RESIDUE REVIEWS

VOLUME 13

RESIDUE REVIEWS

Residues of Pesticides and other
Foreign Chemicals in Foods and Feeds

Edited by

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Preface

That residues of pesticide and other "foreign" chemicals in foodstuffs are of concern to everyone everywhere is amply attested by the reception accorded previous volumes of "Residue Reviews" and by the gratifying enthusiasm, sincerity, and efforts shown by all the individuals from whom manuscripts have been solicited. Despite much propaganda to the contrary, there can never be any serious question that pest-control chemicals and food-additive chemicals are essential to adequate food production, manufacture, marketing, and storage, yet without continuing surveillance and intelligent control some of those that persist in our foodstuffs could at times conceivably endanger the public health. Ensuring safety-in-use of these many chemicals is a dynamic challenge, for established ones are continually being displaced by newly developed ones more acceptable to food technologists, pharmacologists, toxicologists, and changing pest-control requirements in progressive food-producing economies.

These matters are also of genuine concern to increasing numbers of governmental agencies and legislative bodies around the world, for some of these chemicals have resulted in a few mishaps from improper use. Adequate safety-in-use evaluations of any of these chemicals persisting into our foodstuffs are not simple matters, and they incorporate the considered judgments of many individuals highly trained in a variety of complex biological, chemical, food technological, medical, pharmacological, and toxicological disciplines.

It is hoped that "Residue Reviews" will continue to serve as an integrating factor both in focusing attention upon those many residue matters requiring further attention and in collating for variously trained readers present knowledge in specific important areas of residue and related endeavors; no other single publication attempts to serve these broad purposes. The contents of this and previous volumes of "Residue Reviews" illustrate these objectives. Since manuscripts are published in the order in which they are received in final form, it may seem that some important aspects of residue analytical chemistry, biochemistry, human and animal medicine, legislation, pharmacology, physiology, regulation, and toxicology are being neglected; to the contrary, these apparent omissions are recognized, and some pertinent manuscripts are in preparation. However, the field is so large and the interests in it are so varied that the editor and the Advisory Board earnestly solicit suggestions of topics and authors to help make this international book-series even more useful and informative.

"Residue Reviews" attempts to provide concise, critical reviews of timely advances, philosophy, and significant areas of accomplished or needed endeavor in the total field of residues of these chemicals in foods, in feeds, and in transformed food products. These reviews are either general or specific, but properly they may lie in the domains of analytical chemistry and its methodology, biochemistry, human and animal medicine, legislation, pharmacology, physiology, regulation, and toxicology; certain affairs in the realm of food technology concerned specifically with pesticide and other food-additive problems are also appropriate subject matter. The justification for the preparation of any review for this book-series is that it deals with some aspect of the many real problems arising from the presence of residues of "foreign" chemicals in foodstuffs. Thus, manuscripts may encompass those matters, in any country, which are involved in allowing pesticide and other plant-protecting chemicals to be used safely in producing, storing, and shipping crops. Added plant or animal pest-control chemicals or their metabolites that may persist into meat and other edible animal products (milk and milk products, eggs, etc.) are also residues and are within this scope. The so-called food additives (substances deliberately added to foods for flavor, odor, appearance, etc., as well as those inadvertently added during manufacture, packaging, distribution, storage, etc.) are also considered suitable review material.

Manuscripts are normally contributed by invitation, and may be in English, French, or German. Preliminary communication with the editor is necessary before volunteered reviews are submitted in manuscript form.

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Insecticide use in livestock production

By

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I. Introduction

Insecticides are necessary for a productive livestock economy in the United States. A wide range of insects, ticks, and mites must be controlled to protect the health and productivity of livestock. Since an efficient livestock economy also requires the production of adequate amounts of nutritious feeds, it is equally important that insecticides be employed for the control of the many kinds of insects and mites which attack forage and grain crops.

It is difficult to estimate with accuracy the extent of losses that result from direct attack of livestock by insects and related arthropods and the losses in productivity and value of livestock feed crops. However, in a report recently issued by the *U.S. Department of Agriculture* (Agricultural Handbook 291) estimates are given of the losses caused by the more important insects and other pests affecting livestock production. Estimated losses due to the many pests would no doubt be several times higher if the many

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excellent insecticides developed through the joint effort of industry and public-supported research institutions were not available to growers and regulatory agencies for the control of the pests involved.

Arthropods not only cause losses due to direct attack on the animal or plant hosts, but also they are responsible for the transmission of many diseases affecting crops and livestock.

Chemicals provide the principal means of dealing with insect problems. Unfortunately, their use often results in the appearance of chemical residues in meat, milk, and other essential food products derived from livestock. When applied to livestock or poultry, minute amounts of certain insecticides or their metabolites are absorbed through the skin and stored in meat, milk, or eggs. Residues may also occur in these products when livestock consume forage or grains bearing insecticide residues. Such residues generally occur on feeds as the result of insecticides applied to the crops to prevent insect damage. However, drift of insecticides on to forage crops when they are applied to adjacent or sometimes even remote areas also constitutes a source of such residues. The occurrence of residues in raw or processed animal products has led to public concern over the safety of certain insecticides, which in turn has resulted in more stringent laws, regulations, or recommendations governing their registration, sale, and use. As a consequence, the residue problems represent a major aspect of the research on the development of new insecticides for the control of insects affecting livestock and livestock feeds.

As previously noted, insect control chemicals are essential for a productive agricultural economy. At the same time, however, the safety and wholesomeness of food products derived from livestock must be assured. Thus, meeting insect problems with chemicals with safety has required the development of a wide variety of candidate insecticides by industry. Extensive research by entomologists on application rates and treatment schedules is necessary to determine the effectiveness of candidate materials for controlling the many insects affecting livestock and livestock food crops. In turn, extensive and intensive investigations by toxicologists on the toxicity of the insecticides and their metabolites are necessary to appraise the toxicity of promising materials to man and animals. Finally, complex and precise research by chemists is required to develop methods for assaying the generally minute amounts of insecticide residues or their metabolites that may be present in agricultural products. In relation to livestock production, the residue problem involves not only the determination of residues in the animal products but also extensive determinations of residues on or in grain and forage crops and other products utilized as animal feeds.

In order to appraise the scope, complexity, and significance of the insecticide residue problems in relation to livestock production, it seems desirable to present a brief discussion of the more important insect problems involved, and the insecticide treatments employed for insect control. Such discussion will be followed by a review of the information that has been obtained on residues in meat and milk. The progress that has been made in the development of insecticides and in meeting the residue problems in relation to the use of the insecticides represents the joint effort of State Land-Grant institutions, Federal agencies, and the chemical industries.

II. Insects affecting livestock and insecticides required for their control

Livestock are subject to attack by external parasites throughout the year in the United States. During the warmer months, a variety of blood-sucking and annoying flies attack livestock, principally dairy and beef cattle. In the winter, the most important external parasites are the lice on all of the major classes of livestock, the sheep tick on sheep, and the cattle grubs *Hypoderma lineatum* (deVill.) and *H. bovis* (L.), which affect both beef and dairy cattle. Several species of ticks are prevalent during the summer but one species, the winter tick *Dermacentor albipictus* (Pack.), a pest of horses and cattle, is most active during the cooler months. The screwworm *Cochliomyia hominivorax* (Coq.), one of the more important pests of livestock primarily in the southern states, is now being controlled by biological means but special insecticide formulations have been used extensively in the past to protect animals from attack.

The horn fly *Haematobia irritans* (L.), stable fly *Stomoxys calcitrans* (L.), a large number of species of Tabanids (horse flies and deer flies), mosquitoes, black flies (simulids) and sand flies (Culicoides) are important blood-sucking pests of livestock and may substantially reduce weight gains and milk production. In addition to the blood-sucking flies, the house fly *Musca domestica* (L.) is regarded as one of the most important pests because of their abundance on livestock farms where they may contaminate milk with disease organisms and filth, and also possibly spread diseases or parasites to animals. The newly introduced face fly (*Musca autumnalis*) is rapidly spreading in the United States and is becoming of major importance as a pest of cattle and other livestock.

The benefits derived from the use of insecticides are often difficult to assess in monetary terms. However, results by LAAKE (1946) showed that beef cattle protected from attack from horn flies gained about 50 pounds more during the fly season than untreated cattle subject to heavy attack by this insect. BRUCE and DECKER (1947) published data showing that herds of dairy cows subject to attack by horn flies and stable flies produced from ten to 20 percent less milk than did treated herds. Total losses caused by various livestock insects are difficult to measure because of variations in abundance from year to year, season to season, and in different areas. Cattle grubs are among the most costly pests of cattle, causing substantial losses each year because of damaged hides, depreciation in value of beef carcasses, and loss in health or productivity of beef and dairy cattle because of the presence of larvae in the host and annoyance caused by the adult heel flies when they attack cattle for oviposition in the spring. These are only examples of the importance of insects in relation to livestock production. Those mentioned and others affecting livestock and poultry cause losses including cost of control which in the aggregate amount to about four percent of the farm value of livestock and poultry production.

Insecticides now provide the only practical means for controlling most livestock insects. However, sanitary measures and proper management practices are necessary features for good control of such insects as house flies, stable flies, screw-worms, mosquitoes, and certain arthropods affecting livestock and poultry.

A number of insecticides and repellents are used for livestock insect control. The type of formulations, methods of application, amounts applied, and the frequency of treatments vary with the material used and the kinds of pests and hosts involved. It is not practical to discuss in detail the manner of use of the insecticides and repellents for controlling each individual pest on every host. However, Table I has been developed to show the principal insecticides[1] employed, the kinds of insects and hosts involved, and a general indication of the way the materials are used. This should help orient the reader as to the scope of the problem, the nature of the residue hazards that may be involved, and the type of residue studies that have been necessary to establish safe use of insecticides in relation to the control of livestock pests.

III. Insects affecting grain and forage crops and insecticides required for their control

Several major pests of grain and forage crops are widely distributed in the United States and cause substantial losses each year to cultivated grain and forage crops and range grasses. In addition, there are many pests which are sporadic in their abundance but in the aggregate they also cause heavy losses locally or regionally. According to estimates in "Agricultural Handbook 291," the annual yield of corn is reduced by about 12 percent because of damage caused by such insects as the corn ear worm, European corn borer, corn root worms and a number of other pests. It is not practical to discuss all of the insect problems involved. However, a few of the insect problems will be discussed briefly and the nature of the more critical residue problems will be mentioned.

The European corn borer, *Ostrinia nubilalis* (Hubner), is an introduced pest that occurs in most of the corn growing areas. Losses due to this insect will average about 3.5 percent of potential production each year but may substantially exceed this amount in some years. The protection of corn, especially during years of high corn borer density, is necessary to prevent even greater losses. Insecticide residues in relation to the corn borer problem, for the most part, involve the fodder that growers utilize in the field following

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[1] Pesticides mentioned in text are identified chemically in Table XIII.

Table I. *Some of the more commonly used insecticides for the control of insects, ticks, and mites affecting livestock and poultry*

Insecticide[a]	Arthropods and hosts	Methods of use	General restrictions
Pyrethrum & synergists[b]	Bloodsucking flies and mosquitoes, face fly, house fly, lice, and ticks on livestock, including dairy cows	Most commonly used daily for fly control at conc. ranging from 0.05–0.1% pyrethrins employing oil or emulsion mist spray applied at rate of 1–2 fluid oz./cow. For longer lasting protection against flies and for control of lice and ticks, animals are sprayed with water emulsion using conc. mentioned and rates of application ranging from a qt. to a gal. or more	——
Rotenone	Cattle grubs and lice on dairy and beef cattle, lice on poultry, and sheep keds	Generally employed as suspension spray made from 5% rotenone dust. Amount used varies from less than 1 lb. to 7½ lbs. of rotenone material/100 gal. water, depending on insect. Dusts contain from 1–1.5% rotenone for control of lice on livestock and poultry	——
Lethane 384, Thanite	For fly control on cattle	Generally employed each day as oil mist spray containing 3% active ingredient applied at rate of 1–2 fluid oz./animal	——
Methoxychlor	Horn fly on dairy and beef cattle, lice on livestock other than dairy cattle	For dairy cows 1 tablespoon 50% wettable powder conc. is applied to backs and flanks. For other livestock, insecticide is applied as wettable powder or emulsion spray or dip employing conc. ranging from 0.25–0.5% methoxychlor	Methoxychlor not to be used as a spray or dip on dairy cattle
DDT	Horn fly on beef cattle; lice on sheep, goats and swine; and sheep keds	Used to treat back rubber cable with 5% DDT in oil for horn fly; for lice on sheep, goats, and swine and for sheep keds, wettable powder or emulsion sprays or dips contain from 0.25–0.5% DDT	30-day preslaughter limitations to meet 7 p.p.m. tolerance in fat. Not to be used on dairy cows

Table I. (Continued)

Insecticide	Arthropods and hosts	Methods of use	General restrictions
Toxaphene	Horn fly, lice, and ticks on beef cattle; lice on swine; lice and sheep keds on sheep and goats	Used as wettable powder or emulsion spray or dip at conc. ranging from 0.25–0.5%	28-day preslaughter limitation to meet tolerance of 7 p.p.m. in fat. Not to be used on dairy cows
Lindane	Lice, ticks on beef cattle, sheep, goats, and swine. Sheep keds; screw-worm smear	Used as wettable powder or emulsion sprays or dips containing conc. ranging from 0.025–.06% lindane. Employed as wound treatment smear containing 3% lindane for screw-worms (including dairy cows)	30-day preslaughter limitation for lindane for listed uses, except 60 days when used as dip for swine. Not to be used as spray or dust on dairy cows
Malathion	Horn fly, lice, and ticks on beef cattle; lice and sheep keds on sheep and goats; lice on swine and poultry; dust for horn fly on dairy cattle	Dust containing 4–5% malathion applied to back, neck, and flanks of dairy cows using up to 2 oz. Dusts also used as complete coverage for lice control on other livestock and on poultry. Generally used as spray or dip at conc. of 0.5% for other livestock pests	Not to be used as spray on dairy cows; 5-hour pre-milking limitation when dusts are applied to dairy cows
Dioxathion	Horn fly, ticks, and lice on beef cattle; lice on sheep, goats, and swine; sheep keds	Employed as emulsion spray or dip at conc. of 0.15%; used in 1.5% conc. to treat backrubbers	Tolerance of 1 p.p.m. for dioxathion in fat of livestock. Not to be used on dairy cows
Coumaphos	Cattle grubs, horn fly, lice, ticks on beef cattle; lice on sheep, goats, swine, and poultry; sheep ked; spray or dust for screw-worms on livestock	Employed as wettable powder spray or dip at conc. generally ranging from 0.125–0.25%. Action systemic for cattle grub control. Used as 0.5% dust on poultry and as 5% dust to protect wounds from screw-worm attack	Tolerance of 1 p.p.m. in meat and fat. Preslaughter limitation of 45 days for most uses to avoid excessive residues in meat and fat. Not to be used on dairy cows
Ronnel	Horn fly, lice, ticks, and cattle grubs on beef cattle; lice on sheep, goats, and	Employed as wettable powder or emulsion spray or dip at conc. ranging from 0.25–0.5%, except 0.75% for ticks on beef cattle; used as	To meet zero tolerance, preslaughter limitation of 56–60 days for cattle, 84 days

Insecticide	Arthropods and hosts	Methods of use	General restrictions
	swine; sheep keds; screw-worm smear or spray	spray (0.5%) and in smear (5%) for screw-worms. Used as feed additive for cattle grub control	for sheep and goats, and 42 days for swine. Not to be used on dairy cows
Ruelene	Cattle grubs on beef cattle, hornflies and lice on beef cattle	Employed as 0.5% wettable powder or emulsion spray or as a pour-on treatment to back using 8.3% emulsion or 9.4% solution at 1 oz./100 lbs. body weight; action systemic against cattle grubs	28-day preslaughter limitation. Not to be used on dairy cows
Carbaryl	Hornflies, ticks, and lice on beef cattle, and swine; poultry lice and mites	Used as 0.5% spray on livestock, as 5% dust or 0.5% mist spray on poultry	7-day preslaughter limitation for treatment of livestock; 5 p.p.m. tolerance in poultry meat and zero tolerance in eggs
Ciodrin	Horn flies, face flies, lice, ticks on dairy cows	As wet emulsion spray at conc. from 0.15–0.5%; cable treatment for horn flies, 1% solution; mist oil spray at 2% conc.	Certain restrictions on frequency of treatment as a wet spray
Dichlorvos	Horn flies, face flies, and mosquitoes on dairy cows	Mist spray using 1–2 oz. 1% solution	———
Trichlorfon	Cattle grubs	Used as 1% soluble powder spray	14-day preslaughter limitation

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[a] Pesticides mentioned in text are identified chemically in Table XIII.

[b] A variety of synergists are used in combination with pyrethrum, generally at a ratio of 10 parts synergist to 1 part pyrethrins. The synergists employed include piperonyl butoxide, sulfoxide, propyl isome, sesame oil, MGK 264, and others.

the harvest of corn in the fall. The application of insecticides on corn for corn borer control also creates serious residue problems if the corn is to be harvested for ensilage. Substantial progress has been made, however, in the selection of insecticides and formulations that minimize such residues.

The corn earworm, *Heliothis zea* (Boddie), is another major pest of corn which on the average reduces corn yields by about four percent each year. The insect is nationwide in distribution. Insecticides generally are not practical for application to field corn to control this insect because high dosages and several applications are required to achieve a satisfactory degree of control. However, in view of the high value of sweet corn and the need to maintain minimum insect infestations in fresh or processed sweet corn, insecticides are employed in large quantities. Critical residue problems have developed as a result of the growers' efforts to use or market the by-products of sweet corn production as animal feeds, but new insecticides which are less prone to store in animal tissues or appear in milk are coming into general use for treating sweet corn.

A wide variety of insects attack alfalfa and, if not controlled, they can cause partial to virtually complete loss of the alfalfa crop. The potential production of forage alfalfa is reduced by about 15 percent due to insect attack. The insects involoved include the alfalfa weevil *Hypera postica* (Gyllenhal) and the spotted alfalfa aphid *Therioaphis maculata* (Buckton). The high losses to alfalfa during recent years are due in large part to the introduction and spread of these two highly destructive pests. In view of the importance of alfalfa as a dairy feed, the use of insecticides to control insects on alfalfa has caused a number of serious residue problems.

Grasshoppers are major pests of range grasses, as well as for most cultivated forage and grain crops. Grasshopper infestations are generally sporadic on cultivated crops, but are serious pests in localized areas each year. Because of the omnivorous feeding habits of grasshoppers it is necessary during grasshopper outbreaks to apply insecticides to a wide range of crops to prevent damage.

The above are examples of some of the insect problems encountered in the production of grain, forage, and range grasses. Many other insect pests affect grain and forage crops, which necessitates the use of insecticides to deal with them. Other methods of insect control have been investigated in the past and the search for alternate control methods continues. Research is concentrated on the development of resistant varieties, the use of various biological control agents, and cultural control methods. However, insecticides now provide the chief means of dealing with most of the insects affecting animal feed crops. Table II lists the more important insecticides, the major insects and crops treated, the methods of use, and general instructions that must be followed in connection with the safe use of insecticides on crops grown for livestock production.

Table II. *Insecticides employed for controlling the more important insect pests of cultivated forage, grain crops, and range grasses*

Insecticide	Insects and crops treated	Methods of use	General restrictions
Chlorinated hydrocarbons			
DDT	Plant bugs, leafhoppers, thrips, and pea aphid on alfalfa seed crops; corn earworm, armyworms, cutworms, and European corn borer on corn, peanuts, and sorghum; sorghum midge on grain sorghum; velvet bean caterpillar on soy beans and peanuts; white-fringed beetles, Japanese beetles and other soil insects on a variety of crops	Wettable powder and emulsion sprays, dusts, or granules. Dosage generally ranges between 1 and 2 lbs. /acre, but may be as high as 10–25 lbs./acre for controlling soil insects or as low as 5 oz. for thrips	DDT-treated forage is not to be fed to dairy animals being finished for slaughter
Methoxychlor	Alfalfa weevil, alfalfa caterpillar, clover leaf weevil, spittle bugs, and leafhoppers on alfalfa; Mexican bean beetle, Japanese beetle, and velvet bean caterpillar on soybeans; velvet bean caterpillar on peanuts; vetch bruchid on vetch	Emulsion or wettable spray or dust, rates ranging from ½ to 1½ lbs./acre	To meet tolerance up to 100 p.p.m. methoxychlor on no. of forage crops, waiting period of 7 days between treatment and harvest of crop is generally specified
Toxaphene	Grasshoppers, plant bugs, armyworms, and velvet bean caterpillars on alfalfa, soybeans, and/or peanuts; European cornborer on corn; chinch bugs and armyworms on sorghum; stink bugs on rice; grasshoppers, armyworms, and cutworms on small grains	Emulsion spray or dust, dosages range from 1 to 2 lbs./acre; granules on corn for European corn borer	Not to be used on forage intended for dairy cows. Tolerance of 7 p.p.m. established in fat of livestock and for certain forage crops. Also, 5 p.p.m. on certain small grains; 7–14 days specified between treatment and harvest of certain small grains

Table II. (Continued)

Lindane	Alfalfa weevil, spittle bugs on alfalfa and/or clover	Emulsion spray, 4–8 oz./acre	30 days between treatment and harvest of crop specified
Aldrin and dieldrin	Uses of aldrin and dieldrin greatly restricted because of residuc problems. Especially useful in non-grazing areas for grass and turf insects and as barriers for such insects as chinch bugs. Among the most useful insecticides for controlling soil insects.	Dosages from 1–3 oz. for grasshoppers to ¼ to ½ lbs./acre for insects feeding above ground. Aldrin for root worms from 1–2 lbs./acre; for white-fringed beetle larvae dosage up to 3 lbs./acre in waste lands	Tolerances generally zero but range up to 0.25 p.p.m. on certain crops with proposals to reduce tolerances to 0.1 p.p.m. When employed on grazing lands, waiting period of 90 days or longer specified
Endrin	European corn borer and southwest corn borer on corn, armyworms in grain crops, sugarcane borer	Emulsion spray or granules, generally from 3–8 oz. for European corn borer, southwestern corn borer, armyworms, and sugarcane borer	To avoid residues on forage, waiting period from 35-45 days generally specified
Heptachlor	Rootworms and other soil insects to protect corn, certain insects on turf not employed for grazing, grasshoppers on range lands not to be grazed for 90 days	Emulsions, granules, or in fertilizer mixtures; dosage as low as 2 oz. for grasshoppers to as much as 2 lbs. /acre for soil insects	To be used in manner that will not result in residues on forage at time of harvesting or grazing
Organophosphorus insecticides			
Malathion	Alfalfa weevil, spotted alfalfa aphid, and pea aphid on alfalfa; grasshoppers on forage and grain crops; stink bugs on rice; mites on grains; cereal leafbeetle	Emulsion spray at 10 oz. to 1 lb. or more/acre; low vol. sprays for certain insects generally permit lower application rates	Tolerance up to 135 p.p.m. established for no. of forage crops and 8 p.p.m. for certain grain crops; 7 days between treatment and harvest generally specified for use on grain crops
Parathion	Aphids and mites on forage and grain crops, alfalfa weevil, armyworms and cutworms on forage and grain crops, rootworms resistant to other insecticides	Emulsion sprays, granules, or dust at 4–8 oz./acre	To meet a 1 p.p.m. tolerance on variety of crops, 12–15 days between treatment and harvest generally specified

Insecticide	Insects and crops treated	Methods of use	General restrictions
Demeton	Systemic for aphids and mites on alfalfa, aphids on small grains and sugarbeets	Emulsion spray at 4 oz./acre	21 days between treatment and harvest specified to meet 5 p.p.m. tolerance on alfalfa forage and 12 p.p.m. on the hay; waiting periods up to 45 days to meet 0.75 p.p.m. in grains
Phorate	Thrips on peanuts, corn root worms	Granules at 1 lb./acre	No tolerance established
Trichlorfon	Alfalfa caterpillar, cutworms, webworms and armyworms on alfalfa; lygus bugs, leafhoppers, and thrips on alfalfa seed crops	Formulation dissolved in water, sprayed at 4–5 oz./acre, except for cutworms and armyworms which require 1 lb./acre	14 days between treatment and harvest specified to avoid residues
Mevinphos	Aphids, grasshoppers, cutworms, and the alfalfa caterpillar on alfalfa; webworm and corn earworm on sorghum	Emulsion sprays at ¾ to 1½ lbs./acre	To meet 1 p.p.m. tolerance, 1–3 days between treatment and harvest required
Naled	Grasshoppers in alfalfa and clovers, pea aphid on alfalfa, webworms on sorghum, lygus bugs on sugarbeets	Emulsion sprays at ¾ to 1½ lbs./acre	4 days between treatment and harvest required
Diazinon	Alfalfa weevil and spotted alfalfa aphid on alfalfa; grasshoppers in various crops; soil treatments for corn rootworms on peanuts and corn; European corn borer; sod webworms, chinch bugs, and other turf insects	Emulsion spray on alfalfa at 4 oz. for aphids and 8 oz. for grasshoppers; granules or emulsion sprays for soil treatments at 1 lb. for rootworm on corn and 3 lbs. for rootworm on peanuts	To meet 40 p.p.m. tolerance for green and 12 p.p.m. for cured alfalfa hay, 4–7 days between treatment and harvest required. Tolerances on grain crops range from 0.75 p.p.m. in the grain to 40 p.p.m. on forage

Table II. (Continued)

Guthion	Alfalfa weevil larvae	Emulsion at 8 oz./acre	Tolerance of 2 p.p.m. on forage and 5 p.p.m. on hay with 21-day waiting period
Carbamates			
Carbaryl	Corn earworm on corn, sorghum, peanuts, soybeans, and other crops; grasshoppers on grain and forage crops; European corn borer on corn; fall armyworm on variety of crops; leafhoppers and variety of other insects	Wettable powder spray or dust at from about ¼ to 2 lbs./acre	Tolerance ranging from 5–10 p.p.m. established for certain grain crops and beans, 25 p.p.m. on corn fodder, and up to 100 p.p.m. on certain forage crops. Periods ranging up to 21 days specified between treatment and harvest

IV. Other insecticide uses that create residue problems in relation to livestock production

In addition to insect problems involving the control of livestock insects and insects affecting grain and forage crops, there are other uses of insecticides that directly or indirectly create important residue problems in meat and milk. The use of insecticides to control mosquitoes, primarily for the protection of man from their attack, may involve the control of mosquito larvae on irrigated fields or pastures or the application of sprays or aerosols against adult mosquitoes in areas where livestock graze, causing residues on animal feed crops or range. The drift of insecticides on grazing areas or on grain or forage crops when insecticides are used in adjacent areas to protect cotton, vegetables, fruit trees, or forests may create special residue problems in meat and milk. The grower of animals or the feeds may not be aware of such a problem or may have no direct control over the insecticides used in such cases. Another problem that is of major concern involves the use as animal feeds of by-products from crops grown primarily for human consumption and for the production of fiber. Many crops grown primarily for human consumption yield by-products that are excellent feeds for livestock. Some of the more important items include citrus pulp, pea vines, sugar beet tops, sugar beet pulp, apple pomace, and the by-products of sweet corn production. The awareness of this source of residues in livestock products and the development of insecticides less prone to store in animal tissues have greatly lessened the seriousness of this problem. However, when such by-products cannot be used safely for livestock feeds because of residues, they not only represent an economic loss of the feed value of the product but actually create a liability because of disposal problems.

V. Residues resulting from the use of insecticides on livestock

As indicated earlier, some insecticides that are applied directly to livestock to control external parasites may be absorbed through the skin, stored in the body tissues, and excreted in the milk of lactating animals. The organochlorine insecticides present the greatest problem, in this respect. HOWELL *et al.* (1947) were the first to report on the occurrence of DDT residues in milk of dairy cows following the use of DDT as a livestock spray. Due to their stability and solubility in fats and oils, organochlorine insecticides are readily stored in the fatty tissues, and some of them may persist for long periods of time. For the same reasons, they may be excreted in the milk of lactating animals, in solution in the butterfat. When a substantial concentration of an organochlorine insecticide is present in the fat of an animal, it may be slowly released and excreted in the milk over a period of days or weeks, even if there is no further intake.

The organophosphorus insecticides, as a class, are much less stable than the organochlorides and are usually changed quite rapidly to non-toxic compounds by chemical or enzymatic reactions both inside and outside the animal body. Many of the metabolites, and some of the parent compounds, are more soluble in water than in fats and are excreted quite rapidly after absorption. Residues, if present at all, are of short duration.

Carbamates are coming into wide use as insecticides. This class of compound has a tendency to store in fatty tissues, although at much lower levels than the organochlorides.

Pyrethrins, rotenone, Lethane 384, and Thanite may be used in spray or dust form for direct application to animals without causing any residues in meat or milk.

Table III shows some typical data giving residues in tissues after a single spray treatment with several organochlorine insecticides (CLABORN *et al.* 1960a). Table IV, from the same source, shows residues during the spraying period and at several intervals after the last spray where multiple sprays were applied. These data show the extent to which several of the more common organochlorine insecticides will accumulate in the fat of animals under the conditions of the experiments. Lindane, at the concentration required for control purposes, did not cause residues in the fat at any time. Methoxychlor has far less tendency to accumulate in the fat than the other compounds in this class.

Table III. *Insecticides in p.p.m. in the fat of cattle after a single spray treatment*

Insecticide	Weeks after treatment					
	2	6	10	16	22	27
	p.p.m.	*p.p.m.*	*p.p.m.*	*p.p.m.*	*p.p.m.*	*p.p.m.*
DDT, 0.5%	11.2	8.1	5.3	2.3	2.8	1.7
TDE, 0.5%	11.0	5.2	3.9	1.1	0.7	0.5
Methoxychlor, 0.5%	2.8	1.7				
Lindane, 0.03%	None at any time					

ROBERTS and RADELEFF (1960) sprayed hogs twice with 0.5 percent toxaphene emulsion and analyzed the fatty tissues for residues. The tissues contained less than 2 p.p.m. of toxaphene six weeks after the last spray.

Table V gives examples of the residues found by CLABORN *et al.* (1960 a and b) in the tissues of livestock sprayed with organophosphorus insecticides. The levels, it will be noted, are low, and it was further shown that they were rapidly reduced to a point where they could no longer be detected. PLAPP *et al.* (1960) sprayed a steer with 0.15 percent P-32 labelled dioxa-

Table IV. *Insecticides in p.p.m. in the fat of cattle after multiple spray treatments*

Insecticide	Spray Interval (weeks)	After indicated sprayings[a], p.p.m.						After last spraying, p.p.m.		
		1st	2nd	3rd	4th	5th	6th			
								12 weeks	*24 weeks*	*36 weeks*
DDT, 0.5%	3	18	31	—	33	—	35	8	5	2
TDE, 0.5%	3	13	33	—	37	—	28	14	1	1
Methoxychlor, 0.5%	3	1.5	1.5	—	0.8	—	2.4	—	—	—
Lindane, 0.03%	3	None after any spraying						—	—	—
								11 weeks	*28 weeks*	—
Dieldrin, 0.05%	3	7	10	16	24	—	—	17	6	—
								8 weeks	*16 weeks*	—
Heptachlor, 0.5%	2	11	14	14	20	18	19	16	2	—
Chlordane, 0.5%	2	—	—	—	—	—	28	—	4	—
Gamma chlordane, 0.5%	2	9	10	16	17	18	24	16	—	—
								6 weeks	*10 weeks*	*14 weeks*
Strobane, 2%	2	—	—	—	—	—	29	9	4	3
								4 weeks	*6 weeks*	—
Toxaphene, 0.5%	2	0	0	1	7	10	6	4	4	—

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[a] Fat samples were taken at the end of the intervals between spraying.

Table V. *Insecticides in p.p.m. in the fat of livestock after a single spray treatment*

Insecticide	Treatment	Days after treatment	P.p.m. in				
			Omental fat	Renal fat	Muscle	Liver	Kidney
Coumaphos[a]	58 mg./kg.	14 (calves)	0.04	0.03	0.005	0.5	0.2
Dioxathion[a]	16 mg./kg.	7 (calves	1.5	1.07	0	0.2	0.07
Malathion[b]	1%	7 (hogs)	0	0	0	—	—
		7 (sheep)	Trace	Trace	0	—	—
		7 (goats)	Trace	Trace	0	—	—
Malathion[c]	0.5%	7 (cattle)	<0.5	—	—	—	—
Coumaphos[c]	0.5%	7 (cattle)	0.4	—	—	—	—

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[a] CLABORN *et al.* (1960 a).
[b] ROBERTS *et al.* (1960 a).
[c] CLABORN *et al.* (1960 b).

thion and detected small amounts of radioactivity in the fatty tissue immediately after treatment. The residues dropped to zero in seven days. JACKSON *et al.* (1962) sprayed sheep, goats, and cattle with 0.15 percent dioxathion and found that significant amounts were stored in the fatty tissues. The maximum levels, however, were less than 1.0 p.p.m. Fat from cows sprayed six times at two-week intervals contained a maximum of 0.28 p.p.m.

RADELEFF and CLABORN (1960) sprayed dairy cattle with 0.5 percent and 0.75 percent solutions of coumaphos. Maximum levels of organo-soluble compounds of 0.2 and 0.25 p.p.m. occurred in the milk five hours after spraying. These declined to a trace ten days after treatment. Paper chromatography showed that only a part of the residue was coumaphos, the remainder being a mixture of metabolites.

ROBERTS *et al.* (1960a) sprayed sheep, goats, and hogs with 1.0 percent malathion emulsion and analyzed tissues one week after treatment. No malathion was found in hogs but traces were detected in goats and sheep.

Some typical data on the excretion of insecticides in milk, as reported by CLABORN *et al.* (1960a), are shown in Table VI. The organochlorine compounds, it will be noted, are excreted in significant amounts, over a period of 14 to 21 days following a single spray treatment. The organophosphorus compounds were excreted, also, in small but detectable amounts.

Methoxychlor residues were determined in milk by CLUETT *et al.* (1960) after application of dust or spray formulations. No residue was detected

Table VI. *Residues in milk from cows sprayed with various insecticides*

Insecticide	Conc. of spray, %	Max. residue, days after spraying	Max. residue, p.p.m.	Duration of detectable residues, days
DDT	0.5[a]	2	2.8	>21
Dieldrin	0.5[a]	3	7.0	>21
Methoxychlor	0.5[a]	1	0.70	<21
	0.5[b]	2	0.44	<21
Dilan	0.5[b]	2	0.75	>14
Malathion	0.5[a]	5[c]	0.08	3
	1.0[a]	5[c]	0.20	3
	0.5[b]	5[c]	0.27	3
	1.0[b]	5[c]	0.36	3
Strobane	0.5[a]	1	0.74	14
	0.5[b]	2	0.61	14
Toxaphene	0.5[a]	2	0.61	14
	0.5[b]	2	0.74	14
Coumaphos	0.5[a]	5[c]	0.20	14
	0.75[a]	5[c]	0.25	14
Perthane	0.5[a]	3	0.17	<21

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[a] Emulsion.
[b] Suspension.
[c] Hours.

in the milk after treatment with ten grams of a 50 percent dust. Spraying with an aqueous suspension or emulsion caused residues of less than 0.1 p.p.m. that reached a maximum in one day and persisted for one week. Application of four times the normal dosage caused a residue of 0.23 p.p.m. in the milk.

BUTTRAM and ARTHUR (1961) reported that dairy cows, sprayed with a 0.5 percent concentration of Bayer 22,408 excreted small but detectable amounts of the insecticide in the milk for a period of six days after treatment.

CHENG *et al.* (1961) treated dairy barns and loafing pens with 0.5 percent and 1.0 percent dimethoate sprays for fly control. No dimethoate was detected in the milk of dairy cows held in the barn and pens. CHENG *et al.* (1962) treated dairy cows with aerosol and spray formulations of dimethoate and were unable to detect residues of this compound in the milk with the exception of one sample that was thought to have been accidentally contaminated.

Coumaphos was used as a spray on dairy cows at 0.5 percent and 0.75 percent concentrations by CLABORN *et al.* (1962a). Maximum concentrations of 0.2 and 0.25 p.p.m., respectively, were detected in the milk five hours after treatment. There was a gradual decline until only a trace was detected ten days after treatment.

CLABORN *et al.* (1963) sprayed cattle, sheep, goats, and hogs four times in two weeks with a 1.0 percent suspension of carbaryl. Animals were

Table VII. *Carbaryl residues in tissues of cattle, sheep, goats, and hogs after four sprayings with a 1.0 percent concentration*

Days after spraying	Carbaryl in tissues, p.p.m.[a]						
	Omental fat	Renal fat	Muscle	Liver	Heart	Kidney	Brain
Cattle							
1	0.57	0.54	0.13	0.00	0.05	0.10	0.10
7	<0.04	< 0.04	<0.04	<0.04	<0.04	<0.04	<0.04
Sheep							
1	0.21	0.10	0.07	<0.04	0.07	0.04	0.15
7	<0.04	<0.04	<0.04	<0.04	<0.04	<0.04	<0.04
Goats							
1	0.38	0.24	0.18	<0.04	<0.04	0.06	23.0
7	0.70–0.90	0.12–0.25	<0.04	<0.04	<0.04	0.04	0.30
Hogs							
1	<0.04	<0.04	<0.04	<0.04	<0.04	<0.04	<0.04

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[a] Values shown are net values from which readings of control samples have been subtracted.

slaughtered at intervals of one and seven days after the last spraying. Residues of carbaryl, 1-naphthol, and conjugates of 1-naphthol were detected in the body tissues one day after spraying but were not found in the tissues of any of the animals after seven days except in the fat and in the brain of a goat; it was believed that the latter samples were contaminated accidentally in some manner. Table VII summarizes the data for carbaryl and Table VIII shows the 1-naphthyl conjugates found in kidney tissue.

Table VIII. *Residues of chemically bound 1-naphthol (conjugates) in unwashed kidney tissue of animals sprayed with carbaryl*

Animal	1-Naphthol in kidney tissue, p.p.m.[a]	
	1 day after spraying	7 days after spraying
Cattle	1.50	0.12
Sheep	0.34	0.26
Goats	4.00	0.10
Hogs	<0.02	<0.02

[a] Values shown are net values from which readings of control samples have been subtracted.

ROBERTS *et al.* (1960b) sprayed milking cows with a 0.5 percent suspension of carbaryl, at four-day intervals for a total of four sprays. No residues were detected in the fatty tissue or milk. EHEART *et al.* (1962) applied either a 0.5 percent or 50 percent dust to dairy cows immediately after morning or evening milking. No residue of carbaryl was detected in subsequent milkings.

VI. Residues in meat and milk resulting from ingestion of insecticides

The widespread use of insecticides on crops of all kinds inevitably results in the consumption of varying amounts of these compounds by livestock. Extensive use of insecticides is required for the production of forage and grain crops, but the compounds that may be used are strictly limited by their residual life on the crops and their propensity for storage in animal tissues or excretion in milk. Table IX shows the accumulation of some organochlorine insecticides in the fat of cattle when known amounts were fed [CLAYBORN *et al.* (1960 a)]. It will be noted that low levels of intake did not, at a rule, cause unduly high residues in the body fat, and the residues that did accumulate were reduced quite rapidly after the ces-

Table IX. *Residues of various insecticides stored in the fat of cattle fed known amounts added to their diet*

Insecticide	Dosage, p.p.m. in diet	Residue, p.p.m.									
		Weeks after feeding started				Weeks after feeding ceased					
		4	8	12	16	4	8	16	20	24	32
Aldrin	25	50	78	—	—	51	36	—	20	—	8
	2.5	4	5	5	5	—	2	—	—	—	—
BHC	100	159	223	230	250	84	—	17	—	—	—
Chlordan	10	11	13	10	11	—	—	—	—	—	—
	25	12	18	—	—	14	5	—	0	—	—
DDT	25	22	34	42	40	19	—	11	—	6	—
Dieldrin	2.5	7	11	10	14	6	—	—	—	—	—
Endrin	2.5	1	3	1	1	—	—	—	—	—	—
Heptachlor	2.5	1	1	1	0.2	—	—	—	—	—	—
Methoxychlor	25	0	0	—	0	—	—	—	—	—	—
Lindane	10	—	7	8	4	5	—	—	—	—	—
	100	70	76	111	60	12	—	—	1	—	—
Toxaphene	25	2	4	10	12	—	—	—	—	—	—
	100	26	34	33	38	14	3	—	—	—	—

sation of feeding. Of the compounds shown, dieldrin gave the highest deposit in fat per unit fed.

LONG *et al.* (1961) grazed lambs on pasture that had received six applications of endrin at 0.25 lb./acre each, applied as a granular formulation. After grazing for 55 days, residues of about 20 p.p.m. were found in the fatty tissues of the lambs. The residues were reduced to about ten p.p.m. by a subsequent feeding period of 42 days on endrin-free feed.

IVEY *et al.* (1961) determined aldrin and dieldrin in the body tissues of cattle, sheep, and hogs that received aldrin in their feed. At a feeding level of ten p.p.m. the dieldrin residues in the fat were 39 p.p.m., while the lowest level fed, 0.25 p.p.m., resulted in about one p.p.m. in the fat. Much lower amounts were found in liver, kidney, and muscle tissues. At the ten p.p.m. feeding level, muscle tissue contained 0.17 to 0.26 p.p.m. The residues were not significantly reduced by cooking. Aldrin was found only in very small amounts at the highest feeding levels.

SMITH *et al.* (1960) fed dairy cows alfalfa hay containing eight p.p.m. of malathion for three weeks. This insecticide was not detected in the fatty tissues, or in the milk, at any time.

CLABORN *et al.* (1960a) fed toxaphene to dairy cows over an eight-week period and determined residues in milk during this period and for three weeks after feeding had ceased. At an intake of 20 p.p.m., based on total feed consumed, a maximum of about 0.4 p.p.m. was excreted in the milk. At an intake of 100 p.p.m., the maximum level in milk was about 1.1 p.p.m. The residues decreased rapidly after feeding was discontinued.

ZWEIG *et al.* (1963b) fed low levels of toxaphene to dairy cattle in their daily ration. Feeding continued for 77 days, followed by a 14-day period on toxaphene-free feed. Twenty p.p.m. of toxaphene in the feed resulted in the excretion of a maximum of 0.22 p.p.m., reached in 21 days. At five p.p.m., about 0.1 p.p.m. was present in the milk after the same time. By extrapolation it was estimated that one p.p.m. of toxaphene in the feed would produce 0.03 p.p.m. in the milk. Residues at all levels declined to zero after 14 days on uncontaminated feed.

ZWEIG *et al.* (1961) had also carried out investigations designed to establish a maximum level for DDT that could be fed to milk cows without causing the excretion of detectable amounts in the milk. In this work, anything less than 0.01 p.p.m. was considered to be zero. Table X shows some of the data. Examination of these data reveals the fact that there undoubtedly was DDT present in the milk of cows fed the lowest amount (0.5 p.p.m.), although the amounts were consistently below 0.01 p.p.m. It appears probable, too, that an extension of the feeding period would eventually have resulted in detectable residues.

A recent paper by WESTLAKE *et al.* (1963) illustrates the effect of very low-level intake of organochlorine pesticides in terms of excretion in milk. In this instance, technical chlordan was applied to pasture and milk cows were grazed thereon. While at no time was there any detectable heptachlor

Table X. *DDT residues in milk from cows fed daily rations containing varying amounts of added DDT*

Time, days	DDT (p.p.m.) in milk from Jersey (*J*), Guernsey (*G*), and Holstein (*H*) cows									
	0.5 p.p.m added[a]		1.0 p.p.m added[a]		2.0 p.p.m added[a]		3.0 p.p.m added[a]		5.0 p.p.m added[a]	
	J	H	J	H	G	H	J	H	G	H
	DDT feeding									
0	0	0	0	0	0	0.01	0.01	0	0	0
2	0	0	0	0.01	0.01	0.05	0.06	0.04	0.16	0.02
5	0	0	0	0.01	0.03	0.03	0.06	0.03	0.24	0.02
7	0	0	0.02	0.02	0.03	0.01	0.12	0.08	0.32	0.08
9	0	0	0.01	0	0.04	0.01	0.12	0.05	0.25	0.06
12	0	0	0.02	0.01	0.06	0.03	0.18	0.07	0.22	0.07
16	0	0	0	0.02	0.06	0.04	0.15	0.09	0.25	0.09
19	0	0	0.01	0.02	0.05	0.02	0.14	0.08	0.18	0.09
24	0	0	0.02	0.01	0.04	0.02	0.11	0.09	0.20	0.10
27	0	0.02	0.03	0.02	0.10	0.05	0.12	0.10	0.31	0.10
31	0	0.01	0.01	0.03	0.05	0.05	0.06	0.09	0.21	0.10
	DDT feeding discontinued									
2	0	0	0.01	0.01	0.03	0.02	0.04	0.04	0.07	0.02
4	0	0.01	0.02	0.01	0	0.01	0.02	0.04	0.10	0.03
6	0	0.01	0.01	0	0.05	0.06	0.04	0.01	0.05	0
8	0	0	0	0	0	0	0	0.03	0.09	0
10	0	0	0.01	0.01	0	0	0.04	0.04	0.06	0.03
12	0	0	0	0.01	0	0.01	0	0.03	0.04	0

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[a] DDT added to ration.

or heptachlor epoxide on the grass or in the soil, the milk of the cows contained detectable amounts of the latter at the lowest rate of application (0.25 lb./acre). This heptachlor epoxide was presumed to result from about six percent of heptachlor occurring in the technical grade chlordan. In this experiment, heptachlor epoxide levels below 0.01 p.p.m. were considered to be zero. Chlordan was not detected at the lowest treatment level but was found in the higher treatments. The lower limit of the analytical method for chlordan was set at 0.02 p.p.m. Tables XI and XII show the residues found at 0.25 and 0.5 lb./acre, respectively.

Table XI. *Heptachlor epoxide in the milk of cows grazed on pasture treated with 0.25 lb. of chlordan/acre*

Sample date	Heptachlor epoxide, p.p.m.[a]	
	Animal 1	Animal 14
9/6 (pretreat)	0	0
9/14 (pretreat)	0	0
9/29[b]	0	0
10/2	0	0
10/4	0	0
10/7	0.02	—
10/9	0.03	0.02
10/18	0.01	0.01
10/25	0	0.02
11/8	0	0
11/21	0	0

[a] Check sample readings were zero.
[b] Cows exposed on September 26, 1961 after application on September 25, 1961.

GANNON and DECKER (1960) grazed dairy cows on pastures that had been treated with 0.5 lb./acre of dieldrin or heptachlor, or three lbs./acre of DDT. The cows were placed on the pasture immediately after treatment. The maximum concentrations found in the milk were 3.0 to 4.0 p.p.m. of dieldrin, 7.0 to 8.0 p.p.m. of DDT, and 0.2 p.p.m. of heptachlor epoxide. These occurred in three to seven days after the cows were placed on the pastures and declined steadily thereafter.

BACHE *et al.* (1960) fed heptachlor epoxide to dairy cattle at the rates of 0.5 or 1.0 p.p.m. of the roughage intake. These levels simulate the heptachlor epoxide residues found on forage after the applications of heptachlor. During a two-week feeding period the heptachlor epoxide residue in the butterfat reached a maximum of 0.4 and 1.9 p.p.m. for the two levels, respectively, and decreased steadily after feeding was discontinued. The maximum amounts found correspond to about 0.01 and 0.08 p.p.m., respectively, in the whole milk, calculated on the basis of four percent butterfat.

Table XII. *Chlordan and heptachlor epoxide in the milk of cows grazed on pasture treated with 0.5 lb. of chlordan/acre*

Sample date	Chlordan (*C*) and heptachlor epoxide (*HE*) residues in milk, p.p.m.[a]															
	Animal 5[b]		Animal 10[b]		Animal 4[c]		Animal 11[c]		Animal 6[d]		Animal 12[d]		Animal 2[e]		Animal 3[e]	
	C	HE	C	HE	C	HE	C	HE	C	HE	C	HE	C	HE	C	HE
9/14 (pretreat)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
9/29	0.02	0	0.07	0	—	—	—	—	—	—	—	—	—	—	—	—
10/2	0.02	0	—	—	—	—	—	—	—	—	—	—	—	—	—	—
10/3	—	—	0.02	0.02	—	—	—	—	—	—	—	—	—	—	—	—
10/4	<0.02	0	—	—	0	0	0	0	—	—	—	—	—	—	—	—
10/7	<0.02	0	—	—	0.04	0.02	<0.02	0.03	—	—	—	—	—	—	—	—
10/9	0.05	0.02	0.05	0.05	0	0.02	0.05	0.02	—	—	—	—	—	—	—	—
10/11	—	—	—	—	0.03	0.01	0.03	0.01	0.04	0	0	0	—	—	—	—
10/13	—	—	—	—	<0.02	0.04	—	—	0.04	0	—	—	—	—	—	—
10/15	—	—	—	—	—	—	<0.02	0.02	<0.02	0	0	0.02	—	—	—	—
10/18	0.04	0.03	<0.02	0.03	0	0.03	0.04	0.02	<0.02	0	0.04	0.04	—	—	—	—
10/20	—	—	—	—	—	—	—	—	0	0.02	<0.02	0.03	—	—	—	—
10/25	—	0.06	0.07	0.04	0	0.06	0.03	0.02	0	0.03	0.04	0.03	0.03	0	0.02	0
10/28	—	—	—	—	—	—	—	—	0.03	0.03	—	0.06	0.03	0	0.03	0
10/31	—	—	—	—	—	—	—	—	<0.02	0.05	0	0.05	—	0.04	0.02	0.03
11/1	—	—	0.05	0.04	0	0.05	0.04	0.03	0.06	0.06	0.05	0.05	0.04	0.02	0.02	0.03
11/4	—	—	—	—	0.04	0.03	—	—	—	—	—	—	—	—	—	—
11/8	0	0.04	0.07	0.04	0	0.03	0.04	0.03	—	—	0.05	0.04	0.05	0	0	0.03
11/21	0	0.04	0.07	0	0	0.04	0.04	0.02	0	0.03	0	0	0.08	0.03	0.04	0.03

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[a] Check sample readings were zero for heptachlor epoxide. Correction made for chlordan average check value of 0.01 p.p.m. (range: 0 to 0.02 p.p.m.).

[b] Exposed on September 26, 1961 after application on September 25, 1961.

[c] Exposed on October 2, 1961 after application on September 25, 1961.

[d] Exposed on October 9, 1961 after application on September 25, 1961.

[e] Exposed on October 23, 1961 after application on September 25, 1961.

ZWEIG *et al.* (1963 a) fed dairy cattle a ration containing 1.0 p.p.m. of Kelthane for 39 days and one containing 2.0 p.p.m. of this insecticide for 71 days. At the 2.0 p.p.m. level, the butterfat produced by the cows contained 0.2 to 0.4 p.p.m. of the insecticide. No significant residues were found in the milk of the cows fed at the 1.0 p.p.m. level.

GYRISCO *et al.* (1960) fed carbaryl to dairy cows at the rates of 50, 150, or 450 p.p.m., based on their average consumption of hay and silage. No residues were detected in the milk during and after a two-week feeding period.

LYNN *et al.* (1963) found that bromide was secreted in the milk of cows as a result of naturally-occurring bromide in their diet and as a result of added sodium bromide, or of inorganic bromide resulting from methyl bromide fumigation. The levels detected in milk were proportional to the amounts in the feed. Cows that were fed methyl bromide-treated grain containing 53 p.p.m. of total bromide secreted from four to 12 p.p.m. in their milk. At a feeding level of 100 p.p.m., the milk contained seven to 12 p.p.m., and at a 220 p.p.m. level, ten to 20 p.p.m. were detected.

FERTIG and SCHREIBER (1961) fed dairy cows forage containing 300 p.p.m. of dalapon based on dry weight of feed. A maximum level of 2.45 p.p.m. was reached in the milk seven days after feeding was begun. After feeding of dalapon was stopped, the level of the pesticide in the milk dropped to 0.1 p.p.m in three days. KUTSCHINSKI (1961) fed dairy cows feed containing 200 p.p.m. of dalapon for eight weeks, and 20, 50, and 100 p.p.m. for four weeks. The levels in milk reached a maximum of about 0.9 p.p.m. in five days, at the 200 p.p.m. feeding level. The maximums for the 100, 50, and 20 p.p.m. levels were 0.4, 0.3, and 0.05 p.p.m., respectively. Residues in milk were reduced to zero one week after the feeding of dalapon was discontinued.

VII. Problems in meeting tolerances

Tolerances have been established in the United States for many insecticides in meat. These tolerances are at levels, in many instances, that permit the proper and recommended use of the compounds without danger of excessive residues occurring. A reasonable period of feeding on insecticide-free feed will reduce the residues rapidly if they are accidentally exceeded by improper usage. Where very low tolerances (0.25 p.p.m. or less), exist, however, or when there is a zero tolerance (e.g., endrin) any exposure to the organochlorine pesticides may lead to excessive residues and must be avoided. There is little difficulty to be anticipated from the proper use of the organophosphorus insecticides, as residues resulting from their use are of a low order of magnitude and are metabolized and excreted rapidly. The one carbamate reported upon (carbaryl) is in the same category.

The situation regarding milk is much more difficult, because a tolerance of zero exists for all insecticides in milk. Any appreciable exposure to organochlorine insecticides must, therefore, be avoided. Trace amounts may perhaps be present in the feed of dairy cattle without causing detectable residues in milk, but there is risk involved at any level. It is obvious from the data available that the organochlorine compounds will be excreted in the milk at rates proportional to the amounts ingested or absorbed. The levels, therefore, that will result in detectable residues in milk depend upon the sensitivity of the analytical methods employed in the analyses. It is obviously impossible to determine a true "zero" level and the actual value of "zero" can only be the smallest amount that can be detected. The rapid development of more sensitive and specific analytical methods now taking place is constantly reducing the "zero" level, necessitating a continual review of insecticide uses on dairy cattle and in their feed.

Table XIII. *Chemical names of pesticides mentioned in text*

aldrin—not less than 95% of 1,2,3,4,10,10-hexachloro-1,4,4a,5,8,8a-hexahydro-1,4-*endo,exo*-5,8-dimethanonaphthalene
Bayer 22,408—*O,O*-diethyl *O*-naphthalimido phosphorothioate
benzene hexachloride—1,2,3,4,5,6-hexachlorocyclohexane, consisting of several isomers and containing a specified percentage of *gamma*-isomer
carbaryl—1-naphthyl *N*-methylcarbamate
chlordan—at least 60% of 1,2,3,4,5,6,7,8,8-octachloro-2,3,3a,4,7,7a-hexahydro-4,7-methanoindene and not over 60% of related compounds
Ciodrin—*alpha*-methylbenzyl 3-hydroxycrotonate dimethyl phosphate
coumaphos—*O,O*-diethyl *O*-(3-chloro-4-methyl-2-oxo-2H-1-benzopyran-7-yl) phosphorothioate
dalapon—2,2-dichloropropionic acid
DDT—1,1,1-trichloro-2,2-bis(*p*-chlorophenyl) ethane
demeton—mixture of *O,O*-diethyl *S* (and *O*)-2(ethylthio) ethyl phosphorothioate
diazinon—*O,O*-diethyl *O*-(2-isopropyl-4-methyl-6-pyrimidinyl) phosphorothioate
dichlorvos—2,2-dichlorovinyl dimethyl phosphate
dieldrin—not less than 85% of 1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4-*endo,exo*-5,8-dimethanonaphthalene
Dilan—a mixture of 1 part of 1,1-bis(*p*-chlorophenyl)-2-nitropropane and 2 parts of 1,1-bis(*p*-chlorophenyl)-2-nitrobutane
dimethoate—*O,O*-dimethyl *S*-(*N*-methylcarbamoylmethyl) phosphorodithioate
dioxathion—*p*-dioxane-2,3-diyl ethyl phosphorodithioate
endrin—1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4-*endo,endo*-5,8-dimethanonaphthalene
Guthion—*O,O*-dimethyl *S*-[4-oxo-1,2,3-benzotriazin-3(4*H*)-ylmethyl] phosphorodithioate
heptachlor—3a,4,5,6,7,8,8-heptachloro-3a,4,7,7a-tetrahydro-4,7-methanoindene
heptachlor epoxide—3a,4,5,6,7,8,8-heptachloro-2,3-epoxy-2,3,3a,4,7,7a-hexahydro-4,7-methanoindene
Kelthane—1,1-bis(*p*-chlorophenyl)-2,2,2-trichloroethanol
Lethane 384—2-(2-butoxyethoxy) ethyl thiocyanate
lindane—not less than 99% *gamma*-isomer of 1,2,3,4,5,6-hexachlorocyclohexane
malathion—*S*-[1,2-bis(ethoxycarbonyl) ethyl]*O,O*-dimethyl phosphorodithioate
methoxychlor—1,1,1-trichloro-2,2-bis(*p*-methoxyphenyl) ethane
mevinphos—methyl 3-hydroxy-*alpha*-crotonate dimethyl phosphate
MGK-264—*N*-(2-ethylhexyl)-5-norbornene-2,3-dicarboximide

naled—1,2-dibromo-2,2-dichloroethyl dimethyl phosphate
parathion—*0,0*-diethyl *0-p*-nitrophenyl phosphorothioate
Perthane—a mixture of 1,1-dichloro-2,2-bis(*p*-ethylphenyl) ethane (95%) and related reaction products (5%)
phorate—*0,0*-diethyl *S*-(ethylthio)methyl phosphorodithioate
piperonyl butoxide—*alpha*-[2-(2-butoxyethoxy)ethoxy]-4,5-(methylenedioxy)-2-propyltoluene
propyl isome—dipropyl 5,6,7,8-tetrahydro-7-methylnaphtho-(2,3-*d*)-1,3-dioxole-5,6-dicarboxylate
ronnel—*0,0*-dimethyl *0*-(2,4,5-trichlorophenyl) phosphorothioate
Ruelene—4-*tert*-butyl-2-chlorophenyl methyl methylphosphoramidate
Strobane—terpene polychlorinates (65% chlorine)
sulfoxide—1,2-(methylenedioxy)-4-[2-(octylsulfinyl)propyl] benzene
Thanite—a mixture of isobornyl thiocyanoacetate (82%) and related compounds
toxaphene—chlorinated camphene containing 67–69% chlorine
trichlorfon—dimethyl (2,2,2-trichloro-1-hydroxyethyl) phosphonate

Summary

Insecticides are necessary in the United States to control a wide range of insects, ticks, and mites to protect the health and productivity of livestock. Equally important is the control of insects and mites that attack forage and grain crops necessary to provide adequate feed supplies. Chemicals provide the principal means of control but their use often results in chemical residues in meat and milk, either by absorption through the skin when applied to the animals, or through ingestion where treated feeds are used. Extensive research is required to determine the effectiveness of pesticides, to appraise their toxicity to man and animals, and to develop methods of analysis for residues in animal products and feeds.

Livestock are subject to attack by many external parasites in the United States, including the sheep tick, the cattle grubs *Hypoderma lineatum* (de Vill.) and *H.bovis* (L.), the screw-worm *Cochliomyia hominivorax* (Coq.), the horn fly *Haematobia irritans* (L.), the stable fly *Stomoxys calcitrans* (L.), various species of Tabanids, mosquitos, and others. The benefits from control of these pests are great, and the only practical means for controlling them is the use of insecticides. Various insecticides and repellants are used for this purpose.

Many pests of grain and forage crops cause heavy damages in the United States and require control by the use of insecticides. Among the more important are the European corn borer *Ostrinia nubilalis* (Hubner), the corn earworm *Heliothis zea* (Boddie), the alfalfa weevil *Hypera postica* (Syllenhal), many species of grasshoppers, and others.

There are uses of insecticides other than for controlling insects on livestock or grain and forage crops that may create residue problems in animal products. For example, application of insecticides to control mosquitoes may result in residues in grain and forage crops through drift to areas planted in these crops. The same is true when insecticides are used on cotton, fruits and vegetables, forests, and the like. By-products from crops grown for food and fiber may be used for livestock feed and thus cause insecticide residues in meat and milk.

Of the insecticides now in use, the organochlorine compounds are most likely to cause residues in animal products. Many of this class of insecticides are accumulated and stored in the fat of animals and remain there for long periods of time. In lactating animals, they are excreted in the milk. The organophosphorus insecticides are usually much less stable than the organochlorides, and are changed quite rapidly to non-toxic compounds both inside and outside the animal body. The carbamates used as insecticides have some tendency to store in fatty tissues, but only in small amounts.

Extensive research has been done to determine the fate of insecticides in animals after absorption from direct application or ingestion in feeds to determine the uses that may be permitted without causing residues that exceed the established legal tolerances. Usually, when a finite tolerance exists, insecticides may be successfully used to control the insect pests without exceeding these tolerances. In some instances, however, a "zero" tolerance exists (in milk, the tolerance for any insecticide is "zero") and the problem becomes more complicated.

Résumé*

Les insecticides sont nécessaires aux Etats Unis pour lutter contre une grande variété d'insectes, de tiques et d'acariens et protéger ainsi la santé et la productivité du bétail. Il est également important de lutter contre les insectes et les acariens qui s'attaquent aux fourrages et aux graines nécessaires pour assurer une nourriture adéquate du bétail. Les produits chimiques fournissent les principaux moyens de lutte mais leur emploi donne souvent des résidus chimiques dans la viande et le lait, soit après absorption par la peau lorsqu'ils sont appliqués sur les animaux, soit après ingestion lorque des aliments traités ont été donnés au bétail. Une recherche extensive est nécessaire pour déterminer l'efficacité des pesticides, pour apprécier leur toxicité pour l'homme et les animaux et pour développer des méthods d'analyse des résidus dans les produits animaux et les fourrages.

Le bétail est sujet à de nombreux parasites externes aux Etats Unis, tels que la tique des moutons, les oefres *Hypodermalineatum* (de Vill.) et *H. bovis* (L.), la Lucilie bouchère, *Cochliomyia hominivorax* (Coq.), les mouches piqueuses *Haematobia irritans* (L.), les mouches d'écurie *Stomoxys calcitrans* (L.), différentes expèces de tabanides, de moustiques etc. Les bienfaits de l'éradication de ces parasites sont grands, et le seul moyen pratique d'y parvenir est l'emploi des insecticides. Divers insecticides et répulsifs sont utilisés à cette fin.

De nombreux parasites des graines et des fourrages provoquent de lourds dommages aux Etats Unis et exigent l'utilisation des insecticides. Parmi les plus importants il faut citer *Ostrinia nubilalis* (Hubner), la noctuelle du

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* Traduit par R. MESTRES.

maïs *Heliothis zea* (Boddie), le charançon de la luzerne *Hypera postica* (Syllenhal), de nombreuses espèces de sauterelles, etc.

L'emploi des insecticides pour d'autres raisons que la lutte contre les parasites du bétail, des graines et des fourrages peut aussi créer des problèmes de résidus dans les produits animaux. Par exemple, l'épandage des insecticides pour la démoustication peut entraîner la présence de résidus dans les graines et les fourrages à la suite de dérive vers les zones cultivées. Il en est de même lorsque les insecticides sont utilisés sur le coton, les fruits et les légumes et les produits similaires. Les sous-produits de récoltes destinées à la préparation d'aliments et de fibres peuvent être utilisés pour l'alimentation du bétail et entrainer ainsi des résidus d'insecticides dans la viande et le lait.

Parmi les insecticides actuellement utilisés, les composés organo-chlorés sont les plus susceptibles de donner des résidus dans les produits animaux. De nombreux corps de ce groupe sont accumulés et conservés dans les graisses des animaux où ils persistent longtemps. Chez les animaux laitiers, ils sont eliminés par le lait. Les insecticides organo-phosphorés sont généralement beaucoup moins stables que les organo-chlorés et se transforment très rapidement en composés non toxiques à la fois in vivo et hors de l'animal. Les carbamates utilisés comme insecticides ont quelque tendance à subsister dans les matières grasses, mais seulement en petites quantités.

Des recherches extensives ont été faites pour déterminer le sort des insecticides dans les animaux après leur absorption due à une application directe ou à une ingestion avec des aliments pour préciser les utilisations qui peuvent être autorisées sans provoquer des résidus excédant les tolérances légales établies. Habituellement, lorsqu'une tolérance précise existe, les insecticides peuvent être utilisés avec succés pour lutter contre les parasites sans dépasser ces tolérances. Dans quelques cas cependant, il existe une tolérance "zéro" (dans le lait, par exemple, la tolérance est "zéro" pour tout insecticide) et le problème devient beaucoup plus compliqué.

Zusammenfassung*

Insektizide werden in den Vereinigten Staaten benutzt, um eine Vielzahl von Insekten, Zecken und Milben zu bekämpfen und Gesundheit und Produktivität des Viehs zu erhalten. Von gleicher Bedeutung ist die Bekämpfung von Insekten und Milben, die Grün- und Körnerfutter angreifen, die zur Bereitstellung angemessener Futtervorräte notwendig sind. Bei der Bekämpfung sind Chemikalien die Hauptwerkzeuge, aber oft bewirkt ihr Gebrauch Rückstände in Fleisch und Milch. Sie kommen zustande entweder durch Absorption durch die Haut, wenn die Tiere behandelt werden, oder durch orale Aufnahme, wenn behandeltes Futter verwendet wird. Umfangreiche Forschungsarbeit wird benötigt, um die Wirkung von Pestiziden festzustellen, ihre Toxizität auf Mensch und Tier zu beurteilen und analytische

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* Übersetzt von H. MARTIN.

Methoden zur Feststellung der Rückstände in tierischen Produkten und in den Futtermitteln zu entwickeln.

Das Vieh wird in den U.S.A. von vielen Ektoparasiten angegriffen. Dazu gehören die Schafzecke, die in der Rinder-cutis schmarotzende Larve von *Hypoderma lineatum* (de Vill.) und *H. bovis* (L.), die Schmeissfliegenlarve *Cochliomyia hominivorax* (Coq.), die Stechfliege *Haematobia irritans* (L.), die Stallfliege *Stomoxys calcitrans* (L.), verschiedene Tabaniden-Arten, Mücken und andere. Die Bekämpfung dieser Schädlinge bietet grosse Vorteile, und die einzige praktische Möglichkeit, sie zu bekämpfen, liegt im Gebrauch von Insektiziden. Verschiedene Insektizide und Repellents werden für diesen Zweck verwendet.

In den Vereinigten Staaten verursachen zahlreiche Insektenarten schwere Schäden an Getreide- und Futterpflanzen, so dass eine Kontrolle mit Insektiziden notwendig ist. Unter denen mit grösserer Bedeutung sind der Maiszünsler *Ostrinia nubilalis* (Hubner), der Maisschädling *Heliothis zea* (Boddie), der Luzernekäfer *Hypera postica* (Syllenhal), viele Heuschreckenarten u.a.m.

Es gibt aber auch Anwendungsarten von Insektiziden, die nichts mit der Bekämpfung von Insekten auf Vieh, Getreide und Futter zu tun haben, die Rückstandsprobleme in tierischen Produkten zur Folge haben können. So können Insektizide bei der Mosquito-Kontrolle Rückstandsprobleme durch Abdrift auf Getreide- oder Futterbestände bewirken. Das gleiche gilt, wenn Insektizide in Baumwolle, im Obst- und Gemüsebau und in der Forstwirtschaft angewendet werden. Nebenprodukte aus der Nahrungsmittel- und Fasererzeugung können als Viehfutter Verwendung finden und so insektizide Rückstände in Fleisch und Milch hervorrufen.

Von den gegenwärtig verwendeten Insektiziden erzeugen die Chlorkohlenwasserstoffe mit grosser Wahrscheinlichkeit Rückstände in tierischen Produkten. Viele Repräsentanten dieser Insektizidklasse werden in tierischem Fett angereichert und gespeichert und verbleiben hier über lange Zeiträume. Bei lactierenden Tieren werden sie mit der Milch ausgeschieden. Die organischen Phosphorverbindungen sind für gewöhnlich viel weniger stabil als die Organochloride und werden recht schnell zu ungiftigen Verbindungen innerhalb und ausserhalb des tierischen Körpers abgebaut. Die als Insektizide angewandten Carbamate haben einige Tendenz, sich im Fettgewebe zu sammeln, aber nur zu geringen Mengen.

Es wurde umfangreiche Forschungsarbeit geleistet, um das Schicksal von Insektiziden in Tieren nach Adsorption aus direkter Behandlung oder Aufnahme mit der Nahrung zu bestimmen und Anwendungsformen zu entwickeln, die zugelassen werden können, ohne dass Rückstände entstehen, welche die gesetzlich festgesetzten Toleranzen überschreiten. Wenn eine festgesetzte Toleranz besteht, dann können Insektizide gewöhnlich erfolgreich zur Insektizidbekämpfung eingesetzt werden, ohne dass diese Toleranzen überschritten werden. Es gibt jedoch in einigen Fällen eine "Null"-Toleranz (bei Milch besteht eine "Null"-Toleranz für Insektizide aller Art) und das Problem wird komplizierter.

References

BACHE, C. A., G. G. GYRISCO, S. N. FERTIG, E. W. HUDDLESTON, D. J. LISK, F. H. COX, G. W. TRIMBERGER, and R. F. HOLLAND: Effects of feeding low levels of heptachlor epoxide on residues and off-flavors in milk. J. Agr. Food Chem. 8, 408 (1960).

BRUCE, W. N., and G. C. DECKER: Fly control and milk flow. J. Econ. Entomol. **40**, 530 (1947).

BUTTRAM, J. R., and B. W. ARTHUR: Absorption and metabolism of Bayer 22408 by dairy cows and residues in milk. J. Econ. Entomol. **54**, 446 (1961).

CHENG, Tieu Hsi, D. E. H. FREAR, and H. F. ENOS, JR.: Fly control in dairy barns sprayed with dimethoate and the determination of dimethoate residues in milk. J. Econ. Entomol. 54, 740 (1961).

—, —, — The use of spray and aerosol formulations containing R-1207 and dimethoate for fly control on cattle and the determination of dimethoate residues in milk. J. Econ, Entomol. **55**, 39 (1962).

CLABORN, H. V., R. C. BUSHLAND, H. D. MANN, and M. C. IVEY: Meat and milk residues from livestock sprays. J. Agr. Food Chem. 8, 439 (1960 a).

—, R. D. RADELEFF, and R. C. BUSHLAND: Pesticide residues in meat and milk. *U. S. Department of Agriculture,* ARS-33-63, 46 pp. (1960 b).

—, R. H. ROBERTS, H. D. MANN, M. C. BOWMAN, M. C. IVEY, C. P. WEIDENBACH, and R. D. RADELEFF: Residues in body tissues of livestock sprayed with Sevin or given Sevin in the diet. J. Agr. Food Chem. **11**, 74 (1963).

CLUETT, M. L., W. K. LOWEN, H. L. PEASE, and C. A. WOODHOUSE: Determination of methoxychlor and/or metabolites in milk following topical application to dairy cows. J. Agr. Food Chem. 8, 277 (1960).

EHEART, J. F., E. C. TURNER, and J. DICKINSON: Residues of Sevin in whole milk from sprayed and dusted cows. J. Econ. Entomol. **55**, 504 (1962).

FERTIG, S. N., and M. M. SCHREIBER: Effect of Dalapon ingestion on performance of dairy cattle and levels of residue in the milk. J. Agr. Food Chem. **9**, 369 (1961).

GANNON, N., and G. C. DECKER: The excretion of dieldrin, DDT, and heptachlor epoxide in the milk of dairy cows fed on pasture treated with dieldrin, DDT, and heptachlor. J. Econ. Entomol. **53**, 411 (1960).

GYRISCO, G. G., D. J. LISK, S. N. FERTIG, E. W. HUDDLESTON, F. H. FOX, R. F. HOLLAND, and G. W. TRIMBERGER: The effects of feeding high levels of Sevin on residue, flavor, and odor of the milk of dairy cows. J. Agr. Food Chem. 8, 409 (1960).

HOWELL, D. E., H. W. CAVE, V. G. HELLER, and W. G. GROSS: The amount of DDT found in milk of cows following spraying. J. Dairy Sci. **30**, 717 (1947).

IVEY, M. C., H. V. CLABORN, H. D. MANN, R. D. RADELEFF, and G. T. WOODARD: Aldrin and dieldrin content of body tissues of livestock receiving aldrin in their diet. J. Agr. Food Chem. **9**, 374 (1961).

JACKSON, M. B., R. D. RADELEFF, R. H. ROBERTS, L. M. HUNT, and W. B. BUCK: Acute toxicity of Delnav and its residues in tissues of livestock. J. Econ. Entomol. **55**, 699 (1962).

KUTSCHINSKI, A. H.: Form and magnitude of 2,2-dichloropropionic acid (Dalapon) residues in milk. J. Agr. Food Chem. **9**, 365 (1961).

LAAKE, E. W.: DDT for the control of horn flies in Kansas. J. Econ. Entomol. **39**, 65 (1946).

LONG, W. H., L. D. NEWSOM, and A. M. MULLINS: Endrin residues in the fat of lambs grazed on endrin-treated pasture. J. Econ. Entomol. **54**, 605 (1961).

LYNN, G. E., S. A. SHRADER, O. H. HAMMER, and C. A. LASSITER: Occurrence of bromides in the milk of cows fed sodium bromide and grain fumigated with methyl bromide. J. Agr. Food Chem. **11**, 87 (1963).

PLAPP, F. W., JR., W. S. BIGLEY, and D. L. DANROW: Studies on the metabolism and residues of P-32 labelled Delnav in a Hereford steer. J. Econ. Entomol **53**, 60 (1960).

RADELEFF, R. D., and H. V. CLABORN: Excretion of Co-Ral in the milk of dairy cattle. J. Agr. Food Chem. 8, 437 (1960).

ROBERTS, R. H., and R. D. RADELEFF: Toxaphene residues in hogs. J. Econ. Entomol. **53**, 322 (1960).

— —, and H. G. WHEELER: Malathion residues in the tissues of sheep, goats, and hogs. J. Econ. Entomol. **53**, 972 (1960 a).

—, J. B. JACKSON, W. E. WESTLAKE, A. J. ACKERMAN, and H. V. CLABORN: Residue studies of livestock sprays containing Sevin. J. Econ. Entomol. **53**, 326 (1960 b).

SMITH, C. T., F. SHAW, R. LAVIGNE, J. ARCHIBALD, H. FENNER, and D. STERN: Residues of malathion on alfalfa and in milk and meat. J. Econ. Entomol. **53**, 495 (1960).

WESTLAKE, W. E., C. CORLEY, R. T. MURPHY, W. F. BARTHEL, H. BRYANT, and R. L. SCHUTZMANN: Chemical residues in the milk of cows grazed on chlordan-treated pastures. J. Agr. Food Chem. **11**, 244 (1963).

ZWEIG, G., L. M. SMITH, S. A. PEOPLES, and R. COX: DDT residues in milk from dairy cows fed low levels of DDT in their daily rations. J. Agr. Food Chem. **9**, 481 (1961).

—, L. PYE, and S. A. PEOPLES: Residues in butterfat and body fat of dairy cows fed at two levels of Kelthane (1.0 and 2.0 ppm). J. Agr. Food Chem. **11**, 72 (1963 a).

— —, SITLANI, and S. A. PEOPLES: Residues in milk from dairy cows fed low levels of toxaphene in their daily ration. J. Agr. Food Chem. **11**, 70 (1963 b).

Iodine-131 in foods

By

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Contents

I. Introduction

Iodine is one of the 64 elements that make up 0.4 percent of the earth's mass and occurs in an abundance of 0.3 p.p.m. (*Chilean Iodine Educational Bureau* 1956). Although there are iodine-deficient areas in the world, traces of this element can be found in practically all plant and animal life. The significance of iodine in the metabolism of animals and man has been well established. The traditional sources of iodine in the diet are primarily the marine fishes and certain vegetables and fruits, including asparagus, cabbage, green beans, potatoes, lettuce, grapes, pears, and pineapple. On the other hand, foods derived from terrestrial animal sources, such as milk, eggs, and meat, are relatively low in iodine.

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The present paper reviews the available data[1] on physical and chemical characteristics of iodine-131 (^{131}I) in fallout, behavior in the environment, levels observed in milk, and methods to reduce the levels in milk.

A superficial consideration of ^{131}I would lead one to the conclusion that the radioelement would be distributed in much the same way as the stable iodine, but this indeed is not the case. The several investigations made to determine ^{131}I content of the American diet clearly show that the presence of this radionuclide is transient and that the only significant source is fresh fluid milk (EISENBUD *et al.* 1962). In countries such as Japan, where fluid milk makes up only a minor portion of the diet, exposure to ^{131}I comes largely through the consumption of leafy vegetables (YAMAGATA and IWASHIMA 1962). The major pathways followed by ^{131}I from the atmosphere to man are summarized in Figure 1.

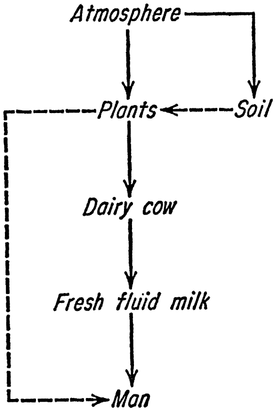

Fig. 1. Pathways of iodine-131 from atmosphere to man: — major route, - - - minor route. Source: nuclear weapons testing and reactor discharges

When ingested, ^{131}I is concentrated in the thyroid gland. Similar concentrations give a higher dosage to the smaller thyroids of children, because of size and sensitivity, than to those of adults. Although levels of ^{131}I higher than those found in the environment are used for diagnostic and therapeutic purposes, the significance of relatively low levels in the environment from nuclear weapons testing fallout or reactor operations has not been clearly elucidated.

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[1] Radiochemical terminology and symbology in text conform to *National Bureau of Standards* recommendations. Throughout this review *Ci* = Curie, *mCi* = milli-Curie, *μCi* = microCurie, and *pCi* = picoCurie; also, *rad* is the unit of absorbed dose in terms of 100 ergs/gram in any medium, and *rem* is Roentgen equivalent man.

II. Sources of production

The radionuclides of iodine are produced in several different ways. By far the largest *uncontrolled* source of radioiodine is the detonation of nuclear devices either in weapons tests or in possible beneficial applications such as the "Plowshare" programs. The largest *controlled* source of radioiodine production is in the fission of uranium or plutonium in nuclear reactors. Radioiodine also results from the activation of specific elements by neutron bombardment in reactors, by heavy nuclei particles in accelerators, or by other high energy devices.

Table I. *Kinds of iodine radionuclides*

Mass number	Half-life[a]	Fission yield (%)		Mass number	Half-life[a]	Fission yield (%)	
		Indep't	Chain[b]			Indep't	Chain[b]
117	~ 10 min.	None	None	129	1.6x10^7 yr.	0.0	0.8
118	17 min.	None	None	130	12.5 hr.	0.0	0.0
119	17 min.	None	None	131	8.08 days	0.0	2.9
120	1.4 hr.	None	None	132	2.3 hr.	0.2	4.4
121	1.5 hr.	None	None	133	20.8 hr.	0.6	6.6
122	3.5 min.	None	None	134	52.5 min.	2.2	7.8
123	13 hr.	None	None	135	6.7 hr.	2.9	5.5
124	4.5 days	None	None	136	86 sec.	2.9	3.9
125	60 days	None	None	137	22 sec.	2.2	2.7
126	13.3 days	None	None	138	5.9 sec.	1.3	1.5
127	Stable	0.0	0.1	139	2.7 sec.	0.8	0.8
128	25 min.	0.0	0.0	140	~ 1.5 sec.	0.3	0.3

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[a] SULLIVAN 1957.

[b] WEAVER *et al.* 1963. For thermal neutron fission of uranium-235 chain, yields do not include isobars of atomic number greater than 53(I).

The radioiodines (see Table I) produced by each of these operations vary widely in kind and amount (HOLLAND 1963). For example, several radioiodines produced during fission include ^{129}I, ^{131}I, ^{132}I, ^{133}I, ^{134}I, and ^{135}I. Some of them eventually find their way into the environment. Any or all may be liberated during nuclear tests or may be found in the accidental discharges from nuclear reactors. In a nuclear test, the principal contributor during the first day is ^{133}I followed by ^{131}I, whereas when a reactor is at equilibrium, ^{134}I is most abundant, with ^{132}I being most abundant at one day and ^{131}I predominating after two days. In accidental releases, ^{133}I, with a half-life of 20.8 hours, may also be of some significance as a contaminant of the environment and food of man.

In chemical reprocessing of fuel elements, the iodine released depends on the age of the fuel elements before reprocessing and the iodine-retention efficiency of waste-treatment devices before release. Only ^{131}I, with its half-life of 8.08 days, may be of any consequence from chemical reprocessing.

Iodine-131 could also be found in the effluents from hospitals where it had been used for diagnostic and therapeutic purposes, but the amount released and its possible effects on the environment would be minimal.

III. Forms of iodine-131

Iodine reacts with many of the trace substances normally encountered in the atmosphere and with many of the inorganic and organic compounds found in the terrestrial environment. Although solid at ordinary temperature, elemental iodine has a high vapor pressure and sublimes readily. It normally dissolves in water droplets to form iodide and hypoiodate ions, especially in the presence of alkaline impurities. It is reduced readily to iodide by hydrogen sulfide, and can in turn be oxidized to free iodine by ozone, hydrogen peroxide, or possibly by air and sunlight. If the iodine is acidified sufficiently, gaseous hydrogen iodide is liberated. Those chemical forms considered to be relatively common are identified in Table II.

Table II. *Chemical forms of iodine* (HOLLAND 1963)

Valence	Common chemical species
-1	I^-, HI, NaI, HI_nH_2O
0	I_2
$+1$	$IC1$, IBr, HOI
$+5$	I_2O_5 IO_3^-, HIO_3, $NaIO_3$
$+7$	IO_4^-, HIO_4, $NaIO_4$
Organic	CH_3I, CH_2I_2, CHI_3, C_2H_5I, $C_2H_4I_2$

The chemical forms of iodine in the environment vary markedly, and in many instances identification of the particular form present is quite difficult. Studies have been carried out to identify the form present in both nuclear debris and the waste effluents from chemical reprocessing plants. As a first step, the usual practice is to separate the radioiodine into two physical fractions, that associated with particulate matter of a specific size and that which may be chemically absorbed or adsorbed by various chemically treated materials or charcoal filters. In addition, there is a third fraction that goes through all of these materials; it is generally considered to be in some organic form.

Another form of separation results when the iodine associated with particles or in the gaseous form is solubilized in water or acid. Studies show that about a third (1/10 to ½) of the ^{131}I, on the average, can be leached out of the particulate fraction in four hours (KEISH and KOCH 1963). The ^{131}I in the leach solution occurs principally as iodide ion (36 to 80 percent) or iodate ion (15 to 55 percent), with a slight preference in favor of the iodide. Use of this technique, however, does not differentiate between iodine and iodide since both appear in the leach as the iodide. These data show that the elemental, reduced, and oxidized states have a significant probability of occurrence. Only the periodate appears relatively rare. No information was available on the chemical form of the 50 to 90 percent of ^{131}I remaining in the particles after leaching. Similar distributions were observed for precipitation samples; however, there seems to be a greater predisposition toward iodide. Here the percentage of ^{131}I in the aqueous phase varied from 23 to 65 percent, that in the settled fraction from 28 to 56 percent, and that in the suspended, fine particulates from six to 25 percent (KEISH and KOCH 1963).

Additional data on the physical and chemical forms of iodine given by PERKINS (1963) show that a large and varying amount of ^{131}I is in the gaseous state, presumably in the reduced form of I_2, hydrogen iodide, or organic acids (Table III). During 1962, about half (ten to 90 percent) of the ^{131}I collected from air passed through the membrane filter used for particle separation and was collected in the charcoal trap.

Table III. *Chemical forms of iodine-131 collected on membrane filters* (PERKINS 1963)

Date	^{131}I leached from filters (%)	Fractionation of leached ^{131}I (%)		
		$I_2 + I^-$	IO_3^-	IO_4^-
10/24/62	44	~ 57	~ 38	< 5
10/25/62	42	~ 66	~ 29	< 5

If the assumption is made that the nonleached fraction of iodine on particulates has the same chemical form as that combined in the leached fraction, about 80 to 85 percent of the fallout ^{131}I is in the reduced state, 15 to 20 percent is present as IO_3^-, and a few percent or more is present as IO_4^-.

Studies at Hanford (PERKINS 1963) with stack discharges of ^{131}I showed the fraction associated with particulates to increase with distance from the source. At one mile 12 percent was associated with the particulate matter, at three miles eight percent, at five miles 20 percent, at ten miles 38 percent, and at 25 miles 34 percent. Ground level measurements taken at the same time showed about 60 percent of the normal fallout radioiodine associated with particular matter. PERKINS (1963) feels that very little, if any, of the gaseous compounds of ^{131}I found exist as elemental iodine or hydrogen iodide.

Evidence is also accumulating (LANE 1963) that ^{131}I sublimes after deposition and can be recycled via the atmosphere.

IV. Transport mechanisms

According to MACHTA (1963), three meteorological processes account for the transport of ^{131}I from the source to the site of deposition: (1) drift of nuclear clouds from low-level tests, (2) transport debris to surface of southern edge of a high-pressure system (subsiding anticyclones), and (3) scavenge by precipitation. Low-altitude drift permits dry depositions, but there is no need to exclude precipitation. The importance of rainfall is illustrated by the data shown in Figure 2, which relates radionuclide levels in air and milk in the St. Louis area from September through December, 1961. The initial high ^{131}I levels observed in milk resulted from the deposition of dry fallout around September 18 to 21, 1961.

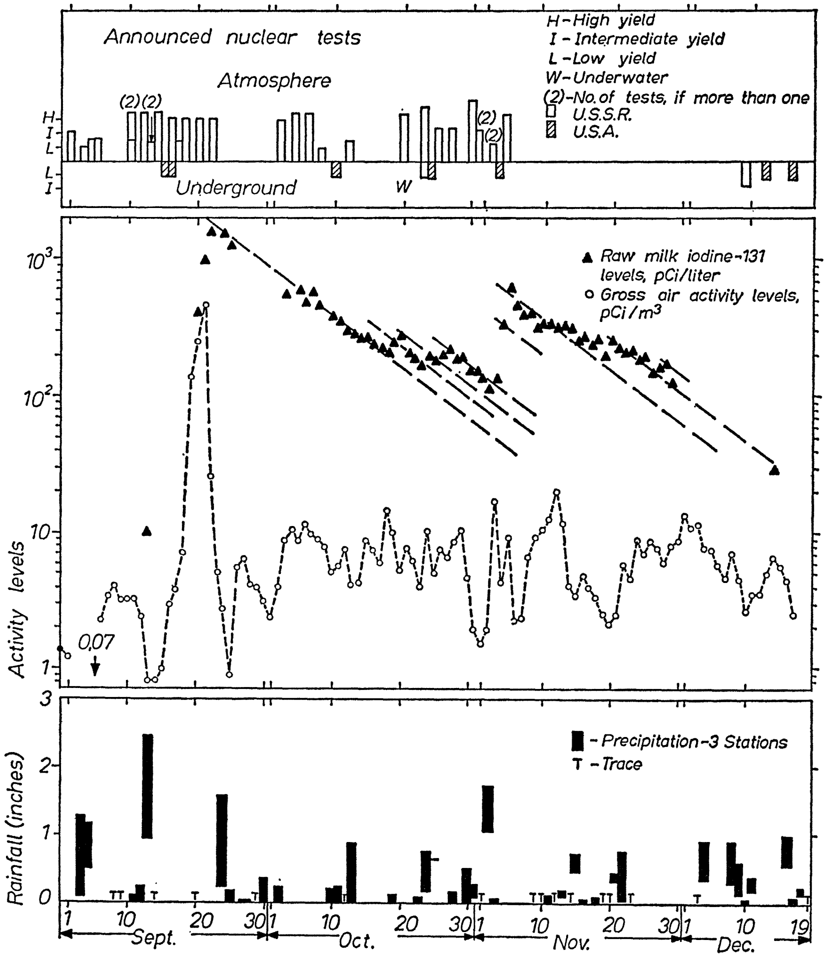

Fig. 2. Environmental data for a portion of the St. Louis milkshed

Scavenging of particulates by precipitation may also take place from high altitudes. During May, 1962 the lower stratosphere over the United

States contained debris from Christmas Island tests, and intense nocturnal thunderstorms scavenged debris from high altitudes. Radar showed some

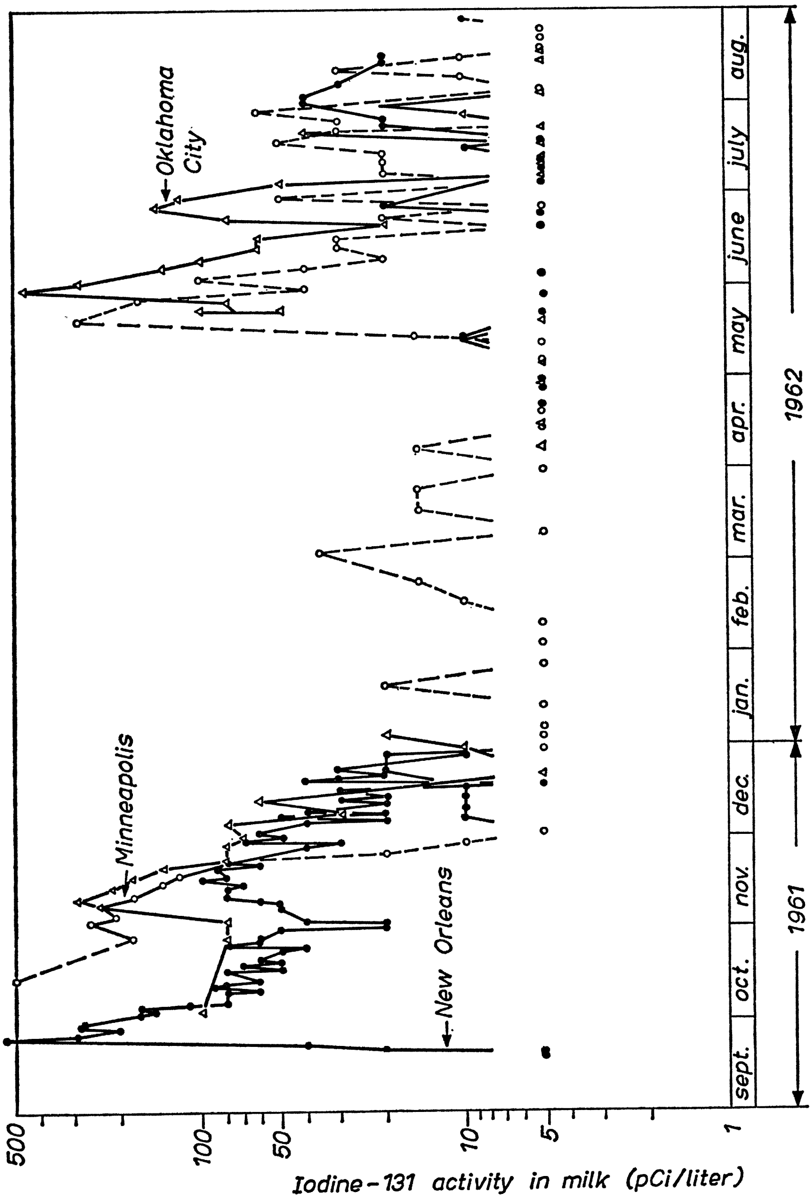

Fig. 3. Iodine-131 levels in milk at Minneapolis, Minnesota, Oklahoma City, Oklahoma, and New Orleans, Louisiana, September 1961 to August 1962

cloud tops at 10,000 feet (3,050 meters) and others as high as 60,000 feet (18,300 meters) during these storms. Evidence of deposition of ^{131}I from this phenomenon is shown in Figure 3 for Oklahoma City in the rainy months of May and June, 1962. Figure 3 also indicates that deposition of ^{131}I and its identity in milk is a highly random phenomenon that depends upon many factors, the most important of which may be local meteorological and climatological conditions.

V. Entry into food chain and metabolism

The distribution of ^{131}I in food depends upon the time the isotope is produced, its presence in the environment, and the degree of contamination. As indicated earlier, the foods that are considered potential dietary sources of ^{131}I are milk and leafy vegetables, and some fruits produced and consumed locally.

Other than perhaps during the period of the formation, the only other time in the history of the earth that it has been exposed to appreciable quantities of ^{131}I was during the periods of active weapons tests from 1951 to 1958 and from 1961 to 1962. During most of 1951 to 1958, however, ^{131}I was regarded as a relatively innocuous fission product compared to strontium-90 (see DAVIS 1965), and the mechanism of ^{131}I transfer from fallout to man was of little interest. At the time of the moratorium on testing, understanding of the chemical forms of ^{131}I in fallout, the importance of rain in its deposition, and the importance of foliar deposition in contrast to root uptake was only superficial. The potential hazards of ^{131}I were not generally recognized until after the calculations of LEWIS (1959) relating the radiation dose to the thyroid of children with the consumption of milk during periods of weapons tests. Since that time, much new information has been developed from studies of the additional amounts of ^{131}I released into the environment from weapons testing during 1961 to 1962 and from chemical separation operations at Hanford, Washington, Aiken, South Carolina, Idaho Falls, Idaho, in the United States and at Windscale in England. Yet many questions concerning the fate of ^{131}I in fallout from atmospheric nuclear explosions remain unanswered.

In a comprehensive series of theoretical and practical investigations, CHAMBERLAIN (1961) developed a basis for quantitative consideration of the deposition of ^{131}I and introduced the concept of velocity of deposition, or V_g, expressed as cm./sec. This parameter is defined as

$$V_g = \frac{\text{Rate of deposition on ground } (\mu Ci/\text{m.}^2/\text{sec.})}{\text{Volumetric concentration in air } (\mu Ci/\text{m.}^3)}\,.$$

This concept is applicable to dry deposition of ^{131}I vapor and to particulate forms provided the particle sizes are sufficiently small to have settling velocities approaching zero.

CHAMBERLAIN has also summarized the deposition velocities calculated during the Windscale accident, in connection with operations at a number of chemical separation plants, and from field experiments (CHAMBERLAIN 1961, CHAMBERLAIN and CHADWICK 1953, BARRY and CHAMBERLAIN 1963) and has found the values to be from 0.3 to 2.8 cm./sec.

Although the importance of the scavenging action of rain in connection with the deposition of ^{131}I is generally recognized, few systematic studies have been made in this connection. PIERSON and KEANE (1962) evaluated this phenomenon and developed a concept of a rainout factor, W_g, defined as

$$W_g = \frac{\text{Concentration in rain } (\text{p}Ci/\text{kg.})}{\text{Concentration in air near ground level } (\text{p}Ci/\text{kg.})}$$

These observations indicate W_g to be in the neighborhood of 500.

In an effort to make the concept of deposition velocity more directly applicable to the deposition of ^{131}I on plants, BARRY and CHAMBERLAIN (1963) investigated some of the factors influencing the uptake of ^{131}I by plants (*Vicia faba*). Their results demonstrated that the rate of ^{131}I vapor absorbed on the leaves is controlled by the rate of diffusion of the gas through the stomata on the leaf surface. Under humid conditions, which favor stomatal opening, the rate of uptake of ^{131}I vapor was about ten times greater than under dry conditions, which favor stomatal closure.

In a series of studies on foliar absorption of ^{131}I by plants HUNGATE *et al.* (1963) observed that the amount of ^{131}I vapor absorbed was proportional to the concentration of the ^{131}I vapor in the surrounding air throughout a wide range of concentration. Subsequent to exposure they found only a minor (about five percent) translocation from leaves to other portions of the plants. In a field experiment simulating a nighttime reactor accident, dispersal of melted fuel elements was made upwind of the plants to be exposed. About ten percent of the ^{131}I was found on the leaves; a little more than half of it could easily be removed by washing.

Although the ground surface was also contaminated, a very low level of activity was found in the root portion of radishes used in the study. These results reinforce the laboratory data on the low rate of translocation and also suggest that uptake by the root system is not an important mechanism.

Although ^{131}I has a physical half-life of 8.08 days, its half-life on contaminated vegetation is approximately 5.5 days. Experimental evidence developed in connection with this observation (MARTIN 1963) indicates that this difference may be due either to mechanical removal of the particles from the vegetation or by volatilization after deposition.

The uptake of ^{131}I from environmental sources by animals is largely from ingestion of contaminated food. A small proportion may be incorporated through inhalation (WEHMAN 1963). Once past the gut barriers and in a soluble form, ^{131}I is metabolized in the same manner as the stable element, i.e., the vast majority is concentrated in the thyroid glands and lesser quantities are deposited in salivary glands, stomach, and skin. Iodine-131 is eliminated from the animal through a number of routes including exhaled air, urine, feces, and milk.

In view of the importance of fresh, fluid cows' milk as a source of ^{131}I in the diet of man, a vast amount of experimental work has been conducted on the uptake, metabolism, and excretion of ^{131}I by dairy cattle (BUSTAD *et al.* 1963, GARNER 1959 and 1960, GARNER *et al.* 1960, GLASSOCK 1954, LENGEMANN and SWANSON 1957, LENGEMANN and COMAR 1964, MILLER and SWANSON 1963, SQUIRE *et al.* 1959, STEENBERG 1959). The most relevant studies for extrapolation to metabolism of ^{131}I from environmental sources are those of BUSTAD *et al.* (1963) and of LENGEMANN and COMAR (1964) in which cows were dosed with ^{131}I twice daily for an extended period rather than with a single dose. In the latter work, samples of milk and blood were taken at regular intervals and thyroid counts were made by use of a scintillation probe. After seven days of experiment, the ^{131}I in the thyroid was 70 percent of the daily dose and in one liter of milk, one percent. The milk-to-plasma ^{131}I ratio was 2:3. Approximately five percent of the ^{131}I of the milk was found to be protein bound. The concentration of ^{131}I in the milk reached a maximum after three days of dosing. The data also showed that the level of stable iodine in the diet has an influence on the movement of ^{131}I into milk.

MILLER and SWANSON (1963) have investigated the transfer of ^{131}I from blood to milk and ^{131}I metabolism in the udder. Their findings indicate that ^{131}I passes easily in both directions between the cows' mammary gland and her blood, and that it can enter the milk independently of the secretory process. Protein-bound iodine does not appear to cross the membranes but is rather synthesized in the udder. Observation that the higher ^{131}I secretion by high-yield cows is primarily a function of the milk volume is consistent with the findings of LENGEMANN and COMAR (1964).

Considerable attention has been given to determine the relationship between ^{131}I in pasture grass and in milk of the dairy cow (SOLDAT 1963, GARNER 1960). HULL (1963) carried out one of the more definitive studies in this connection in the immediate environs of the Brookhaven National Laboratory. Deposition velocity of approximately 0.5 cm./sec. and a rainfall factor on grass of 0.5 were observed, and the values for the average ^{131}I concentration in grass and in milk were found to be one µCi ^{131}I/kg. of grass to 0.07 µCi ^{131}I/liter of milk. These values are in approximate agreement with those found in connection with the Windscale incident.

VI. Sampling and sampling techniques

a) Milk

The method used for collecting milk samples for ^{131}I analysis depends to a large extent upon whether they are intended to determine relationships between environmental media such as air, water, and precipitation and milk, or to relate ingestion through milk and man.

To estimate fallout contamination for surveillance purposes, raw milk from the farm (CAMPBELL *et al.* 1959) or processed milk from milk plants (*U.S. Department of Health, Education, and Welfare* 1961, 1962, and 1963) was collected. Raw milk samples were collected from discrete geographical areas composed of a number of individual farms. A minimum of 1,000 cows was included so that collection of collateral field data such as feeding practices, water supply, and breeds of cattle for each farm was feasible. The milk samples were from a supply that was part of a metropolitan milkshed, and the conditions under which milk was received were such that each sample was representative of the same groups of farms in the production area.

Processed milk collected in a major metropolitan city is a composite of sub samples from each processing plant, collected in proportion to the plant's average sales in the community served. At most stations the sample represents 80 to 100 percent of the milk processed.

Sampling of milk around nuclear facilities may be from herds grazing within the expected range of contamination, usually ten miles or 20 to 30 miles, depending upon the wind direction. A random pooled sample is obtained one day each week for immediate gamma-ray spectrometric examination.

Usually one gallon of milk is collected, preserved with a suitable preservative (formaldehyde, merthiolate, etc.), and shipped to laboratories for analysis.

Earlier surveillance studies by CAMPBELL and MURTHY (1963) indicated that single observations made at monthly intervals would not represent the actual levels during the intervening period and that during active fallout samples should be collected frequently for analysis (Fig. 4). Based on this observation milk samples were collected once a week and in periods of increased fallout, twice a week, or even every day. Where problems of high levels are observed, special projects are established in cooperation with the local agencies concerned to assess the problem. All surveillance data are subject to continuing review and evaluation to observe unusual patterns that may require immediate attention.

When the general fallout is heavy and localized, the effect of ^{131}I on thyroids of children is usually assessed. Under such circumstances samples from individual milk plants may have to be monitored. Since the fallout deposition is not uniform, the milk received by plants may vary accordingly and, therefore, contamination to various degrees is possible. Limited studies were made (EISENBUD *et al.* 1963) in New York City during 1962 to determine variability of ^{131}I concentration among 21 milk plants on four separate days. The results show that the variations from dairy to dairy were random in nature, and therefore, the claim is made that at the end of an extended period of sampling the mean concentrations (180 ± 20 pCi/liter) of the milk from the various dairies would have been the same. The variability of samples within a single dairy and the variability observed among 28 samples from all the plants on the same day ranged from 130

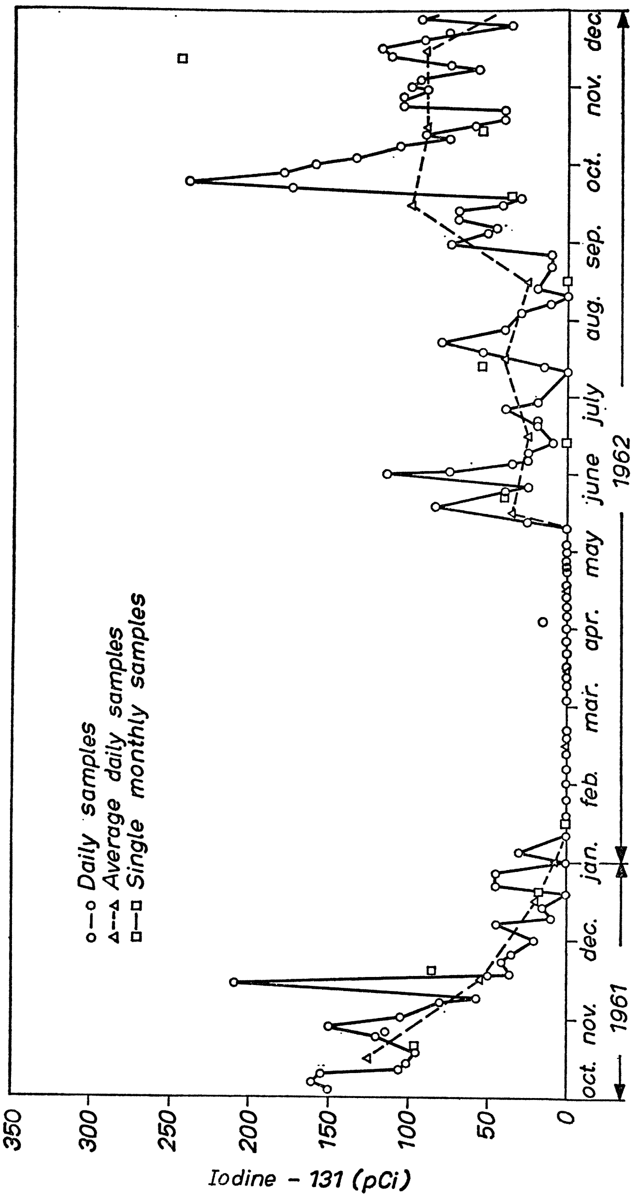

Fig. 4. Influence of sampling frequency on observed concentration of iodine-131 in Cincinnati, Ohio

to 480 pCi/liter. The average was 230 ± 76 pCi/liter and the coefficient of variation was 32.5 percent. More study is needed in this regard.

To determine the relationship between air, vegetation, and precipitation with milk, areas of study are selected either prior to any indication of increase in airborne radioactivity or during times of fallout and immediately following reactor accidents. Representative samples of air, precipitation, vegetation, and milk are collected simultaneously until no significant radioactivity is observed.

b) Vegetation and other environmental samples

The collection of vegetation or other samples for determination of ^{131}I is considered pertinent inasmuch as, in most cases, the presence of other fission products will obscure the ^{131}I present and one must resort to the longer and expensive radiochemical separation techniques for ^{131}I. Only in the case of an accident wherein ^{131}I and some other radionuclides are released preferentially, and are present in high concentration, would it be possible to use simple gamma-ray counting equipment to measure the increased radioactivity in air or vegetation samples.

VII. Nature of iodine-131 in milk

Iodine-131 may be present in milk in the inorganic form as the iodide, or it may be bound with the protein. Tracer studies have shown that ^{131}I appears mostly in the inorganic form in human milk (HONOUR *et al.* 1952), and in goat and rabbit milk (WRIGHT *et al.* 1955). In dogs' milk (VAN MIDDLESWORTH *et al.* 1953) 30 to 60 percent is protein bound, and in lactating rats (POTTER and CHAIKOFF 1956) 86 percent is protein bound. In milk from cows injected with a single dose of carrier free ^{131}I, all the iodine was in the inorganic form (GLASSOCK 1954), whereas in the milk from cows dosed continuously with carrier free ^{131}I approximately 81.7 to 91.4 percent was in the inorganic form, 4.7 to 13.1 percent was protein bound, and less than 0.1 percent was associated with the fat. The distribution of ^{131}I between inorganic and protein fractions was affected by the amounts of stable iodine in the cows' diet (LENGEMANN and SWANSON 1957). In market milk contaminated with fallout from nuclear weapons testing (MURTHY *et al.* 1962, MURTHY 1963, *U.S. Department of Health, Education, and Welfare* 1961, 1962, and 1963) or from a reactor accident (DUNSTER *et al.* 1958), all of the ^{131}I was present in the inorganic form as the iodide.

VIII. Concentration of iodine-131 in milk

As indicated earlier, milk is the major source by which ^{131}I is ingested by children. Consequently attempts are made to relate the observed concentration of ^{131}I in milk to the dosage of thyroids. Some discussions are therefore included in this regard while reviewing ^{131}I levels in milk.

Table IV. *Concentration of iodine-131 in milk* (CAMPBELL 1961)

Month (s)	No. of weapons tests reported		Concentration (pCi/liter)								
	Continental U.S.	Elsewhere	New York	Cincinnati	St. Louis	Salt Lake City	Sacramento	Atlanta	Austin	Chicago	Spokane
1957											
Jan.-Apr.	0	11	—	—	—	—	—	—	—	—	—
May	1	3	—	—	—	—	—	—	—	—	—
June	4	1	40	370	410	440	250	—	—	—	—
July	4	0	—	—	—	—	—	—	—	—	—
Aug.	4	1	50	230	210	200	10	—	—	—	—
Sept.	7	3	300	430	970	740	10	—	—	—	—
Oct.	1	3	250	300	890	990	20	—	—	—	—
Nov.	0	1	100	<10	<10	<10	<10	—	—	—	—
Dec.	0	1	<10	<10	<10	50	<10	—	—	—	—
1958											
Jan.	0	0	<10	<10	<10	<10	<10	—	—	—	—
Feb.	0	2	<10	<10	<10	40	<10	—	—	—	—
Mar.	0	7	<10	<10	<10	<10	<10	—	—	—	—
Apr.	0	2	50	30	100	30	10	—	—	—	—
May	0	3	30	<10	<10	<10	<10	—	—	—	—
June	0	6	<10	<10	<10	<10	240	—	—	—	—
July	0	5	70	110	625	155	45	20	<10	125	—
Aug.	0	4	70	40	60	80	20	65	50	45	55
Sept.	7	6	10	<10	20	15	<10	20	15	15	10
Oct.	24	12	60	30	35	10	10	<10	10	20	<10
Nov.	0	2	35	180	125	35	15	30	130	55	65
Dec.	—	—	<10	<10	25	<10	15	20	40	<10	30

In an earlier study (CAMPBELL *et al.* 1959) raw milk samples were collected on a monthly basis from nine discrete geographical areas around New York City, Cincinnati (Ohio), St. Louis (Missouri), Salt Lake City (Utah), Sacramento (California), Atlanta (Georgia), Austin (Texas), Chicago (Illinois), and Spokane (Washington). In general, the ^{131}I data (Table IV) showed a positive correlation between the announced nuclear weapons tests conducted in the United States and high concentrations of ^{131}I in milk from all stations except Sacramento. The Sacramento area is, of course, in the opposite direction from the prevailing west-to-east movement of air over the United States. The short-lived ^{131}I circling the globe decayed or dispersed before reaching Sacramento. The levels of ^{131}I and dates of mainland weapons tests appeared to be directly related. Analysis of these data led to the conclusion that collecting single monthly milk samples is unsatisfactory for monitoring ^{131}I. This conclusion was reaffirmed during a period of time (October to November, 1961) when the frequency of sampling was greatly increased. Experience derived from the earlier raw milk sampling network led to the activation of the Pasteurized Milk Sampling Program which consisted of collecting from 62 stations.

During 1957-1958 dose rates to thyroids of young children in the United States averaged 0.1 to 0.2 rad/year (LEWIS 1959). From the same measurements the estimated dose rate of 1.8 rads to infants of St. Louis for May 1957 to April 1958 has been made (KNAPP 1960).

After the resumption of nuclear weapons testing by the Soviet Union in September 1961, and because in the meantime the significance of implications of ^{131}I to health of children had been emphasized by the *International Committee on Radiological Protection* and the *Federal Radiation Council*, many of the surveillance groups initiated programs to determnie ^{131}I levels in various environmental media.

Since then numerous data on ^{131}I levels in milk have become available (BENNINSON and RAMOS 1962, *Canadian Department of National Health and Welfare* in the *U.S. Department of Health, Education, and Welfare* 1961, 1962, and 1963, CIGNA and POLVANI 1962, GRUMMIT and KAMATH 1963, JEANMARIE and MICHON 1962, MODELMONT and MICHON 1964, MOISIO and MEITTIEN 1963, and various releases from the *U.K. Atomic Energy Authority* during 1961–1962). The data in general vary widely with the location of the sampling point in relation to the test site and with meteorological and climatological factors.

When the high radioactivity levels during September 1961 became apparent, the airborne particulates were found to pass from the Northeastern region of the United States southward to the Gulf Coast and up through the Mississippi-Missouri valley. The ^{131}I levels in the milk collected along the Mississippi-Missouri valley were highly variable (see Fig. 3), but ^{131}I was present throughout the area. During the winter months ^{131}I levels in milk occasionally reached a maximum of 40 pCi/liter in milk from northern states. No ^{131}I was observed in milk from southern states.

Although several underground tests were conducted in the United States from December 10, 1961 through April 24, 1962 the observed values in the milk from northern states could not have been due to these tests, but rather to the exposure of cows to feed probably contaminated with fallout prior to December 1961.

Of particular interest are the data obtained from a portion of the milkshed in St. Louis area (see Fig. 2). The relationship between radioactivity levels in air, precipitation, and milk are there illustrated. Analysis of data indicated that most of the radioactivity deposited initially was from air intrusion; very little, if any, was by rain. The ^{131}I values in milk reached a maximum of 1,600 pCi/liter on September 22, 1961. For the next 26 days the level appeared to decrease at a rate comparable to a half-life of eight days; therefore a single high air intrusion on September 18 was indicated. No milk samples were collected, however, between September 26 and October 1. The literature shows the half-life of ^{131}I in milk from a single intrusion to be about 4.5 days. Since no significant radioactivity was deposited by rain and the ^{131}I levels in milk decreased with a half-life of eight days, the contribution from inhalation of air by cows cannot be ignored. Evidence to support this conclusion is lacking, however.

As shown in Figure 5, ^{131}I maximum concentrations in individual milk samples from the Pasteurized Milk Network in the United States varied considerably from September 1961 through December 1963. The variation is probably due to meteorological conditions. As pointed out previously, a cause-and-effect relationship exists between the penetration of thunderstorms into high concentrations of nuclear debris in the lower stratosphere and the subsequent ^{131}I in milk.

The probable sources of ^{131}I contamination recorded by the Pasteurized Milk Network are listed in Table V. The high levels of ^{131}I in milk pro-

Table V. *Sources of iodine-131 contamination $\geqq$ 300pCi/liter of milk in Public Health Service Pasteurized Milk Network* (LIST *et al.* 1964)

No. of milksheds	Probable source
13	Sept. to Dec. 1961 U.S.S.R. atmospheric tests
5	May to early June 1962 U.S. Christmas Island atmospheric tests
1	Late June 1962 Vented Nevada underground test
2	July to Aug. 1962 Nevada atmospheric and cratering tests
6	Sept. 1962 U.S.S.R. atmospheric tests
8	Nov. to Dec. 1962 U.S.S.R. atmospheric tests

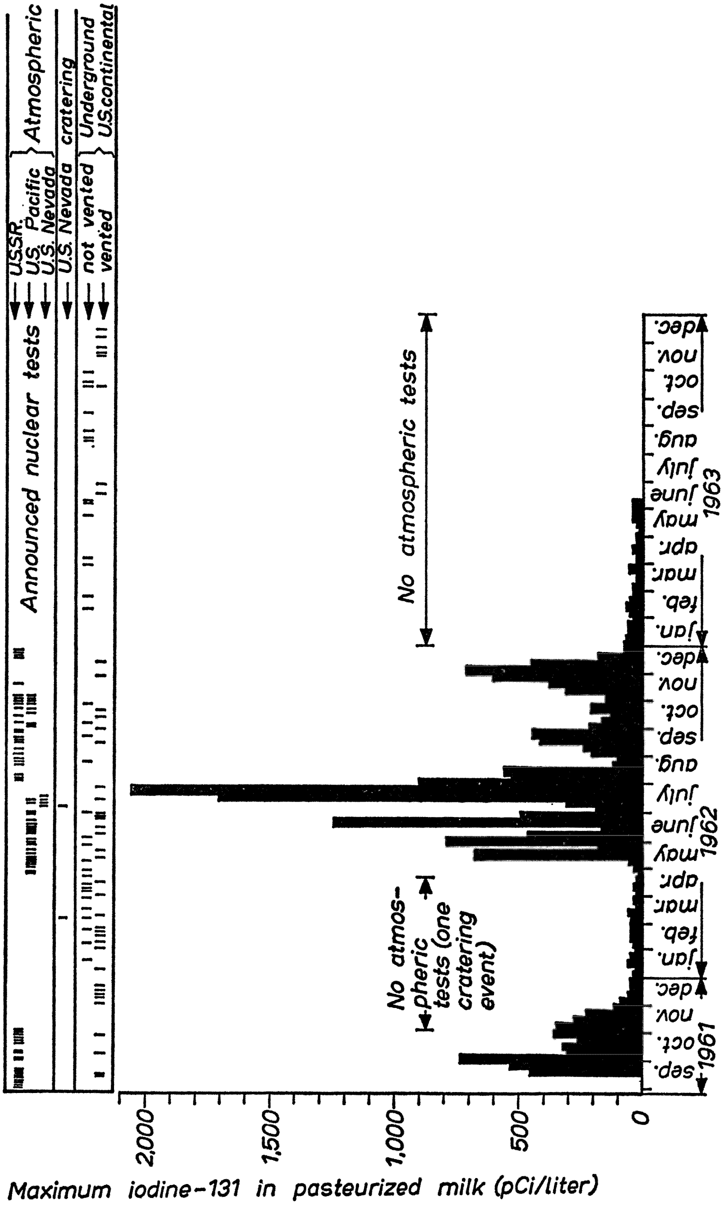

Fig. 5. Maximum concentration of iodine-131 found in any individual milk sample from the Pasteurized Milk Network in the contiguous United States during each week from September 1961 through December 1963

duced in midwestern states during May and June of 1962 have been attributed to atmospheric nuclear tests at the Christmas Islands in the Pacific Ocean. The effect of underground testing in Nevada on ^{131}I in milk has been ruled out except in one case when a milk sample contained more than 300 pCi/liter (LIST *et al.* 1964).

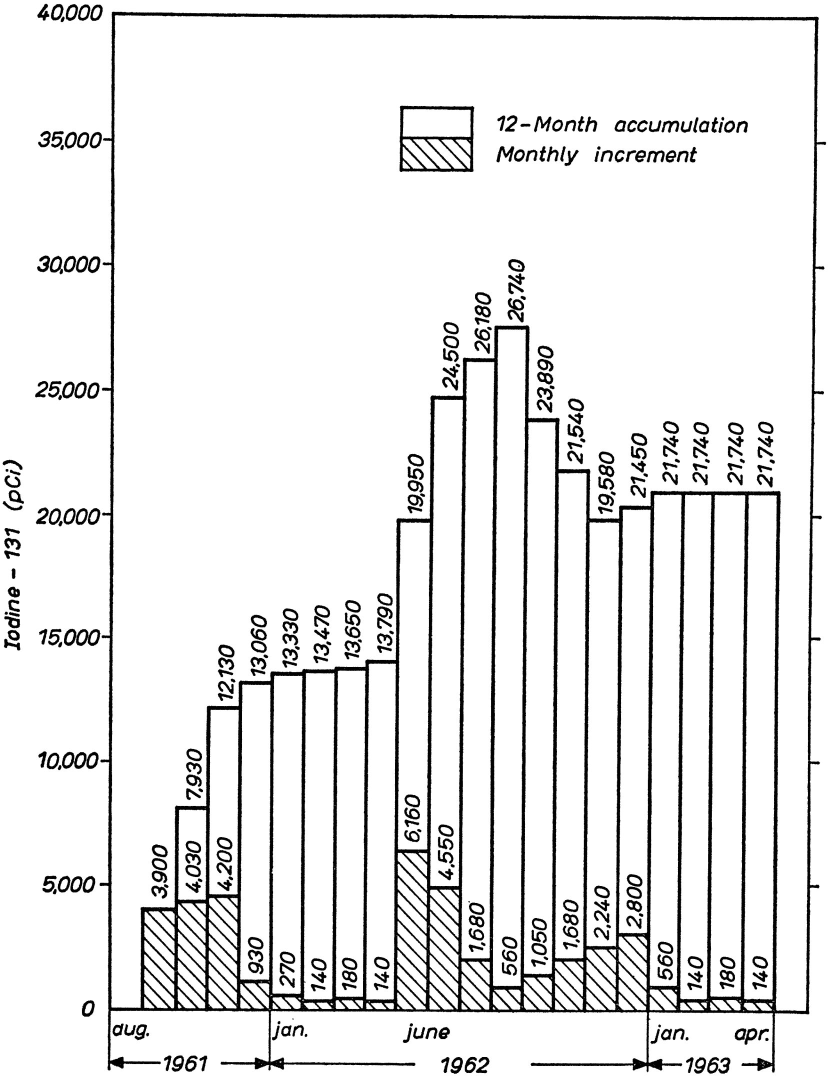

Fig. 6. State of Pasteurized Milk Network in Wichita, Kansas from August 1961 through April 1963

In addition to the high levels of fallout that occurred in 1961, four other special cases of localized fallout were studied: (1) Wichita (Kansas)

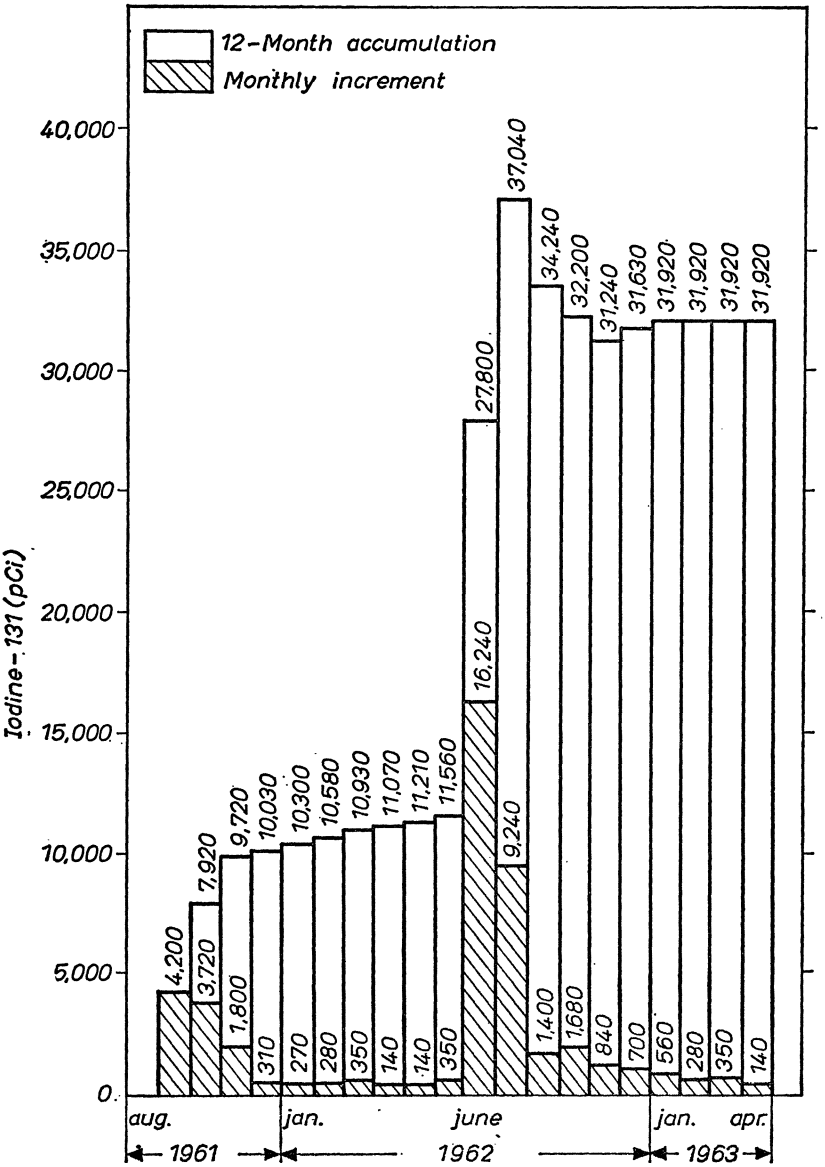

Fig. 7. State of Pasteurized Milk Network in Salt Lake City, Utah from August 1961 through April 1963

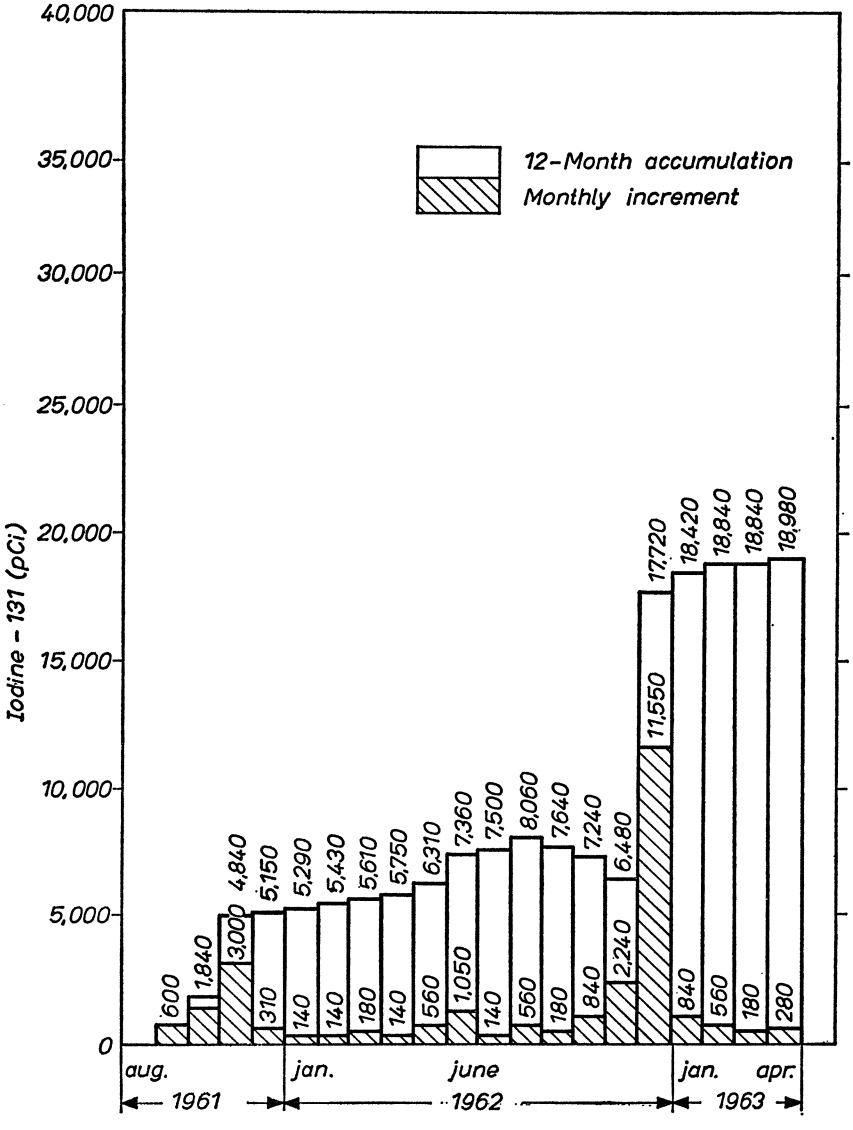

Fig. 8. State of Pasteurized Milk Network in Dallas, Texas from August 1961 through April 1963

—May 1962, (2) Salt Lake City (Utah)—July 1962, (3) Dallas (Texas) —November and December 1962, and (4) Palmer (Alaska)—1961 to 1963 (DAHL 1963). The results are shown in Figures 6, 7, 8, and 9.

In Wichita (Kansas) the ^{131}I levels were as high as 670 pCi/liter. The

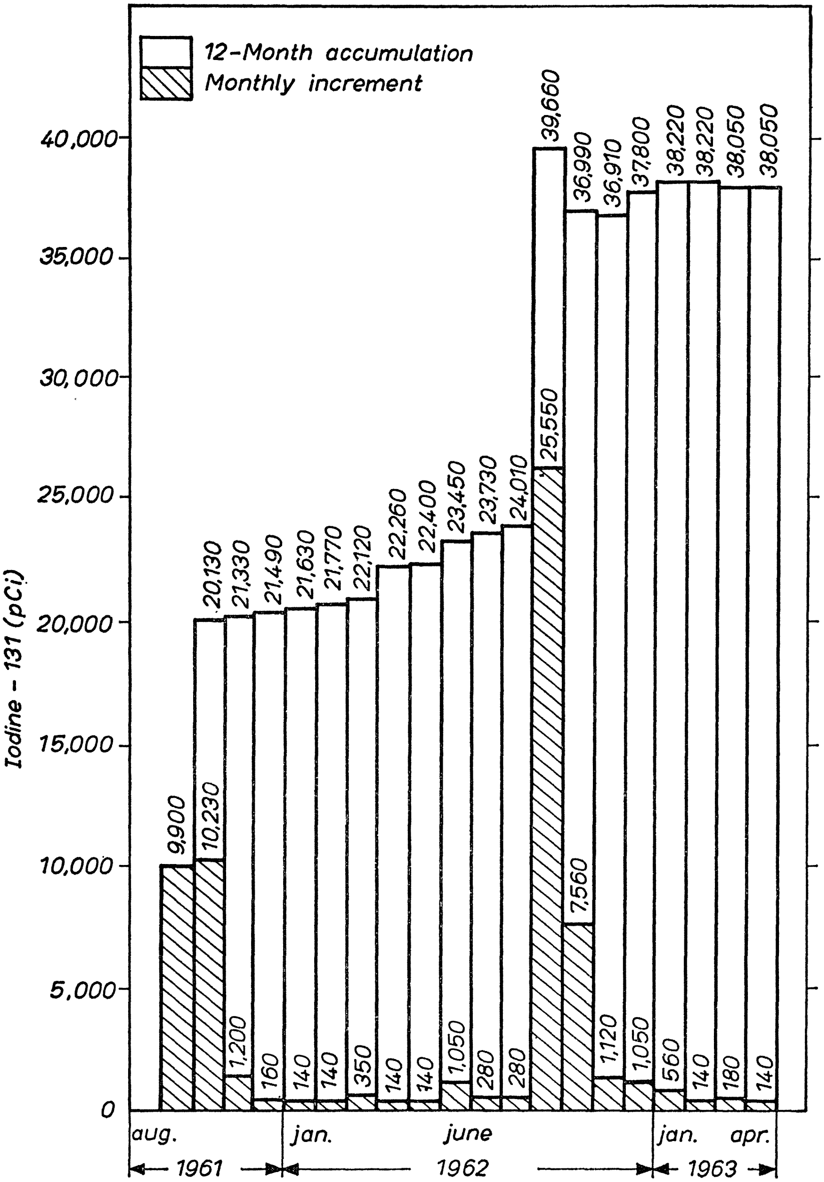

Fig. 9. State of Pasteurized Milk Network in Palmer, Alaska from August, 1961 through April, 1963

increased levels were due to air intrusion which was not monitored at the time. The significance of this case was that milk was the alerting medium.

Appreciable increases in ^{131}I levels of milk samples in Salt Lake City were due to nuclear testing activity in Nevada. The first deposit between July 7 and 10 was attributed to dry fallout and inhalation of airborne ^{131}I by cows; the second, between July 13 and 19, was due to precipitation; and the third between July 20 and 26 was due to rain and dry fallout.

In Texas during November and December 1962 the deposition was associated with high airborne radioactivity and rainfall. Precipitation deposited 0.32 µCi/m.^{2} of gross beta activity; ^{131}I in milk was 200 pCi/liter on November 27, and on November 29 the level rose to 600 pCi/liter. By December 20 the level fell to 70 pCi/liter. The significance here was that ^{131}I levels in milk indicated the extent of air intrusion.

After extensive rainfall in Palmer (Alaska), the ^{131}I levels rose immediately to 2,530 pCi/liter. Some individual samples showed up to 7,000 pCi/liter.

The ^{131}I levels in milk from these special cases have been compared with the *Federal Radiation Council* recommendations for peacetime uses of atomic energy. Only Palmer (Alaska) and Salt Lake City (Utah) values were found to exceed the top of "Range II" for the year (36,500 pCi), and on few occasions the levels exceeded the top of "Range III" (1,000 pCi/liter), but only for short periods of time.

Another case of heavy fallout occurred in the Troy (New York) area on April 26, 1953 as a result of Nevada testing. LAPP (1962) estimates the ^{131}I contamination in the range of two to four Ci/mi.^{2} (0.77 to 1.54 µCi/m.^{2}). Based on British experience in the Windscale incident, a single square inch of plant would be exposed to 650 pCi ^{131}I (a pasture level of 1 µCi/m.^{2}) which, when translated, would show a level of 100,000 pCi/liter in milk. LAPP (1962) further discusses the possibility that if children (0.5 to 1.5 years old) drank this milk in a single "shot" they could have received a total dose of up to 30 rads. Although such a theoretical interpretation is possible, there is always some doubt as to whether the cows were on pasture at the time; therefore, caution should be exercised before predictions are made.

During the Chinese test in October 1964, measurable amounts of ^{131}I were found in some of the milk samples from the Pasteurized Milk Network. None of the levels exceeded 100 pCi/liter; following the Chinese test, however, milk samples from Kansas City (Missouri) and Des Moines (Iowa) during May 1965 showed 220 and 170 pCi/liter, respectively.

In addition to atmospheric testing, underground testing of weapons are also of some concern. On December 10, 1962, in connection with "Project Gnome" at Carlsbad (New Mexico) (*U.S. Department of Health, Education, and Welfare* 1962 a through j), some gaseous material escaped. The highest radioactivity concentration in air was ^{131}I, 1.7 pCi/m.^{3}; ^{133}I, 18 pCi/m.^{3}; and ^{135}I, 3.5 pCi/m.^{3} No activity was found in milk. Of the five animal thyroids counted, one showed as high as 1,100 pCi/gram. Data on ^{131} activity in milk in the vicinity of underground test sites are lacking.

Table VI. *Iodine-131 concentrations in milk from countries other than United States* (pCi/liter); *values in parentheses are averages for the month*

Country	1961				1962				1963
	Sept.	Oct.	Nov.	Dec.	Sept.	Oct.	Nov.	Dec.	Jan.
Argentina[a]	—	—	—	—	← (130) →			—	—
Australia[b]	—	—	—	—	← (15–62) →			—	—
Canada[c]	— (19)	— (64)	— (109)	— (77)	76–372 (138)	81–238 (134)	14–114 (51)	—	—
France[d]	62–92 (80)	107–299 (202)	9–488 (158)	0–164 (34)	—	—	—	—	—
Germany[e]	← (143) →			—	—	—	—	—	—
Great Britain[f]	← (62–136) →			—	—	—	—	—	—
Italy[g]	—	← (158–28) →		—	—	—	—	—	—
Japan[h]	—	36–80 (66)	50–116 (83)	12–33 (24)	27–136 (99)	20–249 (85)	46–317 (112)	30–88 (52)	20–75 (41)
Norway[i]	100–800 (386)	30–1050 (308)	30–800 (222)	40–60 (50)	—	—	—	—	—

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[a] BENNINSON and RAMOS 1962.
[b] BONNYMAN and KEAN 1962.
[c] GRUMMIT and KAMATH 1963.
[d] JEANMARIE and MICHON 1962.
[e] KNOOP and PAAKOLA 1962.
[f] *Great Britain Agricultural Research Council* 1961.
[g] CIGNA and POVLAM 1962.
[h] YAMAGATA and IWASHIMA 1962 and 1963.
[i] HVINDEN *et al.* 1963.

Although several case studies have unusual patterns in the ^{131}I fallout, the fallout pattern observed in Utah due to weapons testing activity in Nevada has always been peculiar and not satisfactorily explainable. For example, during "Sedan Shot" on July 6, 1962 and "Small Boy" on July 14, 1962 appreciable fallout was observed in Utah (PENDELTON 1963). On July 15, 1962 milk showed ^{131}I levels in excess of 2,000 pCi/liter, and occasionally the milk in tank trucks had ^{131}I levels of more than 5,000 pCi/liter. These samples were, therefore, directed to cheese and powdered milk plants. These tests may have caused a total intake of zero to 800,000 pCi ^{131}I by Utah residents consuming one liter of milk per day. The corresponding average infant thyroid doses were approximately one rad, with a peak of 14 rads (PENDELTON 1963).

In contrast to the high levels of ^{131}I observed in the United States, the level of ^{131}I in milk from other parts of the world was considerably lower except in Norway where the concentrations were as high as 1,050 pCi/liter (Table VI).

Milk and food may also become contaminated with ^{131}I from nuclear reactors [*Savannah River Plant* (Aiken, South Carolina), *Hanford Atomic Products Operation* (Richland, Washington), *National Reactor Testing Station* (Idaho Falls, Idaho), *U.K. Atomic Energy Research Establishment* (Windscale)]. The radioactivity releases around nuclear establishments, except for radioactive materials associated with coolants, arise from the dissolution of fuel elements, many of which have been out of the reactor for many days. Direct discharges from reactors, except as already noted, result from accidental releases. For the most part, ^{131}I releases have been held below one Ci per day, and the level of radioactivity in milk under these conditions was found to be below 100 pCi/liter (*U.S. Department of Health, Education, and Welfare* 1962 a through j). In general, one of the reasons the activity releases are monitored is to predict ^{131}I levels in milk. For example, a continuous release of 20 mCi ^{131}I per day from a stack 20 meters high would result in ^{131}I concentration of less than 400 pCi/liter of milk produced from pasture at 7,500 meters from the stack (BRYANT 1964).

During 1956 the plant at Savannah (Georgia) released 1,576 Ci ^{131}I. Milk showed ^{131}I values as high as 5.4 nCi/liter. The average for 48 farms was 890 pCi/liter. In terms of dosage, if a child regularly consumed one liter of milk per day containing 5.4 nCi/liter, his total integrated thyroid dose would be 1.3 rem, whereas an adult consuming the same milk would have 140 mrem. On the other hand, with milk containing 800 pCi/liter, the total integrated dose for a child would be 205 mrem and for an adult 25 mrem (MARTER 1963). During 1961-1962 ^{131}I releases averaged below 20 mCi/day.

The Windscale incident during October 1957 released 20,000 Ci ^{131}I (DUNSTER 1958). Milk samples collected subsequently showed 0.001 to one µCi ^{131}I/liter of milk. The highest observed level during the course of the survey (October 13) was 1.4 μCi/liter of milk from a farm ten miles from Windscale. The farm was in the direct path of the main plume and 400 feet above sea level. The initial sharp rise in ^{131}I of milk occurred 12 to 24 hours after the release; thereafter it decreased with a half-life of approximately five to six days.

Measurements made of radioactivity in the thyroids of adults and children showed a maximum integrated dose to the thyroid of the child of 16 rads and for the adult of 9.5 rads. Assumptions were that the intake had all occurred in a short period and that the mass of the adult thyroids was 20 grams and that of the child five grams.

IX. Methods for determining iodine-131 in milk

Methods for determining ^{131}I in milk include chemical analysis (BERGH 1958, DAS GUPTA 1963), gamma-ray spectrometry (HAGEE *et al.* 1960, YAMAGATA and IWASHIMA 1962), gamma-counting of protein obtained from milk preserved with formaldehyde (MURTHY and CAMPBELL 1960), ion-exchange separation and gamma-counting of resin (BONI 1963, MURTHY 1963, SMITH and WHITEHEAD 1963), ion-exchange separation and beta-counting of silver iodide (KAHN 1964), and *in vivo* measurement of the thyroid of cows as an aid to monitoring milk (YAMAGATA and IWASHIMA 1963).

In the chemical methods, time consuming steps necessary to eliminate interfering substances include sample preparation, solvent extraction, and a series of oxidation-reduction reactions before the iodine is precipitated as silver iodide for determining its activity.

Gamma-ray spectrometric methods are simple (see GUINN and SCHMITT 1964); they require no sample preparation and provide considerable savings in time. An appropriate amount of milk sample is transferred to a container and placed over a thallium-activated sodium iodide crystal coupled to a photomultiplier tube and a single or a multichannel analyzer. The ^{131}I activity is measured by observing the counting rate at 364 kev. Correction must be made for the effects of potassium-40, cesium-137, and barium-140 on the iodine-131 photo-peak.

The method of gross gamma-counting of proteins obtained from milk preserved with formaldehyde is based on the reaction in which the formaldehyde transforms quantitatively the inorganic ^{131}I to the protein-bound form. The method is simple and may be used in conjunction with strontium-90 analysis (MURTHY *et al.* 1960); however, the limit of detection is about 300 to 400 pCi ^{131}I.

The ion-exchange method of separating ^{131}I from unpreserved milk and gamma-counting of resin is simple and rapid and may be adapted to field conditions for rapid analysis of milk (BARATTA 1964). The method measures only the inorganic ^{131}I in milk and not the protein-bound ^{131}I. In market milk, however, most of the ^{131}I has been found to be in the inorganic form. The minimum detectable concentration of ^{131}I by this technique is about four pCi.

If ^{131}I levels at the one-pCi level are to be desired, it is possible to elute the ^{131}I absorbed on the resin, which is precipitated as silver iodide prior to counting in a low-level beta counter (KAHN 1965).

In an emergency situation, such as the Windscale incident, milk is monitored for ^{131}I with a gamma-ray detector coupled to a single-channel analyzer. The detector is held against a 50-liter container of milk. The minimum detectable amount of ^{131}I by this technique is 400 pCi/liter.

X. Guides, standards, and regulations

Some of the earliest recommendations regarding maximum permissible doses, body burdens, and concentrations in air and water were promulgated by the *International Commission on Radiological Protection* (1959 and 1960) and the *National Committee on Radiation Protection and Measurement* (1954 and 1959). As Title 10, Part 20 (*Code of Federal Regulations* 1963) these recommendations have formed the basis of the *Atomic Energy Commission* regulations. Originally, the recommendations covered only the industrial worker, but were applied to the calculation of dose and permissible concentrations for the individual in the population, as well as the population in general, through the use of appropriate multiplying factors. Included in the recommendations were maximum permissible body burdens (*MPBB*) and concentrations in water $[(MPC)_w]$ and air $[(MPC)_a]$ for ^{131}I. These values are summarized in Table VII.

Table VII. *Maximum permissible body burdens (MPBB) and concentrations (MPC) in water and air for iodine-131 (International Commission on Radiological Protection* 1960, *National Committee on Radiation Protection* 1959)

Chemical form	Organ of reference	MPBB[a] (μCi)	MPC (μCi/cm.^{3})			
			40-Hour week		168-Hour week	
			Water	Air	Water	Air
Soluble	Thyroid[b]	0.7	$6\text{x}10^{-5}$	$9\text{x}10^{-9}$	$2\text{x}10^{-5}$	$3\text{x}10^{-9}$
	Total body	50	$5\text{x}10^{-3}$	$8\text{x}10^{-7}$	$2\text{x}10^{-3}$	$3\text{x}10^{-7}$
	GI (LLI)[c]	—	$3\text{x}10^{-2}$	$7\text{x}10^{-6}$	$1\text{x}10^{-2}$	$2\text{x}10^{-6}$
Insoluble	GI (LLI)[b, c]	—	$2\text{x}10^{-3}$	$3\text{x}10^{-7}$	$6\text{x}10^{-4}$	10^{-7}
	Lung[b]	—	—	$3\text{x}10^{-7}$	—	10^{-7}

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[a] Sometimes designated as *q*.
[b] Critical organ.
[c] *GI* = gastrointestinal tract; *LLI* = lower large intestine.

These values indicate that the thyroid is the most critical organ for ^{131}I with a maximum permissible dose of 30 rem/year. The maximum permissible concentrations shown for the thyroid as the critical organ (168-hour week) convert to ingestion and inhalation levels of approximately

5×10^{-2} µCi/day. This value is equivalent to 50,000 pCi/day. If one applies the multiplying factors for exposure to the individual and for exposure of the general population, this number becomes 5,000 and 1,700 pCi/day, respectively.

In 1959 the *Federal Radiation Council* was formed (Public Law 86-373) to provide a Federal policy on human radiation exposure including ". . . guidance . . . in the formulation of radiation standards . . ." (*Federal Radiation Council* 1960). Average dose levels [*Radiation Protection Guides* (*RPG*)] to the thyroid recommended by the *Federal Radiation Council* for controlled sources amount to 30 rem/year for the occupational worker, three rem/year for the individual adult in the population at large, and 1.5 rem/year for the individual child in the population at large. For the population average the numbers quoted are 1.0 and 0.5 rem/year for the average adult and average child, respectively. In its second report (*Federal Radiation Council* 1961) guidance was provided on the intake of ^{131}I. Calculations showed that an intake of 80 pCi/day would correspond to the *RPG* of 0.5 rem/year. This has been rounded off to 100 in establishing the action ranges of intake for this radionuclide as shown in Table VIII.

The *Federal Radiation Council* recommended values for general population average intake are somewhat lower than the *International Commission on Radiological Protection-National Committee on Radiation Protection and Measurement* recommended values cited earlier. The difference exists because the latter calculations are based on standard man, whereas those reported by the former are for a one-year-old child having a two-gram thyroid and consuming milk. As a matter of fact, the *Federal Radiation Council* states that "For adults, the *RPG* for the thyroid would not be exceeded by rates of intake higher by a factor of 10 than those applicable to small children."

Table VIII. *Transient rates of intake for iodine-131 and graded scales of action*

Range	Transient rate of intake (pCi/day)	Scale of action
I	0–10	Periodic confirmatory surveillance as necessary
II	10–100	Quantitative surveillance and routine control
III	100–1,000	Evaluation and application of additional control measures as necessary

In July 1964 the *Federal Radiation Council* released another document spelling out more specific guidance in respect to: "(1) planning protective action to reduce potential doses to the population from radioactive fission products which may gain access to food, and (2) doses at which implementation of protective actions may be appropriate." This report recommended adoption of the term "Protective Action Guide" (*PAG*) for Federal use and established a *PAG* for ^{131}I of 30 rads to the thyroid. As an operational technique, it was considered that the *PAG* would not be exceeded if the average projected doses to the thyroids of a suitable sample of the population did not exceed ten rads. A suitable sample was identified as one consisting of children approximately one year of age who consumed milk from a reasonably homogeneous supply. It was further reported that a total intake of ^{131}I of 600 nCi (600,000 pCi) would result in a dose of about ten rads to a two-gram thyroid. This value of total intake may be compared with a total intake of 365,000 pCi obtained by consuming 1,000 pCi/day of milk (the top of "Range III") over a period of one year. The values cited for the *PAG* are applicable to non-controlled sources of release such as fallout from nuclear devices and accidental releases of radioactive iodine.

Some years ago, following the Windscale incident in England, the *British Medical Research Council* in 1959 suggested an emergency ^{131}I total ingestion value of 650,000 pCi, which with 50 percent uptake into the 1.8-gram gland and prolonged retention would give a mean dose of 25 rad to the gland. Although the total ingestion values are the same (about 600,000 pCi) the dosages to the thyroid vary by a factor of about two. This disparity results primarily from the difference in uptake assumed by the *Federal Radiation Council* and the *British Medical Research Council.*

XI. Countermeasures

Any countermeasure taken would have to be after a careful evaluation of the situation, including medical assessment and economics of the decontamination procedures by the responsible authorities. Several avenues have been suggested for reducing the concentration of ^{131}I in milk. If contamination is localized, milk from other areas may be brought in for immediate consumption. If, however, contamination is widespread, importing milk is not practical. Storage of milk for a sufficient period of time or diverting milk to milk plants to manufacture evaporated milk, milk powder, cheese, etc., is not feasible in countries like the United States since the industry does not have the capacity to store milk for long periods of time or the processing capabilities to handle excessively large quantities of milk.

Transfering cows from pasture to sheltered areas and feeding them stored uncontaminated feed have decreased ^{131}I in milk considerably (KAHN *et al.* 1962). The only source of ^{131}I to cows under these circumstances would be inhalation or the ingestion of sheltered feed that had also been contaminated by deposition. Although this method is favored at the present time and has been found to be successful, it depends on obtaining sufficient warning with regard to movement of nuclear debris. Moreover, this arrangement has limitations because of the lack of sufficient stored feed and the economics of storing large quantities of the feed.

Modification of farm practices by fertilization of soil has been shown to decrease the ^{131}I levels in milk by 50 percent. This is mainly attributed to increased pasture yields whereby the concentration of ^{131}I is reduced per unit weight of pasture (HANSEN 1961).

Although fertilizing pasture reduces the concentration of ^{131}I, it would not be effective in an emergency situation involving higher exposures as in the Windscale incident. Under such circumstances treatment of milk would be necessary to reduce the contamination to safe levels.

Iodine-131 may be removed from milk by ion-exchange techniques (COSSLETT and WATTS 1959, DEMOTT *et al.* 1960, GLUEKAUF *et al.* 1959, MURTHY *et al.* 1962). The most effective and useful technique (MURTHY *et al.* 1962) consists of passing milk through a strong base anion-exchange resin column previously charged with a solution containing chlorides, phosphates, and citrates, which has been calculated by means of the formula 4.03 (m*M* chlorides/liter) + 4.64 (m*M* inorganic phosphorus/liter) + 20.4 (m*M* citric acid/liter). The expressions in parentheses are the anionic composition of milk to be treated, and numerical coefficients account for the difference in the affinity of these anions for the resin. With this method approximately 98 percent of the ^{131}I present may be removed from 160 to 200 resin bed volumes of milk. Temperatures of milk ranging from 0° to 30° C. have no effect on the absorption of ^{131}I on the resin. The anionic composition and organoleptic quality of the resin-treated milk are comparable to those observed with untreated milk. The method is simple and compatible with the practices and equipment used in the milk plant and can be used in conjunction with the process used for the removal of cationic radionuclides from milk (MURTHY *et al.* 1961).

Other attempts to reduce ^{131}I levels in milk include feeding of massive daily doses of stable iodide to cows. The radioactivity of milk was found to decrease by half in two days, whereas two-thirds of the isotope was removed in ten days from their thyroids (LENGEMANN and SWANSON 1957).

Limited studies have been made to determine the effect of processing of milk on ^{131}I activity. No ^{131}I activity was eliminated in the process of high-heat treatment (DEMOTT and EASTERLY 1960) or manufacturing evaporated milk, but about 40 percent of the ^{131}I present was eliminated in the manufacture of powdered milk (RAMOND and WILLIAMS 1964).

Summary

Of the several isotopes of iodine formed from nuclear weapons testing or reactor operations, only iodine-131 (^{131}I) with a half-life of 8.08 days, is considered as a potential health hazard. When released into the environment ^{131}I is transported from source to site of deposition by the drift of nuclear clouds. The debris is brought to the surface as dry fallout or by precipitation scavenging, depending upon meteorological and climatological conditions. The chemical nature of iodine in fallout has been identified as

iodide ion plus iodine, and iodate ion, and small amounts of periodate ion.

When deposited on plants, ^{131}I is absorbed through leaves but not translocated into other plant parts, and there is no root uptake. When ingested by animals through contaminated food and partly by inhalation, ^{131}I is concentrated in the thyroid gland. Excretion from the body may be through several routes including milk. Iodine is present in milk mostly in the inorganic form as the iodide, very little being bound by proteins.

In countries where it is abundant, milk is considered as the major dietary source of ^{131}I to children, whereas in countries where milk is a luxury, leafy vegetable may form a major source of intake.

The marked differences in the concentration of ^{131}I in milk produced in different parts of the world are attributed mainly to the geographical location of the milkshed in relation to weapons testing sites and the meteorological and climatological factors. Apart from fallout, milk may also be contaminated from releases due to reactor malfunctions and chemical reprocessing plants. Evaluation of the observed values for ^{131}I has been possible only by comparing them with the *International Commission on Radiological Protection* and *Federal Radiation Council* guidelines.

Various techniques to reduce ^{131}I levels in milk include moving of cows from pasture to barn and feeding them uncontaminated food, and the removal of ^{131}I from milk by commercially feasible ion-exchange techniques.

Despite investigations to understand the behavior of iodine in the environment, our knowledge so far on the potential health hazard associated with observed relatively low levels of ^{131}I is relatively poor. A continued exploration is warranted into the radioactive forms of the element, particularly in (1) identification of chemical forms of iodine in fallout, (2) establishment of metabolic processes of various forms of iodine in plants, animals, and man, (3) identification of the nature of iodine in vegetation, and (4) exploration of reaction of fallout iodine with biological materials. More definitive and conclusive data regarding these areas will help in developing better guides to evaluate human exposure to radioactive forms of iodine.

Résumé*

Des divers isotopes de l'iode produits par les essais nucléaires ou les réacteurs, seul l'^{131}I dont la période est de 8,08 jours, est considéré comme dangereux pour la santé. Lorsqu'il est libéré l'^{131}I est transporté depuis la source jusqu'au point de dépôt par la dérive des nuages radioactifs. Les débrits sont amenés à la surface sous forme de retombées sèches ou de précipitations de boues, selon les conditions météorologiques ou climatiques. La nature chimique de l'iode dans les retombées a été identifiée comme étant $I^- + I_2$, IO_3^- avec de petites quantités de IO_4^-.

Lorsqu'il est déposé sur des plantes, l'^{131}I est absorbé par les feuilles

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* Traduit par R. MESTRES.

mais n'est pas transporté dans les autres parties de la plante et il n'y a pas d'absorption par les racines. Lorsqu'il est ingéré par les animaux par l'intermédiaire d'aliments contaminés et en partie par inhalation, l'^{131}I est concentré dans la thyroide. L'excrétion peut suivre plusieurs voies dont celle du lait. L'iode est présent dans le lait surtout sous la forme inorganique d'iodure, très peu d'iode se trouvant lié aux proteines.

Dans les pays où il est abondant, le lait est considéré comme la source principale d'^{131}I pour les enfants; par contre dans les pays où le lait est un luxe, les légumes verts peuvent constituer la source principale de l'absorption.

Les différences notables dans la concentration de l'^{131}I dans le lait produit en différentes régions du monde sont attribuées principalement à la situation géographique de la région productrice par rapport aux sites d'essais nucléaires et aux facteurs météorologiques et climatiques. En dehors des retombées radioactives, le lait peut également être contaminé par des déchets produits à la faveur de mauvais fonctionnements de réacteurs et par des usines de traitement. L'évaluation des valeurs observées pour l'^{131}I n'a été possible que par comparaison avec celles données par l'ICPR et le FCR.

Les teneurs en ^{131}I dans le lait peuvent être réduites par le retrait des vaches des paturages et leur nourriture à l'étable au moyen de fourrages non contaminés. L'^{131}I peut être éliminé du lait au moyen d'échangeurs d'ions par des techniques applicables commercialement.

Malgré les recherches effectuées pour comprendre le métabolisme de l'iode dans la nature, nos connaissances actuelles sur les dangers que peuvent entraîner pour la santé les taux relativement bas d'^{131}I sont très limitées. Il est justifié de continuer les recherches sur les formes radioactives de cet élément, surtout (1) dans l'identification des formes chimiques de l'iode dans les retombées, (2) l'établissement des métabolismes des diverses formes de l'iode dans les plantes, les animaux et l'homme, (3) l'identification de la nature de l'iode dans les végétaux et (4) l'exploration de la réaction de l'iode des retombées avec les produits biologiques. Des données plus sûres et concluantes sur ces points aideront à développer de meilleures méthodes pour apprécier l'importance de l'exposition de l'homme aux formes radioactives de l'iode.

Zusammenfassung*

Von den verschiedenen Jodisotopen, die bei Kernwaffentesten oder Reaktoroperationen entstehen, ist nur ^{131}I mit einer Halbwertszeit von 8.08 Tagen als mögliche Gesundheitsgefahr anzusehen. Bei Freisetzung in die Umgebung wird ^{131}I vom Ursprungsort durch die Abtrift der Nuclearwolken zur Ablagerungsstelle transportiert. Das Zerfallsprodukt wird auf die Erdoberfläche gebracht als radioaktiver Trocken-Niederschlag oder als

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* Übersetzt von F BÄR.

Reinigungsfällung, in Abhängigkeit von den meteorologischen und klimatologischen Bedingungen. Die chemische Natur des Jods im Niederschlag wurde als $J^- + J_2$ und JO_3^-, und geringe Mengen von JO_4^- identifiziert.

Nach Ablagerung auf den Pflanzen wird ^{131}I durch die Blätter absorbiert, aber nicht in andere Pflanzenteile befördert; und es erfolgt keine Aufnahme durch die Wurzel. Nach Aufnahme durch die Tiere über die verunreinigte Nahrung und teilweise durch Inhalation, wird ^{131}I in der Schilddrüse konzentriert. Die Ausscheidung aus dem Körper kann über verschiedene Wege einschliesslich die Milch erfolgen. In der Milch ist Jod meist in anorganischer Form als Jodid vorhanden, sehr wenig wird durch Proteine gebunden.

In Ländern, in denen die Milch reichlich vorhanden ist, wird sie als hauptsächliche Nahrungsquelle des ^{131}I für Kinder angesehen; während in Ländern, in denen die Milch ein Luxus ist, das Blattgemüse die hauptsächliche Aufnahmequelle bilden kann.

Die ausgesprochenen Unterschiede in der Konzentration von ^{131}I in der Milch, die in verschiedenen Teilen der Welt produziert wird, werden hauptsächlich zugeschrieben der geographischen Lokalisation des Milcheinzugsgebietes in Abhängigkeit vom Ort der Waffenteste und von den meteorologischen und klimatologischen Faktoren. Abgesehen vom radioaktiven Niederschlag kann die Milch auch durch Abgabe bei schlechtem Funktionieren von Reaktoren und chemischen Aufarbeitungsanlagen verseucht werden. Die Bewertung der beobachteten Werte für ^{131}I war nur durch ihren Vergleich mit den ICRP- und FRC-Leitlinien möglich.

Die verschiedenen Methoden zur Herabsetzung der ^{131}I-Konzentration in der Milch umfassen den Abtrieb der Kühe von der Weide in den Stall und ihre Fütterung mit nicht verseuchter Nahrung, und das Entfernen des ^{131}I aus der Milch durch praktikable Jonenaustauschmethoden.

Trotz der Untersuchungen zum Verständnis des Verhaltens von Jod in der Umwelt ist unsere Kenntnis bezüglich der möglichen Gesundheitsgefahr in Verbindung mit den beobachteten relativ niedrigen ^{131}I-Konzentrationen relativ gering. Eine fortgesetzte Erforschung der radioaktiven Formen des Elements ist gerechtfertigt insbesondere zur 1) Identifizierung der Chemischen Formen des Jod im radioaktiven Niederschlag; 2) Feststellung der Stoffwechselprozesse der verschiedenen Formen des Jod in der Pflanze, im Tier und im Menschen, 3) Identifizierung der Natur des Jod in der Vegetation, und 4) Untersuchung der Reaktion des voll ausgebildeten radioaktiven Jod-Niederschlages mit dem biologischen Material. Endgültigere und schlüssigere Daten auf diesem Gebiet werden zur Entwicklung besserer Richtlinien zur Bewertung des Einflusses der radioaktiven Formen des Jod auf den Menschen beitragen.

References

BARATTA, E.: Ion exchange cartridges and rapid determination of ^{131}I in milk under field conditions. *U.S. Public Health Service,* Division of Radiological Health, unpublished 1964.

BARRY, P. J., and A. C. CHAMBERLAIN: Deposition of iodine onto plant leaves from air. Health Phys. **9**, 1149 (1963).

BENNINSON, D., and E. RAMOS: Contaminacion por I^{131} debida al fallout. *Argentina Commission Nacionel de energia Atomica,* Buenos Aires 1962.

BERGH, H.: A method for the determination of I-131 in biological materials, especially in milk. *Norwegian Defense Research Establishment,* Kjeller, Norway 1958.

BONI, A. L.: Rapid determination of I-131 in milk. Analyst **88**, 64 (1963).

BONNYMAN, J., and D. W. KEAM: Iodine-131 levels in milk in Australia during period May-Nov. 1962. Australian J. Sci. **26**, 74 (1963).

BRYANT, P. M.: Derivation of working limits for continuous release rates of iodine-131 to atmosphere in a milk producing area. Health Phys. **10**, 249 (1964).

BUSTAD, L. K., D. H. WOOD, E. E. ELEFSON, H. A. RAGAN, and R. O. MCCLELLAN: I^{131} in milk and thyroid of dairy cattle following a single contamination event and prolonged daily administration. Health Phys. **9**, 1231 (1963).

CAMPBELL, J. E., G. K. MURTHY, A. S. GOLDIN, H. B. ROBINSON, C. P. STRAUB, and K. H. LEWIS: The occurrence of strontium-90, iodine-131, and other radionuclides in milk, May 1957 through April 1958. Amer. J. Public Health **49**, 225 (1959).

— — Summary of results from the raw milk sampling program. Radiological Health Data (*U.S. Department of Health, Education, and Welfare, Public Health Service*) 4, 511 (1963).

— —, C. P. STRAUB, K. H. LEWIS, and J. G. TERRILL: Radionuclides in milk. J. Agr. Food Chem. **9**, 117 (1961).

CHAMBERLAIN, A. C.: Dispersion of activity from chimney stacks. In: Atomic energy waste, its nature, use, and disposal. Ed. E. Glueckauf. Part 3, chapter 4.5. New York: Interscience 1961.

—, and R. C. CHADWICK: Deposition of airbourne radioiodine vapor. Nucleonics **11**, 22 (1953).

—, and H. J. DUNSTER: Deposition of radioactivity in northwest England from the accident at Windscale. Nature **182**, 629 (1958).

Chilean Iodine Education Bureau: Geochemistry of iodine. Annotated biblio. 182. New York: *Chilean Iodine Educational Bureau* 1956.

CIGNA, A., and F. G. GIORECELLI: Iodine-131 thyroid dose from milk in Italy during the period Sept. 1962-Feb. 1963. Science **143**, 379 (1964).

—, and C. POLVANI: Intake of iodine-131 from milk in Italy during the period from October-December 1961. Nature **194**, 1123 (1962).

Code of Federal Regulations: Standards for protection against radiation. Title 10, part 20, p. 96 (1963).

COSSLETT, P., and R. E. WATTS: The removal of radioactive iodine and strontium from milk by ion exchange. *United Kingdom Atomic Energy Authority,* AERE-R 2881 (1959).

DAHL, A. H., R. BOSTRUM, R. C. PATZER, and J. C. VILLFORTH: Patterns of I^{131} levels in pasteurized milk network. Health Phys. **9**, 1179 (1963).

DAS GUPTA, A. K., and H. G. GREEN: *United States Atomic Energy Commission,* RPD-23. A method for radiochemical determination of iodine-131 in milk. Radiation Protection Division, *Department of National Health and Welfare,* Ottawa, Canada (1963).

DAVIS, S.: The analytical determination of strontium-90 in foods. Residue Reviews **12**, . . . (1965).

DEMOTT, B. J., D. G. EASTERLY, and *University of Tennessee Atomic Energy Commission*: Removal of $iodine^{131}$ from milk. J. Dairy Sci. **43**, 118 (1960).

— — Removal of $iodine^{131}$ from milk. J. Dairy Sci. **43**, 1140 (1960).

DUNSTER, H. J., M. HOWELLS, and W. L. TEMPLETON: District surveys following the Windscale incident. In: 2nd Internat. Conf. on Peaceful Uses of Atomic Energy, Geneva, United Nations. A/CONF. 15/P/316 (1958).

EISENBUD, M., Y. MOCHIZUKI, A. S. GOLDIN, and G. R. LAURER: Iodine-131 dose from Soviet nuclear tests. Science **136**, 370 (1962).

—, B. PASTERNACK, G. LAURER, and L. BLOCK: Variability of the I^{131} concentrations in the milk distribution system of a large city. Health Phys. **9**, 1903 (1963).

Federal Radiation Council: Background material for the development of radiation protection standards. Report I. Washington, D. C.: Superintendent of Documents, *U.S. Government Printing Office* 1960.

— Report II. *Ibid.* (1961).

— Report V. *Ibid.* (1964).

GARNER, R. J.: The metabolism of iodine and strontium in cows. Vet. Record **71**, 982 (1959).

— An assessment of the qualities of fission products likely to be found in milk in the event of aerial contamination of agricultural land. Nature **186**, 1063 (1960).

—, B. F. SANSOM, and H. G. JONES: Fission products and the dairy cow. III. Transfer of ^{131}I to milk following single and daily dosing. J. Agr. Sci. **55**, 283 (1960).

GLASSOCK, R. F.: The secretion of a single tracer dose of labeled iodine in the milk of the lactating cow. J. Dairy Research **21**, 318 (1954).

GLUECKAUF, E., P. COSSLET, and R. WATTS: Removal of radioactive iodine from milk. *United Kingdom Atomic Energy Authority.* AERE-c/M 371 (1959).

Great Britain Agriculture Research Council: Interim report. Wantage, Berks, England. A/AC. 82/G/L 762 (1961).

GRUMMITT, W. E., and P. R. KAMATH: Iodine-131 content of Canadian milk during 1961. *Atomic Energy Canada,* Ltd. Chalk River. AECL-1797 (1963).

GUINN, V. P., and R. A. SCHMITT: Determination of pesticide residues by neutron-activation analysis. Residue Reviews **5**, 148 (1964).

HAGEE, G. R., G. J. KARCHES, and A. S. GOLDIN: Determination of I^{131}, Cs^{137}, and Ba^{140} in fluid milk by gamma spectroscopy. Talanta **5**, 36 (1960).

HANSEN, W. G.: Farming practices and concentration of fission products in milk. *United States Public Health Service.* Publ. 999-R-6 (1964).

HOLLAND, J. Z.: Physical origin and dispersion of radioiodine. Health Phys. **9**, 1095 (1963).

HONOUR, A. J., N. B. MYANT, and E. N. ROWLANDS: Secretion of radioiodine in digestive juices and milk in man. Clinical Sci. **2**, 447 (1952).

HULL, A. P.: Vegetation retention and vegetation-milk ratios of fallout I^{131}. Health Phys. **9**, 1173 (1963).

HUNGATE, F. P., J. F. CLINE, R. L. UHLER, and A. A. SELDERS: Foliar absorption of I^{131} by plants. Health Phys. **9**, 1159 (1963).

HVINDEN, T., L. LUND, and P. THORENSEN: Fallout in Norwegian milk in 1961. *Forsvarets Forsknings Institutt,* Kjeller. FFIS-IR-S-15 (1961).

International Commission on Radiological Protection: Recommendations of the International Commission of Radiological Protection. New York: Pergamon 1959.

— Report of Committee II on permissible dose for internal radiation. Stockholm, Sweden (1959); also, Health Phys. **3**, 1 (1960).

JEANMARIE, L., and G. MICHON: Les pollutions radioactives par l'iode-131 in France du les Novembre 1961 au les Fevrier 1962. *Commissariat a l'energie Atomique Center d'edudes Nucleares,* Fontenay-aux-Roses (1962).

KAHN, B.: Radionuclides in milk—determination of picocurie concentrations of iodine-131 in milk. J. Agr. Food Chem. **13**, 21 (1965).

—, C. P. STRAUB, and I. R. JONES: Radioiodine in milk of cows consuming stored feed and of cows on pasture. Science **138**, 1334 (1962).

KEISCH, B., and R. C. KOCH: Status report no. 1, parts I and II, November 1962, and Status report no. 2, February 1963. Pittsburgh: Nuclear Science and Engineering Corp. (1962 and 1963).

KNAPP, H. A.: Iodine-131 in fresh milk and human thyroids following a single deposition. *United States Atomic Energy Commission,* TID-19266 (1963).

KNOOP, E., and O. PAAKKOLA: I^{131}, Cs^{137}, Ba^{140}, and La^{140} of German milk in the period September 1961-January 1962. Milchwissensch. **17**, 289 (1962).

KOREN, K., and A. BELL: Determination of radioiodine in milk. Health Phys. **5**, 225 (1961).

LANE, W. B.: Plowshare program. *United States Naval Radiological Defense Laboratory,* San Francisco, Calif. P.NE-229P (1963).

LAPP, R. E.: Nevada test site and radioiodine in milk. Science **137**, 756 (1962).

LENGEMANN, F. W., and C. L. COMAR: Metabolism of I^{131} by dairy cows during long term daily administration of the radioisotope. Health Phys. **10**, 55 (1964).

—, and E. W. SWANSON: A study of the secretion of iodine in milk of dairy cows using oral doses of I^{131}. J. Dairy Sci. **40**, 216 (1957).

LEWIS, E. B.: Thyroid radiation doses from fallout. Proc. Nat. Acad. Sci. **45**, 894 (1959).

LIST, R. J., K. TELEGADS, and G. J. FERBER: Metereological evaluation of the sources of iodine-131 in pasteurized milk. Science **146**, 59 (1964).

MACHTA, L.: Metereological processes in the transport of weapon radionuclides. Health Phys. **9**, 43 (1963).

MARTER, W. L.: Radioiodine release incident at the Savannah River Plant. Health Phys. **9**, 1105 (1963).

MARTIN, W. E.: Loss of I^{131} from fallout-contaminated vegetation. Health Phys. **9**, 1141 (1963).

MILLER, J. K., and E. W. SWANSON: Some factors affecting iodine secretion in milk. J. Dairy Sci. **46**, 927 (1963).

— —, and R. G. CRAGLE: Relation of milk secretion to iodine in milk. Health Phys. **9**, 1247 (1963).

MODELMONT, C., and G. MICHON: Radioactive contamination of milk consumed in the Paris area. Lait **44**, 19 (1964).

MOISIO, T., and J. K. MIETTINEN: Cesium-137 and iodine-131 in Finnish milk from September 1962-August 1963. Seeomen Kemi **36B**, 175 (1963).

MURTHY, G. K.: Rapid determination of iodine-131 in milk. In: Rapid methods for estimating fission product concentrations in milk. *United States Public Health Service* Publ. 999-R-2 (1963).

—, and J. E. CAMPBELL: A simplified method for the determination of iodine-131 in milk. J. Dairy Sci. **43**, 1042 (1960).

—, J. E. GILCHRIST, and J. E. CAMPBELL: Method for removing iodine^{131} from milk. J. Dairy Sci. **45**, 1066 (1962).

—, E. B. MASUROVSKY, J. E. CAMPBELL, and L. F. EDMONDSON: Method for removing cationic radionuclides from milk. J. Dairy Sci. **44**, 2158 (1961).

National Committee on Radiation Protection and Measurement: Permissible dose from external sources of ionizing radiation. *National Bureau of Standards* handbook 59. Washington, D.C.: Superintendent of Documents, *U. S. Government Printing Office* 1954.

— Maximum permissible body burdens and maximum permissible concentrations of radionuclides in air and water for occupational exposure. *National Bureau of Standards* handbook 69. Washington, D. C.: Superintendent of Documents, *U. S. Government Printing Office* 1959.

PENDLETON, R. C.: Iodine-131 in Utah during July and August 1962. Science **141**, 640 (1963).

PERKINS, R. W.: Physical and chemical form of I^{131} in fallout. Health Phys. **9**, 1113 (1963).

PIERSON, D. H., and R. KEANE: Characteristics of early fallout from the Russian nuclear explosions of 1961. Nature **196**, 801 (1962).

POCHIN, E. E. (Chairman): Maximum permissible dietary contamination after the accidental release of radioactive material from nuclear reactor. Report of the

Medical Research Council by its Committee on Protection Against Ionizing Radiation. Brit. Med. J. **I**, 967 (1959).

POTTER, G. D., and I. L. CHAIKOFF: Identification of radioiodine compounds in the milk of lactating rats injected with iodine-131. Biochim. et Biophys. Acta **21**, 400 (1956).

RAYMOND, A. E., and G. W. WILLIAMS: Effect of the evaporation and powdering processes on the iodine-131 content of milk. Radiological Health Data (*U. S. Department of Health, Education, and Welfare, Public Health Service*) **5**, 70 (1964).

SMITH, H., and E. L. WHITEHEAD: A rapid method for estimation of I-131 in milk. Nature **199**, 503, (1963).

SOLDAT, J. K.: The relationship between I-131 concentrations in various environmental samples. Health Phys. **9**, 1167 (1963).

SQUIRE, H. M., L. J. MIDDLETON, B. F. SANSOM, and C. R. COIR: The metabolism of dairy cow of fission products from operation Buffalo. *United Kingdom Atomic Energy Authority,* T57/58, p. 119 (1959).

STEENBERG, K.: Secretion of I^{131} from a dairy cow after an oral administration of a single dose in aqueous solution. Acta Agr. Scand. **9**, 198 (1959).

STRAUB, C. P., and J. H. FOOKS: Cooperative field studies on environmental factors influencing I-131 levels in milk. Health Phys. **9**, 1187 (1963).

SULLIVAN, W. H.: Trilinear chart of nuclides. Revision sheets no. 1-10, July 1958-June 1962. *United States Atomic Energy Commission* 1957.

TURNER, F. B.: Quantitative relationship between fallout radioiodine on native vegetation and the thyroid of herbivores. Health Phys. **9**, 1241 (1963).

United Nations: Report of the *United Nations Scientific Committee on the Effects of Atomic Radiation.* Gen. Assembly, Official Records; Seventh Seminar, Suppl. no. 16 (A/5216), pp. 234-257. New York: United Nations (1962).

U. S. Department of Health, Education, and Welfare (Public Health Service): I-131 concentration in milk. Radiological Health Data **2**, 484 (1961 a); **2**, 522 (1961 b); **3**, 86 (1962 a); **3**, 122 (1962 b); **3**, 150 (1962 c); **3**, 189 (1962 d); **3**, 249 (1962 e); **3**, 303 (1962 f); **3**, 340 (1962 g); **3**, 416 (1962 h); **3**, 440 (1962 i); **3**, 486 (1962 j); **4**, 24 (1963 a); **4**, 89 (1963 b); **4**, 95 (1963 c); **4**, 140 (1963 d); **4**, 145 (1963 e); **4**, 185 (1963 f); **4**, 194 (1963 g); **4**, 236 (1963 h); **4**, 294 (1963 i); **4**, 362 (1963 j); **4**, 407 (1963 k); **4**, 511 (1963 l).

VAN MIDDLESWORTH, L., A. H. TUTTLE, and A. THREIKLANDS Iodinated protein in milk. Science **118**, 749 (1953).

WEAVER, L. E., P. O. STROM, and P. A. KILLEEN: *U. S. Naval Radiological Defense Laboratory,* San Francisco, Calif. USNRDC-TR-633 (1963).

WEHMANN, G.: Comparison of ingestion to inhalation dose to man from I^{131}. Health Phys. **9**, 1221 (1963).

WOODMAN, F. J., T. G. CLINTON, W. FLETCHER, and G. A. WELCH: Developments in analytical service given to industrial atomic energy. In: 2nd Internat. Conf. on Peaceful Uses of Atomic Energy. Geneva, United Nations. A/CONF. 15/P/1453 p. 12 (1958).

WRIGHT, W. E., J. E. CHRISTIAN, and F. W. ANDREWS: The mammary elimination of radioiodine. J. Dairy Sci. **38**, 131 (1955).

YAMAGATA, N.: Environmental contamination with iodine-131 in Japan. Nature **193**, 892 (1962).

— *In vivo* measurement of iodine-131 in the thyroid of the dairy cow as an aid for monitoring milk. Nature **198**, 169 (1963).

—, and K. IWASHIMA: Environmental contamination of short-lived radionuclides in Japan in 1961. J. Radiation Research **3**, 48 (1962).

Carcinogenic hazards from pesticide residues

By

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Contents

"Ideally the slightest *real* suspicion of carcinogenicity ought to be enough to prevent a substance from being allowed in the human environment whereas the most rigid proof ought to be insisted upon when the result is to be used as evidence upon which to base further work or to support a theory of carcinogenesis. In practice the reverse is true." (from CLAYSON 1962)

I. Introduction

The possibility that residues of pesticides present in food eaten by man could lead to an increased risk that the consumer might develop cancer has been considered twice in the preceding twelve volumes of *Residue Reviews*. OSER (1962) discussed the experimental studies that are made or should be made in experimental animals in order to provide evidence as to whether or not the substance under investigation had a carcinogenic action on these animals. He emphasized the lack of agreement on the best experimental methods to be employed and pointed out the difficulties of interpreting the findings in animal experiments in such a way that would enable anyone to indicate the hazard the substance under test might present to man if present in his food. DURHAM (1963) in a very comprehensive review of the health hazards that might arise from pesticide residues present in food discussed the difficulties of obtaining any information on the effects on man. He also considered in

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some detail the problems raised by residues of Aramite[2-(*p*-tertiary-butylphenoxy)-isopropyl-2-chloroethyl sulphite], aminotriazole, dichlorodiphenyl-dichloroethane (DDT), and dieldrin. In varying degrees suspicion had been cast on these pesticides because of reports in the literature of their ability to produce tumours in experimental animals under some circumstances. Arsenic was also discussed because there is some epidemiological evidence that it may be carcinogenic for man. Neither in the field of animal tests for carcinogenic action nor in that of their interpretation have any striking advances been made since the above reviews were written. A good deal of emotion about health hazards from pesticides has been engendered in the past few years. There is no evidence that pesticide residues found in food from crops and products treated in an approved manner can cause any ill-effects. If any pesticide did eventually prove to be carcinogenic for man it would be some of those who might be heavily exposed during its use who would develop the disease.

In this short review a critical discussion of some of the information on the activity of chemical carcinogens will be attempted in order to try to get the problem of any carcinogenic hazards presented by pesticides residues into some kind of perspective. There is no simple solution to the problem such as the banning of all pesticides. This would not necessarily make human food significantly less hazardous from the point of view of a carcinogenic danger. One of the most powerful chemical carcinogens yet discovered is a natural product from fungal metabolism (BARNES and BUTLER 1964). The fungus *Aspergillus flavus* can produce aflatoxin on a variety of foods and it is conceivable that under some circumstances the use of an effective fungicide, even if it left a residue in the food, might reduce any hazards to the consumer by preventing the appearance of aflatoxin. Similarly the pyrrolizidine alkaloids present in certain weeds common in pastures and which are excreted in milk (SCHOENTAL 1959) may be eliminated from human food by the judicious use of selective herbicides that show none of the carcinogenic activity of the alkaloids.

II. Suspicion of carcinogenic activity

The statement made by CLAYSON (1962) quoted above the introduction must be considered carefully. The essential point is the meaning of "real suspicion". This was well illustrated in the case of Aramite. When this was shown to be able to product malignant tumours in the biliary system of dogs on a diet containing 500 parts per million (p.p.m.) its carcinogenic activity was undisputed (STERNBERG *et al.* 1960). Its use was immediately forbidden under conditions where it might leave residues in human food. However, the possibility that any individual either had or ever would have consumed more than a few microgrammes of Aramite in the form of a residue on fruit seems rather unlikely. It would seem to be far more important that the manufacture and use of Aramite should only be carried out, if at all, under conditions in which no exposure of man can occur. There is no doubt at all that the heaviest exposure of individuals occurs during the application of pesticides and not from their presence as residues in food. However, pesticides as residues in food may be consumed by any section of the population including the very young. There is evidence that newly born and very young animals are much more sensitive than older animals to the action of some carcinogens.

The location of the tumours in the biliary tract of the dogs given Aramite was believed to result from the excretion of Aramite, or a metabolic product, in the bile and the possible concentration of these in the gall bladder. However, the mouse is not affected by Aramite (OSER and OSER 1962) so that the mouse may metabolize or excrete Aramite by different pathways. Thus, we cannot be certain whether Aramite in any dose would be carcinogenic for man but in practice one must assume that any compound carcinogenic for a single animals species will also be carcinogenic for man. However, the probability that a compound is carcinogenic for man will be even greater when it can be shown that many different animals species are all sensitive to its carcinogenic action. If the matter was important it might be possible to study the metabolism and excretion of Aramite in a sensitive species and in an insensitive species and then to compare the ways in which man dealt with Aramite. This would involve a great deal of work as well as an element of risk for the human subjects of such a study and the results might in the end remain impossible to interpret. The alternative to such an investigation is to assume that Aramite is a carcinogen for man as well as for dogs and rats and remove it from the human environment.

Aminotriazole presents rather a different problem because it belongs to a group of substances that have a common action in interfering with the synthesis of thyroxin and causing a hypertrophy of the thyroid gland. If the stimulus persists, the thyroids of rats are liable to develop tumours. The question as to whether aminotriazole is itself a carcinogen or whether it only acts by causing a prolonged stimulation of the thyroid gland through the thyroid-stimulating hormone of the pituitary is a matter for discussion. A good many other compounds with a similar antithyroid action such as thiourea, thioacetamide, and methylthiouracil can also lead to thyroid tumours in animals. Although PURVES and GRIESBACH (1947) found only thyroid tumours in their rats fed thiourea, others have produced liver tumours with thiourea (FITZHUGH and NELSON 1948). Very large doses of aminotriazole can also produce liver tumours. (NAPALKOV 1965). On the other hand the human thyroid is subject to stimuli of a nature similar to that produced by aminotriazole from goitrogens present in certain foods such as members of the brassica family. Furthermore, there are many thousands of human beings in the world who have received drugs of the aminotriazole type for thyroid disorders and there is no published evidence that such people have a higher incidence of thyroid cancer than those people with similar thyroid disorders treated by different means.

The cases of Aramite and aminotriazole have been outlined because they illustrate some of the problems that must be discussed in trying to assess the meaning of "real suspicion" of carcinogenic action and the nature of a carcinogenic hazard from pesticides. Aramite is an undoubted carcinogen for dogs and it must, in the absence of evidence to the contrary, be assumed to be a carcinogen for man. Aminotriazole can produce tumours in animals and in suitable large doses possibly might do so in man but so much more is known about its mode of action and the response of man to drugs with a similar action that it is possible to assess with much greater precision any hazards its use as a pesticide presents to man. In other words there is no doubt that any stimulus to the thyroid provided by residues of aminotriazole in cranberries or other fruit would fade into insignificance when compared with the stimuli received from goitrogens naturally present in other widely consumed foods such as brassica. The same considerations undoubtedly apply to those other pesticides, particularly of the dithiocarbamate series, that may also stimulate the thyroid gland when given in large doses. Whether or not aminotriazole and thiourea should be considered as carcinogens likely to present a hazard because of their effects on other tissues will be considered below in discussing the potency of carcinogens.

III. Nature of chemical carcinogenesis

No one would contemplate the introduction of a compound as a pesticide when animal tests had shown it to have an unmistakable carcinogenic activity. The animal tests for toxicity normally demanded by those who register or approve new compounds for use as pesticides will demonstrate whether a compound has a high degree of carcinogenic action as was the case with 2-acetyl-aminofluorene (WILSON *et al.* 1947). Tumours appeared in some animals within 90 days. However, in some instances, as with Aramite, a final demonstration of carcinogenic action was not achieved until some years after it had been introduced as a pesticide. Thus, it becomes necessary to consider the possible hazard to consumers that might arise from the use of a pesticide for some years before it was withdrawn because new evidence from animal tests showed that it had a carcinogenic action on animals.

The other problem to discuss is that relating to the use of materials whose carcinogenic activity is suspected but where the interpretation of the laboratory experiments is open to doubt. In order to consider any hazard to human health from residues of pesticides falling into either of these above categories it is necessary to take into account such matters as: (a) mechanisms of chemical carcinogenesis, (b) dose-response relations to chemical carcinogens, (c) cumulative carcinogenic effects, (d) additive effects of carcinogens, and (e) co-carcinogenic and other factors affecting the action of carcinogens.

Although there is no agreement or conclusive evidence about the mechanism of carcinogenesis resulting from exposure of animals to chemicals, current interest centres on the importance of reactions that have been demonstrated between some carcinogens and cellular deoxyribonucleic acid (DNA). This has been discussed by MAGEE (1962) with special reference to dimethylnitrosamine. This compound and other simple alkyl nitrosamines are some of the most versatile and active carcinogens known (MAGEE and BARNES 1966). The reaction of dimethylnitrosamine and other nitroso compounds with tissue constituents after giving them to whole animals has been studied in some detail. It has been shown that the guanine of both ribonucleic acid (RNA) and DNA in liver cells is methylated in the seven position. The demonstrations that the infective part of viruses may be either DNA or RNA and that some viruses can produce cancer provides a possible link between the mechanism of chemical and viral carcinogenesis. It should be emphasised again that it is only a hypothesis that chemical changes induced in cellular DNA or RNA are the precursors of carcinogenic transformation. The hypothesis can be further extended. Changes in DNA, provided they are not so severe as to damage the cell so that it dies, could lead to mutations so that in subsequent multiplication abnormal cancer cells developed. Alternatively, changes in RNA by modifying protein synthesis could lead to loss of essential controlling enzymes—"the enzyme deletion" hypothesis or loss of "the identifying proteins" or "recognition units" within cells which could involve an immunological mechanism as a cause of cancer.

Even if one accepts the assumption that changes in DNA and/or RNA are an essential part of the action of chemical carcinogens, it is not yet possible to make use of the hypothesis for predicting carcinogenic activity in chemical compounds, including those proposed as pesticides. It is certain that any changes in the nucleic acids must be of a rather subtle nature and may of course be produced by a metabolite derived from the original chemical under examination. All that can be said is that a chemical which shows evidence of interfering with cell division should be regarded with special suspicion as a possible carcinogen but it cannot be assumed that a substance that does not appear to react with cellular DNA and RNA is not carcinogenic.

IV. Dose-response relations

Despite a very large literature on the response of animals to chemical carcinogens there is very little information on dose-response relationships. The difficulties in acquiring the data necessary for establishing such relationships can readily be appreciated but the virtual absence of such information makes the problem of assessing "safe" or "ineffective" doses of carcinogens very difficult. In the case of all chemical carcinogens studied hitherto there is a delay of at least three to four months and often much longer between the first dose of the carcinogen and the appearance of the malignant change in the animal's tissues. The response to a carcinogen may be measured by the latent period between the first dose and the first tumour and by the types and frequency of the tumours in the treated animals. In studying the acute toxicity of compounds, simple dose-mortality data may make it possible from figures for the doses killing 75 percent and 25 percent of the animals to calculate both the LD_{50}, the LD_1 or $LD_{0.1}$ because it can be assumed that survival beyond a limited time, usually a week or two at the most, can be assumed to indicate indefinite survival. With chemical carcinogens however it is known that as the dose is reduced so does the interval between first exposure and the appearance of the first tumour also increase. With the small laboratory animals frequently used for such tests the latent interval may rapidly approach that of the total life span of the animal. Where healthy rats and mice are used, that is animals free from specific pathogenic organisms, survival may be increased but a larger proportion of the animals will eventually die with malignant tumours even though no specific or known carcinogen has been added to their environment. It would therefore only be possible to test the effects of very low dose levels of those carcinogens which produced a type of tumour that did not occur spontaneously in the test animals. Surveys of spontaneously occurring tumours in rats used in chronic toxicity studies indicate a wide range of organs and tissues that may be affected by such spontaneous tumours (CRAIN 1958).

While it will be impossible to demonstrate experimentally a safe dose of a carcinogen there is a certain amount of evidence that the speed and frequency with which tumours develop is related to the dose of the carcinogen. What is not clear is the importance of total dose in relation to duration and degree of exposure. GLINOS *et al.* (1951) fed groups of rats for different periods on a diet containing 600 p.p.m. of dimethylaminoazobenzene (DAB) and observed them for up to one year. Only one of 22 rats receiving the diet for 30 days developed a liver tumour but 18 out of 24 rats who got the diet for 50 days developed the tumours. This ratio was not increased by prolonging the feeding for 135 days. However it is not certain whether, if observations had been carried on for another year, a greater incidence of tumours might have been observed.

DRUCKREY (1952) summarized many studies with DAB in which rats were given a single dose each day. Assuming the rats ate 15 grams of food per day it is possible to give a theoretical concentration in the food which would have provided the amounts represented by the single doses actually given. At a nominal level of 2,000 p.p.m. rats developed liver tumours within an average of 34 days. At 200 p.p.m. it took an average of 350 days for tumours to appear and at 70 p.p.m., 700 days. At 20 p.p.m. and seven p.p.m. no tumours developed in a total of 306 rats studied. MACDONALD *et al.* (1952) found that of rats given a diet containing 600 p.p.m. DAB for 150 days 13 out of 16 had liver tumours by 210 days whereas on a diet containing 300 p.p.m. none out of 16 had developed tumours. A clear dose-response relation exists for the carcinogenic action of DAB on the liver. However, the data do suggest that there will be a sharp decline in the incidence of tumours as the dose is reduced.

In work in our laboratory it is clear that the very powerful liver carcinogen, dimethylnitrosamine, also displays a steep dose-response curve. Whereas rats fed 50 p.p.m. in their diet all developed liver cancer in less than 364 days, on a diet containing five p.p.m. there were only two out of 27 rats surviving more than 500 days with liver tumours and on a diet containing two p.p.m. none of the 23 rats surviving 525 days or more developed tumours. However, the more active analogue, diethylnitrosamine, has been shown by DRUCKREY and SCHMAHL (1962) to produce liver cancer in 67 percent of rats after an average of 457 days at a daily dose of 0.3 mg./kg. in drinking water which, assuming a food intake of 15 grams daily, is equivalent to four p.p.m. in the diet. However one must emphasize again that carcinogens of this degree of activity would be detected in the early toxicity tests.

In the case of Aramite where a residue of one p.p.m. was at one time permitted on fruit, rats on a diet containing 100 p.p.m. developed no histological abnormalities in their livers. At 200 p.p.m. there were some histological changes in the livers but only at 400 p.p.m. were two tumours found in 96 rats (POPPER *et al.* 1960). It seems very improbable that anyone who consumed fruit containing one p.p.m. during the years Aramite was available would have been exposed to any significant carcinogenic hazard.

If animals are exposed to carcinogens only for limited periods the time between exposure and the development of tumours is increased. There is also convincing evidence that man responds in the same way. When cigarette smokers give up smoking their chances of developing lung cancer decrease rapidly. Within five years of abstaining their chances are halved and after 20 years or more are less than 1/6 of those who continue smoking. However, these people are still two to three times more likely to get lung cancer than non-smokers (DOLL and BRADFORD HILL 1964).

DRUCKREY (1952) who has done a good deal of work on the dose-response relations to carcinogens is convinced that each dose of a carcinogen leaves an indelible mark on the sensitive tissues and that each such insult is additive although presumably only in the case of two or more doses of the same carcinogen. There is evidence that different carcinogens have additive effects upon each other where the target organ is the same. This has been shown to be the case with liver tumours in rats (MACDONALD *et al.* 1952) and bladder cancer in dogs (DEICHMANN *et al.* 1965). However, the reverse may happen and it has been shown that the carcinogen methylcholanthrene, if added to the diet of rats, will inhibit the formation of liver tumours by methyl-DAB, and tumours in four tissues by acetylaminofluorene. The explanation of this action probably centres round the influence of one carcinogen on the metabolism of the other (MILLER *et al.* 1958) (see below). It is of course impossible to prove or disprove the idea that any dose of a carcinogen will produce an indelible effect on the target organ but it is quite clear that if the effect is small enough there will not be enough time in the average life span to acquire a dose large enough to initiate a frank tumour. The fact that the incidence of tumours rises with age in man and animals may reflect the additive effects of many unknown carcinogens. Equally it could represent a breakdown in those defense mechanisms that resist the development of tumours from what, in young tissues, are submaximal stimuli.

DRUCKREY and SCHMAHL (1962) believe that for any carcinogen the expression $d \times t^n = k$ holds good, where d is the total dose of the carcinogen and t is the time during which exposure takes place, while n is a characteristic for each carcinogen and is a measure of its potency.

V. Carcinogenic activity of pesticides

The substances DAB, diethylnitrosamine, and diaminostilbene studied by DRUCKREY and SCHMAHL (1962) will produce a reasonable proportion of cancers when given to the animals continuously at levels corresponding respectively to 400 p.p.m., four p.p.m. and 1.3 p.p.m. in the diet. The figures are derived by extrapolation from the dose given to each rat each day and assuming a food intake of 15 grams per day. Clearly there is a marked difference between the activity (n) of DAB when compared with the other two carcinogens. In our own experiments over 50 percent of rats on a diet containing only 0.1 p.p.m. of aflatoxin, a natural contaminant of some foods, will develop malignant liver tumours (BARNES and BUTLER, unpublished data) so that aflatoxin is probably even more active than the compounds studied by DRUCKREY and co-workers.

To assess the carcinogenic hazard from pesticide residues in food it is necessary to consider three points in respect to the pesticide under suspicion:

a) Can it regularly produce tumours in experimental animals under specified conditions?

b) Does the response show a relation to the dose and duration of exposure?

c) How does the activity as a carcinogen compare with that of compounds generally accepted as being carcinogenic when given under similar conditions?

No one disputes the carcinogenic activity of Aramite yet at 400 p.p.m. only two of 96 rats had liver tumours in exposures up to two years (POPPER *et al.* 1960). At levels of 500 to 1,429 p.p.m., 15 of 19 dogs observed for 462 to 1,220 days had malignant lesions (STERNBERG *et al.* 1960). This indicates that Aramite is certainly not more active than DAB.

Thiourea used as a fungicide has been condemned as a hazard because of its carcinogenic activity. In groups of rats receiving 100, 250, 500, and 1,000 p.p.m. of thiourea in their food 29 survived two years and 14 of these had liver tumours which did not metastasise (FITZHUGH and NELSON 1948). No details of the incidence in the groups on different levels of thiourea are given.

When aminotriazole was fed at 100 p.p.m. in the diets of rats for two years only four of 26 rats had tumours in their thyroids which were interpreted by some pathologists as malignant (JUKES and SCHAFFER 1960). This compound is certainly not a very active thyroid carcinogen for rats. To produce liver tumours 500 mg. of aminotriazole had to be given daily six days a week (NAPALKOV 1965).

If some doubt exists about the true carcinogenicity of thiourea and aminotriazole, even more exists about DDT, aldrin, and dieldrin. Nevertheless, because of the current interest in these last compounds and because of their undoubted presence in the human diet and, in very small concentrations, in our tissues the case against them needs careful scrutiny. The allegations that DDT may have a weak carcinogenic potential was first made by FITZHUGH and NELSON (1947). Rats received diets containing 100, 200, 400, 600, and 800 p.p.m. of DDT and there was no significant differences in the food intake of the groups. Out of 75 rats surviving 18 months or more, 15 had some liver tumours whereas only in one or two rats would such tumours have been expected to have occurred spontaneously. However, the most striking feature of this report is the complete absence of any reference to the frequency of tumours in the different groups. With an eight-fold difference in dose rates there should have been a striking difference in both the frequency and time of onset of the tumours in the groups on the highest and lowest doses if DDT had behaved like a true liver carcinogen of which many are known. Another feature of the report is that while a very detailed account is given of the hepatocellular changes in these animals there is no specific mention of inequality of cell size, of variability in size and character in cell nuclei, or other changes considered to be precancerous in animals receiving liver carcinogens.

The case against aldrin and dieldrin rests on papers by DAVIS and FITZHUGH (1962) in which mice were studied and by FITZHUGH *et al.* (1964) in which rats were studied. Mice fed aldrin and dieldrin at ten p.p.m. had four times as many hepatomas as untreated controls. No histological details are provided but the tumours are described as benign. It is impossible to describe aldrin and dieldrin as carcinogens for mice on this evidence. In the experiment with rats, groups received 0.5 2.0, 10.0, and 50 p.p.m. of aldrin or dieldrin in their diets. Only those rats receiving the two lower dose levels survived in adequate numbers to make possible a valid assessment of the tumour incidence among them. However, the incidence of tumours was 15 out of 42 rats on 2 p.p.m. but 18 out of 41 in those on 0.5 p.p.m. Thus a four-fold increase in dose caused no increase in the tumour incidence.

It is reasonable to conclude that neither DDT, aldrin, nor dieldrin behaved like a typical liver carcinogen in the experiments just outlined so that another cause of the differences in the incidence of liver tumours must be sought.

Maleic hydrazide was developed as a herbicide and sprout depressant and appeared to have virtually no toxicity to mammals. The fact that it could produce chromosome breaks in plant cells led to a suggestion that it might be carcinogenic to mammals. In a very comprehensive series of investigations, including the feeding and injection of maleic hydrazide into rats and mice as well as a number of ancillary biological investigations it was concluded that maleic hydrazide had no carcinogenic action (BARNES *et al.* 1959). Recent work involving the testing of a series of lactones for carcinogenic action led DICKENS and JONES (1965) to inject six rats subcutaneously twice weekly for 65 weeks with a suspension of finely ground maleic hydrazide in arachis oil. Three of these rats developed sarcomata at the site of injection. In discussing their findings they imply that the safety of maleic hydrazide to the consumers of food containing residues after its use as a sprout depressant needs reappraisal. This review is not the place to discuss the significance of tumours formed at the site of injection of compounds that are contained in food and where the hazard to man is only a food contaminant. Clearly a compound that produced tumours at the site of injection must be tested in feeding experiments. In the case of maleic hydrazide such experiments gave completely negative results. By contrast, aflatoxin, another lactone mentioned previously, not only gives tumours at the site of injection (DICKENS and JONES 1965) but also just as readily when given by mouth (BARNES and BUTLER 1964). It may be concluded that under some conditions maleic hydrazide can exert a carcinogenic effect but the experimental conditions under which it does this do not in any way resemble those in which it could present a hazard to the human consumer of food containing maleic hydrazide. Tested under those conditions it showed no carcinogenic action even on the species sensitive to its effects when injected. However, rather than pay too much attention to maleic hydrazide as a hazard it might be more appropriate for DICKENS and JONES to investigate further the substance bovolide which produced sarcoma in five of the six rats injected and which is a natural ingredient of butter.

VI. Factors modifying chemical carcinogenesis

Although it cannot be predicted whether, when two carcinogens are given simultaneously, they will potentiate, annul, or have no effect on each other, a recent paper reports experiments in which groups of rats were given diets containing 80 p.p.m. of Aramite, of DDT, of methoxychlor, and of thiourea and a further group a diet containing 80 p.p.m. of each. There was no difference in the tumour incidence among rats in all these groups and a group on a normal diet (RADOMSKI *et al.* 1965).

The question as to whether other substances not carcinogenic in themselves can influence the development of tumours or increase the sensitivity of animals to carcinogens is largely unexplored in the case of carcinogens given by mouth. The theory of a two-stage development in cancer with "initiators" and "promoters" was derived from a study of skin cancer and the work has not been shown to have any direct relevance to hazards from carcinogens in food. Whether similar mechanisms can operate in the case of carcinogens producing tumours at sites remote from their initial contact with tissues is unknown. In a recent paper discussing the carcinogenic potential of pesticides a possible role for the non-toxic methylenedioxy derivatives (piperonyl butoxide, etc.) is suggested (FALK *et al.* 1965). It is known that these compounds exert a synergistic effect on the action of insecticides by delaying the metabolism of the poison in the insect. Piperonyl butoxide has a similar effect in the mammal and delays the detoxication of insecticides and barbiturates. FALK *et al.* (1965) have shown that piperonyl butoxide will also delay the metabolism of 3, 4-benzpyrene in mammals. They therefore imply that an innocent compound like piperonyl butoxide might enhance the carcinogenic hazards of our environment. They have completely failed to take into account that for some carcinogens a delay in the rate of their metabolism might greatly increase their safety because it is the metabolites that are carcinogenic. Indeed, MILLER *et al.* (1958) as mentioned above found that two active carcinogens given simultaneously might have less effect than when each was given alone because they depressed the rate at which each other was metabolized.

The need to be cautious in interpreting this type of experimental data in terms of hazard must be emphasized.

VII. Conclusions

Only when we have a detailed analysis of the mode of action or modes of action of chemical carcinogens will it be possible to investigate with any accuracy the present or absence of carcinogenic hazards from pesticide residues in food. No one disputes the policy that a substance which displays an unequivocal carcinogenic effect on animals or man should as far as possible be removed from the human environment. No substance with such properties should ever be allowed to contaminate food. Indeed, it should not be allowed to contaminate anyone whether during manufacture or use. The safety of man from hazards presented by pesticide residues will not necessarily be increased by crying "wolf" on every conceivable occasion that some remote suspicion of some possible direct or indirect carcinogenic activity can be detected in a substance filling a valuable role as a pesticide. Without pesticides many people would die for other reasons long before they reached the age at which they might develop cancer. Cancer was widespread long before modern pesticides were synthesized. If chemical carcinogens are responsible for any significant fraction of human cancer of unknown origin it is probable that such carcinogens will be of natural origin. With aflatoxin, cycasin, and the pyrrolozidine alkaloids before us as examples of carcinogens found widespread in nature, it would be unwise, if not irrational, to try to create undue alarm about carcinogenic hazards from pesticides that display no carcinogenic activity even faintly comparable with that of the compounds listed above.

Meanwhile a very great deal more is waiting to be discovered by quite simple, if rather unexciting, experiments about the dose-response relations to known carcinogens so that suitable experiments will make it possible to compare new compounds such as pesticides and give a more reasonable assessment of any carcinogenic activity they may be found to possess under experimental conditions.

Summary

The possible carcinogenic hazard to consumers of food containing pesticide residues is considered. While no substance shown to have a high carcinogenic activity in animals (e.g., 2-acetylaminofluorene) is ever likely to be developed as a pesticide, other compounds may be shown to have some such activity after they have been in use for some time (e.g., Aramite). Other pesticides may come under suspicion owing to disagreement over the interpretation of animal experiments (e.g., aminotriazole, DDT, dieldrin, and maleic hydrazide).

Current ideas on the mechanisms of chemical carcinogenesis are briefly considered and the difficulty of establishing valid dose-response relations for carcinogens is stressed. Nevertheless, carcinogens vary greatly in the activity they show in experimental animals. Some of the most active are natural and not synthetic substances (aflatoxin, cycasin, and pyrrolizidine alkaloids). The effect of one carcinogen upon the activity of another and the modification of carcinogenic activity by other chemicals are mentioned.

The available evidence on pesticides under suspicion as carcinogens does not suggest they are likely to present a hazard as residues in food. The question as to whether a pesticide with undoubted carcinogenic activity should be withdrawn from the human environment and not just from use where it could contaminate food is considered.

Résumé*

L'éventualité de risques cancérogènes pour les consommateurs d'aliments contenant des résidus de pesticides est examinée. Si aucune substance pour laquelle une haute activité cancérogène a été mise en évidence chez l'animal comme par exemple l'acétylamino 2 fluorène ne saurait être appliquée comme pesticide, d'autres composés peuvent manifester une certaine activité de ce type après avoir été utilisés un certain temps, comme par exemple l'aramite. D'autres pesticides peuvent être considérés comme suspects, en raison des divergences sur l'interprétation des résultats des expérimentations sur les animaux de laboratoire, comme par exemple l'aminotriazole, le D.D.T., la dieldrine et l'hydrazide maléique.

Les opinions courantes sur les mécanismes de la cancérogénèse chimique sont sommairement examinées et la difficulté d'établissement de relations valables entre les doses et effets est soulignée. Cependant, l'activité manifestée par les cancérogènes varie grandement selon les spèces animales. Quelques uns des composés les plus actifs sont d'origine naturelle et non pas synthétique (aflatoxine, cycasine, alcaloides à noyau de pyrrazolidine). Il est fait allusion à l'influence que peut exercer un cancérogène sur l'activité d'un autre cancérogène ainsi qu'à la modification de l'activité cancérogène pouvant résulter de l'association avec d'autres agents chimiques.

Les informations concernant les pesticides suspects de posséder une

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* Traduit par R. TRUHAUT.

activité cancérogène ne sont pas en faveur de l'existence de risques liés à leur présence dans les aliments sous forme de résidus. Le problème de l'exclusion des pesticides possédant une activité cancérogène indiscutable non seulement des emplois pouvant être à l'origine d'une contamination des aliments mais encore du milieu humain en général est discuté.

Zusammenfassung*

Die mögliche Gefahr carcinogener Schädigung des Konsumenten durch Lebensmittel, die Schädlingsbekämpfungsmittel-Rückstände enthalten, wird erwogen. Während wahrscheinlich kein Stoff, der im Tierexperiment eine starke carcinogene Wirkung gezeigt hat (z.B. Acetylaminofluoren) jemals als Schädlingsbekämfungsmittel auf den Markt kommen wird, kann bei anderen Stoffen, die sich schon eine Zeitlang im Gebrauch befinden, u.U. eine deratige Wirkung nachgewiesen werden (z.B. Aramite). Andere Schädlingsbekämpfungsmittel können auf Grund von Meinungsverschiedenheiten über die Interpretation tierexperimenteller Ergebnisse in diesen Verdacht kommen (z.B. Aminotriazol, DDT, Dieldrin und Maleinsäurehydrazid).

Es werden die heute gängigen Vorstellungen vom Mechanismus chemischer Carcinogenese kurz betrachtet und die Schwierigkeiten, zu gültigen Dosis-Wirkungsbeziehungen für Carcinogene zu kommen, betont. Nichtsdestoweniger unterscheiden sich Carcinogene hinsichtlich ihrer an Versuchstieren gezeigten Wirksamkeit stark voneinander. Einige der wirksamsten Carcinogene sind natürliche und nicht synthetische Stoffe (Aflatoxin, Cycasin und Pyrrolizidinalkaloide). Die Wirkung eines Carcinogens auf die Wirksamkeit eines anderen und die Beeinflussung der carcinogenen Wirksamkeit durch andere chemische Stoffe werden erwähnt.

Die vorliegenden Beweise gegen Schädlingsbekämpfungsmittel, die carcinogenverdächtig sind, reichen nicht zur Annahme aus, dass sie als Rückstände in Lebensmitteln wahrscheinlich eine Gefahr darstellen. Die Frage, ob ein Schädlingsbekämpfungsmittel mit unzweifelhaft carcinogener Wirkung aus der menschlichen Umgebung ganz entfernt oder nur dort nicht mehr angewendet werden sollte, wo es Lebensmittel verunreinigen kann, wird behandelt.

References

BARNES, J. M., and W. H. BUTLER: Carcinogenic activity of aflatoxin to rats. Nature **202**, 1016 (1964).

—, P. N. MAGEE, E. BOYLAND, A. HADDOW, R. D. PASSEY, W. S. BULLOUGH, C. N. D. CRUICKSHANK, M. H. SALAMAN, and R. T. WILLIAMS: The nontoxicity of maleic hydrazide for mammalian tissues. Nature **180**, 62 (1957).

CLAYSON, D. B.: Chemical carcinogenesis, p. 55. London: Churchill 1962.

CRAIN, R. C.: Spontaneous tumours in wistar rats. Amer. J. Pathol. **34**, 311 (1958).

DAVIS, K. J., and O. G. FITZHUGH: Tumorogenicity of aldrin and dieldrin. Toxicol. Applied Pharmacol. **4**, 187 (1962).

DEICHMANN, W. B., T. SCOTTI, J. RADOMSKI, E. BERNAL, M. COPLAN, and F. WOODS: Synergism among oral carcinogens. II. Results of the simultaneous feeding of bladder carcinogens to dogs. Toxicol. Applied Pharmacol. **7**, 657 (1965).

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* Übersetzt von O. R. KLIMMER.

DICKENS, F., and H. E. H. JONES: Further studies on the carcinogenic action of certain lactones and related substances in the rat and mouse. Brit. J. Cancer **19**, 392 (1965).

DOLL, R., and A. BRADFORD HILL: Human lung cancer and smoking. Brit. Med. J. **1**, 1399 and 1460 (1964).

DRUCKREY, H.: Konstitution und Wirkung cancerogener Substanzen. Arzneimit. Forsch. **1**, 383 (1952).

—, and D. SCHMAHL: Quantitative Analyse der experimentellen Krebserzengung. Naturwiss. **49**, 217 (1962).

DURHAM, W. F.: Pesticide residues in foods in relation to human health. Residue Reviews **4**, 33 (1963).

FALK, H. L., SANDRA J. THOMPSON, and P. KOTIN: The carcinogenic potential of pesticides. Arch. Environmental Health **10**, 847 (1965).

FITZHUGH, O. G., and A. A. NELSON: Chronic oral toxicity of DDT. J. Pharmacol. **89**, 18 (1947).

— — Tumours in rats fed thiourea and thioacetamide. Science **108**, 626 (1948).

— —, and M. L. QUAIFE: Chronic oral toxicity of aldrin and dieldrin in rats and dogs. Food Cosmetic Toxicol. **2**, 551 (1964).

GLINOS, A. D., N. L. R. BUCHER, and J. C. AUB: I. The effect of liver regeneration on tumour formation in rats fed 4-dimethylaminoazobenzene. J. Expt. Med. **93**, 313 (1951).

JUKES, T. H., and C. B. SHAFFER: Antithyroid effects of aminotriazole. Science **132**, 296 (1960).

MACDONALD, J. C., E. C. MILLER, J. A. MILLER, and H. P. RUSCH: Synergistic action of mixtures of certain hepatic carcinogens. Cancer Research **12**, 50 (1952).

MAGEE, P. N.: Biochemical and pathological mechanism in experimental liver cancer. Scient. Basis of Med. Ann. Reviews, p. 172. London: Athlone Press 1962.

—, and J. M. BARNES: Carcinogenic activity of nitroso compounds. Ann. Review of Cancer (to be published).

— — The production of malignant primary hepatic tumours in the rat by feeding dimethylnitrosamine. Brit. J. Expt. Cancer **10**, 114 (1956).

MILLER, E. C., J. A. MILLER, R. R. BROWN, and J. C. MACDONALD: On the protective action of certain polycyclic aromatic hydrocarbons against carcinogenesis by aminoazo dyes and 2-acetylaminofluorene. Cancer Research **18**, 469 (1958).

NAPALKOV, N. P.: On blastomagenic effect of antithyroid drugs. Union Internationale Contre le Cancer Symposium, Paris (1965).

OSER, B. L.: The experimental induction of cancer by pesticide residues and food additives: its rationale and interpretation. Residue Reviews **1**, 1 (1962).

—, and M. OSER: 2-(*p-tert*-Butylphenoxy) isopropyl-2-chloroethyl sulphite (Aramite). II. Carcinogenicity. Toxicol. Applied Pharmacol. **4**, 70 (1962).

POPPER, H., S. S. STERNBERG, B. L. OSER, and M. OSER: The carcinogenic affect of Aramite in rats. A study of hepatic nodules. Cancer **13**, 1035 (1960).

PURVES, H. D., and W. E. GRIESBACH: Studies on experimental goitre. VIII. Thyroid tumours in rats treated with thiourea. Brit. J. Expt. Pathol. **28**, 46 (1947).

RADOMSKI, J. L., W. B. DEICHMANN, W. E. MACDONALD, and E. M. GLASS: Synergism among oral carcinogens. I. Results of the simultaneous feeding of four tumorigens to rats. Toxicol. Applied Pharmacol. **7**, 652 (1965).

SCHOENTAL, R.: Liver lesions in young rats suckled by mothers treated with pyrrolizidine alkaloids. J. Pathol. Bact. **77**, 485 (1959).

STERNBERG, S. S., H. POPPER, B. L. OSER, and M. OSER: Gall bladder and bile duct adenocarcinoma in dogs after long term feeding of Aramite. Cancer **13**, 780 (1960).

WILSON, R. H., F. DEEDS, and A. J. COX: The carcinogenic activity of 2-acetaminofluorene. II. Effects of concentration and of duration of exposure. Cancer Research **7**, 444 (1947).

Insecticide residues in soils

By

C. A. Edwards*

Contents

I. Introduction

In the past twenty years increasing quantities of very stable insecticides have reached agricultural soils. Much of these residues comes from foliage sprays or dusts which miss their targets and fall onto the soil either close

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Table Ia. *Investigations into soil residues of chlorinated hydrocarbon insecticides*

Reference	Residues reported on								
	DDT	Dieldrin	Lindane	Chlordane	Heptachlor	Aldrin	Endrin	Isodrin	Toxaphene
ACKLEY *et al.* (1950)	x								
ALLEN *et al.* (1954)	x		x						x
BANHAM (1961)		x		x		x			
BARLOW and HADAWAY (1955)	x	x	x			x			
BAMI (1961)	x								
BARTHEL *et al.* (1960)					x				
BERTAGNA (1959)	x	x	x						
BOLLEN *et al.* (1958)						x			
BOSWELL *et al.* (1955)	x		x	x					
CHISHOLM *et al.* (1950)	x								
CHISHOLM *et al.* (1951)	x								
EDWARDS *et al.* (1957)			x			x			
FLEMING *et al.* (1951)									x
FLEMING and MAINES (1953)	x								
FLEMING and MAINES (1954)				x					
FOSTER (1951)	x		x	x					x
FOSTER (1956)	x	x	x	x	x	x	x	x	x
GANNON and BIGGER (1958)					x	x			
GALLAHER and EVANS (1961)	x								
GEROLT (1961)	x	x	x			x			
GINSBERG (1955)	x								
GINSBERG and REED 1954)	x								
KIIGEMAGI *et al.* (1958)		x			x	x			
KINCAID *et al.* (1960)	x		x				x		
KIRK (1952)	x								
HARRIS (1961)		x	x		x	x			
HARRIS and LICHTENSTEIN (1961)	x	x	x		x	x			
LICHTENSTEIN (1957)	x								
LICHTENSTEIN (1958 b)	x		x			x			
LICHTENSTEIN and SCHULZ (1959 a)			x			x			
LICHTENSTEIN and SCHULZ (1959 b)	x		x		x	x			
LICHTENSTEIN and POLIVKA (1959 c)			x	x	x	x			
LICHTENSTEIN and SCHULZ (1961 a)	x					x			
LICHTENSTEIN and SCHULZ (1960)					x	x			
LICHTENSTEIN and SCHULZ (1965)					x	x			
LICHTENSTEIN *et al.* (1960)	x		x			x			
LICHTENSTEIN *et al.* (1963)						x			
LICHTENSTEIN *et al.* (1964)						x			
MORRISON and CROWELL (1952)									
MULLA (1960)	x					x	x		
MULLA (1961 a)	x	x				x	x		x
ROBERTS *et al.* (1962)	x		x						
ROBERTS (1963)		x							
SCHREAD (1949)	x		x	x	x				x
SHOREY *et al.* (1958)		x	x		x	x	x	x	
TERRIERE and INGALSBE (1953)	x	x	x	x	x	x			x
WHEATLEY (1962)	x	x	x			x			
WILKINSON *et al.* (1964)					x	x			
YOUNG and RAWLINS (1958)					x				

to the plants, or after drifting, especially as very fine mists from the low-volume sprays now in common use. It has been estimated that as much as 50 percent of the sprays applied to foliage may reach the soil in this way. Even some that is applied to foliage, reaches the soil when it is washed or blown off crops or when the plant remains are ploughed into the soil. The second main source of insecticide residues in soil is from the large quantities of insecticides applied directly to it to control soil-inhabiting pests, as sprays, dusts, or granules: these are cultivated into the soil or mixed and drilled with fertilizer as dressings.

The most common insecticide residues in soils are chlorinated hydrocarbons of which DDT[1] and dieldrin are the most widespread. Organophosphorus and other insecticides in current use in soils break down so rapidly that measurable quantities rarely exist for many months after they are applied. An ideal soil insecticide would not be toxic to mammals, have no harmful effects on crops, microflora or microfauna, be slightly systemic, and would persist for about a year; so far none of the chemicals used has all these desirable qualities.

The breakdown of insecticides in soil has been less studied than that of herbicides, about which several reviews have appeared in recent years (ALDRICH 1953, NEWMAN and DOWNING 1958, BAILEY and WHITE 1964). The persistences of insecticides have been briefly reviewed (EDWARDS 1964).

There has been much work determining levels of residues of chlorinated hydrocarbon insecticides in soils by analytical and bioassay techniques; the

Table Ib. *Investigations into soil residues of organophosphorus insecticides*

Reference	Residues reported on										
	Parathion	Phorate	Dimethoate	Phosdrin	Demeton methyl	Malathion	Guthion	Trithion	Ethion	Diazinon	Di-Syston
BOHN (1964)			x								
FOSTER (1951)	x										
GETZIN and CHAPMAN (1959)				x							
GETZIN and CHAPMAN (1960)		x									
GETZIN (1958)		x		x	x						
HARRIS (1961)		x									
HARRIS and LICHTENSTEIN (1961)	x	x				x					
LICHTENSTEIN (1958 b)	x										
LICHTENSTEIN and SCHULZ (1964)	x										
MULLA (1960)	x		x				x		x		
MULLA (1961 b)							x	x		x	x
MENN *et al.* (1960)								x			
PARKER and DERVEY (1965)		x									
ROBERTS *et al.* (1962)						x	x				
SCHREAD (1949)	x										
SHOREY *et al.* (1958)	x										
TERRIERE and INGALSBE (1953)	x										

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[1] See Table X for chemical designations of pesticides mentioned in text.

more important papers and chemicals investigated are listed in Tables I *a* and *b*. The estimates of persistence have differed greatly; it is not proposed to list the various data in detail except to illustrate the effect of specific factors. EDWARDS (1964) calculated regressions from all results available for the rate chlorinated hydrocarbon insecticides disappear and these are summarized in Figure 1 and Table II.

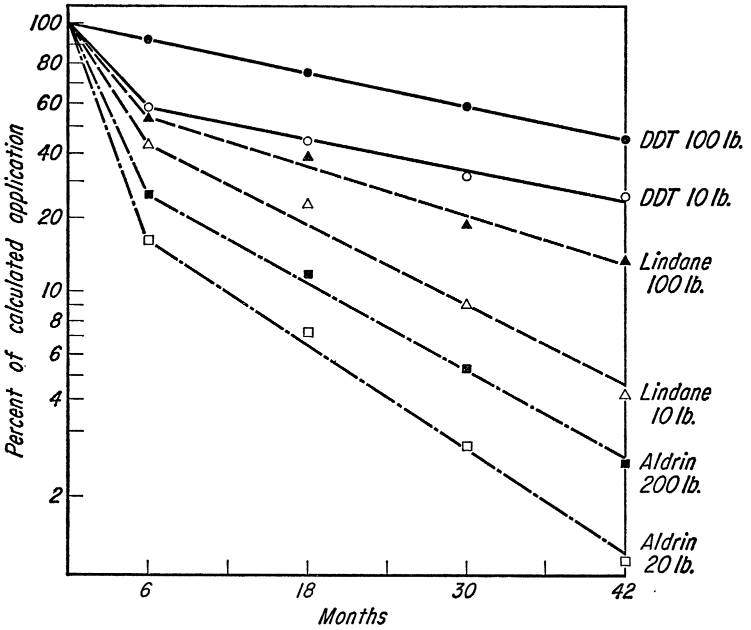

Fig. 1. Effect of concentration on loss of DDT, lindane, and aldrin from a Miami silt loam (after LICHTENSTEIN and SCHULZ 1959)

Table II. *Persistence of some chlorinated hydrocarbon insecticides in soil* (based on all available data)

Chemical	Average dosage active ingredient/acre, lbs.[a]	Time for 95% disappearance,[b] years[b]
Aldrin	1 – 3	1 – 6 (3)
Chlordane	1 – 2 (6)	3 – 5 (4)
DDT	1 – 2½ (10)	4 – 30 (10)
Dieldrin	1 – 3	5 – 25 (8)
Heptachlor	1 – 3	3 – 5 (3½)
Lindane	1 – 2½ (5)	3 – 10 (6½)
Telodrin	¼ – 1	2 – 7 (4)

[a] Bracketed figures are doses which may be used for particular pests in unusual circumstances.

[b] Bracketed figures are average persistences.

On average, DDT persists longest, about 80 percent remaining one year after it was applied compared with 75 percent of dieldrin, 60 percent of lindane, 55 percent of chlordane, 45 percent of heptachlor, and 26 percent of aldrin. After three years the residues are about 50 percent for DDT, 40 percent for dieldrin, 15 percent for chlordane, 10 percent for heptachlor, and 5 percent for aldrin. Aldrin and heptachlor may be insecticidal for longer than these figures indicate, because their breakdown products, dieldrin and heptachlor epoxide, respectively (not included in the above figures), are also insecticidal. By contrast, lindane may be less effective than the figures show, because it breaks down to non-insecticidal chemicals that still react in the nonspecific methods of analysis normally used. Insecticide persistence can differ widely in different soils. For instance, although the average proportion of DDT remaining after three years is about 50 percent, the range is from 26 to 78 percent. It is important to note that chemicals can still be detected in soil long after their biological effects have ended.

The mechanisms of disappearance of insecticides from soil are still poorly understood; volatilization, codistillation, leaching, oxidation, hydrolysis, and microbial activity probably all contribute, with the relative importance of each mechanism depending on the chemical structure of the insecticide. The chemical structure is the most important single factor determining stability, but climate and soil factors such as the combination referred to as soil type, pH, organic matter content, mineral content, drainage, and cropping may also affect the rate of breakdown. The ways in which these factors influence breakdown are complex and interlinked. Factors can be supplementary or can oppose one another; some have been studied in considerable detail but others have hardly yet been considered.

This review summarizes knowledge about how various soil factors affect the breakdown of insecticides, attempts to assess their relative importance, and indicates the possibilities of predicting the breakdown of specific insecticides in particular soil types.

II. Characteristics of the insecticide

The characteristics of the insecticide are most important in determining its persistence in soil. Only relatively stable and inert chemicals will remain in soil for appreciable periods. Two aspects of the structure of the insecticide affect its persistence: firstly its basic chemical stability and secondly its chemical and physical characteristics in relation to soil, and in particular to those soil properties associated with adsorbing and binding chemicals.

a) Type of insecticide

There has been a tendency to select as soil insecticides stable and unreactive chemicals. This is because many soil pests have life cycles of a year or longer and for an insecticide to be successful against them these pests must pick up the insecticide as they move through, or ingest soil. More volatile insecticides have improved vapour toxicity but do not usually persist long enough. Pest control is usually more successful with contact insecticides applied either as broadcast treatments or as seed dressings. Contact action relies on the chemical remaining long enough in the soil for the pest to pick up a lethal dose.

The most successful soil insecticides have been the chlorinated hydrocarbons or organochlorine compounds. These all have very stable structures and are lipophilic compounds of low polarity, but DDT, BHC, chlordane, and heptachlor are less stable in alkali than acid so pH may affect their persistence in soil.

The effective persistence of these materials as insecticides in soil is complicated by their different paths of breakdown. Some, such as BHC, break down to compounds that are not toxic to insects (EDWARDS *et al.* 1957, LICHTENSTEIN and SCHULTZ 1959 a). Others yield a mixture of compounds, some of which are insecticidal and others not; an example is DDT, which breaks down in soil to DDD, DDE, and other compounds. Still others break down to give compounds that are insecticidally active; these include aldrin, which breaks down to dieldrin, and heptachlor, which degrades to heptachlor epoxide (EDWARDS *et al.* 1957, BOLLEN *et al.* 1958, GANNON and BIGGER 1958, LICHTENSTEIN and SCHULTZ 1960, LICHTENSTEIN *et al.* 1964).

The variable persistence reported for chlorinated hydrocarbon insecticides in soil reflects not only the effect of soil characteristics and environmental, climatic, or geographical variations, but also different sampling methods and analytical techniques used by different workers. Some of the more important studies comparing the rates of breakdown of different insecticides in similar soils follow.

ALLEN *et al.* (1954) treated soil with various insecticides and after 4½ years, 38 percent of DDT and six percent of BHC remained in the soil. FOSTER *et al.* (1956) found that endrin was the most persistent chlorinated hydrocarbon, almost 100 percent remaining after two years, followed by dieldrin, dilan, and toxaphene, with about 50 percent recovery after three years, and isodrin and heptachlor with about 35 percent remaining. BHC was least with about five percent remaining and aldrin and chlordane intermediate with about 18 percent remaining after three years. KINCAID *et al.* (1960) stated that 18 percent of DDT, 20 percent of TDE, 24 percent of lindane and 11 percent of endrin disappeared in a year.

LICHTENSTEIN *et al.* (1960) treated several different field soils with chlorinated hydrocarbon insecticides and found that DDT was most persistent, aldrin least, and lindane intermediate. Of the materials studied by ROBERTS *et al.* (1962) DDT was most persistent, about 36 percent remaining at the end of the third year. BHC total isomers were the next most persistent with about 5 percent remaining after three years and only traces of guthion remained. All the malathion disappeared in one year.

LICHTENSTEIN *et al.* (1962) recovered 19.5 percent of the aldrin applied when this was one lb. actual/acre, 17 percent at two lb., and 37 percent at three lb. after one year. The corresponding figures for heptachlor residues were 26, 29, and 36 percent, respectively, of the applied dosages. The breakdown of aldrin to dieldrin and heptachlor to heptachlor epoxide has been more fully studied. BARTHEL *et al.* (1960) showed that when heptachlor was applied to soil there was a rapid initial loss followed by a gradual loss with conversion to heptachlor epoxide. GANNON and BIGGER (1958) concluded that aldrin was converted to dieldrin more rapidly in soil than was heptachlor to its epoxide. Supporting evidence for this came from LICHTENSTEIN and SCHULTZ (1965) who showed that heptachlor as such, was more persistent than aldrin because epoxidation of heptachlor proceeded more slowly than the conversion of aldrin into dieldrin. Environmental influences on the epoxidation of aldrin and heptachlor in soils were studied by LICHTENSTEIN and SCHULTZ (1960).

The organophosphorus insecticides are gradually replacing the chlorinated hydrocarbons as soil-applied insecticides. These are less persistent, probably because many of them are more water-soluble and have a higher vapour pressure and are more readily oxidized and hydrolyzed.

MULLA (1964) evaluated 39 insecticides, mostly organophosphorus compounds, in soil but only seven showed what he termed significant persistence. LICHTENSTEIN and SCHULZ (1964) applied parathion, methyl parathion, and malathion to Carrington silt loam plots at five lb. actual/acre. They found that residues were only 3.1 percent in field soils after eight days for malathion, 30 days for methyl parathion, and 90 days for parathion. In laboratory experiments at 30°C., 95 percent of applied methyl parathion and 30 percent of parathion were lost 12 days after application to a loam soil; 95 percent of malathion had disappeared after seven days. Parathion and methyl parathion seemed to disappear slower when assessed by a nonspecific chemical method than by bioassay, indicating some breakdown to non-insecticidal compounds. In their experiments, parathion disappeared faster from soil in covered than in uncovered vessels, but the reverse was true for aldrin, indicating that parathion was lost principally by hydrolysis whereas aldrin was lost more by volatilization.

PARKER and DEWEY (1965) using *Drosophila* bioassay found that phorate broke down completely in soil within one month, whereas 30 percent of dimethoate remained after one month. BOHN (1964) showed that dimethoate did not persist long having a biological half-life of only four days in soil.

HADAWAY and BARLOW (1964) gave the rates of decomposition of various organophosphorus compounds in soil. Dichlorvos and related vinyl phosphates, phosphoramidothioates, and phosphoramidates decomposed rapidly. Many substituted phenyl phosphates, phosphorothionates, and phosphonates also decomposed fairly rapidly. The most stable compounds they tested were fenthion (Baytex) and the related compound Bayer 37342.

Fenthion was lost from Uganda soil at the rate of about 24 percent in 56 days at 25°C. and 80 percent relative humidity, extrapolation giving a half-life of about three years, whereas the half-life of Bayer 37342 on Uganda soil was 72 days. Sumithion had a half-life at 25° C. and 80 percent relative humidity of ten to 42 days. The decomposition of malathion depended on soil type, over a wide range, but generally this insecticide gave poor results on dried soils.

Insecticides may disappear from soil at different rates either because of differences in their innate stability or because they bond differently when adsorbed on to the soil. Adsorption on to the soil helps the chemical to persist but may bind it so that it is no longer insecticidal. The most effective soil insecticides are probably adsorbed least. HARRIS (1964 a) found that diazinon was 135 times more toxic in wet soil than in dry soil, parathion 28 times, trichlorfon 20 times, dieldrin 16 times, zectran 16 times, DDT 16 times, heptachlor 13 times, and mevinphos 1½ times. The greater the difference between the insecticides' effectiveness in wet and dry soil, probably the more it is adsorbed, because in a wet soil the chemical is freed and can become effective.

HARRIS (1964 b) also found that diazinon, which was as toxic to insects as heptachlor and aldrin by contact action, had a vapour pressure similar to that of heptachlor and became only slightly more toxic in soil. Similarly, parathion which was nearly six times as toxic as aldrin by direct contact action and which also has a vapour pressure intermediate between aldrin and heptachlor was considerably less toxic in soil. Possibly these differences in toxicity result from stronger adsorption of diazinon and parathion in soil thus rendering them less effective both as contact and fumigant poisons.

Trichlorfon and mevinphos have considerable fumigant action but only poor insecticidal action in soil. Trichlorfon is soluble in water, however, and both these chemicals are probably poor insecticides because of adsorption at the water interface. To summarize, chlorinated hydrocarbons persist for years in soil, whereas most organophosphorus insecticides are much more transient.

b) Volatility of the insecticide

A soil insecticide is usually incorporated deep into the soil and unless it is somewhat volatile can disappear only by chemical breakdown or physical leaching. There is considerable evidence to indicate that even the relatively stable chlorinated hydrocarbon insecticides volatilize.

Table III shows that the insecticides with the greater vapour pressures usually disappear faster from soil. GALLAHER and EVANS (1961) found that even non-volatile DDT disappeared more rapidly in the first three months than in the subsequent nine months.

HARRIS (1961) used bioassay with *Drosophila melanogaster* to show that aldrin, heptachlor, heptachlor epoxide, dieldrin, lindane, and phorate were volatile. In terms of rate of volatilization aldrin $\cong$ heptachlor $>$ heptachlor epoxide $>$ dieldrin,

Table III. *Insecticide vapour pressures and disappearance from soils*

Insecticide	Average % insecticide in soil after one year[a]	Vapour pressure at 20°C. (mm. Hg)	
DDT	80	1.0×10^{-7}	Slightly volatile
Dieldrin	75	1.0×10^{-7}	
Lindane	60	9.4×10^{-6}	Moderately volatile
Aldrin	26	6.0×10^{-6}	
Chlordane	55	1.0×10^{-5}	Volatile
Heptachlor	45	3.0×10^{-4}	

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[a] Regression data from EDWARDS (1965 a).

Immediately after being added to the soil, the insecticide volatilized rapidly but later it seemed to become bound to the soil and volatilized more slowly at rates depending on soil types.

When insects were placed above soil treated at the LD_{50} and LD_{95} concentrations (HARRIS and LICHTENSTEIN 1961), mortality after twenty-four hours indicated that aldrin, heptachlor, chlordane, trichlorfon, and mevinphos were moderately volatile; diazinon, parathion, and dieldrin were slightly so; DDT and zectran were non-volatile. In later experiments they also found toxic vapours emanating from soils treated with heptachlor epoxide and phorate but not from soils to which sevin or malathion had been applied.

They investigated the volatilization of aldrin in more detail and found that volatilization of aldrin increased with increases in (a) insecticide concentration in soil, (b) relative humidity of air over soil, (c) temperature, and (d) movement of air over the soil. It decreased in dry soils containing much clay and organic matter. The density of soil in a given volume did not affect volatilization. Over a period of five to seven days 16 to 38 percent of aldrin applied volatilized and 55 to 80 percent of the amount of aldrin applied was still in the soil hence volatilization was important. Aldrin volatilized 37 times as rapidly from a wet Plainfield sand as from a dry sand. They concluded that toxic vapors were only close to the surface of soil treated with aldrin and lindane; farther from the surface they were removed by wind. When air was passed over dry quartz sand for six hours daily, 38 percent of the applied aldrin volatilized, but over field-moist Miami silt loam only 16 percent volatilized. These figures, together with the residual aldrin, accounted for more than 90 percent of the amount applied. Volatilization was greatest during the first day after application, thereafter becoming constant. BARTHEL *et al.* 1960) concluded that volatilization is the most important factor accounting for the loss of heptachlor from granules in the soil. LICHTENSTEIN and SCHULZ (1964) kept parathion-treated soil in dishes, some of which were covered with glass plates and others left open. Parathion disappeared faster from the covered (and wetter) soils than from the uncovered. This suggested to them that the major pathway of degradation of parathion in a loam soil was by hydrolysis and not by volatilization. This was contrary to the results of experiments with aldrin where residues were the most persistent when soils were kept in closed containers which lessened the volatilization of the aldrin. GETZIN (1958) found that within an hour of treatment sandy soil, silt loam, and muck had lost 25, 20 and 18 percent, respectively, of the radioactive thimet applied but after this initial loss very little or no volatilization occurred. BURT *et al.* (1965) concluded that phorate and disulfoton move through soil mainly in the gaseous phase but dimethoate and menazon move through soil dissolved in water. This difference in behaviour can be explained by the greater volatility of phorate and disulfoton.

CHISHOLM and KOBLITSKY (1959) suggested that a large proportion of chlorinated hydrocarbons may be lost by codistillation of insecticide into the atmosphere with the water vapour escaping from the soil.

c) Solubility of the insecticide

Although insecticides are usually formulated in the most water-soluble, miscible, or emulsifiable form possible, their water solubilities are usually small. It is to be expected that its water solubility would greatly influence the persistence of an insecticide in soil by resisting or facilitating leaching. Solubility also seems to be linked with the ease with which chemicals are adsorbed. For herbicides BAILEY and WHITE (1964) stated that there appears to be a relationship between solubility and adsorption and quoted the example of four substituted-ureas which had a degree of adsorption inversely related to the order of their solubilities. They also quote HARRIS and WARREN (1965) as showing that this relationship holds only within related groups of compounds and not for molecules of widely different chemical nature.

There is not much direct information about the effect of water solubility on the persistence of insecticides in soil. However, the most persistent insecticide in soil, DDT, has been quoted as the least water-soluble (0.0002 p.p.m.) organic substance known (FILMER and SMITH 1944). The solubilities of other commonly used chlorinated hydrocarbon insecticides are in the order dieldrin (<0.1 p.p.m.), aldrin (<0.05 p.p.m.), and lindane (10 p.p.m.), which corresponds roughly with the order of their persistence. LICHTENSTEIN (1958) found that parathion leached through soil more readily than lindane which is less water-soluble.

WEIDHAAS *et al.* (1961) found that the loss of parathion from aqueous solution to soil was not a function of the water solubility of this insecticide. HARRIS (1964 b) postulated that effectiveness as a soil insecticide was inversely related to water solubility. Water-insoluble materials such as aldrin and heptachlor (also dieldrin and DDT) were the most effective materials in soils; slightly soluble materials such as parathion and diazinon were intermediate in toxicity; while water-soluble materials such as mevinphos and trichlorfon were ineffective.

It is not clear whether the more water-soluble insecticides are rendered ineffective because they are more readily adsorbed on to soil or because they are more readily leached. This requires much more investigation.

d) Concentration of the insecticide

Doses of soil insecticides ranging from ½ lb. actual/acre to more than 100 lb. actual/acre have been applied to soil experimentally. Not all workers have agreed as to the influence of concentration on persistence. KINCAID *et al.* (1960) used different doses of DDT, TDE, lindane, and endrin in soil. The residues as percentage of amount applied were much less for larger doses than small ones, e.g., after 5½ years at 15 lbs. actual/acre 33 percent of DDT remained, but at 75 lbs. actual/acre only 19 percent remained. Corresponding figures for other insecticides at the same dosages were lindane, 27 and 7 percent and endrin 40 and 37 percent.

Two other groups of workers found no influence of concentration on persistence of insecticide. For example, FLEMING and MAINES (1954) found that the concentration of chlordane in soil had little effect on the percentage remaining even after several years. GALLAHER and EVANS (1961) found that the proportion of DDT from treated pasture soil appeared to be independent of concentration in the range one to eight lbs. actual/acre.

LICHTENSTEIN and coworkers thought that insecticides persisted proportionately longer in large concentrations. For instance, LICHTENSTEIN and SCHULTZ (1959 a) found that in the field, twice as much DDT as a percentage of the original application was recovered after 3½ years from a plot treated with 100 lbs. actual/acre than from one treated with 10 lbs. actual/acre. Corresponding figures were 3.5 for lindane and 2.3 for aldrin. These results were confirmed in laboratory experiments with incubated soil and they concluded that an insecticide disappeared faster when the concentration was smaller. They also showed (1959 a, 1960 c, and 1962) that relatively more aldrin was converted into dieldrin in plots where the insecticide was less concentrated. They also found that aldrin, lindane, and DDT persisted longer in terms of percent of original application in those soils where they were most concentrated (1959 a). They concluded that the reactions going on within the soil and responsible for the disappearance and breakdown of those insecticides investigated, depended to some extent on the concentration of the insecticides in the soil. HARRIS and LICHTENSTEIN (1961) also found that aldrin volatilized from soil at a rate which was proportional to its concentration, between 0.5 and 16.0 p.p.m.

Soil insecticides do not disappear at the same rate when applied as a single large dose as when applied as several small doses adding up to the same total amounts of chemical. For instance, LICHTENSTEIN and SCHULTZ (1965) found that aldrin applied in one massive dosage of 25 lbs. actual/

five-inch acre left much smaller residue levels in soils after five years than when the same amount of aldrin was applied in five five-lb. increments.

Because the conclusions differed so much, EDWARDS (1964) plotted all the published results together with some unpublished data as regressions of percentage of original dose against time. The regressions showed clearly that proportionately more disappeared from a small dose than from a large one. But larger gross quantities of insecticide disappeared from soil with the larger dose, i.e., the rate of breakdown was not constant irrespective of dose but diminished logarithmically with it.

The rate at which a particular dosage disappears seems to be reasonably constant whether the insecticide is a residue of a much larger dose or a new application, except for the rapid losses that often occur immediately after the insecticide is applied.

e) Formulation of the insecticide

Insecticides are formulated in many ways to simplify problems of application. They may be dusts, wettable powders, granules, seed dressings, emulsions, miscible liquids, or solutions. The formulation may considerably affect the way the insecticide interacts with soil and becomes held by it, but opinions differ about this. There is little doubt that the water-soluble forms leach more than the oil-soluble, water-miscible forms (BAILEY and WHITE 1964).

The particle size of insecticide in the formulation is also important; for instance, BARLOW and HADAWAY (1955) stated that the rate particles of lindane were adsorbed was inversely proportionally to their size immediately after application. Hence at first, the effectiveness of the insecticide was directly proportional to particle size because the larger particles are adsorbed slower than smaller ones. However, when all particles had disappeared from the surface and insecticidal effectiveness depended only on fumigant action, the smaller particle treatments were more effective, probably because the insecticide was more thoroughly distributed throughout the soil.

HADAWAY and BARLOW (1963) thought that the initial insect toxicities of deposits of DDT and dieldrin applied to the surface were greater for wettable powders than for emulsions, probably because of the greater ease with which an emulsion can penetrate any surface that is only slightly porous.

BERTAGNA (1959) demonstrated that, even though wettable powders can be adsorbed, they may persist longer on sorptive surfaces than either solutions or emulsions that penetrate beneath the surface rapidly. But these findings refer to persistence at the soil surface; there is evidence that emulsions persist longer in the deeper soil, probably because they are more readily adsorbed. YOUNG and RAWLINS (1958) found that heptachlor as an emulsion persisted longer than when applied as a wettable powder. This difference was greater in muck soil than in silty clay loam. JOHANSON (1950), who placed DDT on the surface of soil columns and then ran water through these, found that although there was some leaching of wettable powder there was more with emulsion.

There is considerable evidence that insecticides applied as granules persist much longer than dusts, wettable powders, miscible liquids, or emulsions, although MULLA (1960) found granules containing 20 lbs. of active DDT /acre were less effective than spray treatments against eye gnats (*Hippelates collusor*). After three months, however, the granules were the more effective. Granular formulations of trithion persisted longer than an emulsion when applied to soil (MENN *et al.* 1960). LICHTENSTEIN *et al.* (1964) applied aldrin to soil (a) as an emulsion left on the soil surface, (b) as an emulsion incorporated into the soil, (c) as granules left on the soil surface, and (d) as granules incorporated into the soil. After one year, 22 percent of the granules applied to the surface were recovered but only 8 percent of the emulsion remained, compared to 51 percent of the aldrin or dieldrin on granules rototilled into the soil and only 36 percent of the aldrin (or dieldrin) applied as an emulsion and cultivated in. Several authors have pointed out that the solvent used in impregnating the granules is important. For instance BARTHEL *et al.* (1960) found that the solvent in which heptachlor was applied to granules affected the persistence of the toxicant in soil. Non-volatiles solids such as Avoclor and rosin made heptachlor in soil last much longer.

MULLA and AXELROD (1960) also studied the effect of solvents used in impregnating insecticide-treated granules on release of the insecticide in soil. By selecting suitable solvents to impregnate granules with the toxicant it is possible to prepare a range of granular formulations which give slow, intermediate, or rapid release of the toxicant. TURNIPSEED *et al.* (1963) used different solvents to apply dieldrin to attapulgite granules and these affected the rate dieldrin was released, which was progressively slower, in the order xylene, heavy hydrocarbon, and non-hydrocarbon (aqueous diethylene glycol).

To summarize, insecticides usually persist longer in soils as granules than as emulsions which in turn persist longer than miscible liquids; they disappear fastest from wettable powders and dusts.

III. Characteristics of the soil

After the characteristics of the insecticide, the type of soil seems to be the most important single factor in determining the persistence and availability of the chemical. The soil type is determined by features such as the proportion of stones, sand, silt, and clay, the mineral content and structure which influences the pH, and the organic matter content. All of these may be important in retaining a chemical such as an insecticide in soil.

a) Soil type

Most of the evidence is that the heavier soils with much clay and organic matter retain insecticides most strongly, but FLEMING and MAINES (1953) studied the persistence of DDT in 84 mineral soils by bioassay with Japanese beetle larvae (*Popillia japanica* Newm.) and found that DDT persisted longest in sand and least so in muck. In the different loams persistence was much the same. This contradicts most of the evidence which shows that heavy soils (with much clay) and soils with considerable organic matter retain insecticides longest. But data based on bioassay estimates only that part of the insecticide which is available to kill the test insect.

The influence of soil type on insecticides first became obvious when it was observed that crops grown in soils treated with very large dosages of chlorinated hydrocarbon insecticides were damaged by the insecticides more in light sandy soils than in heavy clays, mucks, and peats. For instance, APPLEMAN and SEARS (1946) found that plants grown in soil treated with DDT were not so stunted as those grown in sand treated with DDT at the same rates. CHULSKI (1948) found that insecticides were less phytotoxic in Miami clay loam than in sandy soils, and FOSTER (1951) concluded that much more DDT (even up to 1000 lbs. actual/acre) can be safely mixed with muck soil than with mineral soils even when sensitive crops are grown.

LICHTENSTEIN (1959) found that soil type had a remarkable influence on the absorption of lindane into crops. During the first year of an experiment, lindane applied at 100 lbs. actual/acre was very phytotoxic in a sandy loam, less on a Miami silt loam, and almost non-toxic in a muck soil; also carrots grown on soil treated with ten lbs. actual/acre two years before sampling contained six p.p.m. of lindane when grown in a sandy loam, two to four p.p.m. in a Miami silt loam, and 0.4 p.p.m. in a muck soil, although the muck soil contained approximately six times more lindane than the other two soil types.

Large doses of chlorinated hydrocarbon insecticides have sometimes stimulated the growth of some crops, again more in lighter than on heavier soils. CHULSKI (1948) found that BHC stimulated the growth of plants in some soils, which he suggested was probably because of the chemical breakdown of BHC. In the presence of a weak alkali, BHC forms isomers of trichlorobenzene that may stimulate growth. Increases in growth which resulted were less on heavy textured clays than on lighter textured sands and it seemed that the stimulation was inversely proportional to the amount of colloidal material in the soil. This colloidal material could be either the organic or the inorganic colloidal fraction of the soil and he concluded that the organic or inorganic colloids act as a buffering agent and the more colloidal material in the soil the less was the stimulation.

Another indication of the interaction between soils and insecticides is that insecticides are much less toxic in some soils than others. Chlorinated hydrocarbon insecticides are widely used in control of mosquitoes, mainly as sprays applied to the surface of the walls of native mud huts.

HADAWAY and BARLOW (1949, 1951, 1952, 1955, 1956, 1957, 1958, and 1963) studied the effect of the type of soil used in the construction of the huts on the persistence, adsorption, and inactivation of the insecticides. They showed (1955) that solid particles of insecticides deposited from aqueous suspensions could disappear very rapidly from the external surfaces of blocks made from tropical soils. They thought this was a physical process of adsorption and involved complete loss of biological activity of those compounds with no fumigant action, such as DDT and dieldrin. Nevertheless it extended the duration of fumigant action by insecticides such as lindane and aldrin. They considered it to be a physical rather than chemical effect mainly because it was general for all the insecticides examined, and not restricted to one particular group with similar chemical properties. The rates of loss of different insecticides from the surface of an active mud are proportional to their vapour pressures and therefore approximately to their evaporation rates. The loss of an insecticide is not restricted to one adsorbent; it occurs on charcoal and other materials, is reversible, and the insecticide can be recovered from a soil block after adsorption is complete. They found correlation between the capacity of soils to adsorb carbon tetrachloride and DDT. If a soil can adsorb 20 to 30 percent of its weight of carbon tetrachloride it is probably an unsuitable substrate for residual insecticides.

They found (1955) that red soils with much iron oxide most actively sorbed insecticides. British soils sorbed poorly, even peat sorbing much less than most of the African soils. Taveta red soil, which absorbed more slowly than the other red soils, contained a greater proportion of larger hard quartz-like fragments. The insecticides were gradually absorbed from the surface of mud huts into the mud and BARLOW and HADAWAY (1955) found that particles of DDT could be seen to disappear from the surface under the microscope; they disappeared from the surface of Uganda mud at about the same rate as from the surface of charcoal, clearly showing the considerable sorptive power of the lateritic soils (see Table IV).

Table IV. *The residual life of 0 to 10 micron DDT particles on different soils* (from BARLOW and HADAWAY 1955)

Origin of soil	Approx. time to disappearance, days	% carbon tetrachloride sorbed in 24 hours
Uganda, red	3	25
Lagos, red	3	19
Jamaica, red	3	29
Taveta, Kenya, grey	4	22
Mexico, red	10	18
Taveta, Kenya, red	10-14	5
Jamaica, grey	8	13
India, grey	20	3
Weald clay (U.K.)	10-11	9
Liberia, grey	20	11
Upton (U.K.)	No change in 3 months	2
Evesham (U.K.)	No change in 3 months	5
Wantage (U.K.)	No change in 3 months	3
Martock (U.K.)	No change in 3 months	6
Bridgnorth (U.K.)	No change in 3 months	2
Worcester (U.K.)	No change in 3 months	7
Acid peat (U.K.)	No change in 3 months	9
Mexico, grey	No change in 3 months	13
Mexico, brown	No change in 3 months	5

DOWNS *et al.* (1951) noted that DDT lost its activity in weeks or months on various adobe surfaces. Lake-bottom, loamy soil with much organic matter retained insecticidal activity for three years, sandy clay for more than a year, a deltaic deposit and a red clay for only three to six months. The first soil initiated no breakdown, the sandy clay a reaction which decomposed 10 percent of DDT, the others between 20 and 98 percent.

For agricultural soils, EDWARDS *et al.* (1957) showed that aldrin and lindane were adsorbed least in Springfield sand, and by increasing amounts in silty clay loam, light sandy clay loam, coarse silt, silty clay, sandy loam, clay loam, and muck. As much as thirty times the amount of aldrin was needed in sand as in a muck soil to obtain the same kill of fruit flies. In 1957, LICHTENSTEIN published a survey of 24 orchard and crop soils that had been treated with DDT between 1945 and 1955 and found that the sandier soils contained the smallest percentage residues.

Similarly YOUNG and RAWLINS (1958), who analyzed soil for residues one week after the last of six spray applications each of 0.7 lb. actual of heptachlor, found that 31 percent of the total amount of heptachlor applied in muck soil, 11 percent in silty clay loam, and 8 percent in silt loam remained, and these differences were highly significant. ROBERTS (1963) found that to kill test insects required much greater concentrations of dieldrin on muck or loam soils than in sand. One species of test insect, *Cyclocephela immaculata,* ingests soil, but ingestion of treated muck soil did not greatly increase the insecticidal activity of the large amounts of sorbed dieldrin. He suggested that dieldrin may be more tightly bound to soil than either aldrin or lindane. He also showed that the bonding of dieldrin to a particular soil occurred rapidly. LICHTENSTEIN and SCHULTZ (1959 b) found that most aldrin was converted into dieldrin in a sandy soil, less in a Miami silt loam and least in a muck soil. The same workers (1959 a) showed that DDT, lindane, and aldrin all persisted longer in a muck soil (organic matter content 40 percent) than in a silt loam (organic matter content 3.8 percent). Thirty-three percent of the originally applied DDT and lindane were recovered after 3½ years from a muck soil and 4 to 7 percent of the originally applied aldrin; a Miami silt loam still contained 22.7 percent of the DDT, 3.9 percent of the lindane, and 1.1 percent of the aldrin after the same time. Soil type affected the persistence of DDT less than that of the other two chemicals. Persistence of lindane was influenced most by soil type and the difference in different soils increased as time progressed. Six months after application, 1.84 times more lindane was recovered from a muck than from a loam soil, but three years later the recovery ratio was found to be 8.45. In laboratory experiments, 87.5 percent of the applied aldrin was recovered after 56 days in the muck soil, 68.9 percent in a Miami silt loam, and 54.5 percent in the Barnfield sand. The amount of aldrin recovered two months after treatment, from a muck soil was 1.27 times larger than from a loam soil. In the field, 1.94 times more aldrin was recovered from a muck soil than from a Miami silt loam six months after insecticide application.

WIESE (1964) found that after incorporation of aldrin, heptachlor, dieldrin, chlordane, and DDT (with this decreasing order of toxicity to *Trinervitermes trinervoides)* into soil of low organic matter content, the insecticides were only half as active in loam soil, and 1/6 to 1/7 as active in clay soil, as in sandy soil. While there was a considerable parallelism between the regression lines for soil types the slopes for sand tended to be steeper than for loam, which in turn were steeper than for clay; this suggests that the progressive inactivation through adsorption on soil colloids is not indentical for the three soil types. Considerable biological inactivation of all insecticides occurred in soil with much clay.

HARRIS and LICHTENSTEIN (1961) showed that in both wet and dry soils aldrin vapours were released fastest from a quartz sand and slowest from a muck soil (Table V).

Table V. *Release of aldrin vapours from various dry and wet soils* (from HARRIS and LICHTENSTEIN 1961)

Soil type	L.T.$_{50}$ in hours *(Drosophila)*	
	Dry soil	Wet soil
Quartz sand	0.6 ±0.06	0.25
Plainfield sand	22.25±0.25	0.63±0.01
Miami silt loam	51.25±0.25	1.68±0.08
Carrington silt loam	53.00±1.00	1.78±0.08
Muck I	83.50±1.00	6.00±0.15
Muck II	63.00±3.00	9.75±0.25

HARRIS (1964 b) showed that, at field moisture capacity, DDT was 44.1, heptachlor was 61.6, diazinon was 65.7, V-C 13 was 73.3, and parathion was 92.0 times less toxic in muck than in sand, but in dry soil there was no obvious correlation between organic matter content and toxicity. V-C 13 and diazinon were less toxic in dry sand than dry muck, whereas parathion, DDT, and heptachlor were 1.5, 5.8, and 10.2 times more toxic. This indicates that in moist soils, inactivation of the insecticide is proportional to the organic matter content of the soil but in dry soils inactivation is related to the adsorptive capacity of the mineral fraction.

Most work has been on the chlorinated hydrocarbon insecticides because these are stable. Nevertheless, although organophosphorous and other insecticides disappear faster, their persistence also appears to be influenced by the type of soil. GETZIN (1958) found that drenches of phosdrin, thimet, schradan, and demeton killed more aphids and remained active longer in sands and sandy soils than in other soil types such as silt loam, clay loam, and muck. Leaching of radioactive phosdrin through columns of 12 soils showed that most insecticide was bound by peat (127µg./10g. of soil), less by heavy loam soils (10 to 27 μg./100g. of soil), and least by sandy soils (3 to 8 μg./100g. of soil). Malathion, fenthion, and an unnamed insecticide persisted longer in Magugu soil than on highly sorptive red Babati soil (HADAWAY and BARLOW 1963).

The soil type not only influences the rate at which insecticides disappear but also their conversion into other insecticidally active chemicals. For instance, LICHTENSTEIN and SCHULZ (1960) found that aldrin was transformed into dieldrin sooner in a Carrington loam than in a muck soil, and HARRIS and LICHTENSTEIN (1961) showed that more dieldrin was formed in a given time in silt loam soils than in sands or sandy loams; they thought this was because aldrin vaporized less from soils as the clay and organic matter increased.

b) Soil structure

The structure of the soil is one of the main features influencing soil type. It depends partly upon the parent rock from which the soil is formed, on the percentages of the three main soil fractions (sand, silt, and clay), on its mineral content, and on the recent history of the soil. The structure and texture markedly affect aeration and drainage. The rate of movement of water in the soil probably affects the equilibrium between insecticide carried in the soil solution and that adsorbed on to the soil colloids and organic matter.

There has been little work on the direct influence of soil structure on the persistence of insecticides, but as the structure is intimately linked with other features of soil such as pH, organic matter content, and mineral content it must influence the retention of chemicals incorporated into soil.

THURSTON (1953) could find no relationship between the texture of mineral soils and the amount of phytotoxicity from DDT but most workers who considered this found soil structure influenced persistence.

Structure greatly influences porosity of soil and SWANSON *et al.* (1954) found that the retention of lindane in soil was related to porosity. They considered that the movement of moisture is retarded in soils with high capillary porosity and it could be expected that such soils would also retard volatilization and movement of lindane. This may explain why fine-textured soils required larger applications of BHC than did coarser textured ones. By contrast HARRIS (1961) found that air-space porosity of the soil had no effect on the rate at which aldrin volatilized, although the rate of volatilization did decrease with increasing surface area of the soil particles. ROBERTS (1963) considered that the increasing retention of dieldrin by sand, loam, and muck, respectively, may be correlated with the increase in adsorptive surface area corresponding with decrease in particle size.

CHULSKI (1948) found that more BHC can be applied to fine-textured soils than to coarse-textured ones without deleteriously affecting a crop, indicating that ion exchange may play some role in adsorption.

Other ways that soil structure may affect insecticides in soil are considered in the following sections which review factors such as pH, organic matter, clay content, and mineral content; all are intimately related to structure.

c) *Organic matter content*

The amount of organic matter contained in soils ranges from about 40 to 50 percent in muck or peat soils to less than 1 percent in some sands. The surface of organic matter provides a ready substrate on which a wide range of chemicals that reach soils may be adsorbed.

SWANSON *et al.* (1954) did not find much effect of organic matter in soil on the retention of lindane, but this conclusion is not widely supported. Two of the earlier workers, FLEMING and MAINES (1953 and 1954) concluded that the persistence of DDT and chlordane was inversely proportional to the amount of organic matter in soil but they used bioassay for analysis and their results probably reflect the sorption of the insecticides on to the soil and hence their availability to test insects rather than actual persistence. This possibility is supported by the greater amounts retained in the high organic matter soils and compared to those with only little organic matter. After four years, similar amounts of chlordane occurred in all the soils irrespective of the proportion of organic matter. EDWARDS *et al.* (1957) obtained a highly significant correlation between the persistence of lindane and aldrin in soil and organic matter content of the soil (Table VI).

Table VI. *Relation between lindane and aldrin adsorption and soil factors* (from EDWARDS *et al.* 1957)

Soil type	Organic matter, %	Moisture-holding capacity, %	L.D.$_{50}$ after 24 hours (*Drosophila*)	
			Lindane (p.p.m.)	Aldrin (p.p.m.)
Springfield sand	0.5	51	0.25	0.055
Silty clay loam	1.0	71	0.38	0.175
Light sandy clay loam	1.2	52	0.51	0.065
Coarse silt	1.4	58	1.07	0.205
Silty clay	1.8	74	0.67	0.22
Sandy loam	2.6	49	1.25	0.22
Loam	3.8	83	2.65	0.34
Clay loam (lower level)	6.4	92	4.10	0.40
Clay loam (upper level)	10.0	—	5.9	0.54
Muck	40.0	127	8.6	0.85

Correlation coefficients:

L.D.$_{50}$ (aldrin) with organic matter content $r = 0.8935^{**}$
L.D.$_{50}$ (lindane) with organic matter content $r = 0.9141^{**}$
L.D.$_{50}$ (aldrin) with moisture holding capacity $r = 0.8468^{**}$
L.D.$_{50}$ (lindane) with moisture holding capacity $r = 0.8204^{**}$

LICHTENSTEIN *et al.* (1960) also found positive correlations between persistence of aldrin in seven different soils and the amount of organic matter they contain as shown in Figure 2. ROBERTS (1963) assumed that the adsorption of dieldrin by soil organic matter was due to a strong physical

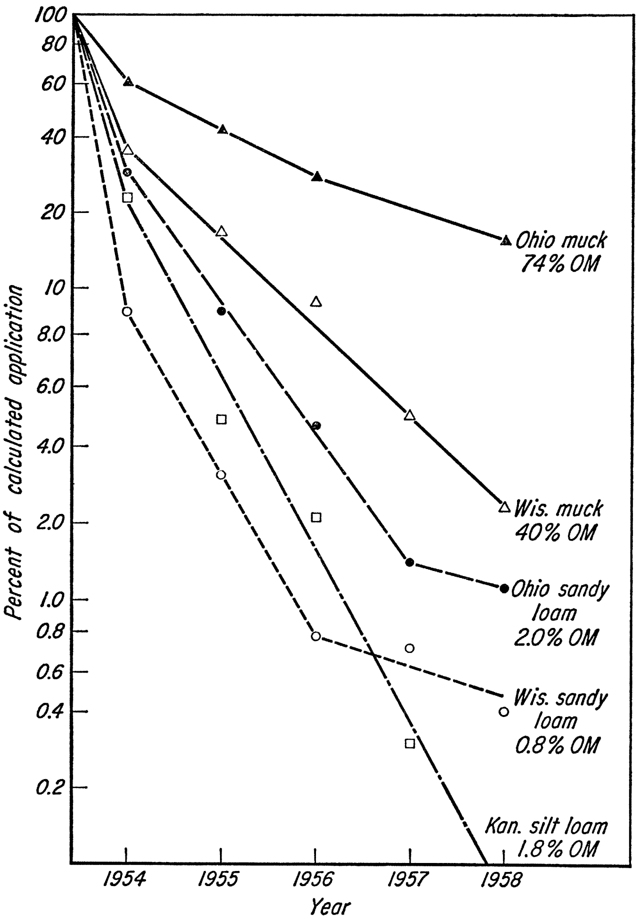

Fig. 2. Rates of loss of aldrin residues from various soils treated at 20 lbs. actual/six-inch acre (after LICHTENSTEIN *et al.* 1960). *OM* = organic matter

bonding because it was not toxic to soil-ingesting insects in a soil with considerable organic matter. By contrast, in Great Britain, WHEATLEY *et al.* (1960) found that dieldrin controlled carrot fly longer in fen soils with much organic matter than in mineral soils, and that it had what they termed a half-life[2] of four years in a mineral soil and one of five to seven years in a fen soil.

HARRIS (1964) showed that the influence of organic matter in the soil can be modified by soil moisture. The organic matter content strongly affects the amount of insecticide adsorbed in moist soil but in dry soil it has little effect.

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[2] See discussion for details of "half-life".

Aldrin can be lost from soil by volatilization, and HARRIS and LICHTENSTEIN (1961) found less was lost in this way from wet soils rich in organic matter than from mineral soils. They thought this was because the surface area for adsorption in a soil increases with organic matter.

Organophosphorus insecticide persistence is also affected by soil organic matter content. Thus GETZIN (1958) found that the amount of phosdrin bound by 12 different soils correlated positively with the organic matter content. MENN *et al.* (1960) studied the persistence of trithion in three different soils and found that it also correlated positively with the amount of organic matter in the soil. PATTERSON (1962) concluded that organic matter, was the major factor of soil structure restricting absorption of phorate by plants from the soil.

Thus there seems little doubt that soils containing much organic matter, although needing more insecticide to kill pests, retain the chemical much longer than soils poorer in organic matter.

d) Clay content

Soils containing much clay might be expected to retain added chemicals longer. They have a very much larger internal surface area than sandy soils and the clay content greatly influences soil acidity. There is not much evidence that clay content influences insecticide persistence, but the few investigations reported indicate that large clay contents increase the persistence.

THURSTON (1953) expected clay content to influence the phytotoxicity of DDT but his experiments using kaolinitic and montmorillonitic clays to adsorb DDT were inconclusive and effects were less than found with ion-exchange resins. SWANSON *et al.* (1954), however, showed that clay content considerably affected lindane adsorption—the more clay the greater the adsorption. Clay/sand mixtures retained lindane more than mixtures of silt/sand and both much more than sand alone. All differences were significant at the 0.01 level.

A considerable influence of clay content on the persistence of lindane and aldrin in soil was suggested by EDWARDS *et al.* (1957) but the correlation was less than for soil organic matter. The issue was a little confused by association of high organic matter with greater clay content in the ten soils tested. Such intercorrelations are common between factors such as organic matter, clay content, moisture capacity, and cation-exchange capacity.

HARRIS and LICHTENSTEIN (1961) found that less aldrin volatilized from soils with a lot of clay and assumed aldrin was adsorbed on clay particles.

e) Soil acidity

Acidity affects the stability of clay minerals and the ion-exchange capacity and may influence the stability and persistence of chemicals added to soil. There is evidence that the adsorption of herbicides of different molecular structure increases as the pH decreased. Chlorinated hydrocarbon insecticides are very inert and unreactive, and although DDT, BHC, heptachlor, and chlordane tend to be unstable in the presence of alkali, most of the evidence suggests that pH does not directly influence their persistence in soil. For instance SWANSON *et al.* (1954) found no correlation between pH and retention of lindane, and DOWNS *et al.* (1951) gave data which indicated that alkalinity was not a significant factor in catalyzing DDT breakdown.

Dieldrin and related compounds are more likely to decompose when sorbed on soil of high "surface acidity" (BARLOW and HADAWAY 1958 b) but such soils are rare in Africa. FLEMING and MAINES (1953) concluded that pH had no influence on the persistence of DDT in soil within the range of 4.0 to 7.5, and SMITH (1948) demonstrated that DDT was stable in both acid and alkaline soils. BOLLEN *et al.* (1958) could not correlate pH with persistence of aldrin and dieldrin in soil. FOSTER (1951) found no damage to sensitive plants in muck soil with amounts of DDT up to 1000 lbs. actual/acre, but when the pH was changed by adding dolomite limestone there was some damage but the amount was inconclusive.

The effect of soil pH on the more reactive organophosphorus insecticides may be more marked. GRIFFITHS (1965) found some indications that organophosphorus insecticides persisted longer in acid soils. Furthermore, GETZIN (1958) found that the amounts of phosdrin bound by 12 different soils were positively correlated with the base-exchange capacity. It seems that further investigations are required on the influence of pH on soil insecticides.

f) Mineral ion content

This is bound up very much with structure and pH. It seems very possible that the types of mineral which predominate in different soils may well influence the retention of insecticidal chemicals there.

BARLOW and HADAWAY (1955) stated that activated iron oxide gels sorbed DDT even faster than charcoal or Uganda mud. This is interesting because the lateritic soils contain large proportions of iron oxide. They later (1958) showed that removing all the materials soluble in organic solvents and the iron compounds had no influence on the rate of sorption of dieldrin and only a slight limiting effect on the sorption of DDT.

PRESS (1959) found that large aluminum and iron contents together with small silica content coincided with an increase in the ability of soils to absorb insecticides. DOWNS *et al.* (1951) showed that those soils which catalyze the decomposition of DDT most effectively have most iron and aluminum. Sorption is probably the first step in the process of catalytic decomposition and sorption takes place very rapidly in soils with much iron. SWANSON *et al.* (1954) found a good correlation between magnesium content of soil and its retention of lindane. GALLAHER and EVANS (1961) examined a range of New Zealand soil types for catalytic effects of clay minerals and inorganic salts and found that some soils derived from volcanic ash promoted decomposition of DDT at high temperatures.

BOLLEN *et al.* (1958) found no correlation between the amount of aldrin and dieldrin recovered from soil and buffer capacity, ammonium binding power, cation-exchange capacity, mechanical analysis, moisture tension, pH, or any other chemical feature. THURSTON (1953), however, found that two strong-base anion-exchange resins IRA 400 and XE 98 when added to nutrient solutions containing DDT lessened its phytotoxicity, and a weak-base anion-exchange resin IR4B had much less effect on phytotoxicity; a cation-exchange resin, IR 120, had no effect on the phytotoxicity of DDT. Wood and bone-activated charcoal effectively adsorbed DDT and prevented phytotoxicity.

Although fuller investigations are required it appeared that minerals do affect the absorption process quite markedly.

IV. Environmental features

The factors that have been considered as influencing the persistence of soil insecticides in the preceding two sections have all been relatively static. A particular insecticide is applied at a known concentration and formulation to a soil. We must now consider the effects of the variable climatic and cultural factors which can also greatly influence the persistence of insecticides in soil.

a) Temperature

Insecticides are lost from soil probably mainly by volatilization, chemical degradation, or bacterial decomposition; all these processes are affected by temperature. GALLAHER and EVANS (1961), however, did not consider that breakdown of DDT was affected by temperature because decomposition rate did not increase over the summer period (25 to 35 weeks) with its high temperatures. Most evidence, however, indicates that increased temperature accelerates the disappearance of insecticides from soil. EDWARDS (1964) kept samples of soil treated with insecticides for 2½ years at −10°C. and for shorter times at 0°C with no significant losses. LICHTENSTEIN and SCHULZ (1959 a) found that insecticides were not lost from frozen soils, but at 6°C. 16 to 27 percent of aldrin or heptachlor was lost in 56 days, at 26°C. 51 to 55 percent, and at 46°C. between 86 and 98 percent (Fig. 3). KIIGEMAGI *et al.* 1958) found a more rapid disappearance of aldrin, dieldrin, and heptachlor residues in summer than in winter. PATTERSON (1962) showed that when the soil temperature was increased from 40° to 100°F. phorate disappeared faster. In the field most of the phorate applied disappeared in two months in summer but persisted longer during the fall and winter. LD_{50}'s of dieldrin, carbaryl, and phosphoramidothioate decreased and that of DDT increased between 20° and 30°C. (HADAWAY and BARLOW 1963).

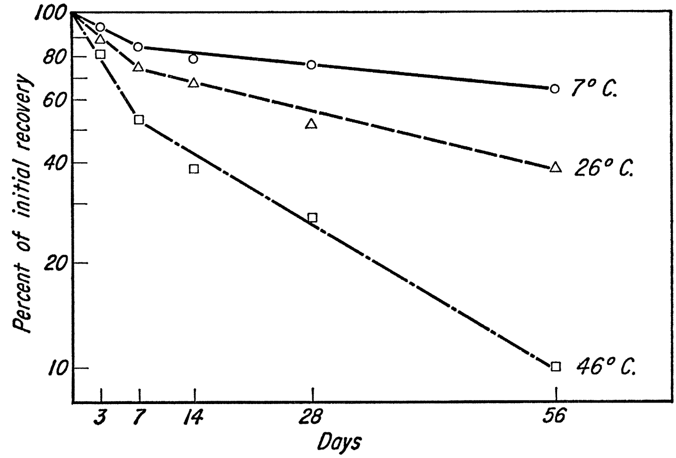

Fig. 3. Loss of aldrin residues from a Plainfield sand as affected by temperature, with applications at 100 lbs. actual/six-inch acre (after LICHTENSTEIN and SCHULZ 1959)

Conversion to other compounds is accelerated by higher temperatures as shown by LICHTENSTEIN and SCHULZ (1959 b) who found that much more of the aldrin applied to soil was converted to dieldrin at temperatures of 26° and 46°C. than at 7°C. In soil at 46°C. there were similar amounts of dieldrin to those in soil at 26°C. after 28 days but after 56 and 84 days there was much less dieldrin in the soil. Probably one of the most important effects of temperature is to increase evaporation of volatile compounds: thus HARRIS and LICHTENSTEIN (1961) found that increased temperature increased the volatilization of aldrin from soil (Table VII).

Table VII. *The volatilization of aldrin from Plainfield sand* (from HARRIS and LICHTENSTEIN 1961)

Temp. (C.)	Percent of aldrin volatilizing in 6 hours
10	2.04±0.26
22	4.88±0.13
30	7.80±0.19
36	10.00±0.59

Temperature may also influence the adsorption, which is usually exothermic, and probably decreases absorption and releases insecticide. With herbicides (HARRIS and WARREN 1965) this rule is not invariable and BARLOW and HADAWAY (1958) showed that increased temperatures increased the rate of sorption of DDT into mud blocks. Solubility of insecticide is also temperature-dependent and so this would also tend to decrease adsorption as the temperature rises and more of the adsorbed insecticide becomes dissolved in soil moisture.

It thus seems that increased temperatures increase the loss of insecticides by increasing conversion to other compounds, volatilization, and desorption, but this is modified by the coincident drying of soil: dry soils hold insecticides much more firmly than wet ones.

Higher temperatures can cause insecticides to break down more rapidly in tropical than in temperate soils, especially during the wet season, but when the soil becomes very dry the rate of breakdown could be considerably slowed. Many parts of the United States have hot summers and cold winters and insecticides could persist in soil there for times comparable to more temperate countries such as Great Britain.

b) Rainfall, leaching, and soil moisture

Soil moisture content is usually closely related to rainfall. Most of the chlorinated hydrocarbon insecticides are sparingly water-soluble with lindane, which has a solubility of 10 p.p.m. in water, the most water soluble. Thus there is unlikely to be much leaching as a direct result of solubility. It is possible, however, that particles of insecticide may be physically washed down through crevices into deeper soil. Leaching may be much more important in the disappearance of organophosphorus insecticides which have relatively greater water-solubilities. There is also considerable evidence that soil moisture exerts a great indirect influence on the adsorption of insecticides by soil.

It is seldom possible to cultivate insecticides into soil to a depth of more than six inches. Several workers have studied the vertical distribution of insecticides at intervals after application. ALLEN *et al.* (1954) applied DDT to soil, worked it into the soil to a depth of six inches, and four years later took cores of soil which they separated into two-inch deep layers and analysed them separately for residues; the top six inches of soil still contained 87 percent of the residues, the next two inches a further 9.5 percent, and the remaining 3.5 percent was in the two inches below this. CHISHOLM and KOBLITSKY (1952) treated turf plots with 25 lbs. of DDT/acre and 92 percent remained in the top inch of soil after one year and the other 8 percent in the next two inches. GINSBURG and REED (1954) in a survey of orchard, potato, corn, and cranberry bog soils found most DDT within the plough and cultivation layer and none below nine inches. GINSBERG (1955) found most of the DDT recovered from soil to be close to the surface with little evidence of downward transport except as a result of cultivation. LICHTENSTEIN (1958) treated a Miami silt loam and a muck soil with aldrin, lindane, or DDT and rototilled them into a depth of four to five inches. After seventeen months, 84 to 96 percent of the insecticides were still in the top three inches of the loam silt; 4 to 12 percent between three and six inches deep, and 0.5 percent below this, whereas in the muck soil 62 to 74 percent was in the top layer, 19 to 29 percent between three and six inches, and 7 to 8 percent in the six-to-nine inch layer. KINCAID *et al.*

(1960) found that average residues in soil of insecticides applied to control tobacco pests were 16.4 lbs. actual/acre in the zero-to-six inch level, 4.1 lbs. actual in the six-to-nine inch level, and 1.0 lb. actual in the nine-to-12 inch level.

LICHTENSTEIN *et al.* (1962) applied aldrin and heptachlor to the soil surface and after four months found nearly all the residues in the top two inches of soil and 75 percent in the top one inch. In six differently treated experimental plots which were rototilled to a depth of five inches, 90 percent of the insecticide residues were located within the upper three-inch soil layer after 15 months, and nearly half of the 90 percent was within the second one-inch layer, whereas the upper (zero-to-one inch) and the lower (two-to-three inch) layers each contained 20 to 30 percent of the totally recovered residues. The zero-to-six inch layers contained 6 to 18 percent of the totally recovered residues but none was found in the six-to-nine inch layer. After three years, 72 to 82 percent of the residues were in the upper three-inch layer, compared with 90 percent after one year. Some 11 to 16 percent were as deep as three to four inches and there were even some small amounts as deep as four to nine inches.

GALLAHER and EVANS (1961) found that DDT applied to the surface of pasture penetrated only to a depth of three to four inches and downward movement into the soil presumably from leaching appeared to be complete after three months. They considered leaching to be most important, although earthworms may contribute to downward movement. Solubility and mechanical transfer of ultra-fine particles was enough to account for migration of DDT downwards.

KIIGEMAGI *et al.* (1958) found no evidence that aldrin, dieldrin, or heptachlor were leached into soil because there was no difference in insecticide content between top and bottom three-inch portions of samples. LICHTENSTEIN *et al.* 1960) noted in experiments in Kansas that the persistence of aldrin, lindane, and DDT was less in one site than in two others. There was a total of 159 inches of precipitation on this site which was thought to have accounted for at least part of the faster disappearance of insecticides.

Drenches of DDT were applied to steeply sloping plots by EDWARDS (1965 b), and samples were analysed for residues after 12 months. There was more insecticide in the lower halves of the plots than in the upper halves, but the differences were not significant although there was insecticide in untreated soil lower down the slope. In a similar experiment, LICHTENSTEIN (1958) found 1.3 to 2.2 times more insecticide in the lower half of sloping plots than in the upper half after three years, with DDT showing smaller differences than lindane or aldrin, and with the greatest differences in the most steeply sloping plots. These differences, however, are most probably caused by run-off from one plot to another. It seems that, in general, leaching is responsible for little loss of chlorinated hydrocarbon insecticides and does not carry much to deeper soil.

These field observations on the distribution of chlorinated hydrocarbon insecticides in soil have been supported by laboratory experiments. JOHAN-

SON (see KIRK 1950) applied DDT wettable powder to the surface of 1.5-inch diameter columns of soil and then ran 30.3 ml. of water through them. Only one to 1.5 percent leached through to the third one-inch layer and none to the sixth. If emulsions or dusts were used leaching was greater. KIRK (1952) placed a one-inch deep layer of DDT treated soil in columns a) at the surface, b) one inch below the surface, and c) three inches below the surface. He found that the deeper the insecticide the more slowly it was lost (Table VIII).

Table VIII. *Leaching of DDT in soil* (from KIRK 1952)

Location	DDT remaining, %	DDT in next layer down, %
Top 1 inch	14.4	3.6
Second 1 inch	25.4	4.1
Fourth 1 inch	34.6	2.1

When LICHTENSTEIN (1958) poured water through columns of soil which had surface insecticide treatments there was some leaching of lindane, but the insecticide could not be detected in water collected at the bottom of the column; less insecticide was leached in soils rich in organic matter than in mineral soils.

Leaching may affect persistence of the more soluble organophosphorus insecticides in soil more than that of the chlorinated hydrocarbons, but experiments with dimethoate (BOHN 1964) showed that rainfall did not cause appreciable leaching into the three-to-six inch zone. Also, PATTERSON (1962) found that although very little of the phorate applied to soil was leached out, the amount lost by leaching depended on the amount of moisture added to the soil. BOHN (1964) showed, however, that half the dimethoate in the top three inches of treated soil disappeared in four days in drought conditions and in 2.5 days after moderate rainfall. Rain did not cause appreciable leaching into the three-to-six inch zone.

Surface run-off down slopes of six to ten in 100 occurred also with parathion (NICHOLSON *et al.* 1962). LICHTENSTEIN and SCHULZ (1964) investigated the persistence of parathion in soils kept under conditions that simulated dry areas, areas of moderate rainfall, and areas in which soils are constantly wet. After 24 days, about 85 percent remained in the dry soil, about 65 percent in the moderately wet, and 35 percent in the permanently wet soil. BARDNER (1960) found that increasing the amount of organic matter in soil increased the retention of phorate seed dressings.

There is some evidence that moisture affects the movement of parathion in soil. LICHTENSTEIN (1958) found that parathion moved from treated soil into soil that was above, below, and to the side of it. WEIDHAAS *et al.* (1961) found that soil would take up parathion and DDT from water, which is evidence that leaching is unlikely.

The moisture content of the soil can greatly influence the availability and hence toxicity of a soil insecticide by influencing the adsorption on various soil fractions. Water can compete for adsorption sites with insecticides because it is a very polar molecule which is strongly adsorbed by soil colloids, so in drier soils there are fewer water molecules to compete with the organic insecticide molecules for adsorption sites. Adsorption seems to be reversible. MENN *et al.* (1960) found that the soil moisture content had little effect on the degradation of trithion in soil but this is unusual and most workers have found soil moisture affected persistence. GEROLT (1961) stated that soil humidity, which is related to soil moisture, can influence the effectiveness of insecticides on the soil surface in three ways: a) it can influence the initial sorption of the particulate insecticide, b) it can influence the rate it diffuses into the soil, and c) it can influence the availability of the sorbed toxicant to the insect. The rate of initial sorption from the particulate state decreases with increasing humidity, particularly on soils of small sorptive capacity. The rate of inward diffusion increases with greater relative humidity. The availability of the sorbed toxicant increases with higher humidity, a ten percent increase in humidity doubling the toxicity.

In studies on the diffusion of insecticides from the surface of dried mud BARLOW and HADAWAY (1958) showed that insecticide particles disappeared from the surface of mud in inverse relation as the humidity decreased. By contrast, the sorbed insecticide diffused faster away from the surface at higher humidities. BARLOW and HADAWAY (1956) have a theory that this depended on the competition between water and insecticides. Water seems to be adsorbed in preference to the insecticide and the increased amounts in soils with high humidity can have two effects. The initial disappearance of the insecticides from the soil particles lying on the surface is retarded because the water molecules already occupy a large proportion of the active surface of the soil. Once the insecticide is adsorbed, the preferential adsorption of water results in an increased mobility of the insecticide molecules and therefore faster diffusion on to and into the insect resting on the soil.

In agricultural soils, ROBERTS (1963) found that moisture content had a considerable effect on the availability of dieldrin in soil to kill chinch bugs. There was a linear relationship between LD_{50} and soil moisture in the ranges 10 to 20 percent soil moisture and 0.8 to 2.0 p.p.m. LD_{50}. Twice as much dieldrin was required at 19.6 percent moisture content as at 10.2 percent. He considered that the principal effect of soil moisture was not on the response of the insects to the insecticide but on the degree of displacement of insecticide from the soil particles, so that more insecticide became available to the insects.

SWANSON *et al.* (1954) showed that moisture greatly lessened the ability of soil to adsorb lindane. When soil is saturated or nearly so, however, vaporization of insecticide is limited. HARRIS and LICHTENSTEIN (1961) also demonstrated that insecticides are strongly adsorbed on to dry soils. Aldrin volatilized 37 times more rapidly from wet Plainfield sand than from dry, and figures for other soils are given in Table IX. The differences in adsorption, as represented by LD_{50} values, become less pronounced as the organic matter content of the soils increases. Not only does the soil moisture content affect volatilization but also the humidity of the air above the soil surface. KALKAT *et al.* (1961) also showed that increased humidities increased the fumigant action of heptachlor, aldrin, diazinon, malathion. and parathion.

Table IX. *Fumigant LT_{50} of vapors released from 60 ml. of either oven-dry or wet soils treated with aldrin at 4 p.p.m.* (from HARRIS *and* LICHTENSTEIN 1961)

Soil Type	Water %	LT_{50} (hours)	
		In dry soil	In wet soil
Quartz sand	3.0	0.60±0.06	<0.25
Plainfield sand	6.6	22.25±0.25	0.63±0.01
Miami silt loam	26.6	51.25±0.25	1.68±0.08
Carrington silt loam	28.2	53.00±1.00	1.78±0.08
Muck I	81.8	83.50±1.00	6.00±0.15
Muck II	147.6	63.00±3.00	9.75±0.25

HARRIS (1964) noted that insecticides differ greatly in competing with water for sites on the soil particles. Parathion and diazinon were both moderately strongly adsorbed by dry soil, yet in damp soils diazinon became 134.6 times more toxic and parathion 28.3 times more toxic, indicating that parathion competes better with water than diazinon. Mevinphos was unusual in that although it was strongly adsorbed by soil it did not compete with water for the active soil sites and in soils containing three percent water or less became more effective. However, with increasing soil moisture it again became less available suggesting the possibility that it was either dissolving in the soil water or becoming sorbed at the water interfaces. HARRIS estimated that, within the range of normal field moisture contents, the toxicity of heptachlor and DDT may vary between two or three times. Using *Gryllus pennsylvanicus* as a test insect, he showed that heptachlor, DDT, diazinon, V-C 13 and parathion were more active in moist than in dry sand. Heptachlor was 7.8, DDT 9.9, parathion 24.4, diazinon 132.1, and V-C 13 188.6 times more toxic in moist sand than dry sand. Dry muck soil, however, did not inactivate the insecticides to the extent that the mineral soil did. Heptachlor and DDT were 1.2 and 1.5 times less toxic in dry muck as in moist muck and diazinon, V-C 13, and parathion were 1.5, 1.7, and 2.5 times more toxic, respectively. The insecticide seems more strongly adsorbed in a dry mineral soil than in a dry muck.

HARRIS also (1964) studied the effect of soil moisture on toxicity of other insecticides to insects in a fine sandy loam soil. The greatest effect was with trichlorfon which was 20.1 times more toxic in wet soil than in dry soil, dieldrin 16.4 times, zectran 16.1 times, DDT 15.9 times, heptachlor 12.7 times, and mevinphos 1.4 times. All these insecticides were strongly adsorbed by dry soil and, except mevinphos, became increasingly toxic with increasing moisture content. Mevinphos was less toxic in dry and wet soils than in those with intermediate water contents. WHEATLEY (1965 b) suggested that the effect of moisture on toxicity may be analogous to adsorption chromatography; the effect of moisture on toxicity is less with the relatively non-polar organochlorine compounds.

Sorption on the soil has also been shown to occur with ethylene dibromide. For instance, WADE (1955) showed that, whereas ethylene dibromide was strongly sorbed by dry soils, the sorptive capacity of the dry soil decreased with increasing humidity. He also demonstrated that less fumigant was adsorbed as soil moisture content increased. In very moist soils a small proportion of the fumigant seemed to dissolve in the soil water. WADE (1954) noted that ethylene dibromide was slightly less available in very moist soils and suggested this was because the fumigant dissolved in the soil water. CALL (1957) thought that the sorption of ethylene dibromide by soils with much moisture was primarily because it was adsorbed at the water interfaces and secondarily by solution in the soil water. PHILLIPS (1959) studied the passage of ethylene dibromide emulsions through soil and found that they passed through slower than through pure water, and they passed through a sandy loam faster than through a peaty soil, which seemed to break the emulsion more. WADE (1955) concluded that water formed a mono-layer on the soil particles which prevented sorption of ethylene dibromide.

The effects of rain and hence soil moisture content on persistence of insecticides in soil can be summarized as follows: rain may leach some of the insecticide away and, because in wet soils less insecticide is adsorbed, it may move more readily through the soil. In dry soils the insecticide is more readily retained but is bound in an inactive form until it is freed by moisture.

c) Plant cover

Open soil is exposed to greater extremes of temperature and rainfall than soil under growing crops. Several workers have studied how crops affect the persistence of soil insecticides. PARKER and DEWEY (1965) used plastic sheets to cover soils treated with phorate and dimethoate but did not find a significant difference in recovery of insecticide between covered and uncovered soils. YOUNG and RAWLINS (1958) shaded soil treated with 0.7 lb. actual heptachlor at six, weekly intervals. Although the difference in accumulation rate between the shaded and exposed soil surfaces was not significant at the five percent level of probability, their results indicated that heptachlor accumulated more in the shaded soil. Temperature may account for this as the mean maximum temperatures were as much as 25°F. higher in the exposed than in the shaded plots.

LICHTENSTEIN *et al.* (1962) found that a dense cover crop of alfalfa considerably increased the persistence of aldrin and heptachlor residues in soil. Three years after 25 lbs. actual aldrin had been applied to plots, 9.1 p.p.m.

remained in plots with an alfalfa cover, whereas bare plots contained only 4.8 p.p.m. Probably the smaller air movement under the cover crop prevented the insecticide residues from volatilizing; differences in temperature and humidity may also have affected disappearance. Movement of air over a soil surface can greatly affect the rate at which insecticide is lost from soil: HARRIS and LICHTENSTEIN (1961) showed that in still air very little insecticide was lost, so volatilization is probably greatly slowed in areas of dense vegetative cover.

A crop can take up insecticide from the soil and some of the insecticide is removed when the crop is harvested. LICHTENSTEIN (1959 and 1965) and GLASSER *et al.* (1958) reported the uptake of insecticide into carrots. LICHTENSTEIN *et al.* (1964) looked into the translocation of aldrin from soil more fully, and found that from soil with 0.24 to 1.4 p.p.m. of aldrin/dieldrin, between 0.13 and 0.37 p.p.m. could be found in harvested carrots. This does not amount to considerable removal of insecticide, as the total weight of carrots/acre is infinitesimal compared with weight of soil. More insecticide was taken up from soil treated with emulsion than from that treated with granules.

To summarize, although cropping affects insecticide persistence, the effect is probably small.

d) Microbial population of soil

The breakdown of weedkillers in soil has been much more studied than that of insecticides and it seems that they are mainly broken down by the microflora (MARTIN 1963). The role of the microflora in the breakdown of insecticides requires more investigation but current evidence indicates that micro-organisms are important. For instance AHMED and CASIDA (1958) showed that soil micro-organisms could metabolize Thimet.

BOLLEN *et al.* (1958), using sterile and non-sterile Chehalis silt loam, found that after 20 hours, 74 percent of aldrin applied could be recovered from non-sterile soil, but 78 percent from sterile soil. In a similar experiment with dieldrin, 10 percent was lost in non-sterile soil, but only 2 percent in sterile soil. The authors did not consider this difference important, but if the short exposure time is considered it is probably a very significant result.

NAUMANN (1959) showed that microorganisms influence the breakdown of parathion. He added one percent parathion to sterile and non-sterile soils and after three weeks 90 percent had decomposed in the non-sterile soil but very little had disappeared from the sterile soil. MENN *et al.* (1960) studied the breakdown of trithion in soil. When 10 p.p.m. actual of five percent granular was added to Santa Cruz sand, 50 percent disappeared in 170 days. When this soil was autoclaved the time required for 50 percent degradation was 525 days. Trithion also persisted up to four times longer in soil treated with Vapam than in untreated soil. LICHTENSTEIN and SCHULZ (1960) investigated the effect of autoclaving wet and dry sand and loam soils on the persistence of aldrin and heptachlor in them. Aldrin disappeared fastest in wet non-autoclaved soil, only 2.06 p.p.m. remaining after 112 days compared with 5.11 p.p.m. in wet autoclaved soil. Most of the difference could be accounted for by aldrin converting to dieldrin. In dry autoclaved soil 7.87 p.p.m. remained. Comparable figures for heptachlor were in wet non-autoclaved soil 3.62 p.p.m. and in wet autoclaved soil 4.54 p.p.m. Between the 56*th* and 112*th* day of the experiment, the rate insecticide disappeared increased gradually, corresponding with an increase in numbers of micro-organisms. The faster disappearance in wet than in dry soil may be because micro-organisms are more active in wet than in dry soil.

In later work, LICHTENSTEIN and SCHULZ (1964) investigated the effect of soil microbes on breakdown of parathion in a loam soil. Samples of the same soil were either left untreated or had glucose solution added and were incubated at 30 °C. for seven days. Other samples were autoclaved at 120 °C. for seven hours daily for five days. All were then treated with 200 p.p.m. of parathion and analysed after six days for residues by the Averell-Norris colorimetric method and by gas-liquid chromatography. The untreated soil had 106.8 to 107.5 p.p.m., the glucose-treated soil 82.9 to 94.7 p.p.m., and the autoclaved soil 163.9 to 171.7 p.p.m. Aminoparathion had formed in the glucose-treated soil and a small amount in the untreated soil, but none in the autoclaved soil. This work suggested that under 'natural' conditions the breakdown of parathion was primarily hydrolytic but larger numbers of certain micro-organisms reduced the parathion molecule. Numbers of bacteria in the untreated and glucose-treated soils differed only slightly, but there were as many as 20 times more yeasts in the glucose-treated soils. The importance of yeasts in parathion breakdown was substantial; adding yeast to solutions of parathion in water increased the rate of breakdown.

e) Cultivation

Insecticides applied to control soil-borne pests are usually cultivated into the soil immediately after being applied, whereas those falling from foliage sprays may remain undisturbed on the surface for weeks or months; hence, the persistence of residues from the two different kinds of treatment can differ greatly. WHEATLEY *et al.* (1961) found that 95 percent of a dosage of one lb. actual/acre of aldrin applied to the surface of sandy loam disappeared in six weeks during summer, little being transformed into dieldrin; by contrast, when the insecticide was mixed into the soil, 40 percent remained as aldrin and 10 percent as dieldrin 20 weeks after treatment.

BIGGER (1955) found that a delay of three to four hours between the time heptachlor was applied to a field and when it was mixed with the soil halved its effectiveness against root-feeding insects compared with when it was immediately cultivated in, suggesting a rapid loss of this insecticide from the soil surface.

LICHTENSTEIN and POLIVKA (1959) concluded that aldrin persisted longer where it had been applied to bare soil and disked in than when it was applied to turf and left uncultivated. MULLA (1960) showed that there was considerable loss of endrin and even more of DDT when surface sprays were not immediately cultivated into the soil.

LICHTENSTEIN *et al.* (1962) applied aldrin and heptachlor emulsions to the surfaces of plots with a watering can. Some plots were rototilled to a depth of five inches and others were left untilled. The tilled plots contained 26 to 50 percent of the applied insecticide four months after treatment and the untilled plots only 2.7 to 5.3 percent. LICHTENSTEIN *et al.* (1964) (see Fig. 1) found that when aldrin was incorporated into the upper four-to-five inch layer of a Carrington silt loam 44 to 62 percent of the amount applied was recovered as aldrin or dieldrin one year after application. When the insecticide was left on the soil surface, only 6.5 to 13 percent was recovered. They attributed this to greater volatilization because of exposure to warmer temperatures and to wind.

They also found that the method of applying insecticide to the soil affected its persistence. Five months after the insecticide had been applied to the soil surface all the residues were in the upper two-inch layer. When the aldrin was incorporated into the upper four to five inches of soil by rototilling, 80 percent of the totally recovered residues five months after treatment, were in the upper two-inch layer, 18 to 19 percent in the two-to-four inch layer, and traces in the five-to-nine inch layer.

PARKER and DEWEY (1965) found that phorate and dimethoate were lost more slowly when mixed thoroughly into an air-dry Lordstown stony silt loam soil and held in a thin layer in the greenhouse than when applied to the soil surface.

LICHTENSTEIN and SCHULZ (1961) showed that regular cultivation decreased amounts of insecticides in soil; they disked a loam soil treated with four lbs. actual of DDT or aldrin/acre daily for three months and found that the residues of DDT diminished by 25 percent and those of aldrin by 38 percent.

The depth to which the insecticide is incorporated into the soil also affects its persistence. YOUNG and RAWLINS (1958) raked weekly applications of heptachlor superficially into soil with a hand rake, but this had little influence on the length of time the insecticide persisted, whereas thoroughly mixing heptachlor with soil in large containers in the laboratory doubled the amount that remained after 21 months: 53 percent as compared with 26 percent which remained in field plots treated at the same time.

It is very difficult to mix insecticides deep into field soils. EDWARDS (1964) rotovated soil sprayed with insecticide, to a depth of nine inches, in two directions at right angles to each other, and found that 46.5 percent was in the top inch of soil, 40.5 percent in the second, 10.2 percent in the third, 1.4 percent in the fourth, and only 1.4 percent had penetrated below a depth of four inches. WHEATLEY and HARDMAN (1959) also showed that dieldrin was not mixed in to the full depth of cultivation when rotovated in, and found that ploughing was more effective in getting insecticides into soil;

it is doubtful however whether insecticides are distributed evenly in the vertical plane by ploughing.

Thus the persistence of insecticides in soil clearly depends on how deeply they are mixed into the soil; even very persistent chemicals disappear during a period of months when on the soil surface.

V. Conclusions

It is much more difficult to predict the disappearance of an insecticide from soil than it is from a simple non-sorptive sprayed surface, where—as found by many workers—the decay curves of many non-volatile insecticides such as DDT fit a simple exponential or logarithmic decay curve for a first-order reaction of the form $r = e^{-kt}$ where t = time after application, k = rate of loss, and r = amount of residue.

In soil none of the insecticides seem to fit such a simple model. Measurements of their decay can best be explained by assuming two, three, or four phases of breakdown for most of the chlorinated hydrocarbon insecticides in soil (Fig. 4). These curves resemble fundamentally those postulated by GUNTHER and BLINN (1955) for the disappearance of insecticides on and in plant tissues. During application of a calculated dose of insecticide to soil, there are various losses, such as loss of spray by drift, dust may be blown away, etc.; some insecticide may remain on the cultivators. Only rarely does the quantity of insecticide intended to be applied actually become incorporated into the soil, as pointed out by many workers.

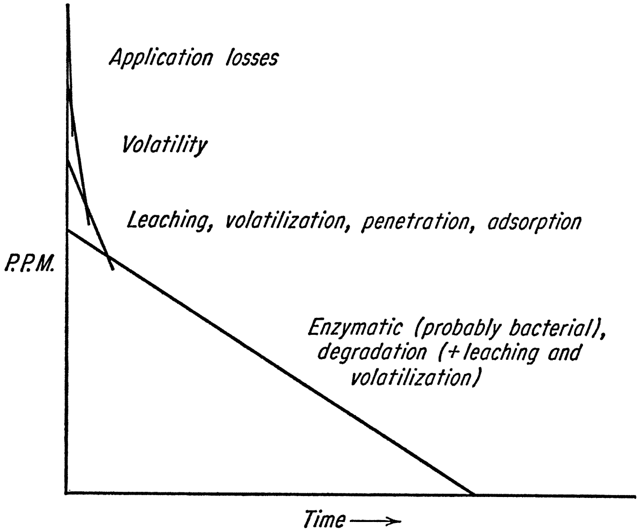

Fig. 4. Theoretical breakdown curve for soil insecticides

After the insecticide has been applied, the period between application and cultivation into the soil may be critical. With a relatively volatile insecticide such as aldrin, I have found that considerable losses can occur even in a few hours. Laboratory evidence for such losses comes from work by HARRIS and LICHTENSTEIN (1961) who found that 100 percent mortality of fruit flies occurred in six hours, when they were exposed to vapour emitted from quartz sand, treated with a dose as small as four p.p.m. of aldrin. Losses between application and cultivation depend very much on the volatility of the insecticide.

There is probably a third phase when the insecticide has been incorporated into the soil but has still to reach a stable form. During this phase there are probably further actual losses due to volatility or leaching if the insecticide is even slightly volatile or soluble, and apparent losses resulting from adsorption or penetration of the insecticide into soil structure. Penetration of most chlorinated hydrocarbon insecticides from the surface of the soil has been adequately demonstrated by HADAWAY and BARLOW (1949 *et seq.*) and DOWNS *et al.* (1951). Adsorption is also a gradual process, as the insecticide molecules gradually find available sites on the soil colloids. This process probably takes from a few days to several months to be completed.

In the final phase, losses may continue to occur by leaching, volatilization, and uptake by plants, but most losses are likely to be by actual degradation, probably mainly from bacterial action. This may approximate much more closely to a true exponential loss. Regressions calculated by the author show good agreement with a logarithmic disappearance during this phase for most chlorinated hydrocarbon insecticides.

Some workers (LICHTENSTEIN 1959 b, WHEATLEY and HARDMAN 1960) have used the term "half-life" to represent the disappearance of insecticides in soil. It can be seen that if the decay curve is built from several overlapping phases it is difficult to define "half-life" values based on the usual expression (GUNTHER and BLINN 1955)

$$t_{1/2} = \frac{2.303 \times \log 2}{k} \text{ where } k = \text{velocity constant slope} \times 2.303.$$

To calculate a "half-life" all the phases would have to be considered separately.

Losses in the first two phases can be greatly lessened by applying the insecticide carefully, but those in the third phase depend much more on the specific insecticide and soil into which it is incorporated. They are probably minimal when a non-volatile insecticide is applied to a non-adsorptive soil; the nearest approach to this is when DDT is applied to a sandy soil with little clay or organic matter. In such a soil there would probably be some leaching but this is much less important than losses from adsorption or penetration. Figure 5 shows that the disappearance of DDT approximates to a simple exponential curve. At the other extreme aldrin differs considerably and this, almost certainly, because of greater volatility.

Before an attempt can be made to predict the persistence of a particular insecticide in a particular soil type, the relative importance of the various

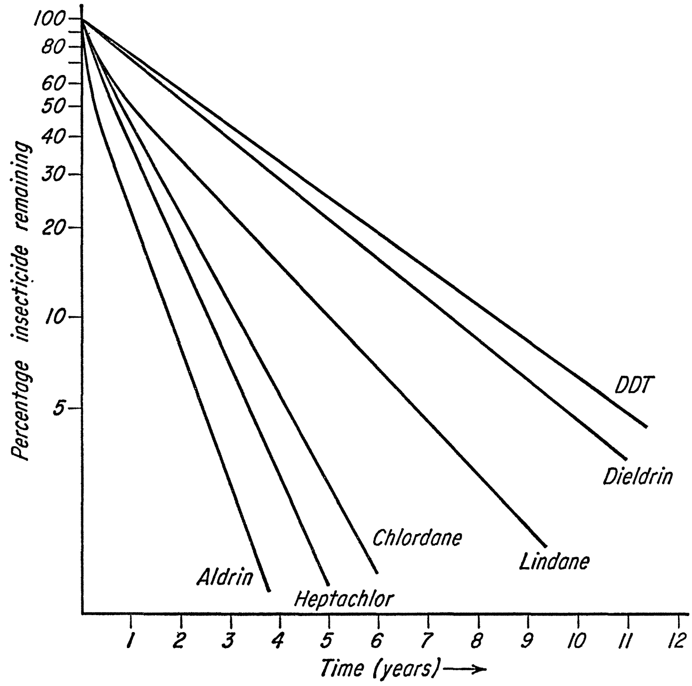

Fig. 5. Breakdown of chlorinated hydrocarbon insecticides in soil. Regression based on all available data

influencing factors reviewed here must be assessed. This can still only be done in a general way; for really accurate predictions much more precise information is required. An idea of the relative importance of the factors can be obtained by separating them into arbitrary classes of gradually decreasing importance.

a) Primary factors

The most important general factor is the chemical structure of the insecticide and its intrinsic stability. Probably the most important properties of the insecticide are its solubility and volatility. The primary importance of the insecticide is demonstrated by Figure 5. Even when the chemical has been applied to a range of soils, in different climatic conditions, the regressions for the various chemicals are still quite distinct.

b) Secondary factors

Of next importance is the type of soil to which the insecticide is applied. The most important single soil characteristic is its organic matter content, as organic matter seems to be the most important single adsorpive site for the insecticide. Other soil features such as clay content and soil structure also influence adsorption but their importance has not yet been fully assessed. The other main factor is the rainfall; losses are greatest by leaching and desorption in soil which are frequently wet.

c) Tertiary factors

The soil temperature has considerable importance in controlling disappearance of soil insecticide residues but it is probably less significant than soil moisture. Changes in soil temperature are usually much less extreme than those in soil moisture; at a depth of six inches in the soil, temperatures show only small fluctuations. The microbial population of soil has an important role in breaking down soil insecticides but it is probably not limiting as it can usually multiply rapidly when and as required. The cultivation of the insecticide into the soil can be considered as of tertiary importance.

d) Quaternary factors

The other factors that have been considered come under this heading; they include the factors of formulation and concentration of the insecticide and for soil its mineral content and acidity. The amount of plant cover probably has relatively little influence on persistence.

These categories are arbitrary and in some situations a particular factor may have greatly increased importance.

There is also little doubt that while we continue to use chemicals to control insects there is need for insecticides persistent enough to fulfil their task but which do not remain in the soil long enough to constitute a hazard to man, crops, or wildlife.

Table X. *Common and chemical names of pesticides mentioned in text*

Aldrin—1,2,3,4,10,10-hexachloro-1,4,4a,5,8,8a-hexahydro-1,4-*endo, exo*-5,8-dimethanonaphthalene
Bayer 37342—*O,O*-dimethyl-*O*-(3,4-dimethyl-4-methylthiophenyl) phosphorothionate
Baytex—Trade name for fenthion
BHC—Mixed isomers of 1,2,3,4,5,6-hexachlorocyclohexane
Carbophenothion—*S*-(4-chlorophenylthiomethyl) diethyl phosphorothiolothionate
Chlordane—1,2,4,5,6,7,10,10-octachloro-4,7,8,9,-tetrahydro-4,7-methyleneindane
DDT—1,1,1-trichloro-2,-2-*bis*(*p*-chlorophenyl) ethane
DDVP—Trade name for dichlorvos
Demeton—*O,O*-diethyl *O*(and *S*)-(2-ethylthio) ethyl phosphorothioate
Diazinon—diethyl 2-isopropyl-6-methyl-4-pyrimidinyl phosphorothionate
Dichlorvos—2,2-dichlorovinyl dimethylphoshate
Dieldrin—1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4-*endo,exo*-5,8-dimethanonaphthalene
Dimethoate—*O,O*-dimethyl *S*-(*N*-methylcarbamoylmethyl) phosphorodithioate
Disulfoton—*O,O*-diethyl *S*-[2-(ethylthio) ethyl] phosphorodithioate
Di-Syston—*O,O*-diethyl *S*-2-(ethylthio) ethyl phosphorodithioate

Endrin—1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4-*endo, endo*-5,8-dimethanonaphthalene
Fenthion—dimethyl 3-methyl-4-methylthiophenyl phosphorothionate
Guthion—*0,0*-dimethyl *S*-4-oxo-1,2,3-benzotriazin-3(4*H*)-ylmethyl phosphorodithioate
Heptachlor—1,4,5,6,7,8,8-heptachloro-3a,4,5,5a-tetrahydro-4,7-*endo*-methanoindene
Heptachlor epoxide—1,4,5,6,7,8-heptachloro-2,3-epoxy-2,3,3a,7a-tetrahydro-4,7-methanoindene
Lindane—gamma isomer of 1,2,3,4,5,6-hexachlorocyclohexane
Malathion—*S*-[1,2-di(ethoxycarbonyl)ethyl]dimethyl phosphorothiolothionate
Menazon—*0,0*-dimethyl *S*-(4,6-diamino-1,3,5-triazin-2-ylmethyl) phosphorodithioate
Methyl parathion—*0,0*-dimethyl *0*-*p*-nitrophenyl phosphorothioate
Mevinphos—*cis*-2-methoxycarbonyl-1-methylvinyl dimethylphosphate
Parathion—*0,0*-diethyl *0*-*p*-nitrophenyl phosphorothioate
Phorate—*0,0*-diethyl-*S*-(ethylthiomethyl)phosphorodithioate
Phosdrin—dimethyl 2-methoxycarbonyl-1-methylvinyl phosphate
Schradan—octamethylpyrophosphoramide
Sumithion—*0,0*-dimethyl *0*-(3-methyl-4-nitrophenyl)phosphorothionate
TDE—1,1-dichloro-2,2-*bis*(*p*-chlorophenyl)ethane
Telodrin—1,3,4,5,6,7,8,8-octachloro-3a,4,7,7a-tetrahydro-4,7-methophthalan
Thimet—Trade name for phorate
Trichlorfon—dimethyl 2,2,2-trichloro-1-hydroxyethylphosphonate
Trithion—Trade name for carbophenothion
Vapam—sodium *N*-methyl-dithiocarbamate
V-C 13—*0*-2,4-dichlorophenyl *0,0*-diethyl phosphorothioate
Zectran—4-dimethylamino-3,5-xylyl methyl carbamate

Summary

The most common and persistent insecticide residues in soil are DDT and dieldrin, and then lindane, chlordane, heptachlor, and aldrin in order of decreasing persistence. Organophosphorus insecticides are much shorter-lived, remaining in the soil for months at most. The chemical structure of the insecticide and its resultant intrinsic stability is the most important single factor governing its persistence in soil. The more volatile an insecticide the shorter time it persists in soil and the effectiveness of a soil insecticide tends to be inversely proportional to its water solubility. The larger the dose of insecticide applied to soil the less disappears in terms of percentage of the original application in a given time. The formulation of an insecticide affects its persistence in soil; granules persist longer than emulsions, which in turn persist longer than miscible liquids; wettable powders and dusts disappear most rapidly of all.

The type of soil treated with an insecticide greatly influences how long residues persist; for instance, heavy clay soils retain insecticides much longer than lighter sandier ones. The phenomenon of adsorption of insecticides on to soil seems to occur with most insecticides; they may, however, become bound to the soil so tightly that they may be non-toxic to either insects or plants. When an insecticide is applied to the surface of mud it gradually becomes adsorbed into the interior of the mud; this must also occur with soil. Organic matter content seems to be the most important single soil factor influencing how long insecticide residues persist in soil, although the amount of clay in a soil can also be correlated with the persistence of residues. There is evidence that organophosphorus insecticides persist longer in acid soils than in alkaline ones and the amount of mineral ions such as Fe, Al, and Mg present can also affect the adsorption and persistence of insecticides in soil.

Climatic factors have a considerable effect on soil insecticide persistence. Increased temperatures accelerate the loss of insecticide by increasing conversion to other compounds, volatilization, and desorption, but coincidentally if the soil becomes dry, the loss of insecticide is retarded. The organochlorine insecticides are very weakly soluble and are seldom leached deeper than plough depth. Soil moisture greatly affects both the adsorption of insecticides and their movement through soil; in dry soils insecticides are tightly adsorbed and have much less insecticidal action.

Soil insecticides are lost less rapidly from soils with a cover crop, probably because the air movement over the soil surface is lessened. It has been demonstrated that soil microorganisms are important in the breakdown of soil insecticides. Insecticide residues left on the soil surface disappear many times faster than if they are cultivated into the soil.

Four overlapping phases are suggested to analyse the breakdown curves of soil insecticides. These include, firstly, application losses, then a phase of losses due to volatilization from the soil surface. After cultivation there is a stage during which the insecticide spreads through the soil and becomes stabilized; during this phase, losses are mainly by leaching, adsorption, and volatilization. In the final phase losses continue by leaching and volatilization, but are mainly by enzymatic degradation, and approximate to a regular exponential curve.

The various factors affecting soil insecticide persistence can be arbitrarily placed in decreasing order of importance as follows:

a) The chemical structure of the insecticide.

b) The type of soil to which the insecticide is applied, especially depending on the organic matter content; amount of rainfall reaching treated soil and the moisture in the soil.

c) The microbial population of the treated soil; depth of cultivation of the insecticide; mean temperatures in the treated soil.

d) The mineral content and acidity of the treated soil; amount of plant cover over treated soil; formulation and concentration of the insecticide.

Résumé*

Les résidus les plus courants et les plus durables d'insecticides dans le sol sont, par ordre de persistance décroissante, ceux du D.D.T. et de la Dieldrine, ensuite, du Lindane, du Chlordane, de l'Heptachlore et de l'Aldrine. Les insecticides organo-phosphorés ont une vie beaucoup plus courte et ne persistent dans le sol que quelques mois au plus. La structure chimique de l'insecticide et la stabilité intrinsèque qui en résulte est le facteur le plus

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* Traduit par R. MESTRES.

important déterminant sa persistance dans le sol. Plus l'insecticide est volatil, moins il persiste dans le sol et l'efficacité d'un insecticide du sol tend à être inversement proportionnelle à sa solubilité dans l'eau. Plus la dose d'insecticide introduite dans le sol est grande, moins est importante la perte en pourcentage de la dose initiale en un temps donné. La formulation d'un insecticide affecte sa persistance dans le sol: les granules persistent plus longtemps que les émulsions, qui, à leur tour, persistent plus longtemps que les liquides miscibles; les poudres mouillables et les poudres sont plus rapidement dissipées.

La nature du sol traité influence beaucoup la persistance: par exemple, les sols très argileux retiennent les insecticides beaucoup plus longtemps que les terrains plus légers plus riches en sable. Le phénomène de l'absorption des insecticides par le sol paraît survenir avec la plupart des insecticides; ils peuvent cependant se lier si fortement au sol qu'ils ne sont alors plus toxiques pour les insectes et les plantes. Lorsqu'un insecticide est déposé à la surface d'une boue il s'adsorbe progressivement à l'intérieur de la boue: ce phénomène doit également se produire avec le sol. La teneur en matières organiques paraît être le facteur particulier le plus important pour la persistance de l'insecticide dans le sol, bien que la quantité d'argile puisse également être en relation avec la persitance des résidus. Les insecticides organophosphorés persistent plus longtemps dans les terrains acides que dans les terrains alcalins et les teneurs en ions minéraux tels que Fe, Al et Mg peuvent également affecter l'adsorption et la persistance des insecticides dans le sol.

Les facteurs climatiques affectent considérablement la persistance des insecticides dans le sol. L'élévation de la température accélère la perte d'insecticide en augmentant la conversion en d'autres composés, la volatilisation et la désorption, mais si elle entraîne un asséchement, la perte d'insecticide est retardée.

Les composés organo-chlorés sont très peu solubles et sont rarement entrainés par l'eau au dessous de la terre arable. L'humidité du sol affecte beaucoup l'adsorption des insecticides et leur mouvement à travers le sol; dans les sols secs les insecticides sont fortement adsorbés et leur activité est bien moindre.

Les insecticides sont moins rapidement perdus des sols recouverts de végétation probablement parce que la ventilation est réduite à la surface du sol. Il a été démontré que les micro-organismes du sol sont un facteur important du catabolisme des insecticides. Les insecticides déposés en surface disparaissent beaucoup plus vite que lorsqu'ils sont enfouis.

Quatre phases interdépendantes sont suggérées pour analyser les courbes de disparrtion des insecticides dans le sol. Elles comportent en premier lieu les pertes à l'application, ensuite les pertes dues à la volatilisation depuis la surface du sol. Après la culture intervient une période pendant laquelle l'insecticide se répand dans le sol et se stabilise; pendant cette phase les pertes surviennent principalement par lessivage, adsorption et volatilisation. Dans la phase finale les pertes continuent par lessivage et volatilisation mais elles surviennent surtout par dégradation enzymatique et se rapprochent d'une

courbe exponentielle normale. Les différents facteurs affectant la persistance des insecticides dans le sol peuvent être arbitrairement classés par ordre d'importance décroissante comme suit:

a) la structure chimique de l'insecticide

b) la nature du sol et notamment sa teneur en matières organiques les précipitations et l'humidité du sol

c) la faune microbienne du sol traité, la profondeur d'enfouissement de l'insecticide et la température moyenne du sol

d) la constitution chimique et l'acidité du sol traité, sa couverture végétale, la formulation et la concentration de l'insecticide.

Zusammenfassung*

Die am meisten verbreiteten und widerstandfähigsten Insektizid-Rückstände im Boden sind DDT und Dieldrin, gefolgt von Lindan, Chlordan, Heptachlor und Aldrin in der Reihenfolge abnehmender Persistenz. Hingegen sind organische Phosphor-Insektizide viel kurzlebiger und verbleiben im Boden höchstens einige Monate. Die chemische Struktur des Insektizids und die daraus resultierende Stabilität ist der wichtigste Einzelfaktor, durch den die Beständigkeit im Boden bestimmt wird. Je flüchtiger ein Insektizid ist, umso kürzer ist die Verweil-Zeit im Boden. Die Wirksamkeit eines Boden-Insektizides ist irreversibel proportional zu seiner Wasser-Löslichkeit. Je höher die Anwendungsdosis des Insektizids ist, umso langsamer verschwindet es aus dem Boden, ausgedrückt im Prozentsatz der Aufwandmenge zu einem gegebenen Zeitpunkt. Auch die Formulierung eines Insektizids beeinflusst dessen Beständigkeit im Boden: Granulate bleiben länger erhalten als Emulsionen, welche hingegen stabiler sind als echte Lösungen; Spritz-Pulver und Stäube werden am schnellsten abgebaut.

Die Persistenz der Insektizid-Rückstände wird wesentlich durch den Boden-Typ beeinflusst: so halten z.B. schwere Lehm-Böden Insektizide viel länger fest als leichte Sand-Böden. Die meisten Insektizide sind der Erscheinung der Adsorption an Bodenteilchen unterworfen: sie können jedoch so fest gebunden werden, dass sie für Insekten und Pflanzen nicht mehr toxisch sind. Wenn ein Insektizid auf die Oberfläche von Schlamm appliziert wurde, so wird es zunehmend im Innern der Schlamm-Masse adsorbiert; das dürfte auch beim Boden der Fall sein.

Der Gehalt an organischem Material scheint der wichtigste Bodenfaktor für die Persistenz von Insektizid-Rückständen zu sein. Jedoch kann auch der Ton-Gehalt mit der Beständigkeit der Rückstände korreliert werden.

Wahrscheinlich bleiben organische Phosphor-Insektizide in sauren Böden länger erhalten, als in basischen. Der Gehalt an Metall-Ionen, wie Fe, Al und Mg kann ebenfalls die Adsorption und die Persistenz der Insektizide im Boden beeinflussen.

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* Übersetzt von H. F. LINSKENS.

Klimatische Faktoren haben einen beträchtlichen Einfluss auf die Beständigkeit der Insektizide im Boden. Steigende Temperatur beschleunigt den Abbau der Insektizide durch zunehmenden Umbau in andere Verbindungen, erhöhte Flüchtigkeit und Desorption. Wenn jedoch der Boden zwischenzeitlich austrocknet, wird der Verlust an insektizider Wirksamkeit verlangsamt. Insektizide auf der Basis chlorierter Kohlenwasserstoffe sind sehr gut löslich und werden selten tiefer ausgewaschen als Pflugschar-Tiefe. Die Bodenfeuchtigkeit beeinflusst in hohem Masse sowohl die Adsorption der Insektizide, als auch ihre Beweglichkeit im Boden. In trockenen Böden sind die Insektizide stark adsorbiert und haben eine sehr geringe insektizide Wirksamkeit.

Boden-Insektizide verschwinden aus Böden, die eine Pflanzendecke tragen langsamer, –wahrscheinlich, weil die Luftbewegung über der Bodenoberfläche reduziert ist. Es konnte gezeigt werden, dass Boden-Mikroorganismen für den Abbau von Boden-Insektiziden von Bedeutung sind. Insektizid-Rückstände verschwinden viel schneller an der Oberfläche des Bodens, als wenn sie durch Bodenbearbeitung in den Boden hinein gebracht wurden.

Für die Analyse der Abbau-Kurven von Boden-Insektiziden werden vier sich überlappende Phasen vorgeschlagen: 1. Verluste bei der Applikation. 2. Verlust durch Flüchtigkeit an der Boden-Oberfläche. 3. Nach der Bearbeitung folgt ein Stadium, in der das Insektizid sich im Boden verbreitet und stabilisiert wird; während dieser Phase kommen Verluste hauptsächlich zustande durch Auswaschung, Adsorption und Verdampfung. 4. In der letzten Phase schliesslich setzen sich die Verluste durch Auswaschen und Verdampfen fort, beruhen jedoch hauptsächlich auf enzymatischem Abbau, sodass eine regelmässige, exponentielle Abbau-Kurve erreicht wird.

Die verschiedenen Faktoren, welche die Persistenz von Boden-Insektiziden beeinflussen, können in erster Annäherung in der Reihenfolge ihrer Bedeutung folgendermassen aufgeführt werden:

a) Die chemische Struktur des Insektizids.

b) Der Bodentyp, auf welchem das Insektizid angewendet wurde. Dabei spielen der Gehalt an organischem Material, die Niederschlagsmenge nach der Bearbeitung, sowie die Bodenfeuchtigkeit eine besondere Rolle.

c) Die Zusammensetzung der Mikroorganismen-Population des behandelten Bodens; die Tiefe, in welche die Insektizide durch Kulturmassnahmen gebracht werden; die mittlere Temperatur im behandelten Boden.

d) Der Gehalt an Metall-Ionen, sowie der Säuregrad des Bodens; die Dichte des Bewuchses über dem behandelten Boden; Formulierung und Konzentration des Insektizids.

References

ACKLEY, W. B., K. C. WALKER, and N. R. BENSON: DDT residues in orchard soils. Proc. Wash. State Hort. Assoc. **46**, 1 (1950).

AHMED, M. K., and J. E. CASIDA: Metabolism of some organophosphorus insecticides by microorganisms. J. Econ. Entomol. **51**, 59 (1958).

Aldrich, R. J.: Herbicides; residues in soils. J. Agr. Food Chem. **1**, 257 (1953).

Alexander, M.: Persistence and biological reactions of pesticides in soils. Proc. Soil Sci. Soc. Amer. **29**, 1 (1965).

Allen, N., R. L. Walker, and L. C. Fife: Persistence of BHC, DDT and toxaphene in soil and the tolerances of certain crops to their residues. U.S. Dept. of Agr. Tech. Bull. **1090**, 19 (1954).

App, B. A., R. H. Carter, and R. E. Ely: Residues on forage, in the soil, and in milk following pasture treatment with granulated dieldrin. J. Econ. Entomol. **49**, 136 (1956).

Appleman, M. C., and O. H. Sears: Effects of DDT on nodulation of legumes. J. Amer. Soc. Agron. **38**, 545 (1946).

Bailey, G. W., and J. L. White: Review of adsorption and desorption of organic pesticides by soil colloids with implications concerning pesticide bioactivity. J. Agr. Food Chem. **12**, 324 (1964).

Bami, H. L.: Sorption of 75% DDT water-dispersible powder on different muck surfaces. Bull. World Health Org. **24**, 567 (1961).

Banham, F. L.: Persistence of certain soil insecticides for control of the tuber flea-beetle, *Epitrix tuberis* (Gent) in the interior of British Columbia. Canad. J. Plant Sci. **41**, 664 (1961).

Bardner, R.: Some factors affecting the toxicity of systemic insecticidal seed dressings. Proc. 11th Internat. Congress Entomol., p. 558. Vienna 1960.

—, K. A. Lord, and S. R. B. Solly: A cholinesterase inhibition method of determining the distribution of organophosphorus insecticides in soils. Chem. & Ind. **3**, 123 (1963).

Barlow, F., and A. B. Hadaway: Studies on aqueous suspensions of insecticides V. The sorption of insecticides by soils. Bull. Entomol. Research **46**, 547 (1955).

— — Effect of changes in humidity on the toxicity and distribution of insecticides sorbed by some dried soils. Nature **178**, 1299 (1956).

— — Studies on aqueous suspensions of insecticides. Pt. VI. Further notes on the sorption of insecticides by soils. Bull. Entomol. Research **49**, 315 (1958 a).

— — Studies on aqueous suspensions of insecticides. Pt. VI. Further notes on the sorption of insecticides by soils. Bull. Entomol. Research **49**, 315 (1958 a).

— — Studies on aqueous suspensions of insecticides. Pt. VII. The influence of relative humidity upon the sorption of insecticides by soils. Bull. Entomol. Research **49**, 333 (1958b).

Barthel, W. F., R. T. Murphy, W. G., Mitchell and C. Caley: The fate of heptachlor in the soil following granular application to the surface. J. Agr. Food. Chem. 8, 445 (1960).

Bertagna, P.: Residual insecticides and the problem of sorption. Bull. World Health Org. **20**, 861 (1959).

Bigger, J. H.: A report given at spray operators school, Urbana, Ill. Jan. 20-21. Agr. Chemicals **10**, 122 (1955).

Birrell, K. S.: Thermal decomposition of DDT by some soil constituents. New Zealand J. Sci. **6** (2), 169 (1963).

Bishop, R. F., and D. Chisholm: Arsenic accumulation in Annapolis Valley orchard soils. Canad. J. Soil Sci. **42**, 77 (1962).

Bohn, W. R.: The disappearance of dimethoate from soil. J. Econ. Entomol. **57**, 798 (1964).

Bollen, W. B., J. E. Roberts, and H. E. Morrison: Soil properties and factors influencing aldrin—dieldrin recovery and transformation. J. Econ. Entomol. **51**, 214 (1958).

Burt, P. E., R. Bardner, and P. Etheridge: The influence of the volatility and water solubility of systemic insecticides on their movement through soil and absorption by plant roots. Ann. Applied Biol. **56**, 411 (1965).

CALL, F.: Soil fumigation IV—Sorption of ethylene dibromide on soils at field capacity. J. Sci. Food Agr. 8, 137 (1957).

CARLO, C. P., D. ASHDOWN, and V. G. HELLER: The persistence of parathion, toxaphene and methoxychlor in soil. Oklahoma Agr. Expt. Station Tech. Bull. M.T. **42**, 11 (1952).

CHISHOLM, D., A. W. MACPHEE, and C. R. MACEACHERN: Effects of repeated applications of pesticides to soil. Canad. J. Agr. Sci. **35**, 433 (1955).

—, L. K. KOBLITSKY, J. E. FAHEY, and W. E. WESTLAKE: DDT residues in soil. J. Econ. Entomol. **43**, 941 (1951).

— — Testing soils for insecticide residues. Agr. Chemicals **7** (2), 47 (1952).

— — Accumulation and dissipation of pesticide residues in soil. Trans. 24th N. Amer. Wildlife Conf., p. 118 (1959).

— —, and W. E. WESTLAKE: The estimation of aldrin and chlordane residues in soils treated for termite control. U. S. Dep't. of Agr., Agr. Research Service, 1962.

CHULSKI, K.: The effect of BHC on some crops grown on various soil types. Mich. Agr. Exp't. Station Quart. Bull. **31**, 170 (1948).

CWILICH, R.: The problem of bioassay of insecticides in soil. Z. Pflanzenkrankh. **69**, 270 (1962).

DECKER, G. C., W. N. BRUCE, and J. H. BIGGER: The accumulation and dissipation of residues resulting from the use of aldrin in soils. J. Econ. Entomol. **58**, 266 (1965).

DOWNS, W. G., E. BORDAS, and L. NAVARRO: Duration of action of residual DDT deposits on adobe surfaces. Science **114**, 259 (1951).

DOWSEY, L. H., D. W. WOODHAM, and C. S. LOFGREN: Heptachlor and heptachlor epoxide residues in truck crops. J. Econ. Entomol. **54**, 1264 (1961).

DURR, H. R. R., C. J. JOUBERTS, and S. S. WALTERS: Effect of application to the soil of aldrin, dieldrin and chlordane on infestations of the Argentine ant (*Iridomymex humilis)* during period of four years. S. African J. Agr. Sci. **1**, 75 (1958).

EDWARDS, C. A.: Persistence of insecticides in the soil. New Scientist **19**, 282 (1963).

— Factors affecting the persistence of insecticides in soil. Soils & Fert. **XXVII**, 451 (1964).

— Effects of pesticide residues on soil invertebrates and plants. Ecology and the industrial society. Vth Symp. Brit. Ecol. Soc. Beackwell, Oxford, p. 239 (1965 a).

— Unpublished data (1965 b).

—, S. D. BECK, and E. P. LICHTENSTEIN: Bioassay of aldrin and lindane in soil. J. Econ. Entomol. **50**, 623 (1957).

—, and G. W. HEATH: The principles of agricultural entomology. London: Chapman & Hall 1964.

—, and K. A. JEFFS: The persistence of some insecticides in soil and their effects on soil animals. Proc. XIIth Internat. Congress Entomol., London, p. 559 (1964 and 1965).

EVANS, W. C.: The breakdown of pesticides in the soil. Proc. 5th Internat. Congress Pesticides, in press. London 1964.

FILMER, R. S., and C. L. SMITH: Residual effect of DDT on *Apis*. J. Econ. Entomol. **37**, 537 (1944).

FLEMING, W. E.: Effect on plants of DDT applied to soil for the destruction of Japanese beetle larvae. U.S. Dep't. of Agr. Bur. Entomol. and Plant Quar. Bull. E-737 (1848).

— Bioassay of soil containing residues of chlorinated hydrocarbon insecticides with special reference to control of Japanese beetle *(p. japonica)* grubs. U. S. Dep't. of Agr. Tech Bull. 1266, p. 44 (1962).

—, L. W. COLES, and W. W. MAINES: Biological assay of residues of DDT and chlordane in soil using *Macrocentrus ancylivorus* as a test insect. J. Econ. Entomol. **44**, 310 (1951).

—, and W. W. MAINES: The effectiveness and duration of treatments with technical DDT in different soils against larvae of the Japanese beetle. U. S. Dep't. of Agr. Bur. of Entomol. and Plant Quar. Bull. E-716, p. 20 (1947).

— — Effect of chlorinated organic compounds on plants grown in treated soil. U. S. Dep't. of Agr. Bur. of Entomol. and Plant Quar. Bull. E-872 (1952).

— — Persistence of DDT in soils of the area infested by the Japanese beetle. J. Econ. Entomol. **46**, 445 (1953).

— — Persistence of chlordane in soils of the area infested by the Japanese beetle. J. Econ. Entomol. **47**, 165 (1954).

— —, and L. W. COLES: Persistence of chlorinated hydrocarbon insecticides in turf treated to control the Japanese beetle. U. S. Dep't. of Agr. Bur. of Entomol. and Plant Quar. Bull. E-829 (1951).

FLETCHER, D. W., and W. B. BOLLEN: The effects of aldrin on soil micro-organisms and some of their activities related to soil fertility. Applied Microbiol. **2**, 349 (1956).

FORD, R. E.: Determination of dieldrin deposits on sprayed mud surfaces. Nature **186**; 994 (1960).

FOSTER, A. C., and V. R. BOSWELL: Some effects of insecticide spray accumulations in soil on crop plants. U. S. Dep't. of Agr. Tech. Bull. 1149, p. 36 (1956).

FOX, C. J. S., D. CHISHOLM, and D. K. R. STEWART: Effect of consecutive treatments of aldrin and heptachlor on residues in rutabagas and carrots and on certain soil arthropods and yield. Canad. J. Plant Sci. 44, 149 (1964).

GALLAHER, P. J., and L. EVANS: Preliminary investigations on the penetration and persistence of DDT under pasture. New Zealand J. Agr. Research **4**, 466 (1961).

GANNON, N., and J. H. BIGGER: The conversion of aldrin and heptachlor to their epoxides in soil. J. Econ. Entomol. **51**, 1 (1958).

GEROLT, P.: Improved persistence of dieldrin deposits on sorptive mud surfaces. Nature **180**, 394 (1951).

— Vapour toxicity of solid insecticides. Nature **183**, 1121 (1954).

— Investigation into the problem of insecticide sorption by soils. Bull. World Health Org. **24**, 577 (1961).

GETZIN, L. W.: The effect of soils upon the efficiency of systemic insecticides with special references to Thimet. Dissertation Abstr. **19**, 625 (1958).

—, and R. K. CHAPMAN: Effect of soils upon the uptake of systemic insecticides by plants. J. Econ. Entomol. **52**, 1160 (1959).

— — The fate of phorate in soils. J. Econ. Entomol. **53**, 47 (1960).

GINSBURG, J. M.: Accumulation of DDT in soils from spray practices. J. Agr. Food Chem. **3**, 322 (1955).

—, and J. P. REED: A survey of DDT accumulation in soils in relation to different crops. J. Econ. Entomol. **47**, 467 (1954).

—, R. S. FILMER, J. P. REED, and A. R. PATERSON: Recovery of parathion, DDT and certain analogs of dichlorodiphenyldichloroethane from treated crop. J. Econ. Entomol. **42**, 602 (1949).

GLASSER, R. F., R. G. BLANK, T. E. DEWEY, B. D. HILTON, and M. M. T. WEIDEN: Occurrence of a toxic non-aldrin residue in carrots grown on aldrin-treated soil. J. Econ. Entomol. **51**, 1038 (1958).

GOULD, E., and E. O. HAMSTEAD: The toxicity of cumulative spray residues in soils. J. Econ. Entomol. **44**, 713 (1951).

GRIFFITHS, D. C.: Persistence of organophosphates used in field trials. Rept. Rothamstead Exp't. Station for 1965 (in press).

GUNTHER, F. A., and R. C. BLINN: Analysis of insecticides and acaricides. p. 143. New York: Interscience 1955.

HADAWAY, A. B., and F. BARLOW: Further studies on the loss of insecticides by absorption into mud and vegetation. Bull. Entomol. Research **40**, 323 (1949).

— — Sorption of solid insecticides by dried mud. Nature **167**, 854 (1951).

— — Studies on aqueous suspensions of insecticides Part III. Factors affecting the persistence of some synthetic insecticides. Bull. Entomol. Research **43**, 281 (1952).

— — The influence of temperature and humidity upon the action of insecticides. II. Temperature during the pretreatment period. Ann. Trop. Med. Parasitol. **51**, 194 (1957 a).

— — The influence of temperature and humidity on the action of insecticides. I. During the post-treatment period. Ann. Trop. Med. Parasitol. **51**, 187 (1957 b).

— — The influence of environmental conditions on the contact toxicity of some insecticide deposits to adult mosquitoes. Bull. Entomol. Research **54**, 329 (1963).

— — A note of the sorption of insecticides on tropical soils. Bull. World Health Org. **30**, 146 (1964).

— —, and J. DUNCAN: The residual action of two organophosphorus compounds and a carbamate on dried muds. Bull. World Health Org. 28, 129 (1963 a).

— — — Some effects of formulations on the initial contact toxicity of insecticides to adult mosquitoes. Bull. World Health Org. 28, 129 (1963 b).

HARRIS, C. I.: Adsorption and desorption of herbicides by soil. Dissertation Abstr. **23**, 2651 (1963).

—, and G. F. WARREN: Weeds, in press, 1965.

HARRIS, C. R.: Factors affecting the volatilization of insecticides from soils. Dissertation Abstr. **22**, 375 (1961).

— Influence of soil moisture on the toxicity of insecticides in a mineral soil to insects. J. Econ. Entomol. **57**, 946 (1964 a).

— Influence of soil type and soil moisture on the toxicity of insecticides in soils to insects. Nature **202**, 724 (1964 b).

—, and E. P. LICHTENSTEIN: Factors affecting the volatilization of insecticides from soils. J. Econ. Entomol. **54**, 1038 (1961).

—, and J. H. MAZUREK: Comparison of toxicity to insects of certain insecticides applied by contact and in the soil. J. Econ. Entomol. **57**, 698 (1964).

HARTLEY, G. S.: Physical factors in pesticide performance. World Rev. Pest Control **2**, 41 (. . . .).

HERNE, D. C., and D. CHISHOLM: Accumulation of DDT in the soil of an Ontario peach orchard. Canad. J. Soil Sci. **38**, 23 (1958).

HETRICK, L. A: Ten years of testing organic insecticides as soil poisons against the eastern subterranean termite. J. Econ. Entomol. **50**. 316 (1957).

HOCKING, K. S.: The practical significance of the sorption of insecticides by soils. Proc. Symp. on Pesticides, p. 40 (1959).

JOHNSON, F. R., and A. M. HILLIS: Fluorescent mineral traces technique to determine fungicide placement in the soil profile. Plant Disease Reporter **42**, 287 (1958).

JOHNSON, H. R.: Soil treatments for subterranean termites. Occ. Paper 152 S. Forest Exp't. Station U. S. Dep't. of Agr., 6pp. (1960).

JONES, L. W.: Stability of DDT and its effect on microbial activity of soil. Soil Sci. **73**, 237 (1952).

— The effects of some pesticides on microbial activity of the soil. Bull. Utah Agr. Exp't. Station, p. 390 (1956).

JURINAK, J. J.: Adsorption of 1,2-dibromo-3-chloropropane vapor by soils. J. Agr. Food Chem. **5**, 598 (1958).

KALKAT, G. S., R. H. DAVIDSON, and C. L. BRASS: The effect of controlled temperature and humidity on the residual life of certain insecticides. J. Econ. Entomol. **54**, 1186 (1961).

KHAMA, S. S.: Effect of chlorinated hydrocarbon insecticides on the soil. Allahabad Farmer **35**, 41 (1961).

KIIGEMAGI, U., H. E. MORRISON, J. E. ROBERTS, and W. B. BOLLEN: Biological and chemical studies on the decline of soil insecticides. J. Econ. Entomol. **51**, 198 (1958).

KIMURA, Y., and V. L. MILLER: Degradation of organomercury fungicides in soil. J. Agr. Food Chem. **12**, 253 (1964).

KINCAID, R. R., F. E. GUTHRIE, R. D. CHISHOLM, L. KOBLIRSKY, F. S. CHAMBERLIN, L. M. MAY, and C. F. ENO: Effects on shadegrown tobacco of certain insecticide residues in the soil. Tobacco New York **151**, 173 (1960).

KIRK, V. M.: A study of the disposition of DDT when used as an insecticide for potatoes. Cornell Univ. Agr. Exp't. Station Mem., p. 312 (1952).

KOBLITSKY, L., and R. D. CHISHOLM: Determination of DDT in soils. J. Assoc. Official Agr. Chemists **32**, 781 (1949).

LICHTENSTEIN, E. P.: The movement of some insecticides in soils. Bull. Entomol. Soc. Amer. **3**, 42 (1957 a).

— DDT accumulation in mid-western orchard and crop soils treated since 1945. J. Econ. Entomol. **50**, 545 (1957 b).

— Movement of insecticides in soils under leaching and non-leaching conditions. J. Econ. Entomol. **51**, 380 (1958).

— Adsorption of some chlorinated hydrocarbon insecticides from soils into various crops. J. Agr. Food Chem. **7**, 43 (1959).

—, and J. T. MEDLER: Persistence of aldrin and heptachlor residues on alfalfa. J. Econ. Entomol. **51**, 222 (1958).

—, L. J. DE PEW, E. L. ESHBAUGH, and J. P. SLEESMAN: Persistence of DDT, aldrin and lindane in some mid-western soils. J. Econ. Entomol. **53**, 136 (1960).

—, C. H. MULLER, G. R. MYRDAL, and K. R. SCHULTZ: Vertical distribution and persistence of insecticidal residues in soils as influenced by mode of application and a cover crop. J. Econ. Entomol. **55**, 215 (1962).

—, G. R. MYRDAL, and K. R. SCHULZ: Effect of formulation and mode of application of aldrin on the loss of aldrin and its epoxide from soils and their translocation into carrots. J. Econ. Entomol. **57**, 133 (1964).

—, and J. B. POLIVKA: Persistence of some chlorinated hydrocarbon insecticides in turf soils. J. Econ. Entomol. **52**, 289 (1959).

LICHTENSTEIN, E. P., and K. R. SCHULZ: Insecticide residues. Colorimetric determinations of heptachlor in soils and some crops. J. Agr. Food Chem. **6**, 848 (1958).

— — Persistence of some chlorinated hydrocarbon insecticides as influenced by soil types, rate of application and temperature. J. Econ. Entomol. **52**, 124 (1959 a).

— — Breakdown of lindane and aldrin in soils. J. Econ. Entomol. **52**, 118 (1959 b).

— — Expoxidation of aldrin and heptachlor in soils as influenced by autoclaving, moisture and soil types. J. Econ. Entomol. **53**, 192 (1960).

— — Effect of soil cultivation, soil surface and water on the persistence of insecticidal residues in soils. J. Econ. Entomol. **54**, 517 (1961).

— — The effects of moisture and microorganisms on the persistence and metabolism of some organophosphorus insecticides in soils with special emphasis on parathion. J. Econ. Entomol. **57**, 618 (1964).

— — Residues of aldrin and heptachlor in soils and their translocation into various crops. J. Agr. Food Chem. **13**, 57 (1965).

— —, and G. T. COWLEY: Inhibition of the conversion of aldrin to dieldrin in soils with methylenedioxyphenyl synergists. J. Econ. Entomol. **56**, 485 (1963).

MACLAGAN, D. S.: Secondary and residual effects of modern insecticides. Scot. Agr. **42**, 81 (1962).

MARTIN, J. P.: Influence of pesticide residues on soil microbiological and chemical properties. Residue Reviews **4**, 92 (1963).

MENN, J. J., G. G. PATCHETT, and G. H. BATCHELDER: The persistence of trithion, an organophosphorus insecticide in soil. J. Econ. Entomol. **53**, 1080 (1960).

MORRISON, H. E., and H. H. CROWELL: Soil insecticide studies in Oregon. J. Econ. Entomol. **45**, 1002 (1952).

MULLA, M. S.: Loss of chlorinated hydrocarbon insecticides from the soil surface in the field. J. Econ. Entomol. **53**, 650 (1960 a).

— Organophosphorus and carbamate insecticides as soil treatments against the eye gnat, *Hippelates collusa* in the laboratory. J. Econ. Entomol. **53**, 1102 (1960 b).

— Effectiveness of granular insecticides against eye gnats and mosquitoes as affected by toxicant concentrations. Mosquito News **20**, 362 (1960 c).

— Control of *Hippelates* gnats with soil treatments using organochlorine insecticides. J. Econ. Entomol. **54**, 636 (1961 a).

— Residual activity of organophosphorus insecticides in soil as tested against the eye gnat, *Hippelates collusa.* J. Econ. Entomol **54**, 865 (1961 b).

— Residues of granular and spray parathion and Baytex in alfalfa hay treated at mosquito larvicidal values. Mosquito News **23**, 150 (1963).

— Effectiveness and residual activity of new compounds in soil against the eye gnat, *Hippelates collusor.* J. Econ. Entomol. **57**, 873 (1964).

—, and H. AXELROD: Efficiency of granulated insecticides influenced by solvents used for impregnation. J. Econ. Entomol. **53**, 938 (1960).

—, M. M. BARNES, and M. J. GARBER: Soil treatments with insecticides for control of the eye gnats, *Hippelates collusa* and *H. hermsi.* J. Econ. Entomol. **53**, 362 (1960).

MURPHY, R. T., W. F. BARTHEL, and C. S. LOFGREN: Residual studies in connection with successive applications of heptachlor for imported fire ant eradication. J. Agr. Food Chem. **10**, 5 (1962).

NAUMANN, K.: Einfluss von pflanzenschutzmittel auf die Bodenmikroflora. Mit. biol. Bund. anst. Berlin **97**, 109 (1959).

NEWMAN, A. S., and C. E. DOWNING: Herbicides and the soil. J. Agr. Food Chem. **6**, 352 (1958).

NICHOLSON, H. P., H. J. WEBB, G. J. LANER, R. E. O'BRIEN, A. R. GRZENDA, and D. W. SHANKLIN: Insecticide contamination in a farm pond. Trans Amer. Fish Soc. **91**, 213 (1962).

OATMAN, E. R., and E. P. LICHTENSTEIN: Control of apple maggot and plum curculio with soil insecticides. J. Econ. Entomol. **54**, 1056 (1961).

PARKER, BRUCE, L., and J. E. DEWEY: Decline of phorate and dimethoate residues in treated soils based on toxicity to *D. melanogaster.* J. Econ. Entomol. **58**, 106 (1965).

PATTERSON, R. S.: Use of phorate and di-syston for potato insect control and a study of factors which influence phorate absorption by plants and loss in the soil. Dissertation Abstr. **23**, 1471 (1962).

PHILLIPS, F. T.: Effect of emulsifiers and organic diluents in soil insecticides and nematicide formulations. Nature **184**, 1512 (1959).

RANDOLPH, N. M., R. D. CHISHOLM, L. KOBLITSKY, and J. C. GAINES: Insecticide residues in certain Texas soils. Texas Agr. Exp't. Station M.P., p. 447 (1960).

REYNOLDS, H. T., and R. L. METCALF: Effect of water solubility and soil moisture upon the plant uptake of granulated systemic insecticides. J. Econ. Entomol. **55**, 2 (1962).

ROBERTS, J. E., R. D. CHISHOLM, and L. KOBLITSKY: Persistence of insecticides in soil and their effects on cotton in Georgia. J. Econ. Entomol. **55**, 153 (1962).

ROBERTS, R. J.: Availability of dieldrin to adult *Blissus leucopterus* and larval *Cyclocephala immaculata* in treated sand loam and muck soils. J. Econ. Entomol. **56**, 781 (1963).

SAN ANTONIO, J. P.: Breakdown of chlorinated insecticides in mushroom compost. Mushroom Sci. **5**, 518 (1962).

SANDS, W. A.: The evaluation of insecticides as soil and mound poisons against termites in agriculture and forestry in West Africa. Bull. Entomol. Research **53**, 179 (1962).

SCHONBERG, J., and B. A. MENDOZA: Persistence of insecticides as affected by fertilizers and their translocation by plants. Boln Soc. Quim. Peru **29**, 159 (1963).

SCHREAD, J. C.: Residual activity of insecticides in control of turf insects. J. Econ. Entomol. **42**, 383 (1949).

SFERRA, P. R: Direct feeding method for bioassay of insecticide residue in soil with *Drosophila.* J. Econ. Entomol. **54**, 822 (1961).

SHAW, W. M., and B. ROBINSON: Pesticide effects in soils on nitrification and plant growth. Soil Sci. **90**, 320 (1950).

SHEETS, T. J., A. S. CRAFTS, and H. R. DREVER: Influence of soil properties on the phytotoxicities of the *s*-triazine herbicides. J. Agr. Food Chem. **10**, 458 (1962).

SHOREY, H. H., W. G. EVANS, R. H. BURRAGE, and G. G. GYRISCO: The residual effect of insecticides applied to meadow and pasture sod for control of European Chafer *(Amphimallon majalis).* J. Econ. Entomol. **51**, 765 (1958).

SMITH, J. V., J. C. STREET, G. R. SHULZ, and L. E. HARRIS: Dieldrin residues on vegetation in irrigated pasture. J. Econ. Entomol. **54**, 1091 (1961).

SMITH, M. S.: Persistence of DDT and BHC in soils. Ann. Applied Biol. **35**, 494 (1948).

STANILAND, L. N: Trials of fluorescent tracers for insecticides in soil. Plant Pathol. **10**, 78 (1961).

STEWART, D. K. R., D. CHISHOLM, and C. R. MACEACHERN: The persistence of certain pesticides in soil and their effect on crop yields. Canad. J. Soil Sci. **40**, 59 (1960).

STRICKLAND, A. H., H. M. BARDNER, and R. A. WAINES: Wireworm damage to Ware potato crops in England and Wales. Plant Pathol. **11**, 93 (1962).

SWANSON, C. L. W., F. C. THORP, and R. B. FRIEND: Adsorption of lindane by soils. Soil Sci. **78**, 379 (1954).

TERRIERE, L. C., and D. W. INGALSBE: Translocation and residual action of soil insecticides. J. Econ. Entomol. **46**, 751 (1953)

THOMPSON, F. B., P. M. BATES, and H. J. TAYLOR: Uptake of DDT from soil by rye-grass *(Lolium perenne).* Proc. 16th New Zealand Weed Control Conf., p. 114 (1963).

THURSTON, R: The effect of some soil characteristics on DDT phytotoxicity. J. Econ. Entomol. **46**, 545 (1953).

TURNER, N. J., M. E. CORDEN, and R. A. YOUNG: Decomposition of vapam in soil. Phytopathol. **52**, 756 (1962).

TURNIPSEED, S. G., and J. K. REED: The rate of leaching of dieldrin from attapulgite clay granules. J. Econ. Entomol. **56**, 410 (1963).

VAN TIEL, N: Field performance of dieldrin/resin wettable powders on sorptive mud surface. Bull. World Health Org. **24**, 593 (1961).

VERONA, O., and G. PICCI: Intorno all'azione esercitata dagli insectticidi sistemici sulla mikroflora del terreno. Agr. Ital. **52**, 61 (1952); through Abstr. Soils and Fertilizers **16**, 36 (1952).

WADE, P.: Soil fumigation. I. The sorption of ethylene dibromide by soils. J. Sci. Food Agr. **5**, 184 (1954).

— Soil fumigation. III. The sorption of ethylene dibromide by soils at low moisture contents. J. Sci. Food Agr. **6**, 1 (1955).

WEIDHAAS, D. E., M. C. BARMAN, and C. H. SCHMIDT: Loss of parathion and DDT to soil from aqueous dispersions and vermiculite granules. J. Econ. Entomol. **54**, 175 (1961).

WESTLAKE, W. E.: The effect of new insecticides on the soil. Proc. 46th Ann. Meet. Washington State Hort. Assoc. p. 169 (1950).

WHEATLEY, G. A.: The assessment and persistence of residues of organochlorine insecticides in soils and their uptake by crops. Ann. Applied Biol. **55**, 325 (1965 a).

— The behavior and rates of loss of organochlorine insecticides in soil. Brit. Soc. Soil. Sci., unpublished talk (1965 b).

—, and J. A. HARDMAN: The bioassay of residues of insecticides in soil. Ann. Applied Biol. 48, 423 (1959).

— — Entomology Report. Bioassay of insecticide residues in soils. Rep't. Nat. Veg. Research Station, Wellesbourne for 1959, p. 38 (1960).

— — Entomology Report. Bioassay of insecticide residues in soils. Rep't. Nat. Veg. Research Station, Wellesbourne for 1961, p. 52, 1962).

— —, and A. H. STRICKLAND: Residues of chlorinated hydrocarbon insecticides in some farm soils in England. Plant Pathol. **11**, 81 (1962).

—, D. W. WRIGHT, and J. A. HARDMAN: The retreatment of soils with dieldrin for the control of carrot fly. Plant Pathol. **9**, 146 (1960).

WIESE, I. H.: Some biological studies on the inactivation of insecticides by various soil types. S. African J. Agr. Sci.. **7**, 823 (1964).

WILKINSON, A. T. S., D. G. FINLAYSON, and H. V. MORLEY: Toxic residues in soil 9 years after treatment with aldrin and heptachlor. Science **143**, 681 (1964).

WITOWSKI, O. O.: Determination of insecticide residues in plant and soil by biological assay. Poznan Inst. Ochrony Roslin. **B17**, 157 (1962).

WOODWELL, G. M: The persistence of DDT in a forest soil. Forest Sci. **7**, 194 (1961).

—, and F. T. MARTIN: Persistence of DDT in soils of heavily sprayed forest stands. Science **145**, 481 (1964).

WRIGHT, W. L: Factors influencing the herbicidal activity of trifluralin in the soil. Dissertation Abstr. **25**, 718 (1964).

YOUNG, W. R., and W. A. RAWLINS: The persistence of heptachlor in soils. J. Econ. Entomol. **51**, 11 (1958).

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