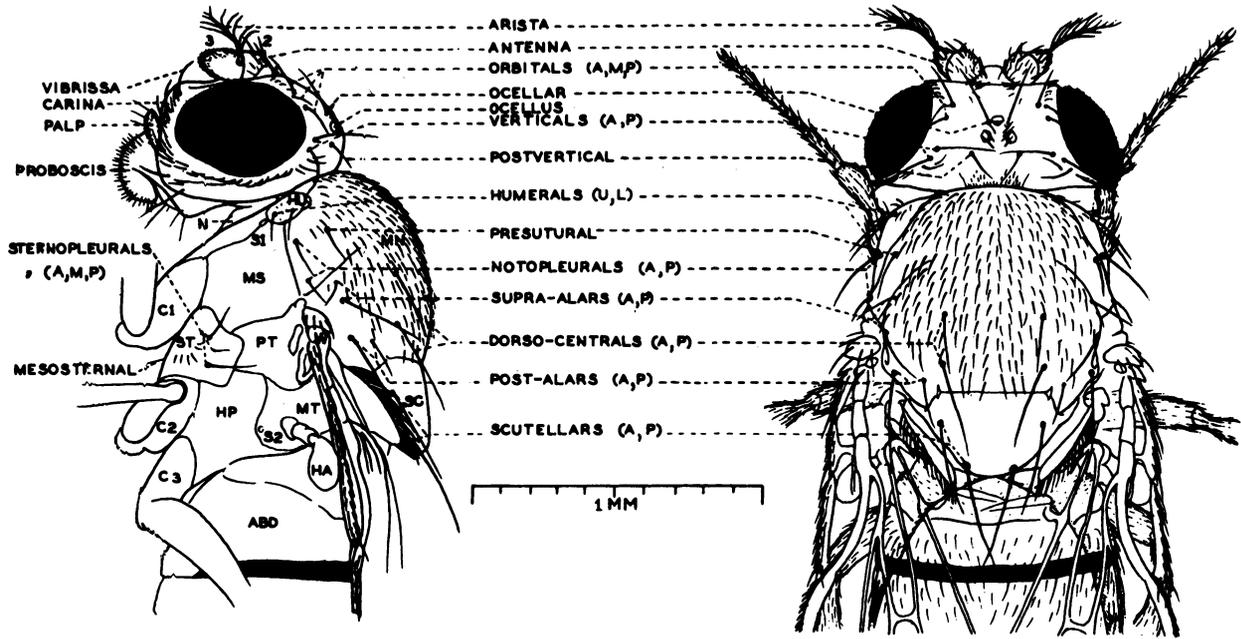
genetics: Homozygous lethal. No *ci* variegation (Stern).

X^O: see *R(1)1*

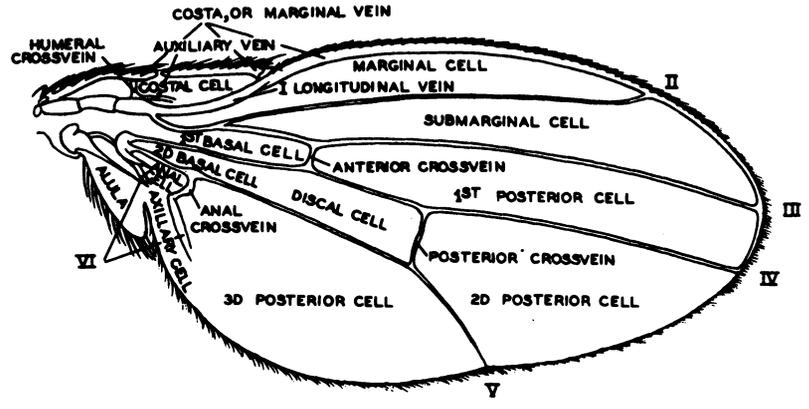
X^c: see *R(1)1*

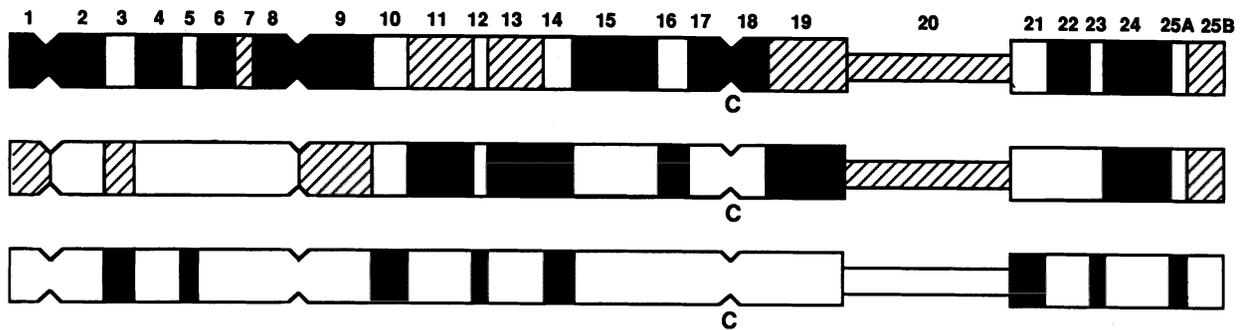
X^{c2}: see *R(1)2*

THE GENOME OF *DROSOPHILA MELANOGASTER*



- LEGEND:-
- A --- ANTERIOR
 - ABD - ABDOMEN
 - C1, C2, C3 - COXAE
 - HA -- HALTERE
 - HP -- HYPOPLEURA
 - HU -- HUMERUS
 - L --- LOWER
 - M --- MIDDLE
 - MN -- MESONOTUM
 - MS -- MESOPLEURA
 - MT -- METANOTUM
 - N --- NECK
 - P --- POSTERIOR
 - PT -- PTEROPLEURA
 - S --- STERNITE
 - S1,S2-THORACIC SPIRACLES
 - SC -- SCUTELLUM
 - ST -- STERNOPLAURA
 - T --- TERGITE
 - U --- UPPER
 - W --- WING





Schematic representation of the prophase differentiation of the Y chromosome. Solid segments are brightly fluorescing or, in the case of N banding, darkly staining; crosshatched regions are moderately fluorescing; hatched regions are dully fluorescing; and empty regions are non staining or non fluorescing. The upper diagram represents the fluorescence of Hoechst-33258-stained chromosomes; the second row depicts the quinacrine staining pattern; the third row indicates chromomycin staining; and the fourth row shows the N-banding pattern (provided by Gatti and Pimpinelli).

NORMAL CHROMOSOME COMPLEMENT

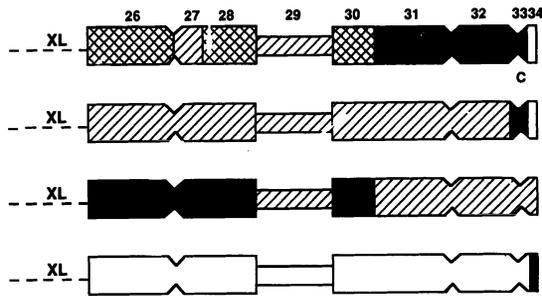
This section contains brief descriptions of the normal chromosome cytology, both in mitotic and polytene cells. Polytene maps are included as foldouts at the end of the book, and combine material from C. B. Bridges (1935, *J. Hered.* 26: 60-64; 1938, *J. Hered.* 29: 11-13), C. B. Bridges and P. N. Bridges (1939, *J. Hered.* 30: 475-76) and P. N. Bridges (1941, *J. Hered.* 32: 64-65; 299-300; 1942, *J. Hered.* 33: 403-408), and Lefevre [1976, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.), Academic Press, London, New York, San Francisco, Vol. 1a, pp. 31-66]. In addition detailed polytene maps based on electron-microscopic examination of thin sections of polytene-chromosome squashes were presented, one numbered section at a time, by Sorsa (1988, *Chromosome Maps of Drosophila*, CRC Press, Inc., Boca Raton, Florida, Vol II). Each of the major chromosome arms is divided into twenty numbered sections, each of which is divided into six subsections, lettered A-F, and within which the bands are numbered sequentially.

Mitotic prophase cytology has been perfected by Gatti and Pimpinelli; using several fluorescent dyes, principally, Hoechst 33258, and N-banding techniques they are able to detect considerable linear differentiation of the pericentric heterochromatin, which is not amplified and thus not amenable to analysis in polytene chromosomes. The main publications dealing with prophase cytology are Gatti and Pimpinelli (1983, *Chromosoma* 88: 349-73) and Dimitri (1991, *Genetics* 127: 553-64).

X chromosome

Also known as chromosome 1. Present in one dose in male and two doses in female. In mitotic figures, the X is a rod-shaped element with a nearly terminal centromere and a minute second arm designated as the right arm, XR. The left arm, XL, is divided into a distal isopycnotic euchromatic region, Xe, in which the chromatids are usually separated, and a proximal heterochromatic or heteropycnotic region, Xh, in which the chromatids are not separated. The heterochromatic region comprises one-third to one-half the DNA content of the X chromosome. Tritiated thymidine incorporation studies (Barigozzi, Dolfini, Fraccaro, Raimondi, and Tiepolo, 1966, *Exp. Cell Res.* 43: 231-34) demonstrate that the heterochromatic region is late replicating. In second meiotic prophase of spermatogenesis, on the other hand, both euchromatin and heterochromatin are visibly double. In mitotic prophase cells, the heterochromatic region can be subdivided into nine segments by means of a variety of staining procedures (Gatti and Pimpinelli); these are designated, from left to right, h26 through h34; h26 is juxtaposed to the euchromatin; the ribosomal cistrons are thought to occupy h29 and the centromere has been placed between h32 and h33. Cooper (1959, *Chromosoma* 10: 535-88) characterized the heterochromatin of aceto-orcein-stained prophase X chromosomes as comprising four major orcein-staining segments designated from right to left hA, hB, hC, and hD and separated by three secondary constrictions, the central one of which corresponds to the nucleolus organizer, *i.e.*, the site of the

bb locus, which comprises a tandem array of repeats encoding the 5.8 S, 18 S, and 28 S ribosomal RNA's; other than the nucleolus organizer, correspondences between Cooper's map on one hand and that of Gatti and Pimpinelli on the other have not been determined.



Schematic representation of the prophase differentiation of the pericentric heterochromatin of the X chromosome. Solid segments are brightly fluorescing or, in the case of N banding, darkly staining, crosshatched regions moderately fluorescing, hatched regions dully fluorescing, and empty regions are non staining or nonfluorescing. The upper diagram represents the fluorescence of Hoechst-33258 stained chromosomes; the second row depicts the quinacrine staining pattern; the third row indicates chromomycin staining; and the fourth row shows the N-banding pattern (provided by Gatti and Pimpinelli).

The polytene X chromosome (see foldout at end of book) extends from region 1 at the tip through region 20 at the centromere; *Xe* comprises approximately 1000 bands (1008 according to Bridges and 979 according to Sorsa), and is thought to extend at least through 20D, with 20E and F perhaps corresponding to the distalmost portion of *Xh* and residing in the chromocenter; all known genes except *bb* and possibly *su(f)* are located distal to 20E [Schalet and Lefevre, 1973, *Chromosoma* 44: 181-202; Lefevre, 1976, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 31-66]. Several polytene landmarks given mnemonic designations by Bridges (1935, *J. Hered.* 26: 60-64) are as follows: the puff at 2B, the four brothers in 9A, the two sisters in 10A and B, the weak spot in 11A, the chains in 15B and D, the turnip in 16A-B, and the offset in 19E.

Y chromosome

In mitotic figures, the Y chromosome appears as an entirely heterochromatic element; tritiated thymidine incorporation studies (Barigozzi, Dolfini, Fraccaro, Raimondi, and Tiepolo, 1966, *Exp. Cell Res.* 43: 231-34) show it to be late replicating. The Y is a two-armed chromosome, the short arm, YS, being about two-thirds the length of the long arm, YL. At metaphase the Y is somewhat shorter than the X chromosome. Sister chromatids are not in evidence in prophase or metaphase Y chromosomes during mitosis, but in second meiotic prophase of spermatogenesis sister chromatids are well separated. In mitotic prophase preparations the Y chromosome can, by a combination of staining procedures, be subdivided into 25 distinct segments (Gatti and Pimpinelli, 1983, *Chromosoma* 88: 349-73); they are designated h1 at the

terminus of YL through h25 at the end of YS (see diagram on the preceding page). Gatti and Pimpinelli have placed the Y male-fertility genes as follows: *kl-5* in h2-h3, *kl-3* in h4-h9, *kl-2* in h10, *kl-1* in h13-h15, *ks-1* in h21-h23, and *ks-2* in h24-h25; in addition, Su(Ste) resides in h11 (Hardy, Lindsley, Livak, Lewis, Sivertsen, Joslyn, Edwards, and Bonaccorsi, 1984, *Genetics* 107: 591-610), and the ribosomal cistrons of the Y are in h20 (Gatti and Pimpinelli). Mapping of eight different satellite DNA sequences with respect to the 25 segments of the Y is described by Bonaccorsi and Lohe (1991, *Genetics* 129: 177-89). In aceto-orcein-stained prophase chromosomes Cooper (1959, *Chromosoma* 10: 535-88) reported that YS is subdivided by the nucleolus constriction into a distal segment, SB, comprising two-thirds of YS, and a proximal segment, SA, comprising the other one-third; YL is divided into three segments of approximately equal length by two constrictions, the more distal of which is the more constant landmark. From the centromere, the segments are designated LA, LB, and LC. Gatti and Pimpinelli have equated the constriction between LB and LC with their brightly fluorescing segments h8-h9 and in general find a correspondence between secondary constrictions seen in aceto-orcein- or Giemsa-stained chromosomes with brightly fluorescing blocks and not with nonfluorescing gaps in Hoechst-stained material.

The Y chromosome does not undergo polytenization. In salivary-gland preparations, Prokofyeva-Belgovskaya (1937, *Genetics* 22: 94-103) observed a small collection of bands that she attributed to the Y. However, Nicoletti and Lindsley (1960, *Genetics* 45: 1705-22) found no evidence of bands attributable to the Y chromosome in a study of *T(1;Y)*'s; their observations were confirmed in the polytene analysis of a large collection to *T(Y;A)*'s (Lindsley, Sandler, Baker, Carpenter, Denell, Hall, Jacobs, Miklos, Davis, Gethmann, Hardy, Hessler, Miller, Nozawa, Parry, and Gould-Somero, 1972, *Genetics* 71: 157-84).

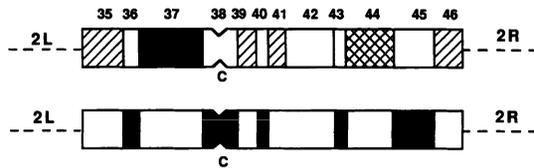
Several complex structural elements in the primary spermatocyte nucleus depend on the presence of the Y chromosome; two, designated tubuli and reticular material in electron micrographs (Meyer, Hess, and Beerman, 1961, *Chromosoma* 12: 676-716), are associated with the male-fertility genes, *kl-5* and *kl-3*, respectively (Hardy, Tokuyasu, and Lindsley, 1981, *Chromosoma* 83: 593-617). These and another associated with *ks-1* can be recognized in the light microscope (Bonaccorsi, Pisano, Puoti, and Gatti, 1988, *Genetics* 120: 1015-34). Their function is unknown.

Addition of Y chromosomes to a normal chromosome complement suppresses variegated position effects, and removal of the Y from the male enhances variegation (1933, Gowan and Gay, *Proc. Nat. Acad. Sci. USA* 19: 122-26). Two or more Y's added to the normal complement cause variegation of otherwise self-colored eyes (Cooper, 1956, *Genetics* 41: 242-64).

Chromosome 2

In mitotic figures, chromosome 2 is less than twice the length of the X and slightly smaller than chromosome 3. It contains two euchromatic arms separated by the centromere and the pericentric heterochromatin. In acetic-orcein-stained preparations of mitotic prophase chromosomes, the pericentric heterochromatin comprises two

segments separated by the centric constriction; Gatti and Pimpinelli, utilizing Hoechst 33258 and N banding, recognize linear differentiation of the pericentric heterochromatin into twelve segments, designated from left to right h35 to h46; h42 is subdivided into 42A and 42B in some strains; the centromere is placed in h38. Dmitri (1991, *Genetics* 127: 553-64) has produced a cytogenetic map of the chromosome-2 heterochromatin; the *l(2)40F* loci, including *lt*, as well as *cta* and *E(SD)* are confined to segment h35 at the euchromatic-heterochromatic junction; the responder sequences, *Rsp*, comprise segment 39 in *2R*, which varies in size according to the sensitivity of the chromosome to distortion by *SD* (Pimpinelli and Dimitri, 1989, *Genetics* 121: 765-72); in addition, *l(2)41Ab* is located at the h39-h40 junction, *l(2)41Aa* and *rl* are in h40, and *l(2)41Ad* is in h43-h44; *l(2)41Ae* and *l(2)41Ah* are located in h46 at the heterochromatic-euchromatic junction. Dimitri has also located a number of heterochromatic breakpoints in chromosome 2.



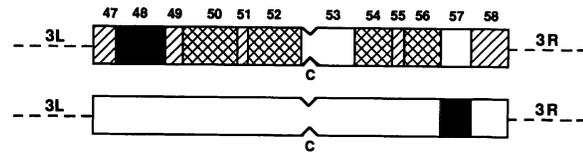
Schematic representation of the prophase differentiation of the pericentric heterochromatin of chromosome 2. Solid segments are brightly fluorescing or, in the case of N banding, darkly staining; crosshatched regions moderately fluorescing; hatched regions dully fluorescing; and empty regions are non staining or non fluorescing. The upper diagram represents the fluorescence of Hoechst-33258 stained chromosomes; the second row depicts the N-banding pattern (provided by Gatti and Pimpinelli).

In polytene preparations the left arm contains 804 bands according to Bridges and 869 as estimated by Sorsa, with an upper limit of 927; its 20 sections are numbered 21-40 from tip to centromere. Land mark regions named by Bridges (1935, *J. Hered.* 26: 60-64) for mnemonic purposes include the dog collar at 21C-D, the shoe buckle at 25A, the shield at 30A, the gooseneck at 31B-F; in addition, regions of the proximal half of *2L* tend to fold back on themselves, frequently imparting a convoluted conformation to this part of the chromosome; these regions are termed the spiral loop in 31B-F, the turn back in 36, and the basal loop in region 37-39. The right arm of chromosome 2 is subdivided into 20 sections labeled 41 at the centromere to 60 at the tip; it contains 1136 bands on the Bridges maps and 1009 to 1152 according to Sorsa, depending on whether doublets are considered single or double; its landmarks are the onion base at 42A, the barrel in section 47 [Lefevre, 1976, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.). Academic Press, London, New York, San Francisco, Vol. 1a, pp. 31-66], goggles at 50A and B (Lefevre), the miniskirt at 50C-D (Strathern), and the huckleberry tip at 60E.

Chromosome 3

In mitotic figures, chromosome 3 is less than twice the length of the *X* and slightly larger than chromosome 2. It contains two euchromatic arms separated by the centromere and the pericentric heterochromatin. In acetic-

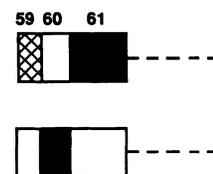
orcein-stained preparations of mitotic prophase chromosomes, the pericentric heterochromatin comprises two segments separated by the centric constriction. Gatti and Pimpinelli subdivide the third-chromosome heterochromatin into twelve sections, numbered from left to right h47 through h58; the centromere is placed in h53, but other than that no further characterization has been undertaken.



Schematic representation of the prophase differentiation of the pericentric heterochromatin of chromosome 3. Solid segments are brightly fluorescing or, in the case of N banding, darkly staining; crosshatched regions moderately fluorescing; hatched regions dully fluorescing; and empty regions are non staining or non fluorescing. The upper diagram represents the fluorescence of Hoechst-33258 stained chromosomes; the second row depicts the N-banding pattern (provided by Gatti and Pimpinelli).

In polytene preparations, the left arm of chromosome 3, *3L*, comprises twenty divisions numbered from tip to centromere 61 through 80 and containing 884 bands according to P. N. Bridges. Sorsa's estimates are considerably higher; considering doublets to be single bands he recognizes 1032 bands and considering them as two bands he enumerates 1073. The mnemonics suggested by Bridges for designating cytological landmarks on *3L* are the barrel at 61C-F, the ballet skirt at 68B-C, the Chinese lanterns, two large puffs, at 74-75, and the graded capsules in 79D-E. The twenty divisions of *3R*, numbered 81F (81A-E do not appear on polytene maps) at the centromere through 100 at the tip contain 1178 bands according to P. N. Bridges. Sorsa estimates that there are 1147 bands if doublets are treated as single bands and 1233 if they are treated as two. Bridges' landmarks for *3R* are the cucumber base in 81 to 83D, the road apple at 85F (Strathern), the duck's head at 89E-91A, and the goblet tip at 100.

Chromosome 4



Schematic representation of the prophase differentiation of the heterochromatin of chromosome 4. Solid segments are brightly fluorescing or, in the case of N banding, darkly staining; crosshatched regions moderately fluorescing; hatched regions dully fluorescing; and empty regions are non staining or non fluorescing. The upper diagram represents the fluorescence of Hoechst-33258 stained chromosomes; the second row depicts the N-banding pattern (provided by Gatti and Pimpinelli).

In mitotic figures, chromosome 4 is a dot-like element that is separated into two segments (*4L* and *4R*) of

grossly unequal size by a sometimes-visible centric constriction. The bulk of chromosome 4 is heterochromatic and in prophase has been differentiated into three segments designated h59, h60, and h61 by Gatti and Pimpinelli. The presence of a short left arm is indicated by its involvement in chromosome rearrangements [e.g., $T(1;4)_{wm}^{11}$ (Panshin and Khvostova, 1938, Biol. Zh. 7: 359-80) and $T(1;4)_{w}^{mA}$ (Griffen and Stone, 1940, Texas Univ. Publ. 4032: 201-07)].

In polytene configurations, chromosome 4 is a short element emerging from the chromocenter. It is divided into two sections, 101 proximally and 102 distally; no reliable bands can be identified proximal to 101D, nor does any of the material in 4L polytenize (Lefevre). Hochman [1976, *The Genetics and Biology of Drosophila* (Ashburner and Novitski, eds.), Academic Press, London, New York, San Francisco, Vol. 1b, pp. 903-28], Lefevre, and Sorsa agree that the 137 bands on the detailed map of chromosome 4 produced by Slizynski (1944, J. Hered. 35: 322-25), and included in the previous edition of this work, cannot be confirmed. They estimate 40-50 bands. Note that the centromere positions of the two subtelocentric elements of the genome differ. It is on the right in the case of the X and on the left in chromosome 4.

4-sim: chromosome 4 from *Drosophila simulans* (H. A. Orr)

A single fourth chromosome from *D. simulans* was introduced into an otherwise *D. melanogaster* genome by Muller and Pontecorvo (1940, *Nature* 146: 199-200; 1940, *Science* 92: 418). Phenotypic effects of 4-sim were described by Muller and Pontecorvo (1942, *Genetics* 27: 157) and Pontecorvo (1943, *Proc. Roy. Soc. Edinburgh B* 61: 385-97; *J. Genet.* 45: 51-66). Although 4-sim/4 is apparently normal, 4-sim/4-sim homozygotes suffer decreased viability and show subtle peculiarities: flattened body, heavy trident, and reduced eyes. 4-sim/4-sim male genitalia also slightly resemble those of *D. simulans* (Muller and Pontecorvo, 1942; Coyne, 1983, *Evolution* 37: 1101-18). Some genes on 4-sim show unusual dominance relations: spa^{Cat} is not seen in 4-sim/ spa^{Cat} heterozygotes. Conversely, 4-sim/ ci shows occasional wing-vein interruptions; 4-sim/ ci^W shows more extreme cubitus-interruptus phenotype than 4/ ci^W (Uphoff, 1949, *Genetics* 34: 315-27). Appearance of ci is not due to the presence of a mutation at ci^+ on the single 4-sim chromosome introduced into *D. melanogaster* as Sturtevant (1946, *Genetics* 31: 259-68) suggested: *D. melanogaster ci*—*D. simulans ci*⁺ species hybrids also occasionally show ci [several *D. simulans* stocks tested (Uphoff, 1949; Orr, unpublished)]. Segregation of 4-sim

and 4 is fairly regular (Muller and Pontecorvo, 1942).

4-sim/4-sim are completely sterile, whereas 4-sim/4 males are fully fertile. All female genotypes are fertile. Testes of sterile males appear normal. Meiosis occurs, but very few motile sperm are produced; about 75% of 4-sim/4-sim males produce no motile sperm at all (Orr, 1992, *Genet. Res.*). Pontecorvo (1943) claimed that sterility results from a breakdown in the last stages of spermiogenesis. The factor(s) causing sterility map to the proximal end of chromosome 4 within a large Minute deletion (Muller and Pontecorvo, 1942). Subsequent deletion and translocation mapping shows that sterility maps to 101F, 102A2-B5, or both, representing less than 15% of the cytological length of the dot chromosome. $Df(4)M101-3/4-sim$ and $Df(4)M101-62f/4-sim$ are male sterile; $Df(4)M101-63a/4-sim$ and $Df(4)G/4-sim$ are fertile (Orr, 1992). Male sterility is therefore apparently due to a single gene, dubbed *hms* (hybrid male sterile). *hms* complements all known genes in the $Df(4)M101$ region and shows no interaction with testis-specific β -tubulin. 4-sim male sterility depends only on zygotic genotype at *hms*.

Comparisons of *D. melanogaster* and *D. simulans* polytene chromosomes were made by Kerkis (1936, *Am. Nat.* 70: 81-86), Horton (1939, *Genetics* 24: 234-43) and Patau (1935, *Naturwissenschaften* 23: 537-43). Chromosome 4 of *D. simulans* differs from that of *D. melanogaster* by a fairly long inversion, including at least 102B1-2 through 102E1-2 and probably 102E3, 4, and 5 (Horton, 1939). A darkly staining terminal ring is sometimes seen at the tip of the *D. simulans* chromosome. The proximal third of chromosome 4 from *D. simulans* tends to be narrower than the distal two-thirds. Slizynski (1941, *Proc. Roy. Soc. Edinburgh B* 61: 95-106) claimed to identify the short left arm of 4-sim and compares it with that of *D. melanogaster*; however, recent workers dispute the claim that 4L can be seen in polytene preparations. Horton (1939) never observed synapsis of chromosome 4 in salivary-gland cells of *D. melanogaster*—*D. simulans* species hybrids. Although 4-sim and 4 in the "4-sim" stock of *D. melanogaster* also do not usually pair, Slizynski (1941) found one nucleus in which the inverted segments paired. In triplo-4 larvae, the *D. melanogaster* 4' s pair and the 4-sim chromosome remains unpaired. As expected, the 4-sim chromosome from the 4-sim stock pairs normally with wild-type *D. simulans* fourth chromosomes in species hybrids (Orr, 1992). In both species hybrids and on a *D. melanogaster* genetic background, the tip of the *D. melanogaster* 4R tends to stick into the chromocenter; the tip of the *D. simulans* 4, however, is always free of the chromocenter (Horton, 1939; Slizynski, 1941).

SPECIAL CHROMOSOMES



Balancers
Compound Chromosomes
X-Y Combinations
Y Derivatives
Autosynaptic Chromosomes

C(1)A

BALANCERS

Balancers are chromosomes that are designed to maintain a homologous chromosome or a segment thereof intact in a cross or stock. They share three properties: (1) they are multiply inverted in order to suppress recombination with homologous chromosomes; (2) they are usually lethal, sterile in one sex or the other, or exhibit low viability in the homozygous condition in order to avoid displacing their homologues in balanced stocks; and (3) they are marked in such a way that their presence in heterozygotes (and, where they survive, as homozygotes) is recognizable, usually with a combination of dominant and recessive mutant alleles. The most crucial aspect of balancers is their ability to suppress recombination, and therefore their rearranged sequence. In this treatment we arrange balancers according to chromosome; balancers with the same rearranged sequence are treated together as a single entry; different marker constitutions as indicated by identifying mnemonics or version designations are tabulated within such entries.

X-CHROMOSOME BALANCERS

asc: see $In(1)sc^{SIL}sc^{8R}+S$

Basc: see $In(1)sc^{SIL}sc^{8R}+S$

Binsc: see $In(1)sc^{SIL}sc^{8R}+dl49$

Binscy: see $In(1)sc^{SIL}sc^{8R}+dl49$

Binsincy: see $In(1)sc^{SIL}sc^{8R}+dl49$

Binsn: see $In(1)sc^{SIL}sc^{8R}+dl49$

Biny: see $In(1)sc^{8L}sc^{S1R}+dl49$

C(1)M4: see section on compound chromosomes

CIB

constitution: $In(1)Cl, sc\ l(1)C\ t^2\ v\ sl\ B.$

synthesis: Muller.

references: 1928, Genetics 13: 279-357.

properties: Male lethal. Suppresses crossing over in the X chromosome. Originally used in recovery of sex-linked recessive lethals; replaced by *Basc* for this purpose.

complete: see $In(1)sc^8+dl49$

finsky: see $In(1)sc^{SIL}sc^{8R}+dl49$

FM0: see $In(1)sc^8+dl49$

FM1: see $In(1)sc^8+dl49$

FM3: First Multiple 3

constitution: $In(1)FM3, y^{31d}sc^8\ dm\ B.$

order: 1A—1B2|20F|16B—19F|3F—4D7|11F2—4E1|11F4—16A|3E—1B|20F.

synthesis: R.F. Grell, 1954.

references: Mislove and Lewis, 1954, DIS 28: 77.

properties: Male lethal owing to presence of two multilocus deficiencies in $In(1)FM3$, which may be covered by B^SYy^+ or by B^SY and y^+Y (Schalet). Effectively suppresses crossing over in the X chromosome. Useful for balancing sex-linked recessive, female-sterile mutants and, in combination with B^SYy^+ , for balancing sex-linked recessive lethal and male-sterile mutants.

FM4

constitution: $In(1)FM4, y^{31d}sc^8\ dm\ B.$

order: 1A—1B2|20F—11F4|4E|3C—4D7|11F2—4F|3C—1B3|20F.

synthesis: R.F. Grell, 1954.

references: Mislove and Lewis, 1954, DIS 28: 77.

properties: Male viable and fertile; homozygous female viable but sterile owing to *dm*. $In(1)FM4$ is the consequence of the approximate reinversion of $In(1)dl49$ in $In(1)sc^8+dl49$ and is similar in sequence to $In(1)sc^8$ but with the transposition of 3C-4F into 11F. Unless this small transposition has an abnormally large effect on crossing over (e.g., see $Dp(2;2)C619$) recombination might be expected to be frequent in $FM4/+$ heterozygotes and especially in $FM4/In(1)sc^8$ heterozygotes. In $FM4/+$ heterozygotes, double crossovers with points of exchange inside or outside the 3C-11F interval produce euploid X chromosomes, and those with one point of exchange inside and one outside produce complementary duplications and deficiencies for 3C—4F. The duplication survives in either sex and exhibits a Confluens phenotype (E.H. Grell); the deficiency might survive in the heterozygote as a Notch Minute female judging from the survival of the slightly smaller $Df(1)w^{m258-44} = Df(1)3C3-4;4D2-E1$. Balancing properties not well determined. Some lines carry w^{55f} and in some y^{31d} replaced with y^+ or B with f or $+$.

FM6

constitution: $In(1)FM6, y^{31d}sc^8\ dm\ B.$

order: 1A—1B2|20F|15E—20F|15D—11F4|4E|3C—4D7|11F2—4F|3C—1B3|20F.

synthesis: R.F. Grell, 55i.

references: Grell and Lewis, 1956, DIS 30: 71.

properties: Male viable and fertile; homozygous females viable but sterile owing to the presence of *dm*. Like *FM4* except for the presence of the additional $In(1)15D-E;20A-B$. Reservations similar to those about the balancing ability of *FM4* apply in *FM6* to the salivary chromosome region from 1B to 15D. In genotypes with a normal recombination rate, *FM6* effectively eliminates recombination in $FM6/+$ heterozygotes but yields viable recombinants when heterozygous for such inversions as $In(1)sc^8$. Used for balancing sex-linked recessive lethal and sterile mutations. Does not effectively balance *cv* or *v* in stocks that are also heterozygous for $In(2LR)SM$ and $In(3LR)Ubx^{130}$. Different markers have been introduced into *FM6*; they include *w* and dm^+ ; the dm^+ is probably a duplication introduced by a double crossover in regions 1B3—3C and 4F—11F2; *FM6*, dm^+ homozygotes are fertile. In addition recessive-lethal-bearing *FM6* chromosomes have been described (e.g., Lifschytz and Falk, 1968, Mutat. Res. 6: 235-44).

FM7

constitution: $In(1)FM7, y^{31d}sc^8\ w^a\ B.$

order: 1A—1B2|20F—20E|15E—20A|15D—11F4|4E1—11F2|4D7—1B3|20F.

properties: Males and homozygous females viable and fertile. An excellent suppressor of recombination with normal-sequence X chromosomes. Commonly used in balancing, even in female fertile versions.

variants:

version	additional markers	ref ^α	comments
FM7a	<i>v</i> <i>Of</i>	1, 2	
FM7b	<i>lz</i> ^{sp}	2	female sterile
FM7c	<i>sn</i> ^{X2} <i>v</i> <i>Of</i> <i>g</i> ⁴	3, 4	female sterile

^α 1 = Merriam, 1968, DIS 43: 64; 2 = Merriam, 1969, DIS 44: 101; 3 = Merriam and Duffy, 1972, DIS 48: 43; 4 = Robbins, 1977, Genetics 87: 665-84.

In(1)dl49+B^{M1}

constitution: *In(1)dl49+B^{M1}, sc v B^{M1}*.

order: 1A—4D7|11F2—4E1|11F4—16A2|20D—16A5|20F.

properties: Males and homozygous females viable and fertile. Effective suppressor of crossing over in X chromosome.

In(1)sc^{4L}sc^{8R}+dl49

constitution: *In(1)sc^{4L}sc^{8R}+dl49, y sc⁴sc⁸w^a*.

order: 1A—1B3|20F—11F4|4E1—11F2|4D7—1B3|20F.

references: Gethmann, 1971, Mol. Gen. Genet. 114: 144-55.

properties: Effective in suppressing crossing over with X chromosome in normal sequence. Deficient for proximal heterochromatin, including *bb*; accordingly X-Y nondisjunction high, and homozygous females lethal.

In(1)sc^{4L}sc^{8R}+S

constitution: *In(1)sc^{4L}sc^{8R}+S, y sc⁴sc⁸w^aB*.

order: 1A—1B3|20F—11A1|6A3—10F10|6A1—1B3|20F.

references: Lindsley, Edington, and Von Halle, 1960, Genetics 45: 1649-70.

properties: Male viable and fertile; homozygous females and X0 males inviable because of deficiency for *bb*. Suppresses crossing over in X chromosome. Used in recovery of Y-suppressed lethals.

In(1)sc⁷AM

constitution: *In(1)sc⁷+AM, sc⁷*.

order: 1A—1B3|6D8—1B4|6D8—8C17|16E2—8D1|16E3—20.

properties: Male viable and fertile; homozygous female viable but sterile because of homozygous *In(1)AM*. Reduces X-chromosome crossing over. May be used to balance sex-linked recessive lethal or sterile mutations.

In(1)sc⁸+dl49

order: 1A—1B2|20F—11F4|4E1—11F2|4D7—1B3|20F.

properties: Effective in eliminating recombination with X chromosomes in normal sequence.

variants:

mnemonic	markers	ref ^α	comments
FMO	<i>y</i> ^{31d} <i>sc</i> ⁸ <i>w</i> <i>cv</i> <i>v</i> <i>Of</i> <i>m</i> ² <i>v</i> <i>f</i> <i>B</i>	3	
FM1 β	<i>y</i> ^{31d} <i>sc</i> ⁸ <i>w</i> ^a <i>lz</i> ^s	1	female sterile
M6	<i>y</i> ^{31d} <i>sc</i> ⁸ <i>w</i> ^a <i>v</i> <i>Of</i> <i>f</i>	2	
M9	<i>y</i> ^{31d} <i>sc</i> ⁸ <i>w</i> ^a <i>mei</i> ^{9L1} <i>vb</i> <i>v</i> <i>Of</i> <i>f</i>	4	

^α 1 = Lewis and Mislove, 1963, DIS 27: 57-58; 2 = Mortin and Lefevre, 1981, Chromosoma 82: 237-47; 3 = Rawls and Porter, 1979, Genetics 93: 143-61; 4 = Schalet (unpublished).

β Originally designated *complete*.

In(1)sc^{8L}sc^{S1R}+dl49

constitution: *In(1)sc^{8L}sc^{S1R}+dl49, y^{31d}sc⁻v^{Of}fB*.

order: 1A—1B2|20F—11F4|4E1—11F2|4D7—1B4|20F.

synonym: *Biny*.

references: Lindsley and Edington, 1957, DIS 31: 131-32.

Lindsley, Edington, and Von Halle, 1960, Genetics 45: 1649-70.

properties: Male lethal owing to deficiency for *sc*. Suppresses crossing over in the X chromosome. Used in the recovery of Y-suppressed, sex-linked recessive lethals.

In(1)sc^{S1}+dl49

order: 1A—1B3|20F—11F4|4E1—11F2|4D7—1B4|20F.

properties: Male viable and fertile; homozygous female viable but sterile owing to homozygosity for *In(1)sc^{S1}*. Reduces crossing over in X chromosome. May be used to balance sex-linked lethal or sterile mutations.

In(1)sc^{S1L}sc^{8R}+dl49

constitution: *In(1)sc^{S1L}sc^{8R}+dl49, sc⁸sc^{S1}*.

order: 1A—1B3|20F—11F4|4E1—11F2|4D7—1B3|20F.

properties: Viable and fertile in both males and homozygous females; effective in suppressing crossing over with a normal-sequence X chromosome. Exists with many marker combinations, most of which were assembled by Muller.

variants:

mnemonic	markers	ref ^α	comments
Binsc	<i>B</i>		
Binscy	<i>y</i> ^{c4} <i>B</i>	4, 5	
Binsincy	<i>y</i> ^{c4} <i>w</i> <i>sn</i> ^{X2} <i>B</i>	1, 2	female sterile
Binsn	<i>sn</i> ^{X2} <i>B</i>		female sterile
flnscy	<i>y</i> ^{c4} <i>v</i> <i>Of</i> <i>f</i>	3	
lncs	—		
lncy	<i>y</i> ^{c4}		
winscy	<i>y</i> ^{c4} <i>w</i>		

^α 1 = Arking, 1975, Genetics 80: 519-37; 2 = Bryant and Zornetzer, 1973, Genetics 75: 623-37; 3 = Gethmann, 1971, Mol. Gen. Genet. 114: 144-55; 4 = Muller, 1952, DIS 26: 113-14; 5 = Muller and Oster, 1963, Methodology in Basic Genetics (W.J. Burdette, ed.). Holden-Day, Inc., San Francisco, pp. 249-78.

In(1)sc^{S1L}sc^{8R}+S

constitution: *In(1)sc^{S1L}sc^{8R}+S, sc⁸sc^{S1}*.

order: 1A—1B3|20F—11A1|6A3—10F10|6A1—1B3|20F.

references: Spencer and Stern, 1948, Genetics 33: 43-74.

Baker, 1973, Dev. Biol. 33: 429-40.

properties: Males and homozygous females viable and fertile; X0 male poorly viable; variegated for *y*, *ac*, *cin* and presumably for other lethals distal to *y*. Suppresses crossing over in X, but less so than *In(1)sc^{S1L}sc^{8R}+dl49*, since *In(1)S = In(1)6A1-3;10F10-11A1* is less effective than *In(1)dl49 = In(1)4D7-E1;11F2-4*. Routinely used in the detection of sex-linked recessive lethals.

variants:

mnemonic	markers	ref ^α
asc	<i>w</i> ^a	
Basc = M5	<i>w</i> ^a <i>B</i>	2
Bascy	<i>y</i> ⁸² <i>w</i> ^a <i>B</i>	1

^α 1 = Blount, 1982, DIS 58: 154; 2 = Spencer and Stern, 1948, Genetics 33: 43-74.

Insc: see *In(1)sc^{S1L}sc^{8R}+dl49*

Inscy: see *In(1)sc^{S1L}sc^{8R}+dl49*

M5: see *Basc* under *In(1)sc^{SIL}sc^{8R}+S*

M6: see *In(1)sc⁸+dl49*

M9: see *In(1)sc⁸+dl49*

Muller 5: see *Basc* under *In(1)sc^{SIL}sc^{8R}+S*

winscy: see *In(1)sc^{SIL}sc^{8R}+dl49*

SECOND-CHROMOSOME BALANCERS

CyO: *Curly of Oster*

constitution: *In(2LR)O, Cy dp^{lv} pr cn²*.

order: 21—22D|33F5—30F|50D1—58A4|42A2—34A1|22D2—30E|50C10—42A3|58B1-60.

synthesis: Oster.

synonym: *Cy, InsO5*.

references: 1956, DIS 30: 145.

O'Donnell, Gerace, Leister, and Sofer, 1975, *Genetics* 79: 73-83.

properties: More effective suppressor of crossing over in chromosome 2 than *In(2L+2R)Cy*; should be superior to *SM1* as a balancer for chromosome 2. Other markers introduced into *CyO* include *Adh^{nA}* and *Adh^{nB}* (O'Donnell *et al.*), *l(2)DTS100*, *l(2)DTS486*, and *l(2)DTS573* (Falke and Wright, 1972, DIS 48: 89), as well as *b, bw, esc², pk^{81h}, Roi, sple^{81h}*, and others.

SM1: *Second Multiple 1*

constitution: *In(2LR)SM1, al² Cy cn² sp²*.

order: 21—22A3|60B—58B1|42A3—58A4|42A2—34A1|22D2—33F5|22D1—22B1|60C—60F.

synthesis: R.F. Grell, 1953.

references: Lewis and Mislove, 1953, DIS 27: 58.

properties: Homozygous lethal. Viability and fertility of heterozygote excellent. Reliable balancer for all of chromosome 2, although there is an occasional double cross-over in 2R if X and 3 are heterozygous for inversions. MacIntyre and Wright (1966, DIS 41: 141-42) found no recombination between *In(2LR)SM1* and *al dp b pr cn c px sp* in females heterozygous for *In(1)sc⁸* and *In(3LR)Ubx¹³⁰*. Nondisjunction of compound autosomes occurs in *SM1/+* females [Lindsley and Grell, 1969, *Genetics* 61 (Supplement): 69-71]; balancer shows interchromosomal effect (Parry, 1973).

**SM2*

constitution: *T(2;3)SM2, al² Cy lt^v cn² sp²*.

synthesis: R.F. Grell, 1953.

references: Lewis and Mislove, 1953, DIS 27: 58. 1954, DIS 28: 77.

properties: Not useful as a balancer.

SM5

constitution: *In(2LR)SM5, al² Cy lt^v sn² sp²*. Series of overlapping inversions and transpositions distributed over length of chromosome (Sankaranarayanan, 1974, *Mutat. Res.* 24: 389-93).

order: 21A—21D2|36C-40F|29D—22D2|34A1—36C|21D3—22A3|60B—58B1|42A3—42D|42D—43A|58B1—58F|53C—42D|53C—58A4|42A2—40F|29E—33F5|22D1—22B1|60C—60F.

synthesis: R.F. Grell, 1953.

references: Mislove and Lewis, 1955, DIS 29: 75.

properties: Homozygous lethal. Heterozygote usually has

good viability and fertility, although may not be as good as *SM1*. Most complete balancer for chromosome 2.

SM6

constitution: *In(1)2LR)SM6, al² Cy dp^{lv} cn^{2P} sp²* combination containing both *In(2LR)O* and *In(2LR)SM1*.

order: 21—22A3|60B—58B1|42A3—50C10|30E—22D2|34A1—42A2|58A4—50D1|30F—33F5|22D1—22B1|60C—60F.

synthesis: Craymer.

references: 1984, DIS 60: 234-36.

properties: Suppresses recombination over entire chromosome 2; carries *cn^{2P}*, an amorphic derivative of *cn²*. Two versions: without *Roi* = *SM6a*; with *Roi* = *SM6b*.

THIRD-CHROMOSOME BALANCERS

C(3)x

constitution: *In(3L)P In(3R)P*. (Probably not the same as *Payne*).

LVM

constitution: *In(3L)P In(3R)P, l(3)LVML pe l(3)LVMR*.

properties: Homozygous lethal; suppresses crossing over in chromosome 3.

MKRS

constitution: *Tp(3;3)MKRS, M(3)76A, kar ry² Sb*.

order: 61—71B2|92E—93C|87F1—92E|71C—87E8|93C—100.

synthesis: Chovnick.

references: 1973, *Genetics* 76: 289-99.

Hilliker, Clark, Chovnick, and Gelbart, 1980, *Genetics* 95: 95-100.

properties: Homozygous lethal. Suppresses crossing over in the proximal regions of both arms of chromosome 3. Version lacking *kar* designated *MRS*.

TM1: *Third Multiple 1*

constitution: *In(3LR)TM1, Me ri sbd¹*.

order: 61—63|72E1—69E|91C—97D|89B—72E2|63C—69E|91C—89B|97D—100.

synthesis: Lewis.

references: 1949, DIS 23: 92.

Lewis and Mislove, 1953, DIS 27: 58.

Shearn, 1974, *Genetics* 77: 115-25.

properties: Homozygous lethal; suppresses crossing over in chromosome 3.

TM2

constitution: *In(3LR)Ubx¹³⁰, emc² Ubx¹³⁰ e^s*.

order: 61A|96A—93B|89D—74|61C—74|89E—93B|96A—100.

synthesis: Lewis.

references: 1952, *Proc Nat. Acad. Sci. USA* 38: 953-61.

1952, DIS 26: 66.

properties: Homozygous lethal. Eliminates crossing over in chromosome 3 except at the end of the right arm. Does not reliably balance mutations in the vicinity of *ca*. MacIntyre and Wright (1966, DIS 41: 141-42) observed about 9% double crossing over in the unbroken segment of the left arm from 61C to 74 on the polytene map and 15% recombination between the breakpoint at 96A and *ca* in *In(3LR)Ubx¹³⁰, Ubx¹³⁰ e/ru h th st cu sr e^s Pr ca* females that were also heterozygous for an X-

chromosome inversion behaving like *In(1)sc*⁸ and *In(2LR)SM1*; no recombination observed in other regions. Some versions also carry *p*^P.

TM3

constitution: *In(3LR)TM3, y⁺ ri p^P sep l(3)89Aa bx^{34e} e* [not *e*^S according to Craymer (1980, DIS 55: 197-200)]. Some *TM3* stocks have lost *y⁺* (Shearn, 1980, DIS 55: 167).

order: 1A1—1A8|61A2—65E|85E—79E|100C—100F2|92D1—85E|65E—71C|94D—93A|76C—71C|94F—100C|79E—76C|93A—92E1|100F3—100F5.

synthesis: Lewis, 55g.

references: Mitchell, 1958, Cold Spring Harbor Symp. Quant. Biol. 23: 279-90.

Lewis, 1960, DIS 34: 53-54.

Tinderholt, 1960, DIS 34: 53-54.

properties: Stocks exist in which *Ser* or *Sb* and *Ser* are present in the balancer, making the *TM3* chromosome homozygous lethal. With normal *X* and *2*, all of chromosome *3* is effectively balanced; however, in the presence of *FM6* and *SM5*, crossing over between *y⁺* and *ri* (*i.e.*, 61A2-65E) is appreciable. Double crossovers that separate *Sb* or *Ser* from the inversion complex are rare, even in the presence of *FM6* and *SM5*.

TM6

constitution: *In(3LR)TM6, Hn^P ss^{P88} bx^{34e} Ubx^{P15} e* [*Ubx^{P15}* listed as *Ubx^{67b}* in CP627 (Craymer, 1980, DIS 55: 197-200)].

order: 61A|89C2—75C|94A—100F2|92D1—89C4|61A2—63B8|72E1—63B11|72E2—75C|94A—92E1|100F3—100F5.

synthesis: Lewis and Bacher, 66i.

references: CP627.

properties: Homozygous lethal. Suppresses crossing over in chromosome *3*. Has unbroken regions with genetic lengths of approximately 10, 15, 20, and 30 units.

TM6B

constitution: *In(3LR)TM6B, Hu e*. Carries same inversions as *TM6* except for substitution of *In(3LR)HR33* for *In(3LR)P88* and addition of *In(3R)Hu*. Exists with several marker combinations, usually carrying *D³* or *Tb* with various combinations of *ca*, *h*, and *Hn^P*.

order: 61A1|87B2—86C6|84F1—86C5|84B6—84F1|84B3—75C|94A—100F2|92D1—87B4|61A2—63B8|72E1—63B11|72E2—75C|94A—92E1|100F3—100F5.

synthesis: Craymer.

references: 1984, DIS 60: 234-36.

properties: Probably most efficient balancer of chromosome *3*.

TM6C

constitution: *In(3LR)TM6C*. Carries same inversions as *TM6B* except that *In(3R)Hu* is absent. Exists with various marker combinations (including *cu* and *Sb*) in addi-

tion to those listed for *TM6B*.

order: 61A1|87B2—75C|94A—100F2|92D1—87B4|61A2—63B8|72E1—63B11|72E2—75C|94A—92E1|100F3—100F5.

synthesis: Craymer.

references: 1984, DIS 60: 234-36.

properties: Homozygous viable, although homozygotes show lower viability and longer development time than wild type. Resembles *TM6B* in balancing efficiency except that the absence of *In(3R)Hu* in *TM6C* lessens its effectiveness in the centromere region.

TM8

constitution: *In(3LR)TM8, l(3)DTS th st Sb e*.

origin: X ray induced in *In(3L)C90 In(3R)C, l(3)DTS4/+*.

synthesis: Marsh.

properties: Effective chromosome *3* balancer that carries dominant temperature-sensitive lethal; shows very few viable and fertile escapers from the DTS phenotype. 87C-92D-E not balanced (Duncan).

TM9

constitution: *In(3LR)TM9, l(3)DTS th st Sb e*.

origin: X ray induced in *In(3L)C90 In(3R)C, l(3)DTS4/+*.

synthesis: Marsh.

properties: Effective chromosome *3* balancer that carries dominant temperature-sensitive lethal; shows very few viable and fertile escapers from the DTS phenotype. 87A-92D-E not balanced (Duncan).

winscy: see *In(1)sc^{SIL} sc^{8R} + dl49*

TMS: Third Multiple of Singson

constitution: *In(3R)87A-B; 97F-98A* superimposed on *Tp(3;3)MKR5, M(3)76A kar ry² Sb P(ry⁺Δ2-3)99B*.

order: 61—71B2|92E—93C|87F—92E|71C—87A|97F—93C|87E—87B|98A—100.

synthesis: Singson.

properties: Useful in maintaining linkage between *Sb* and *A2-3* when passed through heterozygous females.

MULTI-CHROMOSOME BALANCERS

AM1: Autosomal Multiple 1

constitution: *T(2;3)A1, Cy L Ubx¹³⁰*. Reciprocal translocation between balancers *In(2L+2R)Cy* and *TM2*.

synonym: *A1*.

synthesis: Wallace, 1966.

references: Wallace, Zouras, and Krimbas, 1966, Am. Nat. 100: 245-51.

Wallace, 1966, Am. Nat. 100: 565-83.

Thompson, 1977, Genetics 85: 125-40.

1983, DIS 59: 129-30.

properties: Homozygous lethal. Suppresses crossing over in chromosomes *2* and *3*. Recombination on *2* increased in presence of *X* chromosome balancer, *In(1)sc^{SIL} sc^{8R} + dl49* (Thompson).

COMPOUND CHROMOSOMES

Compound chromosomes are monocentric elements in which the material from one chromosome arm is represented twice; they contain the entire diploid complement for the arm involved. They are designated by the symbol *C* followed parenthetically by the designation of the involved arm. Gametes of compound-bearing flies generally carry two or no doses of the chromosome arm. Compound-*X* chromosomes [*C*(1)'s] exist only in females which, unless special steps are taken, carry a *Y* chromosome. Such *C*(1)/*Y* females produce patroclinous sons that inherit the *X* from their father and the *Y* from their mother and matroclinous daughters that inherit two *X*'s from their mother and a *Y* from their father (so-called non-crisscross inheritance). Compound-autosome-bearing flies usually produce no viable progeny unless crossed to flies carrying compounds for the same arm or arms.

Some compounds have arisen repeatedly from certain genotypes; they were studied collectively but not as individual occurrences. In other cases, similar compounds of independent origin were studied individually. Both general classes of compounds and compounds of unique origin are listed.

The two chromosome arms comprising a compound may join (1) by attachment of the base of one to the terminus of the other to form an acrocentric chromosome or (2) by attachment of both proximally to a single centromere to form a metacentric; the ends of either an acrocentric or a metacentric may join to form a compound ring. In addition, the component arms may be in the same sequence or one may be entirely inverted with respect to the other. Thus, the elements of a compound may pair as a spiral (the tandem configuration) or as a hairpin (the reversed configuration). Simple compounds may therefore be classified according to the conventions of Novitski (1954, *Genetics* 39: 127-40) as reversed acrocentrics, reversed metacentrics, reversed rings, tandem acrocentrics, tandem metacentrics, and tandem rings; where applicable, this classification was retained and is used in the designation of compounds.

When the component arms differ in sequence by something other than whole-arm inversion, the classification tandem or reversed becomes ambiguous. Furthermore, when the component arms are separable from each other by a single break, the terms acrocentric and metacentric are descriptive; however, when elements of the two arms become interspersed (as for example by interarm rearrangements), these terms lose meaning. Consequently, the more-complex compounds are given arbitrary symbols.

The chromosomal constitution of compounds in which the chromosome arms remain intact is designated: metacentrics, by the sequences of the component arms separated by a centerpoint (which represents the centromere); acrocentrics, by the sequence of the distal arm separated by an em dash from the sequence of the proximal arm followed by a centerpoint; rings (which are derived from acrocentrics or metacentrics) by origin. In heterozygotes, the gene content of the component arms is listed according to the same conventions, with the genes on the first arm listed in the chromosomal designation followed by those on the second arm. In homozygotes,

the genes are listed in chromosome map order. Complete designation of a compound includes its symbol, its chromosomal constitution, and the gene content of its component arms; e.g., *C*(1)*TM2*, + - *In*(1)*sc*^{4*L*}*EN*^{*R*}, *y cv v · sd · y sn g*. It should be emphasized that the heterozygous gene content of compounds is often highly unstable owing to homozygosis and changes in coupling relations resulting from exchange.

In compounds in which elements of the component arms have become interspersed, it is usually not feasible to designate the chromosomal constitution in terms of the component arms; rather, it is described in terms of the order of chromosome segments as seen in salivary-gland chromosomes. In heterozygotes, the gene content is listed in such a way as to indicate which genes were originally in the different component arms.

· = : see *C*(1)*RM*

:= : see *C*(1)*DX*

2*L* : see *C*(2*L*)*RM*

2*R* : see *C*(2*R*)*RM*

3*L* : see 3(3*L*)*RM*

3*R* : see *C*(3*R*)*RM*

Attached 2*L* : see *C*(2*L*)*RM*

Attached 2*R* : see *C*(2*R*)*RM*

Attached 3*L* : see *C*(3*L*)*RM*

Attached 3*R* : see *C*(3*R*)*RM*

Attached-*X* : see *C*(1)*RM*

C*(1)94-2*A

constitution: Homozygous for *y*; originally heterozygous for *cv*, *sn*, *v*, *g*, and *sd*. Ring shaped in mitotic metaphase. Salivary chromosome analysis shows order to be |1A - 5E|1F - 1A · 20 - 5E|1F - 20|.

origin: Spontaneous (although possibly X ray induced premeiotically) derivative of *C*(1)*TR94-2*. Apparently arose through an asymmetrical or reversed exchange between the 1F region near the centromere and the 5E region near the interstitial heterochromatin of *C*(1)*TR94-2*.

synthesis: Rosenfeld, 1964.

properties: Crossing over in region 1F - 6A produces a single ring carrying *In*(1)94-2*A* = *In*(1)1F-2A;5E-6A. Reversibly convertible to other double-ring configurations by other types of exchange (e.g., Novitski and Braver, 1954, *Genetics* 39: 197-209).

***C*(1)*A*: Compound (1) of Armentrout**

constitution: Homozygous for *y* and probably originally heterozygous for *cv*, *sn*, *v*, *g*, and *sd*. Ring shaped in mitotic metaphase. Salivary chromosome analysis shows order to be |1A - 6F2 |6F2 - 1A|20 - 7A1|7A1 - 20 · |.

origin: Spontaneous stable derivative of *C*(1)*TR94*, which was originally *y cv v · sd · y sn g*. Apparently arose by a process describable as reversed crossing over in region 6F2 - 7A1. Current versions of this chromosome have apparently opened, since they are no longer ring-shaped in metaphase. Shown to have separated at 13E by Traverse and Pardue (1988, *Proc. Nat. Acad. Sci. USA* 85: 8116-20) such that the new order is 13E - 7A1 | 7A1

– 20·1 – 6F2| 6F2 – 1| 20 – 13E| 13E – 20·1 – 6F2| 6F2 – 1| 20 – 7A1| 7A1 – 13E which are interconvertible by exchange between regions 20 and 13. The newly terminal ends at 13E have acquired moderately repeated sequences (He-T DNA) ordinarily encountered at telomeres and in the chromocenter (Traverse and Pardue). Transmission of *C(1)A* is reduced owing to the fact that half of meiotic exchanges lead to the production of dicentric chromosomes.

synthesis: Armentrout, 1964.

properties: An apparently completely stable, compound-ring-X chromosome; cannot produce single-X chromosome derivative by heterochromatic exchange. Should be the best of all compound-X chromosomes for stock purposes.

C(1)DX: Compound (1) Double X

constitution: *C(1)DX, In(1)dl-49 – In(1)sc⁸, y f – y⁻ sc⁸ f[·].*

origin: X ray induced in *In(1)dl-49, y w flIn(1) sc⁸ sc⁸ B* female [stated by Muller to have been *In(1)dl-49/In(1)sc⁸ y^{3PR}*, but the derivative does not carry *y^{3PR}*]. Was originally *y w f – y⁻ sc⁸ B[·]*, but by double exchange *f* became homozygous and *B* was lost.

synthesis: Muller.

synonym: The symbol :=.

references: 1943, DIS 17: 61-62.

Valencia, Muller, and Valencia, 1949, DIS 23: 99-102.

properties: A reversed acrocentric heterozygous for *In(1)dl-49*; it is useful in balancing because it is very stable, which is probably due to little interstitial heterochromatin. *y w f* detachments very rarely produced. Also produces a low incidence of homozygosis for *w*, and *y w f* versions exist. *C(1)DX/0* lethal; deficient for *bb*.

****C(1)M2: Compound (1) Multiple***

constitution: *C(1)M2, In(1)sc⁷ + AM – In(1)FM4[·], sc⁷ – y⁻ sc⁸ dm B[·].*

origin: X-ray-induced exchange between the proximal heterochromatin of *In(1)sc⁷ + AM* and the distal heterochromatin of *In(1)FM4*.

synthesis: Lewis, 54h.

synonym: *FMA2: First Multiple Attached*.

references: 1958, DIS 32: 81.

C(1)M3

constitution: *C(1)M3, In(1)AM – In(1)FM4[·], y² – y⁻ sc⁸ dm B[·].*

origin: Recombinant between *In(1)sc⁷ In(1)AM* element of *C(1)M2* and *In(1)AM, y²* in triploid.

synthesis: Lewis, 55b.

synonym: *FMA3*.

references: 1958, DIS 32: 81-82.

properties: Detachment rare; useful in balancing.

C(1)M4

constitution: *C(1)M4, In(1)w^{m4} + AB – FM7[·], y w^{m4} – y⁻ w^a v^{Of} sc⁸.*

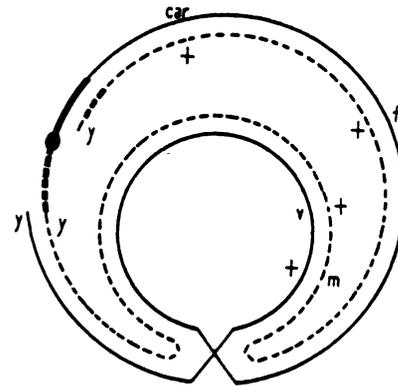
origin: X-ray-induced exchange between the proximal heterochromatin of *In(1)w^{m4} + AB* and the distal heterochromatin of *FM7*.

synthesis: Craymer, 72e.

references: 1974, DIS 51: 21.

properties: Because of the *w^{m4}/w^a* constitution, *C(1)M4* females without a *Y* chromosome display strong variegation, those with *YS* moderate variegation, and those with

YL or a complete *Y* almost no variegation. Useful in maintenance of *XY*-bearing stocks without a free *Y*. Detachment rate approximately 1/15,000. A powerful enhancer of autosomal recombination, but has low viability in combination with autosomal rearrangements. Some derivatives are *y² bb⁻*.



C(1)NB: Compound (1) of Novitski and Braver

From Novitski and Braver, 1954, Genetics 39: 197-209.

C(1)NB: Compound (1) of Novitski and Braver

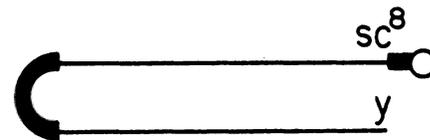
constitution: *C(1)NB, In(1)dl-49 · In(1)sc^{4L} EN^R*; originally *y v f car · y m; In(1)dl-49* and *In(1)EN* attached proximally to a single centromere.

origin: Crossover between the heterochromatic short arm of *In(1)EN* and the proximal heterochromatin of *In(1)dl-49*.

synthesis: Novitski and Braver.

references: 1954, Genetics 39: 197-209 (fig.).

properties: Essentially a tandem metacentric heterozygous for *In(1)dl-49*. Can exist in a number of different configurations interconvertible by crossing over. Generates single rings at different frequencies, depending on configuration of the compound.



C(1)RA: Compound (1) Reversed Acrocentric

Redrawn from Sandler, 1954, Genetics 39: 923-42.

C(1)RA: Compound (1) Reversed Acrocentric

constitution: *C(1)RA, + – In(1)sc⁸.*

origin: Spontaneous from *X · YL/In(1)sc⁸* either by exchange between the proximal heterochromatin of *X · YL* and the distal heterochromatin of *In(1)sc⁸*, or possibly by sister-strand union in one of the heterochromatic segments followed by a normal euchromatic exchange. A frequently recurring event that seems to require the presence of *YL*. More recent attempts to repeat such constructions have been unsuccessful, except in response to x irradiation.

synthesis: Novitski.

synonym: *RA*.

references: Novitski, 1954, *Genetics* 39: 127-40.

Sandler, 1954, *Genetics* 39: 923-42.

1958, Cold Spring Harbor Symp. Quant. Biol. 23: 211-23.

Sandler and O'Tousa, 1979, *Genetics* 91: 537-51.

properties: Yields frequent detachments resulting from exchange between the *Y* chromosome and the interstitial heterochromatin of the reversed acrocentric and preferential recovery of the proximal *X*. Tetrad distribution usually quite abnormal; one-exchange tetrads infrequent and no- and two-exchange tetrads frequent. Exchange frequency increased by addition of *Y* or *y*⁺*YL*, but tetrad distribution remains abnormal (Sandler, 1954). *YL* appended as a second arm to *C(1)RA* normalizes tetrad distribution (Sandler, 1958). Tetrad distribution is normal in more recently recovered *C(1)RA* chromosomes (Sandler and O'Tousa), reason for differences between 1954 and 1979 data is unclear. The presence of a *Y* chromosome or a free-*X* duplication as a homologue markedly increases both exchange between the elements of the compound and fecundity of compound-bearing females.

C(1)RA60g

constitution: *C(1)RA60g*, + - *In(1)sc*⁸.

origin: A spontaneous euchromatic event (perhaps sister chromatid union) in a triploid female heterozygous for *In(1)sc*⁸ + *dl49*.

synthesis: Mohler, 60g.

references: 1960, *DIS* 34: 52.

Gethmann, 1979, *Genetics* 55: 673-79.

properties: Deficient for interstitial heterochromatin and proximal euchromatin, including *su(f)*. Requires a duplication carrying both *su(f)*⁺ and *bb*⁺ in order to survive. Exhibits standard distribution of tetrads in meiosis (Gethmann).

other information: The reciprocal exchange product, *Dp(1:f)60g*, recovered from same fly.

C(1)RA85

constitution: *C(1)RA85*, *y w*¹¹¹⁸ *f*⁵ - *In(1)sc*^{SIL} *sc*^{8R} + *S*, *y*⁻ *sc*⁸ *w*^r *f*⁵.

origin: Spontaneous exchange between the proximal heterochromatin of *y w*¹¹¹⁸ *f*⁵ and the distally inverted heterochromatin of *Basc*, with subsequent loss of *B* and homozygosis of *f*⁵.

references: Mount, Green, and Rubin, 1988, *Genetics* 118: 221-34.

properties: A stable compound *X* chromosome.

C(1)RM: Compound (1) Reversed Metacentric

constitution: *C(1)RM*, + · + ·; two *X* chromosomes in normal sequence attached proximally to the same centromere; exists with many combinations of markers.

origin: Spontaneous. Recurs regularly by exchange between heterochromatin of the short arm of one *X*, *XYS*, or *XYL* and that of the base of the long arm of a sister or homolog. Can be induced in mature *X*·*YL*-bearing sperm (Leigh, 1972, *DIS* 48: 107). Presumably the pericentric heterochromatic constitutions of independently arising *C(1)RM* chromosomes varies.

discoverer: L. V. Morgan, 21b12.

synonym: *Attached-X*; also the symbol · =.

references: 1922, *Biol. Bull.* 42: 267-74.

1938, *Am. Naturalist* 72: 434-46.

properties: Recombination with the *Y* chromosome leads to detachments with a frequency of about 10⁻³ in *C(1)RM/Y* females. Has been extensively used in studies of crossing over (e.g., Anderson, 1925, *Genetics* 10: 403-17; Beadle and Emerson, 1935, *Genetics* 20: 192-206; Welshons, 1955, *Genetics* 40: 918-36).

****C(1)RR1: Compound (1) Reversed Ring***

constitution: *C(1)RR1*, + - *In(1)EN*, *y*⁻ *sc*⁻ - *y*; two *X* chromosomes attached by their normally distal ends to a common centromere and by their normally proximal ends to each other. Marked with *y*.

origin: Spontaneous derivative of *C(1)TR1*.

synthesis: Zimmering.

synonym: *RR*.

references: Novitski, 1954, *Genetics* 39: 127-40.

****C(1)RR2***

constitution: *C(1)RR2*, *In(1)sc*⁸ · *In(1)sc*^{SIL} *EN*^R; originally *y*⁻ *cv v f* · *y m car*. *In(1)sc*⁸ and *In(1)sc*^{SIL} *EN*^R attached proximally to a single centromere and distally at their distal heterochromatic segments.

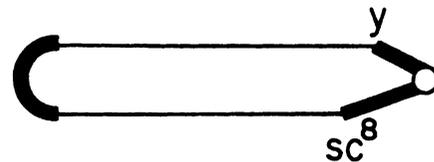
origin: *X* ray induced in an attached-*X* with *In(1)sc*⁸ and *In(1)sc*^{SIL} *EN*^R attached proximally to a single centromere. Recovered as simultaneous loss of *y*⁺ from the tip of both arms.

synthesis: Sandler.

references: 1957, *Genetics* 42: 764-82 (fig.).

1958, Cold Spring Harbor Symp. Quant. Biol. 23: 211-23.

properties: Tetrad distribution abnormal; one-exchange tetrads are infrequent and no- and two-exchange tetrads are frequent. Exchange frequency increased by addition of *Y* or *y*⁺*YL*, but tetrad distribution remains abnormal.



C(1)RR2: Compound (1) Reversed Ring 2

From Sandler, 1957, *Genetics* 42: 764-82.

C(1)RR94-2F

constitution: *C(1)RR94-2F*, + · + ·; two *X* chromosomes of normal sequence attached proximally to a single centromere and joined distally by a segment of heterochromatin.

origin: *X*-ray-induced derivative of *C(1)TR94*.

synthesis: Rosenfeld, 1964.

references: Sandler, 1965, *Nat. Cancer Inst. Monograph* No. 18: 243-72.

properties: Tetrad distribution more nearly normal than in *C(1)RR2*.

C(1)SB: Compound (1) of Sturtevant and Beadle

constitution: *C(1)SB*, + · *In(1)y*⁴; *In(1)y*⁴ and a normal sequence attached proximally to a single centromere.

origin: Recombinant between the uninverted portion of

*In(1)y*⁴ and *C(1)RM* in a triploid.

synthesis: Sturtevant and Beadle.

references: 1936, Genetics 21: 554-604.

Novitski and Sandler, 1956, Genetics 41: 194-206.

properties: A reversed metacentric heterozygous for *In(1)y*⁴. Meiotic behavior similar to that of a tandem metacentric. Crossing over within inversion generates single ring, *R(1)y*⁴.

**C(1)TA1: Compound (1) Tandem Acrocentric*

constitution: *C(1)TA1, In(1)sc*⁴ - *In(1)EN·YL, y sc*⁴ - *y*.

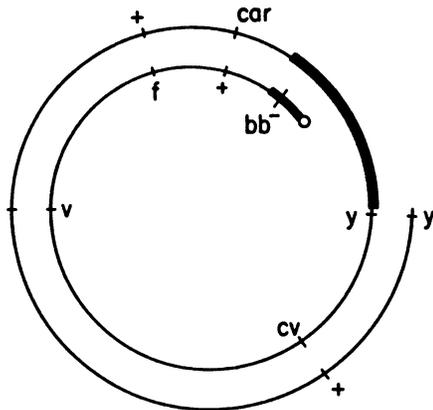
origin: X-ray-induced exchange between the proximal heterochromatin of *In(1)sc*⁴ and *YS* of *YSX·YL*.

synthesis: Novitski.

synonym: *TA*.

references: 1954, Genetics 39: 127-40.

properties: Produces a single, centric, rod-X chromosome and either an acentric, ring-X or a tandem triple-X chromosome by recombination between the proximal and distal X chromosomes.



C(1)TA2: Compound (1) Tandem Acrocentric 2

From Sandler and Lindsley, 1963, Genetics 48: 1533-43.

C(1)TA2

constitution: *C(1)TA2, + - + ·*; originally *y cv f - y f*.

origin: X-ray-induced recombinant in *YSX, y*⁺ *K*^S *y cv v f/XYL, y car K*^L female; origin required triple exchange.

synthesis: Sandler and Lindsley.

references: 1963, Genetics 48: 1533-43 (fig.).

properties: Generates single X chromosomes like *C(1)TA1*. Tetrad distribution about normal. *C(1)TA2/0* lethal, probably deficient for *bb*.

C(1)TA·YL

constitution: *C(1)TA·YL, + - + ·, y - y·KL*; original line segregating for *cv, v, f, and car*.

origin: Spontaneous in a *X·YS, In(1)sc*^{8L} *EN*^R, *y*⁺ *y/X·YL, y cv v f car*; thought to result from an euchromatic exchange between the *X·YL* chromosome and the acentric ring formed by dyscentric exchange between the distal and proximal heterochromatin of the long arm of *X·YS, In(1)sc*^{8L} *EN*^R.

references: Merriam, 1968, Genetics 59: 351-66.

C(1)TA·sc^{V1}

constitution: *C(1)TA·sc*^{V1}, *+ - + ·, y - y·sc*^{V1} *y*⁺.

origin: Derived as a recombinant in the *B - car* region between *C(1)TA·YL* and the *X*^{P4D} element of *T(1;4)B*^{SL} *In(1LR)sc*^{VIR}, *B*^S *car·sc*^{V1} *y*⁺.

references: Merriam, 1968, Genetics 59: 351-66.

C(1)TM1: Compound (1) Tandem Metacentric

constitution: *C(1)TM1, + ·In(1)sc*^{8L} *EN*^R, *y Hw f·y*⁺ *y f*; a normal sequence and *In(1)EN* attached proximally to a single centromere derived from *R(1)2*.

origin: Product of one crossover between *+* and *R(1)2* and one between *In(1)EN* and *R(1)2* in a *+/R(1)2/In(1)EN* triploid.

synthesis: Novitski, 1950.

references: Novitski and Lindsley, 1950, DIS 24: 90-91.

properties: Single crossover between the arms produces single-ring-X chromosome with the same structure as *R(1)2* and an acentric-rod X chromosome. Tetrad distribution about normal (Novitski, 1951, Genetics 36: 267-80; Novitski and Sandler, 1956, Genetics 41: 194-206).

C(1)TM2

constitution: *C(1)TM2, + ·In(1)sc*^{4L} *EN*^R; originally *y cv v sd·y sn g*. The sequence in mitotic prophase is: the normal X euchromatin, two large heterochromatic segments, a small segment, the centromere, a small segment, the inverted X euchromatin.

origin: X-ray-induced exchange between the proximal heterochromatin of a normal X and *YL* of *X·YL, In(1)sc*^{4L} *EN*^R.

synthesis: Lindsley and Sandler, 1963.

synonym: *TMX y*.

references: 1965, Genetics 51: 223-45 (fig.).

properties: Recombination between the arms produces a single-ring-X chromosome and an acentric, rod-X chromosome. Meiotic behavior similar to that of *C(1)TM1*; tetrad distribution about normal.

C(1)TM5

constitution: *C(1)TM5, YSIn(1)EN·+*; originally *y w v B KS·y*.

origin: X ray induced in *YSX·YL, In(1)EN, y w v f B/XYL·YS, y·y*⁺ females.

synthesis: Lucchesi, Mills, and Rosenbleeth.

references: 1965, DIS 40: 57-58.

Pasztor, 1967, DIS 42: 107.

Pasztor, 1971, Genetics 68: 245-58.

properties: Single exchange generates highly unstable single ring chromosomes as seen by very low recovery of ring-bearing daughters and by 16-46% gynandromorphs among single-ring-bearing progeny. Single rings apparently deficient for proximal euchromatic material, as they are male-lethal in combination with a normal Y but survive in combination with *B*^S *Y* or *su(f)*⁺ *Y*.

C(1)TM-H: Compound (1) Tandem Metacentric of Hinton

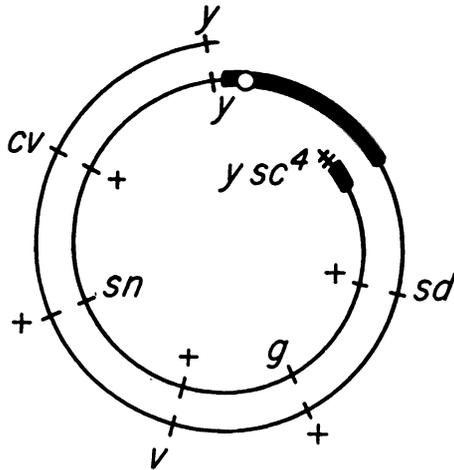
constitution: *C(1)TM-H, In(1)sc*^{4L} *R(1)2*^R [*·In(1)w*^{VC}] + *dl49*; position of centromere indeterminate.

origin: Generated by exchange between the *B*^S duplication of *Dp(1;1)B*^S *-H* and *In(1)dl49*.

references: Hinton, 1957, Genetics 42: 55-65.

properties: These are linear derivatives of the unstable *R(1)2, In(1)w*^{VC}. They exhibit variable stability as indicated by (a) their reduced recovery among the progeny

C(1)TM-H-bearing mothers, (b) the production of *XO* patroclinous sons, and (c) the instability of single rings produced by single exchange between the arms of the tandem metacentric.



C(1)TM2: Compound (1) Tandem Metacentric 2
From Lindsley and Sandler, 1965, *Genetics* 51: 223-45.

C(1)TMB^S9-1: Compound (1) Tandem Metacentric with Bar Stone

constitution: *C(1)TMB^S9-1, Dp(1;1)B^STAG·In(1)sc^{8L}EN^R*; originally *B^Sy cv v sd·y sn g*. The sequence in mitotic prophase is: the normal *X* euchromatin, two large heterochromatic segments, a small segment, the centromere, a small segment, the inverted *X* euchromatin.

origin: X-ray-induced exchange between the proximal heterochromatin of *Dp(1;1)B^STAG* and *YL* of *X·YL, In(1)sc^{8L}EN^R*.

synthesis: Lindsley and Sandler, 1963.

synonym: *TMXB^S9-1*; also designated as *Dp(1;1)B^STRG*.

references: 1965, *Genetics* 51: 223-45.

properties: Recombination between the arms produces a single-ring-*X* chromosome, *R(1)9-1*, and an acentric, rod-*X* chromosome. Recombination between the *B^S* duplication and the homologous region of the inverted arm generates a nontransmissible tandem-ring chromosome. Meiotic behavior similar to that of *C(1)TM2*.

C(1)TMB^S9-4

constitution: *C(1)TMB^S9-4, Dp(1;1)B^STAG·In(1)sc^{8L}EN^R*; originally *B^Sy cv v sd·y sn g*. The sequence in mitotic prophase is: the normal *X* euchromatin, a large heterochromatic segment, a small segment, the centromere, a small segment, the inverted *X* euchromatin.

origin: X-ray-induced exchange between the proximal heterochromatin of *Dp(1;1)B^STAG* and *YL* of *X·YL, In(1)sc^{8L}EN^R*.

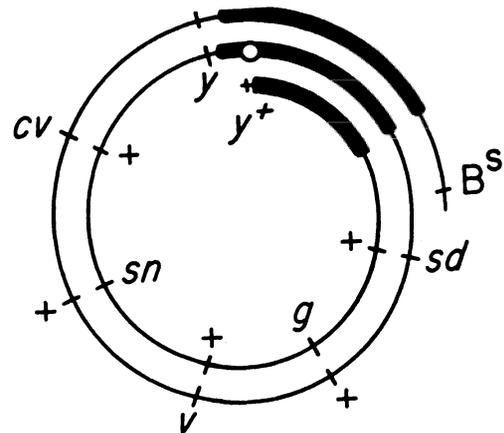
synthesis: Lindsley and Sandler, 1963.

synonym: *TMXB^S9-4*; also designated as *Dp(1;1)B^STRG*.

references: 1965, *Genetics* 51: 223-45 (fig.).

properties: Recombination between arms produces single-ring-*X* chromosome, *R(1)9-4*, and an acentric, rod-*X* chromosome. Recombination between the *B^S* duplication and the homologous region of the inverted

arm produces a tandem-ring chromosome that may be transmissible.



C(1)TMB^S: Compound (1) Tandem Metacentric with Bar-Stone

From Lindsley and Sandler, 1965, *Genetics* 51: 223-45.

****C(1)TR1: Compound (1) Tandem Ring***

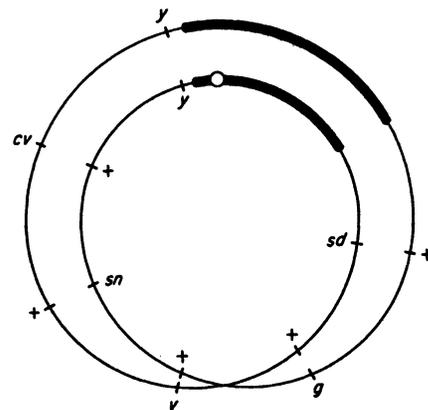
constitution: *C(1)TR1, In(1)sc⁴-In(1)EN·, y⁻sc⁻-y·.*

origin: Spontaneous derivative of *C(1)TA1* in which the *YL* second arm had been replaced by the *X^{P4D}* element of *T(1;4)B^S = T(1;4)15F9-16A1;16A7-B1;102F*. A product of recombination between the duplicated *B^S* second arm and the homologous region of the distal element of the tandem acrocentric.

synthesis: Novitski.

references: 1954, *Genetics* 39: 127-40.

properties: Seems to be poorly transmissible (Novitski, 1954). Produces a centric, single-ring-*X* and either an acentric, single-ring-*X* or a tandem, triple-ring-*X* chromosome by recombination between the two elements of the compound.



C(1)TR94: Compound (1) Tandem Ring 94

From Sandler and Lindsley, 1967, *Genetics* 55: 645-71.

C(1)TR94

constitution: $C(1)TR94, + \cdot In(1)sc^{4L}EN^R \cdot$; originally y
 $cv \ v \ sd \cdot y \ sn \ g$.

origin: Regular but infrequent product of $C(1)TMB^{S9-4}$.
Formed by exchange between the duplicated B^S section
and the homologous region of the inverted arm.

synthesis: Sandler and Lindsley.

references: 1967, Genetics 55: 645-71.

properties: Produces a centric, single-ring- X and either an
acentric, single-ring- X or a tandem, triple-ring- X chromo-
some by crossing over between the two arms of the com-
pound. Transmission higher than that of $C(1)TR1$.
Tetrad distribution about normal. Exhibits $< 0.20\%$
dicentric quadruple rings in mitotic metaphases (Gatti,
Santini, Pimpinelli, and Olivieri, 1979, Genetics
91: 255-74).

***C(1)VM: Compound (1) of Valencia
and Muller**

constitution: $C(1)VM, + - In(1)sc^{S1} In(1)dl-49 \cdot$; origi-
nally $y \ ac \ sc \ pn \ w \ rb \ cm \ ct^6 \ sn^3 \ oc \ ras^2 \ v \ dy \ g \ f \ car - y$
 $sc^{S1} \ lz^s \ B \cdot$.

origin: X ray induced in $+In(1)sc^{S1} In(1)dl-49/YL$ female,
either by exchange between the proximal heterochroma-
tin of the normal sequence and the distal heterochromatin
of $In(1)sc^{S1}$ or by sister-strand union in one of the
heterochromatic elements accompanied by normal
euchromatic exchange. A regularly induced product in
such females.

synthesis: Valencia, Muller, and Valencia.

references: 1949, DIS 23: 99-102.

properties: Essentially a reversed acrocentric in which the
proximal element contains $In(1)dl-49$. Detachment by
crossing over with a Y chromosome relatively frequent.

C(2)EN: Compound (2) ENTire

constitution: $C(2)EN, 2R2L - 2L2R$.

origin: Synthesized by first selecting a $T(Y;2)$ with a break
in YL of $B^S Yy^+$ and an absolutely terminal break in $2L$,
and next transferring the terminal YL to $C(2L)RM$ by
recombination in a $Dp(Y;2)/C(2L)RM/C(2R)RM$ triploid;
the terminal YL was homozygosed to produce
 $C(2LYL)RM, B^S$. Irradiated $C(2LYL)RM, B^S/C(2R)$
females were crossed to $C(2L); F(2R)/F(2R)$ males; this
cross selects for progeny that receive $C(2LYL)RM, B^S$
plus a single copy of $2R$ from their mothers; those with
wider Bar eyes are putative results of a translocation
between the base of $2R$ from $C(2R)RM$ and a terminal YL
of $C(2LYL)RM$ and the surviving offspring are
 $YL2L \cdot 2L2R/F(2R)$; crosses of females of this constitu-
tion to $C(2L)RM; C(2R)RM$ have yielded $C(2)EN$ -bearing
progeny as a consequence of fertilization of an ovum that
has received a derivative homozygous (non B^S) for the
 $2L2R$ arm of the compound and no $F(2R)$ by a nullo-2
sperm. *In situ* hybridization with a telomeric probe
reveals the presence of telomeric sequences at the junc-
tion between $2L$ and $2R$ (Goldstein, Berry, and Novitski,
1984, DIS 60: 117).

references: Novitski, 1977, Genetics and Biology of Dro-
sophila (Ashburner and Novitski, eds.). Academic Press,
London, New York, San Francisco, Vol. 1b, pp. 562-68.
Novitski, Grace and Strommen, 1981, Genetics 98: 257-
73.

properties: Provides all of chromosome 2 necessary for
normal development. $C(2)EN$ -bearing flies produce two

types of meiotic products with respect to chromosome 2;
half disomic and half nullosomic. Accordingly crosses to
normal diploids produce mainly inviable mono- and
triplo-2 zygotes; however crosses of $C(2)EN$ flies to each
other produce progeny. Transmission of $C(2)EN$ by
males versus that from females varies from about 30% to
very low values (see also Robbins, 1977, Genetics
87: 67-81). This is attributed to zygote mortality by
Novitski *et al.*; however, it may be reflected in defects in
spermatogenesis as seen in cross sections of bundles of
elongated sperm tails. Male transmission ratio is sensi-
tive to the particular Y chromosome present (Strommen,
1982, Mol. Gen. Genet. 187: 126-31). Sex-chromosome
disjunction in both sexes influenced by $C(2)EN$ (Falk,
1982, Genet. Res. 41: 17-28); in XY/O males the XY
segregates preferentially from the compound; in X/Y
males and X/X females, sex chromosome nondisjunction
is elevated with the sex chromosomes segregating pre-
ferentially away from $C(2)EN$. In general, $C(2)EN$ -bear-
ing flies perform poorly in stocks and crosses.

C(2;3)EN: Compound (2;3) ENTire

constitution: $C(2;3)EN, 2R2L \cdot 3L3R$.

origin: Induced by irradiation of $C(2)EN; 3R3L \cdot YSy^+$
females. Recovered as a translocation involving the cen-
tric heterochromatin of both elements resulting in the
replacement of one arm of $C(2)EN$ with $3R3L$.

references: Novitski, Grace and Strommen, 1981, Genetics
98: 257-73.

properties: Carries a complete haploid set of the large
autosomes attached to a single centromere. Transmission
by heterozygous females is reduced by nonrandom dis-
junction of heteromorphic dyads resulting from exchange
between the compound and a normal homologue, but it is
high if recombination is suppressed by homologous
inversions. Transmission by heterozygous males is low.
Homozygous viability and fertility not indicated.

C(3)EN: Compound (3) ENTire

constitution: $C(3)EN, 3R3L \cdot 3L3R$.

origin: Synthesized by first selecting a $T(Y;3)$ with a break
in YL of $B^S Yy^+$ and an absolutely terminal break in $3L$.
Females heterozygous for $B^S YL3L \cdot 3R$ and a normal
third chromosome were irradiated and crossed to
 $F(3L)/F(3L)/C(3R)RM$ males; one of the few surviving
products of this cross is non-Bar and comes from ferti-
lization of an ovum containing a half translocation
between the base of $3R$ of the normal third and the distal
 YL material of the $Dp(Y;3)$ fertilized by a sperm contain-
ing $F(3L)$ but no $C(3R)$; the $F(3L)$ in this case was the
 $Y^P 3L^D$ of a $T(Y;3)$ involving breaks in YL of $B^S Yy^+$
and the proximal heterochromatin of $3L$, so that the desired
survivor is $3R \cdot 3L3R/y^+ YS \cdot 3L$ in constitution. One pro-
duct of recombination between $3L$'s in females of the
above constitution is $y^+ YS \cdot 3L3R$. Females homozygous
for the latter derivative were irradiated and crossed to
 $C(3L)RM; C(3R)RM$ males; surviving y offspring carried
 $3R3L \cdot 3L3R = C(3)EN$ from the mother and no third
chromosomal elements from the father. *In situ* hybridiza-
tion with telomeric sequences provides no evidence for
the presence of such sequences at the $3L - 3R$ junction
(Goldstein, Barry and Novitski, 1984 DIS 60: 117).

references: Novitski, Grace and Strommen, 1981, Genetics
98: 257-73.

properties: Provides all of chromosome 3 necessary for

normal development. *C(3)EN*-bearing flies produce two types of meiotic products with respect to chromosome 3: half disomic and half nullosomic; accordingly crosses to normal diploids produce only inviable mono- and triplo-3 zygotes; however crosses of *C(3)EN* flies to each other produce progeny. Transmission of *C(3)EN* by males versus that from females varies widely depending on the particular isolations tested, from 40% relative maternal transmission in some crosses through equal recovery in others, to 30% relative paternal transmission in yet others. In general these chromosomes perform poorly in stocks and crosses.

C(4)RM

constitution: 4.4; exists with various marker combinations. Two right arms of chromosome 4 attached proximally to a single centromere.

origin: X ray induced.

synonym: *E. B. Lewis*.

properties: Produces haplo-4 and triplo-4 progeny in crosses to normal diplo-4 flies. Tetra-4 flies carrying two copies of *C(4)RM* exhibit but slightly reduced viability (Grell, 1972, DIS 48: 69). Segregates quasi regularly from *C(1)RM* in *C(1)RM/0*; *C(4)RM/0* females (Grell, 1963, Genetics 48: 1217-29).

C(A): Compound (Autosomal arm)

Autosomal compound chromosomes may be subdivided into two classes, homocompounds, consisting of two copies of the same autosomal arm attached to a common centromere, and heterocompounds in which two arms from different autosomes are connected through the centromere of one of them. These are discussed separately in the following section.

Homocompounds: Compound chromosomes containing two copies of an autosomal arm [*C(A)*] have been constructed for each autosomal arm, as reversed metacentrics in every case so far. The compound for a particular autosomal arm may be kept in stock either with the compound for the other arm of the same autosome [e.g., *C(AL)*; *C(AR)*] or with free arms [e.g., *C(AL)*; *F(AR)/F(AR)*]. Most compound autosome stocks are of the former constitution; outcrosses to wild type are virtually sterile, the rare survivors being triploids, triploid intersexes, and triploid metafemales (*4X3A*) thought to arise from unreduced ova (Gethmann, 1972, DIS 49: 62) or products of tetraploid oocytes (Kuznetsova and Glotoff, 1978, Genetika 14: 463-69; Kuznetsova and Balakireva, 1980, Genetika 16: 1498-1500; Kuznetsova, 1983, Genetika 19: 775-78); in addition some diploid survivors contain reconstituted *2L·2R* chromosomes. Stocks of the second type are similarly unproductive when outcrossed to wild type; rare survivors carry detachments of the *C(A)* resulting from exchange between the base of the compound autosome and either the *Y* or the *X* chromosome (Chadov, 1975, Genetika 11(1): 80-90; 91-100; 1976, Genetika 12(3): 67-77; Chadov and Chadova, 1977, Genetika 13: 477-89; Chadov, Chadova and Khostina, 1983, Genetika 19: 1111-20). Once the first *C(2L)*; *C(2R)* and *C(3L)*; *C(3R)* strains were constructed (Rasmussen, 1960, DIS 34: 53), it became possible to generate additional compound chromosomes, either spontaneously or by irradiation of normal chromosome complements and by crossing to a compound-autosome-containing line to recover them in

combination with a compound for the complementary arm. Compound autosomal arms result from interchanges involving breaks on opposite sides of the centromere of two chromosomes, either homologues or sisters. The resultant chromosomes have an intact autosomal arm on one side of the centromere and a proximal segment of the other arm on the other side to which is attached the distal majority of the first arm from the sister chromatid or the homologous chromosome. Thus compound arms carry two doses of the majority of the arm but only one dose of a proximal segment of variable length, plus a proximal segment of the opposite arm. New compounds may be induced by irradiation of oocytes (Bateman, 1968, Effects of Irradiation on Meiotic Systems, International Atomic Energy Agency, Vienna, pp. 63-70) and in spermatocytes and spermatids (Leigh and Sobels, 1970, Mutat. Res. 10: 475-87). Compound autosomes with desired marker constitutions may be achieved by recombination in triploids or by inducing them anew in appropriately marked diploids; since the latter is the preferred method, most different marker combinations represent independent occurrences, and are so considered in the following table. Reconstitution of *AL·AR* from *C(AL)*; *C(AR)*, either induced or spontaneous, generates chromosomes that may be duplicated or deficient for regions adjacent to the centromere; these have been used to investigate the genetic content of pericentric regions (Baldwin and Suzuki, 1971, Mutat. Res. 11: 203-13; Hilliker and Holm, 1975, Genetics 81: 705-21). In otherwise normal genotypes *C(AL)* and *C(AR)* segregate from one another regularly in females (Grell, 1970, Genetics 65: 65-74; Holm and Chovnick, 1975, Genetics 81: 293-311); in males they segregate at random with respect to one another (Hilliker, 1981, DIS 56: 61-62; Holm and Chovnick, 1975), generating a trimodal distribution of mature sperm-head volumes; chromosome loss is indicated by the presence of micronuclei in spermatids (Hardy, 1975, Genetics 79: 231-64). Regular segregation in females can be upset by the addition of heterologous inversions or by the addition of an extra *Y* chromosome or an attached *X* (Grell, 1970), compounds that carry only heterochromatic material from the opposite arm segregate randomly in males, whereas those that contain euchromatic material from the opposite tend to segregate from the complementary compound. The table below lists a selection of compound autosomal arms that have been characterized or referenced in some way.

compound ^α	ref ^β	comments
<i>C(2L)CN10</i>	14	complex contains <i>Y</i> fertility genes and <i>bw</i> ⁺ and <i>sp</i> ⁺ from 2R = <i>C(2L)#4</i> ?
<i>C(2L)RM-P, dp</i>	5	
<i>C(2L)RM, b pr</i>	13	
<i>C(2L)RM, j</i>	13	
<i>C(2L)RM-P, b</i>	13	
<i>C(2L)RM-SD72</i>	9, 10	21 — 40F·41A — 42A10 39D3 — 21; induced in <i>In(2LR)SD72</i> ; → 80% segregation from <i>C(2R)RM-SD72</i> in males
<i>C(2L)RM-SH1</i>	7, 10	carries <i>rl</i> ⁺ from 2R; segregates randomly in males
<i>C(2L)RM-SH3, +</i>	7, 10	
<i>C(2L)RM-VH1, lt</i>	7	carries <i>rl</i> ⁺ from 2R; segregates randomly in males
<i>C(2L)RM-VH2, lt</i>		
<i>C(2L)RM-VT1, ho</i>		
<i>C(2R)RM-P, px</i>	3, 5, 13	not duplicated for <i>lt</i> , same as <i>C(2L)RM#4</i> ?

compound α	ref β	comments
<i>C(2R)RM, +</i>		
<i>C(2R)RM, bw</i>		
<i>C(2R)RM, bw</i>	3	
<i>C(2R)RM, cn</i>	3, 13, 15	60F — 41A · 40F — 39 41A — 60F; segregates regularly from <i>C(2L)</i> in males
<i>C(2R)RM, stw bw</i>	13	
<i>C(2R)RM, vg</i>	13	
<i>C(2R)RM-P, px</i>	3	Same as <i>C(2R)RM#4?</i>
<i>C(2R)RM-SD72</i>	9, 10	60F — 42A14 39D4 — 40F · 41A — 60F; induced in <i>In(2LR)SD72</i> ; → 80% segregation from <i>C(2L)RM-SD72</i> in males = <i>C(2R)RM-V43</i>
<i>C(2R)RM-SH1, +</i>	7, 10	
<i>C(2R)RM-SH3, +</i>	7, 10	
<i>C(2R)RM-VHK1, rl cn</i>	8	
<i>C(2R)RM-VK1, bw</i>	14	
<i>C(2R)RM-VK2, bw</i>	7	carries <i>lt</i> ⁺ from 2R; segregates randomly in males
<i>C(3L)RM-P2, ri</i>	11	
<i>C(3L)RM-P5</i>	12	originally <i>In(3L)P ve h th</i>
<i>C(3L)RM, h²</i>	5, 13	
<i>C(3L)RM, se h rs²</i>	5	
<i>C(3L)RM-SH2, +</i>	12	
<i>C(3L)RM-SH3, +</i>	12	
<i>C(3L)RM-VG1, ru st</i>	4	
<i>C(3L)RM-VH3, st</i>		
<i>C(3R)RM, +</i>	5	
<i>C(3R)RM, sbd² gl e²</i>	5, 13	
<i>C(3R)RM-P2, sr</i>	2	
<i>C(3R)RM-P5, sbd² gl e^s</i>	12	
<i>C(3R)RM-SB1, p^P gl³</i>	11	
<i>C(3R)RM-SC1, kar ry</i>		
<i>C(3R)RM-SH3, +</i>		
<i>C(3R)RM-SH3, ry²</i>	1	
<i>C(3R)RM-SH4a</i>	12	originally <i>In(3R)C sb e l(3)elca K-pn</i>
<i>C(3R)RM-SH4b, ca K-pn</i>	12	
<i>C(3R)RM-SH19, +</i>		
<i>C(3R)RM-SH20, +</i>	12	
<i>C(3R)RM-SH21</i>	12	
<i>C(3R)RM-SHK16</i>	1	originally <i>kar² ry⁴⁰⁶ pic IG23 /ry⁺¹⁰</i> <i>Ace</i> ¹²⁶ ; used in half-tetrad analysis or <i>ry</i> fine structure
<i>C(3R)RM-SK2</i>	12	originally <i>p^P ss e^s /cu gl</i>
<i>C(3R)RM-VC1, e^S</i>	4	
<i>C(3R)RM-VK1, e^S</i>		
<i>C(3R)RM-VT2, cu</i>	6	

α In these designations the laboratory of origin is indicated by P = Pasadena, S = Storrs and V = Vancouver. The investigator by B = Baldwin, C = Chovnick, G = Garvin, H = Holm, HK = Hilliker, K = Kiceniuk, and T = Tabatabaie-Harger.

β 1 = Clark, Daniels, Rushlow and Hilliker, 1984, Genetics 108: 953-68; 2 = Evans, 1971 DIS 46: 123-24; 3 = Gethmann, 1976, Genetics 83: 743-51; 4 = Gosh and Mukherjee, 1986, DIS 63: 59-60; 5 = Hardy, 1975, Genetics 79: 231-64; 6 = Harger and Holm, 1980, Genetics 96: 455-70; 7 = Hilliker, 1981, DIS 56: 61-62; 8 = Hilliker, Gibson, Yeomans and Holm, 1977, DIS 52: 32; 9 = Hilliker, Holm and Appels, 1982, Genet. Res. 39: 157-68; 10 = Holm, 1983, DIS 59: 56-59; 11 = Holm, Baldwin, Duck and Chovnick, 1973, DIS 44: 112; 12 = Holm and Chovnick, 1975, Genetics 81: 293-311; 13 = Leigh and Sobels, 1970, Mutat. Res. 10: 475-87; 14 = Nishimura and Gethmann, 1983, Genetics 104: 545; 15 = Sandler,

Heterocompounds: Four types of heterocompounds are possible: *C(2L · 3L)*, *C(2L · 3R)*, *C(2R · 3L)*, and *C(2R · 3R)*; the chromosomal origin of the centromere in such compounds is frequently ambiguous. Pairs of these are produced as the components of 2-3 translocations in which both breakpoints are in the pericentric heterochromatin, e.g., *C(2L · 3L)*; *C(2R · 3R)*, and they may be carried together as translocations in either the homozygous or heterozygous condition. Pairs from two different translocations, which have one autosomal arm in common can be carried in combination with a normal chromosome and a homocompound for the missing arm. The two heterocompounds and the normal compound form a tricomplex; tricomplexes are chromosome constitutions that form a triradial synaptic configuration, involving three entire autosomal arms rather than four as seen in reciprocal-translocation heterozygotes. They are maintained in stock in combination with a compound for the fourth autosomal arm. For example, a triradial synaptic configuration is formed in the constitution *C(2L · 3L)/3L · 3R/C(3R · 2L)*, and it can be maintained in the presence of *C(2R)*. Alternatively a heterocompound may be carried in homozygous condition in combination with homocompounds for the missing arms, e.g., *C(2L)*; *C(2R · 3L)/C(2R · 3L)*; *C(3R)*. New heterocompounds may be induced by crossing irradiated females to heterocompound-bearing males that can generate the complementary gametic types (Parker, 1968, Effects of Radiation on Meiotic Systems, International Atomic Energy Agency, Vienna, pp. 209-18; Puro, 1985, Genet. Res. 46: 287-307). Puro (1973, Hereditas 75: 140-43; 1985, Genet. Res. 46: 287-307) used *T(2;3)N2-29* and *T(2;3)N2-46*, which he determined to be *2L · 3L*; *2R · 3R* and *2L · 3R*; *2R · 3L* respectively, to generate all four types of tricomplex and to characterize their meiotic behaviors. Such tricomplex-bearing flies are produced by crossing *T(2;3)N2-29/T(2;3)N2-46* females to *C(2L)*; *C(2R)* or *C(3L)*; *C(3R)* males. In addition to the heterocompounds recovered from the above two translocations he recovered and characterized *C(2R · 3R)HT10* and *C(2L · 3R)HT26* from irradiated females.

First Multiple Attached: see *C(1)M*

FMA: see *C(1)M*

RA: see *C(1)RA*

RR: see *C(1)RR*

TA: see *C(1)TA*

TMXB^S: see *C(1)TMB^S*

X-Y COMBINATIONS

The X and one or both arms of the Y chromosome may be linked by recombinational events occurring in the heterochromatin. Such X-Y combinations are composed of the X, a centromere (derived from either X or Y), and either YL or YS or both. In the designation of such chromosomes, the component elements are listed in order such that the X precedes the centromere (symbolized by a center point), e.g., YSX·YL. Events that give rise to X-Y attachments are usually recurring so that the same combinations arise repeatedly; however, since the exact points of exchange differ, independent occurrences of similar combinations certainly differ from one another in heterochromatic content. Because similar X-Y combinations of independent origin are not ordinarily designated, studied, or maintained as different chromosomes, and because for most purposes, it is not important that they be distinguished, for the most part, general categories of X-Y combinations are described in the ensuing section. Where a specific combination has been studied, it is listed with the designation of its component elements followed immediately by its specific designation, e.g., X·YLC2.

The complete designation of an X-Y combination consists of the following items in the order given: chromosomal elements, sequence of the X chromosome (if other than normal), gene content. X-Y combinations that differ from one another only with respect to mutant genes or euchromatic inversions are not described separately because it is considered that such mutants and inversions can be removed from or inserted into the component X by euchromatic exchange. When X's differ by an inversion with at least one heterochromatic breakpoint, the chromosomes are described separately because they must differ in their heterochromatic constitution.

FRI: see *YSX*

Fragment 1: see *YSX*

mwh⁺*YSX*: see *Dp(1;Y;3)M1-3*

*sc*⁸*c.o.X*: see *YSX, In(1)sc*⁸

XY⁺: see *X·YL*

X·*bw*⁺*YL*

constitution: X·(*bw*⁺-*ba*⁺) *KL*. X chromosome with prominent *YL* as second arm.

origin: Spontaneous in *y v/bw*⁺*Y*; *bw* stock.

synthesis: Erickson.

synonym: *Dp(1;Y;2)bw*⁺.

references: 1968, DIS 43: 63.

properties: X chromosome originally carried *y* and *v*; Y arm carries *YL* fertility factors with *bw*⁺-*ba*⁺ inserted proximal to *KL*.

X·YL

origin: A recurrent product of exchange between the proximal heterochromatin of *C(1)RM* and either arm of the Y. Also may result from exchange between *YS* and the proximal heterochromatin of a normal X or the interstitial heterochromatin of *C(1)RA*. Have also been recovered as one element of *T(1;Y)'s* induced in *X/Y* sperm with one break in *YS* and the other in X heterochromatin (Kennison, 1981, Genetics 98: 529-48). May be recovered with *YL* marked with various euchromatic

markers, e.g., *bw*⁺ (Erickson, 1968, DIS 43: 63), *y*⁺, or *B*^S.

synonym: *XY*⁺.

references: Stern, 1926, Biol. Zentralbl. 46: 505-08.

1929, Z. Indukt. Abstamm. Vererbungslehre. 51: 253-353.

Kaufmann, 1933, Proc. Nat. Acad. Sci. USA 19: 830-38 (fig.).

properties: An X chromosome in normal sequence with *YL* appended as a second arm. May carry varying amounts of the proximal part of *YS* between the X and the centromere; positions of such breakpoints with respect to *bb*, *ks1*, and *ks2*, as determined by Kennison for *T(X;Y)'s* provided with entries on the translocations. Males carrying X·YL require *KS* in some form for fertility. Chromosome V-shaped in metaphase.

other information: A series of X-ray-induced exchanges between X·YL and *y*⁺*Y* replacing *YL* with *y*⁺*YL* investigated by Frankel (1968, DIS 43: 99; 1973, Genetics 74: 115-32).

X·YLC2

constitution: X·YL, *bb*⁻*KL*.

origin: Recombination between *YS* proximal to *bb*⁺ and *C(1)RM* distal to *bb*⁺.

synthesis: Lindsley.

properties: Like X·YL but deficient for *bb*; X·YLC2/0 lethal. Shows unique behavior in double, first anaphase bridges (Novitski, 1952, Genetics 37: 270-87). Male sterile in combination with *y*⁺*Ymal*⁺ (Stone, 1984, Can. J. Genet. Cytol. 26: 67-77).

X·YL, In(1)EN

constitution: X·YL, *In(1)EN*, *y*·*KL*.

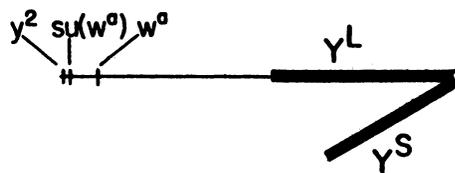
origin: Single euchromatic recombinant from *YSX·YL, In(1)EN/In(1)EN* female.

properties: An entirely inverted X chromosome with *YL* appended as a second arm.

XYL

origin: Detachment of *XYL·X* or *XYL·YLX* (attached X chromosomes synthesized from *XYL·YS*) by interchange with small, free-X heterochromatic duplications, i.e., *Dp(1)f*.

references: Lindsley and Novitski, 1959, Genetics 44: 187-96.



XYL·YS

From Lindsley and Novitski, 1959, Genetics 44: 187-96.

XYL·YS

constitution: *XYL·YS*; originally *y*² *su(w^a) w^a* (*bb*?) *KL·KS*.

origin: X-ray-induced detachment in *C(1)RM*, *y*² *su(w^a) bb/y*⁺*Y* female; Y-chromosome breakpoint distal to *k15*. Also a common product of X-Y translocation induced in *X/Y* sperm, at least when the Y is *B^SYy*⁺ (Kennison,

1981, Genetics 98: 529-48).

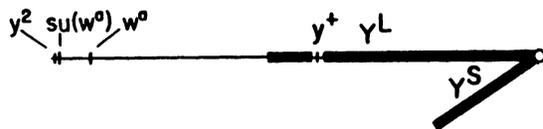
synthesis: Parker.

references: Parker and McCrone, 1958, Genetics 43: 172-86.

Lindsley and Novitski, 1959, Genetics 44: 187-96 (fig.).

properties: Essentially an intact Y chromosome with all of the X euchromatin appended distally to *KL*. Carries all the sex-chromosome material required for male viability and fertility.

other information: Several detachments of this constitution recovered (numbered 2-10T13, 2-10T15, 108-9, 112-17, and 129-11) as well as segregants from translocations with one break in X heterochromatin and one distal to *kl5* in $B^S Yy^+$ (designated E12, E17, F6, K1, N16, P9, V13, and W8).



XYL·YS129-16

XYL·YS129-16

constitution: XYL·YS; originally $y^2 su(w^a) w^a (bb?) y^+$ KL·KS.

origin: X-ray-induced detachment in *C(1)RM*, $y^2 su(w^a) bb/y^+ Y$ female.

synthesis: Parker.

references: Parker and McCrone, 1958, Genetics 43: 172-86.

Parker, 1968, DIS 43: 156.

properties: Essentially an intact Y chromosome with all of the X euchromatin attached to YL distal to y^+ . Interstitial position of y^+ shown by recovery of y^+ reattachment; also interstitial y^+ allele shows strong variegation. The break in the $y^+ Y$ occurs between *l(1)IAc⁺* and y^+ (Parker, 1968). Carries all the sex-chromosome material required for male viability and fertility.

XYL·YS, *bb*⁻

constitution: XYL·YS, $y v bb^- KL \cdot bb^- KS$; rDNA-deficient chromosome carrying most of $B^S Ybb^-$; derived by detachment of the distal [*In(1)dl49*] X of *C(1)DX* and its attachment to YL of $B^S Ybb^-$ with loss of B^S ; *In(1)dl49* replaced by recombination.

synthesis: Komma.

synonym: X-Y^{bb⁻}.

references: Komma and Endow, 1986, Genetics 114: 859-74.

properties: A *Ybb⁻* chromosome with the X euchromatin attached (in normal sequence) distal to YL. Carries all the sex-chromosome material required for male fertility, but is deficient for bobbed.

other information: *bb/XYL·YS*, *bb⁻* females show an increase in the number of 18S + 28S ribosomal genes (magnification) in their offspring.

X·YS

origin: Recurrent product of recombination between the proximal heterochromatin of *C(1)RM* and the Y. Also recovered as one element of *T(1;Y)*'s induced in X/Y sperm with one break in YL and the other in X heterochromatin (Kennison, 1981, Genetics 98: 529-48).

synthesis: Kaufmann.

references: 1933, Proc. Nat. Acad. Sci. USA 19: 830-38.

properties: An X chromosome in normal sequence with YS appended as a second arm. May carry varying amounts of the proximal part of YL between X and the centromere; positions of such breakpoints with respect to *kl5*, *kl3*, *kl2*, *Su(Ste)*, and *kl1* as determined by Kennison for *T(X;Y)*'s provided with entries on the translocations. Males carrying X·YS require KL in some form for fertility. Chromosome J-shaped in metaphase [e.g., Janning, 1970, Mol. Gen. Genet. 107: 150-57 (fig.)].

X·YS, *Basc*

constitution: X·YS, *In(1)sc^{SIL}sc^{8R}+S*, $y^{31d} sc^{S1} sc^8 w^a B \cdot y sc^+$.

origin: Recovered as an X-ray-induced detachment from a *C(1)RM*, *Basc/Dp(1;Y)IE* female.

synthesis: Ehrlich.

references: 1971, DIS 46: 108.

properties: Homozygous females viable and fertile; males viable and fertile with normal Y but lethal with *Dp(1;Y)IE*. Suppresses crossing over in X.

X·YSYL

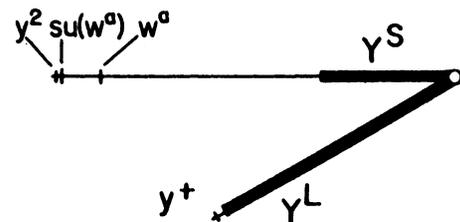
constitution: X·YSYL, $y w \cdot KS KL y^+$; metacentric chromosome.

origin: X ray induced in X·YS, $y w/y^+ YL$ male.

references: Johnsen, 1968, DIS 43: 158.

Johnsen and Zarrow, 1971, Mol. Gen. Genet. 110: 36-39.

properties: Attached XY with X in normal sequence on one side of the centromere and a complete Y on the other. X·YSYL/0 males fertile; transmission of the XY somewhat reduced.



XYS·YL

From Lindsley and Novitski, 1959, Genetics 44: 187-96.

XYS·YL

constitution: XYS·YL; originally $y^2 su(w^a) w^a (bb?) KS \cdot KL y^+$.

origin: X-ray-induced detachment in *C(1)RM*, $y^2 su(w^a) bb/y^+ Y$ female, in which the Y breakpoint is distal to *ks2*. Also a common product of X-Y translocation induced in X/Y sperm, at least when the Y is $B^S Yy^+$ (Kennison, 1981, Genetics 98: 529-48).

synthesis: Parker.

references: Parker and McCrone, 1958, Genetics 43: 172-86.

Lindsley and Novitski, 1959, Genetics 44: 187-96 (fig.).

properties: Essentially an intact y^+Y chromosome with all of the X euchromatin appended distally to YS. Carries all the sex-chromosome material required for male viability and fertility. YL may carry any marker available on the long arm of marked Y chromosomes, e.g., y^+ , B^S , etc.

other information: Two detachments of this constitution recovered (110-8 and 115-9) as well as segregants from translocations with one break in X heterochromatin and one distal to *ks2* in $B^S Yy^+$ (designated G7, N10, N30, P1, V4, V23, V25, V36, and Z14).

YSX

constitution: YSX; originally *KS y cv v f* (Braver).

origin: Spontaneous derivative from $YSX \cdot YL, In(1)EN, KS y \cdot KL/sc cv v f$.

synthesis: Novitski.

synonym: *FR1: Fragment 1*.

references: Novitski, 1952, Genetics 37: 270-87.

Lindsley and Novitski, 1959, Genetics 44: 187-96.

Janning, 1970, Mol. Gen. Genet. 107: 150-57 (fig.).

properties: An X in normal sequence marked with y and with YS appended distal to $l(1)IAa^+$. Reduces crossing over near y.

YSX, In(1)FM7

constitution: $YSX, In(1)FM7, y^+ KS y^- w^a v^{Of}$.

origin: X-ray-induced detachment by exchange between the interstitial heterochromatin of *C(1)M4* with the short arm of $B^S Yy^+$.

references: Craymer, 1974, DIS 51: 21.

YSX, In(1)sc⁸

constitution: $YSX, In(1)sc^8, KS [l(1)IAa-ac]^- sc^8$.

origin: Infrequent product of spermatogonial exchange between YS and the distal inverted heterochromatic segment of $In(1)sc^8$. The incidence increased twenty fold in genotypes undergoing ribosomal DNA magnification [e.g., $In(1)sc^8, bb^1 sc^8/Ybb$], but decreasing with successive generations of magnification (Ritossa, 1973, Proc. Nat. Acad. Sci. USA 70: 1050-54).

synthesis: Sidorov.

synonym: $sc^8 c.o. X$.

references: Sidorov, 1940, Bull. Biol. Méd. Exp. URSS 9: 10-12.

1941, Dokl. Akad. Nauk SSSR, Ser. Biol. 30: 248-49.

Lindsley, 1955, Genetics 40: 24-44.

Ritossa, 1973, Proc. Nat. Acad. Sci. USA 70: 1950-54.

properties: $In(1)sc^8$ with the distal uninverted euchromatic region carrying the normal alleles of $l(1)IAa$ through *ac* replaced by *KS*; deficiency covered in single crossover between $in(1)sc^8$ and $In(1)EN$, i.e., $In(1)sc^8 L EN^R$. Resembles $In(1)sc^8$ in mitotic prophase.

YSXX · YL

constitution: An X-Y combination carrying a *C(1)RA*; the distal X is YSX in normal sequence, and the proximal X is an inverted sequence with YL attached as a second arm.

origin: Exchange in triploid females carrying *C(1)RA*, first replacing the centromere region of the proximal X with that of $YSX \cdot YL, In(1)EN$ and then the terminus of the distal X with that of YSX.

references: Lindsley and Novitski, 1959, Genetics 44: 187-96.

YSX · YL

constitution: $YSX \cdot YL, KS y \cdot YL$.

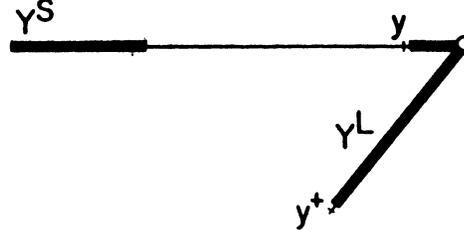
origin: Recombination between YSX and $X \cdot YL$.

synthesis: Lindsley and Novitski.

references: 1959, Genetics 44: 187-96.

properties: An attached XY with the X in normal sequence.

Contains all of the sex-chromosome material required for male viability and fertility. Commonly kept in stock as $YSX \cdot YL/0$ males crossed to $C(1)/0$ females.



YSX · YL, In(1)EN

From Lindsley and Novitski, 1959, Genetics 44: 187-96.

YSX · YL, In(1)EN

constitution: $YSX \cdot YL, In(1)EN, KS y \cdot KL y^+$.

origin: Recovered as recombinant between the proximal heterochromatin of $YSX, In(1)sc^8 L EN^R$ and $y^+ Y$.

synthesis: Lindsley, 1949.

references: Lindsley and Novitski, 1960, DIS 24: 84-85.

1959, Genetics 44: 187-96 (fig.).

properties: Contains all of the sex-chromosome material required for male viability and fertility. Exists without the y^+ marker at the end of the YL arm. A derivative with B^S at the end of YL described by Craymer (1974, DIS 51: 21); also exists with various combinations of sex-linked markers. Two X-ray-induced paracentric-inversion derivatives described by Novitski: $In(1)24$, a nearly complete inversion with one break in the normally proximal euchromatin and the other in the centric heterochromatin, and $In(1)26$ with one break in 10A and one break in heterochromatin, distal or proximal not specified (Novitski, 1951, DIS 25: 122; Lindsley and Novitski, 1959, Genetics 44: 187-96).

YSX · YL, In(1)FM7

constitution: $YSX \cdot YL, In(1)FM7^L EN^R, y^+ KS y v^{Of} \cdot KL B^S$.

origin: Recombination between $YSX, In(1)FM7, y^+ KS y^- w^a v^{Of}$ and $YSX YL, In(1)EN, KS y \cdot KL B^S$ such that the $w^a y^-$ base of FM7 is replaced by the $w^+ y \cdot KL$ of $YSX \cdot YL, In(1)EN$.

synthesis: Craymer, 74d.

synonym: FM7Y.

references: Craymer, 1974, DIS 51: 21.

Mitchell, 1977, Genetics 87: 763-74.

properties: Contains all of the male fertility factors. The FM7 inversions prevents euchromatic crossovers and the y^+ and B^S markers serve to detect recombinational events in the heterochromatin.

YSXYL ·

constitution: $YSXYL \cdot, KS y \cdot KL \cdot$.

origin: Rarely recovered recombinant between $XYL \cdot$ and YSX.

synthesis: Lindsley.

references: Lindsley and Novitski, 1959, *Genetics* 44: 187-96.
Novitski and Brosseau, 1964, *Genetics* 50: 273 (abstr.).
Donady, Seecof, and Fox, 1973, *Genetics* 73: 429-34.
properties: Shows extremely reduced recovery from above

heterozygous females (Lindsley and Novitski), all zygotes carrying *YSXYL* being lethal in some autosomal backgrounds; reciprocal recombinant recovered frequently (Novitski and Brosseau; Donady *et al.*).

Y DERIVATIVES

The Y chromosome consists of a long arm (YL) and a short arm (YS); the long arm is arbitrarily taken as the left arm. Special Y chromosomes include (1) marked Y's which are used as Y tracers and (2) Y fragments, unmarked or marked, which are used as duplications or, when marked, as Y-arm tracers. Y fragments are symbolized either YL or YS plus necessary distinguishing notation, e.g., YS8. The Y chromosome may be marked by mutating the genetically demonstrable elements of the Y or by translocating normal or mutant alleles from other parts of the complement to the Y chromosome. Marked-Y chromosomes are symbolized by combining, without intervening punctuation, the symbol for the normal or mutant gene of primary marker intent with the symbol Y. If the marker is in the long arm, its symbol precedes Y (e.g., y^+Y); if it is in the short arm, its symbol follows Y (e.g., Ybb). Symbols for marked fragments combine the symbol for the appropriate Y arm with that for the marker gene, listed in order. These notations are separated by a center point when the centromere lies between them (e.g. $YL \cdot sc^{S1}$); otherwise they are not separated by punctuation (e.g., y^+YL). The long arm of the Y carries a complex of male fertility genes, *KL*, plus an *ABO* element and *Su(Ste)*, and the short arm carries a normal allele of *bb* and an *ABO* element proximally and a complex of male fertility factors, *KS*, distally. The genetic constitution of the Y chromosome may be designated by listing the above components and the centromere in order from left to right, $kl5^+ kl3^+ kl2^+ ABO-YL Su(Ste) kl1^+ \cdot ABO-YS bb^+ ksl^+ ks2^+$. The genetic constitution of a marker segment is designated by listing the symbols of the most widely separated loci known to be included in it separated by an em dash, e.g., $bw^+ - ba^+$. The constitution of a Y fragment may be designated by listing its genetic elements in order with any ambiguities in order enclosed within parentheses, e.g., $KL(bw^+ - ba^+) bb^+ KS$. When there is a hierarchy of ambiguities in order, a hierarchy of parentheses is used, as in $((ci^+ - spa^+)KL)bb^+ KS$.

4Y

constitution: $((ci^+ - spa^+)KL)bb^+ KS$; tentative.

origin: X ray induced.

synthesis: Edmondson, 1946.

synonym: *Tp4Y*.

references: Muller and Edmondson, 1957, DIS 31: 140-41.

properties: Contains all known loci of chromosome 4 linked to the Y chromosome. Results from recombination between 4Y and *YSX* · *YL* suggest that 4 is inserted into or appended to *YL*. Two doses of this chromosome in the absence of any other Y- or 4-derived material produce viable and fertile flies of both sexes.

 $*B^S w^+ y^+ Y$

constitution: $B^S pdf^+ l(1)20Bb - su(f)^+ Pgd^A - spl^+ y^+ ac^+ KL \cdot bb^+ KS$; inferred from origin.

origin: X-ray-induced deletion of the majority of euchromatin (dm^+ through mal^+) from the recombinant composed of the left end of X^{PYD} element of $T(1;Y)148 = T(1;Y)2D;YL$, which involves B^SY , and the right end of $XYL \cdot YS129-16$, which carries y^+ from y^+Y between X and YL.

synthesis: Nicoletti.

synonym: $Dp(1;Y)B^S w^+ y^+$.

references: Brosseau, Nicoletti, Grell, and Lindsley, 1961, Genetics 46: 339-46.

properties: Meiotic behavior and viability apparently normal. Produces *Co* effect; covers *N*. Has in addition combined marker characteristics of B^SY and y^+Y .

 $B^S w^+ Y$

constitution: $B^S pdf^+ l(1)20Bb - su(f)^+ pn^+ - dm^+ KL \cdot bb^+ KS$; inferred from origin.

origin: X-ray-induced deletion of the majority of euchromatin (rb^+ through mal^+) from a recombinant carrying left end of the X^{PYD} element of $T(1;Y)148 = T(1;Y)2D;YL$, which involves B^SY , and the right end of $XYL \cdot YS$.

synthesis: Nicoletti.

synonym: $Dp(1;Y)B^S w^+$.

references: Brosseau, Nicoletti, Grell, and Lindsley, 1961, Genetics 46: 339-46.

properties: Produces *Co* phenotype in $X/B^S w^+ Y$ male and $XX/B^S w^+ Y$ female. Covers many *N* deficiencies. *B* phenotype as in B^SY .

 $B^S y^+ Y$

constitution: $B^S pdf^+ l(1)20Bb^+ - su(f)^+ y^+ ac^+ KL \cdot bb^+ KS$.

origin: X-ray-induced deletion of the euchromatin (pn^+ through mal^+) from a recombinant carrying the left end of the X^{PYD} element of $T(1;Y)148 = T(1;Y)2D;YL$, which involves B^SY , and the right end of $XYL \cdot YS129-16$, which carries y^+ from y^+Y between X and YL.

synthesis: Nicoletti.

synonym: $Dp(1;Y)B^S y^+$.

references: Brosseau, Nicoletti, Grell, and Lindsley, 1961, Genetics 46: 339-46.

Parker, 1968, DIS 43: 156.

properties: Detachments from $C(1)RM/B^S y^+ Y$ females carry both markers (y^+ and B^S) or neither, so order not $B^S y^+$ but $B^S y^+ Y$ as given.

 $B^S Y$

constitution: $B^S pdf^+ l(1)20Bb^+ - su(f)^+ KL \cdot bb^+ KS$.

origin: X-ray-induced deletion of the euchromatin (including mal^+) from a recombinant carrying the left end of the X^{PYD} element of $T(1;4)B^S = T(1;4)15F9-16A1;16A7-B1;102F$ and the right end of $XYL \cdot YS$.

synthesis: Brosseau.

synonym: $YB^S, Dp(1;Y)B^S$.

references: Brosseau and Lindsley, 1958, DIS 32: 116.

Brosseau, Nicoletti, Grell, and Lindsley, 1961, Genetics 46: 339-46.

properties: Causes extreme *B* phenotype; presence readily scorable in $+/+$, $B/+$, and $+$ but not in *B* or B/B constitutions. Shown to carry pdf^+ in addition to *B* from 16A (Grell) and $l(1)20Bb^+$ through $su(f)^+$ from region 20 (Schalet and Lefevre, 1973, Chromosoma 44: 183-202). Does not cover spa^{pol} . Viability and fertility of $X/B^S Y$, $X/B^S Y/B^S Y$, and $X/X/B^S Y$ good. Eyes of $X/B^S Y$ detectably wider than those of $X/B^S Y/B^S Y$. Three euchromatic bands visible in salivary chromosomes (Nicoletti and Lindsley, 1960, Genetics 45: 1705-22). Prophase chromosomes show the presence of an extra Hochst-bright segment ($B^S Xh$) appended to the end of YL and separated from it by a non-fluorescent junction gap ($B^S Xh_j$) (Gatti

and Pimpinelli, 1983, *Chromosoma* 88: 349-73).

B^SYbb⁻

constitution: $B^S pdf^+ l(1)20Bb^+ - su(f)^+ KL \cdot bb^- KS$; carries *Ybb⁻* deletion on *YS*.

origin: Derived from original *Ybb⁻* chromosome of Schultz by recombination with $B^S Yy^+$.

synthesis: Komma.

references: Endow, 1982, *Genetics* 102: 91-99.

Hawley and Tartof, 1983, *Genetics* 104: 63-80.

properties: *Y* chromosome deficient for most of the rDNA sequences, but carrying some sequences similar but not identical to those on *Ybb⁻* chromosome (Endow, 1982). Marked with B^S . Magnification induced as with *Ybb⁻*.

B^SYy⁺

constitution: $B^S pdf^+ l(1)20Bb^+ - su(f)^+ KL \cdot bb^+ KS ac^+ - l(1)IAa^+$.

origin: Recombination between B^S and $bw^+ Yy^+$.

synthesis: Brosseau.

references: Brosseau, 1958, *DIS* 32: 115-16.

Brosseau, Nicoletti, Grell, and Lindsley 1961, *Genetics* 46: 339-46.

Williamson, 1968, *DIS* 43: 157.

properties: *YL* in mitotic prophase same as that in $B^S Y$; *YS* carries the Hoechst-bright segment (*Xhy⁺*) found on *YL* of $y^+ Y$ (Gatti and Pimpinelli, 1983, *Chromosoma* 88: 349-73).

other information: Several derivatives of $B^S Yy^+$ with newly induced mutant alleles of *y* have been described.

derivative	origin	ref ^α	phenotype
$B^S Yy^{61d}$	tritium	1	yellowish body, bellow bristles, wild type wings
$B^S Yy^{67}$	EMS	3	yellow body, dark bristles
$B^S Yy^{b12}$	X ray	2	wild-type body, yellow bristles

^α 1 = Hughes and Hildreth, 1967, *DIS* 42: 86; 2 = Seattle-LaJolla *Drosophila* Laboratories, 1971, *DIS* 47 (supplement); 3 = Williamson, 1968, *DIS* 43: 65.

B^SYy^{31d}

constitution: $B^S pdf^+ l(1)20Bb^+ - su(f)^+ KL \cdot bb^+ KS ac^+ y^{31d} - l(1)IAa^+$; inferred from origin..

origin: Recombination between $B^S Yy^+$ and $In(1)sc^8, y^{31d} sc^8$ in a female.

synonym: $Dp(1;Y)B^S y^{31d}$.

references: Brosseau, Nicoletti, Grell, and Lindsley, 1961, *Genetics* 46: 339-46.

$B^S - Y$: see $y^+ Yv^+$

B^{SV}Y

origin: Five X-ray-induced derivatives of $B^S Y$.

synthesis: Brosseau.

synonym: $Dp(1;Y)B^{SV}$.

references: Brosseau, 1960, *Genetics* 45: 979.

Moore, Procunier, Cross, and Grigliatti, 1979, *Nature* 282: 312-14.

properties: Show variegation for *B*. With a normal *X*, the eye phenotype is B^+ , with *XY*, it is B^S , and with *XYL* or *YXS*, it is intermediate *B*. The enhancers of variegation, $Df(2R)M41A1$ and $E(var)7$, shift the phenotype of $B^{SV} Y$ flies toward normal (Brosseau, 1960), whereas deficiencies that wholly or partially remove *His* genes [$Df(2L)TW65$, $Df(2L)TW84$, and $Df(2L)TW161$] shift the phenotype toward B^S (Moore *et al.*).

other information: Eleven $B^{SV} Yy^+$ chromosomes of independent origin (The Seattle-LaJolla *Drosophila* Laboratories, 1971, *DIS* 47, supplement) produce a larger eye phenotype in *XY/Y* males than in *X/Y* males.

bw⁺Y

constitution: $KL (bw^+ - ba^+) bb^+ KS$.

origin: Aneuploid segregant from $Tp(2;Y)YL;58F1-59A2;60E3-F1$ (Gersh, 1956, *DIS* 30: 115; Nicoletti).

synthesis: Dempster.

synonym: $Y:bw^+; Dp(2;Y)bw^+$.

references: Muller, 1942, *DIS* 16: 64.

1951, *DIS* 25: 119.

1955, *DIS* 29: 146.

properties: A section of *2R* carrying bw^+ inserted into *YL* proximal to *KL* (Baker, 1955, *DIS* 29: 101-03). Inserted segment known to carry normal alleles of *bw*, *mr*, *or*, *Fo*, *Pin*, *bs*, and *ba* but not *px*, *hy*, *crs*, $M(2)58F$, or $M(2)60C$. Males with two doses of $bw^+ Y$ lethal.

bw⁺Yy⁺

constitution: $KL (bw^+ - ba^+) bb^+ KS ac^+ - l(1)IAa^+$ (Baker, 1955, *DIS* 29: 101-03).

synthesis: Cooper.

synonym: $sc^8 Ybw^+$.

references: Cooper, 1951, *DIS* 26: 97.

cn⁺Y

constitution: Segment of *2R* carrying cn^+ inserted onto *Y*.
origin: $Dp(2;Y)$ segregant from $Tp(2;Y)C = Tp(2;Y)41A;43$.

references: Sullivan, Kitos, and Sullivan, 1973, *Genetics* 75: 651-61.

properties: Covers *cn*.

ct⁺y⁺Y

constitution: $KS cm^+ - ct^+ mal^+ - bb^+ (het) y^+ ac^+ KL \cdot KS$ (tentative).

origin: X-ray-induced deletion of the majority of the euchromatin from a recombinant carrying the left end of the $X^F Y^D$ element of $T(1;Y)131 = T(1;y)6E;YS$ and the right end of $XYL \cdot YSI29-16$.

synthesis: Johnson.

synonym: $Dp(1;Y)ct^+ y^+$.

references: Johnson and Judd, 1979, *Genetics* 92: 485-502.

Nicklas and Cline, 1983, *Genetics* 103: 617-31.

properties: Carries wild-type alleles of *y-ac*, *cm-ct*, and *mal-bb*.

FR2: see $y^+ YL$

Fragment 2: see $y^+ YL$

l(1)1Ac⁺Y

constitution: $l(1)1Ac^+ KL bb^+ KS$.

origin: Neutron-induced derivative of $y^+ Y$.

synthesis: Muller.

synonym: $l(1)J1^+ Y$.

references: Muller, 1954, *DIS* 28: 140-43.

Padilla and Nash, 1977, *Mol. Gen. Genet.* 155: 171-77.

properties: Like $y^+ Y$ except that y^+ and ac^+ , but not $l(1)1Ac^+$ deleted; other $l(1)1A$ loci not tested.

$l(1)J1^+ Y$: see $l(1)1Ac^+ Y$

pn⁻w⁺Y

constitutioci: *kz⁺—spl⁺ KL bb⁺ KS*.

pn⁻w⁺Y

origin: X-ray-induced loss of *pn⁺* in *w⁺Y* chromosome.

synthesis: Lifschytz and Falk.

synonym: *Dp(1;Y)pn⁻w⁺*.

references: Lifschytz and Falk, 1969, *Genetics* 62: 343-52.

properties: Same as *w⁺Y* except for the absence of *pn⁺*.

R(Y)bw⁺

constitution: *KL (bw⁺—ba⁺) bb⁺ KS*; closed to form a ring.

origin: X-ray-induced derivative of *bw⁺Yy⁺*.

synonym: *Y^cbw⁺*; *MYR: Marked Y Ring*.

references: Oster and Lyengar, 1955, *DIS* 29: 159.

Stone, 1982, *Genetics* 102: 245-58.

properties: Ring shaped in mitotic metaphase. Lacks *y⁺* present in the progenitor chromosome. Introduction of *R(Y)bw⁺* via male into certain strains results in death of nearly all male progeny during early embryogenesis. About 10% of strains are subject to such killing of male offspring. Introduction of *R(Y)bw⁺* via female does not result in death of the sons (Oster, 1964, *Genetics* 50: 274). Inviability of *R(Y)bw⁺*-bearing embryos shown by Stone to depend on genotype of mother.

R(Y)F: Ring Y Fillicidal

constitution: Seven unmarked ring-Y chromosomes plus one marked with *su(f)⁺*.

origin: X- and γ -ray-induced derivatives of *B^SYy⁺*.

synthesis: Oster.

references: Oster, 1964, *Genetics* 50: 274 (abstr.).

Stone, 1982, *Genetics* 102: 245-58.

properties: Pre-blastoderm embryos produced by so-called ring-sensitive females and carrying *R(Y)F*, either paternal or maternal in origin, are disorganized and fail to develop; the genetic basis of maternal ring sensitivity is complex (Stone).

R(YL)

constitution: *KL* closed to form a ring.

origin: Selected on the basis of marker loss from singly or doubly marked *Y* chromosomes; recovery from *y⁺Y* requires two breaks; those recovered from *bw⁺Yy⁺* represent a rare subset of derivatives and are attributed to four breaks.

chromosome	origin	source	retains	ref ^α	synonym
<i>R(YL)</i>	spont	<i>y⁺Y</i>	<i>KL</i>	4	<i>Y^{cl}, Y^{Lc}</i>
<i>*R(YL)14</i>	X ray	<i>bw⁺Yy⁺</i>	<i>KL</i>	1, 3	<i>Y^{Lc} 14</i>
<i>R(YL)15</i>	spont	<i>bw⁺Yy⁺</i>	<i>KL</i>	2, 3	<i>Y^{Lc} 15</i>
<i>R(YL)16</i>	X ray	<i>bw⁺Yy⁺</i>	<i>KL</i>	1, 2	<i>Y^{Lc} 16</i>
<i>R(YL)bb⁺</i>	X ray	<i>bw⁺Yy⁺</i>	<i>KL bb⁺</i>	1, 3	<i>Y^{Lc} .bb⁺</i>

^α 1 = Baker, 1955, *DIS* 29: 101-02; 2 = Baker, 1971, *Proc. Nat. Acad. Sci. USA* 68: 2472-76; 3 = Baker and Spofford, 1959, *Univ. Texas Publ.* 5914: 135-54 (fig.); 4 = Muller, 1948, *DIS* 22: 73-74.

properties: *R(YL)* does not respond to the ring sensitivity of certain maternal genotypes (Oster, 1964, *Genetics* 50: 274).

R(YS)bw⁺

constitution: (*bw⁺—ba⁺*) *bb⁺ KS*; closed to form a ring.

Order of elements inferred from origin.

origin: X-ray-induced derivative of *bw⁺Yy⁺*.

synthesis: W. K. Baker.

synonym: *Y^{cS}:bw⁺bb⁺*.

references: Baker and Spofford, 1959, *Univ. Texas Publ.* 5914: 135-54 (fig.).

properties: Ring shaped in mitotic metaphase. Lacks *y⁺* and *KL* from the treated chromosome. Shows some somatic and germinal instability.

RspYy⁺

constitution: *M(2)41A⁺—l(2)41Aa⁺ KL bb⁺ KS ac⁺—l(1)1Aa⁺*.

origin: X-ray-induced deletion of majority of euchromatin from *Y^{P2R^D}* element of *T(Y;2)cb25*, which has one break between *B^S* and *KL* in *B^SYy⁺* and one proximal to *Rsp* in *2R*; retained euchromatin not characterized.

references: Lyttle and Ault, 1985, *Genetics* 110: s23.

phenotype: Two such products isolated, 9 and 11. Both appear to show preferential recovery, with *k* values >0.95, among progeny of *SD/+* males.

other information: See also *Tp(2;Y)Rsp*.

sc · YL: see *YL · sc^{S1}*

sc⁸ ENc.o. Y: see *YL · y⁺*

sc⁸ · Y: see *y⁺Y*

sc⁸ · Y:bw⁺: see *bw⁺Yy⁺*

sc^{8V} · Y: see *y^{v56Y}*

sc^{S1} · YL#2: see *YL · sc^{S1} 2*

sc^{S1} c.o.Y EY80: see *YL · sc^{S1} 3*

sc^{V1} · YS

constitution: *l(1)1Aa⁺—sc^{V1} bb⁺ KS*; tentative.

origin: Spontaneous recombinant from *ln(1)sc^{V1}/Y* male.

synthesis: Muller.

references: 1948, *DIS* 22: 73-74.

properties: Small, two-armed chromosome in mitotic metaphase. Survives in combination with *C(1)DX* and therefore carries *bb⁺*.

sc^{V1} · YS mwh⁺

constitution: *l(1)1Aa⁺—sc^{V1} bb⁺ KS ve⁺ mwh⁺*.

origin: Spontaneous derivative of *Dp(1;1)sc^{V1}* and *T(Y;3)P6 = T(Y;3)62A-B*.

synthesis: Ripoll and Garcia-Bellido.

synonym: *Dp(1;Y;3)H*.

references: 1973, *DIS* 50: 177.

properties: Carries *YS* fertility factors plus *mwh⁺* and *ve⁺*. *y⁺* of the *sc^{V1}* element variegates very strongly in *y* males; no *mwh* variegation.

shi⁺ Y

origin: Deletions of the majority of the euchromatin from a recombinant comprising the left end of *T(1;Y)P12 = T(1;Y)13F1-2;YSy⁺* and the right end of *XYS · YLB^S*.

synthesis: Poodry.

synonym: *Dp(1;Y)shi⁺*.

references: 1980, *DIS* 55: 210.

properties: Covers *sd*, *tc*, and *shi*; marked with *y⁺* and *B^S*.

chromosome constitution

shi⁺ Y1 covers *Df(1)sd72b = Df(1)13F1;14B1* only in the presence of *Dp(1;4)r⁺ = Dp(1;4)14A1-2;16A1-2;102F2-3*

**shi⁺ Y2*

chromosome constitution

sh^rY3 covers *Df(1)sd72b*, but not *r* at 15A1**su(f)⁺Y****origin:** X ray induced in *T(1;Y;4)B^S* males.**synthesis:** Wong, 1965.**synonym:** *Dp(1;Y)su(f)⁺*.**references:** Gethmann, 1967, DIS 42: 39.**properties:** Covers the deficiency in *C(1)RA60g* and carries *su(f)⁺*. *Tp4Y*: see *4Y* *TplY*: see *Dp(3;Y)Tpl* *TplYtrx^D*: see *Dp(3;Y)trx^D***w⁺y⁺Y****constitution:** *Pgd^A—Sgs4⁺y⁺ac⁺KL·bb⁺KS*; inferred from origin and supposed constitution of *B^Sw⁺y⁺Y*.**origin:** X-ray-induced derivative of *B^Sw⁺y⁺Y*.**synthesis:** Nicoletti.**references:** Brosseau, Nicoletti, Grell, and Lindsley, 1961, Genetics 46: 339-46.**properties:** Like *B^Sw⁺y⁺Y*, but with *B^S* and *su(f)⁺* missing.**w⁺Y****constitution:** *Pgd^A—Sgs4⁺y⁺KL·bb⁺KS*; inferred from origin. *Dp(1;Y)2D1-2;3D3-4;Y* (Judd *et al.*). Also *Dp(1;Y)20B;20F* according to Lifschytz and Jakobovitz. Also associated with *T(Y;2)YL;22D* (Schultz) in which the break in *YL* is distal to the *Pgd^A—Sgs4⁺* insertion.**origin:** Spontaneous in *C(1)RAYL/w⁺y⁺Y* female. Seems likely that the progenitor *w⁺y⁺Y* was different from the one described here and was already translocated with chromosome 2.**synthesis:** Nicoletti.**references:** Brosseau, Nicoletti, Grell, and Lindsley, 1961, Genetics 46: 339-46.

Judd, Shen, and Kaufman, 1972, Genetics 71: 139-56.

Gerasimova and Ananjev, 1972, DIS 48: 93.

Lifschytz and Jakobovitz, 1978, Mol. Gen. Genet. 161: 275-84.

McGinnis, Farrell, and Beckendorf, 1980, Proc. Nat. Acad. Sci. USA 77: 7367-71.

properties: Causes a confluens phenotype.**molecular biology:** 3D3-4 breakpoint lies between 1 and 2.5 kb to the right of the right breakpoint of *Df(1)dm75e19* breakpoint (McGinnis *et al.*).**w⁺Y2****constitution:** *Pgd^A—Sgs4⁺y⁺KL·bb⁺KS*; inferred from origin.**origin:** γ-ray-induced loss of *y⁺* from *w⁺y⁺Y*.**references:** Kennison, 1981, Genetics 98: 529-48.**properties:** Like *w⁺Y* but not associated with a *T(Y;2)*.**y⁺bb⁺Ybb⁻****constitution:** *y⁺bb⁺KL·bb⁻KS*.**origin:** *mei-41*-induced exchange between *B^SYbb⁻* and the distal heterochromatin of *In(1)sc⁸*, the *B^S* marker on *YL* of *B^SYbb⁻* being replaced by *y⁺bb⁺* from *In(1)sc⁸*. Site of exchange on the X within the rDNA; on the Y distal to *k15*.**references:** Hawley and Tartof, 1985, Genetics 109: 691-

700.

properties: Male fertile with a normal X.**y⁺bb⁺Ybb⁻****constitution:** *y⁺bb⁺KL·bb⁻KS*.**origin:** Similar to that of *y⁺bb⁺Ybb⁻* except that the site of exchange on the X proximal to the rDNA.**references:** Hawley and Tartof, 1985, Genetics 109: 691-700.**properties:** Male fertile with a normal X.**y⁺bb^{r1}Ybb⁻****constitution:** *y⁺bb^{r1}KL·bb⁻KS*.**origin:** Derived by reduction from *y⁺bb⁺Ybb⁻*.**references:** Hawley and Tartof, 1985, Genetics 109: 691-700.**properties:** Carries *bb^{r1}* = reduced lethal, with about 20 rDNA genes. *y⁺dorY*: see *Dp(1;Y)dor***y⁺g⁺Y****constitution:** *l(1)IAa⁺—ac⁺na⁺—ben⁺KL·bb⁺KS*.**origin:** X-ray-induced deletion of majority of euchromatin from *XYL·YS, In(1)sc²⁹ = In(1)1B;13A2-5*.**synthesis:** Schalet.**properties:** Covers *mus(1)101*, *ty*, and *g* in addition to *ben* and *na*; should also carry *Ste*. Does not rescue *Df(1)HA92 = Df(1)12A6-7;12D3*; although some *Df(1)HA92/y⁺g⁺Y* males survive to late pupal stages, they are unable to complete emergence from the pupal case.**y⁺lz⁺Y****constitution:** *l(1)IAa⁺—ac⁺lz⁺—mus109⁺KL·bb⁺KS*.**origin:** X-ray-induced deletion of majority of euchromatin from *XYL·YS, In(1)y^{A74c} = In(1)1B1-2;9D*.**synthesis:** Schalet.**properties:** Does not cover *t* or *Su(f)*.**y⁺na⁺Y****constitution:** *l(1)IAa⁺—ac⁺na⁺KL·bb⁺KS*.**origin:** X-ray-induced deletion of majority of euchromatin from *XYL·YS, In(1)sc²⁹ = In(1)1B;13A2-5*.**synthesis:** Schalet.**properties:** Covers *na*; neighboring genes not tested; should also carry *Ste*. Covers lethality of *Df(1)RK2 = Df(1)12D2-E1;13A2-5*, but surviving males are sterile and display an abnormal eye shape.**y⁺scY****constitution:** *Dp(1;Y)1E3-4*; Y arm involved not specified.**synonym:** *Dp(1;Y)y⁺sc*.**references:** White, Decelles, and Enlow, 1983, Genetics 104: 433-48.**properties:** Covers *l(1)IAa—su(w^a)*.**y⁺Y****constitution:** *l(1)IAa⁺—ac⁺KL·bb⁺KS*.**origin:** X ray induced in spermatogonial cell of *In(1)sc⁸/Y* male.**synthesis:** Muller.**synonym:** *sc⁸Y*.**references:** Muller, 1948, DIS 22: 73-74.

Hadorn, Grell, and Schultz, 1970, Proc. Nat. Acad. Sci. USA 65: 633-37.

Grell, R., 1971, Mol. Gen. Genet. 110: 218-37.

Baker, 1973, Dev. Biol. 33: 429-40.

properties: Tip of *In(1)sc*⁸ including *l(1)IAa*⁺ through *ac*⁺, but not *sc*, transferred to the tip of *YL* distal to *KL*. *YL* appears to be as long as the *X* in metaphase (Hannah); a large Hoechst-bright segment (*y*⁺*Xh*) appended to the end of *YL* and separated from it by a short non-fluorescent gap (*y*⁺*Xhj*) (Gatti and Pimpinelli, 1983, Chromosoma 88: 349-73). Detachment studies show that *bb*⁺ from *In(1)sc*⁸ has not been transferred to *YL* (Parker). Has dominant *Hw* effect that produces one or more humeral microchaetae in *X/y*⁺*Y* male and *X/X/y*⁺*Y* female and one or more hairs in the membrane of the second and third posterior cells of wing (Schultz) and extra veins, especially associated with the posterior crossveins (Williamson, 1968, DIS 43: 157) in *X/y*⁺*Y/y*⁺*Y* males. *y/y*⁺*Y* males show some variegation for *y* without enhancers of variegation and considerable *y* variegation in the presence of *E(var)7* and *E(var)21* (Hadorn *et al.*, 1970; Grell, 1971).

other information: Derivatives with mutant alleles of *y* have been recovered.

derivative	origin	ref ^α	phenotype
<i>y</i> ⁵³¹ <i>Y</i>	spont	2	
<i>y</i> ^{P59} <i>Y</i>	spont	3	like <i>y</i> ²
<i>y</i> ^{v56} <i>Y</i>	X ray	1	yellow variegation

^α 1 = Hinton and Schmidt, 1956, DIS 30: 121; 2 = Lüning, 1953, DIS 27: 58; 3 = Meyer, 1959, DIS 33: 97.

***y*⁺*Ydor*⁺**

constitution: *l(1)IAa*⁺—*dor*⁺ *fog*⁺—*su(f)*⁺ *KS KL y*⁺.
origin: X-ray-induced deletion in *X* euchromatin of *XYs·YL*, *y*² *su(w^a) w^a KS · KL y*⁺ male.
synthesis: Holm.
synonym: *Dp(1;Y)dor*⁺ *y*⁺.
references: Holm, 1968, DIS 43: 143.
properties: Reduced body size and *Hw* effect in males. Body size normal and little *Hw* effect in compound-X-bearing females with the duplication. Duplication carries mutant alleles, *y*² and *su(w^a)* and a wild-type allele of *y*.

y*⁺*YL

constitution: *l(1)IAa*⁺—*ac*⁺ *KL bb*⁺.
origin: Spontaneous product from *YSX·YL*, *In(1)EN*, *KS y·KL y*⁺ female.
synthesis: Novitski.
synonym: *FR2: Fragment 2*.
references: Novitski, 1952, Genetics 37: 270-87.
properties: Has subterminal centromere and extremely short second arm in mitotic metaphase. Constitution confirmed by analysis of detachments with *C(1)RA*, all of which appear to result from exchange between the interstitial heterochromatin of the compound and the *bb*⁺-bearing short arm of *y*⁺*YL* (Sandler, 1954, DIS 28: 153-54).

***y*⁺*Ymal*⁺1**

constitution: *l(1)IAa*⁺—*ac*⁺ *KL·M(1)I8C*⁺—*su(f)*⁺ *bb*⁺ *KS*.
origin: X-ray-induced euchromatic deletion in *YSX·YL*, *In(1)EN*, *y·y*⁺.
references: Schalet, 1963, DIS 38: 82.

***y*⁺*Ymal*⁺2**

constitution: *l(1)IAa*⁺—*ac*⁺ *KL·l(1)carot2*⁺—*su(f)*⁺ *bb*⁺ *KS*.
origin: Same as *y*⁺*Ymal*⁺1.

references: Schalet, 1963, DIS 38: 82.

other information: A number of X-ray-induced *mal* or *mal*⁺ derivatives partially characterized in table below (Schalet and Finnerty, 1968, DIS 43: 65-66; Chovnick, Finnerty, Schalet, and Duck, 1969, Genetics 62: 145-60).

derivative	characterization
<i>y</i> ⁺ <i>Ymal</i> ¹⁰²	deficient for <i>l(1)carot2—mal</i> ; retains <i>l(1)19Ec</i> ⁺
<i>y</i> ⁺ <i>Ymal</i> ¹⁰⁶	noncomplementing mutation of <i>mal</i>
<i>y</i> ⁺ <i>Ymal</i> ¹⁰⁷	deficient for <i>l(1)carot2—eo</i>
<i>y</i> ⁺ <i>Ymal</i> ¹⁰⁸	deficient for <i>l(1)carot2—unc</i> ; retains <i>lfr</i> ⁺ ; breakpoint at 19E8-F1 (Schalet and Lefevre, 1973, Chromosoma 44: 183-200)
<i>y</i> ⁺ <i>Ymal</i> ¹¹³	deficient for <i>sw—l(1)19Ec</i> ; retains <i>or</i> ⁺ to the left and <i>unc</i> ⁺ to the right
<i>y</i> ⁺ <i>Ymal</i> ¹¹⁶	complementing mutation of <i>mal</i>
<i>y</i> ⁺ <i>Ymal</i> ¹¹⁸	deficient for <i>mal—su(f)</i> ; retains <i>mel</i> ⁺ to the left
<i>y</i> ⁺ <i>Ymal</i> ¹²⁶	deficient for <i>l(1)carot2—l(1)19Ff</i> ; <i>eo</i> ⁺ retained; breakpoint at 19F6-20A1 (Schalet and Lefevre)

***y*⁺*Yv*⁺**

origin: Two X-ray-induced losses of *B^S* from *y*⁺*Yv*⁺*B^S* #1 (Chovnick).
synonym: *B^S·Y*.

y*⁺*Yv*⁺*B^S

constitution: *l(1)IAa*⁺—*ac*⁺ *KL bb*⁺ *KS dy*⁺—*l(1)9Fe*⁺ (*KL?*) *su(f)*⁺—*l(1)20Bb pdf*⁺ *B^S*.
origin: X-ray-induced deletion of the majority of the euchromatin from the recombinant carrying the left end of the *X^PY^D* element of *T(1;Y)124 = T(1;Y)(9F;YL)* and the right end of *XYs·Y1110-8*.
synthesis: Chovnick.
references: Chovnick, 1968, DIS 43: 170.
properties: Three independent isolations; cover *l(1)9Fe* through *v* but not *m* according to Chovnick; #3, however, covers through *dy* but not *fw* (Schalet, 1969, DIS 44: 123). Lethal in combination with *Basc* in males.

***y*⁺*Yy*⁺**

constitution: *l(1)IAa*⁺—*ac*⁺ *KL bb*⁺ *KS ac*⁺—*l(1)IAa*⁺.
origin: X ray induced.
references: Parker and Busby, 1972, Mutat. Res. 16: 49-58.
properties: Carries small duplication, marked with *y*⁺ and *ac*⁺, on the end of each arm. Flies with two *y*⁺*Yy*⁺ chromosomes show a pronounced *Hw* phenotype.

y*²*Y

origin: X-ray- (Lefevre) or γ -ray-induced (Gorelova) deletions of majority of euchromatin from *XYL·YL*, *y*² *su(w^a) w^a*.
synonym: *Dp(1;Y)y*².

chromosome	synthesis	ref ^α	cytology	constitution
<i>y</i> ² Y21T	Gorelova	2, 3	2B6-8	<i>l(1)IAa</i> ⁺ — <i>y</i> ² — <i>hfw KL·KS</i>
<i>y</i> ² Y22T	Gorelova	2	2B9-10	<i>l(1)IAa</i> ⁺ — <i>y</i> ² — <i>frmf KL·KS</i>
<i>y</i> ² Y40T	Gorelova	2	2B9-11	<i>l(1)IAa</i> ⁺ — <i>y</i> ² — <i>frmf KL·KS</i>
<i>y</i> ² Y43T	Gorelova	2	2E1-2	
<i>y</i> ² Y53T	Gorelova	2, 3	2B6-8	<i>l(1)IAa</i> ⁺ — <i>y</i> ² — <i>hfw KL·KS</i>
<i>y</i> ² Y61I	Lefevre	4, 6	1B14	
<i>y</i> ² Y67g19.1'	Lefevre	1, 2, 3, 4, 5	2B17-18; 23A3-4	<i>l(1)IAa</i> ⁺ — <i>y</i> ² — <i>tlc ? KL·KS</i>
<i>y</i> ² Y67g24.2	Lefevre	1, 2, 3, 4	2B6-8;20F	<i>l(1)IAa</i> ⁺ — <i>y</i> ² — <i>hfw KL·KS</i>

^α 1 = Aizenzon and Belyaeva, 1982, DIS 58: 3-7; 2 = Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90; 3 = Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelova, and Zhimulev, 1980, Chromosoma, 81: 281-306; 4 = Craymer and Roy, 1980, DIS 55: 200-04; 5 = Hall and Kankel, 1976, Genetics 83: 517-35; 6 = White, Decelles, and Endow, 1983, Genetics 104: 433-38.

y^{59b}Y

constitution: Carries *l(1)IAa⁺—sta*; Y arm not specified; cytologically the Y carries 1A1-1B1-2.

synthesis: Green.

synonym: *Dp(1;Y)y^{59b}*.

references: Belyaeva, Aizenzon, Semeshin, Kiss, Koczka, Baritcheva, Gorelova, and Zhimulev, 1980, Chromosoma 81: 281-306.

Aizenzon and Belyaeva, 1982, DIS 58: 3-7

Belyaeva, Aizenzon, Kiss, Gorelova, Pak, Umbetova, Kramers, and Zhimulev, 1982, DIS 58: 184-90.

properties: Covers *l(1)IAa-l(1)2Ad*; partially covers *sta* (Aizenzon and Belyaeva, 1982). Marked with *y^{59b}*, which resembles *y¹*.

y^{66d}

constitution: *YSYL·YS, KS bb⁺ y⁺ KL·bb⁺ KS* (Williamson).

origin: Spontaneous derivative of *XYL·YS129-16*.

synthesis: Parker.

references: Parker, 1968, DIS 43: 156.

Williamson, 1973, DIS 50: 102.

properties: Duplicated for *KS* and the nucleolus organizer, the two nucleoli being connected by *YL*. *y scY66d* males are fertile and show more pronounced *y* variegation than *XYL·YS129-16/0* males. *Y66d*, like *XYL·YS129-16*, does not cover *l(1)IAc*; it carries the *y⁺* marker in an interstitial position (Parker, 1968).

YB^S: see **B^SY**

Ybb

This is a group of Y chromosomes that carry insufficient numbers of ribosomal genes to produce wild-type bristle morphology. They are characterized by having 125 or fewer ribosomal cistrons compared to 225 in wild-type Y chromosomes. Since numbers of ribosomal cistrons are unstable and subject to change by magnification or reduction, the estimates of cistron number presented here represent historical observations and need not reflect current compositions. The distinction between chromosomes designated *Ybb⁻* and those designated *Ybb* not always based on objective criteria; however, *Ybb* chromosomes are generally hemizygous viable whereas *Ybb⁻* chromosomes are generally hemizygous lethal [e.g., in *In(1)sc^{4L}sc^{8R}/Ybb⁻*] and therefore have few if any ribosomal cistrons. The original *Ybb⁻* was shown to have a foreshortened short arm; despite this cytological deficiency, however, derivative chromosomes with increased numbers of ribosomal genes have been recovered. Chromosomes designated in both ways are included in the following table.

chromosome	origin	discoverer	ref ^α	phenotype	rDNA gene #
Ybb	spont	Bridges, 1926	3		
Ybb⁻		Schultz, 33k8	3	YS one third normal length	
Ybb^{-1β}		Ritossa	4, 7, 9	hemizygous lethal	100/0/10 ^Y
Ybb^{-2β}		Ritossa	4, 7	hemizygous lethal	66 ^δ

chromosome	origin	discoverer	ref ^α	phenotype	rDNA gene #
Ybb^{-3β}		Ritossa	4, 7	weak <i>bb</i>	125 ^δ
Ybb^{-A1β}		Atwood	2	hemizygous lethal	
Ybb^{-A2β}		Atwood	2, 7	hemizygous lethal	
Ybb^{-S}		Spofford	2	hemizygous semi-lethal	
Ybb^{-Sdf}		Spofford	2	hemizygous lethal	
Ybb^{-T}		Tartof	11	hemizygous lethal	36 ^ε
Ybb^{1ζ}	spont	Ritossa	1	hemizygous lethal	
Ybb^{N2}	spont?		7	weak allele	~100
Ybb^{N3}	spont		7	weak allele	~100
Ybb^{P2}	spont	Tartof	5	moderate to severe allele	50% normal
Ybb^{Rexη}	<i>Rex</i> -induced exchange	Robbins	8, 10	variable-strength alleles	
Ybb^{rv}			7	wild type	>100
Ybb^{Su(var)θ}			5, 7	severe allele	50
Ybb^{ts1}			6	mutant at 18° normal at 29°	117
Ybb^{ts2}			6	mutant at 18° normal at 29°	117
Ybb^X	<i>mei41</i> induced exchange		5	strong allele; <i>Ybb^{+X}</i> derivative	
Ybb^{+X}	<i>mei41</i> induced exchange		5	normal allele; <i>X</i> derived in two steps	

^α 1 = Atwood, 1969, Genetics 61: 319-27; 2 = Baker, 1971, Proc. Nat. Acad. Sci. USA 68: 2472-76; 3 = CP627; 4 = Endow, 1982, Genetics 102: 91-99; 5 = Hawley and Tartof, 1985, Genetics 109: 691-700; 6 = Procnier and Williamson, 1974, Dev. Biol. 39: 198-209; 7 = Ritossa, 1968, Proc. Nat. Acad. Sci. USA 59: 1124-31; 8 = Robbins, 1981, Genetics 99: 443-59; 9 = Spear, 1974, Chromosoma 48: 159-79; 10 = Swanson, 1987, Genetics 115: 271-76; 11 = Tartof, 1973, Genetics 73: 57-71.

^β Derived from the original *Ybb⁻*.

^γ Estimated at 100 non-functional genes (Ritossa), no ribosomal genes (Spear) and 10-12 ribosomal genes, some of them with type 2 repeats (Endow).

^δ Estimates of Ritossa.

^ε Estimate of Tartof.

^ζ Originally lethal with *In(1)sc^{4L}sc^{8R}*, but phenotype can be changed to almost *bb⁺* by magnification.

^η Regularly occurring product of *Rex*-induced mitotic exchange in progeny of crosses of *Rex/+* or *Rex/Rex* females to *YSX·YL/0* males; *X/Ybb^{Rex}* males fertile.

^θ Slight *bb* according to Hawley and Tartof, but strong *bb* according to Ritossa. Suppresses *In(1)w^{m4}* position-effect variegation.

YKpn

constitution: Y carrying the tip of *3R*, presumably *Y^{P3D}* element of a *T(Y;3)*; Y arm not specified.

origin: X ray induced.

synthesis: Falk and Shamay.

synonym: *Dp(3;Y)awd⁺* [= *Dp(3;Y)Kpn⁺*].

references: Lifschytz and Falk, 1969, Genetics 62: 353-58.

properties: Carries *ca⁺* and *Kpn⁺*.

Ymal⁺

constitution: *KL·sw⁺—su(f)⁺ bb⁺ KS*; inferred from origin.

origin: X-ray-induced deletion of majority of euchromatin [*l(1)IAa⁺—car⁺*] from *YSX·YL, In(1)EN*.

synthesis: E. H. Grell.

references: Brosseau, Nicoletti, Grell, and Lindsley, 1961, Genetics 46: 339-46.

other information: Two such chromosomes recovered, only one of which was kept.

Y^cbw⁺: see **R(Y)bw⁺**

Y^{cl}: see *R(YL)*

Y^{CL}:bb⁺: see *R(YL)bb⁺*

Y^{cS}:bw⁺bb⁺: see *R(YS)bw⁺*

YL13

constitution: *KL*.

origin: Spontaneous derivative of *bw⁺Yy⁺*.

synthesis: W. K. Baker.

references: Baker and Spofford, 1959, Univ. Texas Publ. 5914: 135-54.

properties: A large acrocentric chromosome in mitotic metaphase. Lacks *y⁺*, *bw⁺*, and *KS* present in the progenitor chromosome.

YL·sc^{S1}

constitution: *KL·bb⁺ scS1—l(1)IAa⁺*; presence of *bb* tentative.

origin: Recombination between *YS* and distal heterochromatin of *In(1)sc^{S1}*.

chromosome	synonym	ref ^α	cytology
<i>YL·sc^{S1}</i>	<i>sc·YL</i>	1, 4	submetacentric; shorter arm like <i>YL</i>
<i>YL·sc^{S1}2</i>	<i>sc^{S1}·YL#2</i>	3	
<i>YL·sc^{S1}3</i>	<i>sc^{S1} c.o.YEY80</i>	2	resembles <i>Y</i>

^α 1 = Crew and Lamy, 1940, J. Genet. 39: 273-83; 2 = Lindsley, 195; 3 = Parker and McCrone, 1958, Genetics 43: 172-86; 4 = Pontecorvo, 1940, DIS 13: 74.

YL·y⁺

constitution: *KL·(bb⁺) ac⁺—l(1)IAa⁺*.

origin: Spontaneous recombination between *YS* and the distal heterochromatin of *In(1)sc⁸* in males.

references: Lindsley, 1955, Genetics 40: 24-44.

Ritossa, 1973, Proc. Nat. Acad. Sci. USA 70: 1950-54.

properties: One of seven independent cases was deficient for *bb* (Lindsley).

YLC: see *R(YL)*

Yst: Y sterile

constitution: *KL·bb*; in metaphases, the *Y* appears as a rod about the same length as *YL*.

origin: Spontaneous; recovered in a *w^e bb^l* female that was unexpectedly viable, *bb*, and fertile.

synthesis: Bridges, 1926.

references: CP552.

properties: Males with *Yst* sterile in the absence of a *YS* or a normal *Y*. *bb^l/bb^lYst* females fertile and less extreme *bb* than *bb/bb^l* females.

Ysu(f)⁻

constitution: *KL·l(1)20Ac⁺—sw⁺ bb⁺ KS*. The *Y* carries at least one dose of *bb⁺*.

origin: Spontaneous derivative of *Ymal⁺*.

synonym: *Dp(1;Y)su(f)⁻*.

references: Rahman and Lindsley, 1981, DIS 56: 108.

properties: Deficient for *uncl—su(f)*; carries *sw⁺—l(1)20Ac⁺*.

YS8

constitution: *bb⁺ KS*; tentative.

origin: Spontaneous derivative of *bw⁺Yy⁺* recovered from *R(1)l/bw⁺Yy⁺* male.

synthesis: W. K. Baker.

synonym: *YS:bb⁺-8*.

references: Baker and Spofford, 1959, Univ. Texas Publ. 5914: 135-54.

properties: A small, two-armed chromosome in mitotic metaphase. Lacks *y⁺*, *bw⁺*, and *KL* present in the progenitor chromosome.

YSy⁺

constitution: *bb⁺ KS ac⁺—l(1)IAa⁺*.

origin: Derivatives of *bw⁺Yy⁺*.

references: Baker, 1955, DIS 29: 101-12.

Baker and Spofford, 1959, Univ. Texas Publ. 5914: 135-54 (fig.).

derivative	origin	cytology
<i>YSy⁺5</i>	spontaneous in <i>R(1)l/bw⁺Yy⁺</i> male	large acrocentric
<i>YSy⁺6</i>	X ray	metacentric
<i>YSy⁺7</i>	X ray	acrocentric twice length of 4

YS·YS

constitution: *KS bb⁺·bb⁺ KS*.

origin: Three independent cases; spontaneous; *YS·YS2* and *YS·YS3* arose in *XYS/y⁺Y* males.

synonym: *Y''*.

properties: V-shaped chromosome in mitotic metaphase, with both arms the length of *YS*.

chromosome	discoverer	ref ^α
<i>YS·YS1</i>	Stern	2
<i>YS·YS2</i>	Muller	1
<i>YS·YS3</i>	Muller	1

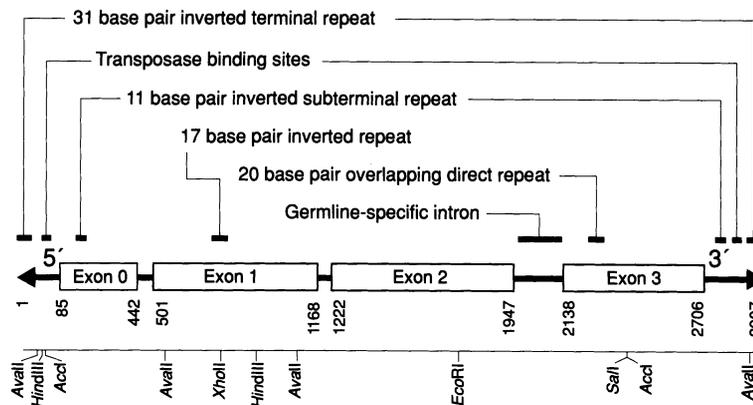
^α 1 = Muller, 1948, DIS 22: 73-74; 2 = Stern, 1929, Z. Indukt. Abstamm. Vererbungsl. 51: 253-353.

AUTOSYNAPTIC CHROMOSOMES

Pericentric-inversion heterozyotes may be recovered and maintained in either of two forms (Craymer, 1981, Genetics 99: 75-97). The conventional heterosynaptic form can be converted to two reciprocal autosynaptic elements by recombination between the inverted segment and a normal homologue. In this sense autosynaptic stocks can be regarded as pericentric inversion heterozyotes in which the two breakpoints have been separated onto homologous centromeres. Craymer termed the complementary products of exchange levosynaptic (*LS*) and dextrosynaptic (*DS*) elements, as the elements carry homologous synapsed regions from the left or the right arm respectively. The levosynaptic element contains two copies of the portion of the autosome to the left of the inversion, one copy of the inversion and no copy of the segment to the right of the inversion; similarly the dextrosynaptic element carries two copies of the region to the right of the inversion, one copy of the inverted region and no copy of the region to the left of the inversion. Heterosynaptic and autosynaptic configurations are interconvertible by single exchange within the inverted segment. In general, reciprocal autosynaptic elements from the same pericentric inversion are carried together; the complex is designated $AS() = LS()/DS()$. Craymer describes a number of methods for generating autosynaptic complexes and indicates their utility in genetic experiments. Gubb, McGill, and Ashburner (1988, Genetics 119: 377-390) have shown how particular autosynaptic stocks can be used to select sets of independently derived *LS* and *DS* elements resulting from interarm translocations between homologous chromosomes; both γ -rays and hybrid-dysgenesis were used to generate such rearrangements. Such an approach selects for autosynaptic chromosomes with breakpoints in the vicinity of those of the selecting $AS()$. In the $LS()$ and $DS()$ chromosomes, those designated with *D* followed by a number were γ ray induced; those with either *P* or *S* are from dysgenic males crossed to autosynaptic-complex-bearing females. Conventions for indicating the allelic content of autosynaptic elements are discussed by Gubb *et al.*

autosynaptic	breakpoint	synthesis	comments
AS(2)bwV1	40F;59D4-E1	Craymer	
AS(2)bwV32g	40F;59E	Craymer	
AS(2)C251	36F;57B	Craymer	
AS(2)ds33k	21C8-D1;60D1-2	Craymer	
AS(2)DTD4	32F;41A-B	Gubb	
AS(2)DTD8	23C-D;41	Gubb	
AS(2)DTD11	28A;41	Gubb	
AS(2)DTD16	23B;41	Gubb	
AS(2)DTD18	23A4-7;41	Gubb	
AS(2)DTD21	23A1-2;41	Gubb	
AS(2)DTD24	26C1-2;41A	Gubb	
AS(2)DTD42	23E3-4;41	Gubb	
AS(2)DTD43	35B1-2;41	Gubb	
AS(2)DTD51	27D1;41	Gubb	
AS(2)DTD52	24D1-2;41	Gubb	
AS(2)DTD86	33B1;41	Gubb	
AS(2)DTD107	32F;41	Gubb	
AS(2)DTD109	25E2-3;41	Gubb	
AS(2)DTD111	29F;41A-B	Gubb	
AS(2)DTD116	26A4-6;41	Gubb	
AS(2)DTD124	24D2-3;41	Gubb	
AS(2)DTD125	31E;41	Gubb	
AS(2)I6	39D3-E1;48F6-49A1	Craymer	
AS(2)Gla	27D;51E		<i>LS(2)</i> deleted and <i>DS(2)</i> duplicated for 22E1-27D

autosynaptic	breakpoint	synthesis	comments
AS(2)IIIG16	40;60E4		
AS(2)noc4	35B1-2;41A	Gubb	
AS(2)px52g	30A;58F	Craymer	
AS(2)S325	21F;41	Gubb	
AS(2)Scorv1	35D1-2;44C4-5	Craymer	
AS(2)Scorv9	35D1-2;41	Craymer	
AS(2)SM1	22A3-B1;60B-C	Craymer	
AS(3)224	69E7-F1;83B7-C1		
AS(3)A114	80;92A2-B1	Craymer	
AS(3)B158	76A;93B	Craymer	
AS(3)bx92	80;89E	Craymer	
AS(3)bx106	72D;89E	Craymer	
AS(3)C190	69F;89D	Craymer	
AS(3)C267	74F;88D	Craymer	
AS(3)C269	78C;98F	Craymer	
AS(3)I7	75A1-2;90A1	Craymer	
AS(3)I19	62A;98A	Craymer	
AS(3)HR33	61A;87B	Craymer	
AS(3)LD1	61C;87B	Craymer	
AS(3)LD3	61F7-62A2;81F5-82A1	Craymer	
AS(3)LD6	62A10-B1;85A2-3	Craymer	
AS(3)O5	62B;88A	Craymer	deficient for 88C-92C; covered by <i>Dp(3;1)O5</i>
AS(3)P10	65C5-9;81	Craymer	
AS(3)P13	63A;81F	Craymer	
AS(3)P21	65A;87F-88A	Craymer	
AS(3)P30	64C;82A	Craymer	
AS(3)P41	64A;88D	Craymer	
AS(3)P42	70F1-2;81F	Craymer	
AS(3)P88	61A1-2;89C1-4	Craymer	
AS(3)P91	67C10-D1;89C2-4	Craymer	
AS(3)P93	64C-E;81F	Craymer	
AS(3)sep	65D2-3;85F2-4	Craymer	
AS(3)Sta	79D;94A	Craymer	
AS(3)Ubx16R	79D;89E	Craymer	
AS(3)Ubx42T	70D;89E	Craymer	
AS(3)UbxU	62A2;89E1-2	Craymer	
DS(2)b81a2	34E3;41	Gubb	
DS(2)CH25	36C;41	Gubb	
DS(2)D2	34D4-5;41B3-9	Gubb	
DS(2)D6	35D5-7;41	Gubb	
DS(2)D32	34F;41	Gubb	
DS(2)P5	34A7-11;41	Gubb	
DS(2)P9	34B7-12;41D	Gubb	
DS(3)TM6			<i>DS(3)P88</i> with <i>TM6</i> inversions
LS(2)b81a2	34D;41D	Gubb	
LS(2)D1	36C;41	Gubb	
LS(2)D3	36D1-2;41	Gubb	
LS(2)D5	36C1-2;42A16-19	Gubb	
LS(2)D7	35F1-2;41	Gubb	
LS(2)D9	35B1-3;41	Gubb	
LS(2)D12	35D1-2;41	Gubb	
LS(2)D15	35C4;41	Gubb	
LS(2)D20	34E4-F2;41	Gubb	
LS(2)P1	35E1-2;41	Gubb	
LS(2)P2	35D5-7;41	Gubb	
LS(2)P3	37B1-2;41C-D	Gubb	
LS(2)P4	25F;35E;41	Gubb	
LS(2)P6	25E-F;28B;35B1-3;41	Gubb	
LS(2)P7	34F5;47A1-2	Gubb	
LS(2)P8	37B;41	Gubb	
LS(2)P10	35B-C;41	Gubb	
LS(2)P11	25E-F;35D;41	Gubb	
LS(2)P12	35E1-2;41	Gubb	
LS(2)P13	35D;41	Gubb	
LS(2)P14	35B;42D1-2	Gubb	
LS(2)P15	37C;47A1-4	Gubb	
LS(2)P16	37D-E;41	Gubb	
LS(2)P17	35F;38C;40;42B	Gubb	
LS(2)P18	36B1-2;41	Gubb	
LS(2)S1	36C;41	Gubb	
LS(2)S2	35D;41	Gubb	
LS(2)S3	26C;35C;42A	Gubb	
LS(2)S4	35B;41	Gubb	
LS(2)S6	36C;42E	Gubb	
LS(2)S8	35D-E;42B	Gubb	

Molecular map of *P* (W. R. Engels)

TRANSPOSABLE ELEMENTS

by: D. J. Finnegan

At least ten percent of the genome of *Drosophila melanogaster* is made up of transposable elements. These are all moderately repeated sequences. There are about fifty families of such elements and these may be divided into groups according to their DNA topologies and putative mechanisms of transposition.

The largest group contains the *copia* -like elements, named after one of the first elements of this type to be discovered. They are similar to retroviral proviruses and have long terminal direct repeats, LTRs, and a long open reading frame that encodes an amino acid sequence related to viral reverse transcriptases. These elements are believed to transpose by a mechanism related to a retroviral life cycle. Eighteen of the elements listed below are of this type (*17.6*, *297*, *1731*, *3S18*, *412*, *BEL*, *blood*, *copia*, *flea*, *gypsy*, *H.M.S. Beagle*, *mdg1*, *mdg2*, *micropia*, *NEB*, *opus*, *roo*, and *springer*).

Six elements, *D*, *F*, *G*, *I*, *Doc*, and *jockey*, are believed to be members of a second class of elements, sometimes called non-viral retrotransposons, that are also believed to transpose by reverse transcription of an RNA intermediate. They have an A-rich sequence at the 3' end of one strand, but no terminal repeats, and encode putative reverse transcriptases. These elements are variable in length and are often truncated at the 5' end of the strand with the A-rich sequence.

P, *pogo*, *hobo*, and *HB* elements have short terminal inverted repeats and are believed to transpose directly from DNA to DNA. The same may be true of *FB* and *BS* elements that have long terminal inverted repeats.

The information given below relates to specific elements, more general discussions can be found in the review by Finnegan and Fawcett [1986, Oxford Surveys of Eukaryotic Genes (N. McClean, ed.). Oxford University Press, Oxford, vol. 3, pp. 1-64] or in articles in "Mobile DNA" [1989, (Berg and Howe, eds.). American Society for Microbiology, Washington]. The restriction maps, terminal sequences and lengths of target-site dupli-

cations are given to aid in the identification of elements encountered during chromosome walks or in the analysis of molecular lesions associated with particular mutations. At least half of all spontaneous mutations in *Drosophila melanogaster* are due to insertions of transposable elements. The terminal sequences shown for an element that is variable in length may not be found at the ends of all copies of that element.

17.6

length: 7.4 kilobases (Saigo *et al.*, 1984).

target site duplication: Four base pairs (Kugimya *et al.*; Inouye *et al.*).

copy number: Forty (Kugimya *et al.*; 1983).

references: Saigo, Millstein, and Thomas, 1981, Cold Spring Harbor Symp. Quant. Biol. 45: 619-28.

Kugimya, Ikenaga, and Saigo, 1983, Proc. Nat. Acad. Sci. USA 80: 3193-97.

Saigo, Kugimya, Matsuo, Inouye, Yoshioka, and Yuki, 1984, Nature 312: 659-61.

Inouye, Yuki, and Saigo, 1986, Nature 310: 332-33.

comments: First described by Saigo *et al.* as a sequence inserted into histone genes and hybridizing to *297* elements. The sequence of the LTR shown here was reported by Kugimya *et al.*. A complete *17.6* element has been sequenced by Saigo *et al.* (1984); the map shown here is derived from this sequence. There are no sites for the enzymes *Ava*I, *Kpn*I, *Sac*I, *Sma*I, or *Xba*I. The sequences of the LTRs of *17.6* and *297* elements are similar. Heteroduplexes between *17.6* and *297* elements show a 1.7 kb region of homology between their right-hand ends (Kugimya *et al.*). All insertions studied so far are associated with duplications of the sequence ATAT.

left end:

AGTGACATAT TCACATACAA AACCCACATAA CATAGAGTAA

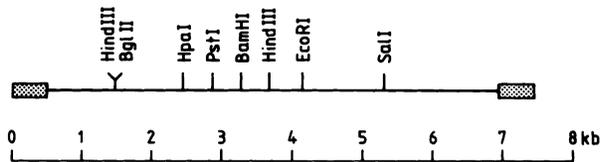
40

right end:

AATAGACTCA AAACATATTTA TTGCAACCAT TTATTGCAA TT

512

restriction map (17.6):



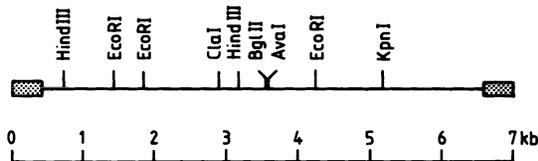
297

length: 7 kilobases (Inouye *et al.*).
target site duplication: Four base pairs (Ikenaga and Saigo; Spradling and Rubin).
copy number: Approximately 30 (Potter *et al.*).
references: Potter, Brorein, Dunsmuir, and Rubin, 1979, Cell 17: 415-27.
 Ikenaga and Saigo, 1981, Proc. Nat. Acad. Sci. USA 79: 4143-47.
 Spradling and Rubin, 1981, Ann. Rev. Genet. 15: 219-64.
 Kugimya, Ikenaga, and Saigo, 1983, Proc. Nat. Acad. Sci. USA 80: 3193-97.
 Inouye, Yuki, and Saigo, 1986a, Eur. J. Biochem. 154: 417-25.
 Inouye, Saigo, Yamada, and Kuchino, 1986b, Nucl. Acids Res. 14: 3031-43.

comments: 297 elements were first described by Potter *et al.* but were originally identified by Wensink and Rubin as being complementary to abundant polyA RNA in tissue-culture cells. The sequence of an LTR shown below was reported by Ikenaga and Saigo. A complete element has been sequenced by Inouye *et al.* (1986a) and the restriction map shown here is based on that sequence. There are no sites for enzymes BamHI, PstI, SalI, SmaI or XhoI. Insertions are preferentially at the sequence ATAT (Spradling and Rubin). The sequences of the LTR's of 297 and 17.6 elements are similar, and heteroduplexes between these elements show a region of 1.7 kb of homology at the right-hand ends of each (Kugimya *et al.*). Inouye *et al.* (1986b) have identified a serine tRNA as the probable primer for reverse transcription.

left end:
 AGTGACGTAT TTGGGTGGAC CAAACCAGCC ACTTCCATTA 40
right end:
 TAGTTCAGAC TCATACATAA AACACAATT TTA 415

restriction map (297):



412

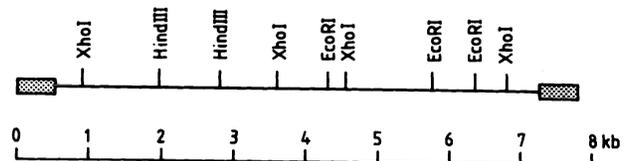
length: 7.6 kilobases (Shepherd and Finnegan).
target site duplication: Four base pairs (Will *et al.*).
copy number: 40 (Will *et al.*).
references: Rubin, Finnegan, and Hogness, 1976, Progress in Nucl. Acid Research. (Cohen and Volkin, eds.). Academic Press, New York, Vol. 19, pp. 221-26
 Finnegan, Rubin, Young, and Hogness, 1978, Cold Spring Harbor Symp. Quant. Biol. 42: 1053-63.
 Will, Bayev, and Finnegan, 1981, J. Mol. Biol. 153: 897-915.
 Shepherd and Finnegan, 1984, J. Mol. Biol. 180: 21-40.

Chang, Wisely, Huang, and Voelker, 1986, Mol. Cell. Biol. 6: 1520-28.
 Yuki, Ishimaru, Inouye, and Saigo, 1986a, Nucl. Acids Res. 14: 3017-29.
 Yuki, Inouye, Ishimaru, and Saigo, 1986b, Eur. J. Biochem. 158: 403-10.
 Micard, Couderc, Sobrier, Girard, and Dastugue, 1988, Nucl. Acids Res. 16: 455-70.

comments: First described as being complementary to abundant poly(A)+ RNA in tissue culture cells (Rubin *et al.*). The sequence of the LTR shown here was reported by Will *et al.*; rare 412 elements have 571 base-pair LTRs, the first 482 base pairs of which correspond to the sequence presented (Will *et al.*). The map shown here is adapted from that published by Shepherd and Finnegan; there are no sites for the enzymes BamHI, SacI, or SalI. The sequence of a complete element has been reported by Yuki *et al.* (1986 a and b). Fourteen of the 18 bases of the putative primer binding sites of 412 and *mdg1* elements are identical, as are 27 bases adjacent to their left-hand LTRs (Will *et al.*). Yuki *et al.* (1986b) have suggested that an arginine tRNA may serve as the primer for reverse transcription of 412 RNAs. Finnegan *et al.* and Micard *et al.* have reported that the majority of 412 transcription is from right to left in tissue culture cells. This would produce antisense RNAs. Prosser and Finnegan have found that both strands of 412 are represented in polyA RNA from adults. Micard *et al.* have shown that the level of 412 transcription in tissue-culture cells is decreased in the presence of 20-hydroxyecdysone. The phenotypes of some mutations associated with 412 insertions are affected by *su(s)* mutations (Yuki *et al.*, 1986a).

left end:
 TGTAGTATGT GCCTATGCAA TATTAAGAAC AATTAATAA 40
right end:
 TTAAAACGGA CTTGTGTTCT GAATTGGAGT TCATCATTAC A 481

restriction map (412):



1731

length: 4.6 kilobases (Peronnet *et al.*).
target site duplication: Five base pairs (Peronnet *et al.*).
copy number: Ten (Peronnet *et al.*).
references: Peronnet, Becker, Becker, d'Auriol, and Best-Belpomme, 1986, Nucl. Acids Res. 14: 9017-33.
 Fourcade-Peronnet, d'Auriol, Becker, Galivert, and Best-Belpomme, 1988, Nucl. Acids Res. 16: 6113-25.
comments: The first 1731 element was identified because its transcription in tissue-culture cells is reduced in the presence of 20-hydroxyecdysone (Peronnet *et al.*). The map, and LTR sequence shown here are taken from Peronnet *et al.*; the sequence of a complete 1731 element has been reported by Fourcade-Peronnet *et al.*; the coding capacity of this element is more closely related to that of *copia* than of longer *copia*-like elements such as 17.6. Reverse transcription of 1731 RNA may be primed by a fragment of the initiator methionine tRNA (Fourcade-Peronnet *et al.*).

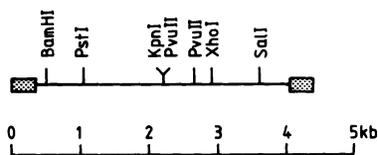
left end:

TGTTGAATAT AGGCAATGCC CACATGTGTG TTGAATATAG 40

right end:

TGTTCCACAC TTGGAGCACC TTTTCAATAA ACAACA 336

restriction map (1731):



3S18

length: 6.5 kilobases (Bell *et al.*, 1985).

target site duplication: Five base pairs (O'Hare *et al.*).

copy number: Approximately 15 (Bell *et al.*, 1985).

references: Fabijanski and Pellegrini, 1982, Nucl. Acids Res. 10: 5979-91.

Mattox and Davidson, 1984, Mol. Cell. Biol. 4: 1343-53.

O'Hare, Murphy, Levis, and Rubin, 1984, J. Mol. Biol. 180: 437-55.

Bell, Bogardus, Schmidt, and Pellegrini, 1985, Nucl. Acids Res. 13: 3861-71.

comments: First identified as an insertion within the non-transcribed spacer of an rDNA repeat (Bell *et al.*; Fabijanski and Pellegrini). Restriction and Southern hybridization data suggest that this element is flanked by direct repeats about 500 base pairs long (Bell *et al.*). A 3S18 probe hybridized to twelve euchromatic sites and the chromocenter of polytene chromosomes from a cross between *gt¹* and *gt^{X11}* strains. The map shown here as been taken from those published by Bell *et al.* and Mattox and Davidson. There are no sites for enzymes BglII, SmaI, or XbaI. The sequences at the ends of the 3S18 and BEL elements are the same suggesting that these elements are closely related if not the same (O'Hare, unpublished). The terminal sequences are from the element associated with the *w^{zm}* mutation (O'Hare *et al.*).

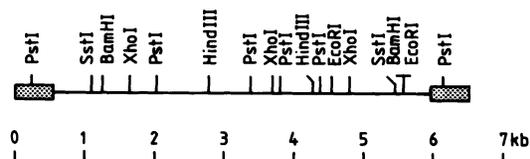
left end:

TGTTTATAAA TAAACCGGCC AGTGTTACGT TTAATTTTCAT

right end:

GTGACTTGTG CGCGTATAACA GGGTGTCTCG TTCCCAAACA

restriction map (3S18):



B104: see *roo*

BEL

length: 7.3 kilobases.

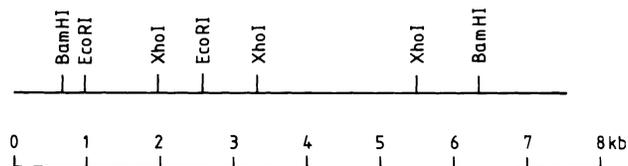
copy number: Twenty five.

references: Goldberg, Sheen, Gehring, and Green, 1983, Proc. Nat. Acad. Sci. USA 80: 5017-21.

comments: First described as an insertion associated with the *w^{a4}* mutation. *In situ* hybridization experiments indicate that BEL elements are located at about twenty five sites throughout the genome, and that their distribution differs from one strain to another. The ends of this element hybridize to each other. Goldberg *et al.* have suggested that it is a *copia*-like elements, although it is not known whether the terminal repeats are direct or

inverted. The sequences at the ends of BEL and 3S18 elements are the same, suggesting that these elements are closely related if not the same (O'Hare, unpublished).

restriction map (BEL):



blood

length: 6 kilobases.

target site duplication: Four base pairs.

copy number: Nine to fifteen.

references: Bingham and Chapman, 1986, EMBO J. 5: 3343-51.

comments: First identified as an insertion associated with the mutation *w^{bl}*. The putative primer binding site is similar to those for *412* and *mdg1* elements, suggesting that the primer may be an arginine tRNA.

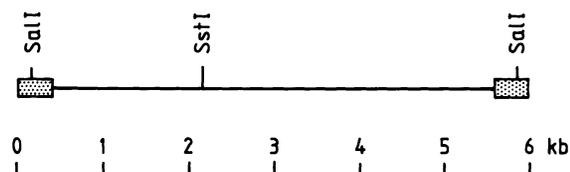
left end:

TGTAGTATGT GCATATATCG AGGGTACACT GTACCTATAA 40

right end:

ACACGTGTTT TCAATTGGTG GCATATATTG GTTTATTACA 400

restriction map (blood):



BS

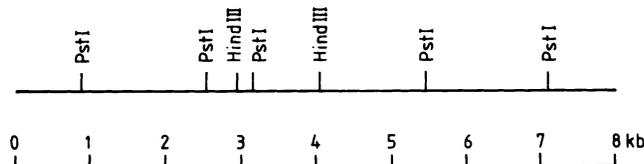
length: 8 kilobases.

copy number: Fifteen.

references: Campuzano, Balcells, Villares, Carramolino, García-Alonzo, and Modolell, 1986, Cell 44: 303-12.

comments: First described as a sequence inserted within the *gypsy* element associated with the *Hw¹* mutation. It has inverted terminal repeats of 2.5 kb. These have an outer domain made up of tandem repeats about 130 base pairs long and a nonrepetitious inner domain. The sequence of the internal domain is well conserved in the genome (Livingstone and Finnegan).

restriction map (BS):



Calypso

length: 7.2 kilobases (Bender).

copy number: Ten to twenty (Bender).

comments: First described as an insertion associated with the *ry³⁰¹* mutation. Three other copies have been cloned; they are identical to the *ry³⁰¹* element as judged by heteroduplex analysis, but have some small variations in their restriction maps.

restriction map (Calypso):



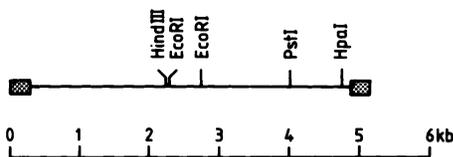
copia

length: 5 kilobases (Emori *et al.*; Mount and Rubin).
target site duplication: Five base pairs (Dunsmuir *et al.*).
copy number: Sixty (Finnegan *et al.*; Potter *et al.*).
references: Finnegan, Rubin, Young, and Hogness, 1978, Cold Spring Harbor Symp. Quant. Biol. 42: 1053-63.
 Potter, Brorien, Dunsmuir, and Rubin, 1979, Cell 17: 415-27.
 Dunsmuir, Brorien, Simon, and Rubin, 1980, Cell 21: 576-79.
 Levis, Dunsmuir, and Rubin, 1980, Cell 21: 581-88.
 Shiba and Saigo, 1983, Nature 302: 119-24.
 Emori, Shiba, Kanaya, Inouye, Yuki, and Saigo, 1985, Nature 315: 773-76.
 Mount and Rubin, 1985, Mol. Cell. Biol. 5: 1630-38.
 Kikuchi, Ando, and Shiba, 1986, Nature 323: 824-26.
 Miller, Rosenbaum, Zbrezna, and Pogo, 1989, Nucl. Acids Res. 17: 2134.
 Sneddon and Flavell, 1989, Nucl. Acids Res. 17: 4025-35.
 Yoshioka, Honma, Zushi, Kondo, Togashi, Miyake, and Shiba, 1990, EMBO J. (in press).

comments: First described by Finnegan *et al.* as a sequence complementary to abundant polyA+ RNA in tissue culture cells. The map is taken from the sequence of Mount and Rubin, and the sequence of the LTR is from Levis *et al.* The sequences of complete *copia* elements have been published by Mount and Rubin and by Emori *et al.* Virus-like particles containing full length *copia* RNAs have been found in tissue culture cells by Shiba and Saigo. The major protein in these particles is translated from a 2 kb spliced mRNA (Yoshioka *et al.*). The sequence of this mRNA has been determined by Miller *et al.* This protein is released from the primary translation product by autocatalytic cleavage (Yoshioka *et al.*). Kikuchi *et al.* have shown that a fragment of the initiator methionine tRNA acts as a primer for reverse transcription of *copia* RNA in these particles. Sequences essential for *copia* expression are located on either side of the major transcriptional start sites (Sneddon and Flavell).

left end:
 TGTTGGAATA TACTATTCAA CCTACAAAAG TAACGTTAAA 40
right end:
 TATTAAGAAA GGAAATATAA ATTATAAATT ACAACA 276

restriction map (copia):



D

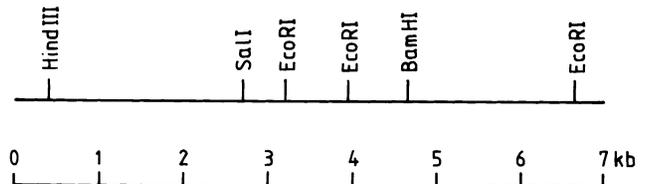
length: Probably variable.
target site duplication: Probably variable.
copy number: 30 to 100 (Pittler and Davis).
references: Pittler and Davis, 1987, Mol. Gen. Genet. 208: 325-28.
comments: Only one *D* element has been reported. This was found at the 3' end of the dunce locus. It was 380 base pairs long, had an A-rich sequence at the 3' end of one strand, and was flanked by fourteen base-pair target-site duplications.

left end:
 TTATTACACC CCAACAGCCT AGCAAGGAAG CTAGGAAGTG 40
right end:
 TGATCAAATA ATAAAAACAT CATCGTAATC GAAAAAATA 380

Delta88

length: 7 kilobases.
references: Karch, Weiffenbach, Peifer, Bender, Duncan, Celnickier, Crosby, and Lewis, 1985, Cell 43: 81-96.
comments: Only one *Delta88* element has been described. This is associated with the *iab8,9^{tuh3}* mutation. It is a moderately repeated element.

restriction map (Delta88):



Doc

length: Variable, up to 5 kilobases (Schneuwly *et al.*).
target site duplication: six to thirteen kilobases (Schneuwly *et al.*; O'Hare *et al.*).
references: Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, Science 221: 23-29.
 O'Hare, Levis, and Rubin, 1983, Proc. Nat. Acad. Sci. USA 80: 6917-21.
 Schneuwly, Kuroiwa, and Gehring, 1987, EMBO J. 6: 201-06.
 Driver, Lacey, Cullingford, Mitchelson and O'Hare, 1989, Mol. Gen. Genet. 220: 49-52.

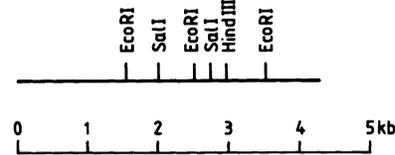
comments: First described as an insertion in the BXC region on a chromosome carrying the *bx³* mutation, although it is not responsible for the mutant phenotype (Bender *et al.*). The map shown here is of this element. Schneuwly *et al.* have found that *Doc* elements lie at both break points of the *Antp^{73b}* inversion; these elements lie in inverted orientation, and the inversion probably resulted from recombination between them. The element reported by O'Hare *et al.* as being associated with the *w¹* mutation is probably a *Doc* element. Driver *et al.* have cloned seven *Doc* elements and have determined the sequences at their termini; their 5' ends are variable, but their 3' ends are conserved.

left end:
 CATTGGGCAT TCCACAGTCT TCGGGTGGAG ACGTGTTCCT
right end:
 ATTCAATAAA TAATAAAAAT TAAAAAATAA AAAAAAATAA

F

length: Variable up to that of consensus element of 4.8 kb (Di Nocera *et al.*).
target site duplication: 8-22 base pairs (Di Nocera *et al.*).

restriction map (Doc):



copy number: Fifty (Di Nocera *et al.*).

references: Dawid, Olong, Di Nocera, and Pardue, 1981, Cell 25: 399-408.

Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, Science 221: 23-29.

Di Nocera, Digan, and Dawid, 1983, J. Mol. Biol. 168: 715-27.

Di Nocera and Casari, 1987, Proc. Nat. Acad. Sci. USA 84: 5843-47.

comments: First described by Dawid *et al.* as an element within a copy of the type I 28S rDNA insertion sequence. The termini shown here are of 101F, the longest *F* element to have been cloned (Di Nocera *et al.*). The map is the consensus for *F* elements (Di Nocera *et al.*). There are no sites for the enzymes ClaI, PvuI, or XhoI. The complete base sequence of a 3.5 kilobase element, *Fw*, has been reported by Di Nocera and Casari. The element *Jiminy*, which was identified within the BXC by Bender *et al.*, is probably an *F* element.

left end:

ATGAAGCATT TCGATCGCCG ACGTGTGAAG ACGTTTTTAT 40

right end:

ATTCAATAAA TAAAAGTAAA GTAAAAA AAAA AAAAAA G 811

restriction map (F):



FB

length: Variable (Truett *et al.*).

target site duplication: Nine base pairs (Truett *et al.*).

copy number: Thirty (Truett *et al.*).

references: Ising and Ramel, 1976, The Genetics and Biology of *Drosophila* (M. Ashburner and E. Novitski, eds.). Academic Press, London, Vol. 1b, pp. 947-54.

Potter, Truett, Phillips, and Maher, 1980, Cell 20: 639-47.

Truett, Jones, and Potter, 1981, Cell 24: 753-63.

Levis and Rubin, 1982, Cell 30: 543-50.

Potter, 1982, Nature 297: 201-204.

Paro, Golberg, and Gehring, 1983, EMBO J. 2: 853-60.

Smyth, Templeton and Potter, 1989, EMBO J. 8: 1887-91.

comments: First described as elements containing inverted repeat sequences (Potter *et al.*, 1980). The inverted repeats at the ends of *FB* elements vary in length; they are made up of different numbers of tandemly repeated sequences, with a maximum repeat length of 155 base pairs (Truett *et al.*; Potter *et al.*, 1982). The DNA between the inverted repeats also varies. The element *FB4* has been sequenced entirely (Potter *et al.*, 1982). Its restriction map and terminal sequences are shown. The only *HinfI* and *TaqI* sites marked, are those that lie in the inverted repeats. There are no sites for the enzymes *AvaI*, *BamHI*, *EcoRI*, *HpaI*, *PstI*, *SacI*, *Sall*, *SmaI*, or

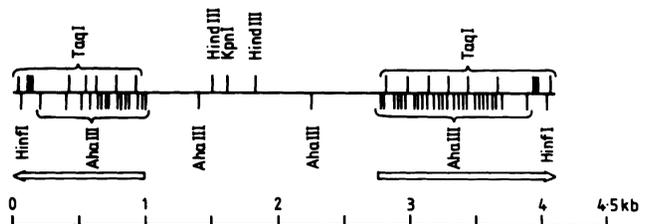
XhoI. Potter *et al.* (1982) have suggested that another transposable element, *HBI*, lies between coordinates 1.1 and 2.75 of *FB4*. The sequence of the central region of an *FB* element related to *FB-w^c*, the element responsible for the *w^c* mutation, has been determined (Smyth, Templeton and Potter). It contains two long open reading frames in one strand, and these authors suggest that they may encode functions required for transposition of *FB* elements. They have evidence that the product of the first open reading frame is a 71 kd polypeptide present in early embryos and egg chambers. *FB* elements have been found at the ends of the *TE* elements (Ising and Ramel; Paro *et al.*). The smallest known element of this type is associated with the mutation *w^{DZL}* (Levis and Rubin).

left end:

AGCTCAAAGA AGCTGGGGTC GGAAAAATCG AATTTTTGAA 40

right end:

TCAAAAATTC GATTTTTCCG ACCCCAGCTT CTTTGAGCT 4089



flea

length: 5.6 kilobases (Kidd and Young).

target site duplication: Six base pairs (Kidd and Young).

references: Kidd, Lockett, and Young, 1983, Cell 34: 421-33.

Kidd and Young, 1986, Nature 323: 89-91.

comments: First described associated with four different *fa* mutations. The map shown here is the reverse of that published by Kidd and Young. There are no sites for the enzyme *BglIII*. These are *copia*-like elements and Kidd and Young have reported sequences from the ends of the LTRs. The elements in the N locus have inserted in target sites within the consensus ATG/GTAT.

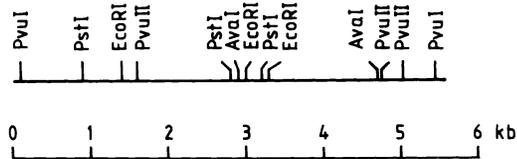
left end:

GTAACATGGA GTAAGGC... 40

right end:

...GCCTTAC TCCATGTTAC

restriction map (flea):



G

length: Variable, up to four kilobases (Di Nocera and Dawid).

target site duplication: Nine base pairs (Di Nocera and Dawid; Di Nocera).

copy number: Ten to twenty (Di Nocera *et al.*).

references: Dawid, Olong, Di Nocera, and Pardue, 1981, Cell 25: 399-408.

Di Nocera and Dawid, 1983, Nucl. Acids Res. 11: 5475-82.

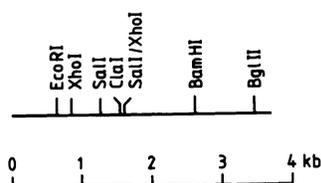
Di Nocera, Graziani, and Lavorgna, 1986, Nucl. Acids Res. 14: 675-91.

Di Nocera, 1988, Nucl. Acids Res. 16: 4041-52.

comments: First described by Di Nocera and Dawid as a sequence inserted within an *F* element. *G* elements have been found in tandem arrays in the non-transcribed spacer sequences of rDNA units. Their chromosomal distribution is fairly stable, as assayed by Southern transfer experiments, and they are concentrated in chromocentric regions (Di Nocera *et al.*). No poly(A)+ RNA complementary to *G* elements has been found in embryos, larvae, pupae, or adults (Dawid *et al.*). The complete base sequence of a 4.3 kb *G* element, *G3A* has been reported by Di Nocera (1988). Its termini are shown here. The map is of the element *G1* (Di Nocera and Dawid). There are no sites for the enzyme *Sma*I.

left end:
 ACAGTCGCGA TCGAACACTC AACGAGTGCA GACGTGCCTA 40
right end:
 TTAATACATA GATCGCTAAA AAAAAAAAAA AAAAAA 4346

restriction map (G):



gypsy

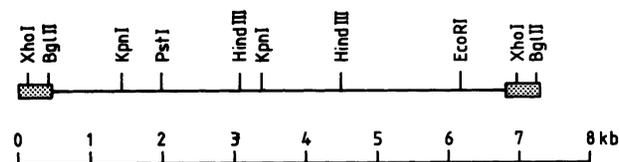
length: 7.3 kilobases (Yuki *et al.*; Marlor *et al.*).
target site duplication: Four base pairs (Kulguskin *et al.*).
copy number: Ten (Bayev *et al.*; Freund and Meselson).
synonym: *mdg4*.
references: Ilyin, Chmeliauskaite, and Georgiev, 1980, Nucl. Acids Res. 8: 3439-57.
 Tchurikov, Ilyin, Skyrabin, Ananiev, Krayev, Zelentsova, Kulguskin, Lyubomirskaya, and Georgiev, 1981, Cold Spring Harbor Symp. Quant. Biol. 45: 655-65.
 Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, Science 221: 23-29.
 Modolell, Bender, and Meselson, 1983, Proc. Nat. Acad. Sci. USA 80: 1678-82.
 Bayev, Lyubomirskaya, Dzhumagliev, Ananiev, Amiantova, and Ilyin, 1984, Nucl. Acids Res. 12: 3707-23.
 Freund and Meselson, 1984, Proc. Nat. Acad. Sci. USA 81: 4462-64.
 Mattox and Davidson, 1984, Mol. Cell. Biol. 4: 1343-53.
 Marlor, Parkhurst, and Corces, 1986, Mol. Cell. Biol. 6: 1129-34.
 Yuki, Inouye, Ishimaru, and Saigo, 1986, Eur. J. Biochem. 158: 403-10.
 Geyer, Green, and Corces, 1988, Proc. Nat. Acad. Sci. USA 85: 8593-97.
 Peifer and Bender, 1988, Proc. Nat. Acad. Sci. USA 85: 9650-54.
 Spana, Harrison, and Corces, 1988, Genes Dev. 2: 1414-23.
 Mazo, Mizrokhi, Karavanov, Sedkov, Krichevskaja, and Ilyin, 1989, EMBO J. 8: 903-11.

comments: First described by Ilyin *et al.* (1980) as *mdg4*, a sequence complementary to double stranded RNA from tissue culture cells, and by Bender *et al.* (1983) as an insertion associated with mutations *bx*³, *bx*^{34e}, *bx*⁵⁵ⁱ, and *bx*^{51j}. Modolell *et al.* have shown by *in situ* hybridization that *gypsy* insertions are associated with many mutations suppressed by *su(Hw)*. The *su(Hw)* product binds to an enhancer-like sequence within *gypsy*, and this

may affect the expression of adjacent genes as well as of *gypsy* itself. This is alleviated by *su(Hw)* mutations (Geyer *et al.*; Peifer *et al.*; Spana *et al.*; Mazo *et al.*). The phenotypes of some mutations caused by *gypsy* insertions are affected by *su(f)* mutations. The LTR sequence shown here was reported by Bayev *et al.*. Freund and Meselson have reported an equivalent sequence of 482 base pairs. The sequences of complete elements have been reported by Yuki *et al.* and Marlor *et al.*. The map shown here is based on those of Bayev *et al.* and Mattox and Davidson. There are no sites for the enzymes *Bam*HI or *Sa*II.

left end:
 AGTTAACAC TAACAATGTA TTGCTTCGTA GCAACTAAGT 40
right end:
 AAATAACATA ACTCTGGACC TATTGGAACT TATATAATT 479

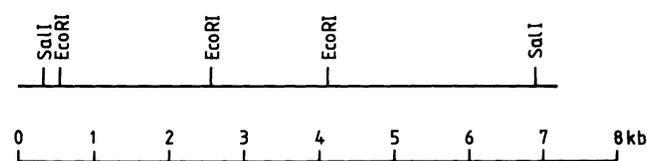
restriction map (gypsy):



Harvey

length: 7.2 kb (Bender).
comments: First described as an insertion associated with the *bx*⁸ mutation. The results of whole genome Southern transfer experiments indicate that this element is repeated within the genome, and that the internal *Sal*I fragment is conserved in length. The ends of the element hybridize to each other.

restriction map (Harvey):



HB

length: 1.6 kilobases (Potter, Brierly, and Potter).
target site duplication: Possibly 8 bp (Potter, Brierly, and Potter).
copy number: Twenty (Brierly and Potter).
references: Potter, 1982, Nature 297: 201-4.
 Brierly and Potter, 1985, Nucl. Acids Res. 13: 485-501.
 Harris, Bailie, and Rose, 1988, Nucl. Acids Res. 16: 5991-98.
 Henikoff and Plasterk, 1988, Nucl. Acids Res. 16: 6234.

comments: The first *HB* element was found as the loop sequence within the *FB* element *FB4* (Potter). Brierly and Potter have shown that *HB* sequences are rarely associated with *FB* sequences and have suggested that they constitute a separate transposable element. The chromosomal distribution of these elements is fairly stable as assayed by Southern transfer experiments. There are 29 bp inverted repeats at the ends of the two elements that have been sequenced, but part of these may comprise target site duplications (Brierly and Potter). If there is an 8 bp target site duplication then the length of the terminal repeat is 20 bp. The sequence of *HB1* contains an open reading frame of 444 bp (Potter). There is about 25% sequence identity between the amino acid sequences

encoded by *HBI* and the *Tcl* transposable element of *Caenorhabditis elegans* (Harris *et al.*; Henikoff and Plasterk). This map is taken from the sequence of *HBI*. There are no sites for the enzymes BamHI, EcoRI, SacI, SalI and XhoI.

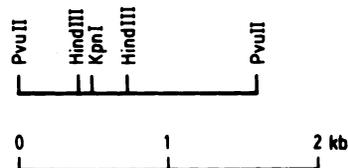
left end:

TACAGCTGTG TTCAGAAAA TAGCAGTGG AAGGAACTA

right end:

TGAAGTCCAA AGCACTGCTA TTATTCTGAA CACAGCTGTA

restriction map (HB):



H.M.S. Beagle

length: 7.3 kb.

target site duplication: Four base pairs.

copy number: 50.

references: Snyder, Kimbrell, Hunkapiller, Hill, Fristrom, and Davidson, 1982, Proc. Nat. Acad. Sci. USA 79: 7430-34.

comments: First described as an insertion within the promoter region of the cuticle protein gene, *Lcp3^{nl}*. The sequence shown here is the reverse complement of that published by Snyder *et al.*. There are no sites for the enzymes Aval, BamHI, HindIII, or KpnI.

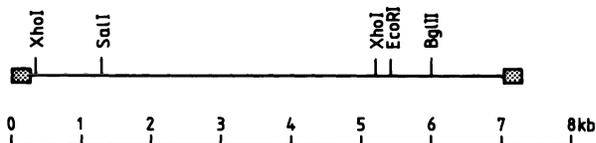
left end:

AGTTATTGCC CTGCAATTGA TTCTCTAACA TCTTGTGGTT 40

right end:

TCTTCAAAAT CAAATCGATA ACTGTAATTA TTAAC 266

restriction map (H.M.S. Beagle):



hobo

length: Variable, up to 3 kb (Streck *et al.*).

target site duplication: Eight base pairs (McGinnis *et al.*; Streck *et al.*).

copy number: 0-50 (McGinnis *et al.*; Streck *et al.*).

references: McGinnis, Shermoen, and Beckendorf, 1983, Nucl. Acids Res. 11: 737-51.

Streck, MacGaffrey, and Beckendorf, 1986, EMBO J. 5: 3615-25.

Yannopoulos, Stamatis, Monastirioti, Haizopoulos, and Louis, 1987, Cell 49: 487-95.

Blackman, Grimaila, Koehler, and Gelbart, 1987, Cell 49: 497-505.

Lim, 1988, Proc. Nat. Acad. Sci. USA 85: 9153-57.

Blackman, Macy, Koehler, Grimaila, and Gelbart, 1989, EMBO J. 8: 211-17.

comments: First described by McGinnis *et al.* as being associated with the *Sgs4* allele in the strain Stromsvreten 8. The sequence and map shown are of *hobo100* (Streck *et al.*). This element contains a single long open reading frame that reads from left to right. Blackman *et al.* have identified a fully functional *hobo* element and have used it to introduce a marked element into the genome. Some

strains appear to lack *hobo* elements, whereas others have 10-50 copies. These are called "E" and "H" strains, respectively (Streck *et al.*). The frequency of *hobo* activity is elevated in some strains and in some cases is increased in the progeny of crosses between E and H strains (Yannopoulos *et al.*; Blackman *et al.*). Lim has evidence that recurring chromosome aberrations that he has found on an unstable X chromosome are due to recombination between *hobo* elements that lie at the breakpoints. The majority of strains derived from natural populations before the mid 1950's harbor few *hobo* homologous sequences. In contrast almost all present-day populations carry numerous *hobo* elements and two specific deletion-derivative elements called *Th1* and *Th2* (Periquet, Hamelin, Bigot, and Lepissier, 1989, J. Evol. Biol. 2: 223-29).

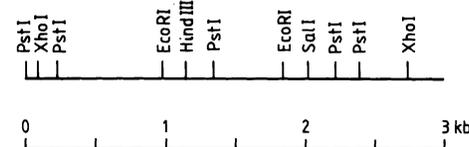
left end:

CAGAGAAGCTG CAGCCCGCCA CTCGCACTCT ACGTCCACCC 40

right end:

TGTGAGTCGA GTGGTAAAA AGTGCCACCC TTGCAGTTCT CTG 1273

restriction map (hobo):



/

length: Variable, up to 5.4 kb.

target site duplication: Variable but usually about twelve base pairs (Fawcett *et al.*).

copy number: 0-10 complete *I* elements plus about 30 incomplete elements (Bucheton *et al.*).

references: Bucheton, Paro, Sang, Pélisson, and Finnegan, 1984, Cell 38: 153-63.

Sang, Pélisson, Bucheton, and Finnegan, 1984, EMBO J. 3: 3079-85.

Fawcett, Lister, Kellett, and Finnegan, 1986, Cell 47: 1007-15.

Crozatier, Vaury, Busseau, Pélisson, and Bucheton, 1988, Nucl. Acids Res. 16: 1999-2013.

Busseau, Pélisson, and Bucheton, 1989a, Nucl. Acids Res. 17: 6939-45.

Busseau, Pélisson, and Bucheton, 1989b, Mol. Gen. Genet. 218: 222-28.

comments: First described by Bucheton *et al.* as insertions associated with *w* gene mutations induced by I-R hybrid dysgenesis. The base sequence of a complete *I* element has been determined by Fawcett *et al.*, and the restriction map shown here is based on this sequence. There are no sites for the enzymes BamHI, EcoRI, SacI, SalI, SmaI, or XhoI. Incomplete *I* elements that have recently inserted in the genome have deleted varying amounts from the 5' end of the sequence of a complete element (Busseau *et al.*, 1989a). Incomplete elements that have been in the genome for a long time are located in pericentromeric regions, and differ from complete elements by many base substitutions and internal or terminal deletions or both (Crozatier *et al.*). Mutations induced by I-R hybrid dysgenesis include apparent point mutations due to insertion of *I* elements and chromosome rearrangements due to recombination between *I* elements (Sang *et al.*; Busseau *et al.*, 1989b).

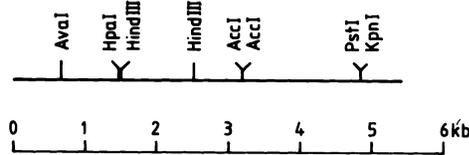
left end:

CATTACCACT TCAACCTCCG AAGAGATAAG TCGTGCCTCT 40

right end:

TAGTTTTGTA AACTATTCTA TCTATCATAA TAATAATAAT AA 5372

restriction map (I):



Isadora

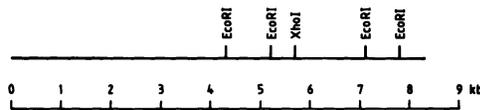
length: 8.3 kilobases.

copy number: 8.

references: Tsubota, Rosenberg, Szostak, Rubin, and Schedl, 1989, *Genetics* 122: 881-90.

comments: Only one *Isadora* element has been studied. It was identified as an insertion at the *Bar* locus of a chromosome carrying the partial revertant of *Bar*, *B*³, and may be responsible for the altered phenotype. It is present at 8 sites on the arms of Oregon R chromosomes but not at the chromocenter. There are no sites for the enzymes BamHI, HindIII, SalI and XhoI.

restriction map (Isadora):



jockey

length: Variable, up to five kilobases (Priimägi *et al.*).

target site duplication: Nine or ten base pairs (Priimägi *et al.*).

copy number: Fifty (Ilyin).

references: Mizrokhi, Obolenkova, Priimägi, Ilyin, Gerasimova, and Georgiev, 1985, *EMBO J.* 4: 3781-87.

Mizrokhi, Georgieva, and Ilyin, 1988, *Cell* 54: 685-91.

Priimägi, Mizrokhi, and Ilyin, 1988, *Gene* 70: 253-62.

comments: First described by Mizrokhi *et al.*, (1985) as an insertion within the *gypsy* element associated with the *ct*^{MR2pN10} mutation. The complete base sequence of a five kilobase element, *jl*, has been reported by Priimägi *et al.* The restriction map of this element and the sequences of its ends are shown here. The genome contains several *jockey* elements that are deleted internally (Priimägi *et al.*; Ilyin). *jockey* elements are transcribed in tissue culture cells using an internal polII promoter (Mizrokhi *et al.*, 1988). The elements *sancho1*, *sancho2*, and *wallaby* appear to be deleted *jockey* elements (Mizrokhi *et al.*, 1988; Corces).

left end:

AAAAATCATT CACATGGGAG ATGAGCAATC GAGTGGACGT 40

right end:

GCGTTGATCA AATAATAAAA ACATCATAAA AAAAAAAAAA 5020

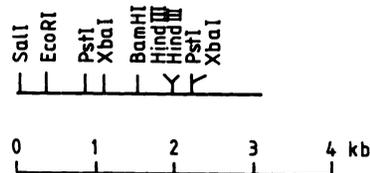
Kermit

length: 4.8 kilobases.

copy number: Thirty.

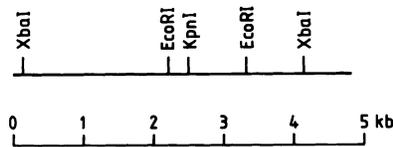
references: Bender, Akam, Karch, Beachy, Peifer, Spierer, Lewis, and Hogness, 1983, *Science* 221: 23-29.

restriction map (jockey):



comments: Only one copy of this element has been described; it was found within the 87E1-6 region in Canton S but not Oregon R DNA. It has been mapped by Bender. There are no sites for the enzymes BamHI, HindIII, or SalI, and there may be additional sites for EcoRI at the ends of the element.

restriction map (Kermit):



mdg1

length: 7.3 kilobases (Ilyin *et al.*).

target site duplication: Four base pairs (Kulguskin *et al.*).

copy number: 25 (Ilyin *et al.*).

references: Georgiev, Ilyin, Ryskov, Tchurikov, and Yenikolopov, 1977, *Science* 195: 394-97.

Georgiev, 1978, *Cold Spring Harbor Symp. Quant. Biol.* 42: 959-69.

Ilyin, Chmeliauskaite, and Georgiev, 1980, *Nucl. Acids Res.* 8: 3439-57.

Ilyin, Chmeliauskaite, Kulguskin, and Georgiev, 1980, *Nucl. Acids Res.* 8: 5347-61.

Kulguskin, Ilyin, and Georgiev, 1981, *Nucl. Acids Res.* 9: 3451-63.

Will, Bayev, and Finnegan, 1981, *J. Mol. Biol.* 153: 897-915.

Yuki, Inouye, Ishimaru, and Saigo, 1986, *Eur. J. Biochem.* 158: 403-10.

comments: First described by Ilyin *et al.* (1978) and Georgiev *et al.* (1978) as being complementary to abundant poly(A)⁺ RNA. The sequence of the LTR shown here is the reverse complement of that published by Kulguskin *et al.*, and the map is the reverse of that published by Ilyin *et al.* (1978). The direction of major transcription is left to right (Ilyin *et al.*). Fourteen of the eighteen bases of the putative primer binding sites of *mdg1* and *412* elements are identical, as are the 27 bases adjacent to their left-hand LTRs (Will *et al.*). Yuki *et al.* have identified an arginine tRNA as being the probable primer for reverse transcription of both *mdg1* and *412* RNAs.

left end:

TGTAGTTAAT TTGAATTCTA ATACTTCTGA TGTAGTTAAT 40

right end:

ATTATTGTTA TTATTATTGT TATTATATTC GTATATACTA CA 442

restriction map (mdg1):



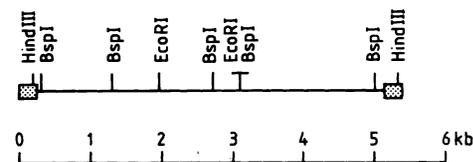
mdg3

length: 5.4 kilobases (Ilyin *et al.*, 1980a).
target site duplication: Four base pairs (Bayev; Mossie *et al.*).
copy number: 15 (Ilyin *et al.*, 1980a).
references: Bayev, Krayev, Lyubomirskaya, Ilyin, Skryabin, and Georgiev, 1980, Nucl. Acids Res. 8: 3263-3273
 Ilyin, Chmeliauskaite, Ananiev, and Georgiev, 1980a, Chromosoma 81: 27-53.
 Ilyin, Chmeliauskaite, Kulguskin, and Georgiev, 1980b, Nucl. Acids Res. 8: 5347-61.
 Mossie, Young, and Varmus, 1985, J. Mol. Biol. 182: 31-43.

comments: First described by Ilyin *et al.* (1980a and b) as being complementary to double-stranded RNA from tissue culture cells. The sequence of the LTR shown here is the reverse complement of that published by Bayev *et al.* (1980); this was originally thought to be 268 base pairs but was subsequently found to be 267 base pairs (Bayev; Mossie *et al.*). The reported direction of major transcription is right to left (Ilyin, 1980a); the map shown is taken from Ilyin (1980a); there are no sites for the enzyme BamHI.

left end:
 TGTAGTAGGC TGCTCCTTCT ACCCTCTTCC TTTACTCTTA 40
right end:
 TGTATTAGAA TATTAACCTC TGTAACCGGC GCCTAAA 267

restriction map (mdg3):



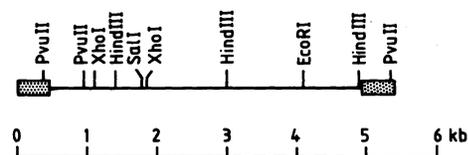
mdg4: see *gypsy*

micropia

length: 5.5 kilobases.
target site duplication: Four base pairs.
references: Lankenau, Huijser, Jensen, Miedma, and Hennig, 1989, J. Mol. Biol. 204: 233-46.
comments: First identified because it hybridizes with a *copia*-like element found on the Y chromosome of *Drosophila hydei*. Only one element from *D. melanogaster* has been studied in detail. The complete sequence of this element has been reported by Lankenau *et al.*; the map is taken from this sequence.

left end:
 TGTCGTGGCG AAAATAATGA GTATGCGTGT AGTCGCTGTT 40
right end:
 GACGGACGCG AGGCCCTGA TATTCTTAAC CCGACA 476

restriction map (micropia):



NEB

length: 5.5 kilobases.
references: Paro, Goldberg, and Gehring, 1983, EMBO J. 2: 853-60..
comments: Only one copy of the *NEB* element has been described. It was found on the large transposable element *TE987A* near the end carrying the *rst* gene. The ends of this element cross hybridize. No inverted repeats could be detected by electron microscopy, suggesting that *NEB* is a *copia*-like element. *NEB* elements occur at multiple dispersed sites in the genome and are located at different positions in different strains. An incomplete element has been found near one end of *TE77*. There are no sites for the enzymes BamHI or Xhpl.

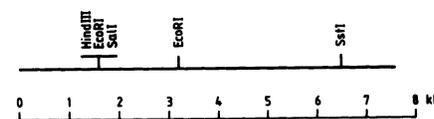
restriction map (NEB):



Nijinski

length: 7.6 kilobases.
copy number: More than 20.
references: Tsubota, Rosenberg, Szostak, Rubin, and Schedl, 1989, Genetics 122: 881-90.
comments: Only one *Nijinsky* element has been studied. It was identified as an insertion at the *Bar* locus of the *Basc* chromosome. It is present at about 20 sites on the arms of Oregon R chromosomes and at the chromocenter. There are no sites for the enzymes BamHI and XhoI.

restriction map (Nijinski):

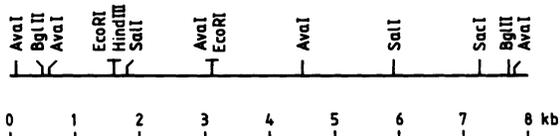


opus

length: 8 kilobases.
target site duplication: Probably six kb.
references: Kidd and Young, 1986, Nature 323: 89-91.
comments: First identified as insertion associated with the mutation *fa*. This is a *copia*-like element. The map and sequences from the ends of the LTRs were reported by Kidd and Young. The only element studied so far was flanked by the sequence TATATA. This is probably the target site duplication, although it could be part of the inverted repeats at the ends of the LTRs.

left end:
 AGTTCACCTT GCATCAGGGT TCTCG..... 40
right end:
AAGAAGAGG GTTCTTAACT

restriction map (opus):



P (W.R. Engels)

length: 2907 base pairs or fewer (O'Hare and Rubin). Longer elements can be produced *in vitro*.

structure: Perfect terminal repeats of 31 base pairs plus several internal repeat structures. Defective *P*-elements are derived from the complete 2907 base pair sequence by internal deletions of various sizes and positions (O'Hare and Rubin). Approximately 150 base pairs at each terminus are usually intact, and are thought to be needed for mobility (Mullins *et al.*).

target site duplication: Eight base pairs (O'Hare and Rubin).

copy number and distribution: (Bingham *et al.*; Kidwell; Anxolabéhère *et al.*) The older laboratory strains, dating from 1950 or earlier, have no *P* homologous sequences, and they are called "M strains." Most natural populations in North and South America and Africa are "P strains", *i.e.*, they have multiple copies of both complete and defective *P*-elements in scattered and highly variable genomic positions. The total copy number is usually 30-50. Most European and Asian populations are "M' strains," meaning that they have mostly defective *P*-elements and a different kind of regulation. The total copy number tends to be less than P strains. Australia has both P and M' strains.

references: Kidwell, Kidwell, and Sved, 1977, *Genetics* 86: 813-33.

Bingham, Kidwell, and Rubin, 1982, *Cell* 29: 995-1004.

Rubin and Spradling, 1982, *Science* 218: 348-53.

Rubin, Kidwell, and Bingham, 1982, *Cell* 29: 987-94.

Kidwell, 1983, *Proc. Nat. Acad. Sci. USA* 80: 1655-59.

O'Hare and Rubin, 1983, *Cell* 34: 25-35.

O'Hare, Levis, and Rubin, 1983, *Proc. Nat. Acad. Sci. USA* 80: 6917-21.

Engels and Preston, 1984, *Genetics* 107: 657-78.

Karess and Rubin, 1984, *Cell* 38: 135-46.

Laski, Rio, and Rubin, 1986, *Cell* 44: 7-19.

Rio, Laski, and Rubin, 1986, *Cell* 44: 21-32.

Engels, Benz, Preston, Graham, Phillis, and Robertson, 1987, *Genetics* 117: 745-57.

O'Kane and Gehring, 1987, *Proc. Nat. Acad. Sci. USA* 84: 9123-27.

Simmons, Raymond, Boedigheimer, and Zunt, 1987, *Genetics* 117: 671-85.

Anxolabéhère, Kidwell, and Périquet, 1988, *Mol. Biol. Evol.* 5: 252-69.

Cooley, Kelley, and Spradling, 1988, *Science* 239: 1121-28.

Robertson, Preston, Phillis, Johnson-Schlitz, Benz, and Engels, 1988, *Genetics* 118: 461-70.

Roiha, Rubin, and O'Hare, 1988, *Genetics* 119: 75-83.

Engels, 1989, *Mobile DNA* (Berg and Howe, eds.). American Society for Microbiology, Washington, pp. 437-84.

Mullins, Rio, and Rubin, 1989, *Genes Dev.* 3: 729-38.

Sved, Eggleston, and Engels, 1989, *Genetics* (in press).

transposase: Exons 0-3 (see map) encode an 87 kilodalton protein needed for both transposition and excision. The 2-3 splice is germline-specific, but this intron can be artificially removed to yield transposase in somatic cells (Karess and Rubin; Rio *et al.*; Laski *et al.*).

insertion sites: *P*-elements insert at random, but their distribution is not uniform. There is a strong preference for euchromatin and the 5' untranslated regions of genes.

They also tend to insert adjacent to other *P*-elements and have a slight preference for target sequences resembling GGCCAGAC. Precise insertional hotspots have been seen in several genes, suggesting that there are additional specificities not yet characterized (O'Hare and Rubin; Engels).

regulation: (Engels) *P* strains and *M'* strains have relatively little transposition and excision activity, and several regulatory mechanisms are thought to be involved. One of these, called the *P* "cytotype," is found only in *P* strains and has a partial maternal inheritance resulting in relative stability in *P*-elements in the progeny of *P* strain females crossed to *M* strain males. However, the elements are active in progeny from the reciprocal cross. Certain defective *P*-elements have been shown to encode a negative regulator of *P*-element activity.

hybrid dysgenesis: Mobilization of *P*-elements results in a syndrome of abnormal traits called hybrid dysgenesis (Kidwell *et al.*; Engels). This syndrome includes high frequencies of chromosome rearrangements and male recombination, both of which occur preferentially at the insertion sites of *P*-elements (Engels and Preston; Sved *et al.*). Elevated mutation rates result from insertion mutations and other genomic changes. At high temperatures there is considerable cell death either in the germline or in somatic tissues depending on the transposase source (Simmons *et al.*; Engels *et al.*).

use as transformation vectors: When *P*-elements are injected into *M* strain embryos in the presence of transposase, they will jump from the injected DNA into chromosomal locations, carrying along any sequence that has been inserted into the internal portion of the element (Rubin and Spradling). Genes transformed in this way usually display approximately normal expression and regulation. However, there is usually some position effect, the degree of which depends on the particular transformed sequence. The transposase source can be a gene coinjected with the *P* vector such as the "wings clipped" element (Karess and Rubin) or a stable genomic source such as P[ry⁺Δ2-3](99B) (Robertson *et al.*).

use in mutagenesis: Selecting *P*-element insertion mutations is useful for cloning genes through "transposon tagging" and for generating variability. The most effective approach is to mobilize defective elements with an immobile transposase source (Robertson *et al.*). The mobilized elements can be either naturally occurring defective *P*-elements or marked elements introduced by transformation (Cooley *et al.*).

use as 'enhancer traps': (O'Kane and Gehring) Genes with specific expression patterns can be identified by mobilization of a *P*-element carrying the β-galactosidase gene fused to the transposase promoter. The spatial and developmental pattern of β-galactosidase expression appears to depend on the surrounding sequences.

left end:

CATGATGAAA TAACATAAGG TGGTCCCGTC GAAAGCCGAA 40

right end:

TCTTGGCCGAC GGGACCACCT TATGTTATTT CATCATG 2907

pogo

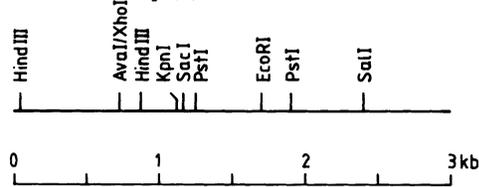
length: Variable, up to at least 2.2 kb (O'Hare).

target site duplication: 0 or 2 base pairs (O'Hare).

references: O'Hare (unpublished).

comments: First copy to be analyzed was a 190 base pair

restriction map (P):



element within the *w¹* insertion on chromosomes carrying the white-eosin mutation. Other copies have been cloned, the longest of which is 1.1 kb. Southern transfer experiments suggest that the genome contains elements of 2.2 kb. Each element has either a 23 base pair terminal inverted repeat and no target site duplication or a 21 base pair inverted repeat flanked by duplication of the sequence TA. The map and terminal sequences are from O'Hare.

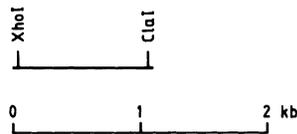
left end:

TACAGTATAA TTCGCTTAGC TGCCTCGAGT ACTTTGCACA 40

right end:

TGCAGCTAAC TATCGATGCA GCTAAGCGAA TTATACTGTA

restriction map (pogo):



roo

length: 8.7 kilobases (Scherer *et al.*, 1982).
target sites duplication: Five base pairs (Scherer *et al.*, 1982).
copy: Eighty (Scherer *et al.*, 1982).
synonym: *B104*.
references: Scherer, Telford, Baldari, and Pirrotta, 1981, *Dev. Biol.* 86: 438-47.
 Meyerowitz and Hogness, 1982, *Cell* 28: 165-76.
 Scherer, Tschudi, Perera, Delius, and Pirrotta, 1982, *J. Mol. Biol.* 157: 435-52.
 Swaroop, Paco-Larsen, and Garen, 1985, *Proc. Nat. Acad. Sci. USA* 82: 1751-55.
comments: Described as *B104* by Scherer *et al.* (1981; 1982) and as *roo* by Meyerowitz and Hogness. *B104* elements were found because they are complementary to abundant poly(A)⁺ RNA in embryos (Scherer *et al.*, 1981), whereas a *roo* element was found inserted near the *Sgs3* gene (Meyerowitz and Hogness). The LTR sequence shown here was reported by Scherer *et al.* (1982), and the map is adapted from those of Scherer *et al.* (1982) and Swaroop *et al.*

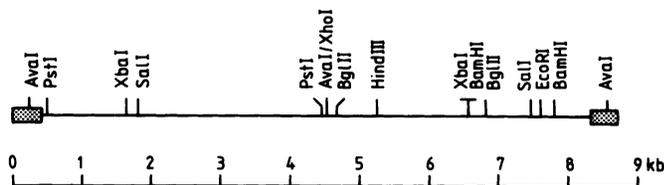
left end:

TGTTACACAGA TGAACACGAA TATATTTAAA GACTTACAAT 40

right end:

AAACTCAACG AGTAAAGTCT TCTTATTGG GATTTTACA 429

restriction map (roo):



sancho1

length: 4.5 kb (Campuzano *et al.*).
copy number: Fifty (Campuzano *et al.*).
references: Campuzano, Balcells, Villares, Carramolino, García-Alonzo, and Modolell, 1986, *Cell* 44: 303-312.
 Mizrokhi, Georgieva, and Ilyin, 1988, *Cell* 54: 685-91.
comments: First described by Campuzano *et al.* as an insertion in a chromosome carrying the *sc^{DI}*, although it is probably not responsible for the mutant phenotype. The restriction map of *sancho1* suggests that it is a deleted *jockey* element (Mizrokhi *et al.*). *sancho1* hybridizes with *sancho2*. The 1.45 kb HindIII fragment is repeated about 50 times in the genome of strains Oregon R, Canton S, and Vallecas (Campuzano *et al.*).

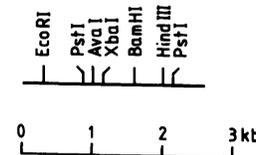
restriction map (sancho1):



sancho2

length: 2.7 kilobases.
copy number: Thirty (Campuzano *et al.*).
references: Campuzano, Balcells, Villares, Carramolino, García-Alonzo, and Modolell, 1986, *Cell* 44: 303-312.
 Mizrokhi, Georgieva, and Ilyin, 1988, *Cell* 54: 685-91.
comments: First described as an insertion on a chromosome carrying the *Hw¹* mutation, although it probably does not contribute to the mutant phenotype. The restriction map of *sancho2* suggests that it is a deleted *jockey* element (Mizrokhi *et al.*). The 1.7 EcoRI-HindIII fragment is repeated about 30 times in the genome of strains of Oregon R and Vallecas. *sancho2* hybridizes with *sancho1* (Campuzano *et al.*).

restriction map (sancho2):



springer

length: 8.8 kilobases (Karlik and Fyrberg).
target site duplication: Probably six base pairs.
copy number: Six (Karlik and Fyrberg).
references: Karlik and Fyrberg, 1985, *Cell* 41: 57-66.
 Kidd and Young, 1986, *Nature* 323: 89-91.
comments: First described by Karlik and Fyrberg as an insertion in the gene for an indirect-flight-muscle-specific tropomyosin isoform associated with the mutation *Tml*. The map and sequence of an LTR were reported by Karlik and Fyrberg. There are no sites for the enzyme BamHI. This element and a copy associated with the mutation *fa³* are both flanked by the sequence TATATA; this is probably the target site duplication, although it could be part of the inverted repeats at the end of the LTR.

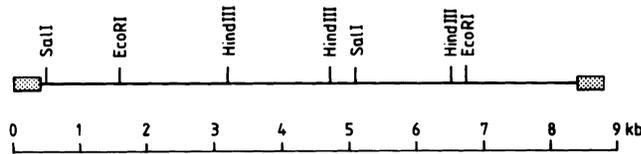
left end:

AATTAATTAA ATGTATGGTG CAGGTCCTC GCCCGGCTCT 40

right end:

TGTGCGGACG ATCAGTCCGG TTAACCTAGT TAACT 405

restriction map (springer):



wallaby

references: Geyer, Green, and Corces, 1988, Proc. Nat. Acad. Sci. USA 85: 3938-42.

comments: This element is associated with a phenotypic revertant of the y^m mutation. It has inserted into the *gypsy* element associated with y^m . The sequence of its termini and its restriction map indicate that it is a *jockey* element.

TE

The transposable elements (*TE*) first investigated by Ising carry the loci of *w* and *rst* flanked by foldback element sequences. The original transposition was from the normal location of *w rst* to 48F on 2R; secondary and tertiary transpositions were located both by recombination and cytologically. The following table is extracted and modified from one by Ising and Block (1980, Cold Spring Harbor Symp. Quant. Biol. 45: 527-44). Here and throughout this volume we designate those transposed elements whose cytological positions have been determined according to those determinations; where the cytology has not been done we retain the original numerical designations but with a # sign to differentiate them from cytological positions.

symbol	location	origin	synonym	homozygote
TE#3	3-45.3	TE48F		viable
TE#5	2-85	TE48F		lethal
TE#14	2-78.9	TE48F		lethal
TE#21	3-69.7	TE48F		lethal
TE#22	3-11.8	TE48F		viable
TE#24	3-42.7	TE48F		viable
TE#37	2-53.0	TE48F		viable
TE#40	3-20.1	TE16B		viable
TE#41	3-87.9	TE48F		viable
TE#44	3-46.8	TE48F		viable
TE#46	3-10.4	TE48F		viable
TE#53	2-56.3	TE48F		viable
TE#63	2-80.6	TE48F		lethal
TE#64	2-58.8	TE48F		viable
TE#68	3-47.3	TE16B		viable
TE#73	2-54.2	TE48F		viable
TE#77	3-58.7	TE48F		viable
TE#91	2-79	TE#5		viable
TE3C	1-1.5	TE48F	TE8	viable
TE7A	1-19.8	TE21EF	TE100	viable
TE16B	1-57.2	[TE#7]	TE6	viable
TE21A	2-0.1	TE21Bb	TE75	lethal
TE21Ba	2-0.1	TE60F	TE133	lethal
TE21Bb	2-0.2	TE48F?	TE30	lethal
TE21C	2-0.4	TE21Bb	TE99	viable
TE21Da	2-0.7	TE7A	TE186	viable
TE21Db	2-0.9	TE57Eb	TE141	lethal
TE21EF	2-0.8	TE48F	TE61	lethal
TE21F22A	2-2.4	TE48F	TE90	viable
TE22A	2-2.3	TE21Bb	TE55	viable
TE22Ba	2-1.5	TE21Bb	TE56	viable

symbol	location	origin	synonym	homozygote
TE22Bb	2-2.4	TE48F	TE114	viable
TE22Bc	2-2.6	TE21Bb	TE57	viable
TE22Bd	2-2.8	TE21Bb	TE81	lethal
TE23CD	2-6.7	TE48F	TE52	viable
TE24D	2-11.7	TE48F	TE17	viable
TE25D	2-19.5	TE48F	TE9	lethal
TE28A	2-26.4	TE48F	TE62	viable
TE28C	2-28.5	TE34Cb	TE49	viable
TE28D	2-32.9	TE21Bb	TE80	lethal
TE29Aa	2-31.0	TE60F	TE128	lethal
TE29Ab	2-31.5	TE48F	TE109	viable
TE29F	2-33.4	TE48F	TE35	lethal
TE30C	2-35.7	TE16B	TE16	viable
TE31A			TE301	?
TE33B	2-44	TE34Cb	TE150	?
TE33E34A	2-47.4	TE48F	TE59	viable
TE34Ca	2-48.3	TE48F	TE60	lethal
TE34Cb	2-48.3	TE48F	TE13	lethal
TE34Cc	2-48.3	TE21Bb	TE94	lethal
TE35A	2-49.1	TE60B	TE146	viable
TE35BC	2-49.8	TE48F	TE36	lethal
TE36A	2-50.6	TE48F	TE116	lethal
TE37C	2-53.3	TE48F	TE42	viable
TE38A	2-54.0	TE#22?	TE48	viable
TE44C	2-57.9	TE48F	TE78	viable
TE47AB	2-60.0	TE48F	TE65	viable
TE47BC	2-60.1	TE48F	TE58	viable
TE47C	2-60.1	TE16B	TE96	viable
TE47EFa	2-61.5	TE48F	TE119	viable
TE47EFb	2-62.0	TE48F	TE126	viable
TE48F	2-58.9		TE1	lethal
TE51D	2-73.6	TE48F	TE19	lethal
TE54B	2-79.8	TE48F	TE45	viable
TE56B	2-86.7	TE48F	TE124	viable
TE57Ea	2-95.2	TE48F	TE33	viable
TE57Eb	2-97.1	TE48F	TE10	lethal
TE60B	2-106.7	TE48F	TE47	viable
TE60F	2-108.5	TE21Bb	TE93	lethal
TE61A	3-1.0	[TE#2?]	TE23	viable
TE61D	3-0.0	TE48F	TE51	viable
TE62DE	3-6.0	TE48F	TE26	lethal
TE65CD	3-22.7	TE48F	TE120	viable
TE65D	3-19.9	TE48F	TE67	lethal
TE67Ea	3-29.8	TE16B?	TE31	viable
TE67Eb	3-33.8	TE48F	TE66	viable
TE69A	3-38.0	TE48F	TE32	viable
TE70F	3-41.7	TE48F	TE38	viable
TE73C	3-44.3	TE48F	TE43	viable
TE76EF	3-46.6	TE48F	TE4	viable
TE86A	3-48.5	TE48F	TE27	viable
TE86EF	3-50.3	TE3C	TE28	viable
TE87A	3-50.6	TE48F	TE98	lethal
TE88A	3-53.0	TE48F	TE39	viable
TE88Ba	3-53.5	TE48F	TE71	lethal
TE88Bb	3-54.1	TE48F	TE72	lethal
TE88Bc	3-55.5	TE48F	TE34	viable
TE89D	3-58.7	TE48F	TE77	viable
TE91E	3-66.8	TE16B	TE70	viable
TE94A	3-74.3	TE21Bb	TE54	viable
TE96D	3-87.6	TE48F	TE48F	viable
TE96E	3-88.4	TE16B	TE20	lethal
TE97D	3-91.3	TE21C	TE137	viable
TE98Ba	3-93.9	TE62DE?	TE29	viable
TE98Bb	3-94.0	TE48F	TE15	viable
TE98F	3-97.1	TE48F	TE89	viable
TE100A	3-102.4	TE48F	TE25	viable
TE100D	3-102.9	TE16B	TE69	viable
TE100E	3-103.0	TE21Bb	TE84	lethal
TEh10-14	Y	TE48F	TE50	viable

DEPARTURES FROM DIPLOIDY

The diploid chromosome complement of *Drosophila melanogaster* may be designated *X/X; 2/2; 3/3; 4/4* for females and *X/Y; 2/2; 3/3; 4/4* for males. Addition to or subtraction from either of these complements of one or more chromosomes produces a departure from diploidy. The non-diploid constitutions are designated by a name but not a symbol except as included in the name, e.g., *X/O* male. Constitutions are described by listing their component chromosomes, homologous chromosomes being separated by slash bars and nonhomologous chromosomes by semicolons. When two homologous chromosomes are attached to the same centromere, components are listed without separation, e.g., *XX, XY*, and *44*.

diploid metafemale

constitution: *X/X/X; 2/2; 3/3; 4/4*; sex chromosome constitution may also be *XX/X*.

source: Produced by triploid and compound-*X*-bearing females. May result from two-*X* gametes produced by nondisjunction.

discoverer: Bridges.

synonym: *superfemale*.

references: 1921, *Science* 54: 252-54.

1922, *Am. Nat.* 56: 51-63 (fig.).

1925, *Am. Nat.* 59: 127-37.

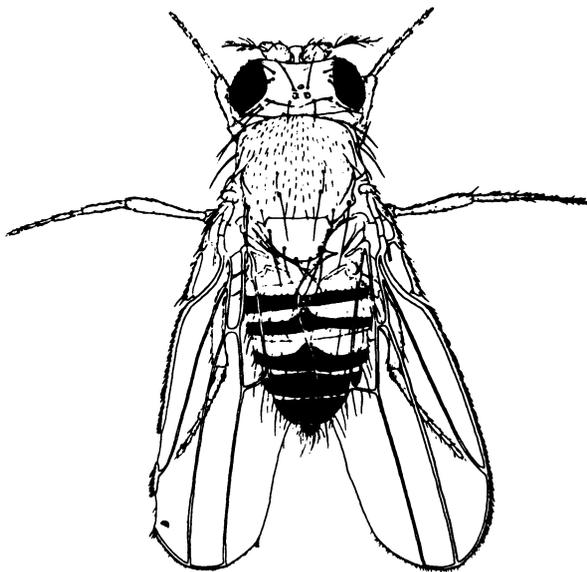
Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 153-62 (fig.).

properties: Wings crumpled or incised on inner margin.

Rear legs often malformed. Viability low, usually less than 0.5%. Flies die mostly in late larval and pupal stages; at 25°C, puparium formation delayed 1-2 days (Brehme, 1937, *Proc. Soc. Exp. Biol. Med.* 37: 578-80). Survival increased by rearing at 20° instead of 25°; five days after oviposition most sensitive (Neeley, 1968, *DIS* 43: 83; 1969, *Genetics* 61: s43). Survivors sterile; two fertile metafemales were apparently mosaic for triploid tissue [Rolfes and Hollander, 1961, *J. Heredity* 52: 61-66 (fig.)]. Larval ovaries from metafemales transplanted into sterile diploids have produced a few progeny (Beadle and Ephrussi, 1937, *Proc. Nat. Acad. Sci. USA* 23: 356-60). Crossing over between the *X* chromosomes appears to be infrequent. Studies of *C(1)RM/R(1)w^{VC}//C(1)RM/O* mosaics (Schüpbach, Wieschaus, and Nöthiger, 1978, *Wilhelm Roux's Arch. Dev. Biol.* 184: 41-56) demon-

strate the focus of lethality to be in a region near the ventral prothoracic discs and that of female sterility to be in the external genitalia. Mosaics with diploid external genitalia and $3X:2A$ germlines produce about three-fourths as many eggs as diploid females but only two percent of such eggs develop into adulthood, most of them failing to complete embryogenesis. Transcriptional activity of each X chromosome in $3X:2A$ larvae, as measured by uridine incorporation by polytene chromosomes, is two-thirds that of each X in $2X:2A$ larvae according to Lucchesi, Rawls, and Maroni (1974, *Nature* 248: 564-67) and equal to that of each X in $2X:2A$ larvae according to Gvozdev, Leibovitch, and Ananiev (1983, *DIS* 59: 48-9). G6PD level per X -chromosome gene, as measured either by enzyme activity (Lucchesi *et al.*) or immunologically (Gvozdev *et al.*), in $3X:2A$ animals is two-thirds that in $2X:2A$ animals.

other information: The term metafemale instead of superfemale was suggested by Stern (1959, *Lancet* 12: 1088).



diploid metafemale

From Bridges, 1922, *Am. Nat.* 56: 51-63.

haplo-4

constitution: $X/X; 2/2; 3/3; 4;$ sex chromosome constitution may also be X/Y .

source: Produced after occasional loss or nondisjunction of chromosome 4 during meiosis. Produced in quantity from crosses of $C(4)RM/0$ with normal or from heterozygous $T(2;4)$ or $T(3;4)$ females.

discoverer: Bridges, 20a30.

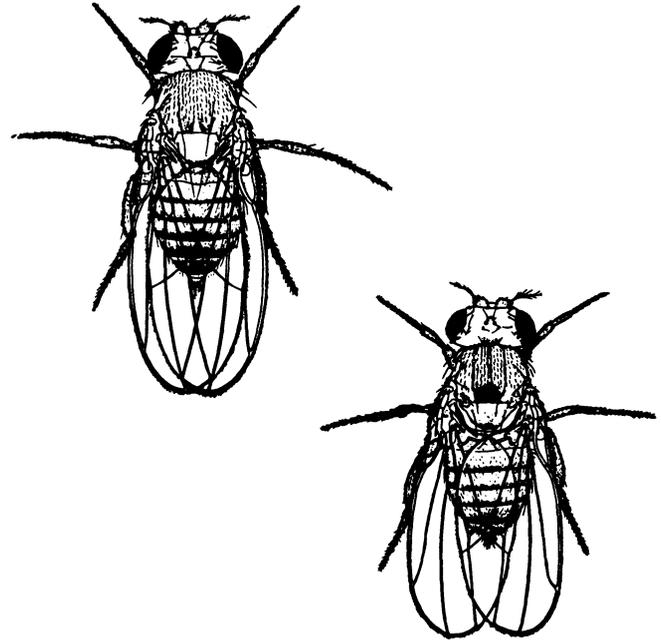
synonym: *Diminished*.

references: 1921, *Proc. Nat. Acad. Sci. USA* 7: 186-92.

1922, *Am. Nat.* 56: 51-63 (fig.).

Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 135-43 (fig.).

properties: Minute phenotype caused by deficiency for $M(4)$. Pale body with prominent trident pattern on thorax. L5 often does not reach wing margin. Eclosion delayed 2-4 days. Viability erratic, usually below 80% of normal. Usually sterile. Male tends to be more viable and fertile than female.



haplo-4

From Bridges, 1922, *Am. Nat.* 56: 51-63.

haploid

constitution: $X; 2; 3; 4.$

source: Recorded as patches of tissue.

discoverer: Bridges.

references: 1925, *Proc. Nat. Acad. Sci. USA* 11: 706-10.
1930, *Science* 72: 405-6.

properties: Eye facets small in haploid patches. A haploid foreleg bore no sex comb; the tissue is therefore probably female, as expected on the basis of balance theory of sex determination. Gynogenetic haploid embryos are produced in crosses of normal females with homozygous $ms(3)k81$ males; a considerable fraction of such embryos continue development to reach the final stages of embryogenesis, producing larvae with differentiated cuticles but which never hatch (Fuyama, 1986, *Genetics* 112: 237-48). That haploid embryos are females is indicated by the failure of gynogenetic haploids produced by *da* females to develop and the failure of the androcidal spiroplasma *SR*, derived from *Drosophila nebulosa* to affect such development (Fuyama, 1987, *DIS* 66: 53). Nuclear division cycle of haploid embryos protracted compared to diploids, and the syncytial blastoderm nuclei undergo an extra division to produce twice the normal density prior to cellularization (Edgar, Kiehle, and Schuberger, 1986, *Cell* 44: 365-72).

intersex

constitution: $X/X; 2/2/2; 3/3/3; 4/4/4;$ presence of Y and number of fourth chromosomes variable.

source: Regularly found among progeny of triploid females.

discoverer: Bridges, 20l.

references: 1921, *Science* 54: 252-54.

1922, *Am. Nat.* 56: 51-63 (fig.).

Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 153-62 (fig.).

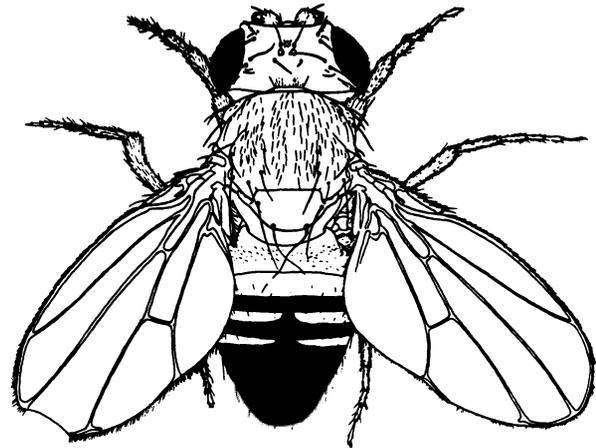
Bridges, 1939, *Sex and Internal Secretions* (E. Allen, C. H. Danforth, and C. A. Doisy, eds.). The Williams

and Wilkins Co., pp. 15-63.

properties: Large-bodied fly with coarse bristles, roughish eyes, and scalloped wing margins. Small hairs on surface of wing more sparsely distributed than in diploids. Usually has sex combs and a mixture of male and female genitalia; genitalia may be malelike or femalelike. Flies contain a patchy distribution of male and female tissue rather than intermediate sexuality; foreleg contains male sex-comb bristles or female bristles or both, but not bristles of intermediate position and morphology as seen in *dsx*; abdomen may consist of a sharply delineated mosaic of male and female pigmentation (Stern, 1968, Genetic Mosaics and Other Essays, Harvard University Press, Cambridge). This genotype is apparently unstably situated at the point of decision in sexual differentiation, and the mosaic phenotype seems to be a product of alternative sexual determination. The probability of male versus female development appears to be subject to extraneous influences. For example, Fung and Gowan (1956, J. Exp. Zool. 134: 515) reported that a triploid line producing intersexes with predominantly female genitalia carried several fourth chromosomes; raising intersexes at 27°-30° reported to have a feminizing effect on development (Laugé, 1969, Ann. Embryol. Morphog. 2: 245-69, 273-99). Gonads of triploid intersexes may be male like, female like or mosaic; *XO*- type crystals are present in spermatocytes of *XXAAA* but not *XXYAAA* intersexes; spermatocytes of the latter type can produce abnormal spermatozoa, which subsequently degenerate (Laugé, 1969, Ann. Soc. Entomol. Fr. (NS) 5: 253-514). Flies are uniformly sterile. Addition of sections of *X* chromosome shifts intersexes toward femaleness [Dobzhansky and Schultz, 1934, J. Genet. 28: 349-86 (fig.); Pipkin, 1940, Univ. Texas Publ. 4032: 126-56]. Addition of sections of the second or the third chromosome has not resulted in a shift of sexuality (Pipkin, 1947, Genetics 32: 592-607; 1960, Genetics 45: 1205-16). Gene activity per *X* chromosome in *2X;3A* intermediate between that of *2X;2A* or *3X;3A* and *1X;2A* as measured by enzyme activity in homogenates of adult flies (Faizullen and Gvozdev, 1973, Genetika 9: 106-07; Lucchesi and Rawls, 1973, Genetics 73: 459-64) and as measured by uridine incorporation by polytene chromosomes (Maroni and Plaut, 1973, Chromosoma 40: 361-77; Ananiev, Faizullen, and Gvozdev, 1974, Chromosoma 45: 193-201; Leibovitch, Belyaeva, and Zhimulev, 1976, Chromosoma 54: 349-62).

metamale

constitution: *X/Y; 2/2/2; 3/3/3; 4/4/4*; inferred from markers inherited. May also be diplo-4.
source: Occurs among progeny of triploid female.
discoverer: Bridges, 201.
synonym: *supermale*.
references: 1921, Science 54: 252-54.
 1922, Am. Nat. 56: 51-63 (fig.).
 Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 153-62 (fig.).
properties: Male has small body and spread wings. Late hatching, poorly viable, and completely sterile. Single polytene *X* of *XY:3A* metamale incorporates 90% as much uridine as all three *X* chromosomes in triploid larvae; G6PD and 6GPD levels in metamales over 80% those of triploid females (Lucchesi, Belote, and Maroni,



metamale

From Bridges, 1922, Am. Nat. 56: 51-63.

1977, Chromosoma 65: 1-7).

nullo-X

constitution: *Y/Y; 2/2; 3/3; 4/4*.
source: One-fourth the progeny from crosses between certain compound-*X*-bearing females [e.g., *C(1)RM/Y*] and normal males.
properties: Dies as embryo (Li, 1927, Genetics 12: 1-58). Cleavage nuclei abnormally distributed and blastoderm not formed (Poulson, 1940, J. Exp. Zool. 83: 271-325). According to Scriba (1964, Zool. Jahrb. Abt. Anat. Ontog. Tiere 81: 435-90), migration of cleavage nuclei to surface of egg is normal, blastoderm formation irregular, and germ band development frequently incomplete.

nullo-X nullo-Y

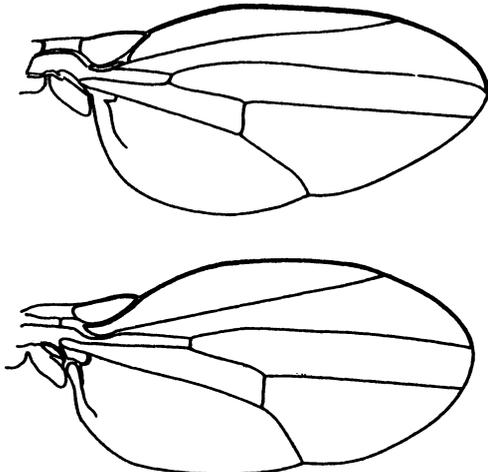
constitution: *2/2; 3/3; 4/4*.
source: One-fourth the progeny of crosses such as *C(1)RM/0* females with *Y^SX^L/0* males.
properties: Most embryos die after 10-12 cleavages (von Borstel and Rekemeyer, 1958, Nature 181: 1597-98). Embryology like that of *nullo-X* (Scriba, 1964, Zool. Jahrb. Abt. Anat. Ontog. Tiere 81: 435-90).

superfemale : see *diploid metafemal*

supermale : see *metamale*

tetra-4

constitution: *X/X; 2/2; 3/3; 4/4/4/4*. Sex chromosome constitution may also be *X/Y*; that for chromosome 4 may be *4/4/4/4* or *44/44*.
references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 135-43.
 Li, 1927, Genetics 12: 1-58.
 Bridges, 1935, Tr. Dinam. Razvit. 10: 463-74.
 Grell, 1961, Genetics 46: 1177-83 (fig.).
 Grell, 1972, DIS 48: 69.
properties: Viability slightly reduced; 68-76% of diplo-4 sibs and 58-63% that of tripl-4 (Moore and Grell, 1972, Genetics 70: 567-81, 583-93). Wings of survivors longer and more pointed than normal.
source: Synthesized as females homozygous for *T(1;4)w^{m4}* + *T(1;4)B^S* formed by recombination in



tetra-4

above: tetra-4; below: diplo-4

From Grell, 1961, *Genetics* 46: 1173-83.

region 3C4-15F8 between $T(1;4)_w^{m5} = T(1;4)3C3-4;101F1-2$ and $T(1;4)_B^S = T(1;4)15F9-16A1;16A7-B1;102F$ (Grell, 1961). Also recovered from progeny of crosses between males and females that carry $C(4)RM$ (Moore and Grell).

tetraploid

constitution: $X/X/X/X; 2/2/2/2; 3/3/3/3; 4/4/4/4$.

source: Seen on a few occasions as a tetraploid daughter of a triploid female or as a patch of tetraploid gonial tissue in an otherwise diploid female. Extensive attempts to produce tetraploid males have failed. (see Novitshi, 1984, *DIS* 60: 157).

discoverer: Bridges.

references: 1925, *Am. Nat.* 59: 127-37.

Morgan, 1925, *Genetics* 10: 148-78.

properties: Recognized by production of progeny that are almost exclusively triploids and intersexes.

triplo-4

constitution: $X/X; 2/2; 3/3; 4/4/4$. Sex chromosome constitution may be X/Y ; that for chromosome 4 may be $44/4$.

source: Product of nondisjunction of chromosome 4. Regular product of cross between $C(4)RM$ and normal diplo-4 flies.

discoverer: Bridges, 21b13.

references: 1922, *Am. Nat.* 56: 51-63.

Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 21 (fig.), 135-43.

properties: Phenotypic departure from normal very slight. Body darker than normal and trident pattern subdued. Eyes small. Body and wings elongate. Preferential segregation of the different fourth chromosomes in triplo-4's described by Sturtevant (1936, *Genetics* 21: 444-66).

triploid

constitution: $X/X/X; 2/2/2; 3/3/3; 4/4/4$. Sex chromosome constitution may also be $X/X/X/Y$, XX/X , or $XX/X/Y$. Triploids from stocks kept for several generations usually carry only two fourth chromosomes, i.e., diplo-4 triploids.

source: Spontaneous from unreduced eggs; incidence increased by treatment with cold (Bauer, 1946, *Z. Naturforsch.* 1: 35-38; Gloor, 1950, *DIS* 24: 82) or with colchicine (Braungart and Ott, 1942, *Sci. Counselor* 8: 105; Schultz). Produced in relatively high frequency by triploid females and by $c(3)G/c(3)G$ females (Gowen, 1933, *J. Exp. Zool.* 65: 83-106).

discoverer: Bridges, 1920.

references: 1921, *Science* 54: 252-54.

1922, *Am. Nat.* 56: 51-63 (fig.).

Morgan, Bridges, and Sturtevant, 1925, *Bibliog. Genet.* 2: 135-43.

properties: Eye facets larger and hairs on surface of wings more sparsely distributed than in diploid, giving eyes and wings a coarse texture; bristles also coarse. These characteristics are diagnostic for three sets of autosomes and result from increased cell size. Body thickset. Ventral bristles between first and second pairs of legs often missing. Discernible from diploid with practice. Triploid females have the same wet weight and the same amount of DNA per fly as do diploid females (Lucchesi and Rawls, 1973, *Biochem. Genet.* 9: 41-51); however, they have fewer and larger cells than diploids (Dobzhansky, 1929, *Wilhelm Roux's Arch. Entwicklunsgmech. Org.* 115: 363-79). Perhaps owing to a reduced number of neurons, triploid females show reduced mating success when competing with their diploid sisters (Novitski and Dews, 1970, *DIS* 45: 101). Genetic activity per X chromosome as estimated from uridine incorporation into salivary chromosomes (Maroni and Plaut, 1973, *Chromosoma* 40: 361-77) or by G6PD and 6GPD activities (Lucchesi and Rawls) the same in diploid and triploid females; thus triploid cells with 50% more chromatin exhibit 50% more activity per cell, but as there are two-thirds as many cells in triploids as diploids, the activity per fly is the same in the two genotypes. Fertility poor owing to production of aneuploid classes of gametes. Because equal numbers of chromosomes tend to go to each pole during first meiotic division, euploid gametes are produced with lower-than-expected frequencies; gametes with one X and two sets of autosomes and with two X's and one set of autosomes far outnumber those with one X and one set of autosomes or two X's and two sets of autosomes (Bridges and Anderson, 1926, *Genetics* 10: 418-41). Triploids that carry an attached X (attached X triploids) are more fertile and produce a higher proportion of triploid progeny than free X triploids. Triploids are of necessity female and their progeny include metafemales, metamales, intersexes, triploid and diploid females, and diploid males. Crossing over is markedly increased in triploids; Sturtevant (1951, *Proc. Nat. Acad. Sci. USA* 37: 405-7) has mapped chromosome 4 in diplo-4 triploids. *B*, *Bl*, *Bx*, *Cy*, *D*, *Dfd*, *H*, *Hw*, *J*, *L*², *Me*, and *Sb* are classifiable in a single dose in triploids. *Dl*, *G*, *N*, *bw*^{VI}, *Px*, *S*, and all Minutes are recessive in a single dose. Two doses of *D*, *Dl*, *G*, *H*, *bw*^{VI}, *Px*, and *Sb* produce an extreme phenotype, whereas two doses of *M* or *Me* are lethal (Schultz, 1934, *DIS* 1: 55).

triploid metafemale

constitution: $X/X/X/X; 2/2/2; 3/3/3; 4/4/4$; third 4 may be absent.

source: Found among progeny of tetraploid female (Morgan). Also produced by nondisjunction of sex chromo-

somes in *C(1)RM/In(1)sc⁸/Y* triploid (Frost).

discoverer: L. V. Morgan.

references: 1925, *Genetics* 10: 147-78.

Frost, 1960, *Proc. Nat. Acad. Sci. USA* 46: 47-51.

properties: Coarse eyes, wing texture, and bristles. Resembles triploid except body smaller and eyes more bulging. Inner wing margins often incised. Using exceptional triploid females as a standard, Frost (1960) determined that triploid metafemales have 25% viability, 24-54% lay eggs (1 to 150 eggs), and about 11% of the eggs develop into adults.

XO male

constitution: *X; 2/2; 3/3; 4/4*.

source: Product of primary nondisjunction of the sex chromosomes in either father or mother in cross of *X/Y* male with *X/X* female. Forms one-fourth the progeny of crosses, such as *X/X* female by *Y^{SX}·Y^L/0* male or *C(1)RM/0* female by *X/Y* male.

discoverer: Bridges.

references: 1916, *Genetics* 1: 1-52.

properties: Male morphologically normal but entirely sterile. No motile sperm produced. Spermatogenesis described by Kiefer (1966, *Genetics* 54: 1441-52) and by Meyer (1968, *Z. Zellforsch.* 84: 141-75; *Genetics* 61 Supplement: 79-92); the phenotype is the consequence of the simultaneous deletion of the six *Y*-linked fertility genes plus *Su(Ste)* and *bb*; the effects of removal of these genes one at a time have been described (Hardy, Tokuyasu, and Lindsley, 1981, *Chromosoma* 83: 593-617; Goldstein, Hardy, and Lindsley, 1982, *Proc. Nat. Acad. Sci. USA* 79: 7404-09; Hardy, Lindsley, Livak, Lewis, Sivertsen, Joslyn, Edwards, and Bonaccorsi, 1984, *Genetics* 107: 591-610). The phenotype includes abnormal distribution of chromosomes and mitochondrial material at meiosis 1, the presence of micronuclei in spermatids, depression of *Ste* on the *X*, and the presence of needle-shaped proteinaceous crystals in primary spermatocytes, spermatids, and extracellularly; these phenotypes are attributable to the absence of *Su(Ste)*. Nucleoprotein structures ordinarily present in primary spermatocyte nuclei attributable to *Kl-5*, *kl-3*, and *ks-1* are missing. The outer dynein arms associated with the peripheral microtubular doublets of the axoneme are reduced or absent owing to the absence of *kl-5* and *kl-3*, and three sperm proteins in the 300-350 kd range attributable to *kl-5*, *kl-3* and *kl-2* are missing. The combined effects of these early defects lead to the general disorganization of structure as spermiogenesis continues. The contributions of *kl1*, *bb*, and *ks2* to the *XO* phenotype have not been sorted out, although their individual effects have been described.

XXY female

constitution: *X/X/Y; 2/2; 3/3; 4/4*. Sex chromosome constitution may also be *X/XY* or *XX/Y*.

source: Product of either primary or secondary nondisjunction in either male or female. Also produced from cross of an *XY*-bearing parent with a normal-*X*-bearing parent. Condition usually found in compound-*X*-bearing female.

discoverer: Bridges.

references: 1916, *Genetics* 1: 1-52.

properties: Phenotype and fertility like those of normal female. Nondisjunction of *X* chromosomes in *X/X/Y* much higher than in *X/X* female; about 4% exceptions with two normal *X* chromosomes and much higher if *X*'s are heterozygous for inversions (Sturtevant and Beadle, 1936, *Genetics* 21: 554-604).

XXYY female

constitution: *X/X/Y/Y; 2/2; 3/3; 4/4*. Sex constitution may also be *XX/Y/Y*, *X/XY/Y*, or *XY/XY*.

source: A common product of crosses such as *Y^{SX}·Y^L/Y* male by *X/Y^{SX}·Y^L* or *X/X/Y* female, or *X/Y/Y* male by *X^{SX}·Y^L/X*, *X/X/Y*, or *C(1)RM/Y* female.

discoverer: Stern.

references: 1929, *Biol. Zentr.* 49: 261-90; 727.

Cooper, 1956, *Genetics* 41: 242-64.

properties: Eye color mottled to varying degrees. Posterior and middle legs often malformed. Fertility and viability reduced. Gametes preponderantly *X/Y* in constitution owing to the regular segregation of both the *X*'s and the *Y*'s at the first meiotic division. Sometimes sterile.

XYY male

constitution: *X/Y/Y; 2/2; 3/3; 4/4*. Sex chromosome constitution may also be *XY/Y*.

source: About one-fourth the progeny of crosses such as *X/X/Y* female by *X/Y* male, *C(1)RM/Y* female by *Y^{SX}·Y^L/Y* male, and *X/Y^{SX}·Y^L* female by *X/Y* male.

discoverer: Bridges.

references: 1916, *Genetics* 1: 1-52.

properties: Phenotype normal; usually fertile; with certain normal *Y* chromosomes, completely sterile (R. F. Grell). The two *Y* chromosomes tend to separate at the first meiotic division to a degree depending on the source of the *Y*'s and the *X* (Grell, 1958, *Proc. Intern. Congr. Genet.* 10th., Vol. 2: 105).

XYYY male

constitution: *X/Y/Y/Y; 2/2; 3/3; 4/4*. Sex chromosome constitution may also be *XY/Y/Y*.

discoverer: Stern.

references: 1929, *Biol. Zentr.* 49: 261-90.

Morgan, Bridges, and Schultz, 1934, *Carnegie Inst. Wash. Year Book* 33: 274-80.

Cooper, 1956, *Genetics* 41: 242-64.

properties: Morphologically normal male but with mottled eyes as in *XXYY* female. Almost entirely sterile owing to the presence of three doses of *kl-3* (Kennison, 1981, *Genetics* 98: 529-48). Cooper (1956) suggests that the few offspring may result from *X/Y/Y* cysts produced by mitotic loss of a *Y* chromosome.

SATELLITE SEQUENCES

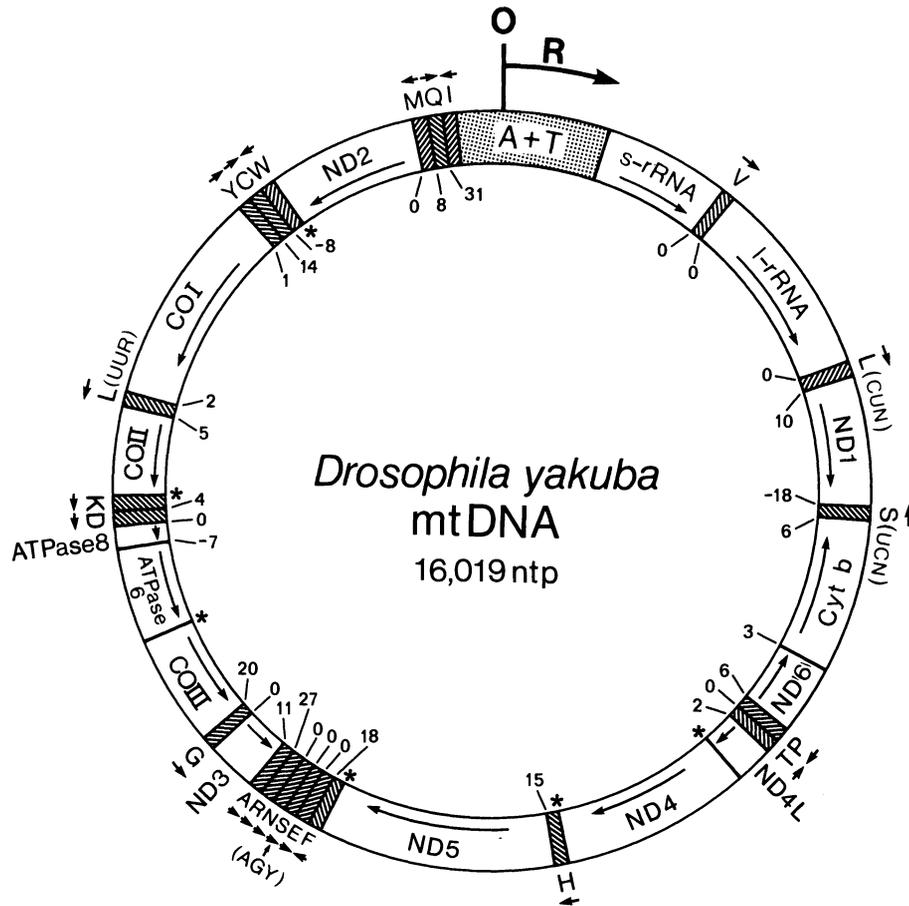
Long arrays of highly repeated simple oligonucleotide sequences with characteristic buoyant densities on cesium chloride gradients performed either with or without the action of DNA-binding antibiotics or metal ions. Comprise approximately 20% of the *Drosophila melanogaster* DNA complement. Confined to, and comprise the majority of, the pericentric heterochromatin of all chromosomes in the complement. Four satellite peaks in cesium chloride gradients can be separated from the main band, which contains the euchromatic DNA (Peacock, Brutlag, Goldring, Appels, Hinton, and Lindsley, 1973, Cold Spring Harbor Symp. Quant. Biol. 38: 405-16). These sequences do not become amplified in polytene chromosomes and remain at the diploid-cell concentration (Gell, Cohen, and Polan, 1971, Chromosoma 33: 319-44). Large clones of 1.672, 1.686, and 1.705 g/ml satellite DNA sequences in *E. coli* are unstable, but segments of 300 to 600 base pairs can be stably cloned in pBR322; it is found that samples of such clones derived from a single peak of the gradient may contain one major and several minor repeating sequences (Lohe and Brutlag, 1986, Proc. Nat. Acad. Sci. USA 83: 696-700).

buoyant density (g/ml)	sequence 5' → 3' ^α	density class (g/ml)	fraction of total DNA (%)
1.663	(AACAA) _n	1.672	0.06
1.669	(AATAAAC) _n	1.672	0.23
1.672	(AATAT) _n	1.672	3.1
1.680	(AATAC) _n	1.672	0.52
		1.686	
1.686	(AATAACATAG) _n	1.686	2.1
1.688	(359 bp) _n	1.688	5.1
1.688	(AATAGAC) _n	1.686	0.24
1.689, 1.701	(AAGAC) _n	1.686	2.4
1.693	(AATAG) _n	1.686	0.23
	(GAGAG) _n		
1.705	(AAGAG) _n	1.705	5.6

^α designated according to the sequence of the purine-rich strand.

The five-, seven-, and ten-base-pair repeats in such clones of either major or minor satellite sequences are quite homogeneous, displaying zero, one, or at most two deviant repeat units in stretches of approximately fifty

such units (approximately one base pair per kilobase). The sequences are not random, but conform to the pattern (RRN)_m(RN)_n, where R represents a purine (usually A) and N represents C, T, or G. The 1.688 g/ml satellite is complex, consisting of a 359-base-pair repeating unit. This sequence is quite variable among repeat units at about a dozen sites, but is otherwise highly conserved (Hsieh and Brutlag, 1979, J. Mol. Biol. 135: 465-81). Hsieh and Brutlag (1979, Proc. Nat. Acad. Sci. USA 76: 731-35) identified and partially purified a protein that binds specifically to the 1.688 g/ml satellite sequence. *In situ* hybridization of satellite sequences to polytene-chromosome preparation show labeling of the chromo-center, and (some at lower stringency) of a few euchromatic bands [e.g. 3C (359 bp), 7F (AATAC), 21D (AAGAG), telomeres (AAGAG) (Lohe and Roberts, 1988, Heterochromatin: Molecular and structural aspects (R.S. Verma, ed.). Cambridge University Press, Cambridge, pp. 148-86)]. Hybridization of radio labeled RNA transcribed from DNA of the different buoyant-density classes to mitotic chromosomes reveals the presence of homologous sequences in the pericentric heterochromatin (Steffensen, Appels, and Peacock, 1981, Chromosoma 82: 525-41). 1.672 g/ml DNA sequences are detected at the tip and in the middle of *YL*, and in the heterochromatin of chromosome 4 and to a lesser extent at the tip of *YS* and in the centromere regions of chromosomes 2 and 3. 1.705 g/ml sequences are detected in substantial quantities at the tip and base of *YL*, the base of *YS*, and in the pericentric heterochromatin of both *2L* and *2R* and in lesser amounts at the tip of *YS*, the centromere region of the *X*, near the heterochromatic-euchromatic junction of *3R*, and in the right arm of chromosome 4. The 1.688 g/ml satellite is mostly localized to two blocks in the *X* heterochromatin, one near the middle in the vicinity of the nucleolus organizer and the other adjacent to the centromere and in a third block near the center of *YL*; it is also detected in order-of-magnitude lower amounts at the tip and base of *YL*, the nucleolus organizing region of *YS*, at the base of one arm each of chromosomes 2 and 3, and on the short arm of chromosome 4 (Peacock, Lohe, Gerlach, Dunsmuir, Dennis, and Appels, 1977, Cold Spring Harbor Symp. Quant. Biol. 42: 1121-35).



Gene map of the *Drosophila yakuba* mitochondrial genome provided by Wolstenholme. The circular molecule of 16,019 nucleotide pairs has been completely sequenced. The 13 protein genes are COI, COII, and COIII (subunits 1, 2, and 3 of cytochrome *c* oxidase), Cyt b (cytochrome b), ATPase 6 and 8 (subunits 6 and 8 of the F₀ ATPase complex), and ND1-ND6 and ND4L (components 1-6 and 4L of the respiratory chain NADH dehydrogenase). Each tRNA gene (hatched) is identified by the one letter amino acid code, and serine and leucine tRNA genes are also identified by the codon family (in parenthesis) that the corresponding tRNAs recognize. s-rRNA and l-rRNA indicate the small and large rRNA genes, respectively. Arrows within and outside the molecule indicate the direction of transcription of each gene. The numbers of apparently non-coding nucleotides that occur between the genes are shown at the gene boundaries on the inner side of the map. Negative numbers indicate overlapping nucleotides of adjacent genes. Asterisks identify possible incomplete termination codons (T or TA). The location of the origin of replication (O) within the A+T-rich region (dotted) and the direction of replication (R) were determined by electron microscope studies (Goddard and Wolstenholme, 1980, *Nucleic Acids Research* 8: 741-757). This map is a modification of that given by Clary and Wolstenholme (1985, *J. Mol. Evol.* 22: 252-27).

NONCHROMOSOMAL INHERITANCE

mitochondria

Mitochondrial DNA (mtDNA) is a covalently closed duplex circle of about 19,500 base pairs. Contained within *Drosophila* mtDNA is a region exceptionally rich in adenine + thymine. This A+T-rich region comprises 5-6 kb in *D. melanogaster*, but is shorter in related species (Fauron and Wolstenholme, 1980, *Nucleic Acids Res.* 8: 2439-52); some intraspecific variation in the length of this region is recorded; e.g., 5.9, 5.5, and 5.1 kb in three strains of *D. melanogaster*; these differences used to confirm maternal inheritance of *Drosophila* mitochondria (Fauron and Wolstenholme, 1980, *Nucleic Acids Res.* 8: 5391-5410). Mitochondria of *D. melano-*

gaster replaced by those of *D. mauritiana* by embryo injection (Niki, Chigusa, and Matsuura, 1989, *Nature* 341: 551-52). The origin of replication is located within the A+T-rich segment and proceeds unidirectionally around the circle, with the first strand becoming as much as 100% completed before the second strand initiates. Extensive sequencing of mtDNA of both *D. yakuba* and *D. melanogaster* has shown the two molecules to be highly homologous and to encode thirteen hydrophobic polypeptides, which have been identified as subunits of enzyme complexes associated with the inner mitochondrial membrane, a large and a small ribosomal RNA and

22 transfer RNA's. The polypeptides include cytochrome b, two subunits (6 and 8) of the ATPase, three subunits of the cytochrome c oxidase, and seven subunits of the NADH reductase complex (Chomyn, Marioeini, Cleeter, Ragan, Matsuno-Yagi, Hatefi, Doolittle, and Attardi, 1985, *Nature* 314: 592-97). These coding regions are densely packed, with few if any nucleotides separating them and in a few cases overlapping, and none of them containing introns (de Bruijn, 1983, *Nature* 304: 234-41; Wolstenholme and Clary, 1985, *Genetics* 109: 725-44; Garesse, 1988, *Genetics* 118: 649-63). The direction of replication and the order and directions of transcription of these mitochondrial genes are indicated on the diagram of the *Drosophila yakuba* mitochondria at the beginning of this section; the A+T-rich region of *D. yakuba* is considerably shorter than that of *D. melanogaster*. Mouse mitochondrial DNA contains the same genes, but the order differs from that of *Drosophila* by three inversions, and there has been some shuffling of tRNA-encoding sequences [Wolstenholme, Clary, Macfarlane, Wahleithner, and Wilcox, 1985, *Organization and Evolution of Invertebrate Mitochondrial Genomes* (Quagliariello, Slater, Palmieri, Saccone, and Kroon, eds.). Elsevier, Amsterdam, New York, Oxford, Vol. 2: pp. 61-70]. The polypeptide encoding genes use ATA, ATT, or ATG as initiation codons; ATC appears only as an internal codon. TAA is the most frequently used termination codon with ATG found in one case; some genes end in T or TA, and the stop codon is generated by polyadenylation (Garesse). UGA → tryptophan instead of stop; AUA → methionine instead of isoleucine; unlike mammalian mitochondria, AGA → serine instead of arginine as in chromosomally derived messages or stop as in mammalian mitochondria. Overall codon usage reflects the relatively high ratio of AT/CG in mitochondrial *vis-à-vis* chromosomal DNA.

picornaviruses

Three small RNA viruses known as picornaviruses are found in *Drosophila melanogaster* but only two of these viruses, DAV and DPV are transmitted from females to their offspring in the egg. The third DCV, is transmitted by contamination of the surface of the egg.

origin: Transmitted vertically in eggs of young, naturally-infected females of both natural and lab populations (old naturally-infected females and artificially-infected females cannot transmit DAV or DPV in this way); viruses also transmitted by contact, ingestion and inoculation.

references: Plus and Duthoit, 1969, *C.R. Acad. Sci., Paris* 268: 2313.

David and Plus, 1971, *Ann. Inst. Pasteur*, 120: 107-119.

Jousset, Plus, Croizier, and Thomas, 1972, *C.R. Acad. Sci. Paris* 275: 3043-46.

Teninges and Plus, 1972, *J. Gen. Virol.* 16: 103-17.

Brun and Plus, 1980, *The Genetics and Biology of Drosophila* (Ashburner and Wright, eds.). Academic Press, London, New York, San Francisco, Vol. 2d, pp. 625-702.

Ashburner, 1989, *Drosophila, a Laboratory Handbook*, Cold Spring Harbor Press, Cold Spring Harbor, New York, pp. 101-02.

phenotype: Both DAV and DPV are ribosomal viruses of 625-702 nm that are resistant to ether, ethanol, and low

pH treatment. Eggs infected with these viruses can be cured by dechlorinating them using sodium hypochlorite and transferring them to fresh medium. Unlike DCV, which kills the flies a few days after injection, the viruses DAV and DPV are not immediately pathogenic to their hosts; however, they reduce the longevity and female fertility of the infected flies. No carbon dioxide sensitivity is observed in *Drosophila* as a result of these viruses. DAV and DPV multiply in the gut, Malpighian tubules, and ovaries of *Drosophila melanogaster* and are only found in the cytoplasm (not the nucleus) of the infected cells. Molecular weights of major capsid polypeptides of the viruses are:

DAV 31,600, 41,200, 72,900

DPV 26,000, 19,400, 48,000.

sigma: sensitivity to carbon dioxide

references: L'Héritier and Teissier, 1937, *Comp. Rend.* 20665: 1099-1101.

1938, *Comp. Rend.* 206: 1193-9, 1683.

1945, *Publ. Lab. Ecole Norm. Super. Biol. (Paris)* 1: 35-74.

L'Héritier, 1948, *Heredity* 2: 325-48.

1951, *Cold Spring Harbor Symp. Quant. Biol.* 16: 99-112.

1958, *Advances in Virus Research* (K.M. Smith and M.A. Lauffer, eds.). Academic Press, London, New York, San Francisco, pp. 195-245.

L'Héritier, and Plus, 1963, *Biological Organization at the Cellular and Supercellular level* (R.J.C. Harris, ed.). Academic Press, London, New York, San Francisco, pp. 59-71.

Gay, 1978, *Mol. Gen. Genet.* 159: 269-83.

L'Héritier, 1979, *Handbook of Genetics* (King, ed.). Plenum Press, New York and London, Vol. 3, pp. 813-18.

Brun and Plus, 1980, *The Genetics and Biology of Drosophila* (Ashburner and Wright, eds.). Academic Press, London, New York, San Francisco, Vol. 2d, pp. 625-702.

Fleuriet, 1980a, *Genetics* 95: 459-65.

1980b, *DIS* 55: 43.

Teninges and Bras-Herreg, 1987, *J. Gen. Virol.* 68: 2625-38.

Fleuriet, 1988, *Evol. Biol.* 23: 1-30.

Ashburner, 1989, *Drosophila, a Laboratory Handbook*, 1989, Cold Spring Harbor Press, Cold Spring Harbor, New York, pp. 101-116, 1192.

phenotype: Whereas normal *Drosophila melanogaster* recover in a short time after carbon dioxide anesthesia, flies belonging to certain strains suffer paralysis eventually followed by death after a short exposure to carbon dioxide. These strains were found to carry the rhabdovirus sigma (L'Héritier and Teissier, 1937). This RNA virus resembles vesicular stomatitis virus (VSV) of horses both in size (70 nm wide and 180 nm long) and shape (bullet-like); VSV and the fish rhabdoviruses PFR and SVC can multiply and produce carbon dioxide sensitivity in *Drosophila* (Brun and Plus, 1980; Teninges and Bras-Herreg, 1987). The sigma virus, however, does not multiply in any vertebrate host and normally is transmitted from *Drosophila* females to their offspring by way of the egg. The expression of sigma is correlated with the presence of the virus in nerve centers. The virus grows well in *Drosophila* tissue culture cells (Richard-Molard,

Blondel, Wyers, and Dezelee, 1984, *J. Gen. Virol.* 65: 91-99).

Carbon dioxide-sensitive sigma strains may be divided into two types: stabilized and nonstabilized. Artificial inoculation regularly leads to the nonstabilized condition. In this state, males do not transmit sensitivity to progeny but females do transmit it to part of their progeny, indicating the presence of the sigma virus in some of the eggs of infected individuals. Some flies of a nonstabilized strain achieve the stabilized state. In the stabilized state, the females transmit the virus and the stabilized condition to approximately 100% of their offspring (Fleuriet, 1980a, 1988). The stabilized males transmit the virus but not the stabilized condition to part of their progeny; electron micrographs show the virus in male germ cells (Brun and Plus, 1980).

The *ref* (refractory) mutants, found on *Drosophila* chromosomes *X*, *2*, and *3*, prevent flies from being infected by sigma. Some of the virus strains are defective and do not produce carbon dioxide-sensitivity in stabilized flies. Other mutant strains of sigma are heat sensitive and unable to infect flies at 30° (Contamine, 1973, *Mol. Gen. Genet.* 124: 233-46). Flies stabilized for one of these temperature-sensitive mutants lose their sensitivity to carbon dioxide at the restrictive temperature but can transmit the virus to their offspring.

molecular biology: Sigma virus from a *Drosophila* cell line was used to prepare genomic RNA (Teninges and Bras-Herreg, 1987). The sigma strain used gave a high yield of virus but was unable to establish a persistent stabilized-type infection when inoculated into *Drosophila* tissue culture cells. After preparation, the genomic RNA was purified, labelled with ³²P, and used to probe for mRNA in sigma-infected cells. A cDNA copy of the complete coding region of the mRNA was cloned and its nucleotide sequence and deduced amino acid sequence determined (Teninges and Bras-Herreg, 1987). There is a long open reading frame (ORF) in the nucleotide sequence starting at position 47 and ending at position 1624. The putative amino acid sequence consists of a chain of 526 amino acids. Within the long ORF is a short frame-shifted ORF (starting at position 643 and ending at position 763) which could encode a peptide of 40 amino acids. About 20% amino acid sequence identity has been established between the glycoprotein precursor of this strain of sigma virus and that of VSV (New Jersey strain).

SR: Sex Ratio

origin: Artificially inoculated into *D. melanogaster* from SR-bearing *D. willistoni* and *D. nebulosa*.

references: Poulson and Sakaguchi, 1961, *Genetics* 46: 890-91.

1962, *Ann. Rep. Nat. Inst. Genetics* (Misima, Japan) 12: 18-19; 19-21.

Poulson, 1963, *Methodology in Basic Genetics* (W.J. Burdette, ed.). Holden-Day Inc., pp. 404-24.

Sakaguchi and Poulson, 1963, *Genetics* 48: 841-61.

Ikeda, 1965, *Science* 147: 1147-48.

Oishi and Poulson, 1970, *Proc. Nat. Acad. Sci. USA* 67: 1565-72.

Sakaguchi, 1970, *DIS* 45: 145.

Oishi, 1971, *Genet. Res.* 18: 45-56.

Tsuchiyama and Sakaguchi, 1972, *DIS* 48: 29.

Miyamoto and Oishi, 1975, *Genetics* 79: 55-61.

Watanabe and Yamada, 1977, *Jpn. J. Genet.* 52: 9-14.

Tsuchiyama, Sakaguchi, and Oishi, 1978, *Genetics* 89: 711-21.

Williamson and Poulson, 1979, *The Mycoplasmas* (Whitcomb and Tully, eds.). Academic Press, London, New York, San Francisco, Vol. III, pp. 175-208.

Laugé, 1980, *The Genetics and Biology of Drosophila* (Ashburner and Wright, eds.). Academic Press, London, New York, San Francisco, Vol. 2d, pp. 33-106.

Niki, 1988, *Jpn. J. Genet.* 63: 11-21.

Ashburner, 1989, *Drosophila, a Laboratory Handbook*, 1989, Cold Spring Harbor Press, Cold Spring Harbor, New York. p. 1193.

phenotype: The SR or Sex Ratio phenotype found in several groups of *Drosophila* carrying spirochaete-like microorganisms now classified as spiroplasmas in their hemolymph (Williamson and Whitcomb, 1975, *Science* 188: 1018-20) and is characterized by a failure on the part of females to produce male offspring (Williamson and Poulson, 1979). Spiroplasmas are normally transmitted from female to offspring in the egg (Nikki, 1988). The SR agent can be extracted from *D. willistoni*, *D. nebulosa*, *D. paulistorum*, or *D. equinoxialis* and can be transferred by inoculation into *D. melanogaster*, and *D. simulans* (Poulson, 1963; Sakaguchi and Poulson, 1963; Ikeda, 1965; Oishi and Poulson, 1970; Watanabe and Yamada, 1977; Tsuchiyama *et al.*, 1978). Two strains of SR microorganisms, WSR from *D. willistoni* and NSR from *D. nebulosa*, are quite stable in *D. melanogaster*, the degree of stability of the infection differing among *D. melanogaster* strains. XY male offspring of females carrying the infection die as embryos. A few infected gynandromorphs with small areas of XO tissue survive, but the majority do not survive when XO cells carry the infection (Tsuchiyama *et al.*, 1978). Triploid intersexes are not killed by the SR agent, nor are females transformed by *tra*, *tra^D*, *ix*, or *dsx* (Sakaguchi and Poulson, 1963; Miyamoto and Oishi, 1975). The spiroplasmas of each species of *Drosophila* carry their own viruses and these viruses can clump and lyse the spiroplasmas of related species of *Drosophila* (Oishi, 1971; Poulson and Oishi, 1973, *Genetics* 74: s216).

other information: The name SR was proposed by Magni (1953, *Nature* 172: 81) for *D. bifasciata* females (inseminated before capture) that produced only female offspring (Laugé, 1980) and was used by Poulson and colleagues for these maternally-transmitted cytological factors (spiroplasmas) found naturally or by infection in *Drosophila*.

CYTOGENETIC MAP

by: Michael Ashburner

This is a cytogenetic map of *D. melanogaster* sorted by determined or estimated genetic map position. Estimated map positions are based on cytological positions, and where the published genetic map position is at variance with the known cytological position, an estimated map position (enclosed in brackets) is substituted for the published genetic position. There is approximate but not complete concordance between the map positions in this table and those in the individual entries in the body of this work. Loci mapped only to a chromosome, chromosome arm, or to a very long region (*e.g.*, between *v* and *f* on chromosome *I*) are excluded from this table. The other fields in the table are cytogenetic map position, gene symbol, and map index. The gene symbols in the table have been reconciled with those used in the body of the text insofar as possible; loci indicated in the table that are not represented in the text are designated with the symbol "†".

Map index has been introduced to indicate absolute physical order within well mapped regions, this order usually being determined by deletion mapping or molecular methods. The reason for doing this is that published map positions, either genetic or cytogenetic, are inadequate to indicate the absolute order in many, if not most

cases. So as to allow this order to be generated by a program, rather than by hand, genes within well mapped regions are given an index which indicates this order. The map index has three components: a numbered division (1-104; numbers 103 and 104 are for the long and short arms of the *Y* chromosome respectively); a lettered subdivision, in lower case so as to avoid confusion with cytogenetic positions, and an index number. If, within a lettered subdivision, two independent fine scale maps cannot be related with respect to each other, then the lower case letter of the index may be repeated. Examples of map indices are 1a100, 24e1000, 24ee100. If two loci have not been separated, then they will have identical index numbers. Physical relationships are indicated by the fact that within a series of map indices (that is within a division or subdivision) a locus with the numerically lower index is physically to the left of one with a higher index. The index number is enclosed with () if only *relative* order is known. Map indices are not permanent attributes of loci (at least, not until the map is "complete"). They will change as these tables are updated. Note that the sorting of loci in this table may well give an incorrect impression of physical order.

THE GENOME OF *DROSOPHILA MELANOGASTER*

genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol
1-0.0			<i>cc</i>	1-[0.3]	1E3-2A3	1e400	<i>l(1)IEFg</i>	1-0.6			<i>l(1)HM1</i>
1-0.0			<i>clw1</i>	1-[0.3]	1E3-2A3	1e400	<i>l(1)IEFh</i>	1-0.6			<i>l(1)YD23a[†]</i>
1-0.0			<i>cpl</i>	1-[0.3]	1E3-2A3	1e400	<i>l(1)IEFi</i>	1-0.6	1E1-2B9		<i>rey</i>
1-0.0			<i>dar</i>	1-[0.3]	1E3-2A3	1e400	<i>l(1)IEFj</i>	1-0.6			<i>srđ</i>
1-0.0			<i>double</i>	1-[0.3]	1F	1e400	<i>l(1)IFa</i>	1-0.7			<i>l(1)ds1</i>
1-0.0			<i>ffi-385</i>	1-[0.3]	1F	1e400	<i>l(1)IFb</i>	1-0.8			<i>l(1)3063</i>
1-0.0			<i>ffiB</i>	1-[0.3]	1F	1f100	<i>l(1)IFc</i>	1-0.8			<i>l(1)dn19</i>
1-0.0	1B10-1E3		<i>fs(1)A147</i>	1-[0.3]	1F4	1f100	<i>l(1)IFd</i>	1-0.8			<i>mk</i>
1-0.0			<i>s(1)M4</i>	1-0.4	1F5-2A		<i>aperC</i>	1-0.8			<i>yea3</i>
1-0.0			<i>l(1)S5</i>	1-0.4			<i>l(1)HM7</i>	1-0.9			<i>fc</i>
1-0.0			<i>l(1)HM9</i>	1-0.4			<i>l(1)ts538</i>	1-0.9			<i>ovi</i>
1-0.0			<i>l(1)HM11</i>	1-0.4			<i>mwi</i>	1-0.9			<i>sbs</i>
1-0.0			<i>l(1)HM12</i>	1-0.4			<i>pop</i>	1-0.9	2E2-2E3	2e150	<i>pcx</i>
1-0.0			<i>l(1)Mc28</i>	1-0.4	2A-2C		<i>tonock[†]</i>	1-0.9	2E3	2e200	<i>kz</i>
1-0.0			<i>l(1)X10</i>	1-0.4	2B6	2b300	<i>dor</i>	1-[0.9]	2E2	2e100	<i>l(1)2Ea</i>
1-0.0	1A1-1A8		<i>pch</i>	1-0.4	2B6-2B10	2b450	<i>fmf</i>	1-[1]	2D1-2D3		<i>l(1)ESHS14</i>
1-0.0			<i>pld</i>	1-[0.4]	2B		<i>l(1)VA335[†]</i>	1-[1]	2D3-2F1		<i>l(1)ESHS15</i>
1-0.0			<i>saw</i>	1-[0.4]	2B15		<i>arm</i>	1-[1]	2F3-3C5		<i>l(1)ESHS16</i>
1-0.0			<i>supact</i>	1-[0.4]	2A2-2B18		<i>cau</i>	1-[1]	2F3-3C5		<i>l(1)ESHS17</i>
1-0.0			<i>tdd</i>	1-[0.4]	2A4		<i>fs(1)N</i>	1-[1]	2C		<i>slgB</i>
1-0.0	1A4	1a100	<i>l(1)1Aa</i>	1-[0.4]	2B4-2B5		<i>l(1)2Bab</i>	1-[1]	2F1	2f100	<i>crn</i>
1-0.0	1A6	1a300	<i>l(1)1Ac</i>	1-[0.4]	2A2-2A4		<i>l(1)ESHS3</i>	1-[1]	2F1-2F3	2f200	<i>l(1)2Fb</i>
1-0.0	1A5	1a400	<i>l(1)1Ad</i>	1-[0.4]	2A2-2A4		<i>l(1)ESHS4</i>	1-[1]	2F1-2F3	2f300	<i>l(1)2Fc</i>
1-0.0	1A7	1a500	<i>cin</i>	1-[0.4]	2B3-2B12		<i>l(1)ESHS5</i>	1-[1]	2F1-2F3	2f400	<i>l(1)2Fd</i>
1-0.0	1A7-1A8	1a600	<i>l(1)1Af</i>	1-[0.4]	2B17-2C2		<i>l(1)ESHS6</i>	1-[1]	2F6	2f500	<i>phl</i>
1-0.0	1A8	1a700	<i>ewg</i>	1-[0.4]	2B17-2C2		<i>l(1)ESHS7</i>	1-[1]	2F6	2f525	<i>ptr[†]</i>
1-0.0	1A5-1A8	1a800	<i>arth</i>	1-[0.4]	2B17-2C2		<i>l(1)ESHS8</i>	1-1.0	3A5		<i>A</i>
1-0.0	1B1	1b100	<i>y</i>	1-[0.4]	2B17-2C2		<i>l(1)ESHS9</i>	1-1.0			<i>fb</i>
1-0.0	1B2-1B3	1b200	<i>ac</i>	1-[0.4]	2B17-2C2		<i>l(1)ESHS10</i>	1-1.0			<i>hypoA</i>
1-0.0	1B3-1B4	1b300	<i>sc</i>	1-[0.4]	2B17-2C2		<i>l(1)ESHS11</i>	1-1.0			<i>fs(1)M103</i>
1-0.0	1B3-1B4	1b350	<i>l(1)sc</i>	1-[0.4]	2B5		<i>npr1</i>	1-1.0	3A6-3C2		<i>msd(gl)</i>
1-0.0	1B3-1B4	1b375	<i>ase</i>	1-[0.4]	2B17-2C2		<i>oljB</i>	1-1.0			<i>ws</i>
1-0.0	1B	1b400	<i>l(1)1Bb</i>	1-[0.4]	2A1	2a100	<i>l(1)2Aa</i>	1-1.0	3A2	3a100	<i>gt</i>
1-0.0	1B	1b500	<i>l(1)1Bc</i>	1-[0.4]	2A1	2a100	<i>l(1)2Ab</i>	1-1.0	3A2	3a200	<i>iko</i>
1-0.0	1B7	1b600	<i>svr</i>	1-[0.4]	2A2	2a200	<i>l(1)2Ac</i>	1-1.0	3A3	3a250	<i>z</i>
1-0.0	1B4-1B8	1b700	<i>su(b)</i>	1-[0.4]	2A2-2A3	2a200	<i>l(1)2Ad</i>	1-1.0	3A4	3a300	<i>l(1)3Ac</i>
1-0.0	1B9	1b800	<i>Appl</i>	1-[0.4]	2A	2a300	<i>l(1)2Ae</i>	1-1.1			<i>bsc</i>
1-0.0	1B9	1b900	<i>vnd</i>	1-[0.4]	2A3-2A5	2a300	<i>sta</i>	1-1.1			<i>ftřdB</i>
1-0.0	1B10-1B11	1b1000	<i>l(1)1Bg</i>	1-[0.4]	2A1-2B4	2b50	<i>Cp70</i>	1-1.1			<i>ung[†]</i>
1-0.0	1B	1b1100	<i>l(1)1Bh</i>	1-[0.4]	2B6	2b100	<i>br</i>	1-1.2			<i>ftřdA</i>
1-0.0	1B	1b1200	<i>l(1)1Bi</i>	1-[0.4]	2B5	2b200	<i>rds</i>	1-1.2	3A6	3a400	<i>l(1)3Ad</i>
1-0.0	1B11-1B12	1b1250	<i>M(1)1B</i>	1-[0.4]	2B4-2B9	2b225	<i>l(1)2Bc</i>	1-1.2	3A7	3a500	<i>l(1)3Ae</i>
1-0.0	1B11-1B13	1b1300	<i>su(s)</i>	1-[0.4]	2B4-2B10	2b250	<i>l(1)2Bd</i>	1-1.2	3A8	3a600	<i>mit(1)15</i>
1-0.0	1B	1b1400	<i>l(1)1Bk</i>	1-[0.4]	2B6	2b400	<i>hfv</i>	1-1.2			<i>pcb</i>
1-0.0	1B13	1c200	<i>mul</i>	1-[0.4]	2B12-2B14	2b500	<i>l(1)2Bg</i>	1-1.2	3B1-3B2	3b150	<i>per</i>
1-[0.0]	1A1		<i>Ars12</i>	1-[0.4]	2B12-2B14	2b500	<i>l(1)2Bh</i>	1-1.2	3B1-3B2	3b175	<i>anon-3B1.2[†]</i>
1-[0.0]	1A8		<i>l(1)DA659[†]</i>	1-[0.4]	2B15	2b600	<i>tlc</i>	1-1.3			<i>mis</i>
1-[0.0]	1B9-1E2		<i>l(1)ESHS1</i>	1-[0.4]	2B	2b700	<i>l(1)2Bj</i>	1-1.3	3A9	3a700	<i>l(1)3Ag</i>
1-[0.0]	1B9-1E2		<i>l(1)ESHS2</i>	1-[0.4]	2B	2b700	<i>l(1)2Bk</i>	1-1.3	3A9	3a800	<i>l(1)3Ah</i>
1-[0.0]	1C1-1C5		<i>NK2</i>	1-[0.4]	2B	2b700	<i>l(1)2Bl</i>	1-1.3	3B1	3b100	<i>sgg</i>
1-[0.0]	1B4-1B9	1b800	<i>elav</i>	1-[0.4]	2B	2b700	<i>l(1)2Bm</i>	1-1.4			<i>l(1)HM13</i>
1-[0.0]	1C	1c100	<i>l(1)1Ca</i>	1-[0.4]	2B	2b700	<i>l(1)2Bn</i>	1-1.4			<i>mit(1)10</i>
1-[0.0]	1C5-1D4	1c300	<i>rw</i>	1-[0.4]	2B	2b700	<i>l(1)2Bo</i>	1-1.4	3B3		<i>par</i>
1-0.1			<i>gen</i>	1-[0.4]	2B	2b700	<i>l(1)2Bp</i>	1-1.4			<i>pte</i>
1-0.1			<i>l(1)CP9[†]</i>	1-[0.4]	2B	2b700	<i>l(1)2Bq</i>	1-1.4			<i>ves</i>
1-0.1			<i>l(1)ne</i>	1-[0.4]	2B	2b700	<i>l(1)2Br</i>	1-1.4	3B2	3b200	<i>l(1)3Bb</i>
1-0.1			<i>om</i>	1-[0.4]	2B	2b700	<i>l(1)2Bs</i>	1-1.4	3B3	3b300	<i>l(1)3Bc</i>
1-0.1	1E1-1E2		<i>su(w^a)</i>	1-[0.4]	2B	2b700	<i>l(1)2Bt</i>	1-1.4	3B4	3b400	<i>l(1)3Bd</i>
1-0.2	2B17		<i>su(w^{sp})</i>	1-[0.4]	2B	2b700	<i>l(1)2Bu</i>	1-1.5	3B4	3b400	<i>e(fa^{swb})</i>
1-0.3			<i>ctt</i>	1-[0.4]	2C1	2c100	<i>l(1)2Ca</i>	1-1.5			<i>l(1)TS56</i>
1-0.3			<i>l(1)63</i>	1-[0.4]	2B17-2D2	2c200	<i>l(1)2Cc</i>	1-1.5			<i>smh</i>
1-0.3			<i>l(1)dn24</i>	1-[0.4]	2B17-2D2	2c200	<i>l(1)2Ce</i>	1-1.5	3B6	3b600	<i>l(1)3Bf</i>
1-0.3			<i>l(1)EN2</i>	1-0.5	2E2-2F1	2e175	<i>fs(1)K10</i>	1-1.5	3C1	3c100	<i>crm</i>
1-0.3			<i>l(1)rr</i>	1-0.5			<i>fs(1)M120</i>	1-1.5	3C2	3c200	<i>w</i>
1-0.3			<i>l(1)ite</i>	1-0.5			<i>gtđ</i>	1-[1.5]	3C		<i>Fcp3C[†]</i>
1-0.3			<i>rsi</i>	1-0.5			<i>l(1)Mb16</i>	1-[1.5]	3C4-3C11		<i>fs(1)K93</i>
1-[0.3]	1E		<i>l(1)IEe</i>	1-0.5	1B14-2B18		<i>mus102</i>	1-[1.5]			<i>fs(1)M2</i>
1-[0.3]	1E		<i>l(1)IEf</i>	1-0.5	2D3-2D4		<i>ph</i>	1-[1.5]	3B4-3B6		<i>fs(1)Ya</i>
1-[0.3]	1F		<i>l(1)IEg</i>	1-0.5	2D4-2D6	2d200	<i>Pgd</i>	1-[1.5]	3B4-3B6		<i>fs(1)Yb</i>
1-[0.3]	1F		<i>l(1)IFf</i>	1-0.5	2D4-2D6	2d300	<i>wapl</i>	1-1.6	3B5	3b500	<i>dwg</i>
1-[0.3]	1E3-1E4		<i>l(1)air2[†]</i>	1-[0.5]	2E		<i>anon-2E[†]</i>	1-1.8			<i>cripA</i>
1-[0.3]	1D	1d100	<i>l(1)1Da</i>	1-[0.5]	2D1-2D3		<i>l(1)air3[†]</i>	1-1.8			<i>fs(1)M104</i>
1-[0.3]	1D	1d200	<i>brc</i>	1-[0.5]	2C2-2D1		<i>l(1)ESHS12</i>	1-1.9			<i>fs(1)M105</i>
1-[0.3]	1D	1d200	<i>l(1)1Dc</i>	1-[0.5]	2D1-2D1		<i>l(1)ESHS13</i>	1-1.9			<i>l(1)ts1</i>
1-[0.3]	1E2	1e100	<i>l(1)IEa</i>	1-[0.5]	2D1-3A		<i>yok</i>	1-2.2	3C5	3c300	<i>rst</i>
1-[0.3]	1E3	1e150	<i>l(1)IEb</i>	1-[0.5]	2C9	2c200	<i>l(1)2Cd</i>	1-2.3	3C5-3C6		<i>vr</i>
1-[0.3]	1E4	1e200	<i>l(1)IEc</i>	1-[0.5]	2C9	2c200	<i>usp</i>	1-2.4			<i>fla</i>
1-[0.3]	1E5	1e400	<i>l(1)IEd</i>	1-[0.5]	2C3	2c600	<i>Actn</i>	1-2.4			<i>PL(1)sp-S1</i>
1-[0.3]	1E3-2A3	1e400	<i>l(1)IEFa</i>	1-[0.5]	2D1-2D2	2d75	<i>l(1)2Da</i>	1-2.5			<i>cpw</i>
1-[0.3]	1E3-2A3	1e400	<i>l(1)IEFb</i>	1-[0.5]	2D2	2d100	<i>csw</i>	1-2.5			<i>l(1)mt</i>
1-[0.3]	1E3-2A3	1e400	<i>l(1)IEFc</i>	1-[0.5]	2D	2d150	<i>l(1)2Dg</i>	1-2.5			<i>l(1)TW3</i>
1-[0.3]	1E3-2A3	1e400	<i>l(1)IEFd</i>	1-[0.5]	2E1	2e50	<i>pn</i>	1-3			<i>l(1)EN12</i>
1-[0.3]	1E3-2A3	1e400	<i>l(1)IEFe</i>	1-0.6	2D3-2F3		<i>aperB</i>	1-3			<i>l(1)M55</i>
1-[0.3]	1E3-2A3	1e400	<i>l(1)IEFf</i>	1-0.6			<i>ebo</i>	1-3			<i>we</i>

CYTOGENETIC MAP

genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol
1-[3]	3C2-3C5		<i>olff</i>	1-10.2	4E2	4e200	<i>ovo</i>	1-18			<i>dep</i>
1-[3]	3C4-3C5	3c250	<i>irreC</i> [†]	1-10.8			<i>mus108</i>	1-18			<i>Eyl</i>
1-[3]	3C11-3C12	3c500	<i>Pig1</i>	1-11			<i>l(1)M47</i>	1-18			<i>fs(1)A508</i>
1-3.0	3C7	3c350	<i>N</i>	1-11			<i>lzl</i>	1-18			<i>l(1)ER</i>
1-3.0	3C11-3C12	3c600	<i>Sgs4</i>	1-11.0	4E	4e300	<i>rg</i>	1-18			<i>l(1)tsUC34</i>
1-[3.0]	3C5-3D4		<i>l(1)ESHS18</i>	1-11.3			<i>apx</i>	1-18]	6C11-6E5		<i>gustE</i>
1-[3.0]	3C11-3C12	3c400	<i>ngp</i> [†]	1-11.5			<i>rgt</i>	1-18]	6D		<i>l(1)6Da</i>
1-3.1			<i>im</i>	1-11.7	4F7-4F11	4f100	<i>fs(1)A1621</i>	1-18]	6D		<i>l(1)6Db</i>
1-3.2			<i>sfc</i>	1-12			<i>l(1)M46</i>	1-18]	6D		<i>l(1)6Dc</i>
1-3.3			<i>fbr</i>	1-12			<i>nob</i>	1-18]	6D		<i>l(1)6Dd</i>
1-3.3			<i>rud</i>	1-12.5	4F7-5A2		<i>fs(1)MY18</i> [†]	1-18]	6D		<i>l(1)6De</i>
1-3.6			<i>mtb</i>	1-12.8			<i>omm</i>	1-18]	6D		<i>l(1)6Df</i>
1-3.7			<i>sth</i>	1-13			<i>Cb</i>	1-18]	6D		<i>l(1)6Dg</i>
1-3.7	3D4	3d100	<i>sam</i>	1-13			<i>l(1)EN8</i>	1-18]	6D		<i>l(1)6Dh</i>
1-3.9	3D4	3d200	<i>dnc</i>	1-13			<i>l(1)M64</i>	1-18]	6C-7A3		<i>l(1)ESHS24</i>
1-[4]	3D6	3d300	<i>l(1)3Da</i>	1-13			<i>l(1)XN75b</i> [†]	1-18]	6C-7A3		<i>l(1)ESHS25</i>
1-[4]	3E1	3e100	<i>slc</i>	1-13	5D5-5D6		<i>sqh</i>	1-18]	6C-7A3		<i>l(1)ESHS26</i>
1-[4]	3E2	3e200	<i>l(1)3Eb</i>	1-[13]	5A8-5C1		<i>fs(1)M14</i>	1-18]	6C-7A3		<i>l(1)ESHS27</i>
1-[4]	3E3	3e300	<i>l(1)3Ec</i>	1-[13]	5A1-5A8		<i>l(1)air5</i> [†]	1-18]	6C-7A3		<i>l(1)ESHS28</i>
1-4.0	3D5	3d300	<i>dm</i>	1-[13]	5A1-5A8		<i>l(1)air6</i> [†]	1-18]	6C-7A3		<i>l(1)ESHS29</i>
1-4.0			<i>ftrdC</i>	1-[13]	5A6-5A13		<i>M(1)5A</i>	1-18]	6C-7A3		<i>l(1)ESHS30</i>
1-[4.0]	3D4-3D5		<i>l(1)ESHS19</i>	1-[13]	5A8-5C6		<i>vrw</i>	1-18.0			<i>tnl</i>
1-4.4			<i>rv</i>	1-13.3			<i>dvw</i>	1-18.3	6E1-6E2	6e100	<i>l(1)6Ea</i>
1-4.5			<i>rta</i>	1-13.3			<i>dwf</i>	1-18.3	6E4	6e200	<i>l(1)6Eb</i>
1-4.5			<i>Sc</i>	1-13.3			<i>fs(1)42</i>	1-18.5			<i>l(1)dn13</i>
1-5			<i>fs(1)M103</i>	1-13.4			<i>l(1)EN13</i>	1-18.5			<i>l(1)ds10</i>
1-5			<i>opht</i>	1-13.6	5A-5B		<i>cx</i>	1-18.8			<i>iav</i>
1-5			<i>su(dx)</i>	1-13.6	5A10-5B3		<i>Tre</i>	1-18.8	6E2-6E4	6e300	<i>ogre</i>
1-[5]3	E2-3E8		<i>l(1)ESHS20</i>	1-13.7	5D-6A		<i>M(1)5D6A</i>	1-18.8	6E1-6E5	6e400	<i>l(1)6Ed</i>
1-[5]	3E2-3E8		<i>l(1)ESHS21</i>	1-13.7	5A10-5C3		<i>mus105</i>	1-18.9	6E5-6E6	6e450	<i>l(1)6Ee</i>
1-[5]	3E2-3E8		<i>l(1)ESHS22</i>	1-13.7	5B	5b100	<i>cv</i>	1-18.9	6E5	6e500	<i>cm</i>
1-[5]	3E2-3E8		<i>l(1)ESHS23</i>	1-13.9			<i>dfa</i>	1-19			<i>fs(1)A1304</i>
1-[5]	3E1		<i>l(1)VE807</i> [†]	1-14	5C7-5D6		<i>l(1)M13</i>	1-[19]	6F1-6F2	6f50	<i>nullo</i> [†]
1-[5]	3E3-3E4		<i>M(1)3E</i>	1-14			<i>l(1)jl</i>	1-[19]	6F5	6f200	<i>l(1)6Fb</i>
1-[5]	3E4-3E6	3e400	<i>l(1)3Ed</i>	1-14			<i>refl1JH</i>	1-[19]	6F7	6f300	<i>l(1)6Fc</i>
1-[5]	3E7	3e500	<i>l(1)3Ee</i>	1-[14]	5A6		<i>Mlc-c</i>	1-19.2	6F5	6f100	<i>Sxl</i>
1-5.0			<i>rub</i>	1-[14]	5D5-5E1		<i>fs(1)K1214</i>	1-19.3			<i>l(1)J1024</i>
1-5.5			<i>l(1)55a</i>	1-[14]	5C-5D		<i>fs(1)ph</i> [†]	1-19.3	6A3-6F11		<i>scp</i>
1-5.5			<i>mf</i>	1-[14]	5C		<i>Hsc70-6</i> [†]	1-19.4	7A3	7a100	<i>l(1)7Aa</i>
1-5.5	3E8-3F1	4b100	<i>ec</i>	1-[14]	5A1-5E8		<i>l(1)air4</i> [†]	1-19.5			<i>l(1)ds8</i>
1-5.5	3F1-4B1	4b200	<i>cho</i>	1-[14]	5C	5c(100)	<i>anon-5C</i> [†]	1-19.5			<i>ryb-2</i>
1-[5.5]	3F1-4B1	4b250	<i>mdl</i>	1-[14]	5C2-5C5	5c(200)	<i>Act5C</i>	1-19.6	7A6-7A8	7a200	<i>l(1)7Ab</i>
1-5.6			<i>te</i>	1-14.1			<i>l(1)CP7</i> [†]	1-19.8	7A6	7a300	<i>l(1)7Ac</i>
1-5.7			<i>Oce</i>	1-14.3			<i>mur</i>	1-19.9	5C-6C11		<i>Fum</i>
1-5.8			<i>l(1)TS45</i>	1-14.4			<i>rmp</i>	1-19.9	7A8	7a400	<i>l(1)7Ad</i>
1-5.9			<i>e(g)</i>	1-14.5			<i>l(1)dn4</i>	1-20			<i>l(1)ts6225</i>
1-6	4A5-4B1		<i>l(1)C</i>	1-14.6			<i>pls</i>	1-20			<i>l(1)XN82b</i> [†]
1-6	4B1-4C12	4b300	<i>fs(1)se</i> [†]	1-14.7			<i>stb</i>	1-20	7A2-7C1		<i>olfC</i>
1-[6]	4B1-4B2		<i>Fas2</i> [†]	1-15			<i>syn</i>	1-20			<i>rde</i>
1-6.0			<i>l(1)ts504</i>	1-15	5C5-5C6		<i>ccd</i>	1-[20]	7A5-7C1		<i>iav</i>
1-6.5	4B4-4B6	4b400	<i>mei9</i>	1-15	5C5-5D6		<i>fs(1)K646</i>	1-[20]	7A2-7A8		<i>l(1)ESHS31</i>
1-6.5	4B6-4C1	4b500	<i>norpA</i>	1-15			<i>fs(1)M3</i>	1-20.0	7B3	7b100	<i>ct</i>
1-6.7			<i>mo</i>	1-15.0	5D2-5D6		<i>fs(1)M17</i>	1-20.2			<i>tre</i>
1-6.8	4C5-4C6	4c300	<i>Qd</i>	1-15.1	5C5-5D6		<i>l(1)5Cda</i>	1-20.4			<i>stu</i>
1-6.8	4C7-4C8	4c600	<i>amb</i>	1-15.2			<i>Ext</i>	1-20.7	7B4-7C1		<i>ag</i>
1-[6.8]	4B5-4C6		<i>M(1)4BC</i> [†]	1-15.2			<i>l(1)E7</i>	1-20.7			<i>l(1)E34</i>
1-6.9	4C5-4C6	4c100	<i>bi</i>	1-15.2			<i>pra</i>	1-20.8	7C3		<i>lgh</i>
1-7			<i>l(1)Mb7</i>	1-15.3			<i>mit(1)2</i>	1-20.8			<i>dec1</i>
1-7	4C5-4C6	4c800	<i>hnt</i>	1-15.5			<i>l(1)E25</i>	1-20.9			<i>pvt</i>
1-[7]	4C		<i>ecl</i>	1-15.5			<i>l(1)Mb28</i>	1-20.9			<i>sht</i>
1-[7]	4C5-4C6	4c200	<i>l(1)bi</i> [†]	1-15.5			<i>torp</i>	1-21	7D8		<i>bis</i>
1-7.3			<i>l(1)ts958</i>	1-15.7			<i>mit(1)7</i>	1-21	7C4-8C2		<i>clw</i>
1-7.3	4C5-4C6	4c400	<i>lac</i>	1-15.9	5E6-5E7		<i>swa</i>	1-21	7D1-7D6		<i>fs(1)M111</i>
1-7.5	4C5-4C6	4c500	<i>omb</i>	1-16			<i>yea2</i>	1-21	7D5-7D6	7dd100	<i>gs</i>
1-7.5	4C5-4C7	4c600	<i>peb</i>	1-16.0			<i>sct</i>	1-21]	7C1-7C9		<i>fs(1)h</i>
1-7.5	4C6	4c600	<i>rb</i>	1-16.2			<i>wgo</i>	1-[21]	7C-7D		<i>Fcp7C</i> [†]
1-[7.5]	4C7-4C15		<i>rap</i>	1-16.3			<i>l(1)1074-ts</i>	1-[21]	7C-7D		<i>fs(1)M73</i>
1-8			<i>l(1)ts</i>	1-16.3	6B1		<i>vs</i>	1-[21]	7C-7D		<i>fs(1)M122</i>
1-8.0			<i>dow</i>	1-16.5			<i>cb</i>	1-[21]	7C5-7C9		<i>L(1)7C</i>
1-8.0			<i>l(1)trs</i>	1-16.6			<i>l(1)ts1126</i>	1-[21]	7D1-8A5		<i>Is</i> [†]
1-8.7			<i>mib</i>	1-17	5D5-6E1		<i>fs(1)K254</i>	1-[21]	7C4-7C9		<i>l(1)air8</i> [†]
1-8.8			<i>cripB</i>	1-[17]	5D1-6E1		<i>fs(1)K741</i>	1-[21]	7C9-7D1 or		<i>l(1)air9</i> [†]
1-9			<i>fs(1)A1371</i>	1-[17]	5D5-6E1		<i>fs(1)K1274</i>	1-[21]	7D5-7D10		
1-9.5			<i>l(1)dn15</i>	1-[17]	5E		<i>Ubi-f</i> [†]	1-[21]	7C9-7D1 or		<i>l(1)air10</i> [†]
1-9.8	4E1-4E2	4e100	<i>svb</i>	1-17.0			<i>dx</i>	1-[21]	7D5-8A5		
1-10			<i>l(1)EN9</i>	1-17.0	6A3-6A11		<i>grg</i>	1-[21]	7B2-7C1		<i>l(1)ESHS32</i>
1-10			<i>l(1)ml</i>	1-17.0			<i>l(1)ts612</i>	1-[21]	7B2-7C1		<i>l(1)ESHS33</i>
1-[10]	3E8-4F11		<i>ft(1)302</i>	1-17.4			<i>rss</i>	1-[21]	7C3-7D1		<i>l(1)ESHS34</i>
1-[10]	4F1-5A2		<i>fs(1)A456</i>	1-17.5			<i>lem</i>	1-[21]	7C3-7D1		<i>l(1)ESHS35</i>
1-[10]	4F1-5A2		<i>fs(1)A1059</i>	1-17.5			<i>ov</i>	1-[21]	7C5-7C9		<i>l(1)hen</i> [†]
1-[10]	4E1-4E2		<i>Pr4E</i> [†]	1-17.5			<i>tmc</i>	1-[21]	7C		<i>M(1)7C</i>
1-10.2			<i>fs(1)M1</i>	1-17.9			<i>l(1)sd</i>	1-[21]	7C1-7C9		<i>olfA</i>
1-10.2			<i>fs(1)M38</i>	1-17.9	6A3-6E5		<i>shf</i>	1-[21]	7C1-7C2		<i>rex</i>
									7C5-7C9		<i>RpS14A</i>

THE GENOME OF *DROSOPHILA MELANOGASTER*

genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol
1-[21]	7C5-7C9		<i>RpS14B</i>	1-[23]	8A4	8a400	<i>l(1)8Ad</i>	1-29.6			<i>fin</i>
1-[21]	7D22		<i>sdt</i>	1-23.0			<i>dbl</i>	1-29.7			<i>l(1)dd9</i>
1-[21]	7D13-7D14		<i>sesD</i>	1-23.1	7E10-8A5		<i>dec2</i>	1-29.8			<i>sesE</i>
1-[21]	7B5	7b300	<i>l(1)7Bc</i>	1-23.1			<i>l(1)8</i>	1-29.8			<i>sto</i>
1-[21]	7B5	7b400	<i>l(1)7Bd</i>	1-23.1			<i>smb</i>	1-29.9			<i>sma</i>
1-[21]	7B	7b400	<i>l(1)7Be</i>	1-23.1	7E11	7e700	<i>pt</i>	1-30	8E-9B1		<i>Yp1</i>
1-[21]	7B	7b400	<i>l(1)7Bf</i>	1-23.1	7F10	7f500	<i>gg</i>	1-30	8E-9B1		<i>Yp2</i>
1-[21]	7C1	7c100	<i>l(1)7Ca</i>	1-23.1	8A1-8A2	8a300	<i>oc</i>	1-[30]	9B-9C		<i>fs(1)A1561</i>
1-[21]	7C2	7c200	<i>l(1)7Cb</i>	1-23.2	8A5		<i>ptg</i>	1-[30]	9A2-9B2		<i>l(1)ESHS40</i>
1-[21]	7C4	7c300	<i>l(1)7Cc</i>	1-23.3			<i>sesA</i>	1-30.0	8F1-8F2		<i>M(1)8F</i>
1-[21]	7C9-7D1	7c350	<i>ofjE</i>	1-23.4			<i>ccw</i>	1-30.1			<i>l(1)Mb15</i>
1-[21]	7C5	7c500	<i>l(1)7Cd</i>	1-23.5			<i>l(1)Mc39</i>	1-30.2	9A2-9A5	9e100	<i>mus109</i>
1-[21]	7C	7c600	<i>l(1)7Ce</i>	1-23.6			<i>firdM</i>	1-30.5			<i>Hk</i>
1-[21]	7C	7c600	<i>l(1)7Cf</i>	1-23.6	7E10-8A5		<i>fs(1)M72</i>	1-30.6			<i>scd</i>
1-[21]	7C	7c600	<i>l(1)7Cg</i>	1-23.6	7F1	7f100	<i>Nrg</i>	1-31	8A5-9A1		<i>btd</i>
1-[21]	7C8	7c900	<i>l(1)7Ch</i>	1-23.8			<i>ch-b</i>	1-31			<i>fs(1)A97</i>
1-[21]	7D	7d200	<i>l(1)7Dc</i>	1-24			<i>jyx</i>	1-31			<i>oml</i>
1-[21]	7D	7d200	<i>l(1)7Dd</i>	1-24			<i>l(1)EN16</i>	1-31	9C	9e150	<i>ftw</i>
1-[21]	7D	7d200	<i>l(1)7De</i>	1-24.3	7C4-8C2		<i>dd</i>	1-31			<i>pby</i>
1-[21]	7D	7d200	<i>l(1)7Df</i>	1-24.6			<i>bre</i>	1-31	9D1-9E1		<i>gual</i>
1-[21]	7D	7d700	<i>l(1)7Dg</i>	1-24.6			<i>svs</i>	1-[31]	9C1-9C2		<i>Pp1b-9C</i>
1-[21]	7D1	7d800	<i>l(1)7Dh</i>	1-24.9			<i>dlg2</i>	1-31.6	9A2-9B1		<i>mw</i>
1-[21]	7D	7d900	<i>l(1)7Di</i>	1-25			<i>l(1)Mc56</i>	1-31.8	9D1-9E2	9e200	<i>Hmr</i>
1-[21]	7D12	7d1000	<i>l(1)7Dj</i>	1-25			<i>ms(1)413</i>	1-32			<i>aw</i>
1-[21]	7D	7d1100	<i>l(1)7Dk</i>	1-25			<i>sml</i>	1-32			<i>e(w^e)</i>
1-[21]	7D	7d1200	<i>l(1)7Dl</i>	1-25	7E1-8C2		<i>tbd</i>	1-[32]	9E		<i>Cg9E</i>
1-[21]	7D5-7D6	7dd200	<i>mys</i>	1-25.0			<i>l(1)ts445a</i>	1-[32]	9E3-11B2		<i>fs(1)M78</i>
1-21.0	7B2-7C1		<i>fiiC</i>	1-25.1			<i>elr</i>	1-[32]	9E3-10A1		<i>fs(1)M116</i>
1-21.0	7D1-7D2	7c400	<i>sn</i>	1-25.1	7E10-8A5		<i>fs(1)M112</i>	1-[32]	9E1-9F3		<i>l(1)air12⁺</i>
1-21.1			<i>sch</i>	1-25.4			<i>rea</i>	1-[32]	9E4		<i>l(1)DC701⁺</i>
1-21.3	7D12-7D14		<i>l(1)adl1</i>	1-25.5			<i>fiiD</i>	1-[32]	9F7		<i>l(1)EA61⁺</i>
1-21.4			<i>mit(1)11</i>	1-25.6			<i>smp</i>	1-[32]	9F1		<i>l(1)EA79⁺</i>
1-21.6			<i>dfw</i>	1-25.7			<i>l(1)dd2</i>	1-[32]	9E1-9E4		<i>l(1)ESHS41</i>
1-21.7			<i>l(1)nc1</i>	1-25.8	8A5-9A2		<i>ctl</i>	1-[32]	9F9		<i>l(1)VE828⁺</i>
1-21.7	7D14-7D22		<i>lon</i>	1-25.9			<i>dlv</i>	1-[32]	9F		<i>mit(1)21⁺</i>
1-22			<i>rdp</i>	1-26			<i>asx</i>	1-[32]	9E1-9E2	9e300	<i>l(1)9Ea</i>
1-22			<i>l(1)dn25</i>	1-26	7E-8A		<i>fs(1)A1242</i>	1-[32]	9E3-9E4	9e600	<i>l(1)9Ec</i>
1-22			<i>l(1)HM22</i>	1-26			<i>l(1)dn11</i>	1-[32]	9E7-9E8	9e700	<i>l(1)9Ed</i>
1-22			<i>sws</i>	1-26			<i>l(1)YL21⁺</i>	1-[32]	9E7-9E8	9e800	<i>l(1)9Ee</i>
1-[22]	7D14-7E10		<i>fs(1)201⁺</i>	1-26.0			<i>l(1)ts982</i>	1-[32]	9F3-9F4	9f100	<i>fiiK</i>
1-[22]	7D14-7E10		<i>fs(1)A5⁺</i>	1-26.3			<i>l(1)ds11</i>	1-[32]	9F7	9f350	<i>l(1)9Fb</i>
1-[22]	7E-8A		<i>fs(1)A473</i>	1-26.3			<i>l(1)TW1</i>	1-[32]	9F9-9F12	9f500	<i>l(1)9Ff</i>
1-[22]	7D14-7E10		<i>fs(1)gb3⁺</i>	1-26.3	8A4-8C6		<i>rdgA</i>	1-32.2			<i>dwx</i>
1-[22]	7D14-7E10		<i>fs(1)gb4⁺</i>	1-26.5	8D10-9A2		<i>mex⁺</i>	1-32.2			<i>l(1)ds2</i>
1-[22]	7D5-7D10		<i>l(1)ESHS36</i>	1-26.8			<i>fiiE</i>	1-32.4			<i>l(1)Mc32</i>
1-[22]	7D5-7D10		<i>l(1)ESHS37</i>	1-26.8			<i>l(1)Mb26</i>	1-32.4			<i>pat</i>
1-[22]	7D10-8A5		<i>l(1)ESHS38</i>	1-27			<i>l(1)M82</i>	1-32.4			<i>pur1</i>
1-[22]	7D10-8A5		<i>l(1)ESHS39</i>	1-27			<i>l(1)Mc52</i>	1-32.4	9E3-9E4	9e400	<i>ras</i>
1-[22]	7D22	7d1300	<i>l(1)7Dm</i>	1-27			<i>Lg</i>	1-32.4	9E7-9F4	9e550	<i>sesB</i>
1-[22]	7E	7e100	<i>l(1)7Ea</i>	1-27			<i>mus111</i>	1-32.5			<i>df</i>
1-[22]	7E4	7e200	<i>l(1)7Eb</i>	1-27.1	7E1-8C2		<i>con</i>	1-32.6			<i>clm</i>
1-[22]	7E6	7e300	<i>l(1)7Ec</i>	1-27.2			<i>ddl</i>	1-32.6	9F5-9F11	9f200	<i>sbr</i>
1-[22]	7E	7e400	<i>l(1)7Ed</i>	1-27.2			<i>l(1)dd11</i>	1-32.6	9F5-9F11	9f400	<i>l(1)9Fe</i>
1-[22]	7E	7e500	<i>l(1)7Ee</i>	1-27.3			<i>dss</i>	1-32.7	9F12		<i>fs(1)BP</i>
1-[22]	7D10-7F2	7e600	<i>l(1)7Ef</i>	1-27.3			<i>tar</i>	1-32.7	9F12	9f300	<i>fiiG</i>
1-22.0			<i>scr</i>	1-27.5			<i>l(1)dd3</i>	1-32.8	9E		<i>mus112⁺</i>
1-22.0			<i>su(stn)</i>	1-27.5			<i>l(1)dhl</i>	1-32.9			<i>ww</i>
1-22.1			<i>aperA</i>	1-27.5	8C3-8C17		<i>t</i>	1-33			<i>brd</i>
1-22.1			<i>coi</i>	1-27.7	8D4-8E2		<i>amx</i>	1-33			<i>l(1)Xi08⁺</i>
1-22.1			<i>l(1)CP8⁺</i>	1-27.7	8D8-8D9		<i>lz</i>	1-[33]	10A-10BC		<i>Cg10AB</i>
1-22.4			<i>l(1)Mb24</i>	1-27.7			<i>su(r)</i>	1-[33]	10A2-11B2		<i>fs(1)M26</i>
1-22.4			<i>shm</i>	1-27.8			<i>tha</i>	1-[33]	10A2-11B2		<i>fs(1)M46</i>
1-22.6			<i>spx</i>	1-28			<i>l(1)M58</i>	1-[33]	10A2-11B2		<i>fs(1)M62</i>
1-22.7			<i>ha</i>	1-[28]	8B-8C		<i>fs(1)C3</i>	1-[33]	9F3-9F6		<i>l(1)ESHS42</i>
1-23			<i>ceb</i>	1-28.0			<i>mit(1)4</i>	1-[33]	9F3-9F6		<i>l(1)ESHS43</i>
1-22.7	7F1		<i>otu</i>	1-28.1	8D8-8D9		<i>dvr</i>	1-[33]	10A2-10A3		<i>l(1)L13⁺</i>
1-23			<i>depl</i>	1-28.1			<i>firdD</i>	1-33.0			<i>osh</i>
1-23			<i>dis</i>	1-28.3			<i>l(1)ts340</i>	1-33.0			<i>wgv</i>
1-23			<i>Etd</i>	1-28.3			<i>opb</i>	1-33.0	9F12	9f350	<i>fs(1)9F⁺</i>
1-23			<i>l(1)HM8</i>	1-28.5			<i>gra</i>	1-33.0	9F13-10A1	9f600	<i>l(1)9Fg</i>
1-23			<i>l(1)XC78⁺</i>	1-28.6			<i>ke</i>	1-33.0	9F13-10A1	9f800	<i>l(1)9Fh</i>
1-23			<i>lix</i>	1-28.8	8A5-9A2		<i>bly</i>	1-33.0	10A1-10A2	10a100	<i>v</i>
1-[23]	7F1-7F2		<i>Cp36</i>	1-29			<i>l(1)tsUC13</i>	1-33.1	10A1-10A2	10a200	<i>csk</i>
1-[23]	7F1-7F2		<i>Cp38</i>	1-29			<i>fs(1)A59</i>	1-33.4	10A1-10A2	10a300	<i>sev</i>
1-[23]	7E10-8A3		<i>fs(1)2448⁺</i>	1-[29]	8F-9A		<i>fs(1)K79</i>	1-33.4	10A1-10A5	10a400	<i>ms(1)10A</i>
1-[23]	7E10-8A3		<i>fs(1)B4⁺</i>	1-[29]	8E-9B1		<i>fs(1)M49</i>	1-33.5	10A3-10A5	10a600	<i>tny</i>
1-[23]	7E10-8A3		<i>fs(1)ga6</i>	1-[29]	9A		<i>fs(1)M52⁺</i>	1-33.5	10A8	10a950	<i>fs(1)10A</i>
1-[23]	7E10-8A3		<i>fs(1)ga7⁺</i>	1-[29]	8E-8F		<i>l(1)mbn⁺</i>	1-33.6	10A6-10A7	10a700	<i>l(1)10Ad</i>
1-[23]	7E10-8A3		<i>fs(1)ga8⁺</i>	1-[29]	8D		<i>l(1)air11⁺</i>	1-33.6	10A6-10A7	10a800	<i>l(1)10Ae</i>
1-[23]	7F1-8C6		<i>su(Cbx)</i>	1-29.0	8A5-9A2		<i>me</i>	1-33.6	10A8	10a900	<i>l(1)10Af</i>
1-[23]	7F3-7F4	7f300	<i>l(1)7Fc</i>	1-29.0			<i>fitF</i>	1-33.7			<i>dfi</i>
1-[23]	7F	7f400	<i>l(1)7Fd</i>	1-29.1	8E-9D		<i>dot⁺</i>	1-33.7	10A3-10A5	10a500	<i>slm</i>
1-[23]	8A1	8a100	<i>l(1)8Aa</i>	1-29.2			<i>Hex-A</i>	1-33.8	10A6-10B3		<i>gs(1)N26</i>
1-[23]	8A2	8a200	<i>l(1)8Ab</i>	1-29.2	8D4-8E1						

CYTOGENETIC MAP

genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol
1-34			<i>l(1)HM3</i>	1-[37]	10E4		<i>l(1)VE817[†]</i>	1-43	12A6-13A5		<i>ota1</i>
1-34			<i>l(1)M53</i>	1-[37]	10F1-10F6	10f100	<i>l(1)10Fa</i>	1-[43]	12A6-12D3		<i>fs(1)J288</i>
1-34			<i>l(1)ts5141</i>	1-[37]	10F1-10F6	10f100	<i>l(1)10Fb</i>	1-[43]	12AB-12C		
1-[34]	10B1-10B17		<i>fs(1)M43</i>	1-[37]	10F1-10F6	10f100	<i>l(1)10Fc</i>	1-43.0	11F2-12A2	11f400	<i>s</i>
1-[34]	10B8-10B11		<i>GsII</i>	1-[37]	10F1-10F6	10f100	<i>l(1)10Fd</i>	1-43.2			<i>bla</i>
1-[34]	10B3-10C		<i>l(1)ESHS44</i>	1-[37]	10F5	10f100	<i>l(1)10Fe</i>	1-43.3			<i>cop</i>
1-[34]	10A9-10A12	10a1100	<i>l(1)10Ah</i>	1-[37]	10F7	10f600	<i>l(1)10Ff</i>	1-43.5			<i>l(1)Mc23</i>
1-[34]	10A9-10A12	10a1200	<i>l(1)10Ai</i>	1-[37]	10F6	10f600	<i>l(1)10Fg</i>	1-43.9			<i>mit(1)17[†]</i>
1-[34]	10A9	10a1300	<i>rtv</i>	1-[37]	10F8	10f600	<i>l(1)10Fh</i>	1-43.9			<i>ten</i>
1-[34]	10B4-10B17	10b500	<i>ny</i>	1-[37]	10F9	10f900	<i>l(1)10Fi</i>	1-44			<i>l(1)M4</i>
1-[34]	10B4-10B9	10b300	<i>l(1)10Bc</i>	1-[37]	10F10	10f1000	<i>l(1)10Fj</i>	1-44			<i>l(1)M52</i>
1-34.3			<i>stt</i>	1-[37]	10F11	10f1000	<i>l(1)10Fk</i>	1-44	12A6-12D3		<i>Yp3</i>
1-34.3	10B1-10B3	10a1100	<i>tu(1)Sz</i>	1-[37]	10F1-10F10	10f1200	<i>qs</i>	1-[44]	12D		<i>fs(1)M79</i>
1-34.3	10B4-10B9	10b100	<i>l(1)10Ba</i>	1-[37]	11A2	11a100	<i>cac</i>	1-[44]	12E1-12E2	12e100	
1-34.3	10B4	10b100	<i>sis-a</i>	1-[37]	11A3	11a400	<i>tsg</i>	1-[44]	12E1-12E2	12e200	
1-34.4	10B4-10B9	10b200	<i>l(1)10Bb</i>	1-[37]	11A	11a600	<i>l(1)11Ac</i>	1-[44]	12E1-12E2	12e300	
1-34.5	10B6	10b400	<i>dsh</i>	1-[37]	11A	11a600	<i>l(1)11Ad</i>	1-[44]	12E1-12E2	12e400	<i>tRNA:ser7:12Ed</i>
1-34.6	10B7	10b500	<i>hop</i>	1-37.0	11A6	11a800	<i>l(1)11Ae</i>	1-[44]	12E1-12E2	12e500	<i>tRNA:ser4:12Ee</i>
1-34.7			<i>rdp</i>	1-37.1			<i>twt</i>	1-[44]	12E1-12E2	12e600	<i>tRNA:ser774:12Ef</i>
1-34.8	10B8-10B9	10b600	<i>dlg1</i>	1-37.2	10		<i>e(y)6</i>	1-[44]	12E1-12E2	12e700	<i>tRNA:ser7:12Eg</i>
1-34.9			<i>fnc</i>	1-37.2			<i>ob</i>	1-[44]	12E1-12E2	12e800	<i>tRNA:ser474:12Eh</i>
1-34.9			<i>mit(1)6</i>	1-37.2			<i>plw</i>	1-44.2	12B1-12B2		<i>mus101</i>
1-35			<i>l(1)M66</i>	1-37.5			<i>fs(1)5e</i>	1-44.4	12B6-12B7		<i>g</i>
1-35			<i>l(1)ts2864</i>	1-38			<i>fs(1)A214</i>	1-44.4	12B2-12B6		<i>l(1)dd4</i>
1-[35]	10C3-10D5		<i>dsl</i>	1-38			<i>l(1)ts3733</i>	1-44.4			<i>l(1)Mc19</i>
1-[35]	10C		<i>e(y)2</i>	1-38			<i>ms(1)516</i>	1-44.5	12A6-12D3		<i>ty</i>
1-[35]	10C5		<i>l(1)DF912[†]</i>	1-[38]	11A6-11A7		<i>mfd</i>	1-44.6			<i>dyb</i>
1-[35]	10C8		<i>l(1)VE623[†]</i>	1-[38]	11A6-11A9		<i>Ten11A[†]</i>	1-44.6			<i>frdG</i>
1-[35]	10B17-10C1	10b750	<i>l(1)10B1</i>	1-38.0			<i>frdN</i>	1-44.6			<i>frdP</i>
1-[35]	10B17-10C1	10b750	<i>l(1)10Bm</i>	1-38.0			<i>gli</i>	1-44.7	12D3-12E1		<i>fs(1)K1563</i>
1-[35]	10B17-10C2	10b1000	<i>l(1)10Bi</i>	1-38.2			<i>shakA</i>	1-44.9	11A7-13F10		<i>l(1)air14[†]</i>
1-[35]	10B17-10C2	10b1100	<i>l(1)10Bj</i>	1-38.3			<i>alo</i>	1-45			<i>cbf</i>
1-[35]	10B17-10C2	10b1200	<i>l(1)10Bk</i>	1-38.4			<i>mn</i>	1-45			<i>gustA</i>
1-35.0	10A8-10A11		<i>stf</i>	1-38.9	11A-11B		<i>agn</i>	1-45			<i>hypoC</i>
1-35.1	10B8-10B15	10b700	<i>l(1)10Bg</i>	1-38.9			<i>wrw</i>	1-45			<i>l(1)tsUC32</i>
1-35.3	10B8-10B15	10b800	<i>l(1)10Bh</i>	1-39			<i>l(1)M39</i>	1-45			<i>l(1)tsUC88</i>
1-35.4	11A7-13F10		<i>l(1)air13[†]</i>	1-39.0			<i>shb</i>	1-45			<i>yea6[†]</i>
1-35.4	10C2-10D4		<i>Rst(1)JH</i>	1-39.1			<i>cff</i>	1-[45]	12E1-12E11		<i>har[†]</i>
1-35.7			<i>ano</i>	1-39.2			<i>hypoE</i>	1-[45]	12D3		<i>wapm</i>
1-35.7	10C1-10C2	10c100	<i>Rpl1215</i>	1-39.5	11A7-11A9		<i>Lsp1-a</i>	1-45.2	12E1-13A5		<i>na</i>
1-35.7	10C3-10C5	10c200	<i>tyl</i>	1-39.6	11A7-11B9		<i>gs(1)N41</i>	1-45.3			<i>frdO</i>
1-35.7	10C1-10C5	10c300	<i>l(1)10Cc</i>	1-39.7			<i>l(1)Mb22</i>	1-45.3			<i>slb</i>
1-35.7	10C1-10C5	10c400	<i>l(1)10Cd</i>	1-39.8			<i>brw</i>	1-45.6			<i>abt</i>
1-35.8			<i>l(1)dn21</i>	1-39.9			<i>frdE</i>	1-45.7			<i>smn</i>
1-35.8			<i>tb</i>	1-40			<i>fs(1)K4[†]</i>	1-45.7	12F1-12F2	12e900	<i>Ste</i>
1-35.8	10A1-10A7		<i>Tum</i>	1-40			<i>nonB</i>	1-46			<i>l(1)EN1</i>
1-35.9	10C3-10D5	10c600	<i>l(1)10Ce</i>	1-40.1			<i>comt</i>	1-46			<i>l(1)M25</i>
1-36			<i>cast</i>	1-40.3	11F2-12A2	11f300	<i>crt</i>	1-46			<i>l(1)ts1704</i>
1-36			<i>fs(1)A180</i>	1-40.7	11E	11e800	<i>wy</i>	1-46			<i>l(1)ts1843</i>
1-36	10C2-10C3	10c500	<i>nod</i>	1-40.8			<i>som</i>	1-46	12F5-12F7		<i>rut</i>
1-[36]	10E1-10E2		<i>Ckl1B</i>	1-41			<i>ups</i>	1-46.1			<i>mit(1)3</i>
1-[36]	10E1-10E2		<i>gustB</i>	1-41			<i>cbd</i>	1-46.1			<i>stp</i>
1-[36]	10E1-10E2		<i>gustC</i>	1-41			<i>l(1)M26</i>	1-46.7			<i>l(1)dd10</i>
1-[36]	10E1-10E4		<i>gustD</i>	1-41			<i>l(1)M36</i>	1-46.9			<i>fs(1)A120</i>
1-[36]	10E3-10E4		<i>Hsc70-3[†]</i>	1-41			<i>tu(1)53</i>	1-47			<i>l(1)EN5</i>
1-[36]	10D1		<i>l(1)C154[†]</i>	1-[41]	11C		<i>prd5[†]</i>	1-[47]	12E1-13A5		<i>fs(1)M20</i>
1-[36]	10E6		<i>l(1)DF939[†]</i>	1-[41]	11D7-11E4		<i>PS1a[†]</i>	1-47.5			<i>shp</i>
1-[36]	10D7		<i>l(1)VE742[†]</i>	1-[41]	11D-11E		<i>rad[†]</i>	1-47.8			<i>lme</i>
1-[36]	10D1-10D2		<i>Pt10D[†]</i>	1-[41]	11D	11d100	<i>l(1)11Da</i>	1-47.9	13B2-13F17		<i>pl</i>
1-[36]	10D4-10E1	10d100	<i>l(1)10Da</i>	1-[41]	11D	11d100	<i>l(1)11Db</i>	1-48			<i>gcd</i>
1-[36]	10D4-10E1	10d100	<i>l(1)10Db</i>	1-[41]	11D	11d100	<i>l(1)11Dc</i>	1-48	12E1-12F1		<i>yl</i>
1-[36]	10D4-10E1	10d100	<i>l(1)10Dc</i>	1-[41]	11E	11e200	<i>l(1)11Eb</i>	1-[48]	12F5-13A		<i>irreA[†]</i>
1-[36]	10E	10e100	<i>l(1)10Ea</i>	1-[41]	11E	11e200	<i>l(1)11Ec</i>	1-[48]	12E1-12E9		<i>mud</i>
1-36.0			<i>l(1)M41</i>	1-[41]	11E	11e200	<i>l(1)11Ed</i>	1-48.0			<i>thb</i>
1-36.0			<i>mit(1)13</i>	1-[41]	11E	11e200	<i>l(1)11Ee</i>	1-48.1			<i>rm</i>
1-36.2			<i>And</i>	1-[41]	11E	11e200	<i>l(1)11Ef</i>	1-48.1			<i>twg</i>
1-36.2	10E2	10e200	<i>dy</i>	1-41.0	12A3		<i>up</i>	1-48.4			<i>l(1)TW4</i>
1-36.3			<i>shl</i>	1-41.1			<i>pun</i>	1-48.4			<i>sge</i>
1-36.3			<i>trb</i>	1-41.1			<i>taw</i>	1-48.5			<i>cel</i>
1-36.4			<i>gr</i>	1-41.7	11B12-12C7		<i>cgd</i>	1-48.5			<i>l(1)Mb38</i>
1-36.6	11A2-11A3		<i>cab</i>	1-41.8	11D9-11D10	11e100	<i>sno</i>	1-48.6			<i>sla</i>
1-36.6	11A2		<i>nbA</i>	1-41.9			<i>l(1)dn27</i>	1-48.7			<i>mgt</i>
1-36.7	11A2	11a200	<i>l(1)11Aa</i>	1-42			<i>kk</i>	1-48.9			<i>thm</i>
1-36.8	11A2	11a500	<i>fw</i>	1-42			<i>l(1)ds3</i>	1-49			<i>fs(1)A457</i>
1-36.8	11A1-11A2	11a300	<i>gd</i>	1-[42]	11F1-11F4		<i>M(1)11F</i>	1-49			<i>l(1)ts4975</i>
1-37	10F7-11D1		<i>Flu</i>	1-[42]	11F2-12A2	11f100	<i>l(1)11Fa</i>	1-[49]	12F6-13B6		<i>M(1)13A</i>
1-37	10A2-11B2		<i>fs(1)M19</i>	1-[42]	11F2-12A2	11f100	<i>l(1)11Fb</i>	1-49.0			<i>mit(1)14</i>
1-37			<i>l(1)M9</i>	1-42.2			<i>frdF</i>	1-49.3			<i>vb</i>
1-37			<i>yea1</i>	1-42.3			<i>l(1)P2</i>	1-49.4			<i>acj6</i>
1-[37]	11A7-11A8		<i>ctv2</i>	1-42.5			<i>swy</i>	1-49.5			<i>Su(SD)X</i>
1-[37]	10F1		<i>l(1)DF978[†]</i>	1-42.6	11F1-12A2		<i>Gpt</i>	1-49.6	12E1-13A5		<i>sesF</i>
1-[37]	10E3-11A2		<i>l(1)ESHS45</i>	1-42.7	12C1-12C2		<i>rdgB</i>	1-49.7			<i>thv</i>
1-[37]	11A2-11A7		<i>l(1)ESHS46</i>	1-43			<i>l(1)EN11</i>	1-50			<i>l(1)EN10a</i>
				1-43			<i>l(1)ts1251</i>	1-50			<i>op</i>

THE GENOME OF *DROSOPHILA MELANOGASTER*

genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol
1-50			<i>Tul</i>	1-54.2			<i>vap</i>	1-58.5			<i>siw</i>
1-50			<i>wobb</i>	1-54.2	14C4-14C6	14c100	<i>mei41</i>	1-58.6			<i>ftdR</i>
1-[50]	13C1-13C3		<i>lpou</i> [†]	1-54.4			<i>Had</i>	1-58.6			<i>spIw</i>
1-[50]	13F		<i>Myb</i>	1-54.4			<i>rdt</i>	1-58.7			<i>l(1)ds12</i>
1-50.0	13A2-13A5		<i>eag</i>	1-54.4			<i>un</i>	1-58.7			<i>tms</i>
1-50.0	13E		<i>su(sc)</i> [†]	1-54.5			<i>acc</i>	1-58.7	17A1-17A2		<i>upd</i>
1-50.1			<i>hpa</i>	1-54.5	15A1	14f100	<i>r</i>	1-58.9			<i>sab</i>
1-50.1			<i>sld</i>	1-54.7	15A1-15A4	14f50	<i>sbl</i>	1-59			<i>l(1)tl</i>
1-50.2	12B9-13F		<i>hap</i>	1-55			<i>fs(1)A1578</i>	1-59			<i>l(1)XN82a</i> [†]
1-50.6			<i>Stp1</i>	1-55			<i>l(1)HM4</i>	1-59			<i>l(1)YQ09</i> [†]
1-50.6			<i>vam</i>	1-55			<i>l(1)ts5697</i>	1-59	16E-17B		<i>por</i>
1-50.8			<i>hypoG</i>	1-55			<i>l(1)tsUC19</i>	1-59		17c300	<i>Tu</i>
1-50.8			<i>sesG</i>	1-55	15A1	14f150	<i>if</i>	1-[59]	17C		<i>Pk?4</i>
1-51			<i>l(1)HM14</i>	1-[55]	15A9-16A2		<i>l(1)ESHS50</i>	1-[59]	17C1-17C2		<i>Rho</i> [†]
1-[51]	13E2		<i>Chc</i> [†]	1-[55]	15A9-16A2		<i>l(1)ESHS51</i>	1-[59]	17A1-17A4	17a100	<i>l(1)16Fa</i>
1-[51]	13F1-14B1		<i>exd</i>	1-[55]	15A5	15a200	<i>l(1)15Ab</i>	1-[59]	17A9-17A10	17a300	<i>l(1)16Fb</i>
1-[51]	13F		<i>Gapdh2</i>	1-[55]	15B	15b100	<i>l(1)15Ba</i>	1-[59]	17A11-17A12	17a400	<i>l(1)16Fc</i>
1-[51]	13F		<i>GB13F</i>	1-55.1			<i>fdg</i>	1-[59]	17A12-17B1	17a500	<i>l(1)16Fd</i>
1-[51]	13F10-14B3		<i>l(1)ESHS47</i>	1-55.1			<i>Shu</i>	1-[59]			<i>l(1)16Fe</i>
1-[51]	13C1-13C2		<i>Pp1-13C</i>	1-55.2	13F10-16A2		<i>Ocd</i>	1-59.1			<i>cvl-6</i>
1-[51]	13D-13E7		<i>sog</i>	1-55.2			<i>thvd</i>	1-59.2	17C		<i>e(y)5</i>
1-51.0			<i>ftdH</i>	1-55.5			<i>mit(1)18</i> [†]	1-59.2	17A5-17A6	17a200	<i>os</i>
1-51.0			<i>ftdJ</i>	1-55.5			<i>St</i>	1-59.4	17C2-17C3		<i>hdp-a</i>
1-51.2			<i>slw</i>	1-55.5	15A1		<i>Su(b)</i>	1-59.4	17C2-17C3		<i>hdp-b</i>
1-51.4	13F10-14B1		<i>slrp</i>	1-55.6	15A4-15F3		<i>dsp</i>	1-59.4	17C2-17C3	17c100	<i>Bx</i>
1-51.5			<i>exi</i>	1-55.6	11A7-13F10		<i>l(1)air15</i> [†]	1-59.5	17C4-17C6	17c200	<i>fu</i>
1-51.5			<i>l(1)ts120</i>	1-55.9			<i>l(1)Mc24</i>	1-59.8			<i>bk</i>
1-51.5			<i>PL(1)sp-S2</i>	1-56			<i>ccb</i>	1-59.8			<i>rdm</i>
1-51.5	13F		<i>sd</i>	1-56			<i>cs</i>	1-59.9			<i>bkl</i>
1-51.5	14A1		<i>shi</i>	1-56			<i>de</i>	1-60			<i>abw</i>
1-51.6	13C		<i>Bg</i>	1-56			<i>l(1)dn6</i>	1-60			<i>l(1)dn8</i>
1-51.6	13F		<i>tc</i>	1-56			<i>l(1)M11</i>	1-60			<i>l(1)Mb8</i>
1-51.7			<i>sesH</i>	1-56			<i>l(1)tr</i>	1-60			<i>l(1)XN75a</i> [†]
1-51.9			<i>smt</i>	1-56			<i>l(1)YD23b</i> [†]	1-[60]	17E3-17E9		<i>Gap43</i> [†]
1-52			<i>fs(1)M108</i>	1-56+			<i>l(1)Mc52</i>	1-[60]	17A-18A		<i>phm</i>
1-52			<i>l(1)EN4</i>	1-[56]	15E-15F		<i>Pen15EF</i> [†]	1-60.1			<i>crk</i>
1-52			<i>l(1)M24</i>	1-56.0	15D1		<i>Axs</i>	1-60.1			<i>obl</i>
1-52			<i>l(1)P1</i>	1-56.2			<i>mei218</i>	1-60.1			<i>ton</i>
1-52			<i>rol</i>	1-56.3			<i>snl</i>	1-60.3			<i>sts</i>
1-[52]	13E		<i>KZ30</i> [†]	1-56.4			<i>l(1)Mb46</i>	1-60.7			<i>thl</i>
1-52.0			<i>mch</i>	1-56.6	15D		<i>M(1)15D</i>	1-60.8			<i>pph</i>
1-52.1	14C7-14C8	14c200	<i>para</i>	1-56.7	15A5-15F3		<i>baz</i>	1-60.8	18A4-18B8		<i>sby</i>
1-52.3			<i>drw</i>	1-56.7	15F1-15F2		<i>f</i>	1-61			<i>l(1)HM18</i>
1-52.3	14C1-14C2	14c75	<i>unr</i>	1-56.7			<i>ftdQ</i>	1-61			<i>l(1)XD03</i> [†]
1-52.3			<i>nonA</i>	1-56.7	16B1-16F8		<i>mit</i>	1-[61]	18A		<i>prd10</i> [†]
1-52.4			<i>ber</i>	1-56.7			<i>Mu-β+3Nβ-</i>	1-61.0			<i>l(1)ts1082</i>
1-52.5			<i>us</i>	1-56.8			<i>fil</i>	1-61.1	18A4-18B8		<i>smd</i>
1-52.6			<i>msc</i>	1-56.9			<i>Sl</i>	1-61.5	18A4-18B8		<i>coc</i>
1-52.9			<i>brb</i>	1-57			<i>adel</i>	1-61.9			<i>l(1)dn28</i>
1-52.9			<i>l(1)nc3</i>	1-57			<i>cripC</i>	1-61.9			<i>meg</i>
1-52.9			<i>l(1)ts1891</i>	1-57			<i>l(1)dh2</i>	1-62			<i>ftdL</i>
1-53			<i>fi</i>	1-57			<i>l(1)dn7</i>	1-62			<i>fs(1)A383</i>
1-53			<i>fs(1)M29</i>	1-57			<i>l(1)ds9</i>	1-62			<i>fs(1)A1140</i>
1-53			<i>l(1)ts3803</i>	1-57			<i>l(1)HM2</i>	1-62			<i>fs(1)de3</i>
1-53			<i>yea7</i>	1-57			<i>l(1)HM20</i>	1-62			<i>fs(1)de7</i>
1-[53]	14B13-15A4		<i>l(1)ESHS48</i>	1-57			<i>l(1)M8</i>	1-62			<i>fs(1)de9</i>
1-[53]	14B13-15A4		<i>l(1)ESHS49</i>	1-57			<i>l(1)M16</i>	1-62			<i>fs(1)de15</i>
1-[53]	14B5	14b500	<i>bss</i>	1-57			<i>l(1)M42</i>	1-62			<i>fs(1)ne5</i>
1-[53]	14C1-14C2	14c50	<i>l(1)14Ca</i>	1-57			<i>l(1)XN52</i> [†]	1-62			<i>fs(1)ne9</i>
1-[53]	14C1-14C2	14c60	<i>l(1)14Cb</i>	1-57			<i>l(1)YA13</i> [†]	1-62			<i>l(1)Mc35</i>
1-53.0	14D1-15A1	15a100	<i>l(1)15Aa</i>	1-57			<i>l(1)Yi29</i> [†]	1-62.0			<i>srb</i>
1-53.1	14B3-14B4	14b100	<i>disco</i>	1-57			<i>lh</i>	1-62.2	18C-18D		<i>e(y)3</i>
1-53.2			<i>su(dp)</i>	1-[57]	16A4-16A5		<i>At</i>	1-62.5	18D1-18D2		<i>car</i>
1-53.5			<i>pyp</i>	1-[57]	16-17		<i>Cg16-17</i>	1-62.7	18B9-18D		<i>M(1)18C</i>
1-53.5			<i>sl</i>	1-[57]	16A1-16C3		<i>l(1)air16</i> [†]	1-62.8			<i>l(1)ts613</i>
1-53.5			<i>sln</i>	1-[57]	17A-18A2		<i>mbd</i>	1-62.9			<i>fs(1)1867</i>
1-53.5	14B5-14B18	14b200	<i>eas</i>	1-[57]	16C1-16C4		<i>Spec-b</i>	1-62.9			<i>RD</i>
1-53.6			<i>shd</i> [†]	1-57.0	16A		<i>pdf</i>	1-62.9	18E		<i>Zw</i>
1-53.9	14C7-14F1	14c300	<i>l(1)14DEa</i>	1-57.0	16A1-16A2	16a100	<i>B</i>	1-63			<i>fo</i>
1-53.9	14C7-14F1	14c300	<i>l(1)14DEb</i>	1-57.2			<i>der</i>	1-63			<i>l(1)ds5</i>
1-53.9	14C7-14F1	14c300	<i>l(1)14DEc</i>	1-57.4	16C2-18B11		<i>exo</i>	1-63			<i>l(1)EN6</i>
1-54			<i>l(1)ds4</i>	1-57.6	16F1-16F3	16f100	<i>Sh</i>	1-63			<i>pub</i>
1-54			<i>l(1)ts480</i>	1-57.7	16C6-16E1	16c100	<i>ff</i>	1-[63]	18F-19E1		<i>ms(1)4</i>
1-[54]	14B5-14C3		<i>bas</i>	1-57.8	16C2-18B11		<i>fcl</i>	1-[63]	18F	18f100	<i>l(1)18Fa</i>
1-[54]	14B13-14D1		<i>M(1)14C</i>	1-57.9	16B		<i>e(y)1</i>	1-63.1			<i>unp</i>
1-[54]	14B		<i>tta</i>	1-58			<i>bu</i>	1-63.4			<i>l(1)X27</i>
1-[54]	14B		<i>U2snRNA14B</i> [†]	1-58			<i>fs(1)M36</i>	1-63.8			<i>l(1)carot14</i>
1-[54]	14B		<i>U5snRNA14B</i> [†]	1-58			<i>gnd</i>	1-63.9			<i>kno</i>
1-[54]	14A1	14a100	<i>l(1)14Aa</i>	1-58			<i>ms(1)401</i>	1-64	19+		<i>grt</i>
1-[54]	14A1	14a200	<i>l(1)14Ab</i>	1-58			<i>rab</i>	1-[64]	19A4-19A7		<i>AnnX</i> [†]
1-[54]	14E	14e100	<i>l(1)14Ea</i>	1-58.1	16B		<i>e(y)4</i>	1-[64]	19A5-19E1		<i>Su(GI)27</i>
1-[54]	14F	14f200	<i>l(1)14Fa</i>	1-58.2			<i>cvw</i>	1-[64]	19E7-19E8		<i>vac</i>
1-54.0			<i>mc</i>	1-58.2	16E3-16F3		<i>mnb</i>	1-[64]	19A1	19a100	<i>amn</i>
1-54.2			<i>nrs</i>	1-58.3			<i>dwu</i>	1-[64]	19A	19a200	<i>l(1)19Aa</i>

CYTOGENETIC MAP

genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol
1-[64]	19A3-19A4	19a300	<i>ot</i>	2-0.0	21C1-21C2		<i>M(2)21C</i>	2-9.0	23E1-23F6		<i>msl2</i>
1-[64]	19A	19a400	<i>l(1)19Ac</i>	2-0.0	21B8-21C6		<i>mbm</i>	2-10	24A1-24A2		<i>Alp</i>
1-[64]	19A	19a400	<i>l(1)19Ad</i>	2-0.0			<i>ocr</i>	2-10			<i>Dt</i>
1-[64]	19B1	19b100	<i>l(1)19Ba</i>	2-0.0			<i>tg</i>	2-10	24A3-24C3		<i>for</i>
1-[64]	19C1	19c100	<i>l(1)19Ca</i>	2-0.0	21A1-21C1	21a100	<i>l(2)gl</i>	2-[10]	24B-24C		<i>Shaw</i>
1-[64]	19C4	19c300	<i>l(1)19Cb</i>	2-0.0	21A1-21B5	21b100	<i>net</i>	2-10.4			<i>l(2)jgd2</i>
1-[64]	19C6	19c300	<i>l(1)19Cc</i>	2-[0.0]	21B2-21B5	21b200	<i>l(2)21Ba</i>	2-10.5			<i>ang</i>
1-[64]	19D	19d100	<i>l(1)19Da</i>	2-[0.0]	21B2-21B5	21b200	<i>l(2)21Bb</i>	2-[11]	24D5-24D8	24d200	<i>l(2)24Dc</i>
1-[64]	19D2-19D3	19d300	<i>mell</i>	2-[0.0]	21B3-21B6	21b500	<i>l(2)Gsl</i>	2-[11]	24D5-24D8	24d200	<i>l(2)24Dd</i>
1-[64]	19E3	19e200	<i>shakB</i>	2-0.1	21C3		<i>ex</i>	2-[11]	24D5-24D8	24d200	<i>l(2)24De</i>
1-[64]	19E7	19e600	<i>vao</i>	2-0.3	21D1-21D2	21d100	<i>ds</i>	2-[11]			<i>l(2)24Df</i>
1-64.0	19B3	19b200	<i>sw</i>	2-[0.0]	21B6-21B8		<i>kis</i>	2-11.0	24D2-24D4	24d100	<i>ed</i>
1-64.1	19C2-19C3	19c500	<i>mel</i>	2-0.1	21C		<i>ush</i>	2-11.7			<i>scal</i>
1-64.4			<i>wa</i>	2-[0.1]	21C		<i>dsx-c21C[†]</i>	2-12			<i>aop</i>
1-64.7			<i>mdg</i>	2-[0.1]	21C		<i>RpIII35[†]</i>	2-12			<i>mat(2)cell-D</i>
1-64.8	18B4-19A1		<i>btdl</i>	2-[0.2]	21D		<i>Pkg1</i>	2-[12]	24D7-24D8	24d200	<i>M(2)24D</i>
1-64.8	19D3-19E1	19d200	<i>mal</i>	2-[0.3]	21D1-21D2	21d200	<i>l(2)21Da</i>	2-[12]	24E1-24F2	24e100	<i>l(2)24Ea</i>
1-65	18E1-20A1		<i>fs(1)A273</i>	2-[0.3]	21D1-21D2	21d300	<i>l(2)21Db</i>	2-12.0	24D5-24D7	24d200	<i>ft</i>
1-65			<i>M(Gpdh)</i>	2-[0.3]	21D1-21D2	21d300	<i>l(2)21Dc</i>	2-12.5			<i>bo</i>
1-65	19E1-19E2	19e100	<i>run</i>	2-[0.3]	21D1-21E1	21d500	<i>l(2)21Dd</i>	2-12.9	24F4-24F7	24f300	<i>M(2)24F</i>
1-[65]	19E-19F	19e300	<i>Cg19EF</i>	2-[0.3]	21D1-21E1	21d500	<i>l(2)21De</i>	2-13	24E2-24E5	24e200	<i>dw-24E</i>
1-[65]	19E6-19E8		<i>l(1)ESH52</i>	2-[0.3]	21D1-21E1	21d500	<i>l(2)21Df</i>	2-[13]	25A		<i>anon-25A[†]</i>
1-[65]	19F1-20A1		<i>l(1)ESH53</i>	2-[0.3]	21D1-21E1	21d500	<i>l(2)21Dg</i>	2-[13]	24F1-24F2	124f100	<i>l(2)24Fa</i>
1-[65]	19F		<i>mit(1)20[†]</i>	2-0.4	21C1-21C2	21b600	<i>al</i>	2-[13]	24F4-24F7	24f300	<i>l(2)24Fc</i>
1-[65]	19E1-20B		<i>ms(1)1</i>	2-0.65	21B8-21C8		<i>l(2)ey[†]</i>	2-[13]	25A4-25A8	25a500	<i>l(2)25Ab</i>
1-[65]	19E8-20A1		<i>ms(1)3</i>	2-1			<i>okr</i>	2-[13]	25A4-25A8	25a600	<i>l(2)25Ac</i>
1-[65]	19E6-19F1		<i>ms(1)7</i>	2-[11]	21E1-21E2	21e100	<i>l(2)21Ea</i>	2-[13]	25B1-25B2	25b100	<i>l(2)25Ba</i>
1-[65]	19E8-20A2		<i>ms(1)14</i>	2-[11]	21E2-22A1	21f100	<i>l(2)21Fa</i>	2-[13]	25B5-25B10	25b200	<i>l(2)25Bb</i>
1-[65]	19E1-19E5		<i>ms(1)19E</i>	2-[11]	21E2-22A1	21f100	<i>l(2)21Fb</i>	2-[13]	25B5-25B10	25b200	<i>l(2)25Bc</i>
1-[65]	19F		<i>ms(1)19Fa</i>	2-[11]	21E2-22A1	21f100	<i>l(2)21Fc</i>	2-[13]	25B5-25B10	25b200	<i>l(2)25Bd</i>
1-[65]	19F		<i>ms(1)19Fb</i>	2-[11]	21E2-22A1	21f100	<i>l(2)21Fd</i>	2-[13]	25B5-25B10	25b200	<i>l(2)25Be</i>
1-[65]	19F		<i>ms(1)19Fc</i>	2-1.0			<i>l(2)K534</i>	2-[13]	25C1-25C2	25c100	<i>l(2)25Ca</i>
1-[65]	19F		<i>ms(1)19Fd</i>	2-1.3	21E1-21E2	21e200	<i>S</i>	2-13.0	25A1-25A2	25a200	<i>dp</i>
1-[65]	19F		<i>seg</i>	2-1.4	21D4-21E2		<i>ninaA</i>	2-13.0			<i>Su(var)2-8</i>
1-[65]	19F		<i>tRNA:met3:19F</i>	2-[1.5]	21D	21d500	<i>U1snRNA21D</i>	2-13.5			<i>Acp-g1</i>
1-[65]	19F		<i>tRNA:tyr1:19F</i>	2-1.9			<i>l(2)K215</i>	2-13.5			<i>AcpJ</i>
1-[65]	19E6	19e300	<i>l(1)19Ec</i>	2-1.9			<i>l(2)Sp9a</i>	2-13.9	25B3-25B7		<i>Sgs1</i>
1-[65]	19E4-19E5	19e400	<i>l(1)19Ed</i>	2-1.9	21D3	21d500	<i>Lsp1-b</i>	2-14			<i>pw</i>
1-[65]	19E5-19E6	19e500	<i>lf</i>	2-2.3	22A3-22B1		<i>shr</i>	2-[14]	25C-28B		<i>inaB</i>
1-[65]	19F2	19f100	<i>l(1)19Fb</i>	2-3	21F2-22B1		<i>lea</i>	2-[14]	25A-27E		<i>Kf2</i>
1-[65]	19F1	19f100	<i>lft</i>	2-3			<i>ms(2)1R[†]</i>	2-[14]	25D		<i>tRNA:asp2:25D</i>
1-[65]	19F	19f200	<i>l(1)19Fc</i>	2-3			<i>ms(2)2R[†]</i>	2-15			<i>l(2)1076</i>
1-[65]	19F3-19F4	19f400	<i>fitO</i>	2-3	22D3-22E1		<i>Su(S)</i>	2-15			<i>l(2)cg</i>
1-[65]	19F4	19f500	<i>l(1)19Fe</i>	2-3-			<i>Su(var)326</i>	2-15	25A4-25A5	25a300	<i>slf</i>
1-[65]	19F4	19f500	<i>slgA</i>	2-[3]	22A		<i>Eno</i>	2-[15]	25A		<i>Bsg25A[†]</i>
1-[65]	19F4	19f500	<i>sol</i>	2-[3]	22A		<i>U3snRNA22A</i>	2-[15]	25A5-25A8		<i>Cf2</i>
1-[65]	19F5	19f800	<i>l(1)19Ff</i>	2-[3]	22A1-22A2	21f500	<i>l(2)21Fe</i>	2-[15]	25C		<i>Cg25C</i>
1-[65]	19F6	19f900	<i>l(1)19Fg</i>	2-[3]	22A1-22B1	22a100	<i>l(2)22Aa</i>	2-[15]	25B		<i>Jon25B</i>
1-65.6			<i>cf</i>	2-[3]	22A1-22B1	22a100	<i>l(2)22Ab</i>	2-[15]	25C1-25C3	25c200	<i>l(2)25Cc</i>
1-65.8			<i>l(1)ddl2</i>	2-[3]	22A1-22B1	22a100	<i>l(2)22Ac</i>	2-[15]	25C2-25C5	25c500	<i>l(2)25Cd</i>
1-65.8			<i>npr3</i>	2-[3]	22A1-22B1	22a100	<i>l(2)22Ad</i>	2-[15]	25C2-25C5	25c500	<i>l(2)25Ce</i>
1-65.9	19E8	19e700	<i>unc</i>	2-[3]	22A1-22B1	22a100	<i>l(2)22Ae</i>	2-[15]	25C3-25C9	25c600	<i>l(2)25Cf</i>
1-65.9	20F	20e200	<i>su(f)</i>	2-3.0	22A7-22B7		<i>Gor2</i>	2-[15]	25C8-25D7	25d300	<i>l(2)25Db</i>
1-66			<i>Rex</i>	2-3.2			<i>l(2)Sp7</i>	2-15.0	25C1-25C3	25c200	<i>M(2)25C</i>
1-66			<i>yea5</i>	2-4			<i>smo</i>	2-15.1			<i>Fs(2)Sz13</i>
1-[66]	20A-20B		<i>Cg20AB</i>	2-[4]	22C		<i>pok[†]</i>	2-16			<i>nmy</i>
1-[66]			<i>CR</i>	2-[4]	22B-22C		<i>tRNA:gly3:22BCa</i>	2-16	25D4-25D5	25d200	<i>tkv</i>
1-[66]	20B		<i>ms(1)16</i>	2-[4]	22B-22C		<i>tRNA:gly3:22BCb</i>	2-16	25F1-25F2	25ff100	<i>mid</i>
1-[66]	20A		<i>ms(1)20Aa</i>	2-[4]	22F1-22F2	22f200	<i>tRNA:tyr1:22Fa</i>	2-[16]	25D3		<i>Bsg25D</i>
1-[66]	20A		<i>ms(1)20Ab</i>	2-[4]	22F1-22F2	22f200	<i>tRNA:tyr1:22Fb</i>	2-16.0			<i>Sk</i>
1-[66]	20A1-20A2	20a100	<i>eo</i>	2-4.0			<i>Su(w^{ch})</i>	2-16.7			<i>l(2)K208</i>
1-[66]	20A1-20A2	20a100	<i>tuh1</i>	2-4.0	22F1-22F2	22f200	<i>dpp</i>	2-17			<i>cli</i>
1-[66]	20A3	20a200	<i>wap</i>	2-4.3			<i>vst</i>	2-17	25D7-25E1		<i>mat(2)ea-G</i>
1-[66]	20A	20a300	<i>intro</i>	2-5			<i>fs(2)B[†]</i>	2-17			<i>pi</i>
1-[66]	20A	20a300	<i>l(1)20Ad</i>	2-5			<i>gou[†]</i>	2-[17]	25F		<i>Lam</i>
1-[66]	20A5	20a500	<i>uncl</i>	2-5.5			<i>l(2)K203</i>	2-[17]	25F		<i>Mst25F</i>
1-[66]	20A5-20B	20b100	<i>fog</i>	2-5.9	22D-23C		<i>Pgk</i>	2-[17]	25F3-26A8	25ff200	<i>E(var)26A</i>
1-[66]	20A-20B	20b200	<i>l(1)20Bb</i>	2-6	22D3-22E1		<i>E(S)</i>	2-17.7	26B1-26B2		<i>ade2</i>
1-[66]	20B3	20b300	<i>stn</i>	2-6			<i>Pl</i>	2-17.8	26A5-26A7	25f500	<i>Gpdh</i>
1-[66]	20B	20b400	<i>l(1)20Ca</i>	2-[6]	23C-23D		<i>Tubg[†]</i>	2-18	26D1-26D2		<i>ee</i>
1-[66]	20C-20D	20c100	<i>l(1)20Cb</i>	2-[6]	23D-23E		<i>U5snRNA23DE</i>	2-18			<i>Fs(2)Sz12</i>
1-[66]	20E1	20e100	<i>sph</i>	2-6.1	23A2-23B2		<i>Cy</i>	2-[18]	26A		<i>chic</i>
1-66.0	20F	20f100	<i>bb</i>	2-[7]	23A1-23A3		<i>nrd</i>	2-[18]	25E1-25E2	25e100	<i>l(2)25Ea</i>
1-66.1			<i>fm</i>	2-[7]	23B		<i>Syt[†]</i>	2-[18]	25E1-25E6	25e200	<i>l(2)25Eb</i>
1-66.4			<i>l(1)ds13</i>	2-[7]	23E		<i>tRNA:ser7:23Ea</i>	2-[18]	25E1-25E6	25e300	<i>l(2)25Ec</i>
1-66.5			<i>ftl</i>	2-[7]	23E		<i>tRNA:ser7:23Eb</i>	2-[18]	25E1-25E6	25e300	<i>l(2)25Ed</i>
1-67			<i>l(1)YE17[†]</i>	2-7.6	23A3-23D4		<i>Su(var)2-4</i>	2-[18]	25E1-25E6	25e300	<i>l(2)25Ee</i>
1-67.9			<i>rfr</i>	2-7.7			<i>apg</i>	2-[18]	25E1-25E6	25e300	<i>l(2)25Ef</i>
1-68.9			<i>sme</i>	2-8	24A3-24D4		<i>capu</i>	2-[18]	25F2-25F3	25f100	<i>l(2)25Fa</i>
1-70			<i>Apx</i>	2-8	24A1-24A3		<i>odd</i>	2-[18]	25F2-25F3	25f100	<i>l(2)25Fb</i>
1-70			<i>bord</i>	2-8	24C4-24D3		<i>slp</i>	2-[18]	25F2-26A1	25f300	<i>l(2)25Fc</i>
1-73.1			<i>E(B)</i>	2-8.3			<i>l(2)ay</i>	2-[18]	25F2-26A1	25f300	<i>l(2)25Fd</i>
2-[0]	21B		<i>thg</i>	2-8.4			<i>sq</i>	2-18.5	25F4-26A1	25f500	<i>l(2)25Fe</i>
2-0.0			<i>bhe</i>	2-[9]	24A		<i>Pkg2</i>	2-18.8	25E1-25E2	25e300	<i>cl</i>

THE GENOME OF *DROSOPHILA MELANOGASTER*

genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol
2-19			<i>raw</i>	2-32.9			<i>Su(var)213</i>	2-[46]	33F		<i>trwn</i> [†]
2-[19]	26E		<i>eya</i>	2-32.9			<i>Su(var)215</i>	2-46.2			<i>l(2)K224</i>
2-20			<i>bot</i>	2-33	31B-31E		<i>bsk</i>	2-47			<i>l(2)pup</i>
2-20			<i>kmB</i>	2-33.7			<i>l(2)K202</i>	2-47			<i>mus211</i>
2-[20]	26B		<i>AcrA</i> [†]	2-[34]	30C1-30C6		<i>Pka-C1</i>	2-[47]	34A-34B		<i>U5snRNA34AB</i> [†]
2-[20]	26B1-26B3		<i>H2.0</i>	2-34.7	30F		<i>bib</i>	2-[47]	34C		<i>Vm34Ca</i>
2-[20]	26A-26B		<i>Kr-h</i> [†]	2-34.8			<i>Su(var)210</i>	2-47.0	33F1-33F5	33f100	<i>nub</i>
2-[20]	26A		<i>Mst26Aa</i>	2-35	30A-30C		<i>mat(2)cell-F</i>	2-[47.1]	34A-34B		<i>U2snRNA34ABa</i>
2-[20]	26A		<i>Mst26Ab</i>	2-35			<i>mbmC</i>	2-[47.1]	34A-34B		<i>U2snRNA34ABb</i>
2-[20]	26A2-26A5	26a100	<i>l(2)26Aa</i>	2-[35]	30B		<i>Fbp2</i>	2-47.5			<i>fs(2)E3</i>
2-[20]	26A7-26A9	26a200	<i>Gal</i>	2-[35]	30A		<i>M(2)30A</i>	2-47.5			<i>PL(2)sp-G2</i>
2-[20]	26A7-26A9	26a200	<i>l(2)26Ac</i>	2-[35]	30B		<i>numb</i>	2-47.9			<i>l(2)K201</i>
2-[20]	26A7-26A9	26a200	<i>l(2)26Ad</i>	2-35.0			<i>l(2)Sp1</i>	2-47.9			<i>ms(2)E4</i>
2-[20]	26A7-26A9	26a200	<i>l(2)26Ae</i>	2-35.3			<i>tipC</i>	2-48	33B3-33F2		<i>aret</i>
2-[20]	26A	26aa100	<i>Fcp26Aa</i> [†]	2-35.4			<i>Su(var)209</i>	2-48			<i>Rm2</i>
2-[20]	26A	26aa100	<i>Fcp26Ac</i> [†]	2-35.6			<i>fs(2)E9</i>	2-[48]	34D1-34D2	34d100	<i>l(2)34Da</i>
2-[20]	26A	26aa100	<i>Vm26Aa</i>	2-35.7	29F-30F		<i>pal</i>	2-[48]	34D3	34d200	<i>l(2)34Db</i>
2-[20]	26A	26aa100	<i>Vm26Ab</i>	2-36			<i>Si</i>	2-[48]	34D5-34D6	34d300	<i>Sos</i> [†]
2-[20]	26B9-27A1	26c100	<i>l(2)26CF</i>	2-36			<i>ter</i>	2-[48]	34D4-34D5	34d500	<i>l(2)34Dc</i>
2-20.6	26A7-26A9	26a200	<i>l(2)26Ab</i>	2-36	31A-31C		<i>trk</i>	2-[48]	34D4-34D5	34d600	<i>l(2)34Dd</i>
2-[21]	27D		<i>Gla</i>	2-36.8			<i>rc</i>	2-[48]	34D6-34D8	34d600	<i>l(2)34Df</i>
2-[21]	27D1-27D2		<i>snRNP27D</i> [†]	2-37			<i>chal</i>	2-[48]	34D6-34D8	34d800	<i>l(2)34De</i>
2-21.9	28A1-28A2	28a100	<i>spd</i>	2-[37]	31B		<i>me31B</i> [†]	2-[48]	34D6-34D8	34d800	<i>l(2)34Dg</i>
2-[22]	28A		<i>kdu</i>	2-[37]	31C-31F		<i>Suc</i>	2-[48]	34E3-34E5	34e100	<i>l(2)34Eb</i>
2-[22]	28A1-28A3		<i>ninaC</i>	2-37.0			<i>Su(var)2-12</i>	2-48.2	34E3-34E5	34e300	<i>rk</i>
2-[22]	27C2-27D2	27c100	<i>l(2)27Ca</i> [†]	2-37.2	31B-31E		<i>Mdh1</i>	2-48.5			<i>fs(2)E4</i>
2-[22]	27C2-27D2	27c100	<i>l(2)27Cb</i> [†]	2-37.5			<i>l(2)Sp10</i>	2-48.5	34D4-34D5	34d400	<i>b</i>
2-[22]	27C2-27D2	27c100	<i>l(2)27Cc</i> [†]	2-37.7			<i>l(2)K326</i>	2-48.7	34E3-34E5	34e200	<i>j</i>
2-[22]	27D1-27D2	27d100	<i>ade3</i>	2-38			<i>drg</i>	2-49			<i>cort</i>
2-[22]	27D1-27E2	27d100	<i>l(2)27Db</i> [†]	2-38			<i>Sq</i>	2-49			<i>l(2)IIS9</i> [†]
2-[22]	27D1-27E2	27d100	<i>l(2)27Dc</i> [†]	2-38.2			<i>Su(var)2-7</i>	2-49			<i>l(2)Sp2b</i>
2-[22]	27D1-27D2	27d100	<i>Pcp</i>	2-38.2	31A2-31C1		<i>Su(var)2-13</i>	2-49			<i>mat(2)syn-A</i>
2-[22]	27E1-28A2	27e100	<i>l(2)27Ea</i> [†]	2-39			<i>aub</i>	2-[49]	34F1-34F2	34f100	<i>l(2)34Fa</i>
2-[22]	28A1-28A3	28a100	<i>wg</i>	2-39			<i>fol</i>	2-[49]	34F4-35A1	34f300	<i>l(2)34Fc</i>
2-[22]	28A1-28B4	28a300	<i>l(2)28Aa</i> [†]	2-39	32A-32E		<i>wdl</i>	2-[49]	34F4-35A1	34f400	<i>l(2)34Fd</i>
2-[22]	28A1-28B4	28a300	<i>l(2)28Ab</i> [†]	2-[39]	31B-31D		<i>den</i>	2-[49]	34F4-35A1	34f400	<i>ms(2)34Fe</i>
2-[22]	28A1-28B4	28a300	<i>l(2)28Ac</i> [†]	2-40	31D-31F3		<i>err</i>	2-49.9	34F3	34f200	<i>wb</i>
2-[22]	28B3-28B4	28b100	<i>l(2)28Ba</i> [†]	2-40	31D-31F3		<i>mat(2)ea-E</i>	2-50			<i>l(2)H</i>
2-[22]	28B3-28B4	28b100	<i>l(2)28Bb</i> [†]	2-40			<i>mat(2)syn-B</i>	2-50			<i>lm</i>
2-[22]	28B3-28B4	28b100	<i>l(2)28Bc</i> [†]	2-40	31D-31F		<i>mat(2)syn-E</i>	2-[50]	35A3-35A4	35a100	<i>l(2)35Aa</i>
2-22.0			<i>fs(2)E2</i>	2-40			<i>pg</i>	2-[50]	35A4	35a200	<i>pu</i>
2-22.0	28A1-28B4	28a300	<i>Sp</i>	2-40			<i>reff(2)M</i>	2-[50]	35B1	35a400	<i>l(2)35Ba</i>
2-22.9			<i>lys</i>	2-40			<i>tkd</i>	2-[50]	35B1-35B2	35a500	<i>noc</i>
2-23			<i>cup</i>	2-40			<i>wd</i>	2-[50]	35B1-35B2	35a600	<i>tRNA:gly3:35Ba</i>
2-23.4			<i>cui</i>	2-[40]	31E		<i>cdc2</i> [†]	2-[50]	35B1-35B2	35a620	<i>tRNA:gly3:35Bb</i>
2-23.8			<i>mus201</i>	2-[40]	32D1-32D4		<i>l(2)gd</i>	2-[50]	35B1-35B2	35a650	<i>tRNA:gly3:35Bc</i>
2-24			<i>cr-b</i>	2-40.1	31C-31D		<i>Su(var)201</i>	2-[50]	35B1-35B2	35a660	<i>tRNA:gly3:35Bd</i>
2-24			<i>mat(2)syn-H</i>	2-40.5	31A2-31C		<i>Su(var)2-1</i>	2-[50]	35B1-35B2	35a670	<i>tRNA:gly3:35Be</i>
2-[24]	28D		<i>Lam-B1</i>	2-40.68			<i>bsp</i>	2-[50]	35B4	35b300	<i>l(2)35Bb</i>
2-[24]	28D2-28D4		<i>Pp2A</i> [†]	2-41			<i>l(2)54</i>	2-[50]	35B4-35B8	35b400	<i>l(2)35Bc</i>
2-[24]	29A		<i>Src2</i>	2-41			<i>mat(2)ea-F</i>	2-[50]	35B5-35B8	35b400	<i>l(2)35Be</i>
2-[24]	28C		<i>tRNA:tyr1:28C</i>	2-41			<i>mat(2)syn-I</i>	2-[50]	35B5-35B8	35b400	<i>l(2)35Bf</i>
2-[24]	28C2-28C3		<i>Uro</i>	2-41.0			<i>J</i>	2-[50]	35B4-35B8	35b700	<i>l(2)35Bd</i>
2-24.0			<i>gt4</i>	2-41.1			<i>PL(2)sp-S3</i>	2-[50]	35B9-35B10	35b800	<i>l(2)35Bg</i>
2-24.1			<i>fy</i>	2-41.3	31E		<i>da</i>	2-50.0			<i>l(2)Sp6b</i>
2-27			<i>l(2)H032</i> [†]	2-41.4	31B-32A		<i>mfs(2)31</i>	2-50.0	35A4-35B1	35a300	<i>el</i>
2-[27]	28D		<i>tRNA:gly3:28D</i>	2-42			<i>slip</i>	2-50.1	35B1-35B2	35b100	<i>osp</i>
2-27.5			<i>corr</i>	2-42.8			<i>AcpB</i>	2-50.1	35B3	35b200	<i>Adh</i>
2-27.5			<i>l(2)K225</i>	2-43			<i>bic</i>	2-50.1	35B3	35b250	<i>Adhr</i> [†]
2-27.5			<i>PL(2)sp-G1</i>	2-43			<i>Su(Cy)</i>	2-50.4			<i>fs(2)E5</i>
2-28			<i>ms(2)E3</i>	2-43.7			<i>Cpt</i>	2-50.5	35B9-35B10	35b800	<i>Su(H)</i>
2-28.3			<i>nr</i>	2-43.8			<i>l(2)bl</i>	2-50.7			<i>mfs(2)350</i>
2-29.5			<i>tu(2)48</i>	2-44			<i>an</i>	2-51			<i>fs(2)lto1</i>
2-29.7			<i>Su(var)2-3</i>	2-44	31F-32E		<i>dal</i>	2-51			<i>fs(2)PN48</i> [†]
2-30			<i>fs(2)eo1</i>	2-44	31F-32E		<i>hup</i>	2-51			<i>ms(2)3R</i>
2-30	29C		<i>grk</i>	2-44			<i>inaA</i>	2-51	35C1-35C2	35c300	<i>vas</i>
2-30			<i>meiG87</i>	2-44	33A1-33A2	33a100	<i>sal</i>	2-51	35D1-35D2	35d200	<i>sna</i>
2-[30]	29D		<i>Gl1</i>	2-[44]	32E		<i>Vm32Ea</i>	2-51	35E1-35E2	35e100	<i>BicC</i>
2-[30]	29D		<i>tRNA:asp2:29D</i>	2-[44]	32E		<i>Vm32Ec</i>	2-51	35F1-36A1	36a100	<i>fzy</i>
2-[30]	29A1-29A2		<i>tRNA:lys5:29A</i>	2-44.0			<i>ab</i>	2-[51]	35E-35F		<i>U5snRNA35EF</i> [†]
2-30.3			<i>l(2)pm</i>	2-44.0	32E		<i>abo</i>	2-[51]	35C1-35C2	35c200	<i>l(2)35Ce</i>
2-31	30A-30C		<i>mat(2)cell-K</i>	2-44.0			<i>ms(2)2</i>	2-[51]	35C1-35C2	35c400	<i>l(2)35Cb</i>
2-31			<i>mbmB</i>	2-44.0			<i>tipA</i>	2-[51]	35C3	35c450	<i>l(2)35Cc</i>
2-[31]	31B		<i>flp</i>	2-44.0			<i>apb</i>	2-[51]	35C3	35c500	<i>rd</i>
2-[31]	31B-31F		<i>pim</i>	2-44.8			<i>Su(var)2-9</i>	2-[51]	35C4-35C5	35c700	<i>l(2)35Cd</i>
2-31.0			<i>d</i>	2-45			<i>Bxd</i>	2-[51]	35C4-35C5	35c800	<i>ftg</i> [†]
2-31.1	29A		<i>Su(var)2-5</i>	2-45			<i>kmA</i>	2-[51]	35C4-35C5	35c900	<i>dgl</i>
2-32			<i>eb</i>	2-45			<i>oph</i>	2-[51]	35D1-35D2	35d100	<i>l(2)35Da</i>
2-32			<i>rags</i>	2-45	33C1-33C2	33c100	<i>prd</i>	2-[51]	35D3-35D4	35d300	<i>lace</i>
2-32	30A-30C		<i>rem</i>	2-[45]	33B3-33F2		<i>zuc</i> [†]	2-[51]	35D5-35D7	35d400	<i>l(2)35Dd</i>
2-[32]	29E		<i>tRNA:asp2:29E</i>	2-[45]	33B1-33B2	33b100	<i>esc</i>	2-[51]	35D5-35D7	35d500	<i>l(2)35De</i>
2-32.0			<i>l(2)Sp14</i>	2-45.7			<i>Su(var)2-11</i>	2-[51]	35D5-35D7	35d600	<i>l(2)35Df</i>
2-32.0			<i>Su(var)207</i>	2-46			<i>tu(2)48j</i>	2-[51]	35D5-35D7	35d600	<i>l(2)35Dg</i>
2-32.1			<i>l(2)K204</i>	2-[46]	33F		<i>dim</i> [†]	2-[51]	35D5-35D7	35d600	<i>l(2)35Dh</i>

CYTOGENETIC MAP

genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol
2-[51]	35D5-35D7	35d600	<i>l(2)35Di</i>	2-53.9	37C1-37C4	37c200	<i>l(2)37Cc</i>	2-54.8			<i>ms(2)E7</i>
2-[51]	35E1-35E2	35d600	<i>l(2)35Ea</i>	2-53.9	37C1-37C4	37c400	<i>l(2)37Ca</i>	2-54.8	38F2-38E9	38e100	<i>Bl</i>
2-[51]	35E1-35E2	35e50	<i>ms(2)35Eb</i>	2-53.9	37C1-37C4	37c400	<i>l(2)37Cd</i>	2-54.8	40F(h35)	40f300	<i>cta</i>
2-[51]	35F1-36A1	35f100	<i>l(2)35Fa</i>	2-53.9	37C1-37C4	37c400	<i>l(2)37Ce</i>	2-[54.8]	38A6-39F1		<i>l(2)Co[†]</i>
2-[51]	35F1-36A1	36a200	<i>l(2)35Fb</i>	2-53.9	37C1-37C4	37c400	<i>l(2)37Cg</i>	2-[54.8]	40F		<i>Rev</i>
2-[51]	35F1-36A1	36a200	<i>l(2)35Fc</i>	2-54			<i>fs(2)jeo2</i>	2-[54.8]	40A		<i>tsh[†]</i>
2-[51]	35F1-36A1	36a200	<i>l(2)35Fe</i>	2-54	36E4-38A7		<i>halo[†]</i>	2-[54.8]	40A-40B		<i>U2snRNA40AB</i>
2-[51]	35F1-36A1	36a200	<i>l(2)35Ff</i>	2-54			<i>mat(2)cell-A</i>	2-[54.8]	40A-40B		<i>U4snRNA40AB[†]</i>
2-51.0	35C1-35C2	35c100	<i>ck</i>	2-54			<i>mat(2)cell-G</i>	2-[54.8]	40F(h35)	40f100	<i>l(2)40Fa</i>
2-51.0			<i>Sco</i>	2-54			<i>mat(2)ea-H</i>	2-[54.8]	40F(h35)	40f200	<i>l(2)40Fc</i>
2-51.3			<i>Su(var)206</i>	2-54			<i>MR</i>	2-[54.8]	40F(h35)	40f400	<i>lt</i>
2-51.7	35F1-36A1	36a200	<i>cact</i>	2-54	36E4-37C1		<i>pre</i>	2-[54.8]	40F(h35)	40f500	<i>l(2)40Fd</i>
2-52			<i>baton</i>	2-54	37E2-38A1		<i>spi</i>	2-[54.8]	40F(h35)	40f600	<i>l(2)40Ff</i>
2-52			<i>pys</i>	2-54	37E3-37F1		<i>spir</i>	2-[54.8]	40F(h35)	40f700	<i>l(2)40Fe</i>
2-[52]	36B		<i>Cyt-b</i>	2-54			<i>tup</i>	2-[54.8]	40F(h35)	40f800	<i>l(2)40Fg</i>
2-[52]			<i>Cyt-c1</i>	2-54	37D5-37D6	37d300	<i>Sd</i>	2-54.9			<i>Alu</i>
2-[52]			<i>Cyt-c2</i>	2-[54]	38E		<i>cad</i>	2-54.9			<i>Jag</i>
2-[52]	36C3-36D2		<i>Dlar[†]</i>	2-[54]	37F5-39F1		<i>del</i>	2-54.9			<i>mus205</i>
2-[52]	35F1-36A1	36a200	<i>cni[†]</i>	2-[54]	38A1-38A3		<i>E86[†]</i>	2-55	41		<i>E(da)</i>
2-[52]	35F1-36A1	36a900	<i>l(2)35Fd</i>	2-[54]	37C2-37D1		<i>fs(2)37C[†]</i>	2-55	40F(h35)		<i>E(SD)</i>
2-[52]	36A1-36A5	36a1000	<i>dac[†]</i>	2-[54]	38C		<i>Fs(2)S27</i>	2-55			<i>inb</i>
2-[52]	36A7-37B2	36a1100	<i>l(2)36Aa</i>	2-[54]	37D2-38A1		<i>fs(2)mel1[†]</i>	2-55			<i>l(2)I323</i>
2-[52]	36A7-37B2	36a1100	<i>l(2)36Ac</i>	2-[54]	37D2-38A1		<i>fs(2)mel2[†]</i>	2-55			<i>pads</i>
2-[52]	36A7-37B2	36a1100	<i>l(2)36Ad</i>	2-[54]	37D2-38A1		<i>fs(2)mel3[†]</i>	2-55			<i>pod</i>
2-[52]	36A10-36A11	36a1400	<i>CytC-1</i>	2-[54]	37D2-38A1		<i>fs(2)mel4[†]</i>	2-55			<i>tf</i>
2-[52]	36A10-36A11	36a1400	<i>CytC-2</i>	2-[54]	37D2-38A1		<i>fs(2)mel5[†]</i>	2-55			<i>tri</i>
2-[52]	36A7-37B2	36a1400	<i>l(2)36Ab</i>	2-[54]	37D2-38A1		<i>fs(2)mel6[†]</i>	2-55.0			<i>l(2)35i</i>
2-[52]	36A6-36B1	36a1400	<i>plo</i>	2-[54]	37D2-38A1		<i>fs(2)mel7[†]</i>	2-55.0			<i>wi</i>
2-[52]	36B3-36C4	36b100	<i>l(2)36Ba</i>	2-[54]	37F7-37F8		<i>msl1</i>	2-55.1			<i>Bow</i>
2-[52]	36B3-36C4	36b100	<i>l(2)36Bb</i>	2-[54]	37B-37D		<i>NaCP37B[†]</i>	2-55.1			<i>l(2)Sp9d</i>
2-[52]	36B3-36C4	36b100	<i>l(2)36Bc</i>	2-[54]	38B4-38B6		<i>neb</i>	2-55.1	41A(h39)	41a100	<i>l(2)41Ab</i>
2-[52]	36B3-36C4	36b400	<i>l(2)36Bd</i>	2-[54]	37D2-38A1		<i>ovi[†]</i>	2-55.1	41A(h40-h41)	41a200	<i>l(2)41Aa</i>
2-[52]	36C2-36D1	36c100	<i>l(2)36Ca</i>	2-[54]	37F5-38A7		<i>scw</i>	2-55.1	41A(h40-h41)	41a200	<i>rl</i>
2-52.2	36B1-36B2	36a1400	<i>Mhc</i>	2-[54]	37D2-38C1		<i>Su(Pc)37D</i>	2-55.1	41A(h43-h44)	41a400	<i>uex</i>
2-52.5			<i>cru</i>	2-[54]	38B6-38C2		<i>vls</i>	2-55.1	41A	41a500	<i>l(2)41Af</i>
2-52.8	35F1-36A1	36a200	<i>chif</i>	2-[54]	37C3-37C7	37c800	<i>brat[†]</i>	2-55.1	41A	41a500	<i>M(2)41A</i>
2-52.9			<i>her</i>	2-[54]	37C3-37C7	37d100	<i>fs(2)TW1</i>	2-55.1	41A(h46)	41a800	<i>l(2)41Ae</i>
2-52.9	36C2-36C9	36c200	<i>dl</i>	2-[54]	37D2-37D7	37d200	<i>l(2)37Da</i>	2-55.1	41A(h46)	41a800	<i>l(2)41Ah</i>
2-52.9	36C2-36C3	36c300	<i>BicD</i>	2-[54]	37D2-37E1	37d300	<i>l(2)37Db</i>	2-55.1	41B-41C	41a900	<i>stw</i>
2-53			<i>fs(2)SC46</i>	2-[54]	37D2-37D6	37d350	<i>Top2</i>	2-[55.1]	41A-41B		<i>tRNA:tyr1:41AB</i>
2-53	36C2-36C4	36d100	<i>qua</i>	2-[54]	37D2-37E1	37d400	<i>l(2)37Dd</i>	2-[55.1]	41A(h39)	41a50	<i>Rsp-Sd</i>
2-53	36E1-36E3	36e200	<i>rdo</i>	2-[54]	37D2-37E1	37d500	<i>l(2)37Dc</i>	2-55.2	41A		<i>DipA</i>
2-[53]	36D1-36D2	36e300	<i>Arr1</i>	2-[54]	37D2-37E1	37d500	<i>l(2)37De</i>	2-55.2			<i>fs(2)E7</i>
2-[53]	36E4-38A7		<i>fs(2)jeo3</i>	2-[54]	37D2-37E1	37d500	<i>l(2)37Df</i>	2-55.2	42A2-42A8		<i>mle</i>
2-[53]	36E4-38A7		<i>fs(2)lto2</i>	2-[54]	37D2-37E1	37d900	<i>l(2)37Dg</i>	2-55.2	41A-42A3		<i>msf</i>
2-[53]	36E4-38A7		<i>fs(2)lto3</i>	2-[54]	37D2-37F4	37d1000	<i>l(2)37Dh</i>	2-55.2	41B-41C	41a1000	<i>ap</i>
2-[53]	36E4-38A7		<i>fs(2)lo-F[†]</i>	2-[54]	37D2-37E1	37d1000	<i>l(2)37Di</i>	2-[55.2]	42A		<i>Act42A</i>
2-[53]	36E4-38A7		<i>fs(2)lo-G[†]</i>	2-[54]	37D2-37E1	37d1000	<i>l(2)37Dj</i>	2-[55.2]	42A		<i>Ecr[†]</i>
2-[53]	36F7-37B8		<i>Roi</i>	2-[54]	37D2-37F4	37e100	<i>l(2)37Ea</i>	2-[55.2]	42A		<i>Edg42A</i>
2-[53]	36E1	36e100	<i>Fas3</i>	2-[54]	37E2-38A1	37f100	<i>l(2)37Fa</i>	2-[55.2]	41C1-41C2		<i>sxc</i>
2-[53]	36E1-36E3	36e300	<i>kel</i>	2-[54]	37E2-38A1	37f100	<i>l(2)37Fb</i>	2-[55.2]	42A		<i>tRNA:asn5:42Aa</i>
2-[53]	36E1-36E3	36e400	<i>ninaD</i>	2-[54]	37E2-38A1	37f300	<i>l(2)37Fc</i>	2-[55.2]	42A		<i>tRNA:tyr1:42A</i>
2-[53]	36F2-36F6	36e500	<i>M(2)36F</i>	2-[54]	37E2-38A1	37f400	<i>l(2)37Fd</i>	2-[55.2]	42A	42a(200)	<i>tRNA:asn5:42Ab</i>
2-53.0	36D1-36E4		<i>ApcC</i>	2-[54]	37E2-38A1	37f500	<i>l(2)37Fe</i>	2-[55.2]	42A	42a(300)	<i>tRNA:arg2:42Aa</i>
2-53.0			<i>l(2)K305</i>	2-[54]	37E2-38A1	37f500	<i>l(2)37Ff</i>	2-[55.2]	42A	42a(400)	<i>tRNA:lys2:42Aa</i>
2-53.1	36D1	36d200	<i>l(2)36Da</i>	2-[54]	37E2-38A1	37f700	<i>l(2)37Fg</i>	2-[55.2]	42A	42a(500)	<i>tRNA:arg2:42Ab</i>
2-53.6	37A3-37A6		<i>Tft</i>	2-[54]	38A6-38B2	38b200	<i>l(2)38Ba</i>	2-[55.2]	42A	42a(600)	<i>tRNA:ile:42A</i>
2-53.7			<i>awu</i>	2-[54]	38A6-38B2	38b200	<i>l(2)38Bb</i>	2-[55.2]	42A	42a(700)	<i>tRNA:lys2:42Ab</i>
2-53.7			<i>l(2)K483</i>	2-[54]	38C6-38C9	38c100	<i>ms(2)38C</i>	2-[55.2]	42A	42a(800)	<i>tRNA:lys2:42Ac</i>
2-53.9	36F3-36F5	36f100	<i>l(2)36Fb</i>	2-54.0	37E2-37F3	37e200	<i>ref(2)P</i>	2-[55.2]	42A	42a(900)	<i>tRNA:lys2:42Ad</i>
2-53.9	36F3-36F8	36f200	<i>l(2)36Fc</i>	2-54.1			<i>AcpK</i>	2-[55.2]	42A	42a(1000)	<i>tRNA:asn5:42Ac</i>
2-53.9	36F8	36f400	<i>l(2)36Fd</i>	2-[54.1]	39C2-39C4		<i>crc</i>	2-[55.2]	42A	42a(1100)	<i>tRNA:asn5:42Ad</i>
2-53.9	36F8	36f400	<i>l(2)36Fe</i>	2-[54.1]	39C-39D		<i>Tpl</i>	2-[55.2]	42A	42a(1200)	<i>tRNA:asn5:42Ae</i>
2-53.9	36F9-36F11	36f600	<i>l(2)36Ff</i>	2-[54.1]	39B		<i>U2snRNA39B</i>	2-[55.2]	42A	42a(1300)	<i>tRNA:arg2:42Ac</i>
2-53.9	36F9-36F11	36f600	<i>l(2)36Fg</i>	2-[54.1]	39B		<i>U4snRNA39B</i>	2-[55.2]	42A	42a(1400)	<i>tRNA:asn5:42Af</i>
2-53.9	36F9-36F11	36f600	<i>l(2)36Fh</i>	2-[54.1]	39B		<i>U5snRNA39B[†]</i>	2-[55.2]	42A	42a(1500)	<i>tRNA:asn5:42Ag</i>
2-53.9	36F9-36F11	36f600	<i>l(2)36Fi</i>	2-54.3			<i>bri</i>	2-[55.2]	42A	42a(1600)	<i>tRNA:asn5:42Ah</i>
2-53.9	36F8-37B7	37a100	<i>l(2)37Aa</i>	2-54.3	39E2-40A		<i>M(2)39F</i>	2-[55.2]	42A	42a(1700)	<i>tRNA:lys2:42Ae</i>
2-53.9	36F8-37B7	37a100	<i>l(2)37Ab</i>	2-54.4			<i>fs(2)E6</i>	2-[55.2]	42A	42a(1800)	<i>tRNA:arg2:42Ad</i>
2-53.9	36F8-37B7	37a100	<i>l(2)37Ac</i>	2-54.5	38A7-38B2		<i>tyr1</i>	2-55.3	42A2-42B1		<i>tk</i>
2-53.9	37B3-37B7	37b100	<i>l(2)37Bi</i>	2-54.5	38B1-38B2	38b100	<i>pr</i>	2-55.3	43A1-43A3	43a200	<i>pk</i>
2-53.9	37B3-37B7	37b100	<i>l(2)37Bj</i>	2-54.6			<i>Cu</i>	2-55.3	43A1-43A3	43a300	<i>sple</i>
2-53.9	37B10-37B14	37b300	<i>hk</i>	2-[54.6]	39E-39F		<i>Ej2b</i>	2-55.4	43B1-43B3	43b100	<i>pwn</i>
2-53.9	37C1-37C4	37b300	<i>l(2)37Cb</i>	2-[54.6]	39D3-39E2		<i>His1</i>	2-55.6			<i>ms(2)E8</i>
2-53.9	37B10-37B13	37b400	<i>l(2)37Ba</i>	2-[54.6]	39D3-39E2		<i>His2A</i>	2-55.7	42B1-42B3		<i>bur</i>
2-53.9	37B10-37B13	37b400	<i>l(2)37Bc</i>	2-[54.6]	39D3-39E2		<i>His2B</i>	2-55.8			<i>ae</i>
2-53.9	37B10-37B13	37b400	<i>l(2)37Be</i>	2-[54.6]	39D3-39E2		<i>His3</i>	2-55.9			<i>ti</i>
2-53.9	37B10-37B13	37b700	<i>Dox-A2</i>	2-[54.6]	39D3-39E2		<i>His4</i>	2-56			<i>ifm(2)11</i>
2-53.9	37B10-37B13	37b700	<i>l(2)37Bb</i>	2-54.7			<i>rh</i>	2-56			<i>l(2)hst</i>
2-53.9	37B10-37B13	37b900	<i>l(2)37Bh[†]</i>	2-54.7	38A7-39D1		<i>Su(crc)</i>	2-56			<i>mbs</i>
2-53.9	37B13-37C2	37b1000	<i>l(2)37Bg</i>	2-54.8			<i>l(2)85</i>	2-56			<i>Rw</i>
2-53.9	37B10-37C2	37b1100	<i>l(2)37Bd</i>	2-54.8			<i>ms(2)11R[†]</i>	2-[56]	42C2-42C7		<i>Adf1[†]</i>
2-53.9	37B13-37C1	37b1200	<i>amd</i>	2-54.8			<i>ms(2)E5</i>	2-[56]	42D-42E		<i>Cg42DE</i>
2-53.9	37C1-37C2	37c100	<i>Ddc</i>	2-54.8			<i>ms(2)E6</i>	2-[56]	42C1-43F8		<i>mat(2)twg[†]</i>

THE GENOME OF *DROSOPHILA MELANOGASTER*

genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol
2-[56]	43C		<i>med</i> [†]	2-[59]	46A1-46A2		<i>tRNA:met3:46A</i>	2-67.0	49D3-49E6	49d300	<i>vg</i>
2-[56]	43A1-43A3		<i>Raf2</i> [†]	2-59.4			<i>stmC</i>	2-67.1	49D	49d300	<i>l(2)49Dc</i>
2-[56]	42E		<i>tRNA:tyr1:42E</i>	2-59.9			<i>l(2)K335</i>	2-68			<i>fas</i>
2-[56]	42E	42e(100)	<i>tRNA:lys2:42Ea</i>	2-60			<i>l(2)IA109</i> [†]	2-68			<i>gho</i>
2-[56]	42E	42e(200)	<i>tRNA:lys2:42Eb</i>	2-60			<i>mat(2)cell-C</i>	2-68			<i>mat(2)cell-I</i>
2-[56]	42E	42e(300)	<i>tRNA:lys2:42Ec</i>	2-60			<i>mat(2)ea-B</i>	2-68			<i>Opt</i>
2-[56]	42E	42e(400)	<i>tRNA:lys2:42Ed</i>	2-60			<i>gat</i>	2-68	49E7-50A3		<i>sie</i>
2-[56]	43A1-43A3	43a100	<i>nec</i>	2-[60]	47A		<i>Gca47A</i>	2-68			<i>ts</i>
2-[56]	43B1-43B7	43b100	<i>l(2)43Ba</i>	2-[60]	47A		<i>Mst47A</i>	2-[68]	49F9-49F13		<i>Mp20</i>
2-56.1			<i>bd</i>	2-[60]	47D-47F		<i>Try</i>	2-[68]	49F2-49F13	49f100	<i>l(2)49Fa</i>
2-56.5			<i>std</i>	2-60.1			<i>at</i>	2-[68]	49F2-49F13	49f100	<i>l(2)49Fb</i>
2-56.6			<i>l(2)K300</i>	2-60.5			<i>arch</i>	2-[68]	49F2-49F13	49f100	<i>l(2)49Fc</i>
2-56.6			<i>ta</i>	2-60.5	47E-47F		<i>ix</i>	2-[68]	49F2-49F13	49f100	<i>l(2)49Fd</i>
2-56.8	43B-43C		<i>phr</i>	2-60.7			<i>ad</i>	2-[68]	49F2-49F13	49f100	<i>l(2)49Fe</i>
2-57			<i>stmA</i>	2-60.8			<i>chl</i>	2-[68]	49F2-49F13	49f100	<i>l(2)49Ff</i>
2-57			<i>dil</i>	2-61			<i>brh</i>	2-[68]	49F2-49F13	49f100	<i>l(2)49Fg</i>
2-57	44C		<i>M(2)44C</i>	2-61			<i>cuff</i>	2-[68]	49F2-49F13	49f100	<i>l(2)49Fh</i>
2-57	43E1-43F8		<i>mat(2)cell-J</i>	2-61			<i>fai</i>	2-[68]	49F2-49F13	49f100	<i>l(2)49Fi</i>
2-57			<i>ms(2)E9</i>	2-61			<i>mat(2)ea-C</i>	2-[68]	49F2-49F13	49f100	<i>l(2)49Fj</i>
2-57			<i>M(Sd)</i>	2-61			<i>ms(2)E11</i>	2-[68]	49F2-49F13	49f1000	<i>l(2)49Fk</i>
2-57	43E5-43E8	43e600	<i>tor</i>	2-61			<i>whd</i>	2-[68]	49F13-50A2	49f100	<i>l(2)49Fl</i>
2-[57]	42C1-43E3		<i>crib</i>	2-[61]	47B1		<i>zep</i> [†]	2-[68]	49F13-50A2	49f1100	<i>l(2)49Fm</i>
2-[57]	43F3-43F6			2-61.0			<i>E(Pc)</i>	2-[68]	49F13-50A2	49f1300	<i>Cos</i>
2-[57]	43E		<i>Gapdh1</i>	2-61.5			<i>l(2)Sp8</i>	2-[68]	49F13-50A2	49f1300	<i>l(2)49Fo</i>
2-[57]	43E1-43E4		<i>Jon43E</i>	2-61.5			<i>l(2)Sp12</i>	2-68.2			<i>ms(2)E12</i>
2-[57]	42C1-43E3		<i>sax</i>	2-62			<i>frh</i>	2-[69]	50C1-50C4		<i>tRNA:lys2:50C</i>
2-[57]	43B1-43C7	43b300	<i>l(2)43Bb</i>	2-62			<i>fs(2)lto4</i>	2-[69]	50C1-50C4		<i>tRNA:tyr1:50C</i>
2-[57]	43B1-43C7	43b400	<i>l(2)43Bc</i>	2-62			<i>ret</i>	2-[69]	49F-50B	49ff(100)	<i>tRNA:ile:49Fa</i>
2-[57]	43B1-43B3	43b500	<i>cos1</i>	2-62	47E3-48A6		<i>sha</i>	2-[69]	49F-50B	49ff(200)	<i>tRNA:ile:49Fb</i>
2-[57]	43B1-43C7	43b600	<i>hum</i> [†]	2-62	47E3-48A6		<i>shn</i>	2-[69]	49F-50B	49ff(300)	<i>tRNA:ile:49Fb</i>
2-[57]	43B1-43C7	43b600	<i>l(2)43Bd</i> [†]	2-62			<i>shy</i>	2-[69]	49F-50B	49ff(400)	<i>tRNA:ile:49Fc</i>
2-[57]	43C1-43C7	43c200	<i>l(2)43Ca</i> [†]	2-62			<i>upw</i>	2-[69]	49F-50B	49ff(500)	<i>tRNA:ile:49Fd</i>
2-[57]	43C1-43C7	43c300	<i>l(2)43Cb</i> [†]	2-[62]	47F		<i>tRNA:thr3:47F</i>	2-[69]	49F-50B	49ff(600)	<i>tRNA:ile:49Fe</i>
2-[57]	43C1-43C7	43c300	<i>l(2)43Cc</i> [†]	2-[62]	48A2	48a200	<i>inv</i>	2-[69]	49F-50B	49ff(700)	<i>tRNA:ile:49Fb</i>
2-[57]	43D1-43D7	43d100	<i>l(2)43Da</i> [†]	2-62.0			<i>che</i>	2-69.1			<i>mus210</i>
2-[57]	43D1-43D7	43d200	<i>l(2)43Db</i> [†]	2-62.0	48A2	48a100	<i>en</i>	2-69.7			<i>l(2)K508</i>
2-[57]	43E1-43E5	43e100	<i>l(2)43Ea</i> [†]	2-62.6			<i>fs(2)E8</i>	2-69.7			<i>wx</i>
2-[57]	43E1-43E5	43e200	<i>l(2)43Eb</i> [†]	2-62.7			<i>Eye</i>	2-70			<i>l(2)64</i>
2-[57]	43E1-43E5	43e200	<i>scra</i>	2-63			<i>Ind</i>	2-70			<i>l(2)mr</i>
2-[57]	43E5-43E8	43e400	<i>l(2)43Ed</i> [†]	2-63			<i>mat(2)syn-G</i>	2-70			<i>lop</i>
2-[57]	43E5-43E8	43e500	<i>l(2)43Ee</i> [†]	2-63	48D-49E		<i>stil</i>	2-[70]	50F		<i>dsx-c50F</i> [†]
2-[57]	43E5-43E8	43e600	<i>l(2)43Ef</i> [†]	2-[63]	48B5-48B7		<i>tRNA:met2:48Ba</i>	2-[70]	50E4-50E7		<i>Hsc70-5F</i> [†]
2-[57]	43E5-43E8	43e600	<i>l(2)43Eg</i> [†]	2-[63]	48B5-48B7		<i>tRNA:met2:48Bb</i>	2-[70]	50D-50E		<i>Pen50DE</i> [†]
2-[57]	43E5-43E8	43e900	<i>l(2)43Eh</i> [†]	2-63.3			<i>spt</i>	2-70.1			<i>dv2</i>
2-[57]	43E8-43E13	43e1000	<i>fs(2)43Ei</i> [†]	2-63.5			<i>twl</i>	2-70.1			<i>ft(2)d</i>
2-57.1			<i>buo</i>	2-63.6			<i>sps</i>	2-70.3	50C20-50C23		<i>mam</i>
2-57.1	43B1-43B2	43c100	<i>so</i>	2-64			<i>fs(2)eo4</i>	2-70.8			<i>Pfd</i>
2-57.5			<i>Hg</i>	2-64			<i>mat(2)cell-E</i>	2-[71]	51B4-51B6		<i>tra2</i>
2-57.5			<i>Ps</i>	2-64.0			<i>E(Bic)</i>	2-71.0			<i>bat</i>
2-57.5	43E8-43E13	43e1000	<i>cn</i>	2-[64]	49A		<i>Cal49A</i>	2-71.1			<i>cg</i>
2-57.6			<i>fs(2)E1</i>	2-[64]	48D		<i>Efla48D</i>	2-71.2			<i>cml</i>
2-57.6			<i>Su(var)2-10</i>	2-[64]	48F-49A		<i>tRNA:his:48F</i>	2-71.2			<i>dr</i>
2-57.8			<i>l(2)K513</i>	2-[64]	48E12-48F1	48ef(50)	<i>DebC</i> [†]	2-71.4			<i>Fs(2)Sz4</i>
2-57.9			<i>l(2)K234</i>	2-[64]	48E12-48F1	48ef(100)	<i>DebA</i> [†]	2-71.4			<i>thiv</i>
2-58			<i>flo</i>	2-[64]	48E12-48F1	48ef(200)	<i>DebB</i> [†]	2-71.5	51C-51E		<i>sf</i>
2-58			<i>puf</i>	2-64.7	48A-49D		<i>Dmnd</i>	2-72	51A1-51B4		<i>Asx</i>
2-[58]	44A		<i>Myc</i>	2-65			<i>l(2)ja</i>	2-72			<i>l(2)me</i>
2-[58]	44E-44F		<i>tRNA:leu2:44EF</i>	2-65			<i>Bkd</i>	2-72			<i>M(2)d</i>
2-[58]	44D	44d(100)	<i>Lcp1</i>	2-65			<i>luc</i>	2-72	51A1-51B4		<i>mat(2)ea-A</i>
2-[58]	44D	44d(200)	<i>Lcp1y1</i>	2-65	48E-49B		<i>M(2)40c</i>	2-72	49E7-50A2		<i>psi2</i>
2-[58]	44D	44d(300)	<i>Lcp2</i>	2-65			<i>mat(2)syn-C</i>	2-72			<i>thi</i>
2-[58]	44D	44d(400)	<i>Lcp3</i>	2-[65]	49B		<i>G-a49B</i> [†]	2-72.0	51A2		<i>L</i>
2-[58]	44D	44d(500)	<i>Lcp4</i>	2-65.1			<i>l(2)K255</i>	2-72.3	51C-51E		<i>kn</i>
2-[58]	44D	44d(600)	<i>LvpH</i>	2-65.2			<i>po</i>	2-72.5			<i>PL(2)L4</i>
2-[58]	44D	44d(700)	<i>LvpD</i>	2-65.2			<i>Su(z)6</i> [†]	2-73			<i>cxb</i>
2-[58]	44D	44d(800)	<i>LvpL</i>	2-65.4			<i>l(2)K333</i>	2-73			<i>l(2)IP85</i> [†]
2-58.4			<i>l(2)K207</i>	2-65.5			<i>ms(2)l</i>	2-73			<i>scb</i>
2-58.5			<i>blo</i>	2-65.6			<i>cht</i>	2-73			<i>dke</i>
2-58.6			<i>Pgi</i>	2-66			<i>arr</i>	2-[73]	51F		<i>Mst51F</i>
2-58.6			<i>smk</i>	2-66.2			<i>tu(2)W</i>	2-[73]	51D1-51E1	51d100	<i>l(2)51Da</i>
2-59	44F-46D		<i>flz</i>	2-66.3			<i>Tyr2</i>	2-[73]	51D1-51E1	51d100	<i>l(2)51Db</i>
2-59	44F-46D		<i>lin</i>	2-66.5			<i>ms(2)E10</i>	2-[73]	51D1-51E1	51d100	<i>l(2)51Dc</i>
2-59			<i>mat(2)syn-D</i>	2-66.7	49D1-49D3		<i>sca</i>	2-[73]	51E1-51E5	51d400	<i>l(2)Ta</i> [†]
2-59			<i>misp</i>	2-66.8			<i>Srf</i>	2-[73]	51E5-51F1	51e100	<i>l(2)51Ea</i> [†]
2-59	44D3-44D4		<i>tuf</i>	2-67	49E6-49F1		<i>Arp</i>	2-[73]	51E5-51F1	51e100	<i>l(2)51Eb</i> [†]
2-[59]	46D		<i>Dhr3</i> [†]	2-67	49D3-49E6		<i>bic</i>	2-[73]	51E5-51F1	51e300	<i>l(2)51Ec</i> [†]
2-[59]	46C3-46C11		<i>eve</i>	2-67			<i>mat(2)cell-B</i>	2-[73]	51F1-52A5	51f100	<i>fs(2)51Fa</i> [†]
2-[59]	46C		<i>Fmrf</i>	2-67	49E2-49F1		<i>Psc</i>	2-[73]	51F1-52A5	51f200	<i>fs(2)51Fb</i> [†]
2-[59]	46E-47E		<i>Inr-a</i> [†]	2-67			<i>Str</i>	2-[73]	51F1-52A5	51f200	<i>fs(2)abc</i> [†]
2-[59]	46E		<i>Jra</i> [†]	2-[67]	49F		<i>TpnC</i>	2-[73]	51F1-52A5	51f200	<i>fs(2)sun</i> [†]
2-[59]	44D-46E		<i>ltd</i>	2-[67]	49D	49d100	<i>l(2)49Da</i>	2-[73]	51F1-52A5	51f200	<i>l(2)51F</i> [†]
2-[59]	45C		<i>Pk?3</i>	2-[67]	49D	49d100	<i>l(2)49Db</i>	2-[73]	52A6-52D	52a100	<i>l(2)52AD</i>
2-[59]	45A11-45B5		<i>rad201</i>	2-[67]	49E7-49F1	49e100	<i>l(2)49Ea</i>	2-73.0			<i>Su(z)3</i> [†]
2-[59]	46E-46F		<i>Syb</i>	2-[67]	49E	49e200	<i>Su(z)2</i>	2-73.8			<i>ch</i>

CYTOGENETIC MAP

genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol
2-74			<i>gau</i> [†]	2-[87]	56D		<i>tRNA:tyr1:56D</i>	2-99.2	57F11-58E4		<i>a</i>
2-74			<i>gp</i>	2-[87]	56D3-56D7		<i>tRNA:val4:56D</i>	2-100			<i>fs(2)K</i>
2-74			<i>scrp</i>	2-87.5	56C1-56D2		<i>M(2)56CD</i>	2-100			<i>pkh</i>
2-74			<i>Su(f)</i>	2-88			<i>plu</i> [†]	2-[100]	58A-58B		<i>Ets-2</i>
2-74.5	51B-52E		<i>Hex-C</i>	2-88			<i>rib</i>	2-[100]	58B1-58C5		<i>mei5332</i>
2-75	52D9-52D15		<i>Got1</i>	2-[88]	56F		<i>l8w</i>	2-[100]	58A-58B		<i>tRNA:gly:58AB</i>
2-[75]	52C-52D		<i>Pox-n</i>	2-[88]	56E-56F		<i>Pcna</i> [†]	2-100.5	58F		<i>px</i>
2-[75]	52C5-52D3	52c200	<i>l(2)52Da</i>	2-[88]	56E		<i>tRNA:his:56E</i>	2-101	58F1-60F1		<i>inaD</i>
2-[75]	52D9-52E5	53d100	<i>l(2)52Ea</i>	2-[88]	56E-56F		<i>tRNA:lys2:56EF</i>	2-[101]	59A-59B		<i>CycB</i>
2-75.5	52D2-52D5	52c100	<i>Gpo</i>	2-[88]	56E-56F		<i>tRNA:met3:56EF</i>	2-101.0	58F2-60E2		<i>pa</i>
2-75.5	52D1-52D7	52c300	<i>c</i>	2-[88]	56E-56F		<i>tRNA:phe2:56EF</i>	2-101.2	58F		<i>M(2)58F</i>
2-76			<i>stop</i>	2-[88]	56E-56F		<i>tRNA:thr6:56EF</i>	2-101.8			<i>l(2)K327</i>
2-76			<i>Wr</i>	2-[88]	56D4-56D12		<i>Tubb56D</i>	2-102			<i>stl</i>
2-[76]	53A		<i>U</i>	2-[88]	56E-56F	56ef(100)	<i>tRNA:gly3:56EFa</i>	2-102			<i>tft</i>
2-77			<i>clo</i>	2-[87]	56E-56F	56ef(200)	<i>tRNA:gly3:56EFb</i>	2-[102]	59C5		<i>Pen59C5</i>
2-77			<i>cra</i>	2-[88]	56F	56f200	<i>tRNA:glu4:56Fa</i>	2-[102]	59C3-59D2		<i>twi</i>
2-77	52D		<i>slf</i>	2-[88]	56F	56f210	<i>tRNA:glu4:56Fb</i>	2-102.4	59C1-59C4		<i>Frd</i>
2-[77]	52F-53A		<i>tRNA:glu4:53A</i>	2-[88]	56F	56f220	<i>tRNA:glu4:56Fc</i>	2-103			<i>Su(w^{co2})</i>
2-77.5	52D-53E		<i>M(2)53</i>	2-88.0			<i>tu(2)bw</i>	2-103.3	59D2-59D8		<i>vir</i>
2-77.7	54B1	54b100	<i>AmyB</i>	2-89			<i>rub</i>	2-103.5	59D4-59D10		<i>ord</i>
2-77.7	54B1	54b200	<i>AmyA</i>	2-89			<i>Sdh</i>	2-104			<i>l(2)bw</i>
2-78			<i>mat(2)ea-D</i>	2-89.7			<i>fs(2)TLM</i>	2-104			<i>nuc1</i>
2-[78]	52F10-52F11		<i>Kin</i>	2-89.8			<i>mus208</i>	2-[104]	59D8-60A7		<i>pep</i>
2-[78]	53C		<i>Pk?7</i>	2-90			<i>dsr</i>	2-104.0			<i>hv</i>
2-[78]	53E4-53E7		<i>Pkc1</i>	2-90			<i>l(2)56a</i>	2-104.5	59D4-59E1		<i>bw</i>
2-[78]	53E4-53E7		<i>Pkc2</i>	2-90	56F1-56F9		<i>min</i>	2-104.7	59E1-59E2		<i>mi</i>
2-[78]	53C-53D		<i>RpA1</i>	2-[90]	56F1-56F9	56f100	<i>5SRNA</i>	2-105	59F		<i>egl</i>
2-[78]	53E		<i>tRNA:gly3:53E</i>	2-91.5	56E		<i>sm</i>	2-105			<i>fs(2)eo6</i>
2-78.8	54A-55A		<i>mapA</i>	2-92			<i>shg</i>	2-105			<i>fs(2)eo7</i>
2-78.8			<i>mapP</i>	2-[92]	57A		<i>Act57A</i>	2-105	59D8-60A1		<i>qui</i>
2-79			<i>pw-c</i>	2-92.3	56F5-56F15		<i>l(2)56Fa</i>	2-105	59D8-60A1		<i>shu</i>
2-[79]	54A		<i>anon-54A</i>	2-92.3	56F5-56F15		<i>l(2)56Fb</i>	2-105.1	60B8-60B10		<i>E(w^a)</i>
2-79.6	54A-55A		<i>nw</i>	2-92.3	56F5-56F15		<i>l(2)56Fd</i>	2-105.5	59E2-60B10		<i>abb</i>
2-79.7			<i>mei58</i>	2-92.3	56F5-56F15		<i>l(2)56Fe</i>	2-106	60A7-60B10		<i>sei</i>
2-798			<i>Su(var)2-Z</i>	2-92.3	56F5-56F15		<i>l(2)56Ff</i>	2-[106]	60A		<i>Ca-P60A</i> [†]
2-80			<i>fr</i>	2-92.3	56F5-56F15		<i>l(2)56Fg</i>	2-[106]	60A3-60A7		<i>gcn</i> [†]
2-[80]	54D		<i>Klp54D</i> [†]	2-92.3	56F5-56F15		<i>M(2)56F</i>	2-[106]	60A		<i>Ga60A</i>
2-[80]	55A		<i>PAbp</i>	2-92.8			<i>mus209</i>	2-[106]	59F5-59F8		<i>l(2)tud</i> [†]
2-[80]	54D		<i>prd11</i> [†]	2-93	57A4-57B1	57a400	<i>exu</i>	2-[106]	60B-60C		<i>Mov34</i> [†]
2-[80]	54C		<i>rhi</i>	2-[93]	57A2-57A3	57a100	<i>l(2)57Aa</i> [†]	2-[106]	59F		<i>tRNA:asn5:59F</i>
2-80.6			<i>Bc</i>	2-[93]	57A2-57A3	57a100	<i>l(2)57Ab</i> [†]	2-106.3			<i>slt</i>
2-80.6			<i>Dox-A1</i>	2-[93]	57A2-57A3	57a100	<i>l(2)57Ac</i> [†]	2-106.4	59E2-60B10		<i>pd</i>
2-80.6			<i>Phox</i>	2-[93]	57A4-57B1	57a400	<i>l(2)57Ad</i> [†]	2-106.7			<i>l(2)ax</i>
2-81			<i>rf</i>	2-[93]	57A4-57B1	57a400	<i>l(2)57Ae</i> [†]	2-106.7	59E2-60B10		<i>ll</i>
2-81.5			<i>fj</i>	2-[93]	57A4-57B1	57a400	<i>l(2)57Af</i> [†]	2-106.7	60A8-60B10		<i>mr</i>
2-81.6			<i>tbs</i>	2-[93]	57A4-57B1	57a400	<i>l(2)57Ag</i> [†]	2-106.7	60A8-60A16		<i>or</i>
2-82			<i>inaC</i>	2-93.2			<i>rw</i>	2-107	60B1-60B10		<i>Dat</i>
2-82			<i>l(2)MV</i>	2-93.3	57A-57F		<i>hy</i>	2-107	58E3-60B10		<i>Fo</i>
2-82			<i>Off</i>	2-94			<i>meiW68</i>	2-[107]	60C7-60C8		<i>Acr60C</i>
2-82			<i>Pur-r</i>	2-94			<i>su(B)</i>	2-[107]	60B-60C		<i>brl</i>
2-82.3			<i>rbl</i>	2-96			<i>clt</i>	2-[107]	60E		<i>NaCP60E</i>
2-82.9	54B1-54E8		<i>aldox2</i>	2-96.2			<i>cv-2</i>	2-[107]	60C		<i>tRNA:asn5:60C</i>
2-83			<i>abr</i>	2-97			<i>bie</i>	2-[107]	60C5-60D1	60c100	<i>l(2)60Ca</i> [†]
2-[83]	54F1-54F2		<i>Ntf</i>	2-97	57B4-58B		<i>grau</i> [†]	2-[107]	60C5-60D1	60c100	<i>l(2)60Cb</i> [†]
2-[83]	55A1-55A3		<i>PpY-55A</i> [†]	2-97	57C5	57c300	<i>Pu</i>	2-[107]	60C5-60D1	60c100	<i>l(2)60Cc</i> [†]
2-83.1			<i>sdp</i>	2-97	57C7-57C9	57c600	<i>tud</i>	2-[107]	60C5-60D1	60c100	<i>l(2)60Cd</i> [†]
2-83.4	55A-55C2		<i>adp</i>	2-[97]	57B5-57B6		<i>E97</i> [†]	2-[107]	60D1-60D10	60d100	<i>l(2)60Da</i> [†]
2-83.5	55A1-55A2		<i>stau</i>	2-[97]	57B-57C		<i>Pen57BC</i> [†]	2-[107]	60D1-60D10	60d100	<i>l(2)60Db</i> [†]
2-84			<i>cha</i>	2-[97]	57B-57C		<i>tRNA:gly3:57BCa</i>	2-[107]	60D1-60D10	60d100	<i>l(2)60Dc</i> [†]
2-84			<i>Eco</i>	2-[97]	57B-57C		<i>tRNA:gly3:57BCb</i>	2-[107]	60D1-60D10	60d100	<i>l(2)60Dd</i> [†]
2-84			<i>ms(2)10R</i>	2-[97]	57B18-57B19	57b100	<i>l(2)57Ba</i>	2-[107]	60D1-60D10	60d100	<i>l(2)60De</i> [†]
2-84	55B-55C1		<i>Pcl</i>	2-[97]	57B18-57B19	57b100	<i>l(2)57Bb</i>	2-[107]	60D1-60D10	60d100	<i>l(2)60Df</i> [†]
2-[84]	55B-55E		<i>Treh</i>	2-[97]	57B17-57B19	57b100	<i>l(2)57Bc</i>	2-[107]	60D1-60D10	60d100	<i>l(2)60Dg</i> [†]
2-85			<i>fs(2)eo5</i>	2-[97]	57B11-57B19	57b100	<i>l(2)57Bd</i>	2-[107]	60D9-60D10	60d800	<i>Tubb60D</i>
2-85.2	55E		<i>Pfk</i>	2-[97]	57B20-57C3	57c100	<i>l(2)57Ca</i>	2-107.0			<i>l(2)NS</i>
2-85.2			<i>Su(z)7</i> [†]	2-[97]	57C4	57c200	<i>l(2)57Cb</i>	2-107.0	60B13-60C5		<i>sp</i>
2-85.3			<i>wt</i>	2-[97]	57C6	57c400	<i>l(2)57Cc</i>	2-107.2	60C6-60D1	60c100	<i>Px</i>
2-85.6	55F		<i>tn</i>	2-[97]	57C6	57c400	<i>l(2)57Cd</i>	2-107.3	60C5-60D2		<i>Pin</i>
2-86	55A-55F		<i>grh</i> [†]	2-[97]	57C7-57C9	57c600	<i>l(2)57Ce</i>	2-107.3	60C5-60D2	60c100	<i>bs</i>
2-86			<i>mat(2)cell-L</i>	2-[97]	57D6	57d100	<i>l(2)57Da</i>	2-107.5	60E6-60E10		<i>E(br)</i> [†]
2-86	55A-55F		<i>sub</i>	2-[97]	57D7	57d200	<i>l(2)57Db</i>	2-107.6	60F3		<i>lf</i>
2-86	55A-55F		<i>thr</i>	2-[97]	57D8-57D10	57d300	<i>l(2)57Dc</i>	2-107.6	60F3		<i>Kr</i>
2-86			<i>wbl</i>	2-[97]	57D8-57D10	57d300	<i>l(2)57Dd</i>	2-107.6	60E9-60F1	60f100	<i>nzip</i> [†]
2-[86]	55A-55F		<i>eay</i>	2-[97]	57D8-57D10	57d300	<i>l(2)57De</i>	2-107.6	60E9-60F1	60f200	<i>gsb-p</i>
2-[86]	55E3-55E4		<i>Eip55BD</i>	2-[97]	57D11-57F4	57e100	<i>l(2)57Eb</i>	2-107.6	60E9-60F1	60f300	<i>gsb-d</i>
2-[86]	55A-55F		<i>fs(2)lto5</i>	2-[97]	57E1-57E4	57e100	<i>mat(2)N</i>	2-107.8	60D14-60E2		<i>Ba</i>
2-[86]	55A-55F		<i>hal</i> [†]	2-[97]	57D11-57F4	57e300	<i>l(2)57Ec</i>	2-108	60E3-60E11		<i>M(2)60E</i>
2-[86]	55A		<i>Hsf</i> [†]	2-[97]	57F1	57e400	<i>Egfr</i>	2-108	60E9		<i>Mhc-c</i>
2-[86]	55C		<i>Ote</i> [†]	2-97.6			<i>stmB</i>	2-[108]	60E11-60F5		<i>Peb</i> [†]
2-[86]	55E		<i>tRNA:gly3:55E</i>	2-98	57B4-58B		<i>prat</i>	2-[108]	60E9	60f150	<i>zip</i>
2-86.5			<i>E(f)</i> [†]	2-99			<i>l(2)Su(H)</i>	2-[108]	60E9-60F1	60f350	<i>meso</i> [†]
2-87	56A-56F		<i>enb</i> [†]	2-[99]	58F8-58F11		<i>HmgD</i> [†]	2-[108]	60F1	60f400	<i>Tkr</i>
2-[87]	56A		<i>Dpt</i> [†]	2-[99]	57D		<i>Mst57D</i>	2-109.9			<i>TipB</i>
2-[87]	56D3-56D7		<i>tRNA:ser4:56D</i>	2-[99]	57F8-57F10		<i>TfIID</i> [†]	2-110			<i>tipD</i>

THE GENOME OF *DROSOPHILA MELANOGASTER*

genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol
3-[0]	61F		<i>Klp61F</i> [†]	3-[6]	63E		<i>ImpE2</i>	3-25.8			<i>mle3</i>
3-[0]	61F-62A		<i>M(3)62A</i>	3-[6]	63F		<i>Ubi-p</i>	3-25.9			<i>l(3)ds11</i>
3-[0]			<i>marb</i> [†]	3-[6]	63D3-63F1	63e100	<i>l(3)63Ea</i>	3-25.9			<i>l(3)SG13</i>
3-[0]	61A-64E		<i>Rnase2</i>	3-[6]	63D3-63F1	63e100	<i>l(3)63Eb</i>	3-26			<i>lfb</i>
3-[0]	61A-61E		<i>su(ve)</i>	3-[7]	63E-64		<i>Cg63-64</i>	3-26			<i>On</i>
3-[0]	61D1-61D2		<i>tRNA:met3:61Dd</i>	3-7.7			<i>Coa</i>	3-26	66A-66C		<i>pbl</i>
3-[0]	61D1-61D2		<i>tRNA:met-i</i>	3-[8]	64A2-64B12		<i>Acr64B</i>	3-26			<i>su(t)</i>
3-[0]	61D1-61D2		<i>tRNA:met-i:61Da</i>	3-[8]	64A3-64A5		<i>Gad</i>	3-[26]	66D14	66d200	<i>Arr2</i>
3-[0]	61F		<i>tRNA:thr6:61F</i>	3-10			<i>Gab</i>	3-[26]	66D11-66D15		<i>Cp15</i>
3-[0]	61E2-62A3	61e200	<i>mwh</i>	3-10.6			<i>l(3)F34</i>	3-[26]	66D11-66D15		<i>Cp16</i>
3-[0]	61F1-61F4	61f10	<i>LysB</i> [†]	3-10.7			<i>l(3)SG4</i>	3-[26]	66D11-66D15		<i>Cp18</i>
3-[0]	61F1-61F4	61f40	<i>LysE</i> [†]	3-10.8			<i>Lcp5</i>	3-[26]	66D11-66D15		<i>Cp19</i>
3-[0]	61F1-61F4	61f50	<i>LysP</i> [†]	3-10.8			<i>Lcp6</i>	3-[26]	66C		<i>ImpE1</i>
3-[0]	61F		<i>dsx-c61F</i> [†]	3-10.8			<i>Lcp7</i>	3-[26]	66C		<i>Jon66C</i>
3-[0]	61A1-61A6		<i>E(bx)</i>	3-10.8			<i>Lcp8</i>	3-[26]	66D		<i>Mst66D</i>
3-[0]	61A-61B		<i>fwd</i>	3-10.8			<i>Lcp9</i>	3-[26]	66E1-66E2	66d400	<i>Prm</i> [†]
3-[0]	61F1-61F4		<i>LysS</i> [†]	3-11.9			<i>Su(var)303</i> [†]	3-[26]			<i>pts</i>
3-[0]	61C		<i>prd2</i> [†]	3-12	62D-64C		<i>dib</i>	3-[26]	66B5-66B8		<i>tRNA:leu2:66B</i>
3-[0]	61E-61F		<i>trh</i>	3-12			<i>dw-b</i>	3-26.0	66D13	66d100	<i>se</i>
3-[0]	61D1-61D2		<i>tRNA:met3:61De</i>	3-[12]	64B		<i>ImpL2</i>	3-26.1			<i>E(ade2)</i> [†]
3-[0]	61D1-61D2		<i>tRNA:met3:61Df</i>	3-12.1			<i>l(3)SG5</i>	3-26.5	66D15	66d300	<i>h</i>
3-[0]	61D1-61D2		<i>tRNA:met3:61Dg</i>	3-12.4			<i>l(3)SG6</i>	3-26.6			<i>l(3)SG14</i>
3-[0]	61D1-61D2		<i>tRNA:met-i:61Db</i>	3-13			<i>PL(3)w-S</i>	3-27			<i>abd</i>
3-[0]		61e100	<i>fap</i>	3-13.5	64B2		<i>tipE</i>	3-[27]	67A		<i>Klp67A</i> [†]
3-[0]	61F1-61F4	61f20	<i>LysC</i> [†]	3-14.7			<i>l(3)F55</i>	3-[27]			<i>Difl</i>
3-[0]	61F1-61F4	61f30	<i>LysD</i> [†]	3-15	62D-64C		<i>srw</i>	3-[27]	67A2-67B1		<i>l(3)67Ac</i>
3-[0]	61F1-61F4	61f70	<i>LysX</i> [†]	3-[15]	64A2-64B12		<i>Ras2</i>	3-[27]	67A2-67B1		<i>l(3)67Ad</i>
3-0.0	61E2-62A6		<i>aa</i>	3-[15]	64B		<i>Src1</i>	3-[27]	67A2-67B1		<i>l(3)67Ae</i>
3-0.0			<i>bos</i>	3-15.2			<i>tipF</i>	3-[27]	67A2-67B1		<i>l(3)67Af</i>
3-0.0	61C3-61C4		<i>emc</i>	3-15.6			<i>l(3)SG7</i>	3-[27]	66F		<i>Rdl</i> [†]
3-0.0			<i>kls</i>	3-16.2			<i>vn</i>	3-[27]	67A2-67B1	67a100	<i>l(3)67Aa</i>
3-0.0			<i>l(3)ds14</i>	3-17			<i>btm</i> [†]	3-[27]	67A2-67B1	67a100	<i>l(3)67Ab</i>
3-0.0			<i>l(3)SG1</i>	3-17			<i>ndl</i>	3-[27]	67B1-67D13	67b100	<i>l(3)67BDa</i>
3-0.0			<i>mp</i>	3-17			<i>rai</i>	3-27.2	66B-66D		<i>ldh</i>
3-0.0			<i>she</i>	3-17			<i>spnC</i>	3-[28]	67C		<i>Cg11</i> [†]
3-0.0			<i>tyw</i>	3-18			<i>mus312</i>	3-[28]	67C		<i>Lamb2</i>
3-0.0	61F5-62A3	61f100	<i>ru</i>	3-18.0	64F		<i>ddd</i>	3-[28]	67C		<i>sxt</i> [†]
3-0.0	62B10-62C1	62b900	<i>mu2</i>	3-18.0			<i>l(3)L6A</i> [†]	3-[28]	67C4-67C6		<i>Tuba67C</i>
3-[0.0]	61A1	61a100	<i>Lsp1-g</i>	3-18.2			<i>l(3)F12</i>	3-[28]	67B1-67D13	67b100	<i>l(3)67BDb</i>
3-0.2	61F5-62A6	61f200	<i>ve</i>	3-18.8	65B		<i>ple</i>	3-[28]	67B1-67D13	67b100	<i>l(3)67BDc</i>
3-1.4	62B9-62B11	62b600	<i>R</i>	3-19			<i>Ama1</i>	3-[28]	67B1-67D13	67b100	<i>l(3)67BDd</i>
3-[1.4]	62B3-62B8	62b100	<i>l(3)62Bb</i>	3-19			<i>spo</i>	3-[28]	67B1-67D13	67b100	<i>l(3)67BDe</i>
3-[1.4]	62B3-62B8	62b100	<i>l(3)62Bc</i>	3-[19]	64C		<i>anon-64C</i> [†]	3-[28]	67B1-67D13	67b100	<i>l(3)67BDf</i>
3-[1.4]	62B3-62B8	62b100	<i>l(3)62Be</i>	3-[19]	64C-64D		<i>Edg64CD</i>	3-[28]	67B1-67D13	67b100	<i>l(3)67BDg</i>
3-[1.4]	62B3-62B8	62b300	<i>l(3)62Ba</i>	3-[19]	64D		<i>Klp64D</i>	3-[28]	67B1-67D13	67b100	<i>l(3)67BDh</i>
3-[1.4]	62B3-62B8	62b300	<i>l(3)62Bd</i>	3-[19]	64D		<i>tRNA:ser7:64D</i>	3-[28]	67B1-67D13	67b100	<i>l(3)67BDi</i>
3-1.5	62B9	62b600	<i>Aprt</i>	3-[19]	64D		<i>tRNA:val3a:64D</i>	3-[28]	67B1-67D13	67b100	<i>l(3)67BDj</i>
3-[1.5]	62C		<i>His1v</i> [†]	3-19.2	64C1-65E1		<i>rv</i>	3-[28]	67B1-67D13	67b100	<i>l(3)67BDk</i>
3-[1.5]	62B		<i>Rapl1</i>	3-19.2			<i>l(3)SG8</i>	3-[28]	67B1-67D13	67b100	<i>l(3)67BDl</i>
3-[1.5]	62B1-62B7		<i>Spec-a</i>	3-19.2	64C12-65E1		<i>Me</i>	3-[28]	67B1-67D13	67b100	<i>l(3)67BDm</i>
3-[1.5]	62A		<i>tRNA:lys2:62A</i>	3-19.6			<i>l(3)ds11</i>	3-[28]	67B1-67D13	67b100	<i>l(3)67BDn</i>
3-[1.5]	62A	62a(100)	<i>tRNA:glu4:62Aa</i>	3-19.6	64C12-65E1		<i>Vn</i>	3-[28]	67B1-67D13	67b100	<i>l(3)67BDp</i>
3-[1.5]	62A	62a(200)	<i>tRNA:glu4:62Ab</i>	3-20			<i>Bj1</i>	3-[28]	67B1-67D13	67b100	<i>l(3)67BDq</i>
3-[1.5]	62A	62a(300)	<i>tRNA:glu4:62Ac</i>	3-[20]	64F		<i>pr3</i> [†]	3-[28]	67B1-67D13	67b100	<i>l(3)67BDr</i>
3-[1.5]	62A	62a(400)	<i>tRNA:glu4:62Ad</i>	3-[20]	64F3-65D3		<i>l(3)mbn</i>	3-[28]	67B	67bb100	<i>Hsp-G3</i>
3-[1.5]	62A	62a(500)	<i>tRNA:glu4:62Ae</i>	3-[20]	64F		<i>Pk?6</i>	3-[28]	67B	67bb200	<i>Hsp-G2</i>
3-[1.5]	62B9-62B11	62b700	<i>l(3)62Bg</i>	3-[20]	64F-65A		<i>prd1</i> [†]	3-[28]	67B	67bb300	<i>Hsp22</i>
3-[1.5]	62B11-62B12	62b8008	<i>l(3)62Bi</i>	3-20.0	64C12-65E1		<i>dv</i>	3-[28]	67B	67bb400	<i>Hsp26</i>
3-[1.5]	62B11-62B12	62b900	<i>l(3)62Bh</i>	3-21			<i>l(3)F13</i>	3-[28]	67B	67bb500	<i>Hsp-G1</i>
3-[1.5]	62C1-62D1	62c100	<i>l(3)62Ca</i>	3-[21]	65A		<i>4.SSRNA</i>	3-[28]	67B	67bb600	<i>Hsp23</i>
3-[1.5]	62D1-62D5	62d100	<i>ecd</i>	3-[21]	65C		<i>Go65A</i>	3-[28]	67B	67bb700	<i>Hsp27</i>
3-[1.5]	62D1-62D5	62d100	<i>l(3)62Da</i>	3-[21]	65B		<i>ImpL3</i>	3-28.1			<i>Fs(3)Ven</i>
3-1.8			<i>mei-11</i>	3-[21]	65A		<i>Jon65A</i>	3-28.9	67C1-67C10	67b100	<i>M(3)67C</i>
3-[2]	62E		<i>M(3)62F</i>	3-[21]	65A10-65A11		<i>Lam-A</i>	3-29.2			<i>l(3)SG15</i>
3-2.4			<i>l(3)SG2</i>	3-[21]	65B		<i>Th</i>	3-29.3			<i>l(3)SG16</i>
3-[3]	63A		<i>Shab</i>	3-[22]	65D1-65D3		<i>Cfla</i> [†]	3-29.7			<i>l(3)ds18</i>
3-[3]	63A		<i>tRNA:ala:63A</i>	3-[22]	65D		<i>Klp65D</i> [†]	3-30			<i>cur</i>
3-[3]	63A		<i>USsnRNA63A</i> [†]	3-[22]	65D		<i>pr3</i> [†]	3-30.2			<i>PL(3)sp-T1</i>
3-4.3			<i>l(3)SG3</i>	3-[22]	65E		<i>vvl</i>	3-30.9			<i>l(3)ds12</i>
3-4.4			<i>aly</i> [†]	3-22.2			<i>l(3)SG9</i>	3-31			<i>Ne</i>
3-4.6			<i>l(3)F19</i>	3-22.5			<i>l(3)F23</i>	3-31.4			<i>Su(var)3-1</i>
3-5			<i>meiS282</i>	3-22.7			<i>rwi</i>	3-31.9			<i>l(3)SG18</i>
3-[5]	63C		<i>Fs(3)Lab</i>	3-23			<i>mus301</i>	3-32.6			<i>PL(3)sp-T2</i>
3-[5]	63B-63C		<i>Hsp83</i>	3-[23]	65F10-65F11		<i>M(3)65F</i>	3-[33]	67D8-67D11		<i>ect</i> [†]
3-[5]	63B		<i>tRNA:lys2:63B</i>	3-23.0	66A-66B		<i>Hn</i>	3-33.4			<i>l(3)DTS2</i>
3-[5]	63B-63C		<i>Spec-bH</i> [†]	3-23.3			<i>l(3)SG10</i>	3-33.8			<i>l(3)Sp1</i>
3-[5]	63A8-63B6	63a100	<i>l(3)63Aa</i>	3-24.0			<i>l(3)SG11</i>	3-[34]	67E-67F		<i>ERC33</i> [†]
3-[5]	63A8-63B6	63a100	<i>l(3)63Ab</i>	3-24.5	66F-67A		<i>l(3)DTS4</i>	3-[34]	68A8-68A9	68a600	<i>Sod</i>
3-[5]	63B6-63C1	63b100	<i>l(3)63Ba</i>	3-25			<i>be3</i>	3-34.0			<i>l(3)SG19</i>
3-[5]	63B6-63C1	63b100	<i>l(3)63Bb</i>	3-25			<i>tu(3)be</i>	3-34.0	67E3-67E4	67f100	<i>E(z)</i>
3-[5]	63B6-63C1	63b100	<i>l(3)63Bc</i>	3-[25]	65F10-66A		<i>M(3)66A</i> [†]	3-34.2	67E3-67F2		<i>Dk</i> [†]
3-[5]	63B6-63C1	63b100	<i>l(3)63Bd</i>	3-[25]	66A		<i>Tph</i> [†]	3-34.4	67E3-67F3		<i>hay</i>
3-[5]	63B6-63C1	63b100	<i>l(3)63Be</i>	3-25.2	66B		<i>Argk</i>	3-34.5	68A4-68A9	68a450	<i>lxd</i>
3-[5]	63B6-63C1	63b100	<i>M(3)63B</i>	3-25.5			<i>l(3)SG12</i>	3-34.7			<i>E(var)303</i>

CYTOGENETIC MAP

genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol
3-35			<i>l(3)F60</i>	3-39.0	69A4-69B1		<i>e(sei)</i>	3-[44]	73A4-73A7	73a400	<i>l(3)73Ae</i>
3-[35]	68A		<i>Pen68A</i> [†]	3-39.4			<i>l(3)SG23</i>	3-[44]	73A4-73A7	73a400	<i>l(3)73Af</i>
3-[35]	68A3-68A4	68a200	<i>l(3)68Aa</i>	3-39.5			<i>Fs(3)Apc</i>	3-[44]	73A4-73A7	73a400	<i>l(3)73Ag</i>
3-[35]	68A3-68A4	68a200	<i>l(3)68Ab</i>	3-39.5			<i>pur</i>	3-[44]	73A7-73A9	73a900	<i>l(3)73Ah</i>
3-[35]	68A4-68A6	68a500	<i>l(3)68Ac</i>	3-39.6			<i>Tcr</i>	3-[44]	73A10-73B1	73a1100	<i>mfs(3)73A</i>
3-[35]	68A5-68A9	68a700	<i>l(3)68Af</i>	3-40			<i>ni</i>	3-[44]	73B1-73B2	73a1400	<i>Abl</i>
3-[35]	68A5-68A9	68a800	<i>l(3)68Ae</i>	3-[40]	70C		<i>Fgf-r</i> [†]	3-[44]	73B1-73B2	73b100	<i>l(3)73Bb</i>
3-[35]	68A5-68A9	68a900	<i>l(3)68Ag</i>	3-[40]	70A		<i>ImpL1</i>	3-[44]	73B5-73B7	73b200	<i>l(3)73Bc</i>
3-[35]	68A5-68A9	68a900	<i>l(3)68Ah</i>	3-[40]	70A		<i>l(3)70Aa</i>	3-[44]	73B5-73B7	73b300	<i>Dab</i>
3-[35]	68A5-68A9	68a900	<i>l(3)68Ai</i>	3-[40]	70A		<i>l(3)70Ab</i>	3-[44]	73C5-73D1	73c100	<i>l(3)73Ca</i>
3-[35]	68A5-68A9	68a1200	<i>l(3)68Aj</i>	3-[40]	70A		<i>l(3)70Ac</i>	3-[44]	73C-73D	73c200	<i>l(3)73Cd</i>
3-[35]	68A5-68A9	68a1200	<i>l(3)68Ak</i>	3-[40]	70A		<i>PapB</i> [†]	3-[44]	73C2-73D1	73c200	<i>plk</i> [†]
3-[35]	68A5-68A9	68a1200	<i>l(3)68Am</i>	3-[40]	69F-70A		<i>tRNA:asp2:69F</i>	3-[44]	73D3-73D5	73d100	<i>Rh4</i>
3-[35]	68A5-68A9	68a1500	<i>l(3)68Al</i>	3-[40]	70B-70C		<i>tRNA:val4:70BCa</i>	3-[44]	73D3-73D5	73d100	<i>sina</i>
3-[35]	68A8-68B4	68b100	<i>l(3)68Ba</i> [†]	3-[40]	70B-70C		<i>tRNA:val4:70BCb</i>	3-44.0	73A3-73A4	73a300	<i>st</i>
3-[35]	68A8-68B4	68b100	<i>l(3)68Bb</i> [†]	3-40.0			<i>tt</i>	3-44.1			<i>l(3)ds17</i>
3-[35]	68A8-68B4	68b100	<i>l(3)68Bc</i> [†]	3-40.3			<i>e(dp\$+v\$-)</i>	3-44.1			<i>l(3)SG27</i>
3-[35]	68A8-68B4	68b100	<i>l(3)68Bd</i> [†]	3-40.4			<i>l(3)Sp6</i>	3-44.1	73A8-73A9	73a1000	<i>tra</i>
3-[35]	68A8-68B4	68b200	<i>l(3)68Be</i> [†]	3-40.5	70A3-70A5		<i>Ly</i>	3-44.3	73A9-73A10	73a1200	<i>l(3)73Ai</i>
3-35.0	68A1-68A2	68a100	<i>rs</i>	3-40.7	70C13-70D1		<i>D</i>	3-44.3	73A10-73B1	73a1300	<i>l(3)73Aj</i>
3-35.0	68C2-68C6	68c100	<i>Sgs8</i>	3-41	70D2-70D3		<i>dev</i>	3-44.4	73C2-73D1	73c200	<i>db</i>
3-35.0	68C2-68C6	68c200	<i>Sgs7</i>	3-41			<i>l(3)SG24</i>	3-45			<i>fle</i>
3-35.0	68C2-68C6	68c300	<i>Sgs3</i>	3-41	70D-71C		<i>shd</i>	3-45			<i>mus302</i>
3-35.5	67F2-68D6		<i>Fuc</i>	3-[41]	70D1-70D2		<i>Fbp1</i>	3-45			<i>re-b</i>
3-35.7	68A5-68A9	68a500	<i>l(3)68Ad</i>	3-[41]	70C		<i>Fgf</i> [†]	3-45			<i>ttx</i>
3-35.7			<i>l(3)ds10</i>	3-[41]	70C		<i>Hsc70-1</i>	3-45			<i>xwh</i>
3-35.9	69A1	69a300	<i>Est6</i>	3-[41]	70C		<i>scg</i>	3-[45]	75D1-75F1		<i>Cat</i>
3-35.9	69A1	69a350	<i>EstP</i> [†]	3-[41]	70D-70E		<i>snw</i>	3-[45]	75B		<i>E75</i>
3-36			<i>ome</i>	3-41.0			<i>l(3)Sp5</i>	3-[45]	75C-75D		<i>ftz-fl</i> [†]
3-[36]	68D		<i>Klp68D</i> [†]	3-41.3	70A-70F		<i>Su(var)</i>	3-[45]	75A		<i>Grd</i> [†]
3-[36]	68C8-68C11	68c400	<i>l(3)68Ca</i>	3-41.4	70C2		<i>Gl</i>	3-[45]	76A3-76B2		<i>M(3)76A</i>
3-[36]	68C8-68C11	68c500	<i>l(3)68Cb</i>	3-41.4			<i>l(3)SG25</i>	3-[45]	75C		<i>Mst75C</i>
3-[36]	68C8-68C11	68c600	<i>l(3)68Cc</i>	3-41.7	70D6-70D7		<i>fz</i>	3-[45]	75F-76B		<i>nkd</i>
3-[36]	68C8-68D3	68c700	<i>l(3)68Cd</i>	3-41.7			<i>l(3)Sp10</i>	3-[45]	75C		<i>prd8</i> [†]
3-[36]	68C8-68D3	68c700	<i>l(3)68Ce</i>	3-41.7			<i>rp</i>	3-[45]	75C1-75C2		<i>Term</i>
3-[36]	68C8-68D3	68c700	<i>l(3)68Cf</i>	3-42			<i>wk</i>	3-[45]	74E-74F	74f100	<i>E74</i>
3-[36]	68C8-68D3	68c700	<i>l(3)68Cg</i>	3-[42]	71C-71D		<i>Creb</i> [†]	3-[45]	74F	74f200	<i>Pep</i> [†]
3-[36]	68C8-68D3	68c700	<i>l(3)68Ch</i>	3-[42]	71C3-71D4		<i>gal</i>	3-45.3			<i>mat(3)3</i>
3-[36]	68D3-68D6	68d100	<i>l(3)68Da</i>	3-[42]	71A		<i>Gsg71A</i> [†]	3-45.9			<i>Dnase2</i>
3-[36]	68D3-68D6	68d100	<i>l(3)68Db</i>	3-[42]	71C3-71D2		<i>mex1</i> [†]	3-45.9			<i>ms(3)HO5A</i>
3-[36]	68D3-68D6	68d100	<i>l(3)68Dc</i>	3-[42]	71B		<i>mni</i> [†]	3-46			<i>bch</i>
3-[36]	68D6-68E4	68e100	<i>CycA</i>	3-[42]	71C2-71C3		<i>Sgs6</i>	3-46			<i>je</i>
3-[36]	68D6-68E4	68e100	<i>l(3)68Eb</i>	3-[42]	70F1-70F2		<i>tRNA:met3:70Fa</i>	3-46	76B-76D		<i>kto</i>
3-[36]	68D6-68E4	68e100	<i>l(3)68Ec</i>	3-[42]	70F1-70F2		<i>tRNA:met3:70Fb</i>	3-46			<i>ml</i>
3-[36]	68D6-68E4	68e100	<i>l(3)68Ed</i>	3-[42]	70F1-70F2		<i>tRNA:met3:70Fc</i>	3-46			<i>mus304</i>
3-[36]	68D6-68E4	68e100	<i>l(3)68Ee</i>	3-[42]	70F1-70F2		<i>tRNA:met3:70Fd</i>	3-46			<i>Pdr</i>
3-36.3	68C11-68D3	68c700	<i>vin</i>	3-[42]	71C3-71D4	71c100	<i>z600</i>	3-46	77A3		<i>polo</i>
3-36.5			<i>cr-3</i>	3-[42]	71C3-71D2	71c200	<i>Eip71CD</i>	3-46			<i>trc</i>
3-36.6			<i>E(var)302</i>	3-42.0	71A1-71A2		<i>Brd</i>	3-46			<i>vtd</i>
3-36.8	69C-70A		<i>l(3)SG21</i>	3-42.3			<i>l(3)DTS7</i>	3-[46]	74A-79D		<i>Ars</i>
3-37			<i>rag</i>	3-42.5	70E3-70F6		<i>gnu</i>	3-[46]	76B-76D		<i>ash1</i>
3-37	68C8-68C11		<i>rt</i>	3-42.8			<i>l(3)ds3</i>	3-[46]	77E1-77E2		<i>kni</i>
3-[37]	68E3-68E4	68e600	<i>l(3)68Ef</i>	3-42.8	71C-71F		<i>ms(3)nc16</i> [†]	3-[46]	77E1-77E2		<i>knrl</i>
3-[37]	68E3-68E4	68e600	<i>l(3)68Eg</i>	3-42.9			<i>l(3)SG26</i>	3-[46]	77B1-77B2		<i>rdgC</i> [†]
3-[37]	68E3-68E6	68e800	<i>l(3)68Fa</i>	3-[43]	71F-72B1		<i>l(3)72Ab</i>	3-[46]	77E-77F		<i>ri</i>
3-[37]	68E3-68E6	68e800	<i>l(3)68Fb</i>	3-[43]	71F-72B1		<i>l(3)72Ac</i>	3-[46]	76B		<i>Shal</i>
3-[37]	68E3-68E6	68e800	<i>l(3)68Fc</i>	3-[43]	71F-72B1		<i>l(3)72Ad</i>	3-[46]	76B		<i>Shl</i>
3-[37]	68E3-68E6	68e800	<i>l(3)68Fd</i>	3-[43]	72C-72D		<i>arl</i> [†]	3-[46]	77F	77f100	<i>anon-77F</i> [†]
3-[37]	68E3-68E6	68f600	<i>l(3)68Fe</i>	3-[43]	72D3-73A5		<i>as</i>	3-[46]	77F	77f100	<i>Pka-R1</i>
3-[37]	68E3-68E6	68f800	<i>l(3)68Ff</i>	3-[43]	72A		<i>Pka-C3</i>	3-46.0			<i>dic</i>
3-[37]	68F3-69A3	69a100	<i>l(3)69Aa</i>	3-43.0	72B		<i>brm</i>	3-46.0			<i>mot28</i>
3-[37]	68F3-69A3	69a200	<i>l(3)69Ab</i>	3-43.0			<i>We</i>	3-46.0	75C5-75D3		<i>W</i>
3-[37]	69A1-69A5	69a300	<i>l(3)69Ac</i>	3-43.2			<i>dhm</i>	3-46.3			<i>fs(3)HO5A</i>
3-[37]	69A1-69A5	69a300	<i>l(3)69Ad</i>	3-43.2	72B1		<i>th</i>	3-46.?			<i>l(3)SG28</i>
3-[37]	69A4-69B1	69a800	<i>l(3)69Ae</i>	3-43.3			<i>mb</i>	K?			
3-[37]	69A4-69B1	69a800	<i>l(3)69Af</i>	3-43.5			<i>Cm</i>				
3-[37]	69A4-69B1	69a1000	<i>l(3)69Ag</i>	3-43.6	72E4-72E5		<i>bul</i>				
3-[37]	69A5-69B5	69b100	<i>l(3)69Ba</i>	3-43.6	72D1-72D5		<i>Pgm</i>	3-46.5			<i>si3</i>
3-[37]	69A5-69B5	69b200	<i>l(3)69Bb</i>	3-43.8			<i>Su(var)3-2</i>	3-46.6			<i>Su(var)3-3</i>
3-[37]	69A5-69B5	69b200	<i>l(3)69Bc</i>	3-44	78A3-79E2		<i>ms(3)sa</i>	3-46.8			<i>Crn</i>
3-[37]	69A5-69B5	69b200	<i>l(3)69Bd</i>	3-44			<i>mus305</i>	3-46.9	77D3-77D5		<i>in</i>
3-37.0	68E3-68E4	68e800	<i>Lsp2</i>	3-44	73A		<i>Su(P)</i> [†]	3-47			<i>du</i>
3-37.3	69C	69b500	<i>gv</i>	3-44			<i>vr</i>	3-47			<i>grn</i>
3-37.5	69A2-69A4	69a300	<i>app</i>	3-[44]	75D4-79B1		<i>cp</i>	3-47			<i>kug</i>
3-37.5	69C	69b500	<i>eyg</i>	3-[44]	73A		<i>E111</i> [†]	3-47			<i>l(3)7E103</i> [†]
3-37.7			<i>sb</i>	3-[44]	72E		<i>Mab4A11</i> [†]	3-47			<i>mus310</i>
3-37.8			<i>l(3)SG22</i>	3-[44]	72F3-73A2		<i>MabF7D6</i> [†]	3-47			<i>mus311</i>
3-38.0			<i>md</i>	3-[44]	73C1-73C2		<i>Nrt</i>	3-47			<i>pip</i>
3-38.2	68D-70A		<i>Nuc</i>	3-[44]	72F-73A		<i>tRNA:met2:72Fa</i>	3-[47]	79B		<i>Act79B</i>
3-38.4			<i>l(3)Sp17</i>	3-[44]	72F-73A		<i>tRNA:met2:72Fb</i>	3-[47]	79C-79D		<i>Ape</i>
3-38.8			<i>fr</i>	3-[44]	73A2-73A3	73a100	<i>l(3)73Aa</i>	3-[47]	80A		<i>CklIα</i>
3-39			<i>pyd</i>	3-[44]	73A3-73A4	73a200	<i>l(3)73Ab</i>	3-[47]	79E1-79E2		<i>Csp</i> [†]
3-[39]	69C-69D		<i>E81</i> [†]	3-[44]	73A4-73A7	73a400	<i>l(3)73Ac</i>	3-[47]	79C-79D		<i>Dromsopa</i> [†]
3-[39]	69E1-69F		<i>M(3)69E</i>	3-[44]	73A4-73A7	73a400	<i>l(3)73Ad</i>	3-[47]	78E		<i>Edg78E</i>

THE GENOME OF *DROSOPHILA MELANOGASTER*

genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol
3-[47]	79A4-79B1		<i>eg</i>	3-47.6	84D2	84d100	<i>roe</i>	3-48.5			<i>com</i>
3-[47]	79B		<i>Egon</i>	3-47.7			<i>drb</i>	3-48.5	85F		<i>Scm</i>
3-[47]	78B		<i>M(3)KX305[†]</i>	3-47.7	84D13-84E2		<i>Su(var)3-4</i>	3-48.5			<i>tet</i>
3-[47]	79E5-80F		<i>M(3)80</i>	3-47.7	84D3-84D5		<i>EstC</i>	3-48.5	85C	85b200	<i>pum</i>
3-[47]	78D7-78D8		<i>Pc</i>	3-47.7	84D3-84D4	84d200	<i>rn</i>	3-48.5	85D4-85D7	85d100	<i>Tubb85D</i>
3-[47]	79B		<i>Pen79B[†]</i>	3-47.8			<i>wz</i>	3-48.6			<i>Su(var)319</i>
3-[47]	80		<i>Rp21</i>	3-47.8	84D13-84D14		<i>lds</i>	3-48.7			<i>l(3)ds14</i>
3-[47]	79E3-79E5		<i>Ten79E[†]</i>	3-47.8	84B3-84B6	84b1200	<i>Tuba84B</i>	3-48.7			<i>l(3)SG31</i>
3-[47]			<i>twr</i>	3-[47.8]	84D	84cc100	<i>Mst84Da[†]</i>	3-48.7			<i>l(3)SG32</i>
3-[47]	79F		<i>tRNA:leu2:79F</i>	3-[47.8]	84D	84cc200	<i>Mst84Db[†]</i>	3-48.7	85D11-85E3	85e200	<i>by</i>
3-[47]	80F	80f100	<i>l(3)80Fa</i>	3-[47.8]	84D	84cc300	<i>Mst84Dc[†]</i>	3-48.8	85D7-85D12		<i>sic</i>
3-[47]	80F	80f200	<i>l(3)80Fb</i>	3-[47.8]	84D	84cc400	<i>Mst84Dd[†]</i>	3-49			<i>cno</i>
3-[47]	80F	80f300	<i>l(3)80Fc</i>	3-48	84C8-84D1	84c800	<i>Gld</i>	3-49			<i>PL(3)sp-W1</i>
3-[47]	80F	80f400	<i>l(3)80Fd</i>	3-[48]	85A		<i>bel</i>	3-[49]	85D1-85D2		<i>D1[†]</i>
3-[47]	80F	80f400	<i>l(3)80Fe</i>	3-[48]	85C		<i>Ef2a</i>	3-[49]	85D8-85D13	85d200	<i>l(3)85Da</i>
3-[47]	80F	80f600	<i>l(3)80Ff</i>	3-[48]	84E7-87E11		<i>ImpE3</i>	3-[49]	85D11-85D12	85d300	<i>l(3)85Db</i>
3-[47]	80F	80f700	<i>l(3)80Fg</i>	3-[48]	85E10-85E15		<i>Mtna</i>	3-[49]	85D10-85D14	85d400	<i>l(3)85Dc</i>
3-[47]	80F	80f800	<i>l(3)80Fh</i>	3-[48]	84F4-84F12		<i>nac</i>	3-[49]	85D11-85E3	85d500	<i>l(3)85Dd</i>
3-[47]	80F	80f900	<i>l(3)80Fi</i>	3-[48]	84F-85A		<i>Pox-m</i>	3-[49]	85D11-85E3	85d500	<i>l(3)85De</i>
3-[47]	80F	80f1000	<i>l(3)80Fj</i>	3-[48]	85C-85D2		<i>prd7[†]</i>	3-[49]	85D11-85E3	85d500	<i>l(3)85Df</i>
3-47.0			<i>fs(3)272</i>	3-[48]	84E-84F		<i>tRNA:arg2:84Fd</i>	3-[49]	85D11-85E3	85d500	<i>l(3)85Dg</i>
3-47.0			<i>l(3)ds5</i>	3-[48]	84E-84F		<i>tRNA:arg2:84Fe</i>	3-[49]	85D11-85E3	85d500	<i>l(3)85Dh</i>
3-47.0			<i>l(3)SG29</i>	3-[48]	84E-84F		<i>tRNA:asn5:84Fc</i>	3-[49]	85D11-85E3	85d500	<i>l(3)85Di</i>
3-47.0			<i>ms(3)mc3</i>	3-[48]	85A		<i>tRNA:tyr1:85Aa</i>	3-[49]	85D11-85E3	85d500	<i>l(3)85Dj</i>
3-[47.1]	82F		<i>Cal82F[†]</i>	3-[48]	85A		<i>tRNA:tyr1:85Ab</i>	3-[49]	85D10-85D13		<i>fps85D[†]</i>
3-[47.1]	82C		<i>Dhr6[†]</i>	3-[48]	85A		<i>tRNA:tyr1:85Ac</i>	3-[49]	85E-86B		<i>hth</i>
3-[47.1]	81F		<i>Dsk</i>	3-[48]	85A		<i>tRNA:tyr1:85Ad</i>	3-[49]	85E		<i>l(3)85Ed</i>
3-[47.1]	82A-82C		<i>M(3)82BC</i>	3-[48]	85A		<i>tRNA:tyr1:85Ae</i>	3-[49]	85E		<i>l(3)85Ee</i>
3-[47.1]	82E1-82E3		<i>opa</i>	3-[48]	84D3-84D4		<i>tRNA:val3b:84Da</i>	3-[49]	85E		<i>l(3)hyd[†]</i>
3-[47.1]	83A		<i>Rp118[†]</i>	3-[48]	84D3-84D4		<i>tRNA:val3b:84Db</i>	3-[49]	85E15-85F6		<i>l(3)85Fa</i>
3-[47.1]	81F-82A		<i>Spl</i>	3-[48]	84D3-84D4		<i>tRNA:val3b:84Dc</i>	3-[49]	85E15-85F6		<i>l(3)85Fb</i>
3-[47.1]	82A		<i>str[†]</i>	3-[48]	84D3-84D4		<i>tRNA:val3b:84Dd</i>	3-[49]	85E15-85F6		<i>l(3)85Fc</i>
3-[47.1]	82A4-82A6		<i>tub</i>	3-[48]	84D5-84D8		<i>Tuba84D</i>	3-[49]	85E15-85F6		<i>l(3)85Fd</i>
3-[47.1]	82E		<i>U3snRNA82E[†]</i>	3-[48]	84D11-84E2	84d300	<i>l(3)84Da</i>	3-[49]	85D		<i>Ras1</i>
3-[47.1]	81F	81f100	<i>l(3)81Fa</i>	3-[48]	84D13-84D14	84d400	<i>l(3)84Db</i>	3-[49]	85C		<i>tRNA:arg:85C</i>
3-[47.1]	81F	81f200	<i>l(3)81Fb</i>	3-[48]	84D13-84D14	84d400	<i>l(3)84Dc</i>	3-[49]	86A		<i>tRNA:ser2b:86A</i>
3-[47.1]	82E	82e(100)	<i>U1snRNA82Ea[†]</i>	3-[48]	84D13-84D14	84d400	<i>l(3)84Dd</i>	3-[49]	85E6-85E10		<i>Tub85E</i>
3-[47.1]	82E	82e(200)	<i>U1snRNA82Ec[†]</i>	3-[48]	84F1-84F7	84d500	<i>l(3)84Fe</i>	3-[49]	85E2-85F1	85e300	<i>M(3)85E</i>
3-[47.1]	82E	82e(300)	<i>U1snRNA82Eb[†]</i>	3-[48]	84E2-84E8	84e100	<i>l(3)84Eb</i>	3-49.0	85E		<i>l(3)85Ea</i>
3-47.2			<i>l(3)SG30</i>	3-[48]	84E2-84E8	84e200	<i>l(3)84Eb</i>	3-49.0			<i>l(3)ds16</i>
3-[47.2]	83D-83E		<i>Ki</i>	3-[48]	84E8-84E9	84e300	<i>l(3)84Ec</i>	3-49.0			<i>l(3)SG34</i>
3-47.3			<i>Aph1</i>	3-[48]	84E8-84E9	84e300	<i>l(3)84Ed</i>	3-49.1	85E-86B		<i>knk</i>
3-47.3			<i>Su(var)323</i>	3-[48]	84E8-84E9	84e300	<i>l(3)84Ee</i>	3-49.2	85E		<i>l(3)85Eb</i>
3-47.4			<i>Su(var)316</i>	3-[48]	84E8-84E12	84e600	<i>l(3)84Ef</i>	3-49.2	86D1-86D4		<i>Odh</i>
3-47.4	83E1-83E2		<i>Tpl</i>	3-[48]	84E8-84E12	84e600	<i>l(3)84Eg</i>	3-49.3			<i>E(var)301</i>
3-[47.4]	83E1-83E2		<i>Rm62[†]</i>	3-[48]	84E8-84F1	84e800	<i>l(3)84Eh</i>	3-49.3			<i>l(3)ds6</i>
3-47.5			<i>ale</i>	3-[48]	84E8-84F1	84e900	<i>l(3)84Ei</i>	3-49.3			<i>l(3)SG36</i>
3-47.5	84A		<i>Ama</i>	3-[48]	84F1-85A3	84f100	<i>l(3)84Fa</i>	3-49.3			<i>l(3)SG37</i>
3-47.5			<i>Kg</i>	3-[48]	84F11-84F12	84f200	<i>l(3)84Fb</i>	3-49.4			<i>ants</i>
3-47.5			<i>kkv</i>	3-[48]	84F11-84F12	84f200	<i>l(3)84Fc</i>	3-49.5			<i>l(3)SG38</i>
3-47.5			<i>wp</i>	3-[48]	84F11-84F12	84f400	<i>l(3)84Fd</i>	3-49.5			<i>Rst(3)ns</i>
3-47.5	84A1-84A6	84b200	<i>pb</i>	3-[48]	84F1-84F7	84f500	<i>l(3)84Ff</i>	3-49.6			<i>dlhA[†]</i>
3-47.5	84B1-84B2	84b600	<i>Dfd</i>	3-[48]	84F1-84F7	84f500	<i>l(3)84Fg</i>	3-49.8	85E		<i>l(3)85Ec</i>
3-47.5	84B1-84B2	84b700	<i>Scr</i>	3-[48]	84F4-84F13	84f800	<i>l(3)84Fh</i>	3-49.8			<i>rar3</i>
3-47.5	84B1-84B2	84b800	<i>ftz</i>	3-[48]	84F4-84F13	84f800	<i>l(3)84Fi</i>	3-50			<i>dn</i>
3-47.5	84B1-84B2	84b900	<i>Antp</i>	3-[48]	84F4-84F13	84f800	<i>l(3)84Fj</i>	3-50			<i>dw</i>
3-[47.5]	84A1		<i>Edg84A</i>	3-[48]	84F13-85A3	84f1100	<i>l(3)84Fk</i>	3-50			<i>mu</i>
3-[47.5]	84B1-84B2		<i>Hu</i>	3-[48]	84F13-85A3	84f1100	<i>l(3)84Fl</i>	3-50			<i>snp</i>
3-[47.5]	84B		<i>ma</i>	3-[48]	84F13-85A3	84f1100	<i>l(3)84Fm</i>	3-50			<i>Su(z)4[†]</i>
3-[47.5]	84B1-84B2		<i>Scx</i>	3-[48]	84E-84F	84ff(100)	<i>tRNA:arg2:84Fa</i>	3-[50]	86C1-86C2		<i>Mpl</i>
3-[47.5]	84A1-84B2		<i>Ta</i>	3-[48]	84E-84F	84ff(100)	<i>tRNA:arg2:84Fb</i>	3-[50]	86B		<i>Su(var)3-5</i>
3-[47.5]	84C		<i>tRNA:gly:84C</i>	3-[48]	84E-84F	84ff(100)	<i>tRNA:arg2:84Fc</i>	3-[50]	86C1-86D8		<i>Su(var)3-14</i>
3-[47.5]	83A-83B		<i>tRNA:lys5:83AB</i>	3-[48]	84E-84F	84ff(100)	<i>tRNA:asn5:84Fa</i>	3-50.0	86D1-86D4		<i>cu</i>
3-[47.5]	84A-84B		<i>tRNA:lys5:84AB</i>	3-[48]	84E-84F	84ff(100)	<i>tRNA:asn5:84Fb</i>	3-50.0			<i>Er</i>
3-[47.5]	83F-84A		<i>tRNA:met2:83F</i>	3-[48]	85A	85a100	<i>l(3)85Aa</i>	3-50.0	86D1-86D4		<i>M(3)86D</i>
3-[47.5]	84C		<i>U2snRNA84Ca[†]</i>	3-[48]	85A	85a200	<i>l(3)85Ab</i>	3-50.0	86C1-86C8		<i>neu</i>
3-[47.5]	84C		<i>U2snRNA84Cb[†]</i>	3-[48]	85A	85a400	<i>l(3)85Ac</i>	3-50.3			<i>l(3)ds13</i>
3-[47.5]	84C8		<i>Vin[†]</i>	3-[48]	85A	85a500	<i>l(3)85Ad</i>	3-50.5			<i>l(3)ds16</i>
3-[47.5]	84A1-84A2	84b100	<i>lab</i>	3-[48]	85A	85a500	<i>l(3)85Ae</i>	3-50.5			<i>man</i>
3-[47.5]	84A4-84A1	84b300	<i>zen</i>	3-[48]	85A	85a500	<i>l(3)85Af</i>	3-50.5			<i>mucl</i>
3-[47.5]	84A4-85A1	84b400	<i>zen2</i>	3-[48]	85A	85a800	<i>l(3)85Ag</i>	3-50.6			<i>l(3)631215[†]</i>
3-[47.5]	84A1	84b500	<i>bcd</i>	3-48.0	79E		<i>exb</i>	3-50.8			<i>l(3)ds9</i>
3-[47.5]	84B3	84b1000	<i>l(3)84Bb</i>	3-48.0	84B1-84B2		<i>Msc</i>	3-50.8			<i>l(3)SG41</i>
3-[47.5]	84B2-84B6	84b1100	<i>l(3)84Bc</i>	3-48.0	85A1-85A3	85a300	<i>Rhod</i>	3-50.9			<i>l(3)ds4</i>
3-[47.5]	84B3-84C2	84b1200	<i>mab</i>	3-48.0	85A6	85a500	<i>p</i>	3-50.9			<i>l(3)SG42</i>
3-[47.5]	84B3-84C2	84b1400	<i>stk</i>	3-48.1			<i>bid</i>	3-51			<i>l(3)S1</i>
3-[47.5]	84C1-84C6	84c100	<i>hat</i>	3-48.1			<i>cvl-5</i>	3-51			<i>meil</i>
3-[47.5]	84C1-84C6	84c200	<i>l(3)84Cb</i>	3-48.1	84E1-84E2	84e50	<i>dsx</i>	3-51			<i>PL(3)sp-W2</i>
3-[47.5]	84C1-84C6	84c300	<i>l(3)84Cc</i>	3-48.3			<i>bod</i>	3-51	86F6-87A7		<i>sad</i>
3-[47.5]	84C1-84C6	84c400	<i>rue</i>	3-48.3			<i>moo</i>	3-51			<i>skd</i>
3-[47.5]	84C5-84C6	84c500	<i>sas</i>	3-48.3	85A3-85B1	85a900	<i>hb</i>	3-[51]	87A-87B		<i>Ag72H5[†]</i>
3-[47.5]	84C5-84C8	84c600	<i>l(3)84Ce</i>	3-48.4			<i>fs(3)HO5B</i>	3-[51]	87B6-87B9		<i>DipC</i>
3-[47.5]	84C6-84C8	84c700	<i>ted</i>	3-48.4			<i>hau</i>	3-[51]	87B8-87B9		<i>Gst</i>
3-47.6			<i>dash</i>	3-48.4	85B	85b100	<i>osk</i>	3-[51]	87A9-87B2		<i>l(3)87Ad</i>

CYTOGENETIC MAP

genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol
3-[51]	87C7		<i>l(3)87Cc</i>	3-[53]	87E11-87F1	87e1200	<i>l(3)87Ek</i>	3-[58]	89C		<i>l(3)89Cd</i>
3-[51]	87A9-87B5		<i>Mab77H5</i> [†]	3-53.2			<i>l(3)SG47</i>	3-58.1	89B1-89B4		<i>mor</i>
3-[51]	87B		<i>Mfcp</i>	3-53.3			<i>Su(var)325</i>	3-58.2	89B9-89B10		<i>Sb</i>
3-[51]	87B		<i>Msp</i>	3-53.4			<i>l(3)SG48</i>	3-58.3			<i>Two-b</i>
3-[51]	86E2-87B5		<i>mus309</i>	3-53.5	87E5-87F12		<i>ninaB</i>	3-58.5	89C1-89C2		<i>ss</i>
3-[51]	86E1-86E2		<i>pros</i>	3-53.5			<i>Su(var)3-8</i>	3-58.7	88F9-89B5		<i>dlhC</i> [†]
3-[51]	87C		<i>su(fu)</i>	3-53.6	87F13-88C2		<i>DipB</i>	3-58.8	89E		<i>l(3)89Ea</i>
3-[51]	86F4-86F7		<i>Su(var)3-13</i>	3-53.6			<i>l(3)ds8</i>	3-58.8	89E		<i>l(3)89Ee</i>
3-[51]	87B-87C		<i>tRNA:lys5:87BC</i>	3-53.6			<i>l(3)SG49</i>	3-58.8	89E1-89E2	89e100	<i>Ubx</i>
3-[51]	87B-87C		<i>tRNA:thr3:87BC</i>	3-53.6	88B1-88B2		<i>red</i>	3-58.8	89E1-89E2	89e200	<i>abd-A</i>
3-[51]	86F1-86F7	86f100	<i>l(3)86Fa</i>	3-53.8			<i>l(3)SG50</i>	3-58.8	89E1-89E2	89e300	<i>Abd-B</i>
3-[51]	86F1-86F7	86f100	<i>l(3)86Fb</i>	3-54	88B1		<i>Rp11140</i>	3-[59]	89E4-89E8		<i>l(3)89Ef</i>
3-[51]	86F4-86F7	86f300	<i>l(3)86Fc</i>	3-[54]	88A1-88A2		<i>ems</i>	3-[59]	89E4-89E8		<i>l(3)89Eg</i>
3-[51]	86F4-86F7	86f300	<i>l(3)86Fd</i>	3-[54]	87F1-87F13		<i>E(var)87F</i>	3-[59]	89E4-89E8		<i>l(3)89Eh</i>
3-[51]	86F6-87A2	86f500	<i>l(3)86Fe</i>	3-[54]	87F		<i>Hrb2</i>	3-[59]	89E4-89E8		<i>l(3)89Ei</i>
3-[51]	86F6-87A2	86f500	<i>l(3)86Ff</i>	3-[54]	87F		<i>Mst87F</i>	3-59			<i>mfs(3)G</i>
3-[51]	86F6-87A2	86f500	<i>l(3)86Fg</i>	3-[54]	88C3-88E2		<i>put</i>	3-59			<i>mus307</i>
3-[51]	87A1-87A7	87a100	<i>l(3)87Aa</i>	3-[54]	88A10-88C3		<i>spnB</i>	3-59			<i>Rf</i>
3-[51]	87A1-87A7	87a100	<i>l(3)87Ab</i>	3-[54]	87F		<i>sqd</i> [†]	3-59			<i>Su(sc)</i>
3-[51]	87A6-87A7	87a300	<i>Hsp70A</i>	3-[54]	88A-88B		<i>tRNA:ser2b:88A</i>	3-[59]	89D9-89E2		<i>l(3)89Da</i>
3-[51]	87A9-87B2	87a500	<i>l(3)87Ae</i>	3-[54]	88C		<i>CabP23</i>	3-[59]	89D9-89E2		<i>l(3)89Db</i>
3-[51]	87B1-87B4	87b100	<i>l(3)87Ba</i>	3-54.0	87F2-88B1		<i>atn</i>	3-[59]	89D9-89E2		<i>l(3)89Dc</i>
3-[51]	87B2-87B4	87b200	<i>l(3)87Bb</i>	3-54.2	l(3)nc99Eb			3-[59]	89E1-89E2		<i>FasI</i>
3-[51]	87B2-87B4	87b200	<i>l(3)87Bc</i>	3-54.2	88B1-88B5		<i>trx</i>	3-[59]	89F-90A		<i>Pros35</i>
3-[51]	87B4-87B6	87b400	<i>svp</i>	3-54.4	88C2-88D6		<i>wrl</i>	3-[59]	89D	89d100	<i>l(3)lIB</i> [†]
3-[51]	87B5-87B9	87b500	<i>l(3)87Be</i>	3-54.7	88C		<i>cv-c</i>	3-[59]	89E	89e400	<i>l(3)lIB</i> [†]
3-[51]	87B5-87B9	87b500	<i>l(3)87Bf</i>	3-54.7			<i>Su(var)330</i>	3-59.0	89E7-89E11		<i>Mc</i>
3-[51]	87B5-87B9	87b500	<i>Pp1-87B</i>	3-54.8			<i>mat(3)1</i> [†]	3-59.2	89E7-89F1		<i>l(3)89Ej</i>
3-[51]	87B9-87B13	87b800	<i>l(3)87Bh</i>	3-54.8	88A12-88B2		<i>su(Hw)</i>	3-59.3			<i>l(3)SG53</i>
3-[51]	87B9-87B13	87b800	<i>l(3)87Bi</i>	3-55	88D10-88E1		<i>E(var)88D</i> [†]	3-59.3			<i>l(3)SG54</i>
3-[51]	87B9-87B13	87b800	<i>l(3)87Bj</i>	3-55			<i>fch</i>	3-59.5			<i>cal</i>
3-[51]	87B11-87C1	87b1100	<i>l(3)87Bk</i>	3-55			<i>ifm(3)5</i>	3-59.5			<i>wtl</i>
3-[51]	87B11-87C1	87b1100	<i>l(3)87Bl</i>	3-55			<i>ifm(3)6</i>	3-59.7+			<i>fl</i>
3-[51]	87B11-87C1	87b1100	<i>l(3)87Bm</i>	3-55	87D1-87D2		<i>mus308</i>	3-59.9			<i>Su(var)320</i>
3-[51]	87C1-87C2	87c100	<i>Hsp70B</i>	3-[55]	88B		<i>fs(3)293</i>	3-[60]	90B1-90B2		<i>Eh</i> [†]
3-[51]	87C6	87c300	<i>l(3)87Cb</i>	3-[55]	88F4-88F5	88f100	<i>Tm1</i>	3-[60]	90C1-91A2		<i>l(3)F31</i>
3-[51]	87C9	87c600	<i>l(3)87Cd</i>	3-[55]	88F2-88F3	88f200	<i>Tm2</i>	3-[60]	90B3-90B8		<i>Sgs5</i>
3-51.0			<i>l(3)SG43</i>	3-55.0			<i>l(3)SG51</i>	3-[60]	90C		<i>tRNA:val4:90Ca</i>
3-51.1	88A12-88B1		<i>l(3)88Ab</i>	3-55.4			<i>r(aGPDH)</i>	3-[60]	90C		<i>tRNA:val4:90Cb</i>
3-51.1			<i>l(3)ds15</i>	3-55.5			<i>l(3)DTS8</i>	3-[60]	90B-90C	90c(100)	<i>tRNA:val3b:90CB</i>
3-51.3	86E3-86E10		<i>mgr</i>	3-55.5			<i>Su(var)327</i>	3-[60]	90B-90C	90c(200)	<i>tRNA:pro:90Ca</i>
3-51.3			<i>tr</i>	3-55.6			<i>Su(var)311</i> [†]	3-[60]	90C	90c(250)	<i>tRNA:?:90Ca</i>
3-51.5	87B13-88D14		<i>l(3)S2</i>	3-56			<i>mus306</i>	3-[60]	90B-90C	90c(300)	<i>tRNA:pro:90Cb</i>
3-51.7	87C9-87D2		<i>Men</i>	3-56			<i>sh</i>	3-[60]	90C	90c(400)	<i>tRNA:ala:90C</i>
3-51.7	87C4-87C5	87c200	<i>l(3)87Ca</i>	3-56.4	88D10-88E1		<i>Su(var)3-9</i>	3-[60]	90C	90c(450)	<i>tRNA:?:90Cb</i>
3-51.7	87C8	87c500	<i>kar</i>	3-56.7			<i>jvl</i>	3-[60]	90B-90C	90c(500)	<i>tRNA:thr:90Ca</i>
3-51.7	87D3-87D6	87d100	<i>l(3)87Da</i>	3-56.7			<i>m(Est6)</i>	3-[60]	90B-90C	90c(600)	<i>tRNA:thr:90Cb</i>
3-51.8			<i>l(3)SG45</i>	3-57	88F1-88F2		<i>ea</i>	3-[60]	90C	90c(700)	<i>tRNA:?:90Cc</i>
3-51.9	87D4-87D9	87d500	<i>mesA</i>	3-57			<i>mul</i>	3-[60]	90C	90c(800)	<i>tRNA:?:90Cd</i>
3-51.9	87D8-87D12	87d600	<i>mesB</i>	3-[57]	88E4-88E8		<i>cen185</i> [†]	3-[60]	90C	90c(900)	<i>tRNA:?:90Ce</i>
3-52			<i>l(3)KX209</i> [†]	3-[57]	88D		<i>dsx-c88D</i> [†]	3-60.0	90B1-90D1		<i>osa</i>
3-52	87E12-87F12		<i>yri</i>	3-[57]	88E1-88E13		<i>Hsc70-4</i>	3-60.7			<i>qf</i>
3-[52]	87E12-87F		<i>E(var)8</i> [†]	3-[57]	88D		<i>Su(var)309</i>	3-60.8			<i>Sgs9</i>
3-[52]	87D3-87D6	87d100	<i>l(3)87Dc</i>	3-57.1	88F	88f300	<i>Ac88F</i>	3-60.8			<i>Su(var)314</i>
3-[52]	87D4-87D9	87d400	<i>l(3)87Dd</i>	3-57.1	89A2-89A3	89a200	<i>Po</i>	3-61	89D-90D		<i>ald</i>
3-[52]	87D7-87D12	87d700	<i>l(3)87De</i>	3-57.2			<i>cmt</i>	3-61			<i>Eth</i>
3-[52]	87D6-87D13	87d750	<i>pmst</i> [†]	3-57.2	89A1-89A2	89a100	<i>Aldox1</i>	3-61			<i>Su(ss)</i>
3-[52]	87D6-87D13	87d800	<i>l(3)87Df</i>	3-57.4	89A3-89A5	89a300	<i>c(3)G</i>	3-61.1			<i>Su(var)306</i>
3-[52]	87D	87d1100	<i>Hsc70-2</i>	3-57.5			<i>Cma</i>	3-61.7	89B9-89B13		<i>Su(var)3-10</i>
3-52.0	87D6-87D13	87d900	<i>ry</i>	3-58			<i>eyh</i>	3-61.8	90C2-90F		<i>Dnase1</i>
3-52.1	87E		<i>l(3)S4</i>	3-58			<i>l(3)SG52</i>	3-62			<i>spnE</i>
3-52.1			<i>l(3)SG46</i>	3-58	89B1-89B4	89b100	<i>srp</i>	3-[62]	90D		<i>cpo</i> [†]
3-52.1	87D3-87D6	87d100	<i>l(3)87Db</i>	3-58	89B4-89B10	89b200	<i>pnr</i>	3-[62]	90D-90E		<i>tRNA:gly:90DE</i>
3-52.1	87D10-87D12	87d1000	<i>snk</i>	3-58			<i>psi3</i>	3-62.0	90E-90F		<i>sr</i>
3-52.1	87D11-87D14	87d1200	<i>pic</i>	3-[58]	89A-89B		<i>Ann</i> [†]	3-62.4			<i>l(3)SG55</i>
3-52.2	87E1-87E2	87e100	<i>sim</i>	3-[58]	89A3-89A5	89a300	<i>l(3)89Aa</i>	3-62.4			<i>Su(var)302</i>
3-52.5			<i>dlhB</i> [†]	3-[58]	89A3-89A5	89a300	<i>rec1</i>	3-62.6	90C2-91A3		<i>Mdh2</i>
3-52.5	87E2-87E3	87e400	<i>Ace</i>	3-[58]	89B-89C	89bc(100)	<i>tRNA:val4:89BC</i>	3-[63]	90F		<i>E103</i> [†]
3-52.7			<i>Dly</i>	3-[58]	89B-89C	89bc(200)	<i>tRNA:phe2:89BC</i>	3-63.1	91A1-91A2		<i>gl</i>
3-52.7			<i>ptd152.7</i>	3-[58]	89B5-89B8		<i>l(3)89Ba</i>	3-64			<i>E(lz)</i>
3-53			<i>l(3)S7</i>	3-[58]	89B7-89C2		<i>l(3)89Bb</i>	3-64			<i>gt3</i>
3-53	87F12-87F15	urd		3-[58]	89B7-89C2		<i>l(3)89Bc</i>	3-64			<i>k</i>
3-53	87A7-87A9	87a400	<i>aur</i>	3-[58]	89B7-89C2		<i>l(3)89Bd</i>	3-[64]	91B		<i>fru</i>
3-[53]	87F12-88B1		<i>l(3)88Aa</i>	3-[58]	89B7-89C2		<i>l(3)89Be</i>	3-[64]	91C		<i>pk?2</i>
3-[53]	87E4-87E5		<i>Su(var)3-7</i>	3-[58]	89B7-89C2		<i>l(3)89Bf</i>	3-64.3			<i>Go</i>
3-[53]	87E1-87E2	87e200	<i>l(3)87Eb</i>	3-[58]	89B7-89C2		<i>l(3)89Bg</i>	3-[65]	91C2-91D1	91d100	<i>l(3)91Ca</i>
3-[53]	87E1-87E2	87e300	<i>l(3)87Ec</i>	3-[58]	89B7-89C2		<i>l(3)89Bh</i>	3-[65]	91C2-91D1	91d200	<i>l(3)91Cb</i>
3-[53]	87E2-87E6	87e500	<i>l(3)87Ee</i>	3-[58]	89B7-89C2		<i>l(3)89Bi</i>	3-[65]	91C7-91D1	91d300	<i>l(3)91Cd</i>
3-[53]	87E3-87E6	87e600	<i>l(3)87Ef</i>	3-[58]	89B7-89C2		<i>l(3)89Bj</i>	3-64.6	91C	91d300	<i>Cha</i>
3-[53]	87E5-87E12	87e700	<i>l(3)87Eg</i>	3-[58]	89B7-89C2		<i>l(3)89Bk</i>	3-65			<i>cv-b</i>
3-[53]	87E5-87E12	87e700	<i>l(3)87Eh</i>	3-[58]	89B7-89C2		<i>l(3)89Bl</i>	3-65			<i>cv-d</i>
3-[53]	87E5-87E12	87e700	<i>l(3)87Ei</i>	3-[58]	89C		<i>l(3)89Ca</i>	3-65			<i>Pec</i>
3-[53]	87E5-87E12	87e1000	<i>l(3)87Ej</i>	3-[58]	89C		<i>l(3)89Cb</i>	3-65			<i>sprd</i>
3-[53]	87E9-87E12	87e1100	<i>Act87E</i>	3-[58]	89C		<i>l(3)89Cc</i>	3-[65]	91B-93F		<i>Kfl</i>

THE GENOME OF *DROSOPHILA MELANOGASTER*

genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol	genetic location	cytology	map index	gene symbol
3-[65]	91F6-91F13		<i>l(3)91Fa</i>	3-74.0			<i>l(3)SG60</i>	3-90			<i>act</i>
3-[65]	91F1-91F13		<i>l(3)91Fb</i>	3-74.3			<i>l(3)dsl9</i>	3-90			<i>ref(3)D</i>
3-[65]	91F1-91F13		<i>l(3)91Fc</i>	3-75.5			<i>dg-a</i>	3-90			<i>Su(y^{3P})</i>
3-[65]	91F6-92A2		<i>l(3)91Fd</i>	3-75.7	94A-94E		<i>cd</i>	3-[90]	96F8-96F13		<i>l(3)96Fa</i>
3-[65]	91F1-91F13		<i>l(3)91Fe</i>	3-[76]	94B1-94B2		<i>T-cp1</i>	3-[90]	96F8-96F13		<i>l(3)96Fb</i>
3-[65]	91F6-92A2		<i>l(3)91Ff</i>	3-76.1			<i>l(3)SG61</i>	3-[90]	96F8-96F13		<i>l(3)96Fc</i>
3-[65]	91D1-91D2		<i>Rh2</i>	3-76.2	94A-94E		<i>wo</i>	3-90.0	96F11	96F100	<i>Pr</i>
3-[65]	91F11-92A3	92a100	<i>l(3)92Aa</i>	3-76.4	94D-95A3		<i>Su(var)3-11</i>	3-90.2			<i>l(3)PR</i>
3-[65]	91F11-92A3	92a300	<i>l(3)92Ac</i> †	3-76.5			<i>l(3)SG62</i>	3-90.2	96C-97A		<i>M(3)96CF</i>
3-[65]	91F11-92A3	92a400	<i>l(3)92Ad</i> †	3-76.6			<i>l(3)SG63</i>	3-90.6			<i>Tb</i>
3-65.3	91B-91F		<i>Srd</i> †	3-76.7			<i>l(3)F24</i>	3-91			<i>spnD</i>
3-[66]	92B1-92B9		<i>tRNA:val3b:92Ba</i>	3-77.5			<i>obt</i>	3-91	97D1-97D2		<i>Tl</i>
3-[66]	92B1-92B9		<i>tRNA:val3b:92Bb</i>	3-78			<i>Gd</i>	3-91	97D9-97D15		<i>tne</i>
3-[66]	92B1-92B9		<i>tRNA:val3b:92Bc</i>	3-78			<i>l(3)DTS1</i>	3-91	97B		<i>tx</i>
3-[66]	92B1-92B9		<i>tRNA:val3b:92Bd</i>	3-78.2			<i>l(3)SG64</i>	3-[91]	97D		<i>anon-97D</i> †
3-66.0			<i>Cu-3</i>	3-78.6			<i>mad</i>	3-[91]	97D		<i>Elg</i> †
3-66.0			<i>Tcp</i>	3-[79]	94E	94e200	<i>unk</i> †	3-[91]	97C-97D		<i>His2AvD</i> †
3-66.2	91F12-91F13		<i>nos</i>	3-79.0	94E	94e500	<i>pnt</i>	3-91.1			<i>ju</i>
3-66.2	92A2	92a500	<i>DI</i>	3-79.3			<i>l(3)Sp2</i>	3-91.1	97D3-97D5		<i>ro</i>
3-66.4	92B6-92B7		<i>Bpt</i> †	3-79.7	95A1-95A3		<i>M(3)95A</i>	3-91.3	97D3-97D5		<i>ms(3)K81</i>
3-66.4	92A4-92B1		<i>ninaE</i>	3-79.7			<i>Mrt</i>	3-91.5	97A-97B		<i>Ald</i>
3-66.4	92A4-92B1		<i>ort</i>	3-79.8			<i>l(3)SG65</i>	3-91.8			<i>l(3)XaR</i>
3-[67]	92D			3-80		94e400	<i>l(3)SG83</i> †	3-91.9	97F1-97F8		<i>Bd</i>
3-67.3			<i>gn</i> †	3-80.7			<i>Fs(3)Lev</i>	3-92			<i>Sc</i>
3-67.8	92A11-92B3		<i>qrt</i> †	3-81			<i>bh</i>	3-92	97D13-97E4		<i>spz</i>
3-67.9			<i>eym</i>	3-[81]	95C		<i>Bsg95C</i> †	3-[92]	97F11		<i>l(3)mbt</i> †
3-68			<i>ncn</i>	3-[81]	95A		<i>Hmg</i>	3-[92]	97F-100F		<i>Rnase1</i> †
3-68.0			<i>l(3)ds1</i>	3-[81]			<i>Esm</i>	3-[92]	97E-97F		<i>Tub97EF</i>
3-[68]	92F		<i>cdc2c</i> †	3-[81]	95D		<i>Hsp68</i>	3-93			<i>cmp</i>
3-[68]	92E		<i>MtnB</i> †	3-[81]	95E		<i>Mst95E</i>	3-93.2			<i>sh5</i>
3-68.2			<i>cmd</i>	3-[81]	95E-95F		<i>Mst95EF</i>	3-94			<i>Ble</i>
3-68.5			<i>com-d</i>	3-[81]	95A		<i>nau</i> †	3-94.1			<i>Pw</i>
3-68.7			<i>l(3)SG57</i>	3-[81]	95C		<i>U3snRNA95C</i>	3-95			<i>bf</i>
3-69.5	92D1-92F2		<i>H</i>	3-[81]	95C	95c(100)	<i>U1snRNA95Ca</i> †	3-95	98D2-98D3		<i>fkh</i>
3-69.8			<i>ref(3)O</i>	3-[81]	95C	95c(200)	<i>U1snRNA95Cb</i> †	3-95			<i>spg</i>
3-[70]	93B		<i>AnnIX</i> †	3-[81]	95C	95c(300)	<i>U1snRNA95Cc</i> †	3-[95]	98C	98c100	<i>Mst98Ca</i>
3-[70]	93C3-93D7		<i>gustM1</i>	3-81.2	94D10-94E5	94e100	<i>hh</i>	3-[95]	98C	98c200	<i>Mst98Cb</i>
3-[70]	93B		<i>Na-p</i> †	3-81.2	94E	94e300	<i>cnc</i> †	3-95.4			<i>rsd</i>
3-[70]	93A1-93A2		<i>tRNA:thr4:93A</i>	3-81.4			<i>l(3)ds3</i>	3-95.5			<i>su(pr)</i>
3-[70]	93B1-93B6	93b10	<i>l(3)93Bc</i> †	3-81.4			<i>l(3)SG66</i>	3-96			<i>spnA</i>
3-[70]	93B1-93B6	93b10	<i>l(3)93Bd</i> †	3-81.6			<i>l(3)a</i>	3-96.7			<i>ref(3)V</i>
3-[70]	93B6-93C2	93b30	<i>l(3)93Be</i> †	3-81.7	95D1-95D4		<i>Gdh</i>	3-97.3			<i>ra</i>
3-[70]	93C1-93C2	93b40	<i>l(3)93Bf</i> †	3-81.7			<i>l(3)ds15</i>	3-98			<i>su(vg)</i>
3-[70]	93C1-93C2	93b40	<i>l(3)93Bg</i> †	3-81.7			<i>l(3)SG67</i>	3-[98]	98D-98E		<i>Hrb1</i>
3-[70]	93C1-93C2	93b40	<i>l(3)93Bh</i> †	3-82	95F7-95F15		<i>crb</i>	3-[98]	98A		<i>Klp98A</i> †
3-[70]	93C1-93C2	93b40	<i>l(3)93Bi</i> †	3-82.7			<i>l(3)ds12</i>	3-[98]	98A7		<i>Mlc1</i>
3-[70]	93B11-93C6	93b100	<i>l(3)93Ba</i>	3-82.7			<i>l(3)SG68</i>	3-[98]	98A-98B		<i>RpL1</i>
3-[70]	93B11-93C6	93b100	<i>l(3)93Bb</i>	3-82.9	95F-96A		<i>ms(3)nc32</i>	3-98.3			<i>LapA</i>
3-[70]	93C3-93C6	93c100	<i>l(3)93Ca</i>	3-[83]	96A		<i>Acr96A</i>	3-98.3			<i>LapD</i>
3-[70]	93C3-93C6	93c200	<i>l(3)93Cb</i>	3-[83]	96A		<i>AcrE</i> †	3-99	98F1		<i>Doa</i>
3-[70]	93C3-93D4	93c300	<i>l(3)93Cd</i>	3-[83]	95F		<i>anon-95F</i> †	3-[99]	98F-99A		<i>Cg98F99A</i>
3-[70]	93D2-93D6	93d100	<i>l(3)93Da</i>	3-[83]	95E6-96A5		<i>Tch</i>	3-[99]	98F-100F		<i>Glu</i>
3-[70]	93D4-93D9	93d200	<i>l(3)93Db</i>	3-[83]	96A		<i>AcrF</i> †	3-[99]	98E6-98F1		<i>Pkc3</i>
3-[70]	93D4-93D9	93d200	<i>l(3)93Dc</i>	3-[84]	96A2-96A5		<i>Pp1a-96A</i> †	3-[99]	98E-98F		<i>Sryc</i>
3-[70]	93D4-93D9	93d200	<i>l(3)93Dd</i>	3-[84]	96A	96a(50)	<i>tRNA:asp:96A</i>	3-[99]	98F3-98F10		<i>yem</i>
3-[70]	93D7-93D10	93d600	<i>Hsr93</i>	3-[84]	96A	96a(100)	<i>U6snRNA96Aa</i>	3-99.2			<i>Dr</i>
3-[70]	93D7-93D10	93d600	<i>l(3)93Df</i>	3-[84]	96A	96a(200)	<i>U6snRNA96Ab</i>	3-99.8			<i>dlhD</i> †
3-[70]	93D7-93D10	93d600	<i>l(3)93Dg</i>	3-[84]	96A	96a(300)	<i>U6snRNA96Ac</i>	3-100			<i>Ama2</i>
3-[70]	93D7-93D10	93d600	<i>l(3)93Dh</i>	3-84.5			<i>l(3)DTS3</i>	3-100			<i>spnF</i>
3-[70]	93D7-93D10	93d900	<i>l(3)93Di</i>	3-84.5			<i>l(3)SG69</i>	3-100	99A-99C		<i>sro</i>
3-[70]	93D7-93D8	93d1000	<i>l(3)93Dj</i>	3-84.5	96C1-96C5		<i>M(3)96C</i>	3-100			<i>wdn</i>
3-70.4			<i>l(3)ds17</i>	3-85	96B-96D		<i>tld</i>	3-[100]	99B9-99B10		<i>Fra</i> †
3-70.4			<i>l(3)SG58</i>	3-[85]	96A1-96A7		<i>aor</i>	3-[100]	99B		<i>Jon99B</i>
3-70.4	93B6-93B7	93b20	<i>r-l</i>	3-[85]	96A1-96A10		<i>ash2</i>	3-[100]	99A-100A		<i>kay</i>
3-70.5	93C3-93D4	93c300	<i>l(3)93Cc</i>	3-[85]	96C		<i>bam</i>	3-[100]	99B5-99B9		<i>M(3)99B</i>
3-70.7	93E		<i>mod(mdg4)</i> †	3-[85]	96B1-96B10		<i>mar</i>	3-[100]	99B		<i>msh1</i> †
3-70.7	93D2	93c300	<i>e</i>	3-85.0			<i>slo</i>	3-[100]	99A10-99B2		<i>Ocr</i>
3-71			<i>l(3)SG59</i>	3-85.2	96A20-96B10		<i>asp</i>	3-[100]	99A		<i>Pt99A</i> †
3-71			<i>segB</i> †	3-85.4			<i>l(3)L6B</i> †	3-[100]	99A		<i>stg</i>
3-72			<i>bn</i>	3-87.2			<i>Ndw</i>	3-[100]	99C5-99C6		<i>trp</i>
3-72			<i>Mar</i>	3-88			<i>mah</i>	3-100.6			<i>Bsb</i>
3-[72]	93E		<i>Inr</i>	3-[89]	96F8-96F11	96f200	<i>boss</i>	3-100.7	99B11-99C1		<i>ca</i>
3-[72]	93E3-93E5	93e100	<i>msh2</i> †	3-89.1	96F8-96F9	96f300	<i>md</i> †	3-100.7	99B11-99C1		<i>ncd</i>
3-[72]	93E3-93E5	93e200	<i>NK3</i>	3-89.1	96F8-96F9	96f400	<i>mg</i> †	3-100.9			<i>l(3)Sp19</i>
3-[72]	93E3-93E5		<i>NK4</i>	3-89.1	96F8-96F9	96f500	<i>mb</i> †	3-[101]	99F		<i>Jon99F</i>
3-[72]	93E3-93E4	93e300	<i>NK1</i>	3-89.1	96F8-96F9	96f600	<i>ma</i> †	3-[101]	99E4-99F1		<i>M(3)99E</i>
3-72.5			<i>det</i>	3-89.1	96F8-96F9	96f700	<i>m1</i> †	3-[101]	99D1		<i>Ser1</i> †
3-[73]	93E1-93E2		<i>prd9</i> †	3-89.1	96F8-96F9	96f800	<i>m2</i> †	3-[101]	99D1		<i>Ser2</i> †
3-[73]	94A6-94A8		<i>tRNA:ser2b:94A</i>	3-89.1	96F8-96F9	96f900	<i>m3</i> †	3-[101]	99D1		<i>Ser3</i> †
3-[73]	93F6-93F16		<i>tsl</i>	3-89.1	96F8-96F9	96f1000	<i>m4</i> †	3-[101]	99D3-99D9	99d100	<i>l(3)99Da</i> †
3-[73]	93E6-93F8	93f100	<i>l(3)93Fa</i>	3-89.1	96F8-96F9	96f1100	<i>m5</i> †	3-[101]	99D3-99D9	99d100	<i>l(3)99Db</i> †
3-[73]	93F6-93F8	93f200	<i>l(3)93Fb</i>	3-89.1	96F8-96F9	96f1200	<i>m6</i> †	3-[101]	99D3-99D9	99d100	<i>l(3)99Dc</i> †
3-[73]	93F6-93F8	93f200	<i>l(3)93Fc</i>	3-89.1	96F8-96F9	96f1300	<i>m7</i> †	3-[101]	99D3-99D9	99d100	<i>l(3)99Dd</i>
3-74.0			<i>l(3)ds13</i>	3-89.1	96F8-96F9	96f1400	<i>E(spl)</i>	3-[101]	99D3-99D9	99d100	<i>l(3)99De</i>

CYTOGENETIC MAP

genetic location	cytology	map index	gene symbol
3-[101]	99D3-99D9	99d100	<i>l(3)99Df</i>
3-[101]	99D3-99D9	99d100	<i>l(3)99Dg</i>
3-[101]	99D3-99D9	99d100	<i>l(3)99Dh</i>
3-[101]	99D6-99E1	99d900	<i>l(3)99Di</i>
3-[101]	99D6-99E1	99d900	<i>l(3)99Dj</i>
3-[101]	99D	99dd100	<i>janB</i>
3-[101]	99D	99dd200	<i>janA</i>
3-[101]	99D4-99D8	99dd300	<i>Sry-b</i>
3-[101]	99D4-99D8	99dd400	<i>Sry-a</i>
3-[101]	99D4-99D8	99dd500	<i>Sry-d</i>
3-[101]	99D1-99D9	99dd600	<i>M(3)99D</i>
3-[101]	99D9-99E3	99e100	<i>l(3)99Ea</i>
3-[101]	99E1-99E2	99e100	<i>Mlc2</i>
3-[101]	99E	99ee(100)	<i>Cec</i>
3-[101]	99E	99ee(200)	<i>cecA1</i> [†]
3-[101]	99E	99ee(300)	<i>cec-y11</i> [†]
3-[101]	99E	99ee(400)	<i>cecA2</i> [†]
3-[101]	99E	99ee(500)	<i>cec-y12</i> [†]
3-[101]	99E	99ee(600)	<i>cecB</i> [†]
3-[101]	99E	99ee(700)	<i>cecC</i> [†]
3-101.1	99C5-99C7		<i>Acph1</i>
3-101.1			<i>l(3)Sp9</i>
3-101.3	99D-99E		<i>Tpi</i>
3-102	99C-99F		<i>ld</i>
3-102	100A5-100B2		<i>til</i>
3-[102]	100B7-100C1		<i>bnk</i> [†]
3-[102]	100A		<i>Pka-C2</i>
3-[102]	100B7-100B9		<i>chp</i>
3-[102]	100A5-100A7		<i>Dapa</i> [†]
3-[102]	100C		<i>dsx-c100C</i> [†]
3-[102]	100E		<i>Efla100E</i>
3-[102]	100C5-100F		<i>E(var)100C-F</i>
3-[102]	100B1-100B5		<i>GNDP</i> [†]
3-[102]	100A5-100A7		<i>l(3)dco</i> [†]
3-[102]	100F		<i>mod</i> [†]
3-[102]	100D-100E		<i>Pen100DE</i> [†]
3-[102]	100E		<i>rvn</i> [†]
3-[102]	100F3-100F5		<i>Su(var)3-12</i>
3-[102]	100D3-100D4		<i>Ttk</i>
3-102.7	100B7-100B8+		<i>bv</i>
3-104.7			<i>Snb</i>
3-105	100C-100F		<i>M(3)100CF</i>
3-[105]	100C-100D		<i>awd</i>
3-[105]	100E-100F		<i>Map205</i>
3-[105]	100F5		<i>Telo3R</i>
3-105.1	100C1-100C6		<i>rod</i>
3-106			<i>l(3)SG70</i>
3-108.6			<i>fs(3)T</i>
3-110.9			<i>l(3)SG71</i>
4-0	102A3		<i>ci-D</i>
4-0	101F2-102A5		<i>M(4)101</i>
4-[0]	101E-102B16		<i>ar</i>
4-[0]	101E-102B16		<i>Scn</i>
4-0.0	101F2-102A5		<i>ci</i>
4-0.2			<i>gvl</i>
4-[1]	102B10-102E9		<i>gy</i>
4-[1]	102E-102F		<i>pho</i> [†]
4-[1]	102D-102F		<i>spa</i>
4-1.4	102B10-102E9		<i>bt</i>
4-2.0			<i>ey</i>
4-3.0	102E2-102F10		<i>sv</i>
Y	Y(h2-h3)	103a100	<i>kl5</i>
Y	Y(h7-h9)	103a200	<i>kl3</i>
Y	Y(h10)	103a300	<i>kl2</i>
Y	Y(h11-h13)	103a350	<i>Su(Ste)</i>
Y	Y(h14)	103a400	<i>kl1</i>
Y	Y(h21-h23)	104a100	<i>ks1</i>
Y	Y(h24-h25)	104a200	<i>ks2</i>